

MEAT THE TRUTH

How and why meat consumption is a major public health concern
A review of the evidence

By Dr Justine Butler, Senior Health Researcher, Viva!Health



Meat the Truth

How and why meat consumption is a major public health concern
A review of the evidence

By: Dr Justine Butler
Senior Health Researcher, Viva!Health

Published by: Viva!Health, 8 York Court, Wilder Street, Bristol BS2 8QH

T: 0117 944 1000

E: health@viva.org.uk

W: www.vivahealth.org.uk

©Viva!Health 2017

Registered charity 1037486

ISBN 978-0-9571873-5-1



Viva!Health is the health and nutrition arm of the charity, Viva!, and promotes human health through the promotion of a vegan diet. Viva!Health monitors and interprets research that links diet to health – explaining in simple terms how what we eat affects us, in both positive and negative ways. Viva!Health communicates this information to the media, the public, health professionals, schools and food manufacturers so providing accurate information on which to make informed choices.

CONTENTS

Abbreviations	4	Bugs, superbugs, BSE, bird flu and the	
Introduction	5	horsemeat scandal – what risk now?	71
Red meat, processed meat and white meat	7	Food poisoning	71
Red meat	7	Superbugs	75
Processed meat	7	Viruses	76
White meat	7	BSE	78
Fish	7	Bird flu	79
The undesirable components of meat	9	Horsemeat and beyond	81
Saturated fat, cholesterol and controversy – the sat fat spat!	9	Evolution and the Paleo diet myth	83
The truth about saturated fat, animal protein and cholesterol	12	Wheat-eaters or meat-eaters?	83
Haem iron – too much of a good thing?	14	The Paleo diet myth	83
Salt and sodium	16	Meat made us smart is a dumb idea	83
Caution – hazardous chemicals!	18	Environment	84
NOCs	18	Nutrition – what’s in meat and where else can I get it?	
HCAs	18	Fat	86
PAHs	19	Protein	87
Carnitine	19	Iron	90
Arsenic	20	Zinc	93
The health effects of meat	21	Selenium	94
Meat and death – mortality figures	21	Phosphorus	95
Cancer	26	Vitamin B2 (riboflavin)	96
Breast cancer	32	Vitamin B3 (niacin)	96
Prostate cancer	35	Vitamin B6 (pyridoxine)	96
Lung cancer	39	Vitamin B12	97
Bowel cancer	42	Who eats meat? Comparing carnivores	100
Kidney cancer	45	Global intakes	100
Pancreatic cancer and lymphoma	46	UK meat consumption – going down!	101
Stomach cancer	47	UK meat supply	103
Cardiovascular disease	49	The meat-free market is thriving	105
Obesity and weight gain	54	Conclusion	106
Bone health	58	References	116
The acid-alkaline hypothesis	59		
Diabetes	63		
Type 1 diabetes (insulin-dependent or juvenile-onset diabetes)	63		
Type 2 diabetes	64		
Fertility	70		

ABBREVIATIONS

AGEs	Advanced glycation end products	LRNI	Lower reference nutrient intake
BaP	Benzo[a]pyrene	MelQx	2-amino-3,8-dimethylimidazo (4,5-f)quinoxaline
BMI	Body mass index		
BSE	Bovine spongiform encephalopathy	MRSA	Methicillin-resistant <i>Staphylococcus aureus</i>
COMA	Committee on Medical Aspects of Food and Nutrition Policy	NDNS	National Diet and Nutrition Survey
CVD	Cardiovascular disease	NOC	N-nitroso compounds
Defra	Department for Environment Food and Rural Affairs	NOF	National Obesity Forum
DiMelQx	2-amino-3,4,8-trimethylimidazo (4,5-f)quinoxaline	PAH	Polycyclic aromatic hydrocarbons
EVM	The Expert Group on Vitamins and Minerals	PCRM	Physicians Committee for Responsible Medicine
FAOSTAT	The Food and Agriculture Organisation Corporate Statistical Database	PhIP	2-Amino-1-methyl-6- phenylimidazo[4,5-b]pyridine
FSA	Food Standards Agency	PSA	Prostate-specific antigen
G3CREC	Third-generation cephalosporin- resistant <i>E. coli</i>	RCC	Renal cell carcinoma
GDP	Gross domestic product	RCTs	Randomised control studies
HCA	Heterocyclic amine	RNI	Reference nutrient intake
HRT	Hormone replacement therapy	SACN	Scientific Advisory Committee on Nutrition
IARC	International Agency for Research on Cancer	TMAO	Trimethylamine-N-oxide
IGF-1	Insulin-like growth factor 1	USDA	United States Department of Agriculture
LA-MRSA CC398	Livestock-associated methicillin- resistant <i>Staphylococcus aureus</i>	WCRF	World Cancer Research Fund
		WHO	World Health Organisation

INTRODUCTION

You don't need to eat meat and it is not good for you. A challenging statement to some, but one backed up by a huge body of scientific evidence. How many studies suggest we need five sausages a day? None! Eating meat increases the risk of all the big killers: heart disease and stroke, diabetes, bowel cancer and other cancers. Meat is one of the main causes of obesity, along with dairy foods. It offers no protection for bone health and the animal protein in meat and dairy is linked to weaker bones. Meat is the main cause of food poisoning. Factory-farming is to blame for the emergence of antibiotic-resistant superbugs. Then there's BSE, bird flu and the horsemeat scandal. It's hard to see why anyone would want to eat meat after becoming aware of the facts.

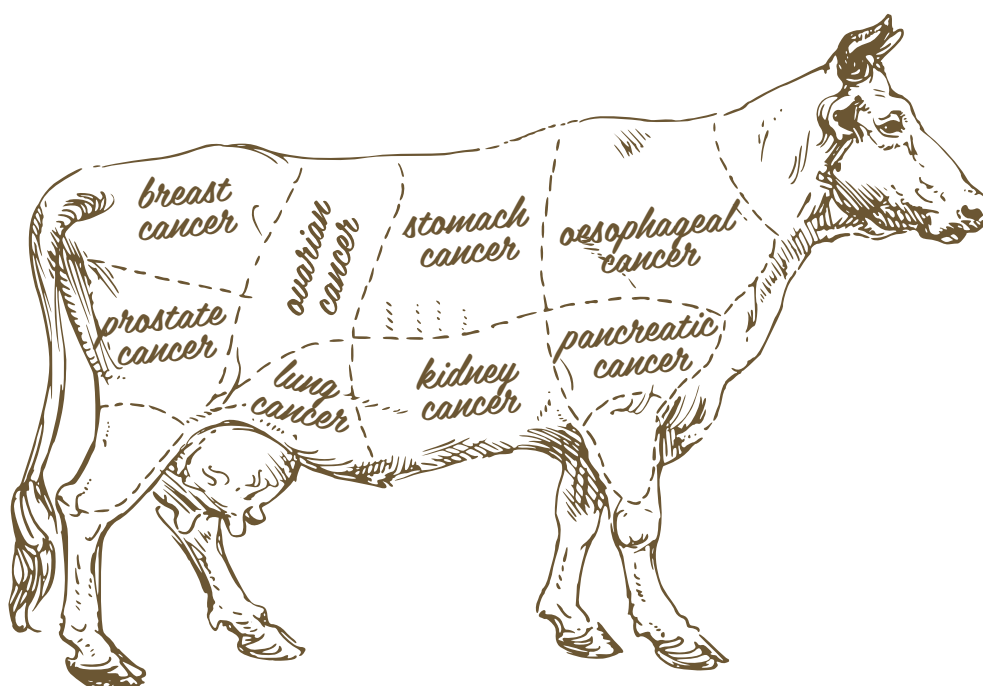
This report investigates the current research looking at all these issues and more. If you are in any doubt then read on, the facts are all here – fully-referenced to the peer-reviewed science from reputable journals.

Heart disease is one of the UK's biggest killers and a leading cause of death worldwide. Type 2 diabetes is rapidly becoming a global epidemic and people who eat meat have a higher risk of developing it. In 2035, the NHS could be spending almost a fifth of its entire budget on treating diabetes. One in every two people born after 1960 will develop cancer at some point in their lives. We are not a healthy nation and diet is largely responsible, and within that, meat plays a

central role. The World Cancer Research Fund (WCRF) says that people should *"Eat mostly foods of plant origin, limit intake of red meat and avoid processed meat."* That means no sausages or bacon – ever. Viva!Health goes one step further and says ***eat no meat ever***. It offers no benefit and it harms health.

The UK Department of Health's advice is based on the links between meat and cancer. The links between meat and heart disease, stroke, diabetes, obesity and other health problems are largely ignored. They say you should limit your intake of red and/or processed meat to no more than 70g a day (or 500g a week). Over a week, if you ate six meals containing meat (so less than one per day) including one full English breakfast, three slices of ham, a quarter pounder burger, spaghetti bolognese, a doner kebab and a Sunday roast, you would have exceeded the government's upper limit by around 100g. The guideline only applies to red and processed meats but most meat-eaters would also consume some white meat (chicken or turkey) pushing up their intake of saturated fat and other harmful substances even further.

Researchers at the University of Cambridge's Institute of Public Health estimated what would happen if meat intake fell and the number of vegetarians doubled. They not only predicted a drop in incidence of heart disease, stroke, diabetes, bowel cancer and other cancers, but also a reduction in greenhouse gas





emissions. The diet that is good for us is also good for the environment. However, there seems to be a reluctance to acknowledge the harmful effects of meat on health and the environment, certainly in terms of changing government policy. This immutable position may finally be getting the push it needs to change because of the links between meat and cancer becoming even more firmly established.

In late 2015, 22 scientists from ten countries met at the International Agency for Research on Cancer (IARC) in Lyon, France, to evaluate the carcinogenicity of the consumption of red meat and processed meat. IARC is the specialised cancer agency of the World Health Organisation (WHO). Their assessments were published on October 26th, 2015, just a few days ahead of World Vegan Day celebrated on November 1st every year.

Their findings made major headlines around the world when it was announced that the WHO declared that eating just 50g of processed meat (less than two slices of bacon) a day increases the risk of bowel cancer by 18 per cent. They also found an increase of 100g of red meat a day increases the risk of bowel cancer by 17 per cent. They also found links between red meat and pancreatic and prostate cancer, and processed meat and stomach cancer.

The meat industry dismissed the report's findings. Norman Bagley, policy director at the Association of Independent Meat Suppliers, said that he believed the industry would continue to thrive (Fortune, 2015). That is interesting as statistics from the Department for

Environment Food and Rural Affairs (Defra) show that UK meat consumption has fallen by 13 per cent since 2007 (Defra, 2015). Within a week of the WHO report being released Bagley said: *"It will not be damaging long-term for the UK meat industry unless they come up with new evidence to show the risk has significantly increased. There is nothing new in this report and nothing to suggest that the risk had changed"* (Fortune, 2015).

However, UK shoppers felt differently. Martin Wood, head of strategic insight-retail at market research company IRI Retail Advantage said: *"While there have been links between certain types of meat and some forms of cancer before, this announcement from a highly respected global body was picked up widely by the media and has had an immediate impact on some people's shopping choices"* (IRI, 2015). IRI said that sales of prepacked sausages were down by 15.7 per cent in the last week of October 2015 compared to the same week in the previous year. In the two weeks following the report, sales of bacon and sausages plummeted 10 per cent (by £3 million) (Gani, 2015). Wood said: *"What we may see here is some people making changes to meat buying, moving away from processed meat to non-processed alternatives..."* (IRI, 2015).

While this may mean some people buying red or white meat or fish instead of processed meats, it also inevitably meant some people reducing or ditching meat altogether. The meat-free market is booming and rising sales suggest that the tide is turning. People are either reducing or dropping meat completely for healthier

options including mock meats (veggie sausages and burgers) as well as dishes made with wholegrains, pulses (peas, beans and lentils) and nuts and seeds.

The research shows that the rising number of vegetarians and vegans not only suffer less illnesses but live longer, healthier lives. Forty per cent of adults in the UK (most men over 60 and women over 65) will soon be advised to take statins to lower their risk of heart disease – which can be prevented and reversed by going vegan. Going into old age disease-free, fit and healthy is all any of us could hope for. If one simple lifestyle change can help you achieve that, isn't it worth making the change today?

RED MEAT, PROCESSED MEAT AND WHITE MEAT

Red meat is red when raw and does not turn white when it is cooked. Most meat from adult mammals fits these criteria. It is also defined as meat with more than a certain level of myoglobin (an iron-containing protein in muscle). Pork is sometimes considered red if the animal is adult, but white if young (a suckling piglet) and the same applies to sheep – mutton is considered red while the flesh of a young lamb is described by some as white.

Generally, the meat from mammals such as cows and calves, sheep, lamb and pigs is considered red, while chicken, turkey and rabbit meat is considered white. Game birds such as pheasants, geese or ducks are sometimes put in a separate category altogether but for the purposes of this report, will be included in the white meat category.

Red meat

The common definition of red meat includes:

- beef and veal
- pork
- goat
- burgers
- mutton and lamb
- venison
- horse
- mince

Burgers and minced meats do not count as processed meat unless they have been preserved with salt or chemical additives.

Processed meat

Processed meat refers to meat that has been preserved by smoking, curing, salting or adding chemical preservatives such as sodium nitrite. Putting fresh meat

through a mincer does not make it processed meat. In general, processed meat has had something done to it to extend its shelf life or change its taste. Most processed meats contain pork or beef, but they may also contain other red or white meats, offal or meat by-products such as blood.

The common definition of processed meat includes:

- sausages
- ham
- salami
- tinned meat
- beef jerky
- pepperoni
- bacon
- hot dogs or frankfurters
- pâtés
- chorizo
- corned beef

White meat

White meat refers to meat that is light-coloured before and after cooking. Chicken is the archetypal white meat. However, the term is generally used for all poultry, even if the meat is technically red, as in duck. Some consider the meat of milk-fed calves (white veal) white too.

Rather more contentious is the inclusion of pork, which may turn white when cooked, but is also from a mammal. From 1987-2011 the US National Pork Board ran the advertising slogan "*Pork. The Other White Meat*" in an effort to suggest that pork was healthier than other red meats. However, neither the UK Food Standards Agency (FSA) nor the United States Department of Agriculture (USDA) classifies pork as a white meat.

White or pale meat can include poultry and game birds as well as rabbit:

- chicken
- duck
- pheasant
- turkey
- goose
- rabbit

Fish

Fish is also referred to by some people as white meat. This may refer to both white fish (which is white before and after cooking) and fatty fish such as salmon and tuna (salmon remains pink when cooked but tuna turns white). Seafood (invertebrates) may also be referred to as white meat, particularly if white before or after cooking, such as shrimp, oysters and scallops. For the purposes of this report, fish will be excluded. See Viva!Health's *Fish Report* for information on the detrimental health effects of fish and advice on where to get healthy omega-3 fats: www.vivahealth.org.uk/go-fish-free.

CURRENT GOVERNMENT ADVICE

Current advice, issued by the Department of Health in 2011, says: *“Adults who eat more than 90g of red and processed meat a day should reduce their intake to 70g a day”* (NHS Choices, 2015). This advice is based on a 2010 report by the Scientific Advisory Committee on Nutrition (SACN) called *Iron and Health*, (SACN, 2010). SACN is a committee of independent nutrition experts which advises the government on diet and nutrition.

This advice followed on directly from a joint report from the WCRF and the AICR published in 2007. The WCRF/AICR's Second Expert Report, *Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective* set out recommendations for cancer prevention based on a comprehensive review of the scientific evidence in this area (WCRF/AICR, 2007). The report involved hundreds of experts in specialist working groups, who reviewed all the evidence to date about the link between food, nutrition, weight gain, being overweight and physical activity and the risk of cancer. The report presented their findings and recommendations to decrease cancer risk.

The report stated that the public health goal for the **population average** consumption of red meat should be **no more than 300g a week, very little if any of which to be processed**. The personal recommendation, for individuals who eat meat, was set at **less than 500g a week, with little, if any, processed meat**. This suggests that it is best to avoid all processed meat, which means no bacon or ham, ever. However, this message has largely become lost as government advice tends to lump red and processed meat together as one category.

One week's intake of red and processed meat amounting to less than 500g could include:

- One cooked breakfast (two sausages and two thin-cut rashers of bacon): 130g
- One slice of ham: 23g
- A quarter pounder beef burger: 78g
- An eight ounce beef steak, grilled: 163g
- One portion of Sunday roast (three thin-cut slices of roast lamb, beef or pork each about the size of half a slice of sliced bread): 90g

Many meat-eaters consume significantly more than this; some might eat this amount in just two days!





THE UNDESIRABLE COMPONENTS OF MEAT

SATURATED FAT, CHOLESTEROL AND CONTROVERSY – THE SAT FAT SPAT!

Every few months the headlines say ‘*butter is back*’, ‘*saturated fat is good for you*’ or ‘*calorie counting is over*’. Then all too often, the study in question is challenged or discredited, but the damage is done...

In March 2014, national headlines declared “*Saturated fat ‘ISN’T bad for your heart’*” (Hope, 2014), suggesting it is perfectly safe to gorge on butter, cheese and sausages. The study, published in the *Annals of Internal Medicine* pooled the results of 72 selected studies looking at the link between fat and heart disease and

suggested that saturated fat may not lead to heart disease after all (Chowdhury *et al.*, 2014). However, the analysis of two of the six studies of omega-6 fats was incorrect – they got the numbers wrong. Results from other relevant studies were not included. They did not mention a review of prospective studies in which a significant reduction in the risk of heart disease was found in relation to polyunsaturated fat. In this analysis, switching from saturated to polyunsaturated fat lowered the risk of heart disease – that was not discussed. They also failed to point out that most of the monounsaturated fat in the studies they looked at was from red meat and dairy sources; so their findings would not necessarily apply to fats from nuts, olive oil and other plant sources. Therefore their conclusions regarding the type of fat being unimportant were wrong. Professor Walter Willett, chair of the Department of Nutrition at Harvard School of Public Health (the Heisenberg of Nutrition!) said: “...*this meta-*

MEAT THE TRUTH

analysis contains multiple serious errors and omissions, the study conclusions are misleading and should be disregarded" (Willett *et al.*, 2014). Two months later a correction was published pointing out the errors in this study (Chowdhury *et al.*, 2014a).

In February 2015, the online journal *Open Heart* published a study suggesting UK dietary guidelines were based on shaky evidence (Harcombe *et al.*, 2015). The authors of the study said that they didn't know what evidence was available when the guidelines were written so they just selected six randomised control studies (RCTs) published before 1983 – all were conducted in men, most of whom already had heart disease. Their results suggested that advice to control saturated fat intake did not affect deaths from heart disease among this small number of unwell men. But that doesn't mean the recommendations are wrong...

Headlines declared *"Butter ISN'T bad for you after all"* (Hope, 2015). However, the study was slammed by experts; Victoria Taylor at the British Heart Foundation said: *"guidance in the UK is based on a consensus of the evidence available"* (British Heart Foundation, 2015) and Professor Christine Williams, Professor of Human Nutrition at the University of Reading, said: *"The claim that guidelines on dietary fat introduced in the 1970s and 80s were not based on good scientific evidence is misguided and potentially dangerous"* (Tran, 2015).

The concept of this study was scientifically flawed. Why they chose to look at old studies retrospectively is unclear – surely the guidelines we have now would be better-challenged if the current research presented a different picture? It doesn't, there is a substantial body of current evidence supporting the case that saturated fat is bad for health. Even the accompanying *Open Heart* editorial questioned the validity of the study; cardiologist Rahul Bahl, of the Royal Berkshire NHS Foundation Trust, wrote: *"Public policies generally do not require RCT evidence, so to advocate their withdrawal here on the basis of the absence of such evidence seems unusual"*.

It turns out, the lead author, pro-fat campaigner Zoe Harcombe, runs a diet-club and has published books on her version of good nutrition. Harcombe advised people to *'ignore public health advice'* in a presentation she wrote for the Weston A Price Foundation, a fringe US organisation which claims to be dedicated to promoting good nutrition by restoring nutrient dense animal products to the diet (Harcombe, 2011). They receive substantial financial support from the animal farming industry and claim that saturated animal fat is essential for good health and that animal fat intake and high cholesterol levels have no link with heart disease. This contradicts what all leading health advisory bodies in the world say.

A month after the publication of the *Open Heart* study, Pascal Meier, Editor-in-Chief of *Open Heart*, issued the following statement: *"Following comments from readers, and post-publication discussions within our editorial team, the authors of this paper were asked to update their competing interest statement. The potential competing interests relate to one of the authors of the article, Mrs Harcombe, who has previously published books on diet and nutrition, and is also a co-director of a company that gives dietary advice (The Harcombe Diet Co.) and co-director of a publishing company (Columbus Publishing) that publishes books on diet and nutrition."*

Meier said *"In this case, Open Heart feels that the books and companies with which Mrs Harcombe has been involved should have been declared. From our point of view, a competing interest exists when professional judgement concerning a primary interest (such as validity of research) could potentially be influenced by a secondary interest (such as financial gain)"* (Harcombe *et al.*, 2015a).

In September 2015, US dietary guidelines were targeted in a similar way in a study in the *British Medical Journal* which questioned the link between saturated fat and heart disease (Teicholz, 2015). The author was journalist Nina Teicholz, who also wrote a book called *The Big Fat Surprise: Why Butter, Meat and Cheese Belong in a Healthy Diet*. In an open letter to the *British Medical Journal*, Dr David Katz of Yale University School of Medicine expressed his concern at them publishing a journalist's commentary as if it were authoritative. Katz said *"It is, in a word, absurd and testimony to the breakdown in integrity where science and media come together"* (Katz, 2015).



Errors in the study were identified and the next month the following correction was published: *"This Feature by Nina Teicholz stated that when the guidelines advisory committee started its work in 2012 there had been several prominent papers, including a meta-analysis and two major reviews (one systematic), that failed to confirm an association between saturated fats and heart disease. This statement did not aptly reflect the findings of the more authoritative of these reviews, by Hooper et al., which found that saturated fats had an effect on cardiovascular events but failed to confirm an effect on cardiovascular mortality."* (Teicholz, 2015a).

There is a substantial body of evidence supporting the case that saturated fat is bad for health. The study by Hooper et al. referred to in the *British Medical Journal's* correction is a Cochrane Review. These are systematic reviews of primary research in human healthcare and health policy; they are internationally recognised as the highest standard in evidence-based healthcare – often referred to as the gold standard. Hooper's review analysed 48 studies including over 65,000 participants. It was found that reducing saturated fat (but not total fat) intake reduced the risk of heart attack and stroke by 14 per cent (Hooper et al., 2012). That was just by reducing saturated fat – not cutting out meat and dairy. The results could have been even more impressive if that had happened!

In May 2016, the National Obesity Forum (NOF) published a report saying that avoiding butter, cream and cheese is actually fueling obesity epidemic and that official advice on low-fat diets and cholesterol is wrong (National Obesity Forum, 2015). National headlines declared that: *"Official advice on low-fat diet and cholesterol is wrong"* (Guardian, 2015). Dr Aseem Malhotra, one of the authors of the report, described Public Health England's new Eatwell Guide as a metabolic timebomb! He says: *"We must urgently change the message to the public to reverse obesity and Type 2 diabetes. Eat fat to get slim, don't fear fat; fat is your friend."*

The NOF report describes calorie counting as a red herring, as calories from different foods have different effects on the human body. It emphasises the fact that in spite of dietary guidelines, the number of people with obesity and type 2 diabetes is rising. But this doesn't prove that the guidelines are wrong; it merely shows that people are ignoring them. Public Health England said *"the report is irresponsible and misleads the public"* (Public Health England, 2016). Professor Susan Jebb from Oxford University condemned the

report as *"non-rigorous and irresponsible"* (Bodkin, 2016) questioning their motives as they accept funding from the pharmaceutical industry (they are supported by GlaxoSmithKline, Sanofi-Aventis, Roche Products as well as the British Meat Nutrition Education Services and weight loss business LighterLife UK). At the time of writing, four of the seven board members of NOF had resigned, while a fifth was considering his options.

Also at the time of writing, the *British Medical Journal* published a review questioning the validity of the cholesterol hypothesis in the elderly (Ravnskov et al., 2016). This led to more sensationalist headlines saying: *"High cholesterol 'does not cause heart disease'"* (Bodkin, 2016a). You'd think they would tread more carefully after last time. A bit of digging around revealed that four of the authors have written books challenging the idea that cholesterol is bad for you and nine are members of a group called The International Network of Cholesterol Skeptics, who oppose the idea that animal fat and cholesterol play a role in heart disease. Dr David Nunan, a senior research fellow at the Nuffield Department of Primary Care Health Sciences at the University of Oxford, performed a post-publication critical appraisal of this review and said: *"Given that the authors failed to account for significant confounding as well as the methodological weaknesses of both the review and its included studies, the results of this review have limited validity and should be interpreted with caution. At this time it would not be responsible, or evidence-based, for policy decisions to be made based on the results of this study"* (Nunan, 2016).

The pro-fat and animal farming lobbies have their sights fixed on sugar as the villain of the piece. It may well be that focusing on saturated fat as the primary dietary villain for heart disease has distracted from the risks posed by sugar, but replacing one villain with another is not helpful. Unfortunately the pro-fat crusade will continue because the meat and dairy industry has money and influence. However, it seems the government will not be swayed on this and the scientific community is well-prepared to stand their ground. It's a shame these few, flawed studies received so much media attention.

THE TRUTH ABOUT SATURATED FAT, ANIMAL PROTEIN AND CHOLESTEROL

There is much evidence linking heart disease and stroke to poor diets, including the high consumption of saturated animal fats, animal protein, salt and refined carbohydrates, and the low consumption of fruits and vegetables. A certain amount of cholesterol in the blood (not the diet) is essential for good health, but high cholesterol levels increase the risk of heart disease and stroke.

Contrary to popular belief, most of our cholesterol does not come from the diet but is produced within the body by the liver. Only a small amount of our cholesterol (15-20 per cent) comes from the diet, and only if you eat animal foods. Cholesterol is found only in animal foods and is particularly concentrated in organ meats and eggs. Even high-fat plant foods (such as avocados, nuts and seeds), contain no cholesterol whatsoever, so a vegan diet is cholesterol-free. We have no actual dietary requirement for cholesterol, in other words we do not need to eat foods that contain it as the liver can make as much as we need. However, there is no mechanism limiting the amount of cholesterol produced by the liver and it can rise to unhealthy levels.

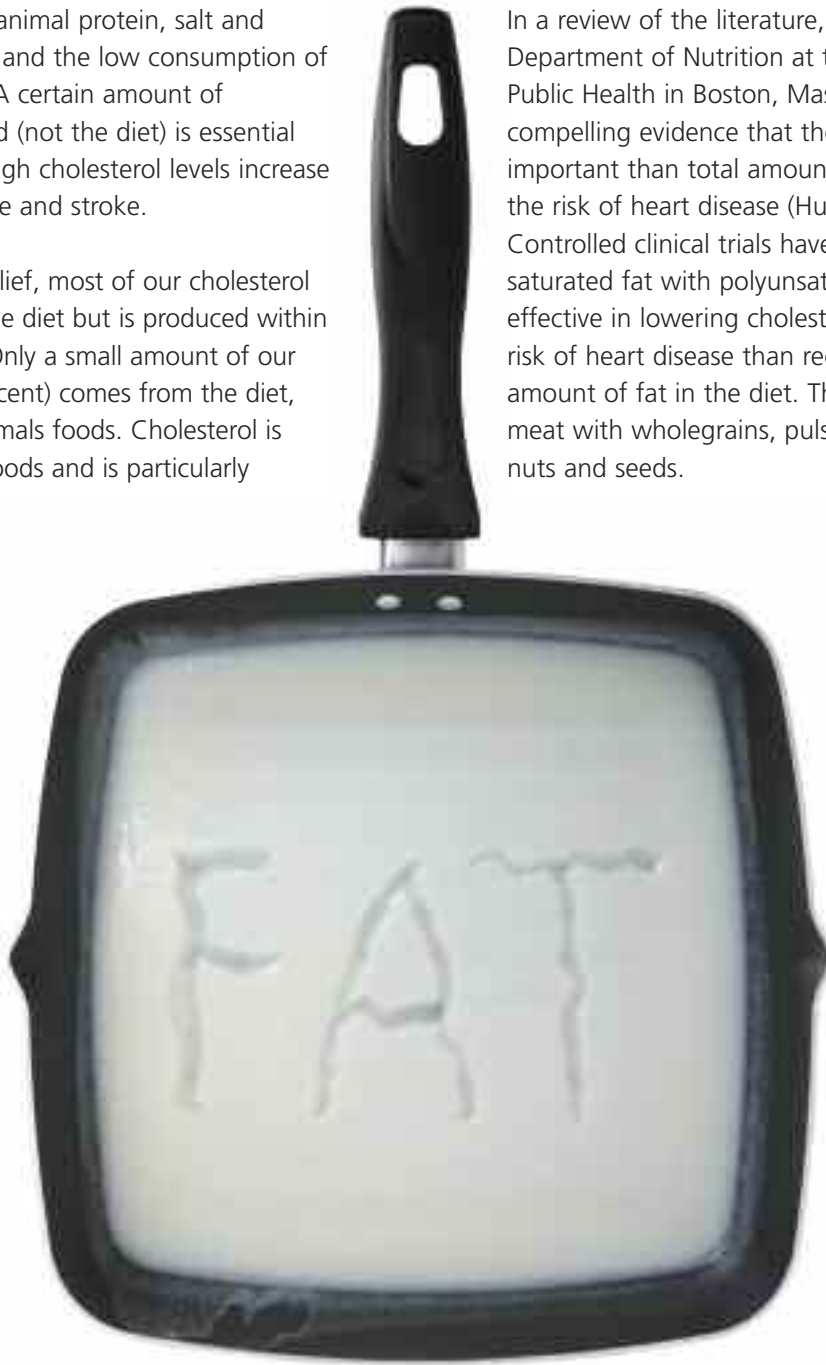
What causes high cholesterol production in the liver? The answer lies in the types of foods we eat: diets high in animal protein and saturated fat have been shown to increase cholesterol. The cholesterol-raising effect of

saturated fat and the links with heart disease are well-documented. All major health organisations (WHO, American Dietetic Association, Dietitians of Canada, British Dietetic Association, American Heart Association, British Heart Foundation, World Heart Federation, British National Health Service, US Food and Drug Administration and European Food Safety Authority) agree that **saturated fat is a risk factor for heart disease**.

In a review of the literature, researchers from the Department of Nutrition at the Harvard School of Public Health in Boston, Massachusetts, found compelling evidence that the types of fat are more important than total amount of fat in determining the risk of heart disease (Hu *et al.*, 2001). Controlled clinical trials have shown that replacing saturated fat with polyunsaturated fat is more effective in lowering cholesterol and reducing the risk of heart disease than reducing the total amount of fat in the diet. This means replacing meat with wholegrains, pulses, fruit, vegetables, nuts and seeds.

It is now widely accepted that diets high in animal fats are unhealthy and that reducing saturated fat helps lower the risk of heart disease. The UK government recommends avoiding or cutting down on fatty foods. The foods high in saturated fat they list include: meat pies, sausages and fatty cuts of meat, butter, ghee, lard, cream, hard cheese, cakes and biscuits and foods containing coconut or palm oil. Trans fats (found naturally

in low levels in meat and dairy products and foods containing hydrogenated vegetable oil, including processed foods such as biscuits, cakes, fast food, pastry, margarines and spreads) can also raise cholesterol levels. However, most people in the UK



don't eat a lot of trans fats as many supermarkets in the UK have removed hydrogenated vegetable oil from their products.

The Oxford Vegetarian Study, a large study comparing 6,000 vegetarians (and vegans) with 5,000 non-vegetarians found that vegans had the lowest cholesterol levels, vegetarians and fish-eaters had intermediate or similar values and meat-eaters had the highest levels (Appleby *et al.*, 1999).

Meat and cheese consumption were positively linked to higher cholesterol and dietary fibre was linked to lower levels. After 12 years of follow-up, the risk of death from heart disease was linked to saturated animal fat and cholesterol in the diet.

A subsequent review of the literature comparing the health of Western vegetarians to non-vegetarians found that vegetarians had lower cholesterol levels and were less likely to die from heart disease than meat-eaters. The authors said that **the widespread adoption of a vegetarian diet could prevent approximately 40,000 deaths from heart disease in Britain each year** (Key *et al.*, 1999).

A Cochrane Review, often referred to as the gold standard in science, found that reducing saturated animal fat, but not total fat intake, reduced the risk of heart attack and stroke substantially. The authors said that lifestyle advice to those at high risk of heart disease and stroke (and probably also to those with a lower risk), should continue to include the permanent reduction of saturated fat and partial replacement by healthier unsaturated fats. There is much evidence that

replacing saturated fat with polyunsaturated fat or monounsaturated fat in the form of olive oil, nuts, seeds and other plant oils can reduce the risk of heart disease and stroke.

In his extensive *China Study*, Professor T. Colin Campbell observed that high intakes of animal protein are linked to heart disease (Campbell and Campbell, 2005). Campbell attributes this to the cholesterol-raising effect of animal protein. Conversely, he noted that plant protein lowers cholesterol. Campbell cites the low rates of heart disease in the southwest Chinese provinces of Sichuan and Guizhou where between 1973-1975 not one single person died of it before the age of 64 among 246,000 men and 181,000 women. Campbell suggests these figures reflect the important protective role of low blood cholesterol levels seen in rural China.

It's not just that animal protein increases cholesterol, but that plant protein can lower it too.

One particular plant protein has been shown to be particularly effective in lowering cholesterol – soya protein. The most popular theory is that soya protein reduces cholesterol production in the liver by increasing the removal of LDL 'bad' cholesterol (Lovati *et al.*, 2000).

Cholesterol rates in the UK are among the highest in the world. High levels are caused by saturated fat, trans fat and animal protein from meat, eggs and dairy products. All reputable health bodies recommend eating less of these and more foods containing unsaturated fats such as avocados, nuts, seeds, plant-based oils such as olive oil and spreads.



HAEM IRON – TOO MUCH OF A GOOD THING?

Not all iron is created equal! There are two types of iron in food:

Haem Iron is found in animal tissue. It is a component of haemoglobin (oxygen-carrying protein in the blood) and myoglobin (oxygen-carrying molecules in muscle) and makes up around half the iron found in red meat, poultry and fish.

Non-Haem Iron makes up the other half of the iron in animal tissue and all of the iron found in plant foods, dairy foods (which contain a very small amount) and eggs.

The haem iron content of different types of meat varies widely. The highest levels are seen in red and processed meat (hamburgers, steak, pork and ham) and the lowest levels in chicken (Cross *et al.*, 2012). The haem iron content of red meat is higher simply because it contains more myoglobin, transporting oxygen to muscles. Put simply, muscles used more frequently are darker (although there are also differences between species). So while chicken meat contains less haem than beef, pork or lamb, the haem content of chicken's thigh meat is considerably higher than breast meat.

Non-haem iron makes up the other half of the iron in animal tissue and all of the iron found in plant foods, dairy foods (which contain a very small amount) and eggs. Most of the iron in the diet is non-haem; the 2003 National Diet and Nutrition Survey (NDNS) found that non-haem iron contributed 94 per cent of dietary iron in the UK (Henderson *et al.*, 2003). The other six per cent of dietary iron was haem iron deriving mainly from the haemoglobin and myoglobin of meat (Johnston *et al.*, 2007).

Haem iron intake in the UK is in decline. A study looking at data from the Medical Research Council National Survey of Health and Development (also known as the 1946 British Birth Cohort) investigated haem and non-haem iron intakes among adults in the UK over a period of 17 years from 1982-1999. Results showed a steady decline in haem iron intake from beef, pork and lamb but a rise in haem iron from poultry (Johnston *et al.*, 2007). This corresponds with Defra's data showing how the consumption of sheep, cattle and pigs in the UK has declined in recent years while the consumption of poultry has increased. The authors suggest that modern farming methods may also be contributing to the falling

level of haem iron in meat as there is evidence that intensive farming results in a lower mineral content of meat (Purchas and Busboom, 2005).

The vast majority of iron in the diet is non-haem iron with just five to 10 per cent coming from haem iron in diets containing meat (Beck *et al.*, 2014). Both types of iron are absorbed in the small intestine, but by different mechanisms. Haem iron is more bioavailable or easily absorbed into the body; 20-30 per cent of haem iron eaten is absorbed while one to 10 per cent of non-haem iron absorbed (Beck *et al.*, 2014). Non-haem iron absorption is more variable because it is subject to a range of influences. It can be influenced by iron status (how much iron there already is in the body) and various dietary factors such as tannins, phytates, calcium, polyphenols and dietary fibre (Johnston *et al.*, 2007). The dietary factors that help or hinder iron absorption are discussed on page 92.

Despite considerable research, the precise mechanism by which haem iron is absorbed remains unclear. It is thought that it is transported across the intestinal membrane (gut wall) into the blood intact where the iron component is removed to enter a common pool of iron along with non-haem iron (Geissler and Powers, 2005). Absorption of haem iron is less affected by iron status or enhancers and inhibitors in the diet than non-haem iron. So haem iron is more easily absorbed and is associated with significantly higher iron stores in the body (Fleming *et al.*, 2002; Liu *et al.*, 2003). The more meat (haem iron) you eat, the higher the level of iron in your blood will be. However, a high absorption rate and/or high iron stores are not necessarily a good thing as the body has no mechanism for disposing of excess iron. In other words, iron from plant foods is more beneficial to the body because its absorption remains safely regulated, whereas, iron from animal sources can accumulate to levels which could be harmful.

Too much iron can have many detrimental effects activating: oxidative responsive transcription factors (molecules in the body which can switch on cancer genes), pro-inflammatory cytokines (lack of regulation of these is linked to atherosclerosis and cancer) and iron-induced hypoxia signalling – a classical feature of cancer (WCRF/AICR, 2007). There are detrimental effects relating to haem iron specifically which are not affected by non-haem iron (Cross *et al.*, 2012). The suggested mechanisms underlying the harmful effects of haem iron are based on its ability to contribute to the formation of **free radicals** and N-nitroso compounds or **NOCs** (Bastide *et al.*, 2011).

Free radicals are unstable and highly reactive molecules capable of damaging biologically important molecules such as DNA, proteins, carbohydrates and lipids (Lobo *et al.*, 2010). The end result can be the generation of toxic molecules that are capable of promoting cancer.

NOCs are produced in the stomach and bowel of people who eat large amounts of red meat (WCRF/AICR, 2007). NOCs may damage cellular DNA, potentially leading to certain cancers. Many NOCs are carcinogens and there is supporting evidence for a role of NOCs in gastric, oesophageal, nasopharyngeal and colon cancers (Loh *et al.*, 2011).

The main sources of external exposure to NOCs are cigarette smoke and diet, particularly processed (nitrite-treated) meats. After eating red and processed meat, the large intestine is rich in nitrogen residues from protein metabolism. These residues may be used by colonic bacteria to produce NOCs. One study of 21 healthy male volunteers, found levels of NOCs on a high red meat diet (420g per day) were significantly higher than on a low meat diet (60g per day) but were no higher when an equivalent amount of vegetable protein was eaten (Cross *et al.*, 2003).

An 8mg supplement of haem iron also increased faecal NOCs compared with the low meat diet, but 35mg ferrous (non-haem) iron had no effect. The finding that haem iron behaves differently suggests that in addition to the bacterial NOC generation that occurs in the gut, the haem iron also contributes to a chemical catalysis (the speeding up of a chemical reaction) that is responsible for the dose-dependent effect of red meat on increasing NOC production in the gut. In other words, haem iron act in multiple ways to drive up NOC production. The authors concluded that if the NOC formed endogenously in the intestine, as a result of haem consumption, is shown to be mutagenic or carcinogenic, this might explain the association between red meat consumption and large bowel cancer risk (Cross *et al.*, 2003).

There exists a widespread conviction that the iron from red meat is somehow superior to that from plant foods. This is what is still written in some nutrition and dietetic textbooks. Numerous studies now show that people with high iron stores suffer a higher incidence of heart disease, diabetes and certain cancers.



HAEMOCHROMATOSIS

Iron overload can be caused by an inherited genetic condition called hereditary haemochromatosis, which affects one in 250 people of Northern European descent (Burke *et al.*, 2000). Normally the liver stores a small amount of iron to make new red blood cells. If you absorb excessive amounts of iron (as some people with this condition do) it builds up in the liver and is deposited in other tissues and organs. Iron overload may cause uncomfortable symptoms such as nausea, abdominal pain, constipation and joint pain. It can also lead to liver damage, heart failure and diabetes. Men are more at risk from haemochromatosis because women regularly lose blood, and therefore iron, during menstruation. This condition illustrates how you can have too much of a good thing.

SALT AND SODIUM

A diet that is high in salt can cause raised blood pressure, which increases the risk of stroke and premature death from cardiovascular disease (CVD). High blood pressure can have no symptoms and it is estimated that in England about one in every three people who have high blood pressure are unaware that they do (NHS Choices, 2014). Cutting down on salt lowers blood pressure, which in turn lowers the risk of CVD.

In 1994, the Committee on Medical Aspects of Food and Nutrition Policy (COMA) recommended a reduction in the daily average salt intake of the population from 9g to 6g because of its role in CVD. A report was prepared in response to the request, for a risk assessment of salt by the Scientific Advisory Committee on Nutrition (SACN). In the 2003 SACN report, the evidence published since 1994 was appraised and the recommendation for a reduction in the population average intake of salt to 6g per day for adults was accepted and targets were also set for children (SACN, 2003). The UK daily recommended maximum intake of salt is:

- 1-3 years – 2g salt a day (0.8g sodium)
- 4-6 years – 3g salt a day (1.2g sodium)
- 7-10 years – 5g salt a day (2g sodium)
- 11 years and above – 6g salt a day (2.4g sodium)

(SACN, 2003).

You might think that salty snacks (like crisps and salted peanuts) contribute most salt to the diets of many. However, the SACN report found that cereals and cereal products (which include bread, breakfast cereals, biscuits, cakes and pastries) provided nearly 40 per cent of average intake and meat and meat products contributed over a fifth of the average salt intake (SACN, 2003).

The 2014 NDNS also found that cereals and cereal products were the largest contributor to sodium intake from food for all age groups (providing 31-37 per cent of which 16-19 per cent came from bread). Meat and meat products were also the second largest contributor for all age groups, providing 19-28 per cent of salt intake from food. Milk and milk products contributed 18 per cent for children aged 1.5-3 and 8-11 per cent for other age groups (Bates *et al.*, 2014).

The findings of these reports were mirrored in those of a recent study of salt intake among children in South London that found savoury snacks only contributed five per cent of the salt intake compared to 36 per cent from cereals (15 per cent of which was from bread) and 18 per cent from meat and meat products (Marrero *et al.*, 2014).

Cutting back on added salt (in cooking and at the table) can help, but 75 per cent of the salt we eat is already in everyday foods such as bread, breakfast cereal and ready meals (NHS Choices, 2014). To really cut down, you need at least to become aware of the salt that is already in the foods you buy and switch to lower-salt or salt-free varieties. You can use the nutrition labels on pre-packed food to see how much salt it contains. Preferably, switch to a wholefood, varied vegan diet to effectively manage your salt intake.

Salt is also called sodium chloride. Sometimes, food labels only give the figure for sodium. The NHS Choices website provides a simple way to work out how much salt you are eating from the sodium figure:

Salt = sodium x 2.5

So, adults should eat no more than 2.4g of sodium per day, which is equal to 6g of salt.

Some foods (such as bread, breakfast cereals), can contribute a lot of salt to our diet, not because these foods are high in salt but because we eat a lot of them. Other foods are high in salt because of the way they are made.

High-salt foods include:

- anchovies
- cheese
- ham
- pickles
- salami
- salt fish
- smoked meat and fish
- yeast extract
- bacon
- gravy granules
- olives
- prawns
- salted and dry-roasted nuts
- soya sauce
- stock cubes

Foods that can be high in salt include:

- bread products such as crumpets, bagels and ciabatta
- ready meals
- sandwiches
- tomato ketchup, mayonnaise, other sauces and condiments
- pasta sauces
- crisps
- pizza
- soup
- sausages
- breakfast cereals
- Soluble vitamin supplements or painkillers

Source: NHS Choices, 2014.

Populations eating mainly vegetarian diets have lower blood pressure than those eating meat and epidemiologic findings suggest that eating fruits and vegetables lowers blood pressure (Sacks *et al.*, 1999).

A recent study investigated the effects of a healthy vegan diet for seven days on the risk factors for CVD

and type 2 diabetes (McDougall *et al.*, 2014). Over 1,600 patients were recruited for the study. Those taking medication for high blood pressure and/or diabetes reduced or stopped taking their medication for the duration of the study to reduce the risk of a huge drop in blood pressure and glucose. They followed a low-fat, high-carbohydrate, moderate-sodium, vegan diet. The kitchen staff used minimal salt, mostly in the form of soya sauce, when preparing meals. The basic meal plan provided roughly 1g of sodium (equivalent to 2.5g salt) per day. However, saltshakers were provided at mealtimes and participants were allowed to use as much table salt as they wanted. The results were remarkable, in just seven days there were statistically significant decreases in cholesterol, weight and blood pressure – despite the fact that many of the participants had stopped taking their medication. The authors suggest that this type of diet could provide a model for a cost-effective therapy to offer patients commonly seen in medical practices and health centres today.

Another study comparing nutritional quality of different diets (vegan, vegetarian, fish- and meat-eaters), found that vegans consumed the least salt; less than half the amount the meat-eaters consumed (Clarys *et al.*, 2014). The authors of this study concluded that the indexing system, which estimates the overall diet quality based on different aspects of healthful dietary models, indicated consistently the vegan diet as the healthiest one. A wordy way of saying a vegan diet is best!

In general, a vegan diet contains less salt or sodium and this is another reason why it is the healthiest diet.



CAUTION – HAZARDOUS CHEMICALS!

“...high consumption of processed foods may lead to an increased intake of saturated fats, cholesterol, salt, nitrite, haem iron, polycyclic aromatic hydrocarbons, and, depending upon the chosen food preparation method, also heterocyclic amines.”
(Rohrmann and Linseisen, 2015).



Among potential dietary carcinogens, there are three groups of compounds that are not naturally present in meat but may develop during preservation or cooking:

- N-nitroso-compounds (NOCs) are formed during food preservation
- Polycyclic aromatic hydrocarbons (PAHs) are formed during cooking
- Heterocyclic amines (HCAs) are formed during cooking

It is difficult to estimate exactly how much of these harmful compounds are present in cooked and processed meat and therefore what a person's intake might be, as they are not naturally present in meat and are not included in standard food composition tables (Jakszyn *et al.*, 2004).

NOCs

NOCs are a class of potent human carcinogens. Haem iron from meat can act as a catalyst in the production of NOCs in the gut. In addition to that, nitrates and nitrites found in processed meats (and smoked cheeses) contribute to the endogenous formation of NOCs in the gut (Abid *et al.*, 2014). Nitrite is used to preserve processed meat as it is extremely toxic to bacteria and serves, for example, as an antibacterial agent against the potential tummy bug *Clostridium botulinum* (Cross and Sinha, 2005). Nitrite also produces the characteristic red-pink colour of cured meats and gives cured meat its flavour. The difference in colour seen in raw cured meat like salami (dark pink, almost red) and cooked cured meat like frankfurters (paler pink) is due to the presence of nitrite. The red colour of raw cured meat is caused by nitrosylmyoglobin (a complex formed by the reaction of myoglobin with nitric oxide), but cooking denatures

globin which then detaches from haem, yielding a pink mononitrosylheme complex, the colour of cooked cured meat (Santarelli *et al.*, 2008).

NOCs are also detectable in food, especially in nitrite-preserved meat such as bacon and ham. One study found the faecal content of NOCs was 60 times higher in volunteers given cured meat than in volunteers given a vegetarian diet (Joosen *et al.*, 2009). While some of this is due to endogenous production of NOCs in the gut that occurs in the presence of haem iron, nitrates and nitrites from red meat, some of the NOCs present are there simply because certain meats contain them.

HCAS

HCAs are formed from the reaction between creatine or creatinine (a compound found in the muscle of meat and fish), amino acids and sugars (found in muscle meats) at high cooking temperatures (Jägerstad and Skog, 1991). The three most abundant HCAs in food are:

- 2-Amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP)
- 2-amino-3,8-dimethylimidazo(4,5-f)quinoxaline (MeIQx)
- 2-amino-3,4,8-trimethylimidazo(4,5-f)quinoxaline (DiMeIQx)

(Abid *et al.*, 2014).

Meat dripping is the animal fat produced either from the fatty unusable parts of a carcass or drained off from a roast piece of meat. It is similar to lard or tallow. Dripping and gravy made from dripping contains considerable amounts of HCAs (Rohrmann *et al.*, 2015). In parts of Yorkshire, when spread on bread, it is known as a 'mucky fat' sandwich!

PAHS

PAHs are formed when meat is cooked over an open flame; the breakdown of fats in meat at high temperatures (pyrolysis) generates PAHs, which then become deposited on the meat (Phillips, 1999). Seven PAH compounds are designated by the US Environmental Protection Agency as probable human carcinogens, including benzo(a)pyrene or BaP (Abid *et al.*, 2014).

Of course these hazardous chemicals are just some of the many different reasons why meat is detrimental to health (evidence for the role of NOCs, HCAs and PAHs in different diseases is provided in later chapters).

CARNITINE

The name carnitine comes from the Latin for flesh (*carnus*) because it was first isolated from meat. Carnitine is found in nearly all cells of the body, it is produced from the amino acids lysine and methionine. It is involved in fat metabolism, transporting fatty acids (fuel) into mitochondria (the cell's powerhouse) to produce energy. This is why carnitine is sold as supplements and used in energy drinks.

We have no dietary requirement for carnitine; we can make all we need in the liver and kidneys, but so do animals – and that is where the problem lies. All meat, including chicken, contains carnitine. Meat is not the only dietary source of carnitine; cow's milk, cheese, wholegrain products and asparagus also contain it, but in much smaller concentrations, and as stated, it is a common ingredient of energy drinks.

Many studies show that vegetarians and vegans have a lower risk of CVD compared to meat-eaters with the inferred mechanism being a lower intake of dietary cholesterol and saturated fat (Fraser, 2009; Key *et al.*, 1999). However, a recent study suggested an additional explanation as to why meat intake may be related to mortality or early death (Koeth *et al.*, 2013). This research suggests that people who eat meat have a different type of bacteria in their gut compared to

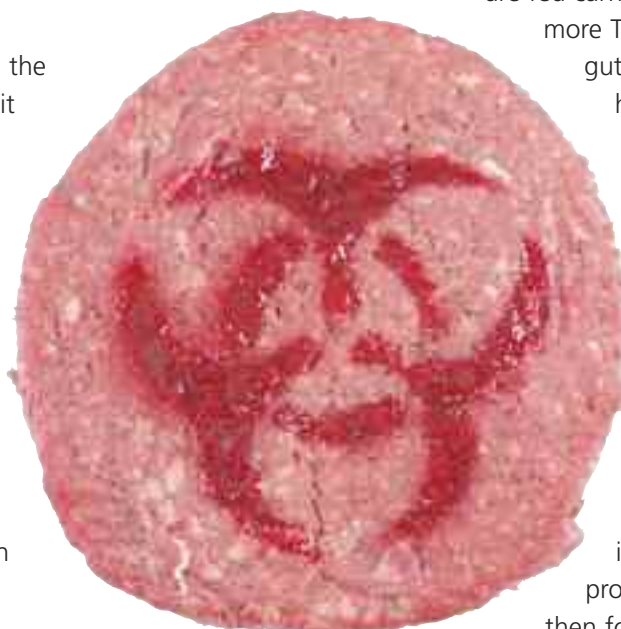
vegetarians and vegans which breaks down carnitine to produce a substance linked to CVD.

It is known that certain gut bacteria use carnitine as an energy source, breaking it down and producing a waste product called trimethylamine. The liver converts this into another substance called trimethylamine-N-oxide (TMAO), which is excreted in urine. TMAO alters cholesterol metabolism in the gut, liver and artery walls increasing the build-up of cholesterol and decreasing the removal of cholesterol from artery walls – a double whammy! This causes a build-up of plaques on the artery walls that can lead to atherosclerosis and CVD.

The study by Koeth *et al.* found that meat-eaters produce higher levels of TMAO than vegans after they are fed carnitine, suggesting that they have more TMAO-producing bacteria in their gut. An unexpected finding was the huge difference in TMAO production between meat-eaters and vegans both before and after the 'carnitine challenge tests' (where carnitine was provided in two forms: a 250mg supplement and an eight ounce sirloin steak). One vegan participant who took the test showed a lower level of TMAO to start with – indicating a lower capacity to produce this harmful substance, then following ingestion of carnitine showed no increase either. On the other hand the meat-eaters responded with a spike in TMAO production. Results suggest that vegans don't have TMAO-producing bacteria in their gut.

This makes sense as people who eat a lot of fibre appear to have more fibre-consuming bacteria. In other words – the diet we choose determines the type of gut bacteria we end up with. Reports have shown significant differences in microbial populations among vegans and meat-eaters (Cordain *et al.*, 2005). More than 100 trillion microorganisms live in our gut, mouth, skin and other mucosal surfaces of our bodies. These microbes offer numerous beneficial functions and understanding our 'microbiome' is fast-becoming the next big thing in nutrition and health research.

The authors of the carnitine study suggest that the safety of carnitine supplementation should be





examined, because consuming high amounts of carnitine may under some conditions prime our gut bacteria with enhanced capacity to produce TMAO and potentially promote atherosclerosis (Koeth *et al.*, 2013).

ARSENIC

You might associate arsenic with the 1944 who-done-it film *Arsenic and Old Lace*, in which affluent elderly gentlemen are poisoned by sweet old ladies. The same year the film was released, the US Food and Drug Administration (FDA) approved the use of the drug 3-nitro-4-hydroxyphenylarsonic acid (Roxarsone) as an animal feed additive. At that time animal drugs containing arsenic were routinely used in animal feed for chickens, turkeys and pigs (but were most commonly used in broiler chickens). In poultry, they were used for growth promotion, feed efficiency and improved pigmentation; they were also approved in combination with other drugs to prevent coccidiosis. Roxarsone was used in poultry feed to kill parasites and promote growth.

In 2011, an FDA study found that chickens that had eaten Roxarsone had higher levels of arsenic in their livers than chickens that had not eaten it (FDA, 2015). Due to technical difficulties, the FDA did not test any other part of the chickens' bodies. However, a more recent study found a range of arsenic species (compounds containing arsenic) in the breast meat of chickens fed Roxarsone (Liu *et al.*, 2016).

Following publication of the FDA study, Pfizer Inc voluntarily suspended sale of Roxarsone. Had they not stopped sales, the FDA probably would have banned the product since arsenic is a known carcinogen. In 2015, the FDA withdrew approval of using Roxarsone in animal feeds. It is banned in the European Union; however, it continues to be legally used in many other countries where its presence in chicken manure significantly enhances the uptake of arsenic species by vegetables (Huang *et al.*, 2014; Yao *et al.*, 2013).

This is just one of the better-known examples of the many different chemicals that factory-farmed animals have been force-fed up until slaughter in recent years.

THE HEALTH EFFECTS OF MEAT

MEAT AND DEATH – MORTALITY FIGURES

Is there a relationship between what you eat and how long you live? Is bacon as bad for you as smoking? What should you be eating if you want a long and healthy life?

Deaths from circulatory diseases, cancers and neoplasms, respiratory diseases, digestive diseases, mental and behavioural disorders, diseases of the nervous system, genitourinary disease, endocrine, nutritional and metabolic diseases, infectious and parasitic diseases or dying as the result of an accident are collectively referred to as *all-cause mortality* – all the deaths that occur in a population, regardless of the cause. It is measured in population studies and clinical trials as an indicator of the safety or danger of a specific activity, such as eating meat.

Several studies suggest that vegetarians and vegans have a greater longevity compared with meat-eaters. Substantial evidence from epidemiological studies shows that meat intake, particularly red and processed meat, is associated with an increased risk of premature death from diabetes, heart disease, stroke and certain cancers (Pan *et al.*, 2012). Numerous large prospective studies have also found that meat intake is linked to all-cause mortality. However, the relationship between meat, disease and mortality has been challenged by the meat industry.

In 2012, a large-scale study from Harvard School of Public Health reported how red meat consumption is associated with an increased risk of early death (Pan *et al.*, 2012). The study warned that each daily serving of red and processed meat increases the risk of dying prematurely. This study was widely reported in the media with headlines such as: “Red meat death study”, “Will red meat kill you?” and “Red meat ‘kills’”.

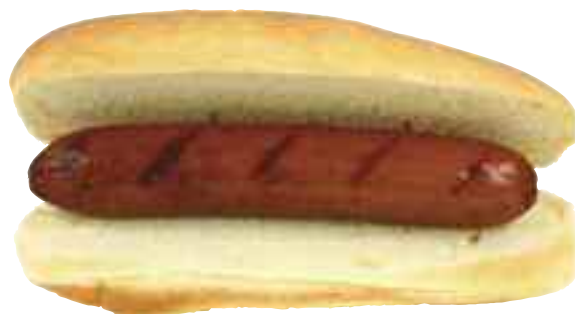
The research analysed data from two large US studies including 37,698 men from the Health Professionals Follow-up Study (1986-2008) and 83,644 women from the Nurses’ Health Study (1980-2008), giving a total of 121,342 participants. Over 22 years of follow-up, there were 23,926 deaths (including 5,910 from CVD and 9,464 from cancer). Results showed that a higher intake of both red and processed meat was associated with a significantly increased risk of all-cause mortality.

Results showed:



One 85g serving of red meat a day (equivalent to the size of a deck of cards) was associated with a:

- 13 per cent increased risk of early death
- 18 per cent increased risk of death from CVD
- 10 per cent increased risk of death from cancer



One serving of processed meat (one hot dog or two slices of bacon) a day was associated with a:

- 20 per cent increased risk of early death
- 21 per cent increased risk of death from CVD
- 16 per cent increased risk of death from cancer

Source: Pan *et al.*, 2012.

MEAT THE TRUTH

The authors estimated that substituting one serving of red meat a day for a healthier source of protein (they suggested poultry, fish, nuts, pulses or wholegrains) would reduce the risk of early death by between 7-19 per cent. Furthermore they suggested that 9.3 per cent of early deaths in men and 7.6 per cent of early deaths in women could be prevented if they consumed just under half a serving (around 42g) a day of red meat.

The authors suggest that saturated fat and haem iron in red meat might partly explain the increased risk of CVD, while the presence of sodium and nitrites might explain the additional risk associated with processed meats. They also point out that some compounds generated in red meat by high temperature cooking are potential carcinogens. This was the first large-scale prospective longitudinal study showing that consumption of red and processed meat is associated with an increased risk of early death.

The findings of this large study were challenged by Dr Carrie Ruxton from the Meat Advisory Panel, a body funded by the meat industry. Ruxton said: *"This US study looked at associations between high intakes of red meat and risk of mortality, finding a positive association between the two. However, the study was observational, not controlled, and so cannot be used to determine cause and effect. The authors' conclusion that swapping a portion of red meat for poultry or fish each week may lower mortality risk was based only on a theoretical model."*

Whereas Dr Rachel Thompson, Head of Research Interpretation at the WCRF said: *"This study strengthens the body of evidence which shows a link between red meat and chronic diseases such as cancer and heart disease. The research itself seems solid and is based on two large scale cohort studies monitored over a long period of time."*

Seventh-day Adventists are a conservative religious group that includes more than 13 million members worldwide. The Adventist church promotes a healthy lifestyle; members are expected to be non-smokers, not drink alcohol and are encouraged to eat a vegetarian diet. Studies among Adventists in California have shown the advantages of a meat-free diet (Butler *et al.*, 2008). Adventists adhere with these recommendations to varying degrees which makes them an ideal group for prospective studies; seeing the relationship between diet and disease over time either within the Adventist group, or comparing them with the general population.

The Adventist Mortality Study (1960-1966) and the first Adventist Health Study (AHS-1) (1974-1988) showed that vegetarian Adventists had a lower risk for most cancers, CVD and diabetes. They also lived longer compared with the general California population; vegetarian Adventist women lived 4.4 years and men 7.3 years longer (Fraser, 2003).

More recently, in an attempt to resolve uncertainty in the literature, the Adventist Health Study 2 (AHS-2) set out to evaluate the link between vegetarian and vegan diets, disease and death (Orlich *et al.*, 2013). The study included 73,308 participants amongst which there were 2,570 deaths during a period of almost six years. Results showed that, compared to meat-eaters, all-cause mortality was 12 per cent lower in vegetarians and 15 per cent lower in vegans. The authors concluded that the evidence that vegetarian and vegan diets may be associated with a lower risk of death should be considered carefully by individuals as they make dietary choices and by those offering dietary guidance. Health professionals, take note!

Another large-scale systematic review of nine prospective studies (from the US, China and Europe including a total of 1,330,352 individuals and 137,376 deaths), was conducted to quantify the association between red and processed meat and all-cause mortality (Larsson and Orsini, 2014). Results also found that a high consumption of red meat, in particular processed meat, is associated with an increased risk of early death. Those with the highest intake of processed meat and total red meat had a 23 per cent and 29 per cent increased risk of death respectively, compared to those with the lowest.

What this study showed was that for both total meat and processed meat intake, the increase in risk of death rises steeply at the lower end of consumption then continues to rise in a dose-response fashion as intake increases. In other words, the increase in risk of early death associated with red and processed meat is not linear; the risk increases rapidly at relatively low intakes. The authors said that the results from their analysis add to the increasing evidence that consumption of red and processed meat should be limited. They point out how this research falls in line with the WCRF report which says that the public health recommendation with regard to cancer risk is to eat no more than 500g per week of red meat like beef, pork and lamb and to avoid processed meats such as ham, bacon, salami, hot dogs and sausages (WCRF/AICR, 2007).

Another study, this time from the US, investigated meat intake and mortality among 322,263 men and 223,390 women enrolled in the National Institutes of Health (NIH)-AARP (formerly known as the American Association of Retired Persons) Diet and Health Study (Sinha *et al.*, 2009). During 10 years of follow-up, 47,976 men and 23,276 women died. Again, results showed that those who ate the most red and processed meat had an increased risk of all-cause mortality, compared to those who ate the least. The authors of this study estimated that 11 per cent of deaths in men and 16 per cent of deaths in women could be prevented if they reduced their red meat intake to the lowest level of intake in this study (14.4g per 1,000 calories).

The UK government says men need around 2,500 calories a day and women around 2,000. The average daily intake of calories per person in the US in 2009 was 3,652 (FAOSTAT, 2015). Based on these figures, people in the NIH-AARP study eating 53g of red meat per day could still be classed in the lowest intake group! In case you are wondering, the highest consumers were eating up to 320g of red and processed meat per day, almost five times what the Department of Health advises people to limit their intake to.

The authors list the potential culprits linking meat to mortality: NOCs, HCAs and PAHs, haem iron (increasing oxidative damage and increasing the formation of NOCs) and saturated fat (associated with breast and bowel cancer). Curiously, in contrast, higher white meat consumption was associated with a small decrease in mortality (more on this to follow).

Studies of UK vegetarians have yielded mixed results. The EPIC-Oxford study was established in the 1990s and included about 65,000 participants. In 2009, they compared mortality rates in vegetarians and meat-eaters among 64,234 participants of which 2,965 died (Key *et al.*, 2009). Results showed no statistically significant differences between vegetarians and meat-eaters. There are a number of possible explanations why the vegetarians did not show a reduced risk. The EPIC-Oxford cohort is not representative of the wider UK population. Recruited through vegetarian societies, health food shops and magazines, they appear to be more health-conscious; they smoke less, weigh less and the meat-eaters consume less meat. The death rates of all participants in this study were much lower than average for the UK and this may have obscured differences in the wider population between



MEAT THE TRUTH

vegetarians and meat-eaters. Also, although fruit and vegetable intake was higher among vegetarians than meat-eaters, the difference was small. The authors say that the relatively low meat intake and high fruit and vegetable intake of the meat-eaters in this UK cohort may have reduced the chance of observing lower mortality rates among vegetarians. Also, this study grouped vegetarians and vegans together so the animal protein (dairy products and eggs) consumed by vegetarians may have masked the harmful effects of meat. Later work has revealed that compared to US Adventist vegetarians, UK vegetarians consume relatively more animal protein and less fibre and vitamin C (Appleby *et al.*, 2016; Orlich *et al.*, 2013).

In 2013, a larger EPIC study investigating the association between meat consumption and early death, combined data from 10 European countries (Rohrmann *et al.*, 2013). There were 448,568 individuals and 26,344 deaths (5,556 died of CVD, 9,861 of cancer, 1,068 of respiratory diseases, 715 of digestive tract diseases and 9,144 of other causes). Initial results showed that high-meat consumers (more than 160g of red meat per day) were 14 per cent more likely to die early than low-consumers (eating 10.0-19.9g per day) and for processed meat, the figure was even higher at 44 per cent. There was no association with mortality and the consumption of poultry (see more on this below). After correction for measurement error (a statistical procedure designed to strengthen the reliability and precision of the results), the risk of early death remained significantly higher only for processed meat; 50g a day of processed meat was associated with an 18 per cent increased risk of early death.

Again, the authors list the usual suspects pointing out how compared to red meat, processed meats tend to contain more saturated fat, cholesterol, salt and additives (some of which are carcinogenic or precursors to carcinogenic substances: NOCs, HCAs and PAHs). They point out that haem iron also links meat consumption to cardiovascular risk but that this is not limited to processed meat. They estimated that three per cent of premature deaths each year could be prevented if people ate less than 20g of processed meat a day and concluded that as processed meat consumption is a modifiable risk factor, public health guidelines should include specific advice on lowering processed meat consumption.

The EPIC-Oxford cohort was revisited more recently in a study comparing mortality rates in vegetarians and meat-eaters. This study also included data from another

prospective study; the Oxford Vegetarian Study. Taken together, they included a total of 60,310 people and 5,294 deaths before the age of 90 (Appleby *et al.*, 2016). Like the 2009 EPIC-Oxford study, the results suggested that UK vegetarians and vegans have a comparable risk of all-cause mortality to meat-eaters. However, when analysing deaths before the age of 75 and excluding participants known to have changed diet group at least once during follow-up, vegetarians and vegans had a 14 per cent lower all-cause mortality than meat-eaters. The decision to switch diet during the study may have occurred in response to the onset of illness, for example, people warned about weight or blood sugar levels going vegetarian or vegan to avoid diabetes and/or heart disease. So it seems logical that excluding individuals who switched diet may give a truer picture of how beneficial a meat-free diet can be. This may not match the substantive evidence seen in US studies, but it shows there is a beneficial effect associated with avoiding meat.

Another possible reason for the discrepancy between the US and UK studies may be the *type* of vegetarian diet followed. The authors suggest that the perceived healthfulness of vegetarian diets is a major motivating factor for Adventist vegetarians, whereas UK vegetarians may be motivated by other factors that are not health-related (animal welfare and/or the environment), possibly making them less likely to adopt a healthy vegetarian diet (Appleby *et al.*, 2016). Furthermore, the amount of animal protein (dairy products and eggs) in the UK vegetarian diet was significantly higher than that in the vegetarian Adventists' diet.

The authors of AHS-2 agree that the lack of similar findings in UK vegetarians is interesting and suggest this difference deserves careful study. In both cohorts, the meat-eaters are a relatively healthy reference group, healthier than the general population. In both cohorts, the vegetarians (especially the vegans) consume less saturated fat and more fibre. However, UK vegetarians and US Adventist vegetarians appear to eat somewhat differently. For instance, the Adventist vegetarians consumed even more fibre and vitamin C than those in the EPIC-Oxford cohort (average dietary fibre in EPIC-Oxford vegans was 27.7g per day in men and 26.4g per day in women compared with 45.6g per day in men and 47.3g per day in women in AHS-2 vegans; the average vitamin C in EPIC-Oxford vegans was 125mg per day in men and 143mg per day in women compared with 224mg per day in men and 250 mg per day in women in AHS-2 vegans).

People choosing to follow a vegetarian or vegan diet for ethical or environmental reasons may eat differently from those who choose vegetarian or vegan diets primarily for health reasons. It seems likely that the potential health benefits of a vegetarian diet is a major motivator of Adventist vegetarians. The authors of AHS-2 say that other large cohort studies have linked increased red and processed meat consumption to higher mortality and that their findings build on this work by demonstrating reduced mortality in those consuming low-meat diets. Notably, the findings of AHS-2 are similar to those of previous North American Adventist cohort studies, demonstrating a consistent association over several decades and replicating prior results in a population with great geographic and ethnic diversity (Orlich *et al.*, 2013).

A different approach was taken by researchers from the Oxford Martin Programme on the Future of Food (Springmann *et al.*, 2016). They estimated both the health and climate change impacts of a global move toward a more plant-based diet. The researchers from the University of Oxford predicted what the effects of four different types of diet would be by 2050. The four different diets were as follows:

- No change
- One that follows health guidelines for meat, fruit and vegetables
- A vegetarian diet
- A vegan diet

Results suggest that dietary change across the globe could have multiple health, environmental and economic benefits; more than five million premature deaths could be avoided globally by 2050 if health guidelines on meat consumption were followed. On a vegetarian diet, the figure rises to more than seven million, **on a vegan diet more than eight million premature deaths could be avoided**. The report suggests the monetary value of health improvements could be comparable with, and possibly larger than, the environmental benefits of the avoided damages from climate change. Lead author, Dr Marco Springmann said: *"Unbalanced diets are responsible for the greatest health burden around the world"*.

The scientific consensus is that both red and processed meats increase the risk of a premature death. Research regarding white meat is somewhat varied, it may decrease mortality when it replaces red meat in the diet but that may just mean that it is just not quite as bad for you as red meat. Avoidance of red and processed

meats (and poultry) and a diet rich in plant-based whole foods including fruits, vegetables, wholegrains, nuts and pulses remains a sound, evidence-based recommendation (Fields *et al.*, 2016).

SUMMARY

- Meat-eating increases the risk of early death. Substituting just one serving of red meat a day for nuts, pulses or wholegrains could reduce the risk by almost 20 per cent. Avoiding meat completely would be even more beneficial. The saturated fat, cholesterol and haem iron in red meat and salt and nitrites in processed meat are implicated along with carcinogenic compounds produced by high temperature cooking.
- US Adventist vegetarians have lower rates of cancer, CVD and diabetes and live longer than the general population. One Adventist study found that vegetarians and vegans were 12 and 15 per cent, respectively, less likely to die early. A UK study found vegetarians and vegans combined had a 14 per cent lower risk.
- Other large-scale studies in the US, China and Europe have reported similar findings. The risk of early death increases rapidly at low intakes; just 50g a day of processed meat a day increases the risk of early death by 18 per cent. Swapping meat for cheese and eggs isn't the answer as these contain harmful animal protein, saturated fat, cholesterol and hormones too.
- On a global scale, more than five million premature deaths could be avoided by 2050 if guidelines on meat consumption were followed. On a vegetarian diet, the figure rises to more than seven million, on a vegan diet more than eight million. Taken together, the research shows a low-fat, high-fibre, vegan diet is the most protective diet against early death.

CANCER

Every year, there are more than 10 million new cases of cancer around the world; they are not spread evenly across the globe (Cross *et al.*, 2007). In 2012, the age-standardised incidence rate (how many out of every 100,000 people will have the disease in a given year) for all cancers for men and women across the globe was 182 people per 100,000. In the UK, the figure was 273 per 100,000 (GLOBOCAN, 2012). For men, the rate varied almost seven-fold from 57 per 100,000 in Niger in Western Africa to 374 per 100,000 in Australia, where prostate cancer represents a significant proportion. Variation among women was nearly five-fold, from 70 per 100,000 in the Gambia to 328 per 100,000 in Denmark, where high rates of breast cancer occur. Denmark has been called the cancer capital as the highest cancer rate for men and women together occurs there with 338 people per 100,000 diagnosed in 2012.

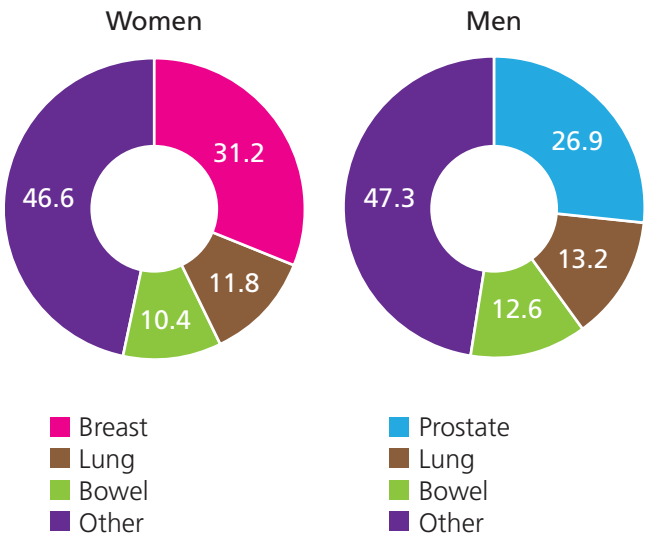
In the UK, one in every two people born after 1960 will be diagnosed with some form of cancer during their lifetime (Ahmad *et al.*, 2015). Although there are more than 200 different types of cancer, the four most common (breast, prostate, lung and bowel cancers) account for more than half of all cancers (NHS Choices, 2014a). Table 1.0 shows the percentages of total cancers these four most common cancers make up.

Table 1.0 Types of cancer registered in the UK in 2014.	
Type of cancer	Percentage of total cancers
Breast*	15.6
Prostate	13.4
Lung	12.6
Bowel	11.5
Other	46.9

Source: Office for National Statistics, 2014.
*46,085 cases in females and 332 in males.

Figure 1.0 shows that three most common cancers account for over half of all cancers in women and men. Lung and bowel cancer make up nearly a quarter of cancers in both genders, while breast and prostate cancer comprise over a quarter of cancers in women and men respectively.

Figure 1.0 Most common cancers in England in 2013 for women and men by percentage.



Source: Bate and Baker, 2015.

Smoking is the most important preventable cause of cancer; responsible for one in four UK cancer deaths and nearly a fifth of all cancer cases (Public Health England, 2016a). Nearly half of all smokers will eventually die from smoking-related diseases. After smoking, poor diet is the most important avoidable cause of cancer.

Countries with relatively high intakes of meat and other animal foods have higher rates of bowel, breast and prostate cancer (Key *et al.*, 2014). Indeed, meat consumption has been recognised as a risk factor for cancer since the early 1900s. A study of cancer rates in different ethnic groups in Chicago from 1900-1907 found that heavy meat-eaters (Germans, Irish and Scandinavians) had higher rates of cancer mortality than pasta-consuming Italians and rice-eating Chinese (Grant, 2014).

Professor T. Colin Campbell, Jacob Gould Schurman Professor Emeritus of Nutritional Biochemistry at Cornell University in the US, is the author of over 300 research papers and co-author of *The China Study* (Campbell and Campbell, 2005), one of America's best-selling books about the impact of diet on our health. His legacy, the China Project, is the most comprehensive study of health and nutrition ever conducted.

The China Project was conceived in 1980-1981 when Dr Chen Junshi (Deputy Director of the Institute of Nutrition and Food Hygiene at the Chinese Academy of Preventive Medicine) visited Campbell's laboratory at the Division of Nutritional Sciences at Cornell University in Ithaca, New York in the US. They were later joined by Professor Richard Peto from the University of Oxford in

the UK and Dr. Li Junyao from the China Cancer Institute at the Chinese Academy of Medical Sciences in China. The project demonstrated important relationships between dietary patterns and cancer risk across different countries (Campbell and Junshi, 1994). It involved 65 Chinese counties and focused on their diets and health.

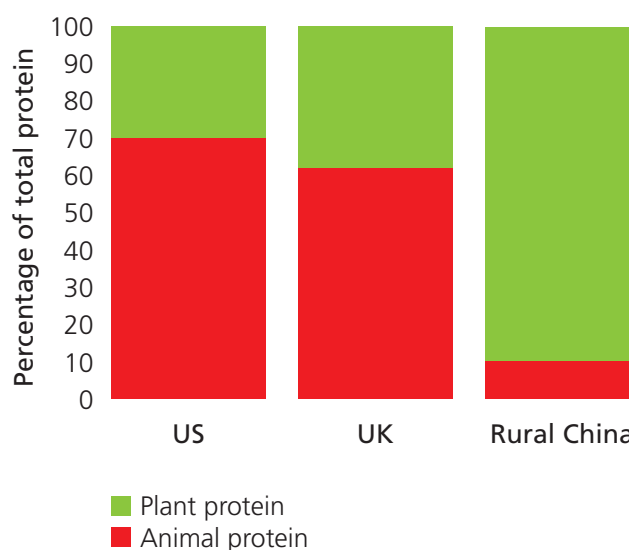
Campbell and Junshi found that several major diseases including a number of different cancers as well as CVD and diabetes were all associated with affluent diets. In other words, these diseases were directly associated with the intake of meat, milk, eggs, animal fat and animal protein whilst diets high in fibre, antioxidants (mainly from fruit and vegetables) and pulses had a preventative effect.

It had previously been suggested that animal protein increases the risk of cancer. The extensive *China Study* found a direct link between dietary protein intake and cancer; the more protein in the diet, the higher the risk of cancer. But this did not apply to all protein, just animal protein.

It is widely acknowledged that the incidence of certain cancers is much greater in some countries than others. What intrigued Campbell was the relationship between animal protein and these cancers. Figure 2.0 shows the differences in animal protein intake between the US, the UK and rural China. In the US, over 15 per cent of total energy intake comes from protein of which 70 per cent is animal protein (Campbell and Campbell, 2005). In the UK, over 16 per cent of food energy comes from protein and of this, 62 per cent comes from animal foods (Henderson *et al.*, 2003). While in rural China, the figures are quite different; nine to 10 per cent of total energy comes from protein and only 10 per cent of that is from animal protein (Campbell and Campbell, 2005).

It could be argued that the difference in cancer incidence between cultures reflects genetic differences between ethnic groups rather than environmental (dietary) effects. However, migrant studies have shown that as people move from a low-cancer risk area to a high-cancer risk area, they assume an increased risk within two generations (WCRF/AICR, 2007). Therefore these vast differences in cancer rates must be largely attributable to environmental factors such as diet and lifestyle. Campbell concluded that animal-based foods are linked to an increased cancer risk whereas a wholegrain plant-based diet including fibre and antioxidants is linked to lower rates of cancer (Campbell and Campbell, 2005).

Figure 2.0 A comparison of animal protein intake in the US, UK and rural China.



Source: Campbell and Campbell, 2005; Henderson *et al.*, 2003.

Campbell has extensively pursued, for many years, the hypothesis that dietary protein enhances tumour development. Several mechanisms have been proposed including how diets rich in animal protein (meat and dairy) drive the synthesis of the growth factor insulin-like growth factor 1 (IGF-1) in the liver, which increases the risk of a range of cancers. A 2006 study published in the *American Journal of Clinical Nutrition* found that compared to a typical Western diet, a low-protein, low-calorie diet was associated with significantly lower IGF-1 levels (Fontana *et al.*, 2006). Campbell acknowledged the importance of this finding but said in his opinion the links between animal protein and IGF-1 production are only one of these countless and highly interdependent cause and effect relations that occur in human nutrition (Campbell, 2007).

Other studies have focused directly on meat. The NIH-AARP Diet and Health Study presents information on the impact of diet and lifestyle factors on risk of cancer. In 1995-1996, nearly half a million US men and women, aged 50-71, none of whom had had cancer, joined the study. They completed a questionnaire about their dietary habits with personal information such as age, weight and smoking history. During an average of seven years follow-up, 53,396 cases of cancer were reported. This information was used to identify links between the amount of meat they ate and the incidence of various cancers (Cross *et al.*, 2007).

Results showed that those who ate the most red meat

MEAT THE TRUTH

had a 20-60 per cent higher risk for oesophageal, bowel, liver and lung cancer compared to those eating the least. Those who ate the most processed meat had a 20-16 per cent higher risk for bowel and lung cancer respectively. The authors discussed different mechanisms linking meat to cancer; saturated fat, haem iron, NOCs, HCAs and PAHs. They suggested that the link between saturated fat and cancer is likely to be related to energy balance; this means too many calories. A diet high in saturated fat, leading to obesity, increases the risk of breast, prostate and bowel cancer as well as heart disease and diabetes. The increased risk of the hormone-related cancers (breast and prostate) may result from increased levels of hormones synthesised in adipose (fat) stores in the body (Gerber and Corpet, 1999).

We know that diets rich in animal protein (meat and dairy) increase IGF-1 which increases the risk of a range

of cancers, including bowel and lung cancer. Iron is also thought to contribute to an increased cancer risk by generating free radicals and inducing oxidative stress. The authors of the NIH-AARP Diet and Health Study also pointed out how exposure to NOCs occurs from eating nitrite-preserved meats as well as endogenous formation in the gut, which is directly related to the haem iron content of meat. They concluded that reducing meat intake to match the lowest intake in the study group could reduce the incidence of cancer at multiple sites. As stated previously, people in the NIH-AARP study eating 53g of red meat per day could still be classed in the lowest intake group. Avoiding meat entirely would be even more effective in preventing cancer.

A UK study from the University of Oxford, looking at how different diets affects cancer risk, revealed that people who don't eat meat have a much lower risk of



getting the disease. The 15-year study was a pooled analysis of two prospective studies including 61,647 British men and women (32,491 meat-eaters, 8,612 fish-eaters, 18,298 vegetarians and 2,246 vegans) among whom there were 4,998 cases of cancer (Key *et al.*, 2014). Results showed that compared with meat-eaters, cancer incidence was 12 per cent lower in fish-eaters, 11 per cent lower in vegetarians and **19 per cent lower in vegans**. This adds to a huge body of evidence showing how meat increases the risk of cancer and other diseases (Key *et al.*, 2014).

The results of the US Adventist Health Study II (AHS-2) were similar, with total cancer risk significantly lower in vegetarians and vegans than in meat-eaters (Tantamango-Bartley *et al.*, 2013). This study examined the association between diet and cancer incidence among 69,120 people among whom 2,939 cancer cases were identified during an average of four years follow-up. In this typically low-risk population, avoiding meat clearly conferred protection against cancer. Vegetarians had an eight per cent lower risk of cancer and **vegans had a 16 per cent lower risk**. In addition to this, vegan women experienced **34 per cent fewer female-specific cancers**. The authors pointed out that the meat-eaters in AHS-2 consume much less meat than the average American. So people eating diets mainly based on meat and dairy are likely to have an even higher risk of cancer than the meat-eaters in AHS-2.

Two key studies found vegetarians have an 8-11 per cent lower risk of cancer and vegans a 16-19 per cent lower risk.

An international study comparing rates of 21 different cancers in 157 countries (reported in 87 studies in 2008), found that people who ate the most animal products had the highest rate of cancers of the breast, kidney, pancreas, prostate and thyroid (Grant, 2014). An interesting feature of this study was the time lag observed between increasing cancer rates and national dietary changes as countries adopt a more Western diet, rich in meat and dairy. Results revealed a 15-30 year lag between diet and cancer mortality; 15-27 years for bowel cancer and 20-31 years for breast cancer in Japan and 10 years for mortality rates for a number of different cancers in several Southeast Asian countries. The author said this study reiterates that animal products are an important risk factor for many types of cancer. Mechanisms discussed, linking animal products to cancer, included increased production of insulin, IGF-1 and sex hormones as well as haem iron giving rise to free radicals, oxidative stress and DNA damage. The

author concluded that eating animal products is as important as smoking for increasing the incidence of all cancer types apart from lung cancer.

Even moderate intakes of animal protein are linked to a higher risk of cancer. A large cross-sectional study from the US looked at protein intake and cancer rates in 6,381 people over 18 years. Results showed **those reporting a high animal protein intake had a 75 per cent higher chance of dying from all-causes** during the follow-up period than people eating much less animal protein. The high-protein group were more than four times more likely to die of cancer than those in the low-protein group (Levine *et al.*, 2014). These associations were either abolished or weakened if the source of protein was plant-based. The authors concluded that taken together, these results indicate that people aged 50-65 consuming moderate to high levels of animal protein display a major increase in their risk for cancer which could be reduced if the protein did not come from an animal source. They suggested high levels of animal protein increasing IGF-1 and possibly insulin may be responsible for the links between animal protein and cancer. These results are in agreement with recent findings on the association between meat consumption and death from all-cause, CVD and cancer (Fung *et al.*, 2010; Pan *et al.*, 2012).

Red and processed meat intake has long-been associated with increased risks of bowel cancer as well as several other cancers (WCRF/AICR, 2007). Several compounds either found in meat or created in it by high-temperature cooking are now known as being potential carcinogens including: NOCs, HCAs and PAHs. Haem iron and iron overload can also increase cancer risk through a range of processes including the promotion of NOC formation, increased colonic cytotoxicity and epithelial proliferation, increased oxidative stress and iron-induced hypoxia signalling (Pan *et al.*, 2012). In other words, it contributes to compounds that can damage the cells in the colon and lead to cancer.

CHICKEN AND PHIPS

Poultry is a source of haem iron too and is a source of potential carcinogens particularly when cooked at high temperatures. Grilling or barbecuing meat results in the formation of HCAs and PAHs, which are potent carcinogens. PhIP is the most abundant HCA detected in the human diet. The IARC has classified PhIP as possibly carcinogenic to humans. PhIP is formed at high temperatures in a reaction between substances found in meats and sugar and depends on the method of

MEAT THE TRUTH

cooking and the variety of meat and increases with the temperature and duration of cooking. Research shows that high levels of PhIP have been found in chicken that was pan-fried, oven-grilled or barbecued. In these samples, PhIP levels were much higher than amounts reported previously in red meats (Sinha *et al.*, 1995).

WHO SAYS MEAT DOES CAUSE CANCER

Viva!Health have been warning people about the links between meat and cancer for years and in 2015, just ahead of World Vegan Day on November 1, the World Health Organisation (WHO), one of the most-respected authorities on health, published a press release stating that processed meat does cause cancer and that red meat probably does too (WHO/IARC, 2015). This was significant because the meat industry has dismissed previous claims linking meat to cancer as 'unscientific' or 'simplistic'. They are still arguing the toss but even they must now realise that they have lost the argument and are clearly trying to defend the indefensible.

The International Agency for Research on Cancer (IARC) is the specialised cancer agency of the WHO. In October 2015, 22 scientists from ten countries met at the IARC in Lyon, France, to evaluate the carcinogenicity of the consumption of red and processed meat to decide if red and processed meats cause cancer. Their findings, published in *The Lancet Oncology*, hit the headlines around the world. They said that eating just 50 grams of processed meat (less than two slices of bacon) a day increases the risk of bowel cancer by 18 per cent.

Here are three main findings from their report:

- Overall, the Working Group classified consumption of processed meat as "*carcinogenic to humans*" on the basis of sufficient evidence for bowel cancer. Additionally, a positive association with the consumption of processed meat was found for stomach cancer.
- The Working Group classified consumption of red meat as "*probably carcinogenic to humans*".
- In making this evaluation, the Working Group took into consideration all the relevant data, including the substantial epidemiological data showing a positive association between consumption of red meat and bowel cancer and the strong mechanistic evidence. Consumption of red meat was also positively associated with pancreatic and with prostate cancer.

Source: Bouvard *et al.*, 2015.

Current guidelines need to be amended to reflect the now indisputable link between meat and cancer.

Cigarettes carry government health warnings, why shouldn't bacon? The government

acknowledge the link between eating a lot of red meat and bowel cancer but they should now go further and warn people sufficiently about the risks associated with eating meat. Although much of the battle against cancer emphasises early detection and treatment, the burden of cancer in terms of both personal suffering and the financial strain on health services, will remain high unless the primary risk factors are understood and addressed.



SUMMARY

- In the UK, one in two people born after 1960 will be diagnosed with cancer during their lifetime. Just four types of cancer (breast, prostate, lung and bowel) make up more than half of all cancers. Migrant studies show that lifestyle and diet play a much larger role than genes. After smoking, poor diet is the most important avoidable cause and people who eat meat have a higher risk.
- The US NIH-AARP Diet and Health Study found people who eat the most red meat had a 20-60 per cent higher risk for oesophageal, bowel, liver and lung cancer. Those eating the most processed meat had a 16-20 per cent higher risk for bowel and lung cancer.
- Vegetarians and vegans have a lower risk of cancer than meat-eaters. In the UK, vegetarians have an 11 per cent lower risk and vegans 19 per cent lower. The AHS-2 had similar results; vegetarians had an eight per cent lower risk and vegans 16 per cent lower. Vegan women had 34 per cent fewer female-specific cancers.
- A study of cancer in 157 countries found high intakes of animal products linked to higher rates of cancers of the breast, kidney, pancreas, prostate and thyroid. Another study found that people eating high levels of animal (but not plant) protein are over four times more likely to die of cancer.
- Chicken is not a healthy option; modern supermarket chickens contain more fat than protein, much of it saturated. High-saturated fat intake increases the risk of breast, prostate and bowel cancer as well as heart disease and diabetes while animal protein increases IGF-1 levels linked to bowel and lung cancer. Haem iron from meat generates free radicals and induces oxidative stress. Chicken also contains higher levels than red meat of the carcinogenic compound PhIP.
- In 2015, the WHO announced that processed meat does cause cancer and that red meat probably does too. Just 50g of processed meat (less than two slices of bacon) a day increases the risk of bowel cancer by 18 per cent. Current guidelines need to be amended to reflect the now indisputable link between meat and cancer. Cigarettes carry government health warnings, why shouldn't bacon and chicken nuggets?



BREAST CANCER

Breast cancer is the most common type of cancer in the UK, with 150 women and at least one man diagnosed every day. Breast cancer incidence rate in the UK has increased by over 70 per cent since the 1970s. Just in the eight years from 1995-2003 it rose by 23 per cent (Bate and Baker, 2015). **Now one in every eight women in the UK will develop breast cancer at some point in their lives.**

Breast cancer rates vary greatly around the world, the age standardised rate now exceeds 100 per 100,000 in Belgium and Denmark and rates in North America, Northern and Western Europe, Australia and New Zealand are not far behind. Moderate rates are seen in South America, Southern Africa and Western Asia, but these are increasing. The lowest rates of breast cancer are found in East and Middle Africa and South-Central and East Asia.

Much is made of the link between genes and breast cancer. However, only 5-10 per cent of all breast cancers are thought to be linked to an inherited breast cancer gene (Apostolou and Fostira, 2013). This means that the vast majority of cancers (90-95 per cent) are not caused by genes. It is also important to remember that having a known breast cancer gene does not mean that a person will definitely develop the disease, it means they are more at risk of developing it.

A range of lifestyle and environmental factors can also increase breast cancer risk including: age, alcohol, obesity, high-animal/low-plant product diet, early puberty, late menopause, late age at first childbirth, hormone replacement therapy (HRT) and the contraceptive pill. Factors that may reduce the risk include: younger age at first pregnancy, high-plant/low-animal product diet, breastfeeding, late puberty, early menopause and physical activity. You can't control all of these risk factors (age, puberty, menopause) but you can change your body weight, the amount of physical activity you do and of course your diet. These three factors have all been linked to breast cancer and are areas where you can take action.

Migration studies show us that the variation in breast cancer rates around the world is not due to genetic factors and that lifestyle factors must be involved. For example, one study found that breast cancer rates among US-born Chinese women were 80 per cent higher than among their foreign-born counterparts (Gomez *et al.*, 2010).

In 2007, a study showing how a Western-style diet is increasing the risk of breast cancer in an Asian population was published. The Shanghai Breast Cancer Study compared the diets of 1,446 women from Shanghai recently diagnosed with breast cancer with those of 1,549 age-matched women without the disease (Cui *et al.*, 2007). Two distinct dietary patterns were identified: 'vegetable-soy' (characterised by tofu, cauliflower, beans, bean sprouts and green leafy vegetables) and 'meat-sweet' (characterised by pork, poultry, organ meats, beef and lamb as well as shrimp, saltwater fish, and shellfish, candy, dessert, bread and cow's milk – notably rich in saturated fats and red meat). The authors said these diets resemble the two primary patterns consistently identified across the US and Europe sometimes referred to as 'prudent' and Western.

Results showed the 'meat-sweet' diet significantly increased the risk of breast cancer in postmenopausal women, especially those who were overweight and with oestrogen receptor-positive breast cancer. These results are consistent with previous analyses of the same cohort that found that red meat, especially well-done red meat, increased breast cancer risk in premenopausal and postmenopausal women. The authors said their findings indicate that red meat intake in Shanghai occurs in a recognisably Western-influenced dietary pattern now emerging in Asian populations.

Another study, this time from Singapore, evaluated the role of diet in breast cancer (Butler *et al.*, 2010). A large group of 34,028 women were followed for up to 12 years during which time 629 breast cancer cases recorded. Two dietary patterns were identified; the 'meat-dim sum' pattern and a 'vegetable-fruit-soy' one. Results showed that **postmenopausal women eating the most plant-based foods had a 30 per cent lower risk of breast cancer. Those on the mostly plant-based diet followed for more than five years had a 63 per cent lower risk.** The authors suggested possible protective roles are conferred by substances found in cruciferous vegetables (cauliflower, cabbage, bok choy, broccoli, Brussels sprouts and similar green leafy vegetables) and soya isoflavones (a phytoestrogen or natural plant hormone). So in these two studies of traditionally low-risk populations, a harmful role for a meaty diet and a protective role for a plant-based diet was observed.

Of course the protective role of a plant-based diet is not limited to any particular country. The ORDET study from Northern Italy found that **women consuming higher levels of plant-based foods had a 34 per**

cent lower risk of breast cancer, those with a BMI over 25 had an even greater risk reduction – **over 50 per cent less risk** (Sieri *et al.*, 2004). The authors concluded that a diet rich in raw vegetables and olive oil protects against breast cancer.

The US Nurses' Health Study II provides some striking evidence; they found that women who ate one-and-a-half servings of red meat per day had a 22 per cent increased risk of breast cancer compared with women who ate one serving of red meat per week (Farvid *et al.*, 2014). In this study, each additional serving of red meat per day was associated with a 13 per cent increase in risk of breast cancer. The authors said: *"When this relatively small relative risk is applied to breast cancer, which has a high lifetime incidence, the absolute number of excess cases attributable to red meat intake would be substantial and hence a public health concern"*. A further finding in this study was that **for each serving per day of red meat, the risk of breast cancer was 54 per cent higher among women using oral contraceptives.** The authors suggest that replacing one serving per day of red meat with pulses (peas, beans and lentils) could lower breast cancer risk by 15 per cent among all women and 19 per cent among premenopausal women.

In a subsequent study, women from the same cohort (Nurses' Health Study II) were asked to provide information about their diets during adolescence (Farvid *et al.*, 2015). Most breast cancer studies look at the diets of women in midlife or later. The hypothesis was that during adolescence and early adulthood mammary glands may be more susceptible to carcinogenic exposure. The authors developed this idea based on the fact that girls and young women exposed to radiation resulting from the atomic bombings of Hiroshima and Nagasaki had a higher risk of breast cancer later in life, but women exposed at age 40 or older did not have an increased risk. Results showed

Why is red meat a potential cause of breast cancer?

"Carcinogenic by-products such as heterocyclic amines and polycyclic aromatic hydrocarbons, created during high temperature cooking of meat; animal fat and haem iron from red meat; and hormone residues of the exogenous hormones for growth stimulation in beef cattle are some of the mechanisms that may explain the positive association between high intake of red meat and risk of breast cancer" Farvid *et al.*, 2014.

MEAT THE TRUTH

higher consumption of red meat during adolescence was associated with a 42 per cent increased risk of premenopausal breast cancer.

Researchers looked at fruit and vegetable intake in relation to breast cancer risk in the Nurses' Health Study II cohort and found an association between higher fruit intake and lower risk of breast cancer (Farvid *et al.*, 2016). For individual fruits and vegetables, **greater consumption of apple, banana and grapes during adolescence and oranges and kale during early adulthood was significantly associated with a reduced risk of breast cancer.**

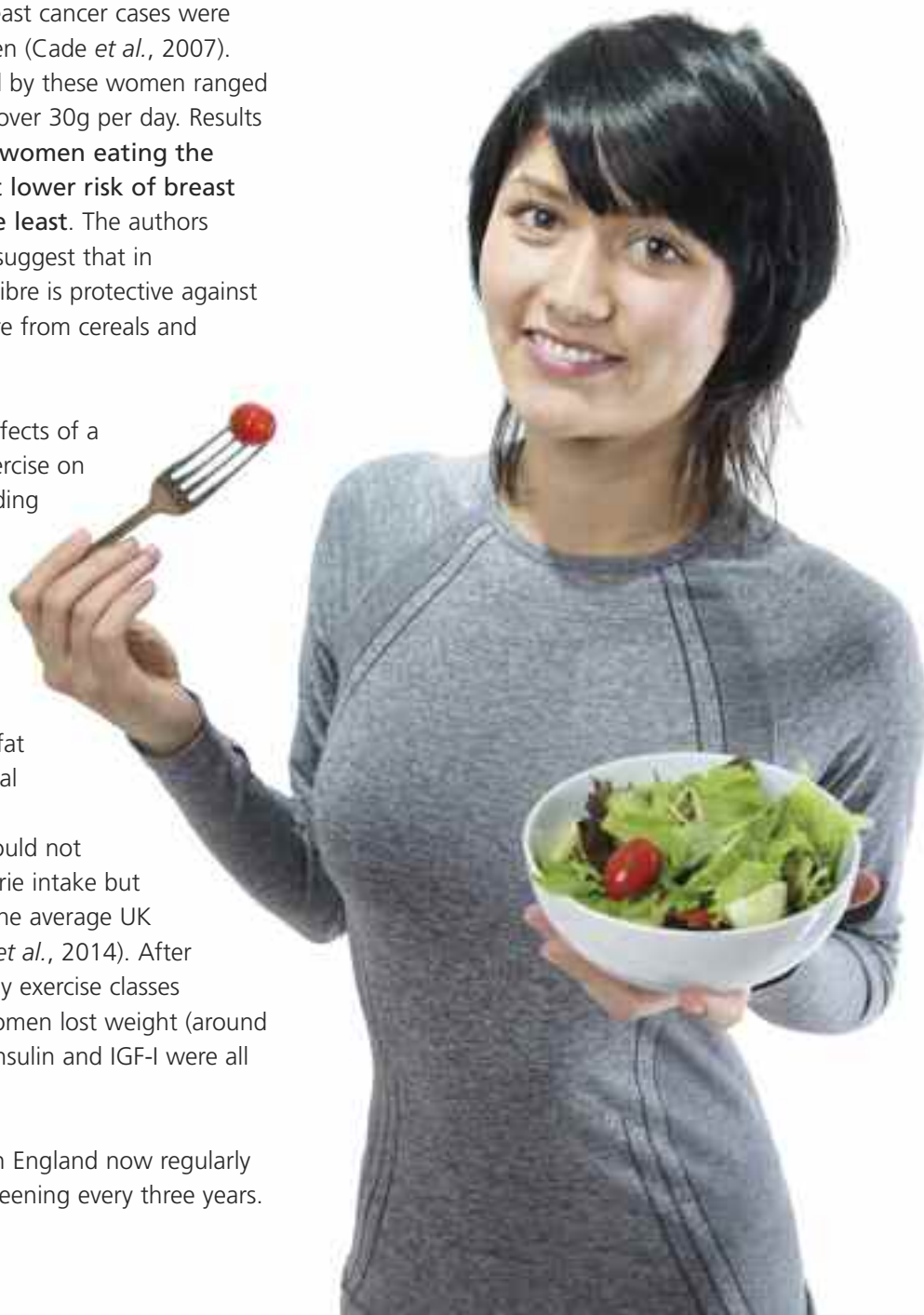
Research from the large EPIC study also found that diets rich in fibre from vegetables were associated with a reduced risk of breast cancer (Ferrari *et al.*, 2013). Similar results were reported by the UK Women's Cohort Study in which 607 breast cancer cases were recorded among 35,792 women (Cade *et al.*, 2007). The amount of fibre consumed by these women ranged from less than 20g per day to over 30g per day. Results showed that **premenopausal women eating the most fibre had a 52 per cent lower risk of breast cancer than those eating the least.** The authors concluded that these findings suggest that in premenopausal women, total fibre is protective against breast cancer; in particular, fibre from cereals and possibly fruit.

Another study looked at the effects of a high-fibre, low-fat diet and exercise on breast cancer risk factors including oestrogen, obesity, insulin and IGF-I in a group of overweight and obese postmenopausal women (Barnard *et al.*, 2006). They were given a diet containing 30-40g of fibre per day and a fat intake of 10-15 per cent of total calorie intake (the government recommends our fat intake should not exceed 35 per cent of our calorie intake but the most recent NDNS found the average UK intake was 36 per cent (Bates *et al.*, 2014). After two weeks of this diet and daily exercise classes (walking on a treadmill) the women lost weight (around 2kg) and levels of oestrogen, insulin and IGF-I were all significantly reduced.

Almost seven million women in England now regularly attend NHS mammography screening every three years.

Assessing their personal breast cancer risk and providing preventative lifestyle advice could help reduce breast cancer incidence substantially. In a survey of 1,803 women, two-thirds associated lifestyle factors with breast cancer but many seemed unaware of specific risk factors such as weight gain, dietary factors, obesity, alcohol consumption and physical inactivity (Fisher *et al.*, 2016). Attendees have already indicated their wish to have lifestyle advice at NHS breast screening clinics, but this is still not routinely provided. Such an initiative could help women be more proactive in preventing breast cancer.

Dietary advice should include how a low-fat, high-fibre, dairy-free, meat-free diet consisting mainly of fruits, vegetables, wholegrains and pulses can result in a major reduction in breast cancer risk factors.



SUMMARY

- Breast cancer rates continue to rise, year on year. One in eight women in the UK will now be diagnosed at some point in their lives. Rates around the world vary widely with highest levels in North America and Europe. Only five to 10 per cent are caused by genes; the vast majority are caused by lifestyle and environmental factors.
- As the Western diet spreads around the world, so does breast cancer. The Shanghai Breast Cancer Study found that women eating Western-style diet containing meat, fish, bread and sugary foods had a higher risk of breast cancer than those eating a traditional diet rich in vegetables and soya.
- The evidence against meat is mounting. The US Nurses' Health Study II found that women eating one-and-a-half daily servings of red meat had a 22 per cent higher risk of breast cancer than those eating one serving a week. Each additional daily serving increased the risk by 13 per cent or 54 per cent among those using oral contraceptives! Women who had eaten the most meat in adolescence had a 42 per cent higher risk of premenopausal breast cancer, but those who had eaten the most fruit had a lower risk.
- Plant-based diets lower the risk of breast cancer. A study from Singapore found that over five years, diets rich in plant foods lowered the risk by 63 per cent in postmenopausal women. The Italian ORDET study found that those eating the most plant foods had a 34 per cent lower risk – it was 50 per cent lower in those with a BMI over 25.
- The EPIC studies show that diets rich in fibre from vegetables lower the risk. The UK Women's Cohort Study found that premenopausal women eating the most fibre had a 52 per cent lower risk than those eating the least.
- Evidence for the harmful role of meat is as strong as that for the protective role of a low-fat, high-fibre, vegan diet consisting mainly of fruits, vegetables, wholegrains and pulses. Providing preventative dietary advice to the seven million women who attend mammography screening every three years in the UK could help reduce breast cancer incidence substantially.

PROSTATE CANCER

Prostate cancer is the most common cancer in men. Across the UK, over 47,000 men are diagnosed with it every year – 130 men every day. Prostate cancer incidence rates have increased by 155 per cent in men in the UK since the late 1970s. Some of this may be down to increased testing but the fact remains that in the UK, **about one in eight men will get prostate cancer at some point in their lives.**

Prostate cancer incidence varies more than 25-fold worldwide; the pattern is similar to that of breast cancer incidence with the highest rates seen in Australia, New Zealand, North America, Western and Northern Europe, where the age-standardised rate is around 100 men per 100,000 (GLOBOCAN, 2012). Incidence rates are also relatively high in some less affluent regions such as the Caribbean (79.8), Southern Africa (61.8) and South America (60.1), but remain low in Asian populations with estimated rates of 10.5 in Eastern Asia and 4.5 in South-Central Asia.

This leading cause of cancer death in most developed countries is now emerging as a major public health problem in developing countries too (Jemal *et al.*, 2011). Prostate cancer incidence rates have been rapidly increasing in China, Korea, Japan and Singapore during the last few decades (Zhang *et al.*, 2012). This increase is thought to be caused by the gradual change towards the Westernised diet with high intakes of energy, animal fat and meat and a low intake of fibre (Gathirua-Mwangi and Zhang, 2014).

Migrant studies provide further evidence that diet plays a role in prostate cancer as remarkable increases in this disease have been observed among men who move from East Asia to North America (Gathirua-Mwangi and Zhang, 2014). High consumption of meat (particularly red, processed and well-done meat), is associated with an increased risk of prostate cancer (John *et al.*, 2011). A review of 46 studies found that saturated fat, well-done meat and calcium are all consistently linked to an increased risk for advanced prostate cancer (Gathirua-Mwangi and Zhang, 2014). Interestingly, calcium from dairy products, but not calcium from other foods was associated with the risk of prostate cancer (Allen *et al.*, 2008).

In a US study of men from the Greater San Francisco Bay Area, weekly consumption of three or more servings of red meat, one and a half or more servings of **processed meat**, one or more serving of **grilled red**

MEAT THE TRUTH

meat and one or more serving of well-done red meat were each associated with a **50 per cent increased risk of developing advanced prostate cancer** (John *et al.*, 2011).

In this study of men from the Greater San Francisco Bay Area, although the increased prostate cancer risk was associated with grilled and well-done red meat, they found no consistent associations with HCAs (except for a possible link with advanced disease and PhIP). However, more than 15 different HCAs are known to accumulate in cooked meat, yet usually only three (PhIP, MeIQx and DiMeIQx) are measured and compounds formed at lower concentrations, which are not measured or not-yet identified, may play a role in prostate cancer (Joshi *et al.*, 2012). While this study also found no consistent association with BaP (the most abundant PAH) the authors said that they couldn't exclude the possibility that other PAHs formed during cooking may underlie the link between grilled red meat and prostate cancer.

Another study, looking at 2,770 cases of prostate cancer among 26,030 men did find a positive association between PhIP from red meat and prostate cancer, particularly high-grade (fast-growing) and possibly also advanced prostate cancer (Rohrmann *et al.*, 2015). However, they noted that while PhIP intake from red but not from white meat was linked with prostate cancer, PhIP intake from white meat is twice as high as that from red meat. This, they said, argues against PhIP (or HCAs in general) as the factor responsible for the link between meat and prostate cancer and suggests that some other mutagenic compounds arising from meat may be the causal factor. More research is required to fully-elucidate the underlying mechanisms.

The research on how diet may affect progression of prostate cancer *after* diagnosis is of great interest given the large numbers of men diagnosed with this disease. Researchers from the Physicians Committee for Responsible Medicine (PCRM) reviewed eight observational studies and 17 intervention studies on the effect of diet on prostate cancer and found that **diets high in saturated fat are associated with a threefold higher risk of cancer progression and death**, compared with a diet low in saturated fat (Berkow *et al.*, 2007). This review revealed a slowing of disease progression in prostate cancer patients consuming flaxseed and lycopene-containing foods (tomatoes are a rich source of lycopene). The authors concluded that **plant-based diets – high in fibre and**

phytonutrients and low in fat and saturated fat – favourably influence health outcomes for prostate cancer patients.

Another study looking at how diet may affect disease progression found that two and a half eggs per week increased the risk of lethal prostate cancer by 81 per cent compared to less than half an egg per week (Richman *et al.*, 2011). They also found a link between poultry and processed meat and progression to lethal prostate cancer, but these associations were of borderline statistical significance. The association with poultry appeared to be driven by poultry and poultry products with skin (chicken or turkey with skin, chicken or turkey hot dogs and chicken or turkey sandwiches). The authors said they had limited power to examine individual poultry items due to low consumption of these foods in their study population. Those eating three and a half servings a week of poultry products were put in the highest consumer group; many people eat a lot more chicken than that. Also, the men who ate the most poultry in this study engaged in more vigorous activity, were less likely to be smokers and consumed less red meat, dairy and coffee than men who ate the least poultry. These factors may have contributed somewhat to masking the harmful effects of poultry in this relatively healthier-living group of low-consumers.

The relationship between eating poultry with skin after diagnosis and clinical outcomes (or disease progression) in men with prostate cancer has been examined before. In the CaPSURE study, **men who ate the most poultry with skin had more than double the risk of prostate cancer progression** compared with men who ate the least (Richman *et al.*, 2010).

The Prostate Cancer Lifestyle Trial was a one-year randomised controlled clinical trial of 93 patients with early-stage prostate cancer who had chosen not to undergo conventional treatment (Ornish *et al.*, 2005). This was a unique opportunity to observe the effects of diet and lifestyle changes without the confounding effects of radiation or surgery. The patients in the experimental group followed a vegan diet consisting of fruits, vegetables, wholegrains and pulses including soya. They also exercised, practised stress management and attended group support sessions. The control patients received the usual care. After one year, **none of the vegan group had required conventional treatment** but six control patients had because their disease had progressed. Prostate-specific antigen (PSA) fell four per cent in the vegan group but went up six per cent in the control group. Although the size of these changes was

modest, the direction of change is important as an increase in PSA indicates disease progression. In a second test, prostate cancer cells *in vitro* were exposed to blood serum from both groups to see if it stimulated or decreased prostate cancer cell growth. Serum from the vegan group inhibited growth almost eight times more than serum from the control group (70 versus nine per cent), suggesting that comprehensive lifestyle changes could affect tumour growth.

In a subsequent study, after two years, just two (five per cent) of the 43 vegan patients had undergone conventional treatment but 13 (almost a third) of the 49 control patients had. Dr Dean Ornish, clinical professor of medicine and founder and president of the Preventive Medicine Research Institute, concluded that **patients with early-stage prostate cancer might be able to avoid or delay conventional treatment for at least two years by making changes in their diet and lifestyle** (Frattaroli *et al.*, 2008).

Taken together, this research provides further evidence that poultry, red and processed meat are associated with increased risk of prostate cancer. While the exact mechanism underlying the links between meat and prostate cancer remain unclear, the association remains firmly established. Research shows that if health professionals can offer qualified, general advice about diet, this may provide men with a focus for action and a means of regaining control (Kassianos *et al.*, 2015). Despite all this evidence, advice from the NHS on the links between diet and prostate cancer remains sparse.

The links between meat and cancer explained

Several explanations have been suggested for the link between red meat and prostate cancer. HCAs formed when meat is cooked at high temperatures may account for the link between poultry after diagnosis and risk of lethal prostate cancer (Richman *et al.*, 2010). HCAs form DNA adducts (this is when DNA becomes bound to a cancer-causing chemical) and so increase the occurrence of numerous cancers including prostate cancer (Richman *et al.*, 2011). Chicken is a primary source of HCAs in the typical Western diet and fried, roast and grilled chicken can contain particularly high amounts of HCAs.

Another obvious culprit is the saturated fat in meat, which has long been proposed as a risk factor. Total and saturated fat intake have both been positively associated with PSA levels (this is a protein produced by the prostate gland used to assess disease progression), increased risk of prostate cancer and aggressive prostate cancer, while saturated fat intake has been associated with fatal prostate cancer (Bishop *et al.*, 2015).

However, results of some studies of dietary fat and prostate cancer are inconsistent, some having a link with red meat but not fat. Other explanations include the presence of haem iron in red meat, which may catalyse oxidative reactions. Finally, an association between prostate cancer and meat intake may be due to potent chemical carcinogens generated during cooking and processing of red meat and poultry, such as NOCs, HCAs and PAHs. The prostate gland is able to metabolise these chemicals into activated carcinogens (Joshi *et al.*, 2012).



The telomere study

In a later study, Ornish focussed on telomeres, stretches of DNA that protect the ends of our chromosomes rather like the plastic caps that prevent shoelaces from unravelling (Ornish *et al.*, 2013). Every time a cell divides, telomeres become shorter. Once they reach a critically short length, the cell stops dividing and dies. As more body cells are lost, signs of ageing begin to appear, which may be followed by age-related diseases such as cancer, stroke, heart disease, vascular dementia, obesity, osteoporosis and diabetes.

Dr Ornish worked alongside Elizabeth Blackburn, who won the 2009 Nobel Prize in Physiology or Medicine for her discovery of telomerase, the enzyme that replenishes telomeres. In this study, as before, one group followed Ornish's lifestyle changes, the

other group carried on as usual. They measured telomere length at the beginning of their study and again after five years. Among the men with low-risk prostate cancer who made comprehensive lifestyle changes (vegan diet, exercise, stress management and support group), telomere length increased significantly by an average of 10 per cent. Telomere length decreased by an average of three per cent in the control group.

This study suggests yet another possible mechanism by which lifestyle changes, avoiding meat and dairy, might help combat prostate cancer and other age-related diseases. Ornish said: *"The implications of this relatively small pilot study may go beyond men with prostate cancer. If validated by large-scale randomised controlled trials, these comprehensive lifestyle changes may significantly reduce the risk of a wide variety of diseases and premature mortality"*.

The data linking the consumption of poultry, red and processed meat to prostate cancer provides a convincing argument for eliminating meat (and all animal foods) from the diet while increasing the intake of wholegrain foods, pulses (including soya), fruit and vegetables, nuts and seeds.

SUMMARY

- Like breast cancer, prostate cancer rates are rising and one in eight men in the UK will get prostate cancer at some point in their lives. Rates vary widely around the world but as the Western diet spreads, so does prostate cancer.
- Meat (particularly red, processed and well-done meat), is linked to a higher risk of prostate cancer. A US study found that during a week, three servings of red meat, one and a half servings of processed meat, one serving of grilled red meat or one serving of well-done red meat were each associated with a 50 per cent increased risk of advanced prostate cancer.
- There is a link with poultry too, especially chicken and turkey with skin as well as hot dogs and sandwiches made with chicken and turkey. In the CaPSURE study, men eating the most poultry with skin had more than double the risk of

prostate cancer progression than those eating the least. The PCRM found that diets high in saturated fat are associated with a threefold risk of cancer progression and death. Disease progression was slower in men who ate flaxseed and lycopene-containing foods like tomatoes.

- The Prostate Cancer Lifestyle Trial showed that men with early-stage prostate cancer might be able to avoid or delay conventional treatment by adopting a low-fat, vegan diet. This sort of dietary advice on how a vegan diet, high in fibre and phytonutrients and low in fat and saturated fat, may prevent and slow prostate cancer, could provide men with a focus for action and a means of regaining control.





LUNG CANCER

Lung cancer is one of the most common and serious types of cancer, making up around 13 per cent of all cancers and responsible for nearly one in five cancer deaths worldwide (GLOBOCAN, 2012). Men are more likely to develop lung cancer than women, probably because they tend to smoke more. It is the most common cancer in men worldwide with the highest rates in Central and Eastern Europe (53.5 per 100,000) and Eastern Asia (50.4 per 100,000). Low rates for men are seen in Middle and Western Africa (2.0 and 1.7 per 100,000 respectively).

In women, the incidence rates are generally lower and the geographical pattern is a little different, reflecting different historical patterns of smoking. The highest rates for women are seen in North America (33.8) and Northern Europe (23.7) with a relatively high rate in Eastern Asia (19.2) and the lowest rates again in Western and Middle Africa (1.1 and 0.8 respectively).

In the UK, lung cancer incidence peaked in the 1960s, remained high until the mid-1980s, and is now declining (WCRF/AICR, 2007). Around 44,500 people are now diagnosed with lung cancer every year in the UK (NHS Choices, 2015b). Smoking is a well-established risk factor for lung cancer and is thought to be linked to 85 per cent of cases. This is because smoking involves regularly inhaling a number of different toxic substances. However, evidence suggests some dietary factors also influence the risk of lung cancer.

In 2007, red and processed meats were classified by the WCRF/AICR as possible causes of lung cancer (WCRF/AICR, 2007). When their report was published the evidence was

somewhat inconsistent, but more recent studies now provide stronger evidence linking diet and lung cancer.

The huge NIH-AARP Diet and Health Study (published the same year but not included in the WCRF/AIRC report) looked at around 500,000 people aged 50-71 and found that those who ate the most red meat had an increased risk of developing lung, bowel, liver and oesophageal cancer compared with those who ate the least. Those eating the most processed meat had an increased risk of developing lung and bowel cancer (Cross *et al.*, 2007). The authors said that one in ten lung and bowel cancers could be avoided if people reduced their red and processed meat intake. Avoiding meat altogether would certainly be even more effective.

A review of 33 studies from Uruguay, Europe, Asia, the US, Canada and Australia found that both red and processed meat consumption increased the risk of lung cancer (Xue *et al.*, 2014). For every additional 120g of red meat per day, the risk of lung cancer increased 35 per cent and for every additional 50g of processed meat per day, the risk increased 20 per cent.

Another review investigating the relationship between meat and lung cancer looked at 34 studies and also found that a high intake of red meat increased the risk of lung cancer by 35 per cent (Yang *et al.*, 2012). They suggested that high levels of saturated fat present in meat may also be associated with the increased risk of lung cancer and said that more studies on meat mutagens, haem iron, meat cooking preferences and doneness level (how well done the meat is) are needed

MEAT THE TRUTH

to fully characterise the meat-lung cancer association. In a study of 242 Iranian men with lung cancer, it was found that refined grains, beef, liver, dairy products, vegetable ghee and animal ghee increased the risk, while fruit, vegetables and sunflower oil were protective (Hosseini *et al.*, 2014). Specifically, sheep and beef livers were found to be a risk factor for lung cancer. The authors said that sheep and beef livers contain heavy metals and other poisons, as it is the organ responsible for metabolising and detoxifying food, antibiotics, vaccine ingredients, pesticide over-spray, tainted water and the synthetic hormones that are frequently given to farmed animals and this may influence cancer risk.

Not all studies have identified the link between meat and lung cancer. The Prostate, Lung, Colorectal and Ovarian Cancer Screening Trial (PLCO) recorded 782 cases of lung cancer over eight years among 99,579 people aged 55-74 years. They found no significant links between red and processed meat and lung cancer (Tasevska *et al.*, 2011). The authors noted that the people enrolled in the PLCO trial were better educated, more physically active, more likely to be married, less likely to smoke and had lower mortality than the general population. It was assumed that they had healthier diets too and the relatively low intake of high temperature cooked meats may have affected this study's ability to detect an association. On average, only 20 per cent of meat consumed in this cohort was cooked at a high temperature. The authors also said the relatively low follow-up period of just eight years was short compared to other studies and may have contributed to them failing to find the links others have.

Potential mechanisms linking meat consumption to lung cancer involve carcinogenic NOCs and haem iron. NOCs are found in nitrite-preserved meat and are also generated by stomach and gut bacteria from nitrites in meat. This endogenous production of NOCs is driven by haem iron also found in meat. Iron overload can activate oxidative responsive transcription factors, pro-inflammatory cytokines and iron-induced hypoxia signalling – a classical feature of cancer (WCRF/IARC, 2007).

Cooking red meat at high temperatures results in the production of HCAs and PAHs which are potent lung carcinogens. The NIH-AARP Diet and Health Study investigated the effects of red and processed meat, HCAs, BaP (the most abundant PAH) and haem iron in almost half a million people followed over eight years (Tasevska *et al.*, 2009). Results showed a significant association between meat and lung cancer. Men who ate the most red meat had a 22 per cent higher risk of

lung cancer and women who ate the most, a 13 per cent higher risk than those who ate the least. Processed meat increased the risk for men by 23 per cent. They found an even higher risk of lung cancer in men with a high intake of haem iron and lower intakes of antioxidants. The authors suggested that an unhealthy diet may disrupt the balance between pro-oxidants and antioxidants in the lung tissue and trigger oxidative damage and carcinogenesis.

High haem iron intake is associated with an increased risk of cancer of the bowel, pancreas and lung (Hooda *et al.*, 2014). Molecular studies have revealed how haem iron may increase cancer progression. Oxygen consumption and haem synthesis are intensified significantly in lung cancer cells compared to normal cells. Thus the increase in haem availability in cancer cells and tumours leads to elevated production of haemoproteins, higher oxygen consumption and cellular energy production which fuels cancer cell progression.

Studies have shown that IGF-1 can stimulate abnormal cell growth and division and some studies have found levels are raised in people with lung cancer (Cross *et al.*, 2007). Animal protein (meat and dairy) increases the production of IGF-1 in the liver and vegans, who don't eat animal protein, have been found to have **lower levels of IGF-1 circulating in the blood**. One study found that the average IGF-I concentration was 13 per cent lower in 92 vegan women compared with 99 meat-eaters and 101 vegetarians (Allen *et al.*, 2002), while another study found IGF-1 was nine per cent lower in 233 vegan men than in 226 meat-eaters and 237 vegetarians (Allen *et al.*, 2000).

A review of eight studies, with 13,548 lung cancer cases among 108,748 people, found that a healthy diet lowered lung cancer risk (Sun *et al.*, 2016). They found that high intakes of vegetables, fruit and soya reduced risk, while red meat and processed meat increased risk. The authors said that the mechanism here is undoubtedly related to anti-tumourigenic (anti-tumour-forming) agents found in the individual components of a healthy plant-based diet, including antioxidants, polyphenols, fibre and minerals. Thus, although stopping smoking is still the most effective approach to lowering lung cancer risk, healthy eating and lifestyle are also necessary for the prevention of lung cancer, especially for former and current smokers.

SUMMARY

- Lung cancer is responsible for nearly one in five cancer deaths. Smoking may cause around 85 per cent of cases but evidence shows that dietary factors also influence the risk. In 2007, the WCRF said red and processed meat were a possible cause. Since then, the evidence has grown stronger.
- The NIH-AARP Diet and Health Study found that those eating the most processed meat had a higher risk of lung and bowel cancer and those eating the most red meat had a higher risk of lung, bowel, liver and oesophageal cancer. Men eating the most red meat had a 22 per cent higher risk of lung cancer and women eating the most, a 13 per cent higher risk. Processed meat increased the risk for men by 23 per cent. High intakes of haem iron and low intakes of antioxidants increased the risk even more.
- Studies from Uruguay, Europe, Asia, the US, Canada and Australia found that that red and processed meat increase the risk of lung cancer. Each additional 120g of red meat a day increased the risk by 35 per cent while an additional 50g of red meat increased it by 20 per cent. Another study found that a high intake of red meat increased the risk by 35 per cent.
- Some studies have found higher levels of the growth hormone IGF-1 in people with lung cancer. Animal protein triggers IGF-1 production in the liver and studies show vegans have lower levels than meat-eaters and vegetarians.
- So meat increases the risk of lung cancer and vegetables, fruit and soya reduce it. One in ten lung (and bowel) cancers could be avoided if people reduced their red and processed meat intake. Avoiding meat altogether would be even more effective. Although stopping smoking is the most effective way to lower lung cancer risk, healthy eating could help prevent it, especially in former and current smokers.

***“The evidence that fruits protect against lung cancer is convincing”
(WCRF/IARC, 2007).***



BOWEL CANCER

Bowel cancer (also called colorectal cancer) is a general term for cancer that includes colon and rectal cancer. There is wide geographical variation in incidence across the world. The highest rates are seen in Australia, New Zealand, Europe and North America and the lowest in parts of Africa, (South Central) Asia and Central America (GLOBOCAN, 2012). In *The China Study*, Professor T. Colin Campbell noted that while North America, Europe, Australia and wealthier Asian countries (such as Japan and Singapore) had relatively high rates, Africa, Asia and most of Central and South America had much lower rates. Campbell noted that the Czech Republic had a death rate of 34.19 per 100,000 males, while in Bangladesh the figure was less than one (just 0.63) per 100,000 males (Campbell and Campbell, 2005).

Bowel cancer is one of the most common types of cancer in the UK, with around 40,000 new cases diagnosed every year. About one in every 20 people in the UK will develop bowel cancer in their lifetime. The varied distribution of this disease suggests that bowel cancer may be linked to diet and lifestyle choices.

The incidence of bowel cancer is much higher in African Americans than in Native Africans (60 per 100,000 versus less than one per 100,000). To explore this further, researchers compared the diets of randomly selected people from both populations and found that surprisingly their fibre intake was the same but that African Americans consumed more protein, fat, meat, saturated fat and cholesterol (O'Keefe *et al.*, 2007). The authors concluded that the higher bowel cancer rate in African Americans is associated with their higher intake of animal products.

High intakes of red and processed meat are consistently associated with an increased risk of bowel cancer. A 2006 review of 19 prospective studies involving almost 8,000 cases of bowel cancer found that those who ate the most red meat and processed meat had a 28 per cent and 20 per cent higher risk of the disease respectively compared with those who ate the least (Larsson and Wolk, 2006). It is because of the links with bowel cancer that in its 2007 report, the WCRF recommended that people should limit their intake of red meat and avoid processed meat (WCRF/AICR, 2007).

Several theories have been put forward to explain how meat increases bowel cancer risk:

- High-fat or high-protein diets can promote carcinogenesis (the initiation of cancer)
- Potentially carcinogenic NOCs are formed in food and/or endogenously by nitrosation of amines and amides
- Cooking meat at high temperature forms mutagenic and carcinogenic HCAs and PAHs
- Haem iron in red meat can promote carcinogenesis through oxidation and DNA damage

Source: Santarelli *et al.*, 2008.



How meat increases the risk of bowel cancer

Fat intake is a major factor that could explain the link between bowel cancer and meat. Epidemiologic studies suggest that a high intake of dietary fat promotes bowel cancer by increasing the secretion of bile acids which can promote tumours. High-fat diets are also high-calorie diets and the balance between energy intake and physical activity is still considered a major risk factor (Santarelli *et al.*, 2008).

HCAs and PAHs are formed during the cooking of meat. As stated, various HCAs are formed according to the type of meat and method of cooking. Processed pork meat (ham and bacon) may not contain as many HCAs as pan-fried beef and chicken, but then processed meats like these contain nitrites which give rise to NOCs. The most abundant HCAs in meat are MeIQx, DiMeIQx and PhIP.

PAHs are produced from the incomplete combustion of organic compounds. Many PAHs, like BaP are mutagens and carcinogens. The main sources of PAHs for people are cooked and smoked meat and fish (notably barbecued meat) and tobacco smoke (Santarelli *et al.*, 2008). Studies suggests that cooking methods and how well-cooked meat is, are related to bowel cancer risk; the higher the temperature, the higher the risk. A Swedish case-control study showed that frequent consumption of fried meat with a heavily browned surface led to a three-fold increase in bowel cancer risk (Gerhardsson-DeVerdier *et al.*, 1991). In summary, HCAs and PAHs are present in the daily diet of meat-eaters; they are proven carcinogens and can lead to bowel cancer.

Another mechanism underlying the links between meat consumption and cancer, is the generation by gut bacteria of carcinogenic NOCs (WCRF/AICR, 2007). NOCs are found preformed in a range of foods including nitrite-preserved processed meats (hot dogs contain 10 times more NOCs than fresh red meat), smoked fish and cheeses (Santarelli *et al.*, 2008). However, the amount of NOCs in human faeces can exceed the amount in the diet by more than 10-fold suggesting endogenous formation in the gut (Holtrop *et al.*, 2012). One study found NOCs in the faeces were 30-fold greater than levels in the diet (Hughes *et al.*, 2001). Another study found the faecal concentration of NOCs was 60 times higher in volunteers given cured red meat than in volunteers given a vegetarian diet (Joosen *et al.*, 2009).

Endogenous production is likely to begin with the formation of nitrosothiols (organic compounds containing a nitroso group) in the stomach, as acidic conditions favour the formation of these compounds (Kuhnle *et al.*, 2007). The gastrointestinal tract then provides favourable conditions for the formation of NOCs. Haem iron stimulates the production of NOCs in the intestines (Cross *et al.*, 2012). In fact, haem from meat strikingly increases NOC formation (Santarelli *et al.*, 2008).

The catalytic effects of haem iron can be inhibited by trapping it with either calcium or chlorophyll from green leafy vegetables and NOC formation can also be inhibited by vitamins C and E. Polyphenols (found in fruit, vegetables, cereals, pulses, tea and wine) can inhibit lipid peroxidation caused by free radicals, the production of which is also catalysed by nitrite-preserved meat and haem (Bastide *et al.*, 2011). The science points to colourful fruit and green leafy vegetables offering protective benefits while all meat has to offer is a range of hidden nasties lurking within it.

Two more studies looking at whether processed meat increases bowel cancer risk by stimulating the production of NOCs, revealed the harmful effects of meat (Joosen *et al.*, 2009). A red meat (beef and pork) diet was compared to a vegetarian diet and then a preserved meat (nitrite-preserved bacon, ham, luncheon meat and corned beef) diet was compared to a vegetarian diet. Faecal homogenates (imagine putting some poo in a blender!) were analysed for haem iron, NOC and genotoxicity. Similar to previous studies, results showed faecal NOC levels were low (3-4 nmol/g) on diets containing no meat. On preserved and red meat diets containing similar amounts of haem iron, faecal NOC levels increased substantially (to around 180 nmol/g). Results also showed that while nitrite-preserved meats have a similar effect as red meat on NOC production, processed meat caused additional DNA damage providing a possible explanation for the increased risk of bowel cancer associated with bacon, sausages, ham and other processed meats.

At the higher levels of meat consumption, concentrations of NOCs are of the same order of magnitude as the concentration of tobacco-specific NOC in cigarette smoke (Bingham *et al.*, 2002). One study found that on a high-meat diet of 420g per day, exposure to NOC was comparable with other sources of NOC, such as tobacco smoke (Hughes *et al.*, 2001).

MEAT THE TRUTH

So it turns out that big meat-eaters may be in need of the same level of health advice as smokers! Which is what then Shadow Secretary of State for Environment, Food and Rural Affairs, Kerry McCarthy suggested in 2015 in an interview for *Viva!life* magazine: *"I really believe that meat should be treated in exactly the same way as tobacco with public campaigns to stop people eating it"* (McCarthy, 2015).

According to the International Agency for Research on Cancer (IARC), the specialised cancer agency of the WHO: *"Ingested nitrate or nitrite under conditions that result in endogenous nitrosation is probably carcinogenic to humans"* (WHO/IARC, 2010). So bacon does cause cancer...

As many studies point out, an additional harmful effect of a diet rich in animal products is that the foods that help protect against bowel cancer (fruit, vegetables, wholegrains and pulses – rich in fibre) are excluded or replaced with meat and dairy. A Canadian study looked at three different dietary patterns among 1,000 people, half of whom had been diagnosed with bowel cancer, to determine each diet's effect on the risk of the disease (Chen *et al.*, 2015). The meaty-diet pattern was characterised by high amounts of red meat, processed meat, fish and processed fish. The plant-based diet featured fruit, vegetables and wholegrains. A third pattern was named sugary-diet as it consisted largely of pies, tarts, desserts and sweets. Results showed that the plant-based diet conferred a protective effect against bowel cancer, while the meat-diet pattern and the sugary-diet pattern were associated with a greater risk. **Those eating the most meat were 84 per cent more likely to have bowel cancer than those eating the least, whereas those eating the most plant-based foods were 45 per cent less likely to have bowel cancer than those eating the least. The sugary pie and biscuit diet fared badly, pushing the risk up to 126 per cent!**

A high consumption of meat, dairy, processed foods, chips, cakes and biscuits, characteristic of the typical Western diet, plays a strong role in bowel cancer risk. The causal mechanism may involve being overweight or obese, which previous studies have found to be important risk factors for bowel cancer (Chen *et al.*, 2015). Other causes are fat, saturated fat, animal protein, NOCs, HCAs and PAHs. It's not always possible to discern which component is doing the damage. Does it matter? A diet rich in one usually contains the others. Bowel cancer is the leading cause of cancer death among non-smokers in affluent countries and its

prevention should be a major goal for public health. Given what we now know about the harmful effects of meat and bowel cancer, isn't it about time the government amended health guidelines to properly reflect the risks associated with meat?

SUMMARY

- Bowel cancer is one of the most common types of cancer in the UK affecting about one in every 20 people. Wide geographical variation indicates a role for diet and lifestyle factors. High intakes of meat are consistently linked with a higher risk, which is why the WCRF recommend that people limit their intake of red meat and avoid all processed meat.
- A review of 19 studies from the US, Europe, Australia and Japan found that those eating the most red meat had a 28 per cent higher risk and those eating the most processed meat, 20 per cent higher. A Canadian study found that people eating the most meat were 84 per cent more likely to have bowel cancer. Those eating the most plant-based foods were 45 per cent less likely to have the disease.
- Including fibre in a meaty diet is not the answer. A study comparing the diets of African Americans and Native Africans and found that surprisingly, their fibre intake was the same, but African Americans ate more protein, fat, meat, saturated fat and cholesterol, suggesting the higher bowel cancer rate in African Americans is linked to their high intake of animal foods.
- Meat consumption plays a strong role in bowel cancer risk. This may be because meat-eaters are more likely to be overweight or obese. It may be the fat, saturated fat, animal protein or the NOCs, HCAs and PAHs in meat. It may not be possible to work out which of these is doing the damage, but does it matter? A diet rich in one usually contains the others.
- Bowel cancer is the leading cause of cancer death among non-smokers in developed countries. Its prevention should be a major public health concern. Given what we now know about the harmful effects of meat, isn't it high time the government amended their health guidelines to properly reflect the risks associated with meat? Meat-eaters need the same level of health advice as smokers but are not being warned sufficiently.

KIDNEY CANCER

Kidney cancer is the eighth most common cancer in adults in the UK, with just over 10,100 people diagnosed each year (NHS Choices, 2014b). Renal cell carcinoma (RCC) is kidney cancer that starts in the lining of the proximal convoluted tubule – the small tubes in the kidney that transport waste molecules from the blood into the urine. RCC accounts for more than 80 per cent of all kidney cancers. In 2007, the WCRF/AICR report deemed the evidence for meat intake and RCC risk to be inconclusive (WCRF/AICR, 2007). Since then, a number of studies have found that mutagens from meat-cooking are associated with RCC risk.



The large NIH-AARP Diet and Health Study cohort of 492,186 people were used to investigate the link between meat-related compounds and the risk of RCC (Daniel *et al.*, 2012). During nine years of follow-up, 1,814 cases of RCC were diagnosed. Results showed that **those eating the most (62.7g per 1,000 calories) red meat had a 19 per cent increased risk of RCC compared to those eating the least (9.8g per 1,000 calories). For every additional 10g of red meat (per 1,000 kcal), the risk increased by 13 per cent.** BaP (used as a PAH marker, indicating the presence of other PAHs) and PhiP (the most abundant HCA) increased the risk by 20-30 per cent. The authors concluded that it seems plausible that epithelial cells in the renal tubule would be sensitive to metabolic stress from haem iron and other dietary carcinogens related to meat intake.

How meat might cause kidney cancer

Several research groups are now working on identifying the precise mechanism linking meat to kidney cancer. There is a group of enzymes (UDP-glucuronosyltransferases or UGTs) that perform a process called detoxification in the AαC and PhiP) can cause the bioactivation of these enzymes, which means activating them in such a way that they can react to DNA and cause mutations which may lead to cancer. The theory is that this enzyme system, working in the kidney (and liver) to clear drugs, hormones, fats and other compounds, can contribute to the genotoxic effects of HCAs by catalysing the production of reactive compounds that bind to DNA (Cai *et al.*, 2016).

Another study, among residents of Texas in the US, examined associations between the meat-cooking mutagens MelQx and PhiP and the risk of kidney cancer in 659 newly diagnosed cases of RCC and 699 controls (Melkonian *et al.*, 2016). In this case-control study, they observed a nearly two-fold increase in RCC risk associated with MelQx and a 54 per cent increased risk associated with PhiP. The authors said this suggests that the intake of meat cooked at high temperatures may impact the risk of RCC through mechanisms related to mutagenic cooking compounds.

SUMMARY

- Kidney cancer is the eighth most common cancer in adults in the UK. Renal cell carcinoma accounts for more than 80 per cent of all kidney cancers.
- The NIH-AARP Diet and Health Study found that those eating the most red meat had a 19 per cent increased risk compared to those eating the least. The mutagenic cooking compounds found in meat cooked at high temperatures are thought to increase the risk. BaP (a PAH marker) and PhiP (the most abundant HCA) increase the risk by 20-30 per cent. For every extra 10g of red meat, the risk increases by 13 per cent.
- A Texan study found a 54 per cent increased risk associated with PhiP and a nearly two-fold increase associated with another HCA called MelQx. Furthermore, HCAs may alter the behaviour of certain enzymes in the kidney (and liver) in such a way that they react with DNA and cause mutations which can lead to cancer.
- Taken together, meat spells trouble for kidney cancer.

PANCREATIC CANCER AND LYMPHOMA

Pancreatic cancer is the **fourth most common cause of cancer death** worldwide with large geographical variation, which implies diet and lifestyle as contributors as risk factors for this disease (Rohrmann *et al.*, 2013). Around 8,800 people are diagnosed with pancreatic cancer in the UK each year (NHS Choices, 2014c).

Meat intake has been positively associated with pancreatic cancer in many studies. The NIH-AARP Diet and Health Study cohort of 537,302 individuals, aged 50-71, investigated the association between meat, cooking methods, meat-mutagen intake and pancreatic cancer (Stolzenberg-Solomon *et al.*, 2007). During five years of follow-up, 836 pancreatic cancer cases were recorded. Results showed that **men consuming the most grilled and barbecued meat had a 50 per cent increased risk of pancreatic cancer**. Men and women consuming the most DiMeIQx (an HCA found abundantly in well-cooked meat) had a 29 per cent increased risk. These findings indicate that meat, particularly meat cooked at high temperatures, plays a role in the development of pancreatic cancer.

A review of eleven studies, six from the US, four from Europe and one from Japan, found that each **50g per day (about one serving) of processed meat was associated with a 19 per cent increased risk of pancreatic cancer** (Larsson and Wolk, 2012).

Few studies have examined associations between dietary fat and pancreatic cancer and their findings have been inconsistent. This may be due to the small number of patients diagnosed with pancreatic cancer and/or to the narrow range of fat intakes in these studies. The NIH-AARP study included a large number of people and wide range of fat intake from diverse food sources. This study observed **significant links between pancreatic cancer and animal fat, particularly from red meat and dairy foods** but did not observe any consistent association with fat from plant foods (Thiébaud *et al.*, 2009). They concluded that **animal fat is associated with an increased risk of pancreatic cancer**.

Combined data from the EPIC-Oxford cohort and the Oxford Vegetarian Study (including 31,470 meat-eaters, 8,516 fish-eaters, 18,096 vegetarians and 2,228 vegans) found that, compared with meat-eaters, **vegetarians and vegans had around 50 per cent lower mortality from pancreatic cancer** (Appleby *et*

How meat might cause pancreatic cancer

In the review of 11 studies from the US, Europe and Japan, the link between pancreatic cancer and red meat was only observed in men. The authors said that red meat consumption was on average higher in men than in women and suggested that if there is a threshold effect with an increased risk of pancreatic cancer only at high levels of red meat consumption, a positive association may be more likely to be detected in men. Another biologically plausible explanation for the observed differences between men and women is haem iron (abundant in red meat), which could enhance the growth of pancreatic cancer tumours (Stolzenberg-Solomon *et al.*, 2007). Due to normal iron loss during menstruation, women do not accumulate such high iron stores as men. Higher levels of iron in the blood and higher percentage of iron saturation (iron to iron binding capacity) were associated with increased risk of subsequent pancreatic cancer in one prospective study (Friedman and van den Eden, 1997).

al., 2016). When they excluded those who changed diet group during the study (possibly reflecting the onset of illness), compared with regular meat-eaters, vegetarians and vegans had around 50-60 per cent lower mortality. This reflects findings from another study of cancer in British vegetarians (Key *et al.*, 2014). They found the risk of **cancer is generally lower in vegetarians and vegans than meat-eaters, specifically 27 per cent lower for pancreatic cancer**.

A link with poultry was found in the huge EPIC study looking at the links between meat and fish with pancreatic cancer (Rohrmann *et al.*, 2013a). During the study, 865 pancreatic cancer cases were recorded among 477,202 participants from 10 European countries. The consumption of red and processed meat was not associated with pancreatic cancer in this study. However, for every 50g of poultry per day (that's just three chicken nuggets or a third of a chicken breast) the risk of pancreatic cancer increased by 72 per cent.

When the same EPIC team found a similar result for lymphomas and poultry they suggested antibiotics and/or coccidiostats (drugs given to poultry or cattle to prevent the growth and reproduction of certain parasites) may be involved (Rohrmann *et al.*, 2011). Chicken and turkeys are often treated with coccidiostats

and antibiotics to enhance their growth and to treat and prevent disease. The frequency of antibiotic use has been associated with the risk of non-Hodgkin lymphoma in some studies (Chang *et al.*, 2005; Kato *et al.*, 2003).

The EPIC team also suggest another possibility, oncogenic animal viruses. Poultry may contain viruses that cause the development of tumours, especially if the meat is not cooked properly. Oncogenic animal viruses have been suspected as a cause of non-Hodgkin lymphoma among people working with animals or in meat-processing for some time but meat consumption has not been connected with transmission of oncogenic viruses yet. However, studies have found a lower risk of non-Hodgkin lymphoma in women consuming well-done meats instead of rare or rare-medium meats (Chiu *et al.*, 1996; Zhang *et al.*, 1999). So you are damned if you cook it, and damned if you don't!

SUMMARY

- Pancreatic cancer is the fourth most common cause of cancer death. Large geographical variation indicates diet and lifestyle as contributing to the risk of this disease.
- Many studies show that meat increases the risk. The NIH-AARP Diet and Health Study found that men eating the most grilled and barbecued meat had a 50 per cent increased risk. Men and women consuming the most DiMeIQx had a 29 per cent increased risk. Another NIH-AARP study found links with fat, particularly from red meat and dairy foods but not from plant foods.
- Studies from the US, Europe and Japan found that each daily 50g serving of processed meat increased the risk in men by 19 per cent. Men tend to eat more meat but it could be that haem iron was involved and women gained protection by losing iron through menstruation.
- The EPIC-Oxford and the Oxford Vegetarian Study found that vegetarians and vegans were 50 per cent less likely to die from pancreatic cancer than meat-eaters. Another study of British vegetarians found the risk of pancreatic cancer was 27 per cent lower than in meat-eaters.
- Another EPIC study found that for every 50g increase in poultry a day (three chicken nuggets or a third of a chicken breast) the risk of pancreatic cancer increased by 72 per cent. The same team found similar results for lymphomas and suggest drugs and antibiotics given to poultry to enhance growth and to treat disease may be involved.

- These findings provide strong evidence that animal fat and meat play a role in the development of pancreatic cancer.

STOMACH CANCER

Stomach cancer – also known as gastric cancer – is the fifth most common cancer worldwide. Around 7,000 people are diagnosed with it each year in the UK (NHS Choices, 2015c). Unfortunately, as stomach cancer isn't often picked up until the later stages, the outlook isn't as good as for some other cancers. In the UK, around 5,000 people die from stomach cancer each year.

We have known about the links between processed meat and stomach cancer for over a decade. Even small amounts increase the risk and the more you eat, the bigger the risk. A 2006 review of 15 studies (including 4,704 stomach cancer patients) found that **consuming just 30g per day (half an average serving) of processed meat increased the risk of stomach cancer by 15-27 per cent** (Larsson *et al.*, 2006). Seven of the studies specifically looked at the effect of bacon and found those eating the most had a 37 per cent higher risk of stomach cancer compared to those eating the least. They concluded that increased consumption of processed meat is associated with an increased risk of stomach cancer.

In 2015, the WHO announced that red and processed meat increases the risk of bowel cancer. They also reported links between red meat, pancreatic cancer and prostate cancer and processed meat and stomach cancer (WHO/IARC, 2015a). Then in 2016, the WCRF published a report from their Continuous Update Project – the world's largest source of scientific research on cancer prevention and survivorship through diet, weight and physical activity (WCRF/AICR, 2016). They analysed worldwide research on how certain lifestyle factors affect the risk of developing stomach cancer. The report included new studies as well as those included in their 2007 report (WCRF/AICR, 2007).

For the first time, drinking alcohol, eating processed meat and being overweight were linked to an increased risk of developing stomach cancer. They said that there is strong evidence that consuming processed meat increases the risk of stomach cancer. Processed meat was defined as meat having undergone salt-preservation, smoking or fermentation, including sausages, bacon, ham, meatballs, burgers, cold meats and hot dogs. The report also said that some evidence suggests consuming grilled or barbecued meat and fish increases the risk of stomach cancer too.

How meat might cause stomach cancer

Potential mechanisms discussed in the WCRF/IARC report included the high levels of salt, nitrite and nitrate that many processed meats contain. Nitrite and nitrate from processed meat may be involved in carcinogenesis, due to reactions they trigger in the body. In the stomach in particular, nitrite and nitrate from meat can react with the degradation products of amino acids from meat to later form NOCs in the gut which are known carcinogens. Smoked meat is also often salted or cured, meaning that it is likely to raise endogenous production of NOCs. Smoked meat may also contain carcinogenic and mutagenic PAHs, depending on the fuel burned to produce the smoke.

A further potential mechanism linking processed meat intake to stomach cancer described in the report was haem iron which, as already stated, contributes to endogenous formation of NOCs, causes oxidative stress and DNA damage. Dietary iron has been identified as a growth factor for the bacteria *Helicobacter pylori*, an established risk factor for ulcers, inflammation and stomach cancer (Ward *et al.*, 2012). Finally, the salt included in cooking, processing and preserving meat can damage the gastric mucosa (the stomach lining) and lead to inflammation (WCRF/IARC, 2016).

A study from Nebraska investigating the role of haem from meat in stomach cancer found the link with endogenous NOCs was present only among people infected with *H. pylori* and those with relatively low blood levels of vitamin C (Ward *et al.*, 2012). The WCRF/IARC report found evidence that consuming little or no fruit increases the risk of stomach cancer and that consuming citrus fruit may decrease the risk of stomach cancer (WCRF/IARC, 2016). So it seems that many factors may work in combination contributing to an increased or decreased risk of stomach cancer but avoiding meat (especially processed meat) is a simple choice you can make to protect yourself.

SUMMARY

- Stomach cancer is the fifth most common cancer worldwide. Processed meat is associated with an increased risk; just 30g a day (half an average serving) increases the risk by 15-27 per cent. People eating the most bacon have a 37 per cent higher risk than those eating the least.
- In 2015, the WHO reported links between processed meat and stomach cancer. Then in 2016, the WCRF published a report saying that that there is now strong evidence that processed meat increases the risk of stomach cancer. They also said that some evidence



suggests consuming grilled or barbecued meat and fish increases the risk of stomach cancer too. They blame high levels of salt, nitrite and nitrate (that can lead to the production of harmful NOCs) as well as carcinogenic and mutagenic PAHs, haem iron and salt.

- So many factors may be at play, contributing to an increased risk of stomach cancer but avoiding meat (especially processed meat) is a simple choice you can make to protect yourself.

To find out about the foods that fight cancer (fruit, vegetables, wholegrain foods, pulses, healthy fats in nuts and seeds) please see *The Incredible Vegan Health Report* at www.vivahealth.org.uk.

CARDIOVASCULAR DISEASE

Cardiovascular disease (CVD) is a general term that describes diseases of the heart and blood vessels such as heart disease and stroke. Blood flow to the heart, brain or body can be reduced as the result of a blood clot (thrombosis), or by a build-up of fatty deposits or plaques inside an artery that cause the artery to harden and narrow (atherosclerosis). Heart attacks and strokes are mainly caused by blockages that prevent blood from flowing to the heart or brain. High cholesterol levels increase the risk and LDL 'bad' cholesterol is the main source of artery-clogging plaques.

Professor T. Colin Campbell's *China Study* observed very low rates of heart disease in the southwest Chinese provinces of Sichuan and Guizhou between 1973-1975 during which time not one single person died of heart disease before the age of 64 among 246,000 men and 181,000 women (Campbell and Campbell, 2005). Campbell suggested these figures reflect the important protective role of low blood cholesterol levels observed in rural China.

According to the WHO, almost a fifth (18 per cent) of global stroke events and over half (56 per cent) of global heart disease is attributable to cholesterol levels above 3.2mmol/l (WHO, 2003), millimole per litre is the standard unit for measuring cholesterol. Current UK guidelines state that we should aim for a cholesterol level below 5.0mmol/l. A joint report between the Medical Research Council and the British Heart Foundation found that the average cholesterol level for people aged 16 and above in the UK is about 5.5mmol/l and about one-fifth of people have levels

above 6.5mmol/l. In China (where there is much less heart disease), the average cholesterol level in the cities is about 4.5mmol/l for men and women aged 35-64 and levels in the countryside are even lower (MRC/BHF, 2006).

It could be argued that genetic differences between races may affect the risk factors for CVD and other diseases. However, Campbell's observations that Japanese men in Hawaii and California have much higher levels of blood cholesterol and incidence of heart disease than Japanese men in Japan confirms that some risk factors are environmental rather than genetic. In other words, the choices we make about the food we eat and how we live can have a significant impact on heart health.

Campbell also observed that the intake of animal protein correlates directly with heart disease incidence, which he attributes to the cholesterol-raising effect of animal protein. Conversely, Campbell notes that eating plant protein lowers cholesterol (Campbell and Campbell, 2005).

About 610,000 people die of heart disease in the US every year, that's one in every four deaths (CDC, 2015). Around 800,000 people in the US have a stroke each year, where stroke is the fifth leading cause of death and a major cause of adult disability (CDC, 2016). Quantifying the role of meat consumption on these events is of great scientific and public health importance. To address this important question, researchers from the Harvard School of Public Health performed a systematic review of the evidence looking for relationships between red meat, processed meat and both types of meat combined (referred to as total meat) with the risk of heart disease and stroke. They examined 20 studies from 10 different countries (including 1,218,380 individuals, 23,889 cases of heart disease and 2,280 strokes) and found a significantly higher risk among those consuming the most processed meat (Micha *et al.*, 2010). They suggest that the high sodium and nitrate preservative levels used in processed meats could be responsible.

The Nurses' Health Study is one of the largest investigations into risk factors for major chronic diseases ever conducted. It began in 1976 when 121,700 female registered nurses aged 30-55, residing in 11 US states, provided detailed information on their medical history and lifestyle. Every two years, follow-up questionnaires have been sent to update information on potential risk factors and to identify newly diagnosed cases of heart

MEAT THE TRUTH

disease and other illnesses. The large number of participants and high follow-up rate with updated dietary information provides an excellent way of identifying the links between certain foods and disease.

In 2010, the Nurses' Health Study reported on the relationship between major dietary protein sources and heart disease in women (Bernstein *et al.*, 2010). **Higher intakes of red meat and whole-fat dairy products were associated with a higher risk of heart disease**, while higher intakes of nuts, fish and poultry were associated with a lower risk. Again, this doesn't mean that fish and chicken are good for you; it may just show that they are not quite as bad as red and processed meat. Also, it should be remembered that this study dates back to 1980 when the composition of chicken meat was different; now the average broiler hen contains more fat than protein (Wang *et al.*, 2010). The Nurses' Health Study found that **replacing a serving of meat with one of nuts had the greatest effect, lowering the risk of heart disease by 30 per cent**. The usual suspects linking meat and disease were discussed; saturated fat, cholesterol, high sodium, HCAs and haem iron.

Two years later, the same research group looked at the risk of stroke in women from the Nurses' Health Study and a large cohort of men from the US Health Professionals Follow-up Study (43,150 men followed for 22 years and 1,397 strokes). Results also showed that **replacing meat with other sources of protein lowered the risk of stroke** (Bernstein *et al.*, 2012). Replacing a serving of meat with nuts lowered the risk of stroke by 17 per cent.

The same year, the association between red meat and CVD was also investigated in these two large cohorts (Pan *et al.*, 2012). They also found **that high intakes of both red and processed meat were associated with a higher risk**. Most processed meat contains pork or beef, but it may also be made using other red or white meats including chicken and turkey. With each serving per day of red meat and processed meat, the risk of cardiovascular death increased by 18 per cent and 21 per cent respectively. Substituting other sources of protein lowered the risk.

In Europe, a large EPIC study including 493,179 individuals followed over 12.7 years and 5,556 deaths from CVD, revealed an increased risk in cardiovascular death in those consuming high levels of processed

meat. **A 28 per cent increased risk was seen in those consuming more than 160g of processed meat per day** compared to those eating less than 20g (Rohrmann *et al.*, 2013). They concluded that those with a high consumption of processed meat are at increased risk of early death, in particular due to CVD but also to cancer and that **health promotion activities should include specific advice on lowering processed meat consumption**.

People typically think of processed meat as only referring to pork and beef, but this category can also include chicken and turkey. Processed meat is treated by salting, curing or smoking to improve the shelf life, colour and taste of the food. These processes lead to an increased intake of carcinogens or their precursors: NOCs, HCAs and PAHs (Rohrmann *et al.*, 2013). So chemicals used in processed meats may play a role in CVD by damaging blood vessels. In particular, sodium and nitrites might explain the additional harm of processed meats (Pan *et al.*, 2012).

Processed meats such as sausages, salami and bacon also have a higher content of saturated fats and cholesterol than red meat which is often consumed after removing visible fat, whereas the proportion of fat in sausages often reaches 50 per cent of the weight or even more (Rohrmann *et al.*, 2013). Both high saturated fat and cholesterol intake are related to the risk of heart disease.



In contrast to the US studies discussed, this EPIC study found no statistically significant association between red meat and cancer or cardiovascular death. The authors suggested this may be explained by the higher meat consumption in the US than in Europe (Rohrmann *et al.*, 2013). EPIC participants tend to be healthier than the general population. It seems likely that the lower meat consumption, and the removal of fat from red meat among the health conscious, coupled to a healthier lifestyle, masked the harmful effect red meat has in relation to the risk of cardiovascular disease that numerous other studies have revealed.

Beef, pork, poultry and lamb all contain high levels of fat, especially saturated fat and eating a lot of saturated fat can raise cholesterol levels in the blood which increases your risk of heart disease. Cholesterol is a type of fat that travels in the bloodstream. Our bodies need some, but high levels in the blood (particularly LDL or “bad” cholesterol) can build up and block the arteries increasing the risk of CVD. If a clot blocks an artery to the heart, a heart attack may follow. If a clot blocks an artery to the brain, a stroke may occur. Foods that are high in saturated fat (meat and dairy) raise cholesterol levels in the blood by promoting production of cholesterol in the liver. In fact, saturated fat is recognised as the single dietary factor that has the greatest negative effect on LDL ‘bad’ cholesterol (Hu *et al.*, 2001).

Cholesterol levels in the UK are among the highest in the world. Studies show that vegetarians and vegans have lower cholesterol levels, lower blood pressure and a lower risk of diabetes than meat-eaters. As a result, the risk of developing CVD in vegetarians and vegans is significantly lower. A study from the University of Oxford found that **British vegetarians have a whopping 32 per cent lower risk of hospitalisation or death from heart disease than meat-eaters** (Crowe *et al.*, 2013).

Cochrane Reviews are often referred to as the gold standard in science. A 2012 review from The Cochrane Library analysed 48 studies including over 65,000 participants. It was found that reducing saturated (animal) fat, but not total fat intake, reduced the risk of heart attack and stroke by 14 per cent (Hooper *et al.*, 2012). The authors concluded that lifestyle advice to those at high risk of CVD (and probably also to those with a lower risk), should continue to include the permanent reduction of saturated fat and partial replacement by healthier unsaturated fats. There is much evidence that replacing saturated fat with polyunsaturated fat or monounsaturated fat in the form

of olive oil, nuts, seeds and other plant oils can reduce the risk of CVD (Willett *et al.*, 2014).

The idea that high iron stores can increase the risk of CVD was first proposed in the *Lancet* in 1981 (Sullivan, 1981). Sullivan proposed that menstrual blood loss could be responsible for the lower risk of CVD seen among premenopausal women compared to men and postmenopausal women. Since then, other studies have confirmed that high iron stores are a risk factor for CVD (Salonen *et al.*, 1992).

Research suggests that haem iron from red meat, but not non-haem iron from plant foods, is associated with heart disease. In the Netherlands, among a group of 16,136 women aged 49-70, a high haem iron intake from meat was associated with a 65 per cent increase in heart disease risk (van der A *et al.*, 2004). These women had relatively high haem intake intakes; an average of 1.8mg per day, much higher than the 0.5mg per day observed in the UK 2003 NDNS (Henderson *et al.*, 2003). Interestingly because women of reproductive age lose iron by menstruation, the authors hypothesised that these women may gain some protection from iron overload and therefore from heart disease too. If this were true, the relationship between high iron intake and heart disease would be stronger in non-menstruating women. To test their theory, they divided the women into those who had periods and those who didn’t and found that menstruating women had a significantly lower risk of heart disease (10 per cent compared to 58 per cent). So it would seem losing blood every month protects against iron overload and therefore heart disease. An earlier report from the Nurses’ Health Study, this time looking at women with type 2 diabetes, also found that haem iron increases the risk of heart disease (Qi *et al.*, 2007). Again, the authors suggested that losing blood every month conferred protection against heart disease as the association between haem iron and heart disease was stronger in postmenopausal women.

As well as menstruation in women, voluntary blood donation is an important cause of blood loss that can affect iron stores. Numerous studies have shown that regular blood donation can reduce the risk of CVD (Meyers *et al.*, 2002; Tuomainen *et al.*, 1998). The loss of iron associated with giving blood could be the reason for the observed reduction in risk. More recently another study showed that high-frequency blood donation was associated with lower iron stores, improved vascular function and reduced oxidative stress in blood donors (Zheng *et al.*, 2005).

MEAT THE TRUTH

Figure 3.0 illustrates the proposed mechanism underlying the harmful role of haem iron in CVD. It involves iron's role as a catalyst in the formation of free radicals and the subsequent oxidation of biological molecules such as DNA and LDL cholesterol (Hunnicut et al., 2014). This mechanism is thought to underlie the basis of neurodegenerative disorders (such as Alzheimer's disease), certain cancers and CVD (Muñoz-Bravo et al., 2013). The oxidation of LDL cholesterol may be a key initial event in the progression of atherosclerosis and CVD (Niki, 2011). It has also been suggested that low body iron stores may protect against CVD through limiting the oxidation of LDL cholesterol (Meyers et al., 2002).

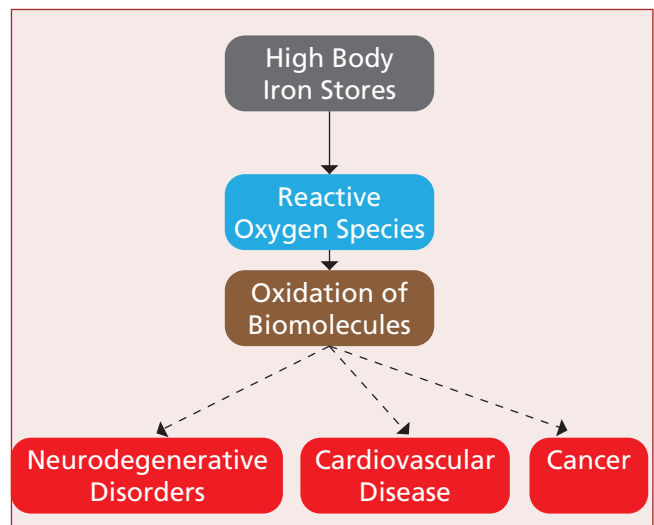
The idea that iron is linked to CVD has been challenged in a small number of studies. In a letter to the *British Medical Journal*, researchers from Finland countered this argument by asserting that all of the negative studies used unreliable measurements of iron status (such as serum iron concentration, transferrin iron saturation) or had other design problem (Hemilä and Paunio, 1997). They said:

"There exists a widespread conviction that the more iron in the diet the better. This is what is still written in medical textbooks. This conviction is, however, the enemy of the truth."

Tomi-Pekka Tuomainen, Research fellow
Riitta Salonen, Senior clinical research fellow
Kristiina Nyyssönen, Clinical biochemist
Jukka T Salonen, Academy professor
Research Institute of Public Health, University of Kuopio,
70211 Kuopio, Finland

So convinced of the link are some researchers that they have suggested reducing our iron intake to reduce the risk of CVD. Some say the evidence may even be strong enough to recommend ending iron fortification and supplementation and to start advising people to donate blood regularly. Of course you could reduce the risk of CVD just by avoiding red meat, processed meat, chicken and seafood which are the main sources of haem iron (Qi et al., 2007). Others caution that given the extent of iron deficiency, any decision to reverse iron fortification and supplementation policy should be based on extremely sound science (Sempos, 2002). The obvious solution is to obtain non-haem iron from plant foods as the body only absorbs as much of this type of iron as it needs. The absorption of haem iron from meat is unregulated; it is absorbed in the gut whether it is needed or not so excessive consumption can lead to iron overload and an increased risk of CVD.

Figure 3.0 Oxidative stress caused by high iron stores in the body.



The NIH-AARP Diet and Health Study found that red and processed meats were linked to early death in both men and women including that from cancer and CVD (Sinha et al., 2009). White meat (chicken and turkey) did not have the same links but the authors of the study did point out that processed meat included luncheon meats and cold cuts made of red and white meat and low-fat hotdogs made from chicken or turkey. They said that red and white processed meat products can overlap as both can include bacon, sausage, ham, chicken and turkey.

The sensible approach is to eat a well-balanced vegan diet containing plenty of iron-rich plant-based foods such as pulses (peas, beans, lentils and soya bean products such as soya milk and tofu), dark green leafy vegetables (such as broccoli, bok choy and watercress), fortified breakfast cereals, wholegrains (such as wholemeal bread, brown rice and wholemeal pasta), dried fruits (such as raisins, prunes, apricots and figs) black treacle and, in moderation, plain dark chocolate.

Taken together, these scientific findings support the links between meat and CVD, one of the UK's biggest killers. This evidence supports the idea that CVD risk can be reduced by a diet that provides more plant-based sources of protein compared with the typical Western diet (Richter et al., 2015). Put simply, ditching meat lowers your risk of suffering a heart attack or stroke.

SUMMARY

- Heart disease and stroke are caused by blockages in the arteries that prevent blood flow to the heart or brain. High cholesterol increases the risk and LDL 'bad' cholesterol is the main cause. The WHO say 56 per cent of heart disease and 18 per cent of strokes are caused by cholesterol levels above 3.2mmol/l. UK guidelines recommend aiming for a level below 5.0mmol/l but levels in the UK are among the highest in the world averaging around 5.5mmol/l.
- Geographical variation and migrant studies show that heart disease is caused by lifestyle and environmental factors. Many studies have looked at the effects of diet and meat specifically. Researchers from the Harvard School of Public Health found a much higher risk among people eating high levels of processed meat. The NIH-AARP Diet and Health Study found that red and processed meats were linked to early death in men and women, from cancer as well as CVD.
- Results from the Nurses' Health Study showed that red meat is linked to a higher risk of heart disease and replacing one daily serving of meat with nuts lowered the risk by 30 per cent. The Nurses' Health Study and the US Health Professionals Follow-up Study together found that replacing meat with healthier protein also lowered the risk of stroke. A third study, combining both groups again, found that each daily serving of red and processed meat increased the risk of death from CVD by 18 per cent and 21 per cent, respectively.
- The EPIC study found that people eating more than 160g of processed meat a day were 28 per cent more likely to have CVD than those eating less than 20g. They did not find the substantive evidence against red meat that other studies have found but EPIC participants eat less meat and are healthier than the general population, this may have masked the harmful effects of red meat. The facts remain that foods that are high in saturated fat (meat and dairy) raise cholesterol which is a risk factor for CVD.
- A review from the Cochrane Library found that reducing saturated animal fat (but not total fat), reduced the risk of CVD by 14 per cent. They say lifestyle advice should continue to include reducing saturated fat and replacing some of it with healthier unsaturated fats.
- Other substances in meat have been linked to CVD; haem iron increases the risk of heart disease, which is why lower rates are seen in women who menstruate and people who donate blood regularly.
- Vegans have lower cholesterol, lower blood pressure and a lower risk of CVD. British vegetarians have a whopping 32 per cent lower risk of hospitalisation or death from heart disease than meat-eaters.
- The risk of heart disease and stroke can be significantly reduced by a diet that provides more plant-based sources of protein compared with the typical Western diet. Put simply, ditching meat lowers your risk of suffering a heart attack or stroke.



OBESITY AND WEIGHT GAIN

Around two-thirds of adults and a third of children in the UK are overweight. Of these, one in every four adults and around one in every five children (aged 10-11), are obese – the UK has become the ‘fat man of Europe’ (NHS Choices, 2015d). The UK has the highest level of obesity in Western Europe, ahead of France, Germany, Spain and Sweden. Obesity levels in the UK have more than trebled in the last 30 years. In 2010, a team of experts, led by Professor Klim McPherson at Oxford University, predicted that by 2020, eight out of 10 men and almost seven in 10 women will be overweight or obese (Brown *et al.*, 2010). By 2050, more than half the population could be obese. McPherson’s study says this would lead to a 23 per cent rise in the prevalence of obesity-related stroke, a 34 per cent rise in obesity-related high blood pressure, a 44 per cent rise in obesity-related heart disease and a 98 per cent rise in obesity-related diabetes.

Being overweight increases the risk of a range of health problems including diabetes, high blood pressure and up to ten different types of cancer. It can also impair a person’s well-being, quality of life and ability to work. Compared with a healthy weight man, an obese man is:

- five times more likely to develop type 2 diabetes
- three times more likely to develop bowel cancer
- more than two and a half times more likely to develop high blood pressure – a major risk factor for stroke and heart disease

An obese woman, compared with a healthy weight woman, is:

- almost 13 times more likely to develop type 2 diabetes
- more than four times more likely to develop high blood pressure
- more than three times more likely to have a heart attack

NHS Choices, 2015d.

McPherson says: *“We are being overwhelmed by the effects of today’s ‘obesogenic’ environment, with its abundance of energy-dense food and sedentary lifestyles”.*

Research has shown that we have a natural tendency to store fat – it’s a survival mechanism that helped early humans survive famine and food shortages. However, a

wide variety of foods are now available in abundance and it can be tempting to overeat and to go for unhealthy types of food – high in sugar and fat.

Most unhealthy saturated fat in the average UK diet comes from: fatty cuts of meat, poultry skin, meat products such as sausages and pies, whole milk and full fat dairy products, coconut oil and palm oil, pastry, cakes and biscuits, sweets and chocolate. The government recommends eating less of these foods and more foods containing unsaturated fats such as avocados, nuts, seeds, plant-based oils and spreads.

Somehow, chicken continues to slip under the net in many health studies that focus on red and processed meat. This does not mean it is a healthy option. Some researchers have suggested that people who replace red meat with chicken do so in an attempt to improve their health. These people are also less likely to smoke, consume less alcohol and take regular physical exercise.



These lifestyle patterns may mask the harmful effects of white meat that would be more apparent otherwise.

Poultry now accounts for nearly half of all the meat bought in the UK; British people are currently eating an estimated 2.2 million chickens per day. But is it really a healthy option? Would you be better off replacing chicken with chickpeas? Is chicken as low in fat as we have been led to believe? Professor Michael Crawford, of London Metropolitan University, found that modern organic and non-organic broiler chickens sold for human consumption provide more energy from fat than from protein (Wang *et al.*, 2010). This is not common knowledge!

In 1976, the Royal College of Physicians and the British Cardiac Society recommended replacing fatty red meat with poultry because it was considered to be lean and therefore lower in fat than other meats. However, the situation has changed drastically since then, with a striking increase in fat content of the standard broiler chicken (that's the common type of bird sold in supermarkets). Professor Crawford set out to put the record straight and provide a snapshot of data on fat in chickens now sold to the public. Crawford's team collected samples between 2004-2008 from UK supermarkets and farm shops. They measured the fat content and found that **the chickens they sampled contained more fat than protein!** (Wang *et al.*, 2010). They concluded that in view of the obesity epidemic, chickens that provide several times the fat energy compared with protein seem illogical and said this type of chicken husbandry needs to be reviewed with regard to its implications for animal welfare and human nutrition.

There are growing concerns about health impacts of the increasing consumption of so-called fast foods and takeaway foods on health, of which chicken nuggets, wings and drumsticks play an increasing role. A 2015 review of the evidence found that frequent consumption of fast foods is accompanied with overweight and abdominal fat gain, impaired insulin and glucose balance, lipid and lipoprotein disorders, induction of systemic inflammation and oxidative stress increasing the risk of diabetes, metabolic syndrome and cardiovascular disease (Bahadoran *et al.*, 2015).

Chemical analyses of 74 samples of fast-food menus consisting of French fries and fried chicken (nuggets/hot wings) bought in McDonalds and KFC outlets in 35 different countries in 2005-2006 showed that the total fat content of the same menu ranged

from 41 to 65g at McDonalds and from 42 to 74g at KFC (Stender, 2007). The government say we should consume no more than 70g of fat a day. So this could be all the day's fat in one meal! KFC's own website says that, for example, their Mighty Bucket For One contains 67.3g of fat of which 11.1g is saturated! We're advised to eat less fat, especially saturated fat. UK health guidelines recommend that the average man should eat no more than 30g of saturated fat a day and the average woman should eat no more than 20g of saturated fat a day.

In the documentary film *Super-Size Me*, independent filmmaker Morgan Spurlock ate McDonald's food three times a day for 30 days and gained 11kg (Spurlock, 2004). His cholesterol went up from just over 4mmol/l (a healthy number) to 6mmol/l, he experienced mood swings, sexual dysfunction and fat accumulation in his liver. It took Spurlock fourteen months to lose the weight gained from his experiment using a vegan diet.

It's not just fast food and takeaway meat products that are causing problems. The National Health and Nutrition Examination Survey (NHANES) study looked at associations between general meat consumption and weight among US adults (Wang and Beydoun, 2009). Results showed that those who ate the most meat were **around 27 per cent more likely to be obese and 33 per cent more likely to have central obesity** compared to those eating the least. This is most likely to be due to their higher energy and fat intake. Indeed, those eating the most meat consumed around 700 more calories per day than those consuming the least.

Meat intake is related to weight gain because of its high energy and fat content. The more calories you eat, the more weight you gain. However, one of the largest nutrition studies ever, the European Prospective Investigation into Cancer and Nutrition-Physical Activity, Nutrition, Alcohol, Cessation of Smoking, Eating Out of Home and Obesity (EPIC-PANACEA) project, found that meat consumption was significantly associated with weight gain and **the link remained even after controlling for calorie intake** (Verghnaud *et al.*, 2010).

They investigated the link between total meat, red meat, poultry and processed meat and weight gain over five years among a total of 103,455 men and 270,348 women aged 25-70, recruited from 10 European countries. Results confirmed meat consumption was associated with weight gain in both men and women, no surprises there. However, an intake of 250g meat per day (≈450 calories) would lead to an annual weight gain

MEAT THE TRUTH

of nearly half a kilogram *more* than the weight gain experienced by someone eating a diet containing the same number of calories but with a lower meat content! After five years the weight difference would be 2kg. The strongest link with annual weight change was observed for poultry. However, when subjects with previous illness and those likely to lie about their diets were excluded, the association of weight gain with poultry dropped and the strongest link was seen with processed meat.

These results contradict the theory behind high-protein diets (such as the Atkins diet) that suggest eating lots of meat and animal protein makes you feel full and want to eat less and so helps you lose weight. The result are however in agreement with most previous studies that show a positive link between meat intake and weight gain suggesting a decrease in meat consumption can help weight management.

The EPIC-PANACEA study was challenged by a group of scientists (one whom serves on a speaker's bureau for the National Cattlemen's Beef Association). They suggested the additional weight gain in the meat-eaters may have been muscle mass and said the study should have assessed body composition by measuring body fat (Astrup *et al.*, 2010). A number of the EPIC-PANACEA team responded repeating the analyses in a smaller group of their study including 91,214 people (Vergnaud *et al.*, 2010a). They measured their waists as an indicator of abdominal fat. In agreement with their original finding, they found that meat consumption was positively associated with an increase in waist circumference (0.76cm increase after five years for every 100 calorie increase in daily meat intake). They acknowledged the usefulness of assessing body composition to address further the association between meat and weight gain. The National Cattlemen's Beef Association did not respond.

It's unclear why meat-eaters appear to gain more weight than people eating the same number of calories but with less or no meat. The EPIC-PANACEA analysis accounted for differences in BMI, physical activity, educational level, smoking status, total energy intake and plausible misreporting. They did suggest a theory; if meat-eaters acquire all detrimental lifestyle features and if the sum of all these together is more than the sum of each individual effect, this could account for the results seen.

Other studies show how meat intake meat is associated with higher fasting glucose and insulin concentrations, which promotes the absorption and conversion of

glucose into either glycogen or fat (Fretts *et al.*, 2015). How the consumption of meat may influence glucose and insulin levels is still open to speculation; it may be to do with NOCs in processed meat or formed within the body, which have a toxic effect on pancreatic beta cells and promote diabetes. Haem iron, advanced glycation end products and amino acids (eg leucine), may also influence pancreatic beta cell function, insulin secretion and the pathogenesis of diabetes. The point is, **meat impairs glucose metabolism and makes people fatter than people eating the same number of calories but without the meat.**

The EPIC-PANACEA study team also found that diets containing high levels of protein, at the expense of fat or carbohydrate, were also positively associated with weight gain, especially when they missed out on carbohydrates that are rich in fibre (Vergnaud *et al.*, 2013). Those eating diets with more than 22 per cent of energy from protein had a 23-24 per cent higher risk of becoming overweight or obese compared to those eating diets with no more than 14 per cent of energy from protein. So high-protein diets are not advisable for weight loss. It has been suggested that in addition to restricting calories, a possible reason Atkin's dieters lose weight is that because their diet is so monotonous, they tend to eat smaller amounts of food when allowed fewer food choices in the meal (Astrup *et al.*, 2004). A bit like the study suggesting that cider vinegar might help with weight loss because it makes people feel nauseous! (Darsi *et al.*, 2014).

Research analysing data from 170 different countries resulted in an important finding in two papers (You and Henneberg, 2016; You and Henneberg, 2016a). After adjusting for factors such as people's activity levels, income, lifestyle and calorie consumption, meat intake was directly and significantly linked to excess weight. In fact, meat turned out to be as bad as sugar and these two food groups together explain almost all the variation in people's body weights. Both studies describe why and how meat is linked to obesity. If we eat more than enough food, the fats and carbohydrates are digested first and supply all the energy we require. Meat protein is digested later and the body only needs to use a small proportion of it so most of the energy it provides is surplus to requirements and is converted to fat which is then stored in your body. The authors conclude that public health strategies should be put in place to reduce meat consumption.



SUMMARY

- Obesity levels in the UK are rising and by 2020 eight out of 10 men and almost seven in 10 women could be overweight or obese. Being overweight increases the risk of diabetes, high blood pressure, heart disease and several different types of cancer.
- The body has a natural tendency to store fat, so if you eat lots of fatty foods like meat, dairy foods, cake and biscuits, you gain weight. Even lean cuts of meat contain relatively high fat levels compared to plant foods. Chicken is not the answer as modern supermarket chickens contain more fat than protein! Just one meal from KFC (or McDonalds) can contain more fat than you should eat in an entire day. It took filmmaker Morgan Spurlock 14 months to lose the 11kg he gained eating at McDonalds three times a day for a month.
- The NHANES study found that those who eat the most meat are around 30 per cent more likely to be obese and have central obesity (a fat tummy), which increases the risk of diabetes.
- People who eat meat tend to consume more calories than vegetarians and vegans. However, the EPIC-PANACEA study found that even when they eat the same number of calories, meat-eaters gain more weight. They also found that people who eat lots of protein, at the expense of fat or carbohydrate (containing fibre), gain more weight too. Both results put the theory behind the Atkins diet in a dim light!
- Research from 170 different countries shows that meat intake is directly linked to weight gain. Another study suggesting that meat is as bad as sugar found that if we eat more than enough food, fats and carbohydrates are digested first for energy and the energy in meat protein (not combined with fibre like plant protein) ends up being stored as fat. They say that public health strategies should be put in place to reduce meat consumption.
- There is a wealth of research showing how a low-fat vegan diet can help achieve and maintain healthy weight. See *The Incredible Vegan Health Report* at www.vivahealth.org.uk for more details.

BONE HEALTH

Osteoporosis (meaning porous bones) occurs when calcium is lost from the bones and they become more fragile and prone to fracture. It is sometimes called the silent disease as there are often no symptoms until a fracture occurs. Hip fracture constitutes the most serious complication of osteoporosis and accounts for the majority of fracture-related deaths and healthcare costs among individuals over 50. In the UK, one in two women and one in five men over 50 experience fractures, mostly as a result of decreased bone density (National Osteoporosis Society, 2016).

Many risk factors for osteoporosis have been identified including a low BMI, low bone mineral density, reduced sunlight exposure (essential for vitamin D production in the skin), early menopause, smoking, alcohol consumption, low physical activity levels and obesity – so being underweight or overweight can increase the risk.

In 2012, the WHO Collaborating Centre for Metabolic Bone Diseases, at the University of Sheffield Medical School in the UK published a review of hip fracture incidence worldwide (Kanis *et al.*, 2012). They observed a greater than 10-fold variation in hip fracture risk between countries. The highest levels were seen in North Western Europe (Iceland, UK, Ireland, Denmark, Sweden and Norway) through to central Europe (Belgium, Germany, Austria, Switzerland and Italy) to the south east (Greece, Slovenia) and onwards (to the Lebanon, Oman and Iran). Other high-risk countries for women were Hong Kong, Singapore, Malta and Taiwan.

Regions of moderate risk included Oceania (the islands of the tropical Pacific Ocean, Australia and New Zealand), the Russian Federation, the southern countries of Latin America and North America. However, if you separate the ethnic groups in the US, then Hispanic, Asian and Black populations would be classed low-risk and Caucasian women, high risk.

Low-risk regions included the northern regions of Latin America, Africa, Jordan and Saudi Arabia, India, China, Indonesia and the Philippines.

In general, fracture rates are highest in Caucasian women living in temperate climates and are lower in women from Mediterranean and Asian countries and lowest still among women in Africa. Countries in economic transition, such as Hong Kong, have seen significant increases in fracture rates in recent decades (WHO, 2003). The incidence of hip fracture is escalating worldwide and 50 per cent of the total hip fracture incidence is projected to occur in Asia by 2050 (Dai *et al.*, 2014). This shows that environmental factors, such as diet, are responsible.

This view is supported by changes in fracture risk in immigrant populations. For example in the US, black Americans have a lower fracture risk than Caucasians, but a much higher risk than black Africans. A similar scenario is seen among the Japanese population of Hawaii compared to those in Japan and Chinese people living in Singapore compared with mainland China (Kanis *et al.*, 2012).

The role of diet was revealed in the Singapore Chinese Health Study which investigated dietary patterns and fracture risk among 63,257 Chinese men and women (Dai *et al.*, 2014). Two distinct dietary patterns were identified: the vegetable-fruit-soy pattern, characterised by vegetables, fruit and soya foods, and the meat-dim-sum pattern, rich in meat and refined starchy foods. Results showed that compared to the meaty diet, the Chinese diet rich in vegetables, fruit and soya products was associated with a substantially lower risk of hip fracture.

In *The China Study*, one of the most comprehensive nutritional studies ever undertaken, Professor T. Colin Campbell said that there is little evidence to show that increasing calcium intake will prevent fractures. In fact, research is moving in the opposite direction, showing that the more dairy and animal protein that is consumed, the higher the incidence of osteoporosis (Campbell and Campbell, 2005). Unfortunately, most medical advice focuses on calcium intake rather than looking for the reasons for calcium loss which include salt, caffeine, tobacco, lack of exercise and maybe alcohol as well as animal protein.



THE ACID-ALKALINE HYPOTHESIS

The hypothesis that a high animal protein diet could be a risk factor for osteoporosis dates back to research conducted more than 40 years ago (Barzel and Jowsey, 1969). The hypothesis proposes that as food is digested, acids are released into the blood and the body tries to neutralise the acid by drawing calcium from the bones. This calcium is then excreted in the urine (the calciuric response). Animal proteins from meat, dairy, fish and eggs are thought to have a particularly bad effect because of the greater amount of sulphur-containing amino acids they contain compared to most plant proteins. Sulphur-containing amino acids give rise to sulphuric acid when they are broken down in metabolism.

We are probably better adapted to the type of diet our ancestors were exposed to during millions of years of evolution than to the diet we have been eating since the agricultural revolution 10,000 years ago – or since industrialisation only 200 years ago (Frassetto *et al.*, 2001). Modern diets are more acid-forming than the alkalisng foods that would have been consumed. Consider a cheeseburger with fries and a fizzy drink compared to nuts, seeds, fruit, leaves and water with the occasional piece of meat and/or fish... Consequently, humans are not adapted to contemporary acid-forming diets which contribute to modern epidemics of chronic disease (Scialla and Anderson, 2013).

A substantial body of evidence links animal protein to a decrease in bone mineral density. A study, looking at hip fracture incidence in 33 different countries in relation to consumption of plant and animal protein, found that the **countries with the lowest fracture rates also had the lowest intakes of animal protein** (Frassetto *et al.*, 2000). In 10 of the 11 countries with the highest fracture rates, animal protein intake exceeded plant protein intake. The authors said that **hip fracture incidence is directly related to animal protein intake** and suggested that bone integrity is compromised by acid that results from the metabolism of animal protein. They suggested that the moderation of animal food intake, coupled to an increased ratio of vegetable to animal food consumption, may confer a protective effect.

Another study of 1,035 elderly women found that **those with a high ratio of animal to vegetable**

protein intake had a greater risk of hip fracture compared to those with a low ratio (Sellmeyer *et al.*, 2001). A further study of 757 young girls in urban Beijing in China, compared the effects of protein intakes from animal and plant sources on bone mass accrual over five years (Zhang *et al.*, 2010). Results showed that **protein from animal foods, particularly meat, had negative effects on bone mineral content**.

Another study compared the effects of animal and plant protein in the diets of overweight and obese postmenopausal women dieting (Campbell and Tang, 2010). They found that **the energy-restricted diet with meat promoted bone loss** compared with the energy-restricted diet without meat. They warned that for postmenopausal women trying to lose weight, choosing a diet containing meat may reduce bone mineral density and increase the risk of osteoporosis. This extends the findings of an earlier study which examined the levels of bone loss in 1,600 older women and found that **vegetarians had lost only 18 per cent bone mineral compared to omnivores who had lost 35 per cent bone mineral by the age of 80** (Marsh *et al.*, 1988).

So, for children and adolescents, while a good protein intake is important for bone development, research suggests that large intakes of animal protein may counter this positive effect. In a study looking at long-term protein intake, dietary acid load and bone status in children, it was concluded that the positive effect of protein could be negated, at least partly, by a high renal acid load (Alexy *et al.*, 2005). These findings support the health benefit of a diet rich in alkali-yielding fruit and vegetables and the authors recommend an integrative approach saying that focusing on single nutrients is not sufficient. Other studies showing that animal protein-based diets with the same amount of protein as a vegetarian diet can increase the risk for uric acid tones (Breslau *et al.*, 1988) have led some to suggest that high calcium losses in the urine caused by animal protein may be a risk factor for the development of osteoporosis.

A number of studies have examined the role of the dietary acid load in people with chronic kidney disease. The evidence supports a direct relationship between a high dietary acid load and chronic kidney

disease progression, bone loss and sarcopenia (loss of skeletal muscle). However, due to a wide variety of techniques and terminology used to quantify the dietary acid load, this theory is not widely appreciated by nephrologists (Scialla and Anderson, 2013). A number of critical reviews of the acid-alkaline hypothesis have been published (Darling *et al.*, 2009; Fenton *et al.*, 2009; Fenton *et al.*, 2011). These reviews argue that a causal association between dietary acid load and osteoporosis is not supported by the research.

One critic argues that if bone is the main source of calcium from which diet-related acid is buffered, all the bone in the body would be dissolved in just a few years (Bonjour, 2005). It is also argued that homeostatic mechanisms ('housekeeping' systems that attempt to keep everything running normally) including renal acid excretion, would not permit a steady-state low-grade metabolic acidosis caused by a typical Western diet. In other words, the body has ways of redressing the balance when, for example, the diet increases acid levels in the blood, and even small increases are countered by these mechanisms – well that's the theory anyway. However, it has been demonstrated that **a high dietary acid load, which lies within the ranges seen in a typical American or European diet, can increase the acidity of the blood** (Frassetto and Sebastian, 2013). So, on the one hand we are told that we can compensate for the acidifying effects of a high-protein diet, while on the other hand, the research shows that we may not be able to balance it out completely. It may be that the truth lies somewhere between these two apparently irreconcilable arguments.

Buffers are chemical substances that can minimise changes in a liquid when it becomes more acidic or alkaline. To maintain equilibrium whilst there is an increased amount of acid in the body, at least three compensatory responses are activated: buffering from the bone (and to some degree skeletal muscle), increased ventilation to eliminate carbon dioxide, and in the kidney, bicarbonate is generated and reabsorbed into the blood while excess hydrogen ions are secreted into the urine. In healthy people, these buffering systems all have a tremendous capacity to maintain the blood pH (acid-alkali balance) within a very narrow margin (Kerstetter *et al.*, 2009). However, the major reservoir of alkalis (in the form of alkaline salts of calcium) is the skeleton,

which provides the buffer needed to maintain blood pH and plasma bicarbonate concentrations (Pizzorno *et al.*, 2010). While kidney metabolism represents a major mechanism by which metabolic acid loads are handled by the body, **if the kidneys are overwhelmed or compromised (kidney function declines with age), calcium from the bones may be called on to compensate for the increasingly acidic environment** and an alkalising diet could help redress the balance (Dargent-Molina *et al.*, 2008; Frassetto and Sebastian, 2013). So, under certain conditions, **the acid-alkaline hypothesis may provide a plausible mechanism in which a vegan diet, rich in fruit and vegetables, could help promote and preserve bone health**. This may go some way in explaining the apparently contradictory evidence concerning animal protein, meat and bone health.

The acid-alkaline hypothesis has also been challenged on the basis of a series of short-term experimental studies suggesting that high-protein diets are not harmful to bone health and might actually be beneficial by improving calcium absorption (Kerstetter *et al.*, 2003). However, while high-protein diets may increase calcium absorption, they also increase calcium *excretion* in the urine. Therefore, the positive effects of protein intake on calcium absorption may only be beneficial under conditions of adequate calcium intake (Mangano *et al.*, 2014).

Growing evidence suggests that calcium and protein may interact in terms of bone health and that the potential harmful effect of a high-protein diet may only be compensated for if there is an adequate calcium intake (Weikert *et al.*, 2005). If there is insufficient calcium in the diet to counter the calciuric effect, calcium may be lost from the bone. At the same time, high calcium intake may have adverse effects (tissue calcification, kidney stones) so is not desirable either.

One study (of just 13 participants) compared a moderate animal protein intake to a high animal protein intake diet (1g per kg body weight compared to 2.1g per kg – the generally accepted daily protein dietary allowance is 0.8g per kg of body weight) and found with 800mg of calcium per day, all participants ended up in negative calcium balance (Kerstetter *et al.*, 2005). This was not anticipated and the authors suggested that that much calcium was not enough to maintain calcium balance. However, because the

extra urinary calcium lost in the high-protein diet was found to come from the diet (as opposed to the bone), the authors concluded that, at least in the short term, high-protein diets are not detrimental to bone health.

While, the increased amount of urinary calcium produced by the high-protein diet may reflect enhanced calcium absorption and not bone resorption, under both the moderate and high protein diets tested, the vast majority (over 90 per cent) of the calcium found in the urine still came from the bones. Adult bones constantly undergo remodelling through bone resorption by osteoclasts and bone formation by osteoblasts. In adults, almost the entire human skeleton may be remodelled over a 10-year cycle.

While this study suggests that higher protein intakes may not be harmful in the short term, it should be remembered that they can lead to kidney problems and increased levels of IGF-1 which are linked to certain cancers. The long-term impact of high-protein diets on bone health is still unclear and the relative contribution of calcium from the bone and/or diet to protein-induced calciuria remains controversial.

Other studies suggest that high-protein diets may increase calcium absorption and help preserve bone mass by stimulating IGF-1, a potent bone growth stimulator (Mangano *et al.*, 2014). However, **increased IGF-1 levels are linked to an increased risk of certain cancers, so high animal protein diets are not desirable and should not be recommended.**

This is a complex issue with a wide range of factors involved, not least the role of the kidney. It should be noted that people who consume a high-animal protein diet have an increased risk of kidney disease and continuing to consume high levels of meat, eggs and dairy foods may present a burden on an already overworked kidney. It seems logical that the harder you make the kidneys work, the more likely they are to struggle to meet the challenge. There is a consensus that in people with kidney disease or poor kidney function (resulting from aging), a high dietary acid load may result in acidosis which may then lead to bone and muscle loss.

One study found that a diet incorporating increased fruit and vegetable intake produced substantially less acid than the typical American diet rich in meat and dairy (Scialla and Anderson, 2013). They suggested that in chronic kidney disease and aging, a high dietary acid load (caused by a higher intake of meat, eggs, cheese and cereal grains and a lower intake of fruits and vegetables) may result in low-grade, subclinical acidosis. In these circumstances, efforts to maintain stable blood pH and boost acid excretion from the kidney may lead to bone and muscle loss and further decline in kidney function, but this may be mitigated by alkali. In other words, this unnecessary strain on the kidneys could be avoided simply by eating less acid-forming foods. They concluded more studies are needed to determine the degree of benefit of a foods-based approach to reducing the dietary acid load in patients with early to moderate chronic kidney disease.

The acid-alkaline hypothesis remains controversial. Currently, research that supports both the proponents

and opponents of the hypothesis exist (Frassetto and Sebastian, 2013). The pattern of incidence of osteoporosis around the world certainly suggests that some aspect of the typical Western lifestyle is a significant contributing factor to bone loss. Furthermore, the evidence shows that **diets rich in animal protein are harmful to bone health** but more research is needed. In the meantime, it seems sensible to observe how the Western diet is accompanied by the so-called Western diseases including osteoporosis and limit, if not eliminate, all meat from the diet.

Research suggests that physical activity or exercise (especially weight-bearing) is the most critical factor for maintaining healthy bones, followed by improving diet and lifestyle; this means eating plenty of fresh fruit and vegetables, and cutting down on caffeine and avoiding alcohol and smoking.





SUMMARY

- Osteoporosis is a serious problem in the UK, where one in two women and one in five men over 50 experience fractures, mostly as a result of low bone strength. The highest rates are seen in the countries that consume the most animal foods. As the consumption of meat and dairy foods increases, so do fracture rates.
- The Singapore Chinese Health Study found that a diet rich in meat and refined starchy foods was linked to a much higher risk of hip fracture than a traditional diet, characterised by vegetables, fruit and soya foods.
- There is little evidence that increasing calcium intake will prevent fractures. In fact, research shows that the more dairy and animal protein is consumed, the higher the incidence of osteoporosis. The acid-alkaline hypothesis offers a plausible explanation as to how this occurs and although it has been challenged, a high dietary acid load (as seen in a typical Western diet), can increase the acidity of the blood.

- The pattern of incidence of osteoporosis around the world certainly suggests that some aspect of the typical Western lifestyle is a significant contributing factor to bone loss. Furthermore, the evidence shows that diets rich in animal protein are harmful to bone health. It seems likely that meat is as damaging, if not more so, than milk as it doesn't even contain calcium – and of course plant-based sources of calcium are best.
- Research shows that physical activity (especially weight-bearing) is the most critical factor for maintaining healthy bones, followed by improving diet and lifestyle; this means eating plenty of fresh fruit and vegetables, and cutting down on caffeine and avoiding alcohol and smoking.

For more information see Viva!Health's fully-referenced scientific report *Break Free – How to Build Healthy Bones and What Really Matters in the Prevention of Osteoporosis* at: www.vivahealth.org.uk/bones.

DIABETES

Diabetes mellitus is a chronic disease caused by too much sugar (glucose) in the blood. High levels of glucose in the blood can cause damage to the nerves and blood vessels if left untreated. Insulin is an important hormone secreted by beta cells in the pancreas. It regulates blood glucose levels but when things go wrong, glucose can accumulate. Without treatment diabetes can lead to long-term health problems including kidney failure, gangrene, sensory loss, ulceration, blindness and CVD.

Type 1 diabetes (insulin-dependent or juvenile-onset diabetes)

Type 1 diabetes occurs when the body produces little or no insulin as the result of the autoimmune destruction of the insulin-producing cells in the pancreas. That's when the body's immune system attacks its own cells. This type of autoimmune response is thought to involve a genetic predisposition (diabetes in the family) coupled to an environmental trigger. However, genetic susceptibility seems to be less of a factor as a prerequisite for developing type 1 diabetes now than it was in the past (Atkinson *et al.*, 2014).

Scientists have identified a number of genes linked to type 1 diabetes and developed a 'hierarchy of susceptibility'; the more of these genes you have, the higher the risk of developing type 1 diabetes.

Researchers from the Diabetes and Metabolism Division of Medicine at the University of Bristol looked at the frequency of these high risk genes in a group of 194 people diagnosed with type 1 diabetes as children over 50 years ago (1922-1946). They compared them to a group of 582 age- and sex-matched people diagnosed between 1985-2002. Results showed that the frequency of high-risk genes was 12 per cent lower in those diagnosed recently compared with the older group (Gillespie *et al.*, 2004). Other studies from Finland and the US have found a similar disparity (Hermann *et al.*, 2003; Vehik *et al.*, 2008). This suggests that increasing environmental exposure is now able to trigger type 1 diabetes in people who are less genetically susceptible than the generation above them. In other words, the rapid rise of type 1 diabetes must be due to major environmental factors rather than genes.

At the moment, there is no cure for type 1 diabetes, so treatment involves regular injections of insulin to keep blood glucose levels as normal as possible to prevent health problems developing later in life.

Globally, the incidence of type 1 diabetes varies substantially (Atkinson *et al.*, 2014). It is most common in Finland with more than 60 cases per 100,000 diagnosed per year. It is uncommon in China, India and Venezuela (around 0.1 cases per 100,000). Wide variations in incidence even occur between neighbouring areas in Europe and North America, but the general



MEAT THE TRUTH

trend is upwards. Over the past 60 years, the worldwide incidence of type 1 diabetes has been increasing by 3-5 per cent per year, doubling approximately every 20 years with a rapid increase in the number of very young children affected (TEDDY Study Group, 2008). In Europe, the most substantial increases have been noted in children under five (Atkinson *et al.*, 2014).

So how does diet affect our risk of diabetes and what can we do to lower our risk? A convincing body of evidence suggests that proteins found in cow's milk and cow's milk infant formulas can act as an environmental trigger leading to type 1 diabetes. This is discussed at length in Viva!Health's *White Lies* report: www.vivahealth.org.uk/resources/scientific-reports/white-lies.

In addition to the harmful effects of cow's milk and dairy products, red and processed meats have been implicated in type 1 diabetes too. The prospective Type 1 Diabetes Prediction and Prevention (DIPP) Study in Finland was launched in 1994. In DIPP, general population new-born babies are screened for type 1 diabetes genes and those with genetic susceptibility are followed for years to see if they develop the disease. By 2012, of the 150,000 children screened, over 8,500 children were identified as carrying an increased genetic risk and over 300 of them had been diagnosed with type 1 diabetes (DIPP, 2012).

The recent DIPP study investigated meat consumption in the diets of 2,939 mother-child pairs (where the infants had genetic susceptibility) and the children were followed for up to 14 years, during which time 172 of them developed preclinical diabetes (where not all of the symptoms are present, but blood sugar is abnormally high) and 81 developed clinical diabetes. Results showed that **maternal consumption of red meat and meat products while breast-feeding increased the risk for preclinical type 1 diabetes by 19 per cent and clinical type 1 diabetes by 27 per cent**. In particular, **consumption of processed meat increased the risk for type 1 diabetes by 23 per cent** (Niinistö *et al.*, 2015). The vast majority of children with diabetes have type 1, but an increasing number are now developing type 2 diabetes.

Type 2 diabetes

Type 2 diabetes occurs either when the body does not produce enough insulin or when it cannot use the insulin produced. This type of diabetes is linked with obesity; people with a BMI of 30 or more are at greater

risk of developing it. In particular, fat around the abdomen (tummy) increases the risk because it releases substances that can upset the body's cardiovascular and metabolic systems. Type 2 diabetes used to occur most frequently in people over 40, but is now increasingly seen in younger people.

The main symptoms for both types of diabetes include: feeling very thirsty, urinating frequently – particularly at night, feeling very tired and weight loss. Other symptoms of type 2 diabetes include: itchiness around the genitals or regular bouts of thrush (a yeast infection), blurred vision caused by the lens of the eye becoming dry, cramps, constipation and skin infections. Not all symptoms occur and those that do might be subtle and can go unnoticed for years. Blood sugar levels in type 2 diabetes can be controlled by lifestyle changes including regular exercise coupled to dieting and weight loss.

In the UK, about 90 per cent of all adults with diabetes have type 2. While rising obesity levels account for the increase in type 2 diabetes, this does not explain the huge increase in type 1 diabetes cases seen over the last few decades. If current trends continue by 2025 it is estimated that five million people in the UK will have diabetes (Diabetes UK, 2012). Most of those will have type 2 diabetes because of our ageing population and rapidly rising numbers of overweight and obese people. This could mean that the NHS's annual spending on diabetes could rise from £9.8 to £16.9 billion in just 25 years and that in 2035, the NHS could be spending almost a fifth of its *entire budget* on treating diabetes (Hex *et al.*, 2012).

Although obesity and physical inactivity are major determinants of type 2 diabetes and account for much of the increase in prevalence, dietary factors also play an important role in its development (Pan *et al.*, 2011). **Meat consumption is considered a major dietary risk factor for type 2 diabetes**. Research suggests that eating just one serving of meat per week significantly increases the risk of diabetes (Vang *et al.*, 2008). This study investigated the link between meat intake and diabetes in 8,401 adult Seventh Day Adventists (none had diabetes at the start of the study). **Those who followed a 'low-meat' diet over the 17 years of this long-term study had a staggering 74 per cent increase in their risk of developing type 2 diabetes compared to those who followed a meat-free diet for the same period**. Part of this difference was attributable to obesity and/or weight gain but even after allowances were made for this, meat intake remained an important risk factor.

The Fat Story

The children and grandchildren of people with type 2 diabetes have an increased risk of getting the disease, even if they are not overweight or obese. In type 2 diabetes, the body may make some insulin, but not enough, or it may not react to insulin properly. This is called insulin resistance and as a result, glucose builds up in the blood. One study investigated insulin resistance in healthy, young, slim adults whose parents or grandparents had type 2 diabetes (Peterson *et al.*, 2004). What was interesting about this study was that compared to the healthy control subjects, those exhibiting insulin resistance had 80 per cent more fat *inside* their muscle cells. Fat inside the muscle cells (intramyocellular lipid), leads to the production of toxic fatty breakdown products and free radicals that can block the signalling pathways insulin uses to transport glucose into the cell. Ultimately this can lead to a build-up of glucose in the blood and type 2 diabetes. It was suggested that the offspring of people with diabetes may be more likely to have an inherited defect in the way fat is metabolised in their cells. If this is the case, it doesn't matter how much insulin they produce, they may still end up with high levels of glucose in the blood unless they alter their diet.

This explains why people who eat a lot of fat end up having more sugar in the blood than people who eat lots of carbohydrate. A paradox given that it is carbohydrate that is broken down in the body into simple sugars. Dr Michael Greger, physician, author and international speaker on nutrition, food safety and public health, provides an elegant explanation of why this phenomenon occurs. He says blood sugar is like a vampire – it needs an invitation to enter our cells! That invitation is insulin; it is the key that unlocks the door that lets glucose into our muscle cells. If there is no insulin (as in type 1 diabetes) glucose can't enter the cells and blood sugar levels rise. However, it may just be that the insulin we have doesn't work. Insulin can be blocked by intramyocellular lipid disrupting the signalling pathways that allow glucose to enter the cell. This is called insulin resistance and it can lead to type 2 diabetes (Greger, 2015).

In order to understand the extent to which a high-fat diet can influence intracellular fat metabolism ten healthy young men were given a high-fat diet containing 50 per cent of its calories from fat – a diet not too different from that which many people in Western countries eat (Sparks *et al.*, 2005). After just three days, intracellular lipids had increased considerably, showing that accumulation of fat inside

cells is extremely rapid. An earlier study found that fat can cause insulin resistance, inhibiting glucose uptake, within three hours! (Roden *et al.*, 1999).

More recent studies confirm that insulin resistance in muscles and the liver is strongly linked to fat storage in these tissues and that impaired metabolism is a key mechanism responsible for insulin resistance and type 2 diabetes (Morino *et al.*, 2006; Delarue and Magnan, 2007; Daniele *et al.*, 2014). These studies show how high levels of fat in the diet and therefore in the blood can lead to insulin resistance by inhibiting the transport of glucose into muscles.

Under normal conditions, fat is metabolised in the cells' powerhouses (mitochondria) but it appears that people with type 2 diabetes have fewer (and maybe slower) mitochondria in their cells and as a consequence, fat accumulates inside the cells (Barnard, 2007). In people with this condition, **a high-fat diet can result in fat building up in the cell which blocks insulin activity and leads to high blood glucose levels and type 2 diabetes.** However, the good news is that it can be reversed; low-fat diets can improve insulin sensitivity in obese people with type 2 diabetes because as the amount of fat in the blood falls, the activity of the mitochondria in muscle cells (burning up food to produce energy) increases, improving metabolism and thus restoring (at least to some extent) insulin sensitivity and glucose transport into the cells (Daniele *et al.*, 2014).

Human evolution provides some answers as to why this happens. Muscle is considered to be a metabolically flexible (or promiscuous) organ because of its capacity to use both glucose and fats as fuel (Kitessa and Abeywardena, 2016). When they had access to energy-rich food, our ancestors developed ways to store fat in the body to keep them going when food was scarce (Barnard, 2007). For some people, this means storing fat inside the cells as well as in layers around the body. We live in very different times now where for most people in developed countries food is rarely, if ever, scarce, yet our bodies are still programmed to store fat when it is available. The research suggests that it is the extent to which we store fat in our cells that is inherited and that people who store more fat in muscle cells are more likely to experience insulin resistance and therefore more likely to develop type 2 diabetes (Hoeks *et al.*, 2010). So the accumulation of intramuscular fat can cause reduction in mitochondrial function and lead to insulin resistance and type 2 diabetes.

MEAT THE TRUTH

In his extensive *China Study*, Professor T. Colin Campbell notes that as fat intake rises and carbohydrate falls, the incidence of type 2 diabetes increases (Campbell and Campbell, 2005). The typical Western diet is high in fat (especially unhealthy saturated fat), animal products and sugary foods and low in complex carbohydrates. Most saturated fat in the average UK diet comes from: fatty cuts of meat, poultry skin, meat products such as sausages and pies as well as dairy products and sweet foods like cakes and biscuits. Add to that the fact that we don't eat nearly enough fruit, vegetables and wholegrain (carbohydrate-containing) foods; no wonder diabetes is reaching epidemic proportions.

The fat in meat is not the only problem. In 2010, researchers from Harvard School of Public Health reviewed 20 studies, including over a million individuals

and found that the consumption of **processed meat was associated with a 19 per cent higher risk of diabetes** (Micha *et al.*, 2010). They suggested that it is the *processing* of meat that determines how strongly it may affect the risk of diabetes. A later review from Harvard School of Public Health looking at the association between red and processed red meat and diabetes in US adults combined data from three large studies (Pan *et al.*, 2011). They followed 37,083 men in the Health Professionals Follow-Up Study, 79,570 women in the Nurses' Health Study I and 87,504 women in the Nurses' Health Study II. They found that **an increase of one serving a day of both red and processed meats was linked to an increased risk of type 2 diabetes. Red meat increased the risk by 12 per cent, processed meat by 32 per cent and red and processed meats combined, by 14 per cent.** They estimated that substituting of one serving of nuts or wholegrains per day for one serving of red meat was associated with a 16-35 per cent lower risk of type 2 diabetes.

In an interview with Harvard magazine, lead author of the study Frank Hu, Professor of Nutrition and Epidemiology at Harvard School of Public Health, was asked "*Why is red meat harmful?*" Professor Hu says: "*Saturated fat, which can lead to cardiovascular disease, is really just the beginning of the story. Even though it is difficult to pinpoint one compound or ingredient as mechanistically linked to diabetes risk, three components of red meat – sodium, nitrites and iron – are probably involved. Sodium is well known to increase blood pressure, but it also causes insulin resistance; nitrites and nitrates have also been shown to increase insulin resistance and to impair the function of the pancreatic beta cells. Iron, although an essential mineral, can cause beta-cell damage in individuals with hereditary hemochromatosis (a disorder in which the gastrointestinal tract absorbs too much iron) and haem iron – the readily absorbable type found in meat – at high levels can lead to oxidative stress (and cell damage) and systemic, chronic inflammation in some people*". They suggested that substituting one serving of red meat per day for nuts and wholegrains could lower the risk of diabetes by 16-35 per cent.

In 2012, more studies had been done and the Harvard School team revisited the evidence. This time they too found that **both red and processed meat are associated with an increased risk of type 2 diabetes; each 50g daily serving of processed meat was linked to a 51 per cent higher risk and each 100g daily serving of red meat was linked to a 19**



per cent higher risk (Micha *et al.*, 2012). They looked at specific meats and found that each daily serving of bacon (two slices) or hot dogs was associated with a two-fold higher risk of diabetes. Importantly, in these studies, processed meats included some processed

poultry meats (chicken or turkey deli meats and hot dogs). Taken together, this research shows that different types of processed meats, including deli meats that may often be processed white meats, have relatively similar associations with type 2 diabetes.

How meat might cause diabetes

A number of potential mechanisms have been suggested, including the saturated fat and cholesterol in meat, although the similar amount of these in both types of meat does not account for the higher risk associated with processed meat.

Haemochromatosis is a condition where people accumulate high body stores of iron. The fact that up to 65 per cent of people with this condition develop diabetes (Adams *et al.*, 1991) has led to speculation that iron overload may cause diabetes. In 2004, an investigation into iron intake and diabetes revealed that while total iron intake was not related to diabetes, haem-iron intake from red meat was (Jiang *et al.*, 2004). Remember, a vegan diet provides iron in its non-haem form, which is absorbed in a more regulated way than haem-iron.

More recently, a review of 11 studies found that higher haem iron intake and increased body iron stores were both significantly associated with a greater risk of type 2 diabetes (Bao *et al.*, 2012).

Dietary haem iron can increase oxidative stress and insulin resistance (Micha *et al.*, 2012).

However, the average haem iron content is lower in processed meat than red meat (as it tends to contain more fat and less protein), so while haem iron may contribute to the risk of diabetes, it cannot explain the stronger association of processed meats with it.

The major difference between substances found in red and processed meats is in the content of preservatives. On average, processed meats contain about 400 per cent more sodium and 50 per cent more nitrates per gram (Micha *et al.*, 2012). Nitrites and nitrous compounds have also been associated with type 1 diabetes in children (Virtanen *et al.*, 1994; Parslow *et al.*, 1997) and in adults, nitrate concentrations have been used as a biomarker for impaired insulin response (Kleinbongard *et al.*, 2005). Thus, higher nitrates and nitrites in processed meats could further explain their stronger relationship with diabetes.

High-temperature cooking or frying, commonly used in preparing processed meats, can introduce HCAs and PAHs, which could increase risk of both heart disease and diabetes (Micha *et al.*, 2012).

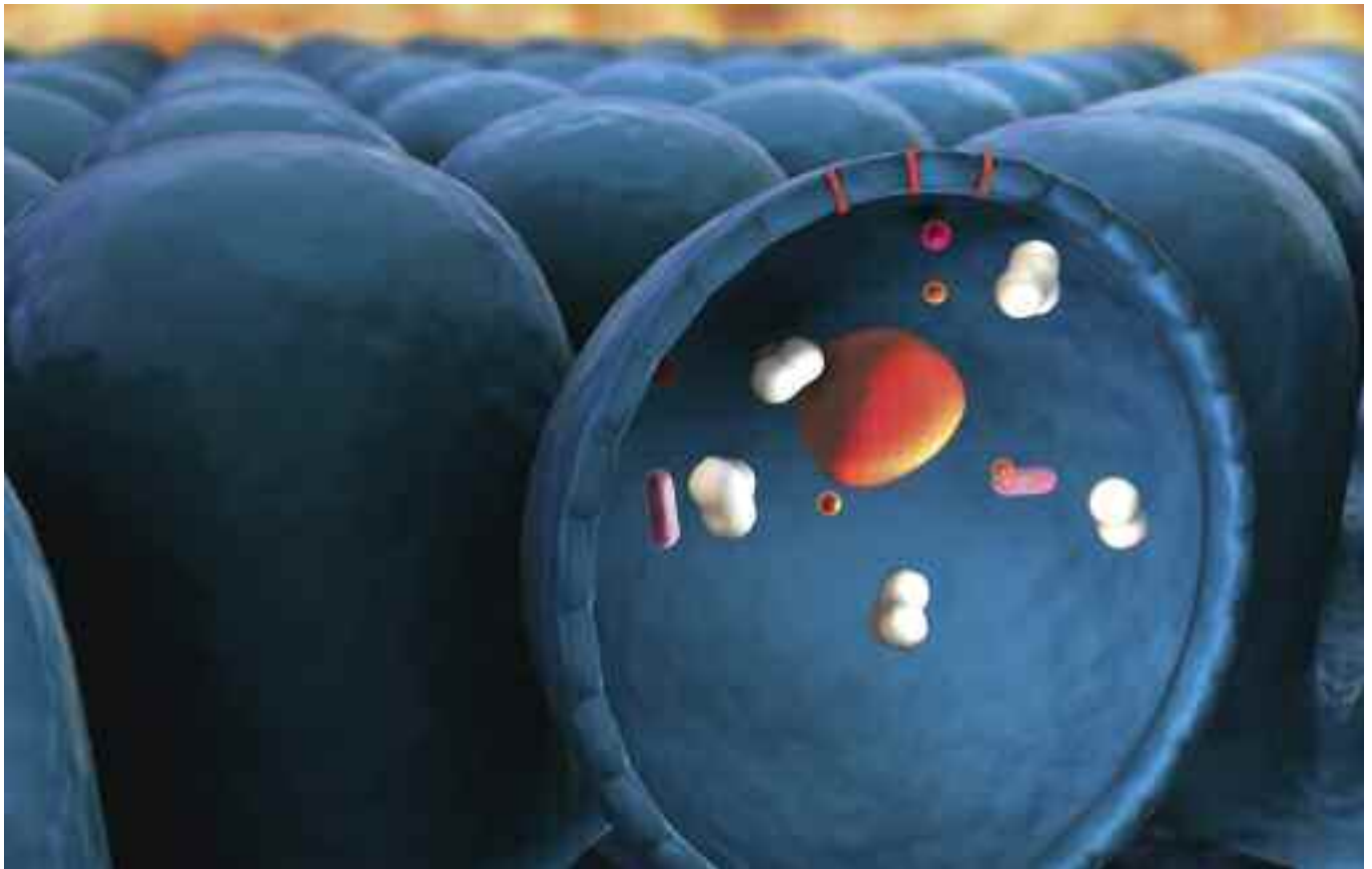
However, there may be another compound in meat contributing to diabetes risk. A study that found positive links between red meat, processed meat and poultry listed the usual suspects: saturated and trans fats, cholesterol, animal protein, haem iron, sodium, nitrites and nitrosamines as well as additional substances called advanced glycation end products (AGEs) also referred to as glycotoxins (Feskens *et al.*, 2013).

AGEs are proteins or lipids that become glycated as a result of exposure to sugars – glycation is the non-enzymatic reaction between glucose and proteins or lipids (Gkogkolou and Böhm, 2012). Put simply, they are formed by the combination of glucose with protein or fat. In addition to AGEs that form within the body, AGEs also exist in foods. Modern diets are largely heat-processed and as a result contain high levels of AGEs (Uribarri *et al.*, 2010).

AGEs are also formed in the body in high amounts during ageing and in diabetes and have been implicated in numerous age- and diabetes-related diseases. It has been suggested that these glycotoxins may be the missing link in the relationship between dietary fat and meat intake and type 2 diabetes (Peppas *et al.*, 2002).

A diet low in AGEs could reduce inflammatory biomarkers in patients with diabetes and renal failure and thus may be an important supportive therapy in diabetes (Gkogkolou and Böhm, 2012). In other words, people with diabetes would be better off avoiding foods that contain these harmful compounds. **Animal foods that are high in fat and protein are generally AGE-rich and prone to additional AGE formation during cooking.** In contrast, carbohydrate-rich foods such as vegetables, fruits and wholegrains contain relatively few AGEs, even after cooking (Uribarri *et al.*, 2010).

MEAT THE TRUTH



In the large EPIC-InterAct study they found a positive association between meat and risk of type 2 diabetes. **Those eating the most meat had a higher risk of developing type 2 diabetes compared with those eating the least** (InterAct Consortium, 2013). According to specific types of meat, they saw a higher risk of type 2 diabetes in: high consumers of red and processed meat, **male high consumers of red meat, processed meat and haem iron and in female high consumers of poultry**. So for women, chicken was the link with a higher risk of diabetes. The usual suspects were discussed; however, this study also described how high levels of diabetes have been reported among workers in the meat industry. It has been proposed that this might be related to exposures to zoonotic infective agents (like viruses) present in fresh cuts of meat, including poultry.

Another possibility is excess food consumption may over-stimulate the mTOR pathway – an intracellular signalling pathway important in regulating the cell cycle and directly related to cellular proliferation, cancer and longevity (Zoncu *et al.*, 2011). The mTOR pathway has evolved to accelerate growth but it also speeds up cancer, metabolic derangement, ageing and may lead to the destruction of insulin-producing beta cells in the pancreas. The multi-protein complex, mTORC1 acts in the mTOR pathway regulating cell growth and proliferation by promoting many anabolic processes.

Dairy proteins and meat stimulate insulin and IGF-1 signalling and provide high amounts of leucine – another stimulator for mTORC1 activation (Melnik, 2012). The simultaneous availability of high levels of glucose, insulin, IGF-1 and leucine in the Western diet results in maximal mTORC1 stimulation, leading to increased pancreatic beta cell proliferation and early cell death, characteristic hallmarks of beta cell disturbance in type 2 diabetes. In other words, meat may cause insulin-producing beta cells in the pancreas to proliferate and die.

Reducing mTORC1 signalling by limiting daily intakes of leucine-rich animal proteins may offer a great chance for the prevention of type 2 diabetes and obesity, as well as other epidemic diseases linked to increased mTORC1 signalling, especially cancer and neurodegenerative diseases which are frequently associated with diabetes (Melnik, 2012). Inhibition of mTOR starting in mid-life could bring significant improvements to human health which may be a critical factor in the diabetes epidemic (Zoncu *et al.*, 2011).

Taken together, these findings suggest that clinical and public health guidance should prioritise reduction of all meat consumption (red, processed and poultry) to reduce not only diabetes but many other diseases too.

SUMMARY

Type 1 diabetes

- Over the last 60 years, type 1 diabetes has been increasing by three to five per cent per year, doubling every 20 years with a rapid rise in the number of children affected. Cow's milk and dairy products are linked to type 1 diabetes and so is meat. The DIPP Study in Finland found that children of breastfeeding mothers who ate red meat and meat products had a 19-27 per cent higher risk. In particular, consumption of processed meat increased the risk.

Type 2 diabetes

- Most people with diabetes have type 2, which is on the rise too and by 2035, the NHS could be spending almost a fifth of its entire budget on just treating this disease. Meat is a major dietary risk factor for type 2 diabetes. Eating just one serving of meat per week significantly increases the risk. A US Adventist study found that those following a low-meat diet over 17 years had a staggering 74 per cent higher risk of type 2 diabetes compared to those following a meat-free diet.
- A Harvard School of Public Health's review of 20 studies found that processed meat was associated

with a 19 per cent higher risk. A later review from the same group found both red and processed meats were linked to an increased risk. They suggested sodium, nitrites and iron could be to blame and said that substituting one serving of red meat a day for nuts and wholegrains could lower the risk of diabetes by 16-35 per cent.

- Other studies agree that both red and processed meat are associated with an increased risk of type 2 diabetes; one found each 50g daily serving of processed meat was linked to a 51 per cent higher risk and each 100g daily serving of red meat, a 19 per cent higher risk.
- The EPIC-InterAct study found a higher risk of type 2 diabetes in: people who ate lots of red and processed meat, men who ate lots of red meat, processed meat and haem iron and women who ate lots of chicken.
- A high-fat diet can result in fat building up inside the cells in the body which can then block insulin activity and lead to high blood glucose levels and type 2 diabetes. However, the good news is that it can be reversed with a low-fat, vegan diet.

For more information see Viva!Health's report *The Big-D: Defeating Diabetes through Diet*:
www.vivahealth.org.uk/diabetes .



FERTILITY

The Mediterranean diet has been related to lower risk of multiple chronic diseases, but it has also been found to have a positive impact on male reproductive potential. A Spanish study looking at 209 male university students found that those consuming a Mediterranean diet with high intakes of vegetables and fruits had a better sperm count than those eating a Western diet characterised by high intakes of processed meats, French fries and snacks (Cutillas-Tolín *et al.*, 2015). The authors concluded that traditional Mediterranean diets may have a positive impact on male reproductive potential.

It was previously shown that meat and dairy foods may be linked to fertility problems. An earlier study found that men who ate the most meat and full-fat dairy products had fewer and slower sperm, while those who ate the most fruit and vegetables had higher quality sperm that swam faster (Mendiola *et al.*, 2009). Lead researcher Professor Jaime Mendiola said *"...among the couples with fertility problems coming to the clinics, men with good semen quality ate more vegetables and fruit, which means more vitamins, folic acid and fibre and fewer proteins and fats, than those with poor sperm quality"*.

Replacing animal protein with plant-based protein can reduce infertility risk in women too according to a study that looked at the diets of 18,555 women who were followed up as they attempted a pregnancy or became pregnant during an eight year period (Chavarro *et al.*, 2008). Results found that the risk of ovulatory infertility was 39 per cent higher in women eating the most animal protein. Consuming just five per cent of total energy intake as vegetable protein rather than animal protein was associated with a more than 50 per cent lower risk of infertility. The authors concluded that replacing animal sources of protein, in particular chicken and red meats, with vegetable sources of protein may reduce the risk of infertility.

Another study investigated the influence of diet on sperm quality and intracytoplasmic sperm injection (ICSI), a form of treatment for men who are infertile used in nearly half of all IVF treatments requiring just one sperm, which is injected directly into the egg (Braga *et al.*, 2012). Two hundred and fifty men undergoing ICSI cycles were followed. Factors positively influencing sperm included fruit and cereal consumption. Factors negatively

affecting sperm included BMI, alcohol consumption, smoking and the consumption of red meat which had a negative impact on the implantation rate.

SUMMARY

- Diet has a huge impact on health and fertility is no exception. Men who eat the most vegetables and fruits (and therefore more vitamins, folic acid and fibre) have a better sperm count than those eating lots of protein and fat found in meat, full-fat dairy foods, French fries and snacks. Infertility in women is also linked to diet with higher rates being found in women who eat the most animal protein. Replacing just five per cent of energy intake of animal protein (meat and dairy) with vegetable protein (pulses or nuts for example) could reduce infertility risk by 50 per cent.
- Given that there is no harm in recommending a healthy diet and lifestyle, couples seeking assisted reproduction treatments should be advised about the drastic effect of both the male and female diet and lifestyle on treatment success.



BUGS, SUPERBUGS, BSE, BIRD FLU AND THE HORSEMEAT SCANDAL - WHAT RISK NOW?

FOOD POISONING

Food poisoning is caused by eating contaminated food. In general, most people get better within a few days without treatment. Occasionally it can lead to serious or long-term conditions or even death. Food poisoning is more likely to result in serious consequences in vulnerable people than healthy adults. This includes the very young, the very old and the immune-compromised. People with some vulnerability may account for nearly 20 per cent of the population in the UK and the US (Lund, 2015).

Symptoms of food poisoning usually begin within one or two days of eating contaminated food, although it may start after just a few hours or even several weeks later. If the poisoning is caused by a bacterial infection, it can take 12-72 hours for toxins from the bacteria to build up. The main symptoms include: feeling sick (nausea), vomiting, diarrhoea, stomach cramps (tummy ache), a lack of energy and weakness, loss of appetite, a high temperature (fever), aching muscles and chills (NHS Choices, 2015e).

In most cases of food poisoning, the food is contaminated by bacteria such as *Salmonella* or *Escherichia coli*, or a virus such as the norovirus. Food can become unsafe by not cooking it thoroughly (especially meat), not storing it correctly if it needs to be chilled (meat again), leaving cooked food for too long at warm temperatures, not sufficiently reheating previously cooked food or being past its 'use by' or 'best before' date. It may become contaminated through poor personal hygiene (being handled by someone who is ill or has dirty hands) or cross-contamination from other food.

Most cases of food poisoning are related to the consumption of animal products (meat, poultry, eggs, fish and dairy) as plants tend not to harbour the types of bacteria capable of causing food poisoning in humans. If plant foods do cause food poisoning it is generally because they have been contaminated with

animal excreta, human sewerage or handled with dirty hands during preparation.

Common food sources of foodborne illness include:

- Raw meat and poultry
- Raw eggs
- Raw shellfish
- Unpasteurised milk
- 'Ready-to-eat' foods, such as cooked sliced meats, pâté, soft cheeses and pre-packed sandwiches



MEAT THE TRUTH

In 2000, the UK FSA published the Report of the Study of Infectious Intestinal Disease in England – IID1 (FSA, 2000). It combined data from national surveillance centres with a comprehensive review of the current scientific literature. The aim was to estimate the extent and causes of infectious intestinal disease (food poisoning) in England. Results suggested that one in five of the population of England (nine and a half million people) suffer food poisoning every year and that two per cent of the population (one and a half million) visit their GP with symptoms.

In 2011, the Second Study of Infectious Intestinal Disease in the Community (IID2) updated IID1 and looked at the UK as a whole. It set out to assess what changes had occurred since the original study, in terms of numbers of people affected and individual pathogens (FSA, 2011a). They estimated that one in four (17 million) people suffer from food poisoning every year in the UK (43 per cent higher than in IID1). However, the number of people visiting their GP was around 50 per cent lower, just two per cent of the population (one million). So it seems more cases were going unrecognised and unreported; they suggested that for every case reported to national surveillance centres, there were 147 unreported cases.

In 2014, the FSA published an extension to the IID2 Study which aimed to identify the specific pathogens and foods that cause most cases of food poisoning (FSA, 2014). Results showed that *Campylobacter* remains the most common foodborne pathogen in the UK with an estimated 280,000 cases and 39,000 GP visits. Other common foodborne pathogens include *Clostridium perfringens* (~ 80,000 cases), norovirus (~ 74,000 cases) and *Salmonella* (~ 33,000 cases). *Salmonella* was ranked first in terms of hospital admissions (~2,500) indicating the severity of the illness caused by this foodborne pathogen.

Poultry was found to be the most common food associated with food poisoning in the UK with an estimated 244,000 cases, 34,000 GP visits and 870 hospital admissions. Around half of all cases and GP visits and a fifth of hospital admissions for foodborne illness are attributable to poultry contamination (FSA, 2014). The FSA put *Campylobacter* at the top of their

priority list as they considered it to be the biggest food safety problem affecting people in the UK. In 2014, they launched a campaign telling people not to wash raw chicken, warning that splashed water droplets can spread *Campylobacter* bacteria on to human skin, work surfaces, clothing and cooking equipment.



Is factory farming making you sick?

The ever-increasing drive to lower the cost of meat inevitably results in more intensive farming methods. The WHO highlighted how safe disposal of manure from large-scale animal and poultry production facilities is a growing food safety problem in much of the world, as manure frequently contains pathogens (WHO/FAO, 2002). For example, in the US, the consolidation of industrial livestock production has led to the fast production of huge amounts of cheap meat. But cheap meat comes at a cost: millions of tons of manure and toxic pollutants which threaten important waterways.

US environmental advocacy organisation Environment America, say that the five major animal agribusinesses (Tyson, JBS, Cargill, Smithfield and Perdue) produce a combined 162,936,695 tons of manure every year! (Environment America, 2016). Environment America said: *"From slaughtering plants run by the company or its subsidiaries, Tyson discharged over 20 million pounds of toxic pollutants to the nation's waters in 2014 – more by volume than even Exxon Mobil or Dupont – according to data the company reported to the federal Toxics Release Inventory. Most of the company's toxic discharges are nitrates, which are linked to blue baby syndrome and some forms of cancer"*.

Agriculture is the probable cause for making more than 145,000 miles of rivers and streams across the US too polluted for swimming, drinking or maintaining healthy wildlife, according to the US Environmental Protection Agency.

The drive for cheap meat has led us into trouble in other ways too. Another problem that recently emerged was how mechanical evisceration (removal of internal organs) of poultry can result in the rupturing of the digestive tracts and the spilling of faecal matter onto

the skin of the animal (Cho *et al.*, 2009). This is obviously a concern with poultry, as people eat the skin. Companies selling this type of equipment boast that their machines can deal with high processing capacities, for example, of up to 13,500 chickens per hour!

Researchers at the University of Minnesota looked for the presence of an antibiotic-resistant strain of *E. coli* in 1,648 different food samples (Johnson *et al.*, 2005). They found contamination in 69 per cent of pork and beef and **92 per cent of poultry samples**. The authors said that the high prevalence of antibiotic-resistant *E. coli* found in meat products is consistent with contamination of animal carcasses with the animal's own faecal flora (gut bacteria) during slaughter and processing and with use of antimicrobial agents in food-animal production. This is probably why 73 per cent of 1,032 samples of fresh shop-bought chicken taken from large retail outlets, small independent stores and butchers in the UK, between February 2014 and February 2015, tested positive for *Campylobacter* (FSA, 2015).

The FSA recently repeated the survey, this time testing 1,009 samples of fresh whole chilled UK-produced chickens taken between January and March 2016 (FSA, 2016). They found that *Campylobacter* was present on 50 per cent of chicken samples. At first glance this might look like good news – down from 76 per cent in the same quarter of the previous year. However, the FSA say that one of the reasons the survey results were lower is because of the decision taken by a number of retailers and their suppliers to remove neck skin from the birds before they are sold. The neck skin is the most contaminated part of the chicken. It is also the part of the bird that the FSA have been testing which they say means that comparisons with previous results are not as reliable as they would like. Therefore, they have stopped this survey and began another in 2016, with a different method of testing *Campylobacter* levels on chicken. Some might call that dodging a bullet!

Table 2.0 shows an estimate of the main pathogen-food combinations, based on studies included in the FSA's extension to the IID2 Study. Foods of animal origin, particularly meat and meat products, are associated with the vast majority of cases of foodborne illness (Lund *et al.*, 2015). While chicken is the clear front-runner responsible for the largest number of food poisoning cases, it is not the main cause of food poisoning death. In 2011, the FSA said that *Listeria monocytogenes* was responsible for the largest number of food poisoning deaths in the UK (causing more deaths than *Salmonella*

Table 2.0 Foods associated with foodborne disease in the UK, Canada, Denmark, the Netherlands, the US and the EU. Source: Lund *et al.*, 2015.

Pathogen	Percentage of food affected
<i>Campylobacter</i>	Poultry 40-90; red meat up to 40 dairy 10-40
Norovirus	Seafood up to 40; produce 20-40
<i>Clostridium perfringens</i>	Beef & lamb 40-50; poultry up to 20; complex foods up to 20
<i>Salmonella</i>	Eggs 10-80; poultry up to 40; pork, beef and lamb each up to 20; produce up to 20
<i>E. coli</i> O157	Beef and lamb 40-70; produce 10-30
<i>Listeria</i>	Unspecified red meat up to 50; dairy up to 40; complex foods up to 40; seafood up to 20, other meats up to 20; produce up to 10

Produce refers to salad vegetables, cooked vegetables, fruit, nuts, seeds (including sprouted seeds) and produce dishes. Complex foods are foods consisting of foods made from more than two ingredients from different categories of foods.

and *E. coli* O157 combined). Although relatively rare, listeriosis (*listeria* infection) can cause severe illness and invariably requires hospitalisation; a third of cases are thought to result in death (FSA, 2011).

Listeria bacteria may be found in a range of chilled, 'ready-to-eat' foods, including pre-packed sandwiches, cooked sliced meats, pâté and soft cheeses (such as Brie or Camembert). These foods should be eaten by their 'use-by' dates. This is particularly important for pregnant women, because listeriosis in pregnancy can cause complications and can result in miscarriage.

E. coli is usually harmless but it can be serious and the strain *E. coli* O157 can cause kidney failure and death. Most cases of *E. coli* food poisoning occur after eating undercooked beef (particularly mince, burgers and meatballs) or drinking unpasteurised milk. The incubation period for food poisoning caused by *E. coli* is typically one to eight days and the symptoms may last for a few days or weeks.

MEAT THE TRUTH

Of course there are many other bugs (bacteria, viruses and parasites) that cause foodborne illness. People of a certain age remember the uproar in the late 1980s caused when the then Health Minister Edwina Currie said that most egg production in this country was affected with *Salmonella*. *Salmonella* bacteria are often found in raw or undercooked meat (especially poultry, pork and beef), as well as dairy products and eggs. Indeed, poultry, pigs and cattle represent an important source of *Salmonella* even though they may show no sign of infection.

In the same way that *Campylobacter* contamination occurs in poultry, contamination of pork and beef can occur from the guts rupturing during evisceration or from the hide during removal and subsequent washing, or transportation of the carcasses. Handling contaminated meat may result in a cross-contamination of hands, tables, kitchenware, towels and other foods (FSA, 2011b). The FSA suggest that contamination of the surface of these meats may be of less concern providing the meat is cooked properly. However, if contaminated meat is minced, *Salmonella* can become incorporated throughout the meat, which if inadequately cooked, may lead to food poisoning.

SUMMARY

- The vast majority of cases of food poisoning are caused by meat, chicken, eggs, fish or dairy. If plant foods are to blame, it is usually because they are contaminated with animal excreta, human sewerage or handled by someone with dirty hands.
- Many cases go unrecognised and unreported and it may be that one in every four people experience food poisoning every year in the UK.
- Chicken is the most common cause, responsible for around half of all cases. *Listeria* is the most fatal, resulting in death in around a third of cases.
- The drive for cheap meat and ever-bigger, industrialised factory farms is a major contributory factor. Mechanical evisceration (removal of internal organs) of poultry has been shown to increase the risk of faecal matter contaminating the meat, which is probably why around 70 per cent of supermarket chickens in the UK are contaminated with *Campylobacter*. Beef and pork are affected in this way too.
- Going vegan is no guarantee that you will avoid food poisoning, but it certainly does lower the risk substantially.



SUPERBUGS

Asking for a rare steak in a restaurant could be like playing a game of Russian roulette! The routine use of antibiotics in farmed animals has led to the rapid increase in antibiotic-resistant strains of bacteria. A 2015 government review found that these 'superbugs' can be passed on to humans through undercooked meat, (O'Neill, 2015). After reviewing the current literature, the government report argues that the case for reducing antibiotic use in agriculture is compelling and that there is a need to take action now. Of the 139 studies the review looked at, only seven (five per cent) argued that there was no link between antibiotic consumption in animals and resistance in humans, while 100 (72 per cent) found evidence of a link. They said we need to take urgent steps to make sure that the use of antibiotics in animals that are important for human use, is restricted and, where necessary, banned. Calls for reduced use of antimicrobials in food production have been made before, but not, perhaps, in such blunt and specific terms.

Bacterial resistance to antibiotics spells trouble for humans and animals. Drugs that were effective for treating community- and hospital-acquired infections are no longer so because the target bacteria are becoming resistant to their action (Lipsitch *et al.*, 2002). Some fear we may be approaching a 'post-antibiotic era' in which common infections are untreatable.

Over half of the antibiotics that are produced in the US are used for agricultural purposes (Lipsitch *et al.*, 2002). Contamination of meat can then transfer these 'superbugs' from the animal's digestive tract to humans. Plant foods (and so vegetarians and vegans) can also be affected via bacteria in manure contaminating crops, fruit and vegetables. Antibiotic resistant strains of bacteria are increasing at an alarming rate; of particular concern are methicillin-resistant *Staphylococcus aureus* (MRSA) and third-generation cephalosporin-resistant *E. coli* (G3CREC). The 'superbug' G3CREC originates directly from the overuse of cephalosporin, an antibiotic used in broiler chicken farms. It was estimated that in the UK in 2007, there were 1,580 cases and 282 deaths associated with poultry-derived G3CREC (Collignon *et al.*, 2013).

Not long after antibiotics were first used widely in humans it was discovered that they could promote more rapid growth when given to farm animals at low levels (O'Neill, 2015). However, the use of antibiotics at low or 'sub-therapeutic' levels encourages the development of resistant bacteria. Despite a 2006 EU-wide ban on growth-promoting antibiotics added to animal feed, huge quantities of antibiotics continue to be given for 'disease prevention'.

Livestock-associated methicillin-resistant *Staphylococcus aureus* (LA-MRSA CC398) is an important cause of animal-to-human infections in many countries. One



MEAT THE TRUTH

study isolated LA-MRSA CC398 from retail meat samples from UK farms (Hadjirin *et al.*, 2015). This study indicates that these superbugs were probably established in UK pig farms and demonstrates a potential pathway for the transmission of LA-MRSA CC398 from livestock to humans in the UK.

Another example of how antibiotic use in animals is creating major human health risks is the emergence of bacteria carrying resistance to the antibiotic colistin (polymyxin E), our last defence against multi-resistant bacteria (O'Neill, 2015). The routine screening of foods from areas in China where colistin is routinely given to pigs revealed a high number of resistant bacteria (Liu *et al.*, 2016a). They found colistin-resistant *E. coli* in more than 20 per cent of animals, 15 per cent of raw meat samples and one per cent of hospital patients.

The alarming thing about this was that the bacteria had resistance that could be transferred between different bacteria, something that had not been reported before. The resistance genes were carried on plasmids – ancillary or extra pieces of DNA carried in the cell in addition to chromosomal DNA. Chromosomes contain all the essential information for living, whereas plasmids are much smaller and contain additional information that may offer some benefits to the bacteria. Rather like if the chromosome was a large briefcase containing all your essential information, the plasmid might be likened to a small purse with some additional notes in it. The important point here is that plasmid DNA can be passed between different strains of bacteria – this is called horizontal gene transfer and it opens up the possibility of antibiotic-resistance spreading even faster. The authors said that **the emergence of plasmid-mediated resistance to colistin heralds the breach of the last group of antibiotics.**

Antibiotic resistance is a problem of our own making, a direct consequence of the inappropriate use of antibiotics. Restrictions of antibiotic use in animals cannot always wait for incontrovertible evidence of harm and a delay may result in a lost opportunity to preserve the usefulness of certain antibiotics in human medicine (Smith *et al.*, 2002).

For more information see Viva!'s film *Swine*: www.viva.org.uk/swine

SUMMARY

- The routine use of antibiotics in farmed animals has led to the rapid increase in antibiotic-resistant strains of bacteria. Calls for reducing their use have been largely ignored, probably because they promote more rapid growth when given to farm animals at low levels. So antibiotics continue to be used routinely for 'disease prevention'. As a consequence, antibiotic resistant strains of bacteria are emerging at an alarming rate; third-generation cephalosporin-resistant *E. coli* are of particular concern, originating from the overuse of antibiotics in broiler chickens. LA-MRSA CC398 is a superbug thought to have emerged from UK pig farms.
- Now we have colistin-resistant *E. coli*, these superbugs are resistant to colistin, a 'last-resort' antibiotic used against multi-resistant bacteria.
- This is a problem of our own making and we simply can't afford to drag our feet anymore.
- So if you undercook meat, you could expose yourself to bacteria which may or may not be antibiotic-resistant, but if you overcook it, you could be at risk from carcinogenic compounds formed in the cooking process. The dilemma is what some people might call a no-brainer!

VIRUSES

Norovirus, sometimes called the 'winter vomiting bug', is a common cause of gastroenteritis (stomach flu) affecting 600,000-1 million in the UK every year. It is a highly contagious virus that can infect anyone. You can get it from an infected person, contaminated food or water, or by touching contaminated surfaces. Globally, the virus affects around 267 million people and causes over 200,000 deaths each year – usually in less developed countries in the very young, elderly and immunosuppressed (Debbink *et al.*, 2012). Outbreaks often occur in closed communities such as hospitals, schools and cruise ships, where infection spreads rapidly by person-to-person transmission or through contaminated food. Norovirus is shed in huge quantities in the faeces and vomit of infected people and as few as ten viral particles can cause infection (Matthews *et al.*, 2012).





Norovirus outbreaks can also occur from bivalve shellfish such as oysters, infected by sewerage-contaminated water. Bivalve shellfish are commonly involved in outbreaks of foodborne viral diseases and can carry norovirus as well as other viruses (hepatitis A and E). Oysters and mussels are filter feeders and if the waters they inhabit are polluted with human sewage, they can accumulate viruses. This gives another meaning to the term 'bottom-feeder'! Other sources include foods contaminated during irrigation or washing and foods contaminated by an infected handler. Most people who are infected can look after themselves at home; antibiotics don't help because the infection is caused by a virus. Some evidence suggests the presence of noroviruses in pigs and cattle, but there is no evidence yet for direct transmission to humans.

Other foodborne viruses (such as hepatitis E) have been found in meat from pigs, wild boar and deer (EFSA, 2011). Although foodborne transmission of hepatitis E may be relatively rare, the virus can be transmitted through consumption of undercooked meat. Several

studies suggest the following food items as risk factors for infection: pork pies, liver pâté, wild boar, undercooked or raw pork and sausages (EFSA, 2011). The European Food Safety Authority say that high risk groups (people with underlying liver disease, immunocompromised people and pregnant women) should be discouraged from eating meat and liver derived from wild boars and domestic pigs without proper cooking for prevention of hepatitis E (EFSA, 2011).

SUMMARY

Norovirus is a highly infective human virus that can be passed from person-to-person or by contaminated food. Food vehicles for it include foods contaminated by an infected handler, contaminated during irrigation or washing and sewage-contaminated bivalve shellfish. These filter feeders can accumulate viruses if the waters they inhabit are polluted with human sewage. They can also be a source of hepatitis A and E. Hepatitis E has also been found in meat from pigs, wild boar and deer.

MEAT THE TRUTH

BSE

Many people can remember the agricultural minister in 1990, John Gummer, cheerfully feeding his four-year-old daughter a beef burger in front of the world's press. It was the height of the outbreak of bovine spongiform encephalopathy (BSE), commonly known as mad cow disease. The fatal neurodegenerative disease causes a spongy degeneration of the brain and spinal cord. During the 1986-1998 outbreak in the UK, more than 180,000 cattle were infected and 4.4 million were slaughtered. In 1990, fears were growing that BSE could infect humans, but Gummer hoped to convince the nation that British beef was perfectly safe.

In 1996, the worst fears were realised when the first case of the human form of the disease, Creutzfeldt-Jakob disease (CJD), was confirmed in the UK. It led to a storm of news reports, changes in government policies regarding the beef industry, a ban on exports of meat, restrictions on blood donations and a widespread fear that anyone could be infected (Diack *et al.*, 2014).

An inquiry into BSE concluded that it was caused by cattle being fed the remains of other cattle in the form of meat and bone meal (Defra, 2000). Remember, cows are natural herbivores so it is very unnatural for them to eat the remains of other cattle. The meat and bone meal had been produced by the rendering (industrial cooking) of carcasses of cattle infected with BSE. The feeding of meat and bone meal to all farmed animals was banned in 1996 and an EU-wide ban has been in place since 2001.

By 2016, CJD had killed 178 people in the UK (National Creutzfeldt-Jakob Disease Surveillance Unit, 2016). The most recent case was in 2016 when a 37-year-old man was diagnosed with the condition only two weeks before he died. It is feared that people infected with CJD may carry the disease for up to 50 years before symptoms develop. A team of researchers at the University College London studied Papua New Guineans with a related disease called kuru (a prion disease caused by cannibalism) in order to work out how long BSE may lurk



in the body before it develops into CJD. They found that some Papua New Guineans, who once feasted on their own relatives, succumbed to prion disease as much as half a century later (Collinge *et al.*, 2006).

The discovery renews concerns that CJD, could also be incubating silently and could rear its head decades from now. Professor John Collinge, who led the study, said *"Recent estimates of the size of the CJD epidemic based on uniform genetic susceptibility could be substantial underestimations"*. An editorial in the *Lancet* accompanying this study stated: *"Any belief that CJD incidence has peaked and that we are now through the worst of this sinister disease must now be treated with extreme scepticism"*.

SUMMARY

It wasn't until humans began dying from CJD in the UK in 1996 that the unhealthy practise of feeding cows the remains of other dead cows was acknowledged as the probable cause of this fatal neurodegenerative disease. Animal feeding practices have now been changed but CJD has a long incubation period and cases are still emerging.

BIRD FLU

Bird flu – as avian influenza has become known – came to international attention in 1997 when it spread through live-poultry markets in Hong Kong and for the first time infected people, resulting in six deaths. The Hong Kong outbreak was controlled by slaughtering the entire poultry population.

Avian influenza viruses occur naturally among wild aquatic birds and have done for millions of years – without making them ill. The virus infects the intestines of ducks, for example, and is then passed on in water from one bird to another, causing no problems. However since the 1950s, avian influenza viruses have caused illness and deaths in large numbers of land-birds, especially poultry (FAO, 2011). In the UK, a highly pathogenic strain of avian influenza virus called H5N1 caused serious disease among chickens in Scotland in 1959 and turkeys in England in 1991. The last reported case of H5N1 in the UK was in early 2008 (NHS Choices, 2015f).

Various different strains of H5N1 have been identified – one particular strain is of huge international concern because it can infect people and has led to the deaths of several hundred. In June 2016, WHO confirmed that since 2003, there had been 851 cases of human infection with H5N1 and 450 deaths reported from 16 countries. Fatalities have been seen in Egypt, Indonesia and Vietnam as well as Cambodia, Thailand and China (WHO, 2016).

While there are low-pathogenic varieties of H5N1, high-pathogenic strains cause death in more than half (60 per cent) of the people they infect. So although it may be relatively hard for humans to become infected, when they are, the odds are not good. Case-fatality rates for past influenza pandemics have ranged from about 0.1-2.5 per cent (Li *et al.*, 2008). Also, avian flu viruses aren't usually transmitted from one human to another, but there have been a number of cases of human-to-human infection among families caring for infected relatives (Qi *et al.*, 2013; Wang *et al.*, 2008; Ungchusak *et al.*, 2005). Imagine the death rate if a highly pathogenic avian influenza virus became able to spread like the common flu!



MEAT THE TRUTH

In 2013, another pathogenic strain (H7N9) appeared, this time in China. Most of the cases of human infection with H7N9 followed recent exposure to live poultry or markets where live birds were sold. By May 2015, there had been 665 confirmed cases and 229 deaths. Most among middle-aged and elderly men but a number of cases were reported in travellers from Hong Kong, Taiwan, Malaysia and Canada.

Bird flu has gone from being a relatively rare occurrence to one that crops up frequently, every year and the UK has not escaped. In November 2014, a low-severity H5N8 virus was confirmed at a farm in Yorkshire, then in February 2015, a low-severity H7N7 virus was found at a farm in Hampshire. It seems that it just won't go away and now H5N1 has infected migratory birds who are spreading it around the world. In 2015, a highly pathogenic H5N1 strain was identified at a chicken farm in the Dordogne in France (Defra, 2015b). It was subsequently detected in a number of other farms in southwestern France.

But what caused bird flu to change into this much more sinister menace? The short answer is intensive factory-farming. Viruses are unlike other life-forms in that they are very simple packages of DNA that hijack the cellular machinery of plants and animals in order to replicate and multiply. They don't breathe, drink, eat or excrete... they just replicate, almost like little machines. Their sole purpose is to infect animals or plants and make more copies of themselves. If a mutation occurs that makes this easier, the mutated version will thrive. Factory farms provide a perfect environment for the emergence of new 'super-viruses'.

In aquatic birds, the virus had found an ideal environment in which it could co-exist without harming the host. However, when these birds were taken to market, the virus could no longer spread from bird to bird in water. This meant there was a new pressure on the virus to mutate or die.

Mutations naturally occur but unless they offer some advantage, the original version of the virus will continue to dominate. In this new environment, mutations occurred and the virus was able to spread – via faeces, nasal secretions, or secretions from the mouth or eyes of infected birds.

Of course, intensive poultry production provides the perfect breeding ground for a mutating virus. Chickens and other commercial birds are raised in closed, crowded, stressful and unsanitary industrial facilities with little or no natural light, offering the bird flu virus a perfect opportunity for infection, mutation and spreading. The genetic profile of birds found in factory farms is often less diverse than those raised in backyards. Due to the industry's reliance on breeding methods, commercially raised broilers are genetically very similar. Broilers and turkeys are bred to produce birds that grow quickly (300 per cent faster than birds raised in the 1960s) to produce as much breast meat as possible, to the point where some birds struggle to stand. This inevitably adds to the stress and lowers their immune function increasing the opportunity for viral infection. A perfect storm of our own making!

An aberrant host is a 'dead-end' host from which viral replication does not normally occur, humans for example. A spillover host is a novel (new) population susceptible to infection that may go on to transmit the virus, poultry for example. Humans are currently regarded as aberrant hosts as, at the moment, we do

not spread infection. However, we are at risk of becoming spillover hosts due to viral evolution, which could result in a global influenza pandemic.

Influenza viruses typically evolve much more rapidly in spillover hosts such as chickens and turkeys than they do in reservoir hosts such as wild water birds (Suarez *et al.*, 2000).

Avian influenza viruses have infected many different animals, including ducks, chickens, pigs, whales, horses, seals and dogs (CDC, 2015a). In 2006, authorities in Germany announced detection of H5N1 in a



domestic cat that was found dead on the northern island of Ruegen where more than 100 wild birds have died from H5N1 infection. In 2003-2004 a number of captive tigers and leopards in a zoo in Thailand who were fed fresh chicken carcasses died of H5N1 infection. Subsequent investigation determined that at least some tiger-to-tiger transmission of the virus had occurred (WHO, 2006).

There is widespread agreement that the pattern of avian influenza infection which occurred during the 2003-2005 Asian epidemic represents a disturbing new evolutionary development in the behaviour of the virus, the full ramifications of which may not yet have unfolded. In his comprehensive book which provides a full and insightful account on the subject of bird flu, Dr Michael Greger warns that:

“There are three essential conditions necessary to produce a pandemic. First, a new virus must arise from an animal reservoir, such that humans have no natural immunity to it. Second, the virus must evolve to be capable of killing human beings efficiently. Third, the virus must succeed in jumping efficiently from one human to the next. For the virus, it’s one small step to man, but one giant leap to mankind. So far, conditions one and two have been met in spades. Three strikes and we’re out. If the virus triggers a human pandemic, it will not be peasant farmers in Vietnam dying after handling dead birds or raw poultry – it will be New Yorkers, Parisians, Londoners, and people in every city, township, and village in the world dying after shaking someone’s hand, touching a doorknob, or simply inhaling in the wrong place at the wrong time.”
(Greger, 2006).

The poultry industry has responded to the bird flu crisis by playing down the risk to humans. The Food and Agriculture Organisation of the UN recommend an integrated programme of vaccination and enhanced biosecurity including separating reservoir and spillover species, introducing restrictions on marketing and movement of birds and separating high risk and lower risk species during marketing (FAO, 2005). However, WHO spokesman for the Western Pacific region, Peter Cordingley said: *“It might be time, although this is none of WHO’s business really, but the bottom line is that humans have to think about how they treat their animals and how they farm them, how they market them – basically the whole relationship between the animal kingdom and the human kingdom is coming under stress.”* (CNN, 2004).

SUMMARY

- Until relatively recently, the bird flu virus co-existed in aquatic birds spreading from bird to bird in water without harming them. When people began taking birds to market, the virus was unable to spread and a new, more virulent virus emerged, one that could spread more easily in the faeces and secretions from the birds. Large-scale factory farms now provide the perfect environment for a mutating virus – closed, dimly lit (UV can harm viruses), crowded, stressful and unsanitary conditions – a perfect storm of our own making!
- The virus has become able to jump species and has infected a range of animals including humans, in whom fatality rates can be very high. While it is still relatively difficult to catch, there have been reports of human-to-human infection and if it mutates further, becoming as easy to catch as a common cold virus, we will be in deep trouble. One way to take control of the situation would be for huge numbers of people to stop eating poultry, pigs and other animals and remove the viral reservoir of factory-farms.

HORSEMEAT AND BEYOND

The 2013 horsemeat scandal arose when people discovered that foods they had eaten, that they thought contained beef, actually contained horsemeat (and no beef at all in some cases). An investigation by the Food Safety Authority of Ireland found horse DNA in frozen ‘beef-burgers’ that were sold in several Irish and British supermarkets. The DNA testing also revealed widespread mislabelling whereby beef-burgers contained pig DNA (Food Safety Authority of Ireland, 2013).

The meat industry would argue that the presence of undeclared meat is not a health issue. However, there is increasing evidence that meat can cause allergic reactions, indeed, a prevalence of beef, pork and chicken allergies has been reported (Tanabe *et al.*, 2007). Furthermore, it’s not unreasonable to want to know what is in your food!

The scandal revealed a major breakdown in the traceability of the food supply chain and showed the potential for harmful ingredients to be included as well. Where did these horses come from? Sports horses could have entered the food supply chain, and with them the veterinary drug phenylbutazone which is banned in farmed animals.

MEAT THE TRUTH

The scandal has since spread to other European countries. In 2015, a study looking at processed meat products from Italian markets and supermarkets found that 57 per cent of products were mislabelled (Di Pinto *et al.*, 2014). The study revealed a high probability of incorrect species declaration in meat products and insufficient labelling information for sausages, pâté and meat patties. It confirmed that fraudulent descriptions, with various undeclared animal species in ready-to-cook meat products, and adulteration of meat products with an undeclared mixture of meats, are widespread.

In 2012, Spanish police found a warehouse in the Galician town of As Neves filled with 15 tons of dead stray dogs which they believed were going to be processed into animal feed. It is feared that stray dogs from Spain may have been used to make pet food, farm animal feed and may have even been used in foods for human consumption.

Viva! found out in 2013 that the UK FSA authorised testing for dog and cat meat in 11 samples from a range of take-away premises in Enfield in London. The samples taken were raw meat and labelled as being beef or lamb. Results were negative but we asked them why they weren't testing a wider area given that they were aware of the scandal in Spain. The FSA said that they didn't think it was necessary. Spanish authorities say they cannot rule out that dog meat has not already entered the human food chain and the contamination could be widespread across Spain and other EU countries.

This raises concerns for British supermarkets and processors who had no idea that horsemeat was in their products. The tests that showed widespread contamination with horsemeat would not have revealed the presence of dog or cat meat. There are fears that meat from euthanised dogs, cats, horses and other sick or unwanted stray animals may have found its way into pet food, farmed animal feed or food for human consumption. The concern here is that residues of antibiotics and other drugs, used to treat those animals, may end up in some meat products. It's a shocking prospect for most that dog and cat meat might have entered the human food chain, but given the depth of deceit the horsemeat scandal exposed, it seems entirely plausible. It may only be a matter of time before dog, cat and perhaps even rat meat is found in a British meat pie. The obvious way to avoid being caught out is to not eat meat.

SUMMARY

The 2013 horsemeat scandal revealed widespread mislabelling of food including 'beef' that was actually horsemeat, as well as other mislabelled foods such as beef-burgers that tested positive for pig DNA. There are health concerns that horsemeat may come from racehorses treated with veterinary drugs. Then there was the warehouse in Spain containing 15 tons of dog meat, it's not known for certain where they were destined. Going meat-free is the only way to make sure you avoid eating horse, dog, cat or rat.



EVOLUTION AND THE PALEO DIET MYTH

WHEAT-EATERS OR MEAT-EATERS?

Carnivores (such as cats, dogs and wolves) have strong jaws that can only move open and shut and sharp teeth and claws to tear off chunks of raw meat and 'wolf' them down. Their acidic stomachs help digest flesh and short intestines allow the quick expulsion of rotting meat remains. On the other hand, herbivores (such as rabbits, horses and sheep) chew from side-to-side, their saliva contains digestive enzymes and they have longer intestines to absorb nutrients.

When asked if humans are herbivores, carnivores, or omnivores, Dr William C. Roberts, Editor-in-Chief, of *The American Journal of Cardiology* said: "Although most of us conduct our lives as omnivores, in that we eat flesh as well as vegetables and fruits, human beings have characteristics of herbivores, not carnivores" (Roberts, 2000). Researchers from Harvard University say that although increased consumption of meat during human evolution certainly contributed to dietary quality, meat-eating alone was insufficient to support the evolution of human traits, because modern humans fare poorly on diets that include raw meat (Carmody and Wrangham RW, 2009). They suggest it was the *cooking* of food that substantially improved the quality of the diet. Indeed, meat-eating may have necessitated cooking because raw meat is difficult and takes time to chew, thus limiting consumption in large quantities (Luca *et al.*, 2010).

THE PALEO DIET MYTH

In the 1980s US anthropologists Boyd Eaton and Melvin Konner suggested the Paleo diet as a model for modern human nutrition (Eaton and Konner M, 1985). The Paleo (Palaeolithic or hunter-gatherer) diet contains high protein (meat and fish but no dairy) and fibre but no grains or pulses. Proponents say the mismatch between this and modern diets is to blame for high levels of obesity, diabetes and heart disease. However, we have evolved to be flexible eaters (Henry *et al.*, 2014) and genetic evidence shows that we continued to evolve over the last 40,000 years,

well into the Neolithic era (Hawks *et al.*, 2007). Modern adaptations include increased production of amylase, an enzyme that helps us digest carbohydrates or starch (Turner and Thompson, 2013). Furthermore, geochemical analysis of grains and pulses from Neolithic sites reveal that early farmers relied much more heavily on plant protein than previously thought (Bogaard *et al.*, 2013).

MEAT MADE US SMART IS A DUMB IDEA

In the 1990's British scientists Leslie C. Aiello and Peter Wheeler proposed the 'expensive-tissue hypothesis' whereby there is a trade-off between the size of the digestive tract and the brain (Aiello and Wheeler P. 1995). The brain is 'expensive' because it requires so much energy and a high-quality diet enabled us to reduce the size of our digestive tract and free up energy to increase brain size. In other words 'meat made us smart'. However, recent research, published in the journal *Nature*, refutes this saying a higher quality diet, coupled to energy saved by walking upright, growing more slowly and reproducing later, fuelled the growth in brain size (Navarrete *et al.*, 2011). Prehistoric humans ate some meat but it didn't make them smart.

SUMMARY

Humans have more characteristics in common with herbivores than carnivores. We are not suited to eating raw meat and cooked meat, even at moderate levels, is associated with a wide range of health problems. The idea that we are suited to a hunter-gatherer diet, rich in meat and fish with no grains or pulses, is flawed. Humans continued evolving past the Palaeolithic era and our Neolithic ancestors adapted to be able to digest carbohydrates for example and we now know that they relied on plant protein more than previously thought. The research simply doesn't support the notion that humans were designed to eat meat, especially in the quantities consumed in some affluent countries.

ENVIRONMENT

Livestock farming requires vast amounts of land, water and fuel, harms biodiversity and leads to species extinctions. It devastates ecosystems, pollutes oceans, rivers, seas and air, uses up water, oil and coal and contributes to climate change. It causes about one-fifth of global greenhouse gas emissions.

It takes far more resources to feed a meat-eater than a vegetarian or vegan; animal protein requires 5-10 times more water than vegetable protein. Leading scientists have issued stern warnings about global food supplies, saying that the world may have to switch almost completely to a vegetarian diet to avoid catastrophic shortages.

The Stern Review on the *Economics of Climate Change* is a report written for the British government in 2006 by economist Nicholas Stern, chair of the Grantham Research Institute on Climate Change and the Environment at the London School of Economics (LSE) and also chair of the Centre for Climate Change Economics and Policy at Leeds University and LSE (Stern, 2006). The report discussed the effect of global warming on the world economy. It is significant as the largest and most widely known and discussed report of its kind. The Stern Report concluded that although dealing with global warming by cutting emissions of greenhouse gases will cost a lot of money (about one per cent of the world's GDP), doing nothing about it will cost the world an awful lot more, anything from five-20 times more. The report warned that we face losing up to a fifth of the world's wealth from unmitigated climate change suggesting that if unchecked, it will devastate the global economy on the scale of the Great Depression or the 20th century's world wars.

The United Nations' report, *Livestock's Long Shadow* says livestock farming is responsible for more greenhouse gas emissions than all the world's transport (cars, buses, trucks, trains, ships and planes) combined (FAO, 2006). Changing the way we eat could have a phenomenal effect on the environment.

And what about soya? The vast majority of soya grown in the Amazon is used for animal feed so people can eat meat and dairy. Vegetarians and vegans eat a tiny fraction of that and if you want to be sure to avoid soya from the rainforests, buy organic.

The links between health and the environment are also beginning to emerge; the diet that is good for us is also good for the planet. A 2012 study modelling consumption patterns in the UK estimates that a 50 per cent reduction in meat and dairy consumption, if replaced by fruit, vegetable and cereals, could result in a 19 per cent reduction in greenhouse gas emissions and up to nearly 43,600 fewer deaths per year in the UK (Scarborough *et al.*, 2012). Pulses were included in the modelling but were not explicitly discussed as they were included in the 'vegetable' category. In other words they were not thought to have an influence on health that is different to vegetables.

As discussed previously, researchers from the Oxford Martin Programme on the Future of Food said that a global switch to diets that rely less on meat and more on fruit and vegetables could save up to eight million lives by 2050, lead to healthcare-related savings and reduce greenhouse gas emissions by two thirds (Springmann *et al.*, 2016). The report said that adhering to health guidelines on meat consumption could cut



global food-related emissions by nearly a third by 2050, but the widespread adoption of a vegetarian diet would bring emissions down by 63 per cent and a vegan diet would reduce them by 70 per cent. Lead author of the report, Dr Marco Springmann, said: *"The size of the projected benefits should encourage individuals, industry and policymakers to act decisively to make sure that what we eat preserves our environment and health"*.

Demands on food production are ever-increasing and meeting them globally is a substantial challenge. Feeding animals food that humans could eat is clearly a waste of precious resources. One study revealed that 36 per cent of calories produced by the world's crops are currently being used for animal feed with only 12 per cent of these calories eventually finding their way into the human diet as meat and other animal products (Cassidy *et al.*, 2013). Growing food solely for human consumption, without feeding it through farmed animals, could increase available calories by as much as 70 per cent, which could feed an additional four billion people! There really is no longer any excuse for wasteful Western diets now.

The EPIC team investigated to what extent an environmentally friendlier diet is also a healthier diet in the EPIC-NL cohort study (Biesbroek *et al.*, 2014). They found that substituting meat with other major food groups was associated with a lower mortality risk and a reduced environmental burden. Especially when vegetables, fruit, nuts, seeds, pasta, rice or couscous were used to replace meat.

A study from German consumer protection organisation Foodwatch, says that giving up meat could drastically reduce your carbon footprint. Meat-eaters' diets are responsible for almost twice the emissions as vegetarians' and **going vegan could cut your emissions more than seven-fold** (Foodwatch, 2008).

Other research shows that you can improve your health and do your part for the environment by dropping meat from the menu. Researchers from California looked at the diets of 34,000 people of which around half were vegetarians. They found that meaty diets required 2.9 times more water, 2.5 times more energy, 13 times more fertiliser and 1.4 times more pesticides than the vegetarian diets (Marlow *et al.*, 2009). Lead author, Dr Hal Marlow, said *"Almost everyone has some knowledge that it costs less environmentally or is healthier to be a vegetarian, but there's no understanding yet of really what that means until you put some numbers behind it"*.

SUMMARY

Livestock farming uses far more resources than agriculture. *The Stern Report* warned of a global catastrophe if nothing is done to stop global warming. The UN's report *Livestock's Long Shadow* described how livestock farming contributes more to global warming than all the world's transport put together! Numerous modelling studies show how greenhouse gas emissions could be substantially reduced if people cut their meat intake. Going vegan could cut your emissions seven-fold. The health benefits are an added bonus! If you care about the environment then it is essential that you adopt a green diet – a vegan diet.

For more information on how what you eat affects the environment see: www.viva.org.uk/what-we-do/our-work/environment





NUTRITION – WHAT’S IN MEAT AND WHERE ELSE CAN I GET IT?

Meat is the best source of protein isn't it? And what about iron and vitamin B12? All nonsense, meat doesn't contain anything of nutritional benefit that you can't find in healthier foods. What it does have though is hormones, antibiotics and a whole host of other unsavoury ingredients that will be discussed later.

All muscle tissue contains protein, including all of the essential amino acids. Most types of meat contain iron, zinc, selenium, phosphorus, vitamins B2, B3, B6, B12 and choline. Some meat also provides vitamin K. Muscle and organ tissue contains little or no carbohydrate and does not contain any dietary fibre. Most meats contain extremely low levels of calcium, negligible amounts of beta carotene and vitamin E, and no vitamin C.

Meat does contain cholesterol and is a rich source of unhealthy saturated fat, linked to obesity and cardiovascular disease (CVD). Other undesirable compounds in processed meat include potentially carcinogenic substances: N-nitroso compounds – nitrosamines or nitrosamides (NOCs), polycyclic

aromatic hydrocarbons (PAHs) and heterocyclic amines (HCAs). The iron in red and processed meat (haem iron) has been implicated in the risk of cancer. Non-haem iron from plant foods offers all the benefits of iron without this risk.

FAT

All fat has nine calories per gram, twice as many calories as carbohydrates and protein. However, not all fats are 'bad'. We need a moderate amount of unsaturated so-called 'good' fats in the diet. These types of fat are essential for cell membranes, eyes, the brain and metabolic functions. These healthy fats are plentiful in plant foods such as nuts, seeds and their oils, avocados and soya foods. Green leafy vegetables contain them too, but not much as they are a very low-fat food.

We have no dietary requirement for saturated fat. Found widely in meat, dairy, eggs, processed foods and fish, this unhealthy type of fat contributes to the risk of CVD by raising blood cholesterol levels. When you eat

saturated fat it is converted into cholesterol by the liver. Saturated fat also slows down how quickly cholesterol is removed from the blood. Cutting down on saturated fat in the diet and replacing it with unsaturated fats is an effective way of reducing cholesterol and therefore lowering the risk of CVD.

Trans fats are an unhealthy type of fat naturally found in low levels in meat and commonly produced industrially from vegetable fats used in processed foods, fried 'fast foods' and margarine (although not so much now). Trans fats have been shown to increase blood cholesterol levels and thus the risk of CVD.

The Department of Health recommends that saturated fat should contribute no more than 11 per cent of the total energy that we get from food (Department of Health, 1991). They say:

- **The average man should eat no more than 30g of saturated fat a day.**
- **The average woman should eat no more than 20g of saturated fat a day.**

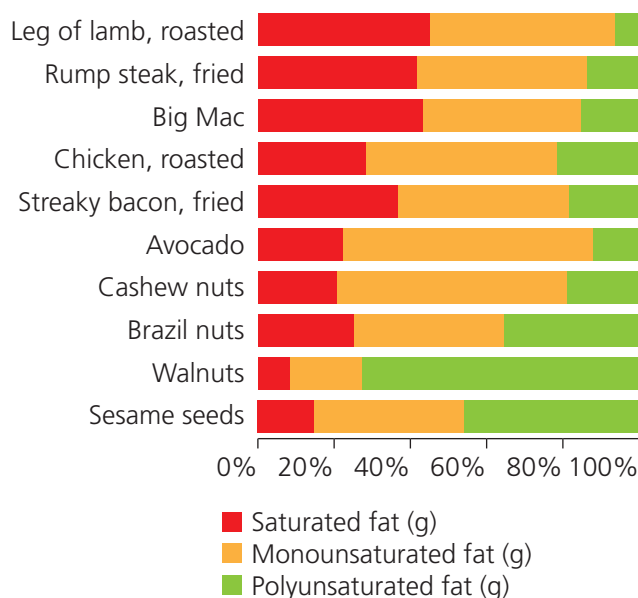
Most people in the UK eat too much saturated fat: about 20 per cent more than the recommended maximum (British Dietetic Association, 2014). Most saturated fat in the average UK diet comes from: fatty cuts of meat, poultry skin, meat products such as sausages and pies, whole milk and full fat dairy products such as cheese and cream, butter, ghee and lard, coconut oil and palm oil, pastry, cakes and biscuits, sweets and chocolate.

The saturated fat content of meat varies widely depending on the species and breed of animal, the way the animal was raised, what the animal was fed, the part of the body from which the meat was taken and the method of cooking. Wild animals such as deer tend to be leaner than farmed animals. However, the fact remains that all meat contains significant amounts of saturated fat that we have no dietary requirement for.

Figure 4.0 shows that meat, nuts, avocados and seeds vary widely in the proportion of 'good' and 'bad' fats they contain. For example, a roast leg of lamb contains 14.2g of fat per 100g, 5.7g of which is saturated and just 0.8g of which is polyunsaturated. Walnuts on the other hand contain 68.5g of fat per 100g (which is a lot of walnuts), 5.6g of which is saturated and a whopping 47.5g is polyunsaturated.

Most health organisations (including the WHO)

Figure 4. Fatty acid composition of different foods shown as a percentage of the total fat content.



Source: FSA 2002.

recommend eating less food rich in saturated fat and more foods containing unsaturated fats such as avocados, nuts, seeds, plant-based oils and spreads. Other rich sources of healthy essential fats include walnuts, flaxseeds, hempseeds, rapeseeds and their oils and oils produced from some species of algae (which contain the omega-3 essential fatty acids EPA and DHA used in our eyes and brains).

PROTEIN

Proteins play an important role in the body, forming the basis of muscle, hair, nails and collagen (the connective tissue that holds the body together). Protein is required for the synthesis of various metabolic products, including neurotransmitters, hormones, haem (found in red blood cells) and DNA.

But where do vegans get their protein?

It's a tired old question vegans get asked by people who think that meat and dairy foods are the only reliable source of protein. They forget that entire populations avoid meat and/or dairy and that humans have been thriving on plant-based sources of protein for thousands of years. If you eat enough calories from a well-balanced vegan diet, it is very difficult to go short of protein. Protein deficiency is rare in industrialised countries and is far more associated with disease or ageing than dietary choices.

WHO suggests that protein should contribute 10-15 per cent of your total energy intake (10-15 per cent of the

MEAT THE TRUTH

Figure 5.0 Protein content of 100g of selected meat-based foods (red) and meat-free alternatives (green).



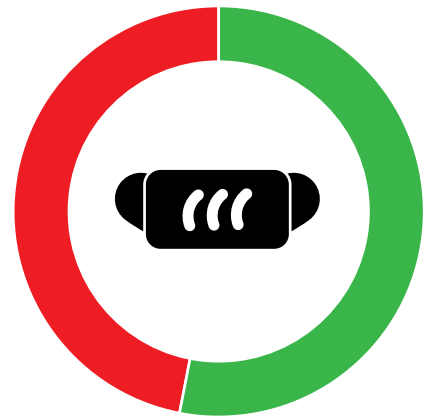
Frankfurter
13.6g

Taifun Organic
Tofu Frankfurter
14.9g



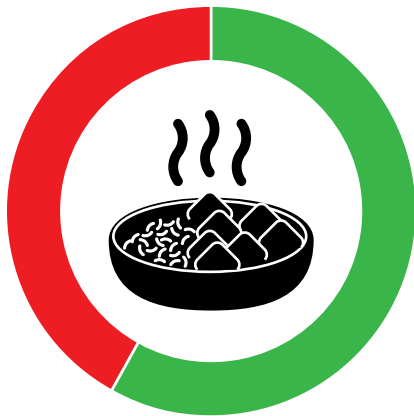
Bacon Rashers,
Streaky 23.8g

Vbites Meat-
free Maple
Cured Rashers 25.5g



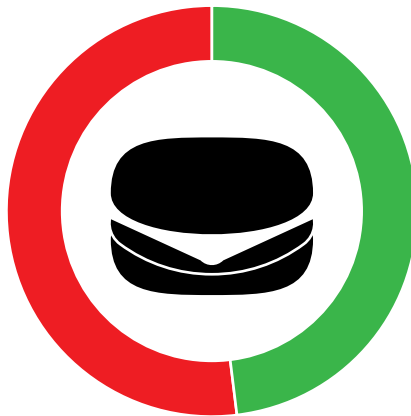
Sausage Rolls
9.9g

Linda McCartney's
Sausage Rolls
11.3g



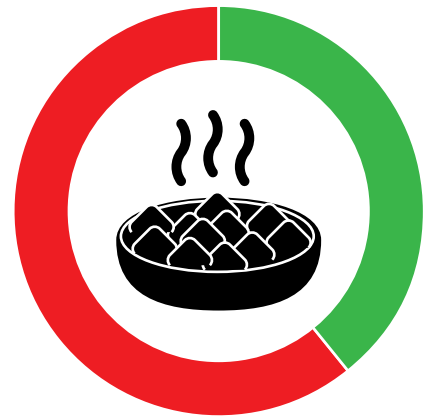
Waitrose Chicken
Jalfrezi 12.1g

Vegan Chicken
Jalfrezi 16.8g



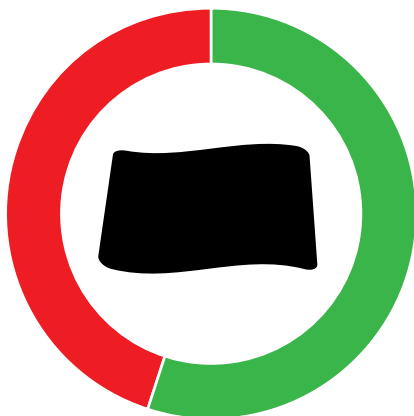
Big Mac 12.4g

Amy's Breakfast
Sandwich 11.5g



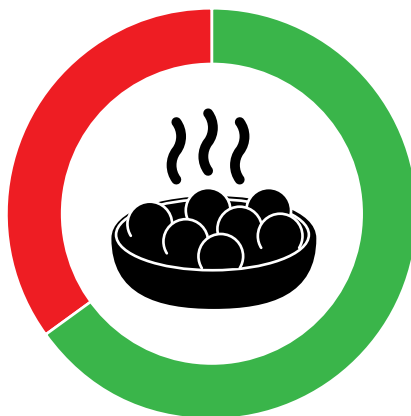
Chicken Breast
in Crumbs 18g

Fry's Golden-
crumbed Schnitzels
11.7g



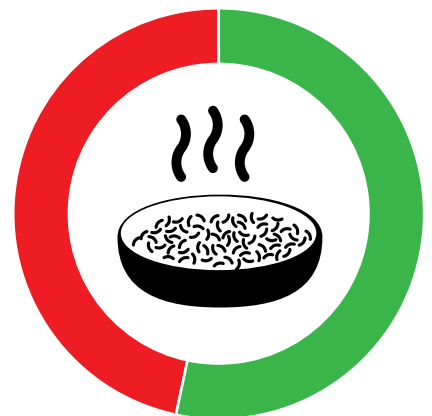
Ham Slices 18.4g

Vbites Meat-free
Ham slices
22.5g



Chicken nuggets
10g

Fry's Chicken-style
Nuggets
18.7g



Beef Mince,
stewed 19g

Meat the Alternative
Beef-style mince
(soya) 21.8g

*Vegan Chicken Jalfrezi: Seeds of Change Indian Jalfrezi sauce with Vbites Chicken Pieces (33%).

*Big Mac: two burgers, bun, sauce, cheese, lettuce and pickles.

*Amy's breakfast sandwich: meatless sausage patty, bun, sauce and scrambled tofu with a Tofutti American Vegan Cheese Slice.

Source: FSA, 2002. The protein contents of vegan products were obtained directly from respective food packaging labels and company websites.

calories you eat). The latest UK NDNS found that average protein intakes met or exceeded that in all groups of people assessed providing 14-15 per cent of food energy for children and 17-18 per cent for adults (Bates *et al.*, 2014).

On average, men should eat around 55g and women 45g of protein daily. That's about two palm-sized portions of tofu, nuts or pulses. Most people find it very easy to eat that much or more. On average, men and women in the UK eat about 45-55 per cent more protein than they need each day (Bates *et al.*, 2014). There is no advantage to eating more protein than you need and too much animal protein is harmful (more on this to follow).

Figure 5.0 shows a comparison of how much protein is in 100g of various meat-based foods and meat-free alternatives. Some meat dishes contain more protein, some vegan ones contain more. Overall, the protein content of the various meat dishes listed is fairly comparable to the vegan alternatives. For example, beef mince contains 21.8g per 100g while soya mince contains 19g. A Big Mac contains 12.4g of protein per 100g and Amy's breakfast sandwich (a meatless sausage patty in a bun with sauce and scrambled tofu plus a slice of Tofutti American Vegan Cheese) contains 11.5g. Sausage rolls, bacon rashers and frankfurters all contain within one or two grams of their vegan alternatives. That said, just as it is not healthy to eat lots of meat, it is not a good idea to eat lots of processed foods as they tend to contain relatively high levels of fat and salt which can increase the risk of obesity and CVD. Perhaps the main difference from a

health perspective is the vegan alternatives are not linked to cancer!

All plant foods contain some protein but some foods provide a better source than others. Table 3.0 shows how much protein may be obtained from an average portion of different plant-based foods. This illustrates how easy it is to get the 45-55g per day recommended without eating meat. For example, if you were to start the day with toast, hummus and cherry tomatoes, then lunch on lentil dahl, spinach and a small portion of brown rice and for dinner have a medium baked potato with a vegan frankfurter and baked beans your protein intake for the day would be 57.6g. On another day, toast and peanut butter for breakfast, two sausage rolls with a green salad for lunch and vegan spaghetti bolognese (with vegan mince) for dinner adds up to 45.9g of protein for the day. A medium-sized bowl of muesli served with soya milk and a banana, a falafel and hummus wrap and a tofu-stir-fry with mixed seeds adds up to 45.8g of protein.

These suggestions do not include drinks, soya milk or fruit so the final figure will be even higher. If you consume enough calories in a varied vegan diet, it is very easy to achieve the desired level of protein.

A high-protein intake is not necessarily a good thing; high intakes of animal protein have been linked to diabetes, cancer and early death. High protein diets also increase the circulating levels of the growth hormone insulin-like growth factor 1 (IGF-1), which increases the risk of cancer.

Table 3.0 The protein content of selected plant-based foods

Men should eat around 55g and women 45g of protein daily

Food (medium portions)	Protein (g)	Food (medium portions)	Protein (g)
Sunflower seeds (1 tbsp)	3.2	Quinoa, cooked (180g)	7.9
Tahini (1 tsp)	3.5	Wholemeal spaghetti (180g)	8.5
Hummus (50g)	3.8	Baked potato with skin (large 220g)	8.6
Peanut butter (20g)	4.5	Mixed nuts (40g)	9.16
Soya yogurt (125g pot)	4.5	Red lentils, cooked (135g)	10.3
Soya milk (200ml)	6.2	Chickpeas, cooked (135g)	11.3
Wholemeal bread (2 medium slices)	6.2	Cauldron Organic Original tofu (100g)	12.6
Heinz baked beans (135g)	6.3	Taifun Smoked Tofu with Almonds &	
Brown rice, boiled (100g)	6.7	Sesame Seeds (100g)	18.8
Almonds (15 almonds)	6.9		

IRON

"Anaemia and iron overload are two of the most prevalent disorders worldwide and affect over a billion people." Anderson and Shah, 2013.

It's a myth that you need to eat meat to get iron. One of the largest studies of vegetarians and vegans, The European Prospective Investigation into Cancer and Nutrition (EPIC) Oxford study, compared over 33,000 meat-eaters, 18,000 vegetarians and 2,500 vegans and found that vegans had the *highest* intake of iron, followed by vegetarians with meat-eaters coming last (Davey *et al.*, 2003). The American Dietetic Association state that iron deficiency is no more common among vegetarians than meat-eaters (Craig and Mangels, 2009).

The meat industry tries to reinforce the idea that you need to eat meat to get iron. A small number of industry-funded studies have fed in to the confusion. The authors of one such study, commissioned by the Meat and Livestock Commission, expressed concern about iron intakes in relation to falling meat intake (Gibson and Ashwell, 2003). The authors (who both run 'independent' nutrition consultancies that have worked with Kellogg's, Danone, Glaxo SmithKline, Coca-Cola, PepsiCo as well as the Meat and Livestock Commission) said that low consumption of red and processed meat has implications for iron intakes and iron status in men and women.

However, contrary to the idea that meat is the main source of iron in a meat-eater's diet, research shows that cereals and cereal products (bread and fortified breakfast cereals) are the principal source of iron in the average UK diet. One study found that the main contribution of iron in a large British cohort was from cereal foods, including breakfast cereals, bread and other cereal products such as pasta, rice, cakes, biscuits and puddings, followed by fruit, nuts and vegetables and then meat coming in last (Johnston *et al.*, 2007). The 2014 NDNS was in agreement – cereals and cereal products were the largest contributor to iron intake for all age groups (Bates *et al.*, 2014). People who don't eat meat can easily get sufficient iron from cereal, pulses and vegetable sources that also provide healthy fats and valuable fibre.

The Reference Nutrient Intake (RNI) is the amount of a nutrient which is sufficient for most people. The RNI for iron is 8.7 mg per day for men and 14.8 mg per day for women up to the age of 50 (Department of Health,

1991). Women have higher requirements as they lose iron during menstruation. If iron intake is low, the amount of haemoglobin in the red blood cells can fall leading to iron-deficiency anaemia. Symptoms include tiredness, weakness, feeling cold and an inability to concentrate.

As mentioned previously (see page 14), iron occurs in two forms; haem iron and non-haem iron. Haem iron is found in animal tissue as it is a component of haemoglobin (oxygen-carrying protein in the blood) and myoglobin (oxygen-carrying molecules in muscle) and makes up around half the iron found in red meat, poultry and fish. Most iron in the diet is non-haem iron (5-10 per cent comes from haem iron in diets containing meat). Haem iron is more readily absorbed; 20-30 per cent of haem iron eaten is absorbed. Absorption of non-haem iron is more variable with 1-10 per cent of non-haem iron absorbed because non-haem iron absorption is subject to a range of influences including a number of dietary factors that can increase or inhibit it. Vitamin C can increase iron absorption while phytates (from high fibre foods), polyphenols found in tea, coffee and red wine, calcium and oxalic acid may inhibit it (see Table 4.0).

Non-haem iron absorption is also affected by iron status – how much iron you already have in the body. People with low iron stores or higher physiological need for iron will tend to absorb more iron and excrete less (Saunders *et al.*, 2013). Haem iron constitutes a smaller part of dietary iron than non-haem iron, but is more bioavailable with 20-30 per cent of haem iron being absorbed in the gut – whether it is needed or not. **A high absorption rate is not necessarily a good thing as the body has no mechanism for disposing of excess iron.** Indeed, excessive iron levels (iron overload) are linked to heart disease, diabetes and bowel cancer. It is well known that many meat-eaters are oversupplied with iron, increasing the risk of heart disease and cancer (Leitzmann, 2005). So, iron from plant foods is more beneficial to the body because its absorption remains safely regulated, whereas iron from meat can accumulate to levels which could be harmful.

That said, iron deficiency is a concern in both developing and industrialised countries; and young women are particularly vulnerable. The NDNS found that iron intakes among some groups in the UK are below the RNI, girls and women are particularly affected. The average iron intake among women aged 19-64 was 78 per cent of the RNI, among girls aged 11-18 it was only just over half of the RNI at 57 per

IRON-RICH FOODS



Edamame (soya beans)



Breakfast cereals:
Shreddies, Weetabix,
Sultana Bran, Ready
Brek and Muesli



Spaghetti (whole wheat)



Cashew nuts



Sesame seeds and
sunflower seeds



Almonds



Lentils (green,
brown and red)



Black-eyed beans



Spinach



Watercress



Pine nuts



Swiss chard



Chick peas



Apricots (dried)



Red kidney beans



Curly kale



Cumin, turmeric
and thyme



Mung beans



Parsley



Bread (wholemeal)



Walnuts



Coconut cream and
dessicated coconut



Spring greens



Hazelnuts



Prunes



Spring onions



Figs (dried)



Raisins and sultanas

Table 4.0 Factors that enhance or inhibit iron absorption

Vitamin C found in abundance in fruit and vegetables, can increase the amount of iron absorbed considerably by converting it into a soluble, more readily absorbable form and by preventing it from forming complexes with phytate or tannin (Fairweather-Tait, 2004). The amount of vitamin C in eight strawberries or 200ml of orange juice can increase iron absorption three- to four-fold (Craig, 1994). To increase your iron absorption substitute your morning cup of tea or coffee with a glass of freshly squeezed orange juice. Combine vitamin C-containing foods with iron-rich meals (beans on toast with apple juice or watercress salad with dates, toasted pumpkin seeds and slices of orange).

Phytate found in unrefined grains, seeds and pulses (which are also a rich source of iron) can bind to iron, calcium, magnesium and zinc and reduce absorption. Soaking pulses and discarding the water can help as can sprouting pulses and grains (Gibson *et al.*, 2014). CookingSprouting can increase iron absorption by 20-62 per cent (Hemalatha *et al.*, 2007). Increasing the amount of time bread is fermented can help. Remember though, wholemeal bread and brown rice contain around two to three times the amount of iron found in white bread and rice (Craig *et al.*, 1994). So even though the percentage of iron absorbed from wholegrain foods may be lower, the total amount of iron absorbed is similar, making wholegrain foods the healthier option as they also contain more vitamins, minerals and fibre.

Polyphenols are chemical substances in plants that possess antioxidant properties that may reduce the risk of CVD and cancer. **Tannins** are a type of polyphenol found in tea and red wine that bind non-haem iron and may reduce iron absorption. So tea (including herbal teas), coffee, cocoa and red wine may inhibit iron absorption. Research shows that tea-drinking does not influence iron status in healthy people who eat a well-balanced diet and have adequate iron stores (Temme and Hoydonck, 2002; Nelson and Poulter, 2004). Try switching to fresh fruit smoothie (as its vitamin C content increases the absorption of iron) and wait at least one hour after eating before drinking tea.

Calcium may inhibit iron absorption, but research suggests that over time, calcium has a limited effect on iron absorption (Saunders *et al.*, 2013). Calcium inhibition may be counteracted by the addition of vitamin C (Walczyk *et al.*, 2014). This doesn't mean you should limit your calcium intake; calcium is a very important mineral. Avoid drinking cow's milk and taking calcium supplements with food (Mangels *et al.*, 2011).

Oxalic acid is a compound found in spinach, Swiss chard and beetroot leaves as well as tea, chocolate and other cocoa products. Some research suggests that oxalic acid may reduce iron absorption. However, recent evidence suggests that it is of minor relevance in iron nutrition (Bonsmann *et al.*, 2008).

cent. When the group surveyed was reassessed taking supplements into account, the average intake across the whole group (including those not taking supplements) increased to 91 per cent of the RNI (Bates *et al.*, 2014).

The Lowest Recommended Nutrient Intake (LRNI) is the amount of a nutrient which is sufficient for only a few individuals. Habitual intakes below the LRNI will almost certainly be inadequate for most people. Nearly half the girls (46 per cent) and almost a quarter of women (23 per cent) in the survey had an iron intake below the LRNI. There was evidence of anaemia plus low iron stores indicative of iron deficiency in nearly five per cent of girls and women. They were not a group of

vegetarians or vegans, so clearly being a meat-eater does not guarantee protection against low iron intakes.

Contrary to the idea that meat is the best source of iron in a meat-eater's diet, grains and cereal products (bread and fortified breakfast cereals) were the main source of iron in the diet, followed by meat and meat products then vegetables. People who don't eat meat can easily get sufficient iron from cereal, pulses, nuts, seeds and vegetable sources that also provide many vitamins, other minerals, healthy fats and valuable fibre.

Table 5.0 shows the iron content of medium-sized portions of selected foods including different types of meat, fruit, vegetables, pulses and wholegrain foods.

Table 5.0 The iron content of selected foods

RNI: 8.7mg/day for men and 14.8mg/day for women

Food (medium portions)	Iron (mg)	Food (medium portions)	Iron (mg)
Tesco Malt Wheats, Sainsbury's Wholegrain		Lamb chop, grilled (edible portion 70g)	1.5
Malties or Waitrose Malted Wheats (40g)	4.8	Spinach, boiled (90g)	1.4
Medium steak, fried (144g)	4.3	Sesame seeds (1 tbsp/12g)	1.2
Tofu, fried (100g)	3.5	Prunes (six dried fruits 48g)	1.2
Wholemeal Spaghetti, boiled (220g)	3.1	Cashew nuts (20 nuts ~ 20g)	1.2
Pumpkin seeds (1 handful/35g)	3.1	Brown rice, boiled (180g)	0.9
Red lentils, boiled (120g)	2.9	Broccoli, boiled (85g)	0.9
Figs (three dried fruits 60g)	2.5	Wholemeal bread (36g)	0.9
Quarter pounder beef burger, grilled (78g)	2.0	*Alpro Soya milk (200ml)	0.7
Baked beans (135g)	1.9	Chicken breast, grilled (130g)	0.5
Curly kale, boiled (95g)	1.9	Bacon, grilled (46g)	0.4
Kidney beans, canned (90g)	1.8		

Source: FSA, 2002 and Alpro*.

The RNI for women under 50 is 14.8mg per day. This amount of iron can be obtained from a bowl of cereal with fruit (blueberries or strawberries) for breakfast, lentil and pasta salad for lunch and smoked tofu stir-fry with brown rice, broccoli and pumpkin seeds for dinner. Alternatively, scrambled tofu and wholemeal toast for breakfast with orange juice, lentil soup for lunch and chickpea and spinach curry for dinner would provide plenty of iron. Including vitamin C-containing foods with your iron-rich meals can boost the amount of iron you absorb substantially.

ZINC

Zinc is an essential trace element that has several important functions; it helps to make new cells and enzymes, helps us process carbohydrate, fat and protein in food, strengthens the immune system and can help with the healing of wounds. Zinc may inhibit the replication of the rhinovirus; the most frequent cause of common cold symptoms. A 2011 Cochrane review suggests that taking zinc supplements within a day of the symptoms starting can speed up recovery and lessen the severity of a cold (Singh and Das, 2011).

The RNI for zinc is 7mg per day for women and 9.5mg per day for men (Department of Health, 1991). Like iron, the bioavailability of zinc may be reduced if there is a high presence of absorption inhibitors such as phytate and polyphenols. However, you can limit the effect of these as described above; soaking and sprouting pulses, grains and seeds, and fermenting grain products (using bread rather than crackers). As stated for iron, although

zinc may be absorbed at a lower rate from wholegrain bread than white bread (due to the phytates), but the higher amount of zinc in wholegrain bread more than compensates for the lower absorption.

It has been suggested that vegans with high intakes of unrefined grains might need slightly more zinc than recommended (Gibson *et al.*, 2014). However, you shouldn't take more than 25mg of zinc supplements a day, unless advised to by a doctor. Taking high doses can lead to a copper deficiency, anaemia and weakening of the bones.

In a large EPIC study of meat-eaters, fish-eaters, vegetarians and vegans, average zinc intakes among women were above the RNI of 7mg in all dietary groups. Among the men, the average intake of all groups except the meat-eaters was slightly below the RNI of 9.5mg per day, but the meat-eaters' intake was only just above it at 9.78mg day (Davey *et al.*, 2003).

These results concur with those of the earlier NDNS from 2008/2009 and 2011/2012 which found that the average daily intake of zinc from food was close to or above the RNI for most people. Boys aged 11-18 years were slightly under at 90 per cent of the RNI and girls that age a bit lower still at 81 per cent of the RNI (Bates *et al.*, 2014). A number of people (nine per cent of 4-10 year olds, 17 per cent of 11-17 year olds and 10 per cent of men over 65) had zinc intakes below the LRNI. Other studies from industrialised countries show that for vegetarians, up to the age of 11, zinc intakes are similar or even higher than those of meat-eaters. This

Table 6.0 The zinc content of selected foods

RNI: 7mg/day for women and 9.5mg/day for men

Food (medium portions)	Zinc (mg)	Food (medium portions)	Zinc (mg)
Pumpkin seeds (1 handful/35g)	2.7	Wholemeal Spaghetti, boiled (220g)	0.7
Lamb chop, grilled (edible portion 70g)	2.5	Wholemeal bread (36g)	0.6
Tofu, fried (100g)	2.0	Sesame seeds (1 tbsp/12g)	0.6
Bacon, grilled (46g)	1.2	Kidney beans, canned (90g)	0.6
Brown rice, boiled (180g)	1.2	Spinach, boiled (90g)	0.5
Red lentils, boiled (120g)	1.2	Figs (three dried fruits 60g)	0.4
Cashew nuts (20 nuts ~ 20g)	1.1	Broccoli, boiled (85g)	0.3
Chicken breast, grilled (130g)	1.0	Prunes (six dried fruits 48g)	0.2
Bran flakes (30g)	0.8	Curly kale, boiled (95g)	0.2
Baked beans (135g)	0.7		

Source: FSA, 2002.

may reflect plant-based foods being the major source for iron and zinc during childhood, irrespective of dietary practices (Gibson *et al.*, 2014).

Table 6.0 shows the shows the zinc content of medium-sized portions of selected foods including different types of meat, fruit, vegetables, pulses, nuts and wholegrain foods. The concentration of zinc in plants varies based on levels of the element in soil. When there is adequate zinc in the soil, the food plants that contain the most zinc are wheat (germ and bran) and various seeds (pumpkin, sunflower and sesame).

While red meat undoubtedly provides a source of zinc, it also contains high levels of unhealthy saturated fat, cholesterol and growth hormones. Furthermore, animal protein (but generally not plant protein) raises levels of IGF-1, a growth hormone linked to several cancers. Red and processed meats are also linked to several cancers including bowel, prostate and pancreatic cancer (WHO/IARC, 2015). You are better off getting your zinc from plant-based sources such as wholegrain foods, nuts and seeds which contain healthy essential fatty acids and valuable fibre.

A daily intake of 10mg could be achieved from wholegrain cereal and soya milk, beans or hummus on wholemeal toast, tofu or tempeh stir-fry with cashews, broccoli and pumpkin seeds and brown rice. Additional snacks of dried fruit, nuts and seeds would provide a substantial boost.

SELENIUM

Selenium is a trace element that is essential for a wide

range of biochemical functions within the body. It plays an important role in our immune system and in reproduction. It also helps to prevent damage to cells and tissues.

The RNI for adults is 75µg a day for men and 60µg a day for women. High intakes of selenium can be toxic. The Expert Group on Vitamins and Minerals (EVM) set a safe upper limit for selenium intake at 450µg per day, in North America the upper level of tolerable intake is set slightly lower at 400µg per day (SACN, 2013).

Selenium intake varies widely around the world, mainly due to the differences in the availability from the soil. Selenium is less readily taken up by plants growing in more acidic, impervious soils (Fordyce, 2005). This makes it difficult to make food composition tables and estimates of dietary intakes may be of limited reliability for determining actual intakes (SACN, 2013). The UK is reported as having low selenium intakes with an average adult intake of 48µg per day from food sources or 51µg including supplements (SACN, 2013). If your food is grown in soil that has low selenium levels it could be a problem regardless of dietary preference. Because European soil and plants are relatively poor sources of selenium, farmed animals are often supplemented with it (Hoeftlich *et al.*, 2010). If you are concerned about getting enough, you could cut out the middleman and take a supplement.

According to the NDNS, teenagers and adults are estimated to have average selenium intakes below the RNI. Only boys and girls aged 1.5-10 years had an average intake above the RNI (which is 15-30µg per day for this age group). It should be noted however, that the selenium dietary reference values were set on very

limited data and caution should be exercised when using the RNI to infer the adequacy of selenium intake in the population (Bates *et al.*, 2014).

The research on vegan diets and selenium has produced mixed results (de Bortoli and Cozzolino, 2009; Hoeflich, 2010; Fayet, 2014). It is well-documented how a well-balanced vegan diet offers a wide range of beneficial health effects. However while a not well-balanced vegan diet (chips and beans), may still be preferable to not well-balanced meaty diet (chips and burgers), it may be low in certain nutrients such as selenium. A vegan diet is not fool-proof, some common sense is required.

The main plant-based sources of selenium in the UK diet are bread and cereals. Table 7.0 shows how Brazil nuts provide a very rich source of selenium. The amount contained in 100g of Brazil nuts can range from 85-690µg per 100g (FSA, 2002). Consuming just two Brazil nuts a day for 12 weeks can increase the amount of selenium in the blood by over 60 per cent (Thomson *et al.*, 2008). Including Brazil nuts in the diet could avoid the need for supplements.

Nuts are a healthy, nutritious food that provide an excellent source of vitamin E and magnesium. People who eat nuts also have higher intakes of folate, beta-carotene, vitamin K, calcium, phosphorus, copper, selenium, potassium and zinc. Nuts provide valuable phytochemicals and their antioxidant power is similar to that of broccoli and tomatoes. Eating 42g (a generous handful) of mixed nuts a day can reduce the risk of heart disease (King *et al.*, 2008).

Table 7.0 The selenium content of selected foods

RNI: 60µg/day for women and 75µg/day for men

Food (100g)	Selenium (µg)
Brazil nuts	85-690
Green or brown lentils, dried, boiled	40
Mushrooms, fried in corn oil	12
Red kidney beans, canned	6
Mung beans, boiled	5
Soya milk	4

Source: FSA, 2002.

PHOSPHORUS

Phosphorus is a mineral that helps to build strong bones and teeth and helps to release energy from food. Phosphorus is abundant in most diets and deficiencies are highly unlikely. The RNI for adult women and men is 550mg of phosphorus per day.

Too much can be harmful. Taking high doses of phosphorus supplements for a short time can cause diarrhoea or stomach pain. Taking high doses for a long time can reduce the amount of calcium in the body, making bones more prone to fracture.

Phosphoric acid is used as a preservative in a variety of fizzy drinks such as Coca Cola, Diet Coke, Coke Zero and Dr Pepper. Phosphoric acid contains a small amount of the mineral phosphorus. Research shows that cola drinks may cause bone loss in older women. The phosphoric acid (and sugar) in cola may be to blame, or it may be that cola is drunk in place of healthier calcium-containing drinks such as calcium-fortified soya milk (Tucker *et al.*, 2006).

Table 8.0 shows that a wide range of wholegrain foods, pulses, vegetables and fruits (especially dried fruits) contain phosphorus. You can get all the phosphorus you need from a varied plant-based diet that does not include meat.

Table 8.0 The phosphorus content of selected foods.

RNI: 550mg/day

Food (100g)	Phosphorus (mg)
Wholemeal bread, toasted	237
Garlic, raw	170
Broad beans, frozen, boiled	150
Brown rice, boiled	120
Kidney beans, canned	130
Red lentils, boiled	100
Apricots, ready-to-eat	82
Chickpeas, canned	81
Parsnip, boiled	76
Broccoli, boiled	57
Asparagus, boiled	50
Blackcurrants	43
Green or brown lentils, dried, boiled	40
Avocado	39

Source: FSA, 2002.

Table 9.0 The vitamin B2 (riboflavin) content of selected foods

RNI: 1.1mg/day for women and 1.3mg/day for men

Food (100g)	Vitamin B2 (riboflavin) (mg)
Yeast extract	11.90
Tesco Malt Wheats, Sainsbury's	
Wholegrain Malties or Waitrose	
Malted Wheats	1.20
Almonds	0.75
Wheatgerm	0.72
Mushrooms, fried in corn oil	0.34
Lentil, green/brown, dried, boiled	0.27
Avocado	0.18
Sesame seeds and tahini	0.17
Apricots, ready-to-eat	0.16
Wheatgerm bread	0.11

Source: FSA, 2002.

Table 10.0 The vitamin B3 (niacin) content of selected foods

RNI: 13mg/day for women and 17mg/day for men

Food (100g)	Vitamin B3 (niacin) (mg)
Yeast extract	64.0
Peanuts	13.8
Peanut butter	12.5
Tahini	5.1
Sesame seeds	5.0
Wholemeal toast	4.5
Sunflower seeds	4.1
Pine nuts	3.8
Mushrooms, raw	3.2
Apricots, ready-to-eat	2.3
Peas, boiled	1.8
Brown rice, boiled	1.3
Avocado	1.1

Source: FSA, 2002.

VITAMIN B2 (RIBOFLAVIN)

Riboflavin helps keep skin, eyes and the nervous system healthy and helps the body release energy from the food we eat. The RNI for riboflavin is 1.3mg a day for men and 1.1mg a day for women (NHS Choices, 2015a). Riboflavin cannot be stored in the body, so you need it in your diet every day. If you take supplements, do not take too much, because this might be harmful. The government say that 40mg or less a day of riboflavin supplements is unlikely to cause any harm (NHS Choices, 2015a).

Table 9.0 shows the riboflavin content of 100g portions of selected foods. You should be able to get all the riboflavin you need by eating a varied and balanced diet. Yeast extract is considered to be an exceptionally rich source. UV light can destroy riboflavin, so ideally these foods should be kept out of direct sunlight.

VITAMIN B3 (NIACIN)

Niacin (vitamin B3 or nicotinic acid), has several important functions, including helping to release energy from the foods we eat and helping to keep the nervous systems and skin healthy. The RNI for niacin is 17mg a day for men and 13mg for women. There are two forms of niacin: nicotinic acid and nicotinamide, both of which are found in food. Taking high doses of nicotinic acid supplements can cause skin flushes and taking

high doses for a long time could lead to liver damage (NHS Choices, 2015a). The Department of Health says you should be able to get all the niacin you need by eating a varied and balanced diet. Taking 17mg or less of nicotinic acid supplements a day or 500mg or less of nicotinamide supplements a day is unlikely to cause any harm (NHS Choices, 2015a).

The average niacin content of various types of beef, lamb and chicken lies in the range of 5.0-10mg per 100g. Table 10.0 shows you can get all the niacin you need from a varied vegan diet.

VITAMIN B6 (PYRIDOXINE)

Vitamin B6 (pyridoxine) has several important functions, including helping the body use and store energy from protein and carbohydrates in food. It also helps to form haemoglobin (the substance in red blood cells that carries oxygen around the body). The RNI for vitamin B6 is 1.4mg a day for men and 1.2mg a day for women. The government say that you should not take more than 10mg of vitamin B6 a day in supplements unless advised to by a doctor. Taking more than 200mg a day of vitamin B6 for a long time can lead to a loss of feeling in the arms and legs (peripheral neuropathy). Taking doses of 10-200mg a day for short periods may not cause any harm. However, there is not enough evidence to say for how long these doses could be taken safely (NHS Choices, 2015a).

Table 11.0 Vitamin B6 (pyridoxine) content of selected foods.

RNI: 1.2mg/day for women and 1.4mg/day for men

Food (100g)	Vitamin B6 (pyridoxine) (mg)
Wheatgerm	3.30
Yeast extract	1.60
Tesco Malt Wheats, Sainsbury's Wholegrain Malties or Waitrose Malted Wheats	1.20
Sesame seeds and tahini	0.75
Peanuts	0.59
Avocado	0.36
Potatoes, boiled	0.33
Banana	0.29
Lentil, green/brown, dried, boiled	0.28
Apricots, ready-to-eat	0.14
Wholemeal toast	0.13

Source: FSA, 2002.

Table 11.0 shows that you should be able to get all the vitamin B6 you need by eating a varied and balanced vegan diet.

VITAMIN B12

All B vitamins help the body produce energy from food. Vitamin B12 also helps maintain healthy nerve cells and helps in the production of DNA, the body's genetic material. B12 works closely with folate, to make red blood cells, to help iron work better in the body and to produce a compound involved in immune function and mood.

B12 deficiency can lead to serious health problems, especially in the very young. Symptoms include: extreme tiredness, lack of energy, pins and needles, muscle weakness, depression and cognitive problems such as impaired memory, understanding and judgement. A lack of B12 can lead to a raised level of the amino acid homocysteine in the blood which has been linked to CVD; this can affect meat-eaters, vegetarians and vegans. You can easily be tested by your doctor and a B12 deficiency may be treated with supplements or a course of injections.

Vitamin B12 is a water-soluble vitamin that can be stored for years in the liver. Excess amounts leave the body in the urine. The UK government suggest an RNI of 1.5µg (micrograms) of B12 per day. In the US, the recommended amount is 2.4µg. However, a recent study suggested the ideal intake lies between 4-7µg per day (Bor *et al.*, 2010). Another study suggested that a daily intake of between 6-10µg should ensure an adequate intake of B12 and minimise the build-up of homocysteine (Vogiatzoglou *et al.*, 2009).

In 2015, the European Food Safety Authority set a new 'adequate intake' dietary reference value for vitamin B12 of 4µg per day (EFSA, 2015). This figure was based on data from 13 dietary surveys in nine European Union countries and is based on preventing B12 deficiency and therefore may not represent the optimum intake. Erring on the side of caution, Viva!Health recommends an intake of 5µg per day from fortified foods with the regular use of supplements to ensure topping this up. This is particularly important for children too.

There are a number of different forms of B12:

Cyanocobalamin is a cheap and stable 'inactive' form used to fortify infant formula, breakfast cereals, vitamin drinks, plant milks and vegan meat replacements, as well as animal and fish feed. It is called inactive because it needs to be converted into an active form to work in the body. Tablets typically contain doses from as low as 2.5µg up to 1,000µg.

Hydroxocobalamin is used as an injectable form of vitamin B12 that is given when there are problems with absorption. In the UK 1,000µg per dose is generally used.

Methylcobalamin is an 'active' form of vitamin B12. It costs more as it is not so stable which is why it is



MEAT THE TRUTH

provided in higher doses from 500-2,000µg. It needs to be stored away from light.

Adenosylcobalamin is another 'active' form of vitamin B12, available as a supplement that needs to be stored away from light.

Hydroxocobalamin may be better for treatment by injection. Methylcobalamin and cyanocobalamin show very similar effects in the body, but on balance, cyanocobalamin may be superior as a supplement because of its stability (Obeid *et al.*, 2015). US physician, author and internationally recognised speaker on public health issues, Dr Michael Greger says: *"Unless you're a smoker or have kidney failure, cyanocobalamin should be fine. That's what I take!"* (Greger, 2012).

Vitamin B12 absorption can be reduced by a number of factors: poor functioning kidneys (McMahon *et al.*, 2015); the diabetes drug Metformin and proton pump inhibitors (PPIs) which inhibit the production of stomach acid (Long *et al.*, 2012); nitric oxide in cigarette smoke (Gabriel *et al.*, 2006); nitrous oxide (laughing gas) used for anaesthesia or recreational use (Rusher and Pawlak, 2013) and heating food and drink in a microwave or other forms of cooking (Watanabe, 1998; Czerwinka *et al.*, 2014).

The Department of Health cautions that if you take vitamin B12 supplements, you should not take too much, because this could be harmful. However, the amount they say you can take before it might be harmful is substantially higher than the RNI. They say that taking 2,000µg or less a day of vitamin B12 in supplements is unlikely to cause any harm (NHS Choices, 2015a).

B12 is made by bacteria in soil and water and to some extent bacteria in the gut (although production in the gut occurs in a different area to where absorption takes place). Traditionally farmed animals got B12 from eating food from the ground because B12 was in the bacteria in the soil. B12 consumed in their diet was then taken up into the cells in their bodies, which is how vitamin B12 ends up in red meat, fish, poultry, eggs and dairy products.

Vitamin B12 is also produced commercially in large vats where bacterial cultures are grown for its extraction. This type of B12 is used in fortified foods: veggie burger mixes, yeast extracts, margarines, breakfast cereals, soya milks and B12 supplements for humans and animals. In modern society, fruit and vegetable production is far more sanitised in that fruit and vegetables are washed in chlorine for sale in supermarkets. This removes the

B12-producing bacteria and so vegans must obtain vitamin B12 from fortified foods and/or supplements. Similarly, modern factory farming methods have changed the nature of the food farmed animals eat meaning that cattle and sheep now need B12 supplements too!

France accounts for 80 per cent of world production of cyanocobalamin (the most common and widely produced form of B12) producing more than 10 tons per year; over half of that (55 per cent of sales) are destined for animal feed, while the remaining 45 per cent is for humans (Kaesler, 2005). This makes the B12-reason to eat animal products somewhat invalid. For those who don't like taking supplements, how is it any better to eat an animal that had been fed supplements? You could just cut out the middleman and get your B12 straight from the source, it's easier to absorb that way too (see below).

Plant foods, fermented soya foods and seaweeds do not provide a reliable source of B12 with the possible exception of the seaweed nori (Watanabe *et al.*, 2014). However, this has yet to be confirmed by more substantive evidence. **Until nori and other plant foods said to provide B12 are shown consistently to correct B12 deficiency, vegans should not rely on them for vitamin B12.**

Vitamin B12 in meat is bound to animal protein and so is more difficult to absorb than the unbound form produced by bacteria. B12 deficiency tends to increase with age; up to 40 per cent of the UK's meat-eating elderly population suffers from low B12 due to a reduction in their ability to absorb this vitamin (Tucker *et al.*, 2000). In fact, mild to moderate B12 deficiency is common in industrialised countries despite the fact that a typical western diet provides around 5-7µg B12 per day (Obeid *et al.*, 2015). This may be explained by an age-related decrease in the ability to release B12 from animal protein or by an impaired intestinal absorption of B12. The most common cause of B12 deficiency in the UK is the loss of intrinsic factor (a protein produced in the stomach) which may result from a genetic predisposition and tends to be age-related (Herbert, 1994). In the elderly, a decline in the amount of acid produced in the stomach can also reduce B12 absorption; again this mainly affects B12 absorption from meat. People with decreased gastric secretion often have difficulty digesting collagen, a major constituent of meat that is primarily digested by the enzyme pepsin, which could prevent the release of vitamin B12 from animal protein (Vogiatzoglou *et al.*, 2009).

Table 12.0 The vitamin B12 content of selected foods.

RNI: 1.5µg/day

Food (medium portions)	Vitamin B12 (µg)	Food (medium portions)	Vitamin B12 (µg)
Medium steak, fried (144g)	2.9	Alpro Heavenly Velvet Vanilla Dessert (125g pot)	0.3
Meridian Yeast Extract (4g serving – enough for one slice of toast)	2.8	Chicken breast, grilled (130g)	Trace
Soya milk – Alpro Soya Original (200ml)	2.5		
Quarter pounder beef burger, grilled (78g)	2.3	Note that some of the organic versions of these products are not fortified with B12.	
Marigold Engevita with Added B12			
Yeast Flakes (5g)	2.2		
Lamb chop, grilled (edible portion 70g)	2.1	*Many cereals fortified with B12 also contain vitamin D (D3) from lanolin, a substance obtained from sheep's wool. At the time of writing, Tesco Malt Wheats, Sainsbury's Wholegrain Malties and Waitrose Malted Wheats all contain 2.1µg of B12 per 100g and no vitamin D, so are suitable for vegans. Kellogg's only use D3 at the moment but are looking into the possibility of using D2, which is suitable for vegans.	
Koko long-life dairy free coconut drink (200ml)	0.8		
Alpro Simply Plain Yoghurt	0.6		
Fortified cereal* (40g)	0.8		
Marmite (4g serving – enough for one slice of toast)	0.6		
Vecon Vegetable Stock (1tsp/5g)	0.5		
Pure Soya margarine (10g)	0.5		
Bacon, grilled (46g)	0.5	Source: FSA, 2002 and respective food packaging labels and company websites.	

Furthermore, while meat and meat products may *contain* vitamin B12, most meat is consumed after cooking, which can cause significant losses of B12 (Watanabe, 2007).

Studies suggest that the bioavailability of vitamin B12 from meat is lower than previously thought. This may be because of losses incurred in cooking or difficulty the body has freeing up B12 bound to animal protein. Either way, meat is not the best source of B12 and you are better off getting it from fortified foods and/or supplements.

In the US, The Institute of Medicine of the National Academy of Sciences recommends that *all* adults over 50 years (including meat-eaters) obtain B12 from vitamin supplements or fortified foods because of the high incidence of impaired B12 absorption from animal foods in this age group (Institute Of Medicine, 1998). It could be argued that vegans have a heads-up on B12 as they routinely include a supplement or fortified foods in their diet.

Table 12.0 shows the amount of B12 you can find in a range of meat and plant-based foods. Note that the 144g steak listed provides more than two times the daily 70g limit of red meat recommended by the government. It is quite easy to ensure your 5µg of B12 per day by including certain foods regularly in the diet; fortified

cereal with soya or coconut milk, yeast extract, vegan spread, Vecon stock in soups and stews, a generous sprinkle of yeast flakes on your dinner and occasionally, certain fortified desserts (Alpro soya yoghurts and soya desserts contain B12). Of course, you need to ensure all these foods are the fortified varieties! Topping up once or twice a week with a supplement can be useful as a safety net to ensure you are getting sufficient B12.

A study in Switzerland found that despite a relatively low B12 intake from food in the vegan group they looked at, deficiency of this vitamin was low thanks to the widespread use of supplements. They concluded that consuming a well-balanced diet including supplements or fortified products, all types of diet can potentially fulfil requirements for vitamin and mineral consumption (Schüpbach *et al.*, 2015).

B12 intakes among vegans are thought to be increasing, reflecting the increase in the number of B12-fortified products available coupled to a raised awareness. This will undoubtedly confer an advantage on vegans in later life who are used to ensuring sufficient B12 is present in their diet. It may be that vegans have gained an advantage in that they are used to routinely consuming B12-fortified foods and are therefore less likely to experience age-related B12 deficiencies.

WHO EATS MEAT? COMPARING CARNIVORES

GLOBAL INTAKES

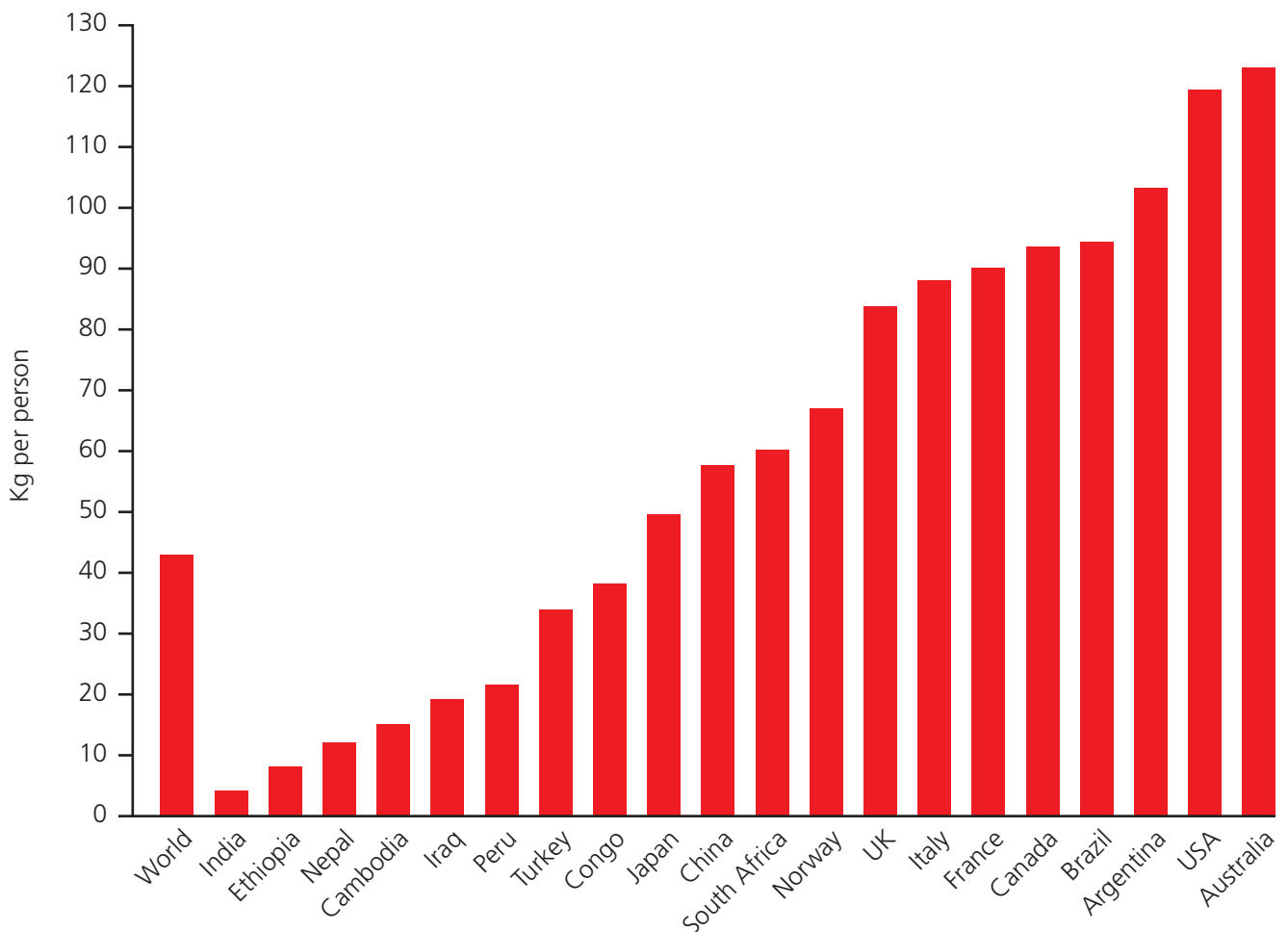
The amount of meat people eat varies widely around the world. The Food and Agriculture Organisation Corporate Statistical Database (FAOSTAT) website disseminates statistical data collected and maintained by the Food and Agriculture Organisation of the United Nations. They produce food balance sheets presenting a comprehensive picture of the pattern of a country's food supply during a specified period.

Figure 6.0 shows the lowest levels of meat consumption are seen in parts of Asia, Africa and the Middle East. In 2011, the average intake of meat in India was just 4kg per person over the year. In Ethiopia it was 8kg and in

Iraq it was 19kg, considerably lower than the world average intake of 42kg. Among the top consumers for 2011 were New Zealand (127kg), Australia (121kg), the US (117kg), Austria (106kg) and Israel (102kg). The average intake per person in the UK was 82kg, a little less than Italy and France and close to the average across the European Union of 83kg.

In general, wealthier countries consume more meat. However, there are exceptions; meat consumption in Brazil (93kg per year) was higher than that in France (89kg per year) although Brazil has a gross domestic product (GDP) per capita less than a third of that in France (World Bank, 2015).

Figure 6.0 Annual meat consumption (kg per person) for 2011 for selected countries.



Source: FAOSTAT, 2015.

UK MEAT CONSUMPTION — GOING DOWN!

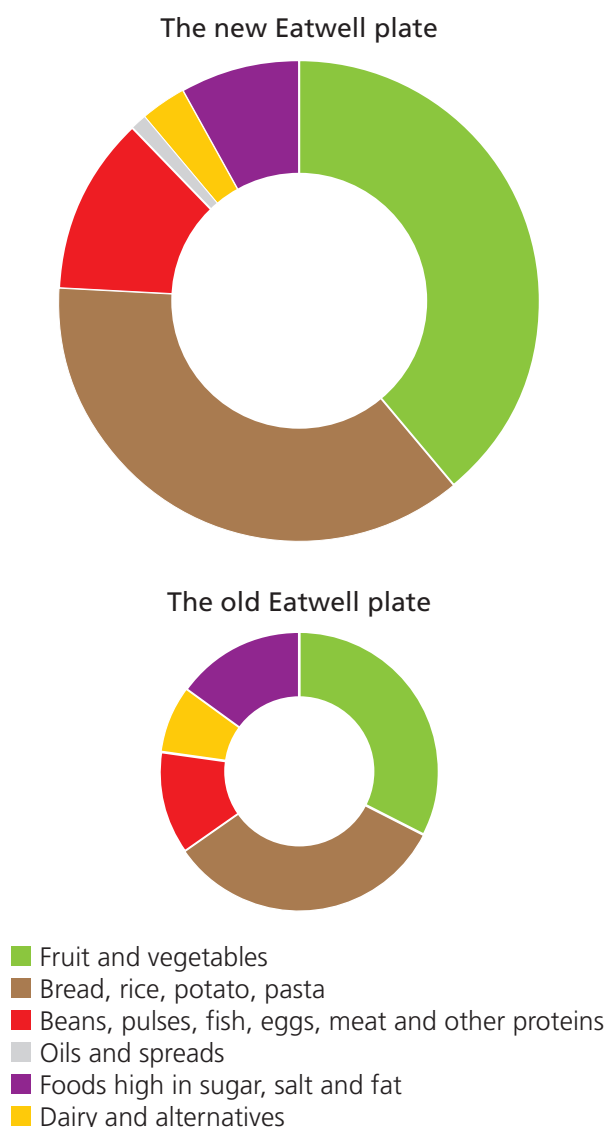
The average amount of meat eaten per person in the UK is almost double the world average. If you convert it into an average daily consumption, it comes out at **226g of meat per day** for the UK. The government recommends eating no more than 70g of red or processed meat (two slices of bacon a day) and don't specify a limit on white meat but, according to Public Health England's new Eatwell guide, they say that protein-rich foods (beans, pulses, fish, eggs, meat and other non-dairy sources of proteins) should make up no more than 12 per cent of the of total energy intake per day (Public Health England, 2016b).

In 2016, Public Health England's new dietary advice recommended people halve their dairy intake and eat less meat, replacing it with beans and pulses. Figure 7.0 shows their redesigned and reworded Eatwell Guide which moved away from animal-based foods favouring more plant-based options. It seems that they are finally responding to the huge body of scientific evidence showing how harmful meat and dairy are to health. The new guidelines represent a small but significant step towards healthier eating, emphasising the importance of fruit, vegetables and complex carbohydrates (such as brown rice and wholemeal bread) in the diet. The shift in emphasis away from meat and dairy is a view more in keeping with the current research, which acknowledges the harm meat and dairy do to our health and the environment. Viva!Health have been campaigning for change for years and this has been a long time coming. The government now need to go one step further and accept the well-documented benefits of a fully vegan diet excluding all meat, fish, eggs and dairy foods.

The new guide says: *"Beans, peas and lentils (which are all types of pulses) are good alternatives to meat because they're naturally very low in fat, and they're high in fibre, protein, and vitamins and minerals. Pulses, or legumes as they are sometimes called, are edible seeds that grow in pods and include foods like lentils, chickpeas, beans and peas. Other vegetable-based sources of protein include tofu, bean curd and mycoprotein*; all of which are widely available in most retailers".*

*Quorn products are made from **mycoprotein** which is a fungal protein; 'myco' is from the Greek word for fungus.

Figure 7.0 Comparison of Public Health England's new Eatwell Guide with the previous Food Standards Agency's recommended Eatwell plate.



Sources: Public Health England, 2016; FSA, 2012.

The dairy category has been renamed 'Dairy and alternatives' and the amount we should eat has been reduced from 15 per cent to eight per cent. Explaining why dairy products have been downgraded a spokesperson for Public Health England said: *"Our independent expert body said you can get calcium from across the diet and not just from dairy products. We are currently meeting or exceeding calcium recommendations whereas we are still consuming too much saturated fat and salt."*

Alison Tedstone, chief nutritionist for Public Health England said: *"Our new Eatwell Guide helps people to*

MEAT THE TRUTH

understand what a healthy balanced diet looks like. The evidence shows that we should continue to base our meals on starchy carbohydrates, especially wholegrain, and eat at least five portions of a variety of fruit and vegetables each day. On the whole, cutting back on foods and drinks that are high in saturated fat, salt, sugar and calories would improve our diets, helping to reduce obesity and the risk of serious illnesses such as heart disease and some cancers.”

This is a small but important departure for government health guidelines. The Carbon Trust sustainability assessment said that the new Eatwell Guide would have a much lower impact on the environment than the current UK diet does (The Carbon Trust, 2016). It seems that we are finally joining up the dots between what is good for us and what is good for the environment.

The National Diet and Nutrition Survey (NDNS) is a study of people’s eating habits across the UK jointly funded by Public Health England and the FSA. Each year about 1,300 people aged 18 months and up take part. The survey gives a snapshot of the country’s diet and

nutritional habits and helps monitor the success of government health initiatives, like the 5-a-day campaign.

Public Health England’s Eatwell Guide suggests that meat, fish, eggs, beans and other non-dairy sources of protein should make up no more than 12 per cent of the of total energy intake per day. The 2000/2001 NDNS found that these foods made up 22 per cent, almost double the recommended amount (17 per cent of which were meat or meat products). Within the protein group of foods, a fifth of was made up of chicken and turkey dishes, 15.5 per cent was beef and veal dishes, the remainder consisted mainly of baked beans (7.0 per cent), eggs (6.7), bacon and ham (6.6), meat pies and pastries (6.3), oily fish (5.4), sausages (4.2), pork and pork dishes (3.9) and white fish (3.9). Nuts and seeds, an excellent source of protein, energy and healthy fat, contributed less than one (0.9) per cent of energy intake.

Foods and drinks high in sugar and fat made up 15 per cent, five times the amount recommended. Over a fifth of this category was made up of chips, the rest consisted of buns, cakes and pastries, fried potatoes and products made from them, biscuits, sugar, chocolate, fizzy drinks, crisps, cereal-based puddings and ice-cream. One of the problems with such high intakes of meat, fat and sugary foods is that it inevitably occurs at the expense of other, healthier foods such as fruit and vegetables, and carbohydrates (bread, rice pasta and potatoes).

More recently, the 2014 NDNS found that again, meat contributed more than the recommended 12 per cent energy intake. Meat was the second largest contributor (after cereals) to energy intake for children aged 11-18 years and adults aged 19-64, contributing 17 per cent of energy intake in both groups, down from 22 per cent in 2000/2001. They found that the average consumption of total meat and red meat was lower in most groups assessed in 2011-2012 compared with an earlier assessment in 2008-2009 (Bates *et al.*, 2014). So although on a global scale, UK meat consumption is relatively high, it is in a steady and consistent decline.

We may be moving in the right direction but the speed at which we are moving is painfully slow with the majority of people in industrialised countries still consuming meat. The 2014 NDNS report found the average consumption of **red meat** for adults aged 19-64 was close to the government’s recommended upper limit of 70g per day coming in at 71g per day. However, men were found to be consuming significantly higher amounts (86g for men and 56g for women). For adults

Table 13.0 A comparison of the new advice from Public Health England’s Eatwell Guide compared to the FSA’s old Eatwell Plate

FOOD GROUP	OLD ADVICE	NEW ADVICE
Fruit and veg (at least 5-a-day)	33 per cent	39 per cent
Potatoes, bread, rice, pasta (choose wholegrain varieties)	33 per cent	37 per cent
Beans, pulses, fish, eggs, meat and other proteins	12 per cent	12 per cent and emphasis shifted from meat to beans and pulses
Dairy and alternatives	15 per cent	eight per cent and now includes dairy-free varieties
Food and drink high in fat and/or sugar	A combined category of eight per cent	three per cent
oils and spreads		one per cent

Sources: Public Health England, 2016; FSA, 2012.

aged 65 or over, average red meat intake was 63g per day, but men still exceeded the 70g limit (75g for men and 54g for women). This was just red meat consumption, chicken, turkey and mixed dishes containing them were the most commonly consumed type of meat for all age groups except those aged 65 and over. For older adults, the most commonly consumed meat was bacon and ham – processed meats that the WCRF said should be avoided and the WHO said cause cancer (see Cancer page 26). The government, undoubtedly under pressure from the meat industry, are dragging their feet. Viva!Health shows in this report why public health recommendations for meat intake should be zero.

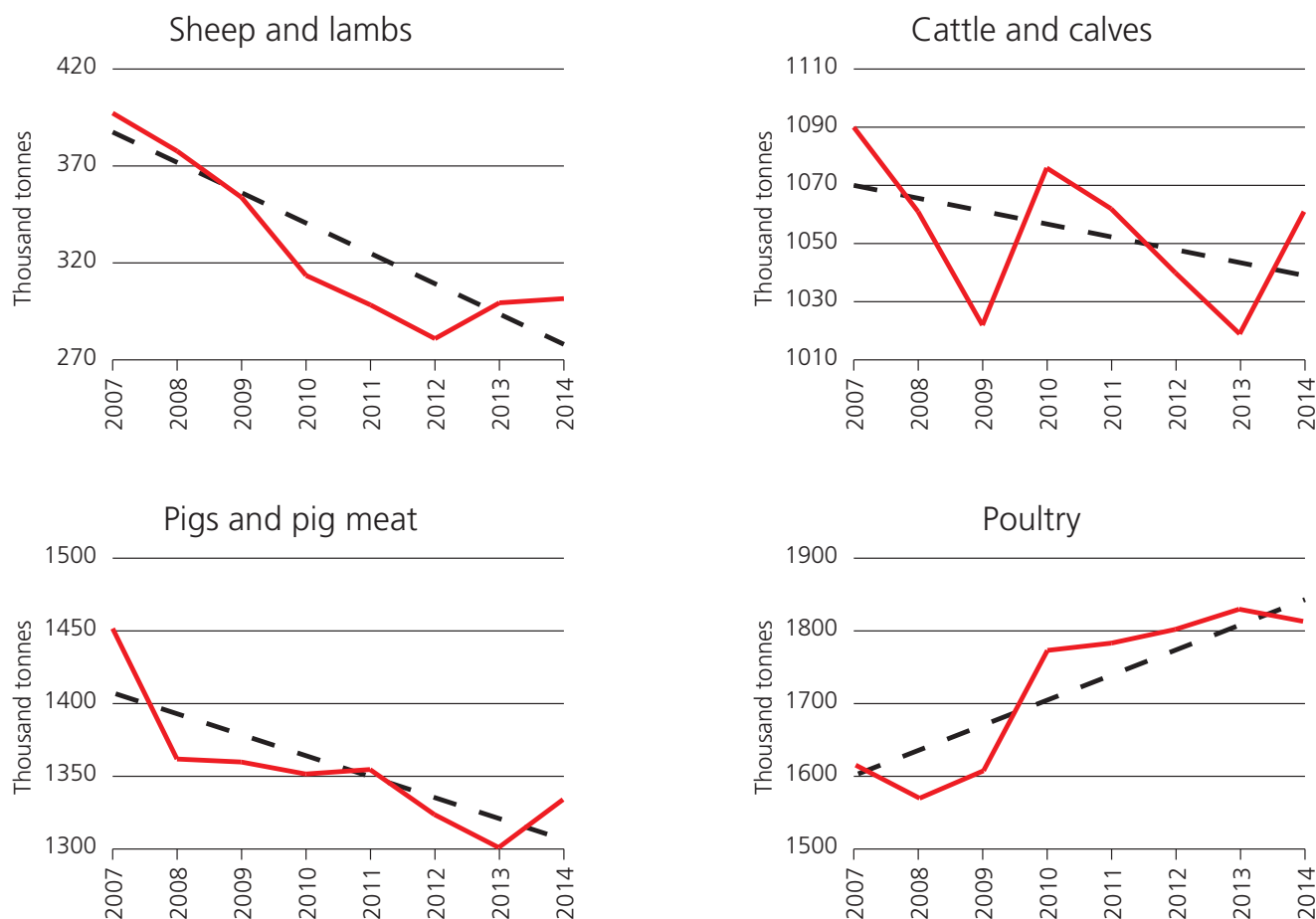
UK MEAT SUPPLY

The Department for Environment, Food and Rural Affairs (Defra) publishes an overview of agriculture in the UK every year called *Agriculture in the United Kingdom*. It contains an extensive range of data including farm incomes, land use, livestock numbers, prices, production

of key commodities (eg meat, milk and vegetables), overseas trade, organic farming and the environment. The information is used widely by government, industry, researchers and other stakeholders to support policy monitoring and development. It is also possible to build up a picture of meat consumption in the UK over time using data from these reports.

Figure 8.0 shows that the consumption of sheep, cattle and pigs in the UK has declined in recent years while the consumption of poultry has increased. The figures are quite erratic for cattle and calves, but the overall trend (shown on the graph as a linear trend line) gives a good indication of what the overall trend in consumption is over time. The drop in meat consumption seen in all four categories in 2008/2009 may reflect the drop in income many experienced during the financial crash of that period. The drop in beef consumption seen in 2013 is probably linked to the horsemeat scandal of that year. However, the overall trend in meat-reduction reflects a growing awareness of the links between meat-eating and

Figure 8.0 Trends in total meat supply (total quantity available after imports and exports are accounted for) in the UK from 2007-2014.



Source: Defra, 2012 and 2015a.

MEAT THE TRUTH

human disease as well as an increasing number of people rejecting the slaughter of animals for food thanks to the campaigning work and undercover exposés undertaken by Viva!

The steady decline in meat consumption in the UK is evident from other sources too. Defra publish a UK household consumption dataset that provides detailed statistics on household purchases per person per week going all the way back to 1974 (Defra, 2015).

Defra's dataset separates carcass meat and non-carcass meat (see Table 14.0). Carcass meat may be bought as a joint or a piece of meat attached to a bone to be fried or roasted, like a steak or a leg of lamb. Pork chops are a smaller item that can be grilled or fried but are also classed as carcass meat. Offal is classed as non-carcass meat, liver for example. All poultry is classed as non-carcass meat, so chicken, turkey, takeaway and

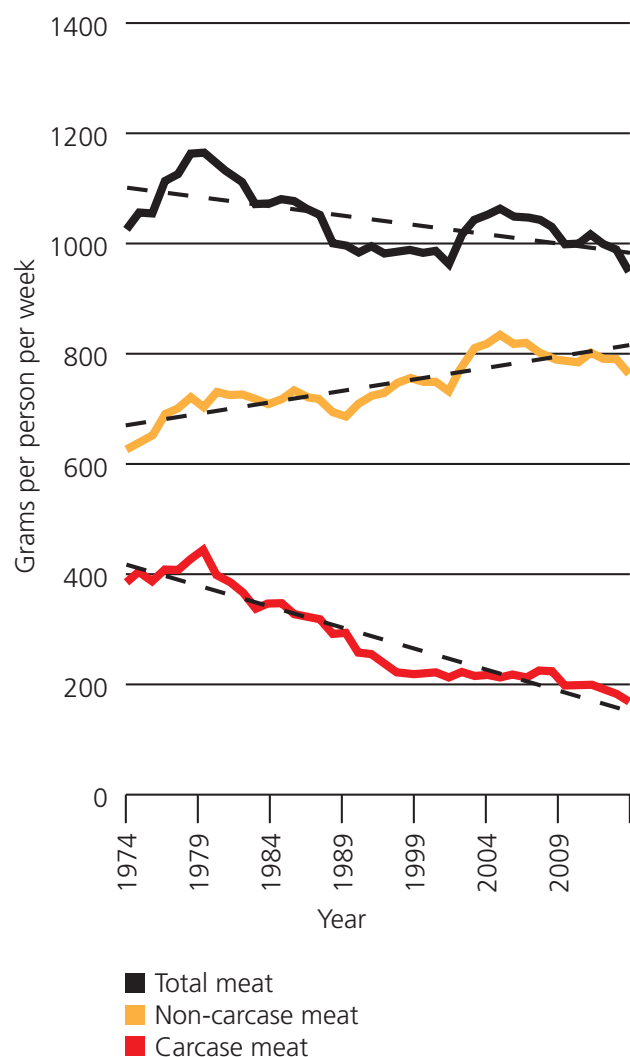
ready meals made with chicken or turkey are included. All sausages, pies and meat-based ready meals are classed as non-carcass meat.

Figure 9.0 shows a steady decline in the quantity of carcass meat purchased for UK households. The trend line suggests that since 1974, the amount of carcass meat purchased, per person per week, has fallen by 265g (the equivalent of a portion of roast beef, a lamb chop and a medium rump steak). On the other hand, the amount of non-carcass meat has increased per person per week by 146g (the equivalent of a quarter-pounder beef burger, one sausage and one rasher of bacon OR six chicken nuggets and a chicken drumstick). **However, taken together, total meat consumption in the UK has dropped by more than 10 per cent (119g per person per week) since 1974 (Defra, 2015).**

Table 14.0 Types of carcass and non-carcass meat as categorised by Defra

Carcass meat	Non-carcass meat	
Beef and veal Beef joints – on the bone Beef joints – boned Beef steak – less expensive Beef steak – more expensive Minced beef All other beef and veal Mutton and lamb Mutton Lamb joints Lamb chops All other lamb Pork Pork joints Pork chops Pork fillets and steaks All other pork	Liver Ox liver Lambs liver Pigs liver All other liver All offal other than liver Bacon and ham, uncooked Bacon and ham joints, uncooked Bacon and ham rashers, uncooked Bacon and ham, cooked Cooked poultry not purchased in cans Chicken and turkey, cooked Takeaway chicken Chicken, uncooked – whole chicken or chicken pieces Other poultry, uncooked (including frozen) Turkey, uncooked Other poultry uncooked Corned beef, canned or sliced Other cooked meat Other canned meat and products Other fresh, chilled and frozen meat	Sausages, uncooked – pork Sausages, uncooked – beef and other Meat pies and sausage rolls, ready to eat Meat pies, ready to eat Sausage rolls, ready to eat Meat pies, pasties and puddings, frozen or not frozen Burgers, frozen or not frozen Ready meals and convenience meat products Complete meat-based ready meals Other convenience meat products Pate and delicatessen type sausage Pâté Delicatessen type sausages Meat pastes and spreads Takeaway meats Takeaway meat pies and pasties Takeaway burger and bun Takeaway kebabs Takeaway sausages and saveloys Takeaway meat based meals Takeaway miscellaneous meats
Defra, 2015.		

Figure 9.0 Quantity of carcase, non-carcase and total meat purchased for UK households from 1974-2013.



Source: Defra, 2015.

THE MEAT-FREE MARKET IS THRIVING

As meat consumption falls, the vegetarian and vegan food market is rapidly expanding. Market researchers Mintel say that the number of new vegetarian and vegan food and drink products doubled between 2009 and 2013 (Mintel, 2015). They say that 12 per cent of global food and drink products launched in 2013 carried a vegetarian claim, up from six per cent in 2009, and two per cent of global food and drink launches carried a vegan claim in 2013, up from one per cent in 2009. This is a substantial portion of the market and veganism is now one of Britain's fastest growing lifestyle movements.

In 2014 Mintel estimated the UK meat-free food market was worth £625 million in 2013, up from £543 million in 2009. Laura Jones, Global Food Science Analyst at Mintel, says: "Globally, the outlook for the meat alternative market is positive and will continue to be driven by an emerging consumer trend towards meat reduction".

Viva!'s campaigns over the last 21 years have played a significant role in changing the way many people eat. Viva! was the first group to film the factory-farming of ducks locked in stinking, overcrowded sheds with no access to water in which to clean or swim. Huge press and TV coverage saw duck slaughter figures shrink from 19 million to 14 million, and they're still falling.

Viva! have filmed in dozens of pig farms and exposed the widespread use of metal farrowing crates for breeding sows (cages so small they can't even turn around) and showing the filthy concrete cells that piglets have to call home. Viva! have also exposed the constant misuse of antibiotics in animals and revealed the now-established link between factory farms and antibiotic-resistance superbugs like MRSA.

Viva!'s undercover investigators have hit the headlines by showing how the UK's leading turkey producers treat their animals; thousands crammed into a shed, dim light, billowing dust, dead and dying birds everywhere. Turkey sales plummeted by four million.

Viva!'s kangaroo campaign got UK supermarkets to empty their shelves of all exotic meats. Oh, and we wiped out the British ostrich industry! Viva! also exposed the cruelty involved in foie gras production, showing how ducks and geese are force fed until their livers almost explode, persuading over 1,000 restaurants and Amazon to stop selling this 'torture in a tin'. Viva! also shamed Gordon Ramsay and made Heston Blumenthal drop it.

Viva!Health keeps abreast of the science and provides a unique major resource on vegan health and nutrition for health professionals and the public. We have shown that chicken meat is not a healthy alternative to red meat, that all fish contains dangerous pollutants and that cow's milk and dairy products are not healthy or natural. This report provides irrefutable evidence that meat is not needed for good health and is linked to a wide range of illnesses and disease including the UK's biggest killers; heart disease, diabetes and cancer.

CONCLUSION

We are told to limit the amount of red and processed meat we eat – chicken is completely ignored. This is not good enough; the government should be encouraging people to replace meat with healthier plant-based protein. The WCRF and WHO have both issued clear warnings about meat and cancer; they have not minced their words! They say processed meats (including processed white meats made from chicken and turkey) **do** cause cancer and red meat probably does too.

There are some signs of change – small beginnings. In 2016, Public Health England's new Eatwell Guide shifted the emphasis from meat as the main source of protein. "*Meat, fish, eggs, beans and other non-dairy sources of protein*" was replaced with: "*Beans, pulses, fish, eggs, meat and other proteins*". The change in wording reflects a small but significant shift in emphasis from meat and dairy to plant foods. A move in keeping with the current research which recognises the harm meat and dairy do to our health and the environment.

Public Health England now says "*Pulses such as beans, peas and lentils are good alternatives to meat because they're lower in fat and higher in fibre and protein, too*". However, they also say: "*Choose lean cuts of*

meat and mince and eat less red and processed meat like bacon, ham and sausages". These guidelines need to be strengthened in light of the WCRF and WHO reports to say "***cut down on all meat and avoid processed meat as it causes cancer***". Otherwise people will continue to buy chicken (thinking it is a healthy meat) along with bacon, ham and sausages.

UK meat consumption is relatively high compared to other countries, but has steadily declined since the 1970s. Consumption of carcass meat (joints or steaks) has fallen while that of non-carcass meat (chicken, turkey, sausages, pies and meat-based ready meals) has increased. However, despite the huge increase in chicken sales, taken together, total meat consumption in the UK has fallen by more than 10 per cent since 1974.

The meat-free market is thriving – worth £625 million in 2013. The number of vegans in the UK has risen by 360 per cent over the last decade with 542,000 now compared to 150,000 in 2006. It's time the government gets on board with what people are working out for themselves – meat is unhealthy, we don't need it and we don't want it.



Meat does not contain any nutrients that you can't find in healthier foods. Most types of meat contain iron, zinc, selenium, phosphorus, choline and B vitamins. All these are widely available in healthier, plant foods. Meat contains little or no carbohydrate and no fibre, which lowers cholesterol and protects against bowel cancer and heart disease.

Meat contains cholesterol and saturated fat, linked to obesity and cardiovascular disease (CVD). The British Dietetic Association says that most people eat 20 per cent more saturated fat than the maximum recommended amount. Most comes from fatty cuts of meat, poultry (particularly chicken skin), sausages and pies as well as dairy products, cakes and biscuits. All reputable health organisations (including the WHO) recommend eating less saturated fat found in meat and dairy foods and more unsaturated fats found in avocados, nuts, seeds, plant-based oils and spreads.

The same is true for protein; most people eat 45-55 per cent more than they need. There is no advantage to eating more protein than you need, in fact it is harmful. If you consume enough calories in a varied vegan diet, it is very easy to achieve the desired level of protein.

Vitamin B12 is often cited as a reason to eat meat. What most people don't realise is that traditionally farmed animals would have got B12 from food eaten from the ground, contaminated with B12-producing bacteria (this is how our ancestors got theirs too in addition to the small amount of meat they ate). Intensive farming methods ensure that sanitised animal feed is free of B12, so animals need supplements too. You could cut out the middleman and take your own. B12 from fortified foods and supplements is also easier to absorb than B12 from meat which is bound to animal protein. In the US, all adults over 50 years (including meat-eaters) are advised to get B12 from fortified foods or supplements because of the high incidence of poor absorption from meat and dairy. Many vegans have a heads-up on B12 as they routinely include a supplement or fortified foods in their diet.

It's a myth that you need meat to get iron, deficiency is no more common among vegetarians and vegans than meat-eaters. Iron deficiency affects large numbers of meat-eaters, so clearly it is not a vegetarian or vegan issue. The NDNS shows that being a meat-eater does not guarantee protection against deficiency. Young women are particularly vulnerable because of iron lost during menstruation. A good level of iron intake can be achieved from a varied vegan diet and combining iron-rich foods

with vitamin C can significantly boost absorption.

Too much iron can be harmful, activating molecules that switch on cancer genes and others linked to atherosclerosis and heart disease. Haem iron, from meat, can contribute to the formation of free radicals and N-nitroso compounds (NOCs) which can damage DNA and lead to cancer. Non-haem iron from plant foods offers all the benefits of iron without these risks. Meat processing (curing with nitrites) can also lead to the formation of NOCs in meat and haem iron in meat encourages NOC formation in the gut. A double whammy!

Cooking meat at high temperatures (pan-frying, grilling and barbecuing) can produce carcinogenic chemicals: polycyclic aromatic hydrocarbons (PAHs) and heterocyclic amines (HCAs). The main sources of PAHs are cooked and smoked meat and fish (notably barbecued meat) and tobacco smoke. BaP is the most abundant PAH in the diet. Chicken is the main source of HCAs in a typical Western diet and fried, roast and grilled chicken can contain particularly high amounts. PhIP is the most abundant HCA and high levels have been found in pan-fried, oven-grilled and barbecued chicken – much higher than in red meats. So much for chicken being the healthy option! Dripping and gravy made from it also contains high levels of HCAs. In parts of Yorkshire, dripping spread on bread is known as a 'mucky fat' sandwich!

Some sportspeople use energy drinks laced with carnitine – a substance found in meat (and some plant foods at lower levels). Carnitine is converted in the body into a harmful compound called TMAO that increases the build-up of cholesterol in the arteries. Vegans may not have the type of gut bacteria that produces TMAO from carnitine. The safety of carnitine supplements has been questioned and it seems likely that carnitine from meat could be just as harmful.

If that's not enough to put you off, consider the arsenic residues found in the breast meat of chickens fed arsenic-containing animal feed additives. Banned in the EU, these additives continue to be used in many other countries.

It's difficult to say which component of meat is the most harmful as there are so many to choose from! Saturated fat, cholesterol, salt, NOCs, HCAs, PAHs, carnitine, arsenic – meat's got the lot!

A large body of evidence links meat to a premature death. Substituting just one serving of red meat a day for a healthier source of protein (pulses, wholegrains,

MEAT THE TRUTH

nuts and seeds) can reduce the risk of early death so imagine what ditching meat altogether can achieve. Vegetarian Adventists have a lower risk of cancer, CVD, diabetes and live longer compared to the general population. Other studies agree that high-meat consumers (especially of processed meat) have an increased risk of early death, this falls in line with the WCRF report which recommends completely avoiding processed meats. That means no bacon, ever.

The EPIC studies in the UK and Europe may not match the substantive evidence seen in US, but taken together they do show the benefits of avoiding meat. The EPIC-Oxford Study failed to find a link between meat-eating and early death but the meat-eaters in this study were more health-conscious than the average UK meat-eater and death rates of all participants were lower than average. An earlier, larger EPIC study in Europe did reveal a link between processed meat and early death. A subsequent EPIC-Oxford Study combined with the Oxford Vegetarian Study also found a lower rate of early deaths before the age of 75 among vegetarians and vegans compared to meat-eaters when participants known to have changed diet group were excluded. It may be that when people fell ill, they switched group joining the healthier vegans!

Another reason the UK studies are not quite as strong as the US ones may be that UK vegetarians and vegans are motivated by factors other than health (animal welfare and/or the environment), making them less likely follow such a healthy diet as the Adventist vegetarians in the US, whose motivation is health. Furthermore, the amount of animal protein (dairy products and eggs) in the UK vegetarian diet was significantly higher than that in the vegetarian Adventists' diet. A diet filled with eggs and cheese may offer little advantage to one packed with meat. A healthy vegan diet is made up of plenty of fruit and vegetables, wholegrains, pulses, nuts and seeds and no animal fat or animal protein.

The scientific consensus is that meat increases the risk of a premature death. Researchers from the Oxford Martin Programme on the Future of Food suggest that, on a vegan diet, more than eight million premature deaths could be avoided by 2050. Replacing meat with a diet rich in fruits, vegetables, wholegrains, pulses, nuts and seeds remains a sound, evidence-based recommendation for improving the quality of life and avoiding an early death.

Then there are the well-documented and irrefutable

links between meat and cancer. In the UK, one in every two people born after 1960 will be diagnosed with cancer at some point in their lives. After smoking, poor diet is the most important avoidable cause of cancer. Meat has been recognised as a risk factor for cancer since the early 1900s. Countries with high intakes have higher rates of bowel, breast and prostate cancer, three of the most common cancers. The NIH-AARP Diet and Health Study found that people who ate the most red meat had a higher risk for oesophageal, bowel, liver and lung cancer and those who ate the most processed meat had a higher risk for bowel and lung cancer.

It's difficult to know which component of meat is responsible for the links with cancer as there are so many candidates: saturated fat, animal protein, haem iron, salt, NOCs, HCAs and PAHs... it's an extensive list. A diet high in saturated fat, leading to obesity, increases the risk of breast, prostate and bowel cancer as well as heart disease and diabetes. Animal protein increases IGF-1 levels, which increase the risk of bowel and lung cancer. Iron overload may increase cancer risk by generating free radicals and inducing oxidative stress. NOCs from nitrite-preserved meats and bacterial production in the gut, bind to DNA and cause mutations that can lead to cancer. HCAs and PAHs produced by cooking meat at high temperatures are carcinogenic. Avoiding meat completely is an effective way to reduce the risk of cancer.

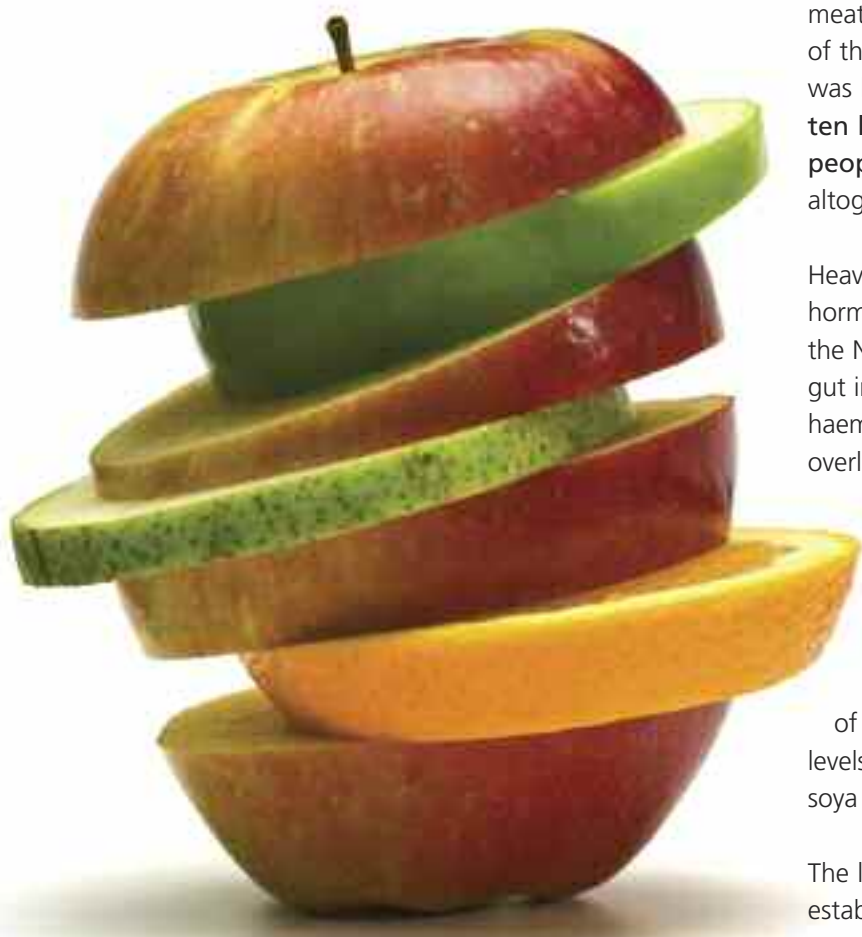
A study from the University of Oxford, found that compared with meat-eaters, cancer incidence was lower in fish-eaters and vegetarians but lower still in vegans. Results of AHS-2 were similar, with total cancer risk significantly lower in vegetarians and vegans than in meat-eaters. This adds to a large body of evidence, not least the substantive WCRF and WHO reports that state clearly that meat consumption is linked to cancer. Current guidelines need to be amended to reflect the indisputable link between meat and cancer. Cigarettes carry a government health warning, why shouldn't bacon?

Links between diet and breast cancer have been suspected for a long time. The wide variation in breast cancer rates around the world and migration studies show that genes are a minor cause, responsible for just 5-10 per cent of breast cancers. Research shows that diets rich in meat and dairy are linked to a higher risk of breast cancer compared to plant-based diets. The Nurses' Health Study II found that each daily serving of meat increased the risk substantially – especially in women using oral contraceptives. Given the high incidence rate for breast cancer (one in eight women in

the UK), they say that the consumption of meat should be regarded as a public health concern.

Girls exposed to radiation from the atomic bombs at Hiroshima and Nagasaki were more likely to get breast cancer later in life than those not exposed, but women over 40 who were exposed did not have an increased risk. This inspired researchers to investigate the adolescent diets of women in the Nurses' Health Study II. Results showed that those who ate a lot of meat when they were young had a much higher risk of premenopausal breast cancer.

Several different substances in meat may be responsible for the link with breast cancer including: HCAs and PAHs created during cooking, animal fat, haem iron and hormone residues. Fruit, vegetables and fibre are all associated with a lower risk. Dietary advice given at mammography screenings would be an effective way of helping women lower their risk of breast cancer. Such advice should include how a low-fat, high-fibre, meat-free diet consisting mainly of fruits, vegetables, wholegrains and pulses can result in a major reduction in the risk of breast cancer.



The global distribution of prostate cancer is similar to that of breast cancer; countries with high levels of one tend to have high levels of the other. As the Western diet takes over more traditional diets in developing countries, the number of men with prostate cancer increases and high consumption of meat (particularly red, processed and well-done meat), is associated with the increased risk. It is suggested that PhiP and other HCAs are responsible. Fried, roast and grilled chicken can contain particularly high amounts of HCAs.

The Prostate Cancer Lifestyle Trial found that patients with early-stage prostate cancer were able to avoid or delay conventional treatment for at least two years by following a vegan diet. A small pilot study suggested a vegan diet may help combat the disease by increasing the length of the telomeres, stretches of DNA that protect the ends of our chromosomes, like the plastic caps that prevent shoelaces from unravelling. Despite the evidence, advice from the NHS on the links between diet and prostate cancer remains sparse.

High intakes of meat and haem iron are also linked to lung cancer, one of the most common and serious types of cancer. In 2007, red and processed meats were classified by the WCRF/AICR as possible causes of lung cancer. The NIH-AARP Diet and Health Study found red meat was linked to lung cancer too as well as cancers of the bowel, liver and oesophagus and processed meat was linked to lung and bowel cancer. They said **one in ten lung and bowel cancers could be avoided if people reduced their meat intake**. Avoiding meat altogether would be even more effective.

Heavy metals and other chemicals (including synthetic hormones) in organ meat may be responsible. It may be the NOCs found in preserved meat and produced in the gut in response to high haem iron intake. It may be the haem iron, which can increase cancer progression. Iron overload can switch on cancer genes, trigger

inflammatory responses and iron-induced hypoxia signalling – a classical feature of cancer.

It could be HCAs and PAHs which are which are potent lung carcinogens. High intakes of animal protein drive up IGF-1 levels in the body and people with lung cancer tend to have higher levels of this growth hormone. Vegans tend to have lower levels of IGF-1 and high intakes of vegetables, fruit and soy reduce the risk of lung cancer.

The link between meat and bowel cancer is well-established, it is one of the best-known diseases associated with meat. A number of substances in meat

MEAT THE TRUTH

are thought to be responsible. It may be the animal fat or the animal protein as both can promote cancer. It could be the carcinogenic NOCs formed in food and/or in the gut, or HCAs and PAHs formed in meat cooked at high temperature or the haem iron in red meat which can promote carcinogenesis through oxidation and DNA damage. Take your pick!

People who eat 400g or more of meat a day might be exposed to as many NOCs as a smoker! So meat-eaters may be in need of the same level of health advice as smokers. Which is what then Shadow Secretary of State for Environment, Food and Rural Affairs, Kerry McCarthy suggested in 2015 in an interview for *Viva!life* magazine. The research supports her suggestion; better dietary advice could save lives. Bowel cancer is the leading cause of cancer death among non-smokers in affluent countries and its prevention should be a major goal for public health. Given what we now know about the harmful effects of meat and bowel cancer, isn't it about time the government amended health guidelines to properly reflect the risks associated with meat?

Studies show that mutagens in cooked meat are associated with renal cell carcinoma, the most common form of kidney cancer. A developing theory is that HCAs activate enzymes in such a way that they behave differently and cause mutations in DNA that can lead to cancer. Meat intake is linked to pancreatic cancer too, the fourth most common cause of cancer death worldwide. Studies show that meat, particularly meat cooked at high temperatures, plays a significant role in this disease. Both animal fat and haem iron are

suspected to play a part in the links between meat and pancreatic cancer too. British vegetarians and vegans have a substantially lower risk of this disease than meat-eaters. The huge EPIC study found strong links with chicken and suggested that antibiotics and/or drugs called coccidiostats given to poultry and cattle to prevent the growth of parasites may be involved. They also suggested that animal oncogenic viruses may cause cancer if meat is not cooked enough. So you are damned if you cook it, and damned if you don't!

The links between processed meat and stomach cancer, the fifth most common cancer worldwide, have been known about for over a decade. In 2015, the WHO reported links between processed meat and stomach cancer. Then in 2016 the WCRF said that there is strong evidence that consuming processed meat increases the risk of this disease. They also said that grilled and barbecued meat was linked to it and eating little or no fruit also increased the risk. High levels of salt, nitrite, nitrate and NOCs in processed meats have been blamed as well as carcinogenic and mutagenic PAHs in smoked meat.

CVD is one of the biggest killers responsible for early death. Researchers from Harvard School of Public Health found a significant link between processed meat and CVD. The Nurses' Health Study reported similar links between red meat and heart disease; replacing one serving of meat with nuts lowered the risk. A later study from the same group combined with the US Health Professionals Follow-up Study found replacing meat with nuts also lowered the risk of stroke. A later study from both cohorts again confirmed that both red and processed



meat increased the risk of CVD and substituting meat with healthier sources of protein lowered it.

In Europe, the large EPIC study found a link between processed meat and CVD. They suggested the chemicals used in processed meats may be responsible for damaging blood vessels. Furthermore, processed meats contain even more saturated fat than red meat and this type of unhealthy fat increases the risk of CVD. Haem iron may be involved too as high iron stores are a risk factor and research shows that women who menstruate and people who donate blood regularly have a lower risk of CVD. It has been suggested we should end iron-fortification of flour and encourage more blood donations to reduce iron stores and lower the risk of CVD. You could just avoid meat and opt for non-haem iron from plant foods as absorption is regulated and you only take up as much as you need. In a nutshell; ditching meat lowers your risk of suffering a heart attack or stroke.

One in every four adults in the UK is obese. People who eat a lot of meat are more likely to be overweight or obese because of the high fat content of meat. Many

people choose chicken thinking that it is a healthier option to red meat. Chicken accounts for nearly half of all the meat bought in the UK with around 2.2 million chickens being eaten every single day! However, selective breeding and intensive farming ensures that chicken is not a healthy option with the average supermarket chicken containing more fat than protein.

An interesting finding from the EPIC-PANACEA study was that a diet rich in meat caused more weight gain than a low-meat diet containing the same number of calories. The strongest links were seen for chicken and processed meat. A pro-meat group (including a speaker from National Cattlemen's Beef Association) suggested the additional weight could be increased muscle mass but this was ruled out after belly fat was measured. They found that animal protein-rich diets were linked to weight gain especially when they missed out fibre from carbohydrates. More research is needed but the fact remains that meat makes people gain more weight than plant-based diets even when they contain the same number of calories. You are better off replacing chicken with chickpeas!



MEAT THE TRUTH

The highest levels of osteoporosis are seen in Europe and the US (particularly among white people in the US) and the lowest rates in South America, Africa and Asia. Diet and lifestyle are clearly involved as black Americans have a lower risk than white Americans, but a higher risk than black Africans. The same scenario is seen in Japanese people in Hawaii compared to those in Japan and Chinese people in Singapore compared with mainland China. The more affluent the diet (rich in meat and dairy), the higher the risk of osteoporosis and fracture is.

The acid-alkaline hypothesis suggests that as food is digested, acids are released into the blood and the body tries to neutralise it by drawing calcium from the bones if there isn't enough calcium readily available. Meat and dairy are particularly bad as they contain more acid-promoting amino acids (the building blocks of protein). So the more meat and dairy you eat, the more acidic the blood, the more calcium is potentially lost from the bone – that's the theory.

The most osteoporosis occurs in the countries that consume the most animal protein. If you want to lower your risk of osteoporosis, ditching all animal protein is a good place to start. Weight-bearing exercise (stair-climbing, walking or dancing) is the best option for improving your bone health while ensuring you get enough plant-based calcium and vitamin D.

The numbers of people with type 1 and type 2 diabetes is rising globally at such a rate that in the UK alone, it is estimated that by 2035, the NHS may be spending nearly a fifth of its entire budget on treating diabetes. A large body of evidence shows how proteins found in cow's milk can trigger type 1 diabetes in some people but more recent research also suggests that children of mothers who eat meat (especially processed meat) while breast-feeding have a higher risk of developing type 1 diabetes later on in childhood.

Meat is also a major risk factor for type 2 diabetes not least because people who eat meat tend to weigh more and people who weigh more have a higher risk. People who eat a lot of fat end up having more sugar in the blood than people who eat lots of carbohydrate. This is because a high-fat, meat-rich diet leads to the build-up of fat globules inside the cells which block insulin activity causing high blood glucose levels, thus increasing the risk of type 2 diabetes. In addition to the harmful effects of fat and cholesterol, haem iron may also increase the risk. The nitrates and nitrites in processed meats are also implicated along with HCAs, PAHs and AGEs. Given the strength of this research, public health guidance should prioritise reducing all meat consumption (red, processed and poultry) to reduce not only diabetes but the secondary diseases it can lead to too.

Meat and dairy foods are also linked to fertility problems; men who eat the most meat and full-fat dairy products tend to have fewer and slower sperm than those eating the most fruit and vegetables. This



may be because of the vitamins, folic acid and fibre and the fewer proteins and fats in the healthier Mediterranean-style diet. Replacing animal protein with plant protein can reduce infertility risk in women too. Couples trying to conceive should be advised about the important effects of diet for men and women.

It is estimated that one in five people in England suffer food poisoning every year. Animal foods, particularly meat and meat products, are responsible for most cases. If plant foods cause poisoning, it tends to be because they are contaminated with animal excreta, human sewerage or were handled with dirty hands. In the UK, *Campylobacter* is the most common foodborne pathogen (while *Salmonella* is responsible for the most hospital admissions). Chicken is the most common food associated with food poisoning in the UK and the FSA recently launched a campaign advising people not to wash raw chicken as the splashes could contaminate clothes, skin and the entire kitchen!

Cheap meat comes at a cost; the expansion of large-scale factory-farms has led to many problems including the safe disposal of millions of tons of manure, making many streams and rivers too polluted for swimming, drinking or maintaining healthy wildlife. Mechanical evisceration (removal of internal organs) of slaughtered animals is now done so rapidly that meat is frequently contaminated with faecal residues from the guts which is especially a concern with poultry as people eat the skin. It can also cause problems when meat is minced for burgers for example, then eaten rare because the bacteria on the outside becomes incorporated throughout the meat. One study found seven out of

ten pork samples and nine out of ten chicken samples were contaminated with an antibiotic resistant bug found in the animals' gut. This is probably why over 70 per cent of fresh shop-bought chickens in the UK tested positive for *Campylobacter* in 2015. More recently the number testing positive dropped to 50 per cent but it was later revealed that some producers were removing the neck skin before testing – neck skin is the most contaminated part of the bird. The FSA abandoned the project and have said they will start a new one with the first results due in 2017.

Most cases of *E. coli* food poisoning are caused by undercooked beef (particularly mince, burgers and meatballs) as well unpasteurised milk. *Salmonella* bacteria are often found in raw or undercooked meat (especially chicken, pork and beef), as well as dairy products and eggs. Chickens, pigs and cows may be infected with *Salmonella* even though they show no symptoms. Going vegan is no guarantee that you will avoid food poisoning, but it certainly lowers the risk substantially.

The routine use of antibiotics in farmed animals has led to the rapid increase in antibiotic-resistant bugs or superbugs which can be passed on to people from undercooked meat. Superbugs are increasing at an alarming rate. Research shows that certain strains of MRSA were established in UK pig farms and third-generation cephalosporin-resistant *E. coli* originate directly from the overuse of antibiotics in broiler chicken farms. Despite an EU ban on growth-promoting antibiotics in animal feed, huge quantities of antibiotics continue to be given for 'disease prevention'. In the UK, nearly half of all antibiotics are given to farmed animals. For more information see Viva!'s report: *Pig Farming, The Inside Story*: www.viva.org.uk/pigreport.

Bacteria resistant to the antibiotic colistin (our last defence against multi-resistant bacteria) have recently emerged. A new development is that these resistance genes can be passed from one strain to another, illustrating how antibiotic use in animals is creating a major human health risk. The need to restrict and even ban the use of certain antibiotics in animals has never been so urgent. We are charging headlong towards a 'post-antibiotic era' where bacterial infections in people may no longer respond to the antibiotics we have been relying on for years. So, if you undercook meat, you could expose yourself to bacteria, which may be antibiotic-resistant, but if you overcook it, you could be at risk from carcinogenic compounds. The dilemma is a no-brainer! For more information see Viva!'s film *Swine*: www.viva.org.uk/swine.



MEAT THE TRUTH

Viruses can cause gastroenteritis (or stomach flu). The 'winter vomiting bug' norovirus is a common cause, affecting up to one million in the UK every year. Outbreaks often occur in hospitals, schools and cruise ships, where infection spreads rapidly. Bivalve shellfish (oysters, mussels and clams) are often the cause. Other foodborne viruses (such as hepatitis E) have been found in meat from pigs, wild boar and deer. Foodborne transmission of hepatitis E is relatively rare but the virus can be passed on in undercooked meat such as: pork pies, liver pâté, wild boar and under-cooked or raw pork and sausages. People with liver disease, immuno-compromised people and pregnant women should be discouraged from eating these types of meats in particular.

Avian influenza (or bird flu), came to international attention in the late 1990s when it spread through live-poultry markets in Hong Kong, infected people and caused six deaths. There are different strains of varying severity; the H5N1 strain has killed 450 people to date while the H7N9 has killed 229. Avian flu viruses aren't usually transmitted from one person to another but human-to-human infection is possible and there have been a number of cases among families caring for infected relatives. The highly-pathogenic H5N1 causes death in 60 per cent of the people it infects. The 1918 flu pandemic, a deadly pandemic that infected 500 million people across the world, caused death in just two per cent of those infected. Imagine what harm bird flu would do if it became easily passed on from person to person!

Bird flu represents a disturbing new evolutionary development in the behaviour of the avian flu virus. It's a disaster of our own making, spreading from aquatic birds (where it has coexisted quite happily for thousands of years) into live-poultry markets and on to factory farms which provide the perfect environment for a mutating virus. The poultry industry has responded to the crisis predictably by playing down the human risk. One way to take control would be for large numbers of people to stop eating poultry, pigs and other animals and remove the viral reservoir of factory-farms.

The reason beef sales plummeted in the 1990s was the BSE crisis when despite agricultural minister John Gummer's reassurances that British beef was perfectly safe, a number of people became ill and died from Creutzfeldt-Jakob disease (CJD), the human form of BSE. An inquiry revealed that BSE was caused by cows being fed the remains of other cows in the form of meat and bone meal. As herbivores, this is obviously a very unnatural practice for them and has since been banned.

CJD has killed close to 200 people in the UK. The most recent was in 2016, when a 37-year-old man was diagnosed two weeks before he died. People infected with CJD may carry the disease for up to 50 years before symptoms develop and it has been suggested that we are not yet through the worst of this sinister disease.

Another dip in red meat consumption occurred in 2013 when it was revealed that various meat product manufacturers were selling mislabelled food that was actually horsemeat. The scandal revealed a major breakdown in the traceability of the food supply chain and showed the potential for harmful ingredients to be included as well. The scandal spread to other countries revealing widespread mislabelling of meat products and insufficient labelling information for sausages, pâté and pies. The discovery of a Spanish warehouse filled with 15 tons of dead stray dogs added further to the scandal. Viva! found out that limited testing for dog and cat meat was done in London but the FSA said the results were negative. The fear is that meat from euthanised dogs, cats, horses or other sick animals may have found its way into pet food, farmed animal feed or human food. If this happens, residues of antibiotics and other drugs could end up in some meat products. People also do have the right to know what they are eating!

We are not designed to eat meat. If you look at a carnivore's teeth and jaws, how highly acidic their stomachs are and how short their colons are, you can see we share more characteristics with herbivores. When did you last see an enthusiastic meat-eater snatch up a live rabbit and tear through the fur and into the flesh, crunching its raw bones with their bare teeth? Humans tend not to eat meat unless it is packaged, cooked and often flavoured with spices, herbs and seasoning. Mandy Pella's photo of a piece of bacon with a nipple on it went viral after she posted it on Facebook with the caption: *"I was going to make BLTs for dinner until I realised my bacon still had a nipple on it"*. The widespread horror shows how most meat-eaters are uncomfortable being reminded that meat is part of a dead animal.

We are not the same as our Palaeolithic 'hunter-gatherer' ancestors (but even then, plant foods were the staples); humans continued evolving into the more recent Neolithic era 10,000 years ago. Modern adaptations include increased production of an enzyme that helps us digest carbohydrates (bread, rice and other wholegrains). In fact, research shows that early farmers relied much more heavily on plant protein than previously thought.

The theory behind the Paleo Diet is wrong. Then there is the outdated notion that meat made us smart – the ‘expensive tissue hypothesis’. A higher quality diet, coupled to the energy saved by walking upright, growing more slowly and reproducing later, fuelled the growth in human brain size, not eating meat.

We simply can’t afford to keep eating meat. A meat-eaters’ diet is responsible for almost twice the greenhouse gas emissions as vegetarians’ and going vegan could cut your emissions more than seven-fold. Meaty diets require more land, water, energy, fertiliser and pesticides than vegan diets – they use far more resources. The 2006 Stern Report warned that if we ignore global warming, the global economy could face devastation on the scale of the Great Depression or the 20th century’s world wars. The UN report, *Livestock’s Long Shadow* says livestock farming is responsible for more greenhouse gas emissions than all the world’s transport (cars, buses, trucks, trains, ships and planes) put together! Changing the way we eat could have a phenomenal effect on the environment, but not changing it could be even more dramatic! The diet that is good for the planet is also good for us. The EPIC-NL study found that replacing meat with nuts, seeds, pasta, rice or couscous was associated with a lower risk of early death and a reduced environmental burden. If you care about the environment then it is essential that you adopt a green diet – a vegan diet.

There is no nutritional benefit from meat that can’t be found in a varied vegan diet; you are much better off getting healthier fats, plant protein, fibre, vitamins and minerals from a range of plant foods. The health benefits of avoiding meat are indisputable which is why, slowly but surely, the world’s most reputable health bodies are beginning to recommend change. The meat industry has been able to influence official dietary guidelines for decades. Just look at how the US Department of Agriculture rejected the advice of their own expert panel by not including considerations of environmental sustainability in the most recent edition of the *Dietary Guidelines for Americans*. Unfortunately the pro-meat crusade will continue while the meat industry has money and influence. However, it seems the UK government and other health bodies are beginning to show some small signs of acknowledging the harm meat does. The evidence presented in this report should help promote a more significant change in public health advice such that people are advised to stop eating meat altogether and go vegan. If the government aren’t brave enough to do it then hopefully people will make the change for themselves anyway.



REFERENCES

- Abid Z, Cross AJ and Sinha R. 2014. Meat, dairy, and cancer. *American Journal of Clinical Nutrition*. 100 Suppl 1:386S-93S.
- Adams PC, Kertesz AE and Valberg LS. 1991. Clinical presentation of hemochromatosis: a changing scene. *American Journal of Medicine*. 90, 44S-9.
- Ahmad AS, Ormiston-Smith N and Sasieni PD. 2015. Trends in the lifetime risk of developing cancer in Great Britain: comparison of risk for those born from 1930 to 1960. *British Journal of Cancer*. 112 (5) 943-947.
- Aiello LC and Wheeler P. 1995. The Expensive-Tissue Hypothesis: The Brain and the Digestive System in Human and Primate Evolution. *Current Anthropology*. 36 (2) 199-221.
- Alexy U, Remer T, Manz F, Neu CM and Schoenau E. 2005. Long-term protein intake and dietary potential renal acid load are associated with bone modeling and remodeling at the proximal radius in healthy children. *American Journal of Clinical Nutrition*. 82 (5) 1107-1114.
- Allen NE, Appleby PN, Davey GK and Key TJ. 2000. Hormones and diet: low insulin-like growth factor-I but normal bioavailable androgens in vegan men. *British Journal of Cancer*. 83 (1) 95-97.
- Allen NE, Appleby PN, Davey GK, Kaaks R, Rinaldi S and Key TJ. 2002. The associations of diet with serum insulin-like growth factor I and its main binding proteins in 292 women meat-eaters, vegetarians, and vegans. *Cancer Epidemiology and Biomarkers Prevention*. 11 (11) 1441-1448.
- Allen NE, Key TJ, Appleby PN, Travis RC, Roddam AW, Tjønneland A, Johnsen NF, Overvad K, Linseisen J, Rohrmann S, Boeing H, Pischon T, Bueno-de-Mesquita HB, Kiemeny L, Tagliabue G, Palli D, Vineis P, Tumino R, Trichopoulos A, Kassapa C, Trichopoulos D, Ardanaz E, Larrañaga N, Tormo MJ, González CA, Quirós JR, Sánchez MJ, Bingham S, Khaw KT, Manjer J, Berglund G, Stattin P, Hallmans G, Slimani N, Ferrari P, Rinaldi S, Riboli E. 2008. Animal foods, protein, calcium and prostate cancer risk: the European Prospective Investigation into Cancer and Nutrition. *Br J Cancer*. 98 (9) 1574-1581.
- Anderson ER and Shah YM. 2013. Iron homeostasis in the liver. *Comprehensive Physiology*. 3 (1) 315-330.
- Appleby PN, Thorogood M, Mann JI and Key TJ. 1999. The Oxford Vegetarian Study: an overview. *American Journal of Clinical Nutrition*. 70 (3 Suppl) 525S-531S.
- Appleby PN, Crowe FL, Bradbury KE, Travis RC and Key TJ. 2016. Mortality in vegetarians and comparable non-vegetarians in the United Kingdom. *American Journal of Clinical Nutrition*. 103 (1) 218-230.
- Apostolou P, Fostira F. 2013. Hereditary breast cancer: the era of new susceptibility genes. *Biomed Res Int*. 747318.
- Astrup A, Meinert Larsen T and Harper A. 2004. Atkins and other low carbohydrate diets: hoax or an effective tool for weight loss? *Lancet*. 364, 897-899.
- Astrup A, Clifton P, Layman DK, Mattes RD and Westerterp-Plantenga MS. 2010. Meat intake's influence on body fatness cannot be assessed without measurement of body fat. *American Journal of Clinical Nutrition*. 92 (5) 1274-1275.
- Atkinson MA, Eisenbarth GS and Michels AW. 2014. Type 1 diabetes. *Lancet*. 383 (9911) 69-82.
- Bahadoran Z, Mirmiran P, Azizi F. 2015. Fast Food Pattern and Cardiometabolic Disorders: A Review of Current Studies. *Health Promot Perspect. eCollection*. 5 (4) 231-240.
- Bao W, Rong Y, Rong S and Liu L. 2012. Dietary iron intake, body iron stores, and the risk of type 2 diabetes: a systematic review and meta-analysis. *BMC Medicine*. 10, 119.
- Barnard RJ, Gonzalez JH, Liva ME and Ngo TH. 2006. Effects of a low-fat, high-fiber diet and exercise program on breast cancer risk factors in vivo and tumor cell growth and apoptosis in vitro. *Nutrients and Cancer*. 55 (1) 28-34.
- Barnard ND. 2007. *Dr. Neal Barnard's program for reversing diabetes: the scientifically proven system for reversing diabetes without drugs*. New York, NY, USA: Rodale Inc.
- Barzel US and Jowsey J. 1969. The effects of chronic acid and alkali administration on bone turnover. *Clinical Science*. 36, 517-524.
- Bastide NM, Pierre FH and Corpet DE. 2011. Heme iron from meat and risk of colorectal cancer: a meta-analysis and a review of the mechanisms involved. *Cancer Prevention Research (Phila)*. 4 (2) 177-184.
- Bate A and Baker C. 2015. *Cancer Statistics in Detail – House of Commons Briefing papers SN02677* [online] Available from: <http://researchbriefings.files.parliament.uk/documents/SN02677/SN02677.pdf> [Accessed 11 May 2016].
- Bates B, Lennox A, Prentice A, Bates C, Page P, Nicholson S and Swan G. 2014. *National Diet and Nutrition Survey Results from Years 1, 2, 3 and 4 (combined) of the Rolling Programme (2008/2009 – 2011/2012). A survey carried out on behalf of Public Health England and the Food Standards Agency* [online]. Available from: www.gov.uk/government/uploads/system/uploads/attachment_data/file/310995/NDNS_Y1_to_4_UK_report.pdf [Accessed 12 January 2016].
- Beck KL, Conlon CA, Kruger R and Coad J. 2014. Dietary determinants of and possible solutions to iron deficiency for young women living in industrialized countries: a review. *Nutrients*. 6 (9) 3747-7376.
- Berkow SE, Barnard ND, Saxe GA and Ankerberg-Nobis T. 2007. Diet and Survival After Prostate Cancer Diagnosis. *Nutrition Reviews*. 65 (9) 391-403.
- Bernstein AM, Sun Q, Hu FB, Stampfer MJ, Manson JE and Willett WC. 2010. Major dietary protein sources and risk of coronary heart disease in women. *Circulation*. 122 (9) 876-883.
- Bernstein AM, Pan A, Rexrode KM, Stampfer M, Hu FB, Mozaffarian D and Willett WC. 2012. Dietary protein sources and the risk of stroke in men and women. *Stroke*. 43 (3) 637-644.
- Biesbroek S, Bueno-de-Mesquita HB, Peeters PH, Verschuren WM, van der Schouw YT, Kramer GF, Tysler M and Temme EH. 2014. Reducing our environmental footprint and improving our health: greenhouse gas emission and land use of usual diet and mortality in EPIC-NL: a prospective cohort study. *Environmental Health*. 13 (1) 27.
- Bingham SA, Hughes R and Cross AJ. 2002. Effect of white versus red meat on endogenous N-nitrosation in the human colon and further evidence of a dose response. *Journal of Nutrition*. 132 (11 Suppl) 3522S-3525S.
- Bishop KS, Erdreich S, Karunasinghe N, Han DY, Zhu S, Jesuthasan A and Ferguson LR. 2015. An investigation into the association between DNA damage and dietary fatty acid in men with prostate cancer. *Nutrients*. 7 (1) 405-422.
- Bodkin H. 2016. Scientists at war over controversial 'eat fat to get thin' obesity report. *Telegraph*. 23 May. [online]. Available from: www.telegraph.co.uk/news/2016/05/23/scientists-at-war-over-controversial-eat-fat-to-get-thin-obesity [Accessed 28 June 2016].

- Bodkin H. 2016a. [online]. High cholesterol 'does not cause heart disease' new research finds, so treating with statins a 'waste of time' Available from: www.telegraph.co.uk/science/2016/06/12/high-cholesterol-does-not-cause-heart-disease-new-research-finds/ [Accessed 28 June 2016].
- Bogaard A, Fraser R, Heaton TH, Wallace M, Vaiglova P, Charles M, Jones G, Evershed RP, Styring AK, Andersen NH, Arbogast RM, Bartosiewicz L, Gardeisen A, Kanstrup M, Maier U, Marinova E, Ninov L, Schäfer M and Stephan E. 2013. Crop manuring and intensive land management by Europe's first farmers. *Proceedings of the National Academy of Sciences*. 110 (31) 12589-12594.
- Bonjour JP. 2005. Dietary protein: an essential nutrient for bone health. *Journal of the American College of Nutrition*. 24, 526S-536S.
- Bonsmann (genannt) SS, Walczyk T, Renggli S and Hurrell RF. 2008. Oxalic acid does not influence non-haem iron absorption in humans: a comparison of kale and spinach meals. *European Journal of Clinical Nutrition*. 62 (3) 336-341.
- Bor MV, von Castel-Roberts KM, Kauwell GP, Stabler SP, Allen RH, Maneval DR, Bailey LB and Nexø E. 2010. Daily intake of 4 to 7 microg dietary vitamin B-12 is associated with steady concentrations of vitamin B-12-related biomarkers in a healthy young population. *American Journal of Clinical Nutrition*. 91 (3) 571-577.
- Bouvard V, Loomis D, Guyton KZ, Grosse Y, Ghissassi F El, Benbrahim-Tallaa L, Guha N, Mattock H and Straif K on behalf of the International Agency for Research on Cancer Monograph Working Group. 2015. Carcinogenicity of consumption of red and processed meat. *The Lancet Oncology* [online]. Available from: [www.thelancet.com/pdfs/journals/lanonc/PIIS1470-2045\(15\)00444-1.pdf](http://www.thelancet.com/pdfs/journals/lanonc/PIIS1470-2045(15)00444-1.pdf)
- Braga DP, Halpern G, Figueira Rde C, Setti AS, Iaconelli A Jr and Borges E Jr. 2012. Food intake and social habits in male patients and its relationship to intracytoplasmic sperm injection outcomes. *Fertility and Sterility*. 97, 53-59.
- Breslau NA, Brinkley L, Hill KD and Pak CY. 1988. Relationship of animal protein-rich diet to kidney stone formation and calcium metabolism. *The Journal of Clinical Endocrinology and Metabolism*. 66 (1) 140-146.
- British Dietetic Association. 2014. *Food Fact Sheet; Cholesterol* [online]. Available from: www.bda.uk.com/foodfacts/cholesterol.pdf [Accessed 10 November 2015].
- British Heart Foundation. 2015. *Study questions guidance on fat consumption in diets* [online]. Available from: www.bhf.org.uk/news-from-the-bhf/news-archive/2015/february/study-questions-guidance-on-fat-consumption-in-diets [Accessed 15 February 2016].
- Brown M, Byatt T, Marsh T, McPherson K. 2010. A prediction of Obesity Trends for Adults and their associated diseases [online]. Available from: www.foresightfordevelopment.org/sobipro/55/1214-a-prediction-of-obesity-trends-for-adults-and-their-associated-diseases [Accessed 8 November 2016].
- Burke W, Cogswell ME, McDonnell SM and Franks A. 2000. *Public Health Strategies to Prevent the Complications of Hemochromatosis*. In: Khoury MJ, Burke W and Thomson EJ eds. *Genetics and Public Health in the 21st Century: using genetic information to improve health and prevent disease*. London: Oxford University Press.
- Butler TL, Fraser GE, Beeson WL, Knutsen SF, Herring RP, Chan J, Sabaté J, Montgomery S, Haddad E, Preston-Martin S, Bennett H and Jaceldo-Siegl K. 2008. Cohort profile: The Adventist Health Study-2 (AHS-2). *International Journal of Epidemiology*. 37 (2) 260-265.
- Butler LM, Wu AH, Wang R, Koh WP, Yuan JM and Yu MC. 2010. A vegetable-fruit-soy dietary pattern protects against breast cancer among postmenopausal Singapore Chinese women. *American Journal of Clinical Nutrition*. 91 (4) 1013-1019.
- Cade JE, Burley VJ and Greenwood DC; UK Women's Cohort Study Steering Group. 2007. Dietary fibre and risk of breast cancer in the UK Women's Cohort Study. *International Journal of Epidemiology*. 36 (2) 431-438.
- Cai T, Yao L and Turesky RJ. 2016. Bioactivation of Heterocyclic Aromatic Amines by UDP Glucuronosyltransferases. *Chemical Research and Toxicology*. 29 (5) 879-891.
- Campbell TC. 2007. Dietary protein, growth factors, and cancer. *Am J Clin Nutr*. 85 (6) 1667.
- Campbell TC and Campbell TM II. 2005. *The China Study*. Dallas, Texas, USA: BenBella Books.
- Campbell WW and Tang M. 2010. Protein intake, weight loss, and bone mineral density in postmenopausal women. *Journals of Gerontology. Series A Biological Sciences and Medical Sciences*. 65 (10) 1115-1122.
- Campbell TC and Junshi C. 1994. Diet and chronic degenerative diseases: perspectives from China. *American Journal of Clinical Nutrition*. 59 (5 Suppl) 1153S-1161S.
- Carmody RN and Wrangham RW. 2009. Cooking and the human commitment to a high-quality diet. *Cold Spring Harbor Symposia on Quantitative Biology*. 74, 427-434.
- Cassidy ES, West PC, Gerber JS, Foley JA. 2013. Redefining agricultural yields: from tonnes to people nourished per hectare. *Environmental Research Letters*. 8: 034015.
- CDC. 2015 Heart Disease Facts [online]. Available from: www.cdc.gov/heartdisease/facts.htm [Accessed 7 October 2016].
- CDC. 2015a. Transmission of Avian Influenza A Viruses Between Animals and People [online]. Available from: www.cdc.gov/flu/avianflu/virus-transmission.htm [Accessed 30 June 2016].
- CDC. 2016. Stroke [online]. Available from: www.cdc.gov/stroke/index.htm [Accessed 7 October 2016].
- Chang ET, Smedby KE, Hjalgrim H, Scholkopf C, Porwit-MacDonald A, Sundstrom C, Tani E, d'Amore F, Melbye M, Adami H-O and Glimelius B. 2005. Medication use and risk of non-Hodgkin's lymphoma. *American Journal of Epidemiology*. 162: 965-274.
- Chavarro JE, Rich-Edwards JW, Rosner BA and Willett WC. 2008. Protein intake and ovulatory infertility. *American Journal of Obstetric Gynecology*. 198, 210.e1-210.e7.
- Chen Z, Wang PP, Woodrow J, Zhu Y, Roebroth B, McLaughlin JR and Parfrey PS. 2015. Dietary patterns and colorectal cancer: results from a Canadian population-based study. *Nutrition Journal*. 14, 8.
- Cho B, Kim MS, Chao K, Lawrence K, Park B and Kim K. 2009. Detection of fecal residue on poultry carcasses by laser-induced fluorescence imaging. *Journal of Food Science*. 74 (3) E154-9.
- Chowdhury R, Warnakula S, Kunutsor S, Crowe F, Ward HA, Johnson L, Franco OH, Butterworth AS, Forouhi NG, Thompson SG, Khaw KT, Mozaffarian D, Danesh J and Di Angelantonio E. 2014. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. *Annals of Internal Medicine*. 160 (6) 398-406.
- Chowdhury R, Warnakula S, Kunutsor S, Crowe F, Ward HA, Johnson L, Franco OH, Butterworth AS, Forouhi NG, Thompson SG, Khaw KT, Mozaffarian D, Danesh J and Di Angelantonio E. 2014a. Correction: Association of Dietary, Circulating, and Supplement Fatty Acids With Coronary Risk. *Annals of Internal Medicine*. 160 (9) 658.
- Chiu BC, Cerhan JR, Folsom AR, Sellers TA, Kushi LH, Wallace RB, Zheng W and Potter JD. 1996. Diet and risk of non-Hodgkin lymphoma in older women. *JAMA*. 275: 1315-21.
- Clarys P, Deliens T, Huybrechts I, Deriemaeker P, Vanaelst B, De Keyser W, Hebbelinck M and Mullie P. 2014. Comparison of nutritional quality of the vegan, vegetarian, semi-vegetarian, pesco-vegetarian and omnivorous diet. *Nutrients*. 6 (3) 1318-1332.
- CNN. 2004. *Bird flu sparks Asian health scare* [online]. Available from: <http://edition.cnn.com/2004/WORLD/asiapcf/01/13/bird.flu/> [Accessed 29 June 2016].

- Collignon, P, Aarestrup FM, Irwin R and McEwen S. 2013. Human Deaths and Third-Generation Cephalosporin use in Poultry, Europe. *Emerging Infectious Diseases*. 19 (8): 1339-1340.
- Collinge J, Whitfield J, McKintosh E, Beck J, Mead S, Thomas DJ and Alpers MP. 2006. Kuru in the 21st century—an acquired human prion disease with very long incubation periods. *Lancet*. 367, 2068-2074.
- Cordain L, Eaton SB, Sebastian A, Mann N, Lindeberg S, Watkins BA, O’Keefe JH and Brand-Miller J. 2005. Origins and evolution of the Western diet: health implications for the 21st century. *American Journal of Clinical Nutrition*. 81 (2) 341-354.
- Craig WJ. 1994. Iron Status of Vegetarians. *American Journal of Clinical Nutrition*. 59, Supp, p1233S-7S.
- Craig WJ and Mangels AR; American Dietetic Association. 2009. Position of the American Dietetic Association: vegetarian diets. *Journal of the American Dietetic Association*. 109 (7) 1266-1282.
- Cross AJ, Pollock JR and Bingham SA. 2003. Haem, not protein or inorganic iron, is responsible for endogenous intestinal N-nitrosation arising from red meat. *Cancer Research*. 63 (10) 2358-2360.
- Cross AJ and Sinha R. 2005. *Impact of food preservation, processing, and cooking on cancer risk*. In: Beck K, ed. *Carcinogenic and anticarcinogenic food components*. Boca Raton, Florida, US: CRC Press.
- Cross AJ, Leitzmann MF, Gail MH, Hollenbeck AR, Schatzkin A and Sinha R. 2007. A prospective study of red and processed meat intake in relation to cancer risk. *PLoS Medicine*. 4 (12) e325.
- Cross AJ, Harnly JM, Ferrucci LM, Risch A, Mayne ST and Sinha R. 2012. Developing a heme iron database for meats according to meat type, cooking method and doneness level. *Food Nutrition Science*. 3 (7) 905-913.
- Crowe FL, Appleby PN, Travis RC and Key TJ. 2013. Risk of hospitalization or death from ischemic heart disease among British vegetarians and non-vegetarians: results from the EPIC-Oxford cohort study. *American Journal of Clinical Nutrition*. 97 (3) 597-603.
- Czerwonka M, Szterk A and Waszkiewicz-Robak B. 2014. Vitamin B12 content in raw and cooked beef. *Meat Science*. 96 (3) 1371-1375.
- Cui X, Dai Q, Tseng M, Shu XO, Gao YT and Zheng W. 2007. Dietary patterns and breast cancer risk in the shanghai breast cancer study. *Cancer Epidemiology Biomarkers and Prevention*. 16 (7) 1443-1448.
- Cutillas-Tolín A, Mínguez-Alarcón L, Mendiola J, López-Espín JJ, Jørgensen N, Navarrete-Muñoz EM, Torres-Cantero AM and Chavarro JE. 2015. Mediterranean and western dietary patterns are related to markers of testicular function among healthy men. *Human Reproduction*. 30 (12) 2945-2955.
- Dai Z, Butler LM, van Dam RM, Ang LW, Yuan JM and Koh WP. 2014. Adherence to a vegetable-fruit-soy dietary pattern or the Alternative Healthy Eating Index is associated with lower hip fracture risk among Singapore Chinese. *Journal of Nutrition*. 144 (4) 511-518.
- Daniel CR, Cross AJ, Graubard BI, Park Y, Ward MH, Rothman N, Hollenbeck AR, Chow WH and Sinha R. 2012. Large prospective investigation of meat intake, related mutagens, and risk of renal cell carcinoma. *American Journal of Clinical Nutrition*. 95 (1) 155-162.
- Daniele G, Eldor R, Merovci A, Clarke GD, Xiong J, Tripathy D, Taranova A, Abdul-Ghani M, DeFronzo RA. 2013. Chronic reduction of plasma free fatty acid improves mitochondrial function and whole-body insulin sensitivity in obese and type 2 diabetic individuals. *Diabetes*. 63(8) 2812-2820.
- Dargent-Molina P, Sabia S, Touvier M, Kesse E, Bréart G, Clavel-Chapelon F and Boutron-Ruault MC. 2008. Proteins, dietary acid load, and calcium and risk of postmenopausal fractures in the E3N French women prospective study. *Journal of Bone and Mineral Research*. 23 (12) 1915-1922.
- Darling AL, Millward DJ, Torgerson DJ, Hewitt CE and Lanham-New SA. 2009. Dietary protein and bone health: a systematic review and meta-analysis. *American Journal of Clinical Nutrition*. 90.1674-1692.
- Darsi J, Frost GS, Montaser R, Yap J and Robertson MD. 2014. Influence of the tolerability of vinegar as an oral source of short-chain fatty acids on appetite control and food intake. *International Journal of Obesity*. 38 (5) 675-681.
- Davey GK, Spencer EA, Appleby PN, Allen NE, Knox KH and Key TJ. 2003. EPIC-Oxford: lifestyle characteristics and nutrient intakes in a cohort of 33 883 meat-eaters and 31 546 non- meat-eaters in the UK. *Public Health Nutrition*. 6 (3) 259-269.
- Debbink K, Lindesmith LC, Donaldson EF and Baric RS. 2012. Norovirus immunity and the great escape. *PLoS Pathogens*. 8 (10) e1002921.
- de Bortoli MC and Cozzolino SM. 2009. Zinc and selenium nutritional status in vegetarians. *Biological Trace Elements Research*. 127 (3) 228-233.
- Defra. 2000. *BSE: Disease control & eradication - Causes of BSE* [online]. Available from: <http://webarchive.nationalarchives.gov.uk/20130822084033/http://www.defra.gov.uk/animalh/bse/controls-eradication/causes.html> [Accessed 4 July 2016].
- Defra. 2012. *Agriculture in the United Kingdom, 2012* [online]. Available from: <http://webarchive.nationalarchives.gov.uk/20130123162956/http://www.w.defra.gov.uk/statistics/files/defra-stats-foodfarm-crosscutting-auk-auk2011-120709.pdf> [Accessed 14 October 2015].
- Defra. 2015. *Family food datasets* [online]. Available from: www.gov.uk/government/statistical-data-sets/family-food-datasets [Accessed 5 January 2016].
- Defra. 2015a. *Agriculture in the United Kingdom, 2014* [online]. Available from: www.gov.uk/government/uploads/system/uploads/attachment_data/file/430411/auk-2014-28may15a.pdf [Accessed 14 October 2015].
- Defra. 2015b. *Highly Pathogenic Avian Influenza H5N1 in poultry in France* [online]. Available from: www.gov.uk/government/uploads/system/uploads/attachment_data/file/480529/poa-h5n1-hpai-france.pdf [Accessed 29 June 2016].
- Delarue J, Magnan, C. 2007. Free fatty acids and insulin resistance. *Current Opinion in Clinical Nutrition and Metabolic Care*. 10 (2) 142-148
- Department of Health, 1991. *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom*. HMSO, London.
- Diabetes UK. 2012. *Diabetes in the UK 2012* [online]. Available from: www.diabetes.org.uk/diabetes-in-the-uk-2012 [Accessed 20 June 2016].
- Diack AB, Head MW, McCutcheon S, Boyle A, Knight R, Ironside JW, Manson JC and Will RG. 2014. Variant CJD. 18 years of research and surveillance. *Prion*. 8 (4) 286-295.
- Di Pinto A, Bottaro M, Bonerba E, Bozzo G, Ceci E, Marchetti P, Mottola A and Tantillo G. 2014. Occurrence of mislabeling in meat products using DNA-based assay. *Journal of Food Science and Technology*. 52 (4) 2479-2484.
- DIPP. 2012. *The DIPP project* [online]. Available from: <http://dipp.utu.fi/index.php?mid=8&language=en> [Accessed 20 June 2016].
- Eaton SB and Konner M. 1985. Paleolithic Nutrition – A Consideration of Its Nature and Current Implications. *New England Journal of Medicine*. 312 (5) 283-289.
- EFSA. 2011. Scientific Opinion on an update on the present knowledge on the occurrence and control of foodborne viruses [online]. Available from: www.efsa.europa.eu/sites/default/files/scientific_output/files/main_documents/2190.pdf [Accessed 27 June 2016].

- EFSA. 2015. Scientific Opinion on Dietary Reference Values for cobalamin (vitamin B12). *EFSA Journal*. 13 (7) 4150
- Environment America. 2016. *Corporate Agribusiness and the Fouling of America's Waterways* [online]. Available from: www.environmentamericacenter.org/reports/ame/corporate-agribusiness-and-fouling-americas-waterways [Accessed 30 November 2016].
- Fairweather-Tait SJ. 2004. Iron nutrition in the UK: getting the balance right. *Proceedings of the Nutrition Society*. 63 (4) 519-528.
- FAO, 2005. *Epidemiology of H5N1 Avian Influenza in Asia and Implications for Regional Control* [online]. Available from: www.fao.org/docs/eims/upload/246974/aj122e00.pdf [Accessed 29 June 2016].
- FAO. 2006. *Livestock's Long Shadow* [online]. Available from: www.fao.org/docrep/010/a0701e/a0701e00.HTM [Accessed 30 June 2016].
- FAO. 2011. *Approaches to controlling, preventing and eliminating H5N1 HPAI in endemic countries*. FAO Animal Production and Health Paper No. 171 [online]. Available from: www.fao.org/docrep/014/i2150e/i2150e.pdf [Accessed 29 June 2016].
- FAOSTAT. 2015. *Food and Agriculture Organisation of the United Nations Statistics Division* [online]. Available from: <http://faostat3.fao.org/download/FB/CL/E> [Accessed 12 October 2015].
- Farvid MS, Cho E, Chen WY, Eliassen AH and Willett WC. 2014. Dietary protein sources in early adulthood and breast cancer incidence: prospective cohort study. *British Medical Journal*. 348 g3437.
- Farvid MS, Cho E, Chen WY, Eliassen AH and Willett WC. 2015. Adolescent meat intake and breast cancer risk. *International Journal of Cancer*. 136 (8) 1909-1920.
- Farvid MS, Chen WY, Michels KB, Cho E, Willett WC and Eliassen AH. 2016. Fruit and vegetable consumption in adolescence and early adulthood and risk of breast cancer: population based cohort study *British Medical Journal*. 353, i2343.
- Fayet F, Flood V, Petocz P and Samman S. 2014. Avoidance of meat and poultry decreases intakes of omega-3 fatty acids, vitamin B12, selenium and zinc in young women. *Journal of Human Nutrition and Dietetics*. 27 Suppl 2: 135-142.
- FDA. 2015. *Arsenic-based Animal Drugs and Poultry* [online]. Available from: www.fda.gov/AnimalVeterinary/SafetyHealth/ProductSafetyInformation/ucm257540.htm [Accessed 28 June 2016].
- Fenton TR, Lyon AW, Eliasziw M, Tough SC and Hanley DA. 2009. Meta-analysis of the effect of the acid-ash hypothesis of osteoporosis on calcium balance. *Journal of Bone and Mineral Research*. 24 (11) 1835-1840.
- Fenton TR, Tough SC, Lyon AW, Eliasziw M and Hanley DA. 2011. Causal assessment of dietary acid load and bone disease: a systematic review & meta-analysis applying Hill's epidemiologic criteria for causality. *Nutrition Journal*. 10, 41.
- Ferrari P, Rinaldi S, Jenab M, Lukanova A, Olsen A, Tjønneland A, Overvad K, Clavel-Chapelon F, Fagherazzi G, Touillaud M, Kaaks R, von Rüsten A, Boeing H, Trichopoulou A, Lagiou P, Benetou V, Grioni S, Panico S, Masala G, Tumino R, Polidoro S, Bakker MF, van Gils CH, Ros MM, Bueno-de-Mesquita HB, Krum-Hansen S, Engeset D, Skeie G, Pilar A, Sánchez MJ, Buckland G, Ardanaz E, Chirlaque D, Rodriguez L, Travis R, Key T, Khaw KT, Wareham NJ, Sund M, Lenner P, Slimani N, Norat T, Aune D, Riboli E and Romieu I. 2013. Dietary fiber intake and risk of hormonal receptor-defined breast cancer in the European Prospective Investigation into Cancer and Nutrition study. *American Journal of Clinical Nutrition*. 97 (2) 344-353.
- Feskens EJ, Sluik D and van Woudenberg GJ. 2013. Meat consumption, diabetes, and its complications. *Current Diabetes Reports*. 13 (2) 298-306.
- Fields H, Millstine D, Agrwal N and Marks L. 2016. *Journal of the American Osteopathic Association*. 116 (5) 296-300.
- Fisher BA, Wilkinson L and Valencia A. 2016. Women's interest in a personal breast cancer risk assessment and lifestyle advice at NHS mammography screening. *Journal of Public Health*. pii: fdv211
- Fleming DJ, Tucker KL, Jacques PF, Dallal GE, Wilson PW, Wood RJ. 2002. Dietary factors associated with the risk of high iron stores in the elderly Framingham Heart Study cohort. *Am J Clin Nutr*. 76 (6) 1375-1384.
- Foodwatch. 2008. Klimaretter Bio? [online]. Available from: www.foodwatch.org/uploads/media/foodwatch-Report_Klimaretter-Bio_20080825_01.pdf [Accessed 4 July 2016].
- Fontana L, Klein S, Holloszy JO. 2006. Long-term low-protein, low-calorie diet and endurance exercise modulate metabolic factors associated with cancer risk. *Am J Clin Nutr*. 84 (6) 1456-1462.
- Fortune A. 2015. No fears over IARC report. *Meat Trades Journal*. 6 November 2015, p1.
- Food Safety Authority of Ireland. 2013. Results of Burgers Analysed [online]. Available from: www.fsai.ie/uploadedFiles/News_Centre/Burger_results_2013_01.pdf [Accessed 30 June 2016].
- Fordyce FM. 2005. *Selenium deficiency and toxicity in the environment*. In *Essentials of Medical Geology* p373-415. eds O Selinus, B Alloway, JA Centeno, RB Finkelman, R Fuge, U Lindh and P Smedley. London: Elsevier.
- Fraser G. 2003. *Diet, Life Expectancy, and Chronic Disease: Studies of Seventh-Day Adventists and Other Vegetarians*. New York: Oxford University Press;
- Fraser G. 2009. Vegetarian diets: what do we know of their effects on common chronic diseases? *American Journal of Clinical Nutrition*. 89 1607S-1612S.
- Frassetto LA, Todd KM, Morris RC Jr and Sebastian A. 2000. Worldwide incidence of hip fracture in elderly women: relation to consumption of animal and vegetable foods. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*. 55 (10) M585-592.
- Frassetto L, Morris RC Jr, Sellmeyer DE, Todd K and Sebastian A. 2001. Diet, evolution and aging—the pathophysiologic effects of the post-agricultural inversion of the potassium-to-sodium and base-to-chloride ratios in the human diet. *European Journal of Nutrition*. 40 (5) 200-213.
- Frassetto L and Sebastian A. 2013. Commentary to accompany the paper entitled 'nutritional disturbance in acid-base balance and osteoporosis: a hypothesis that disregards the essential homeostatic role of the kidney', by Jean-Philippe Bonjour. *British Journal of Nutrition*. 110 (11) 1935-1937.
- Frattaroli J, Weidner G, Dnistrian AM, Kemp C, Daubenmier JJ, Marlin RO, Crutchfield L, Yglecias L, Carroll PR and Ornish D. 2008. Clinical events in prostate cancer lifestyle trial: results from two years of follow-up. *Urology*. 72 (6) 1319-1323.
- Fretts AM, Follis JL, Nettleton JA, Lemaitre RN, Ngwa JS, Wojczynski MK, Kalafati IP, Varga TV, Frazier-Wood AC, Houston DK, Lahti J, Ericson U, van den Hooven EH, Mikkilä V, Kieft-de Jong JC, Mozaffarian D, Rice K, Renström F, North KE, McKeown NM, Feitosa MF, Kanoni S, Smith CE, Garcia ME, Tiainen AM, Sonestedt E, Manichaikul A, van Rooij FJ, Dimitriou M7, Raitakari O, Pankow JS, Djoussé L, Province MA, Hu FB, Lai CQ, Keller MF, Perälä MM, Rotter JI, Hofman A, Graff M, Kähönen M, Mukamal K, Johansson I, Ordovas JM, Liu Y, Männistö S, Uitterlinden AG, Deloukas P, Seppälä I, Psaty BM, Cupples LA, Borecki IB, Franks PW, Arnett DK, Nalls MA, Eriksson JG, Orho-Melander M, Franco OH, Lehtimäki T, Dedoussis GV, Meigs JB, Siscovick DS. 2015. Consumption of meat is associated

with higher fasting glucose and insulin concentrations regardless of glucose and insulin genetic risk scores: a meta-analysis of 50,345 Caucasians. *Am J Clin Nutr.* 102 (5) 1266-1278.

Friedman GD and van den Eeden SK. 1997. Risk factors for pancreatic cancer: an exploratory study. *International Journal of Epidemiology.* 22 (1) 30-37.

FSA. 2000. A Report of the Study of Infectious Intestinal Disease in England (IID1) [online]. Available from: www.esds.ac.uk/doc/4092%5Cmrdoc%5Cpdf%5C4092userguide6.pdf [Accessed 23 June 2016].

FSA. 2002. McCance and Widdowson's *The Composition of Foods, 6th summary edition*. Cambridge, England: Royal Society of Chemistry.

FSA. 2011. *Foodborne disease strategy 2010-15. An FSA programme for the reduction of foodborne disease in the UK* [online]. Available from: www.food.gov.uk/sites/default/files/multimedia/pdfs/fds2015.pdf [Accessed 2 October 2015].

FSA. 2011a. *The Second Study of Infectious Intestinal Disease in the Community (IID2)* [online]. Available from: www.food.gov.uk/sites/default/files/7111-1-1393_IID2_FINAL_REPORT.pdf [Accessed 23 June 2016].

FSA. 2011b. UK Publicly Funded Research Relating to *Salmonella*: Update, May 2009 [online]. Available from: www.food.gov.uk/sites/default/files/multimedia/pdfs/msffgsalmonella1.pdf [Accessed 27 June 2016].

FSA. 2012. Food Standards Agency: *Guidelines for use and reproduction of the eatwell plate model* [online]. Available from: www.food.gov.uk/northern-ireland/nutritionni/eatwellplate/eatwell-guidelines [Accessed 5 July 2016].

FSA. 2014. *Costed extension to the Second Study of Infectious Intestinal Disease in the Community: Identifying the proportion of foodborne disease in the UK and attributing foodborne disease by food commodity* [online]. Available from: www.food.gov.uk/science/research/foodborneillness/b14programme/b14projlist/fs231043ext [Accessed 22 June 2016].

FSA. 2015. *Campylobacter survey: cumulative results from the full 12 months (Q1 - Q4)* [online]. Available from: www.food.gov.uk/news-updates/news/2015/14003/campylobacter-survey-results-12months [Accessed 24 June 2016].

FSA. 2016. *Year 2 of a UK-wide survey of campylobacter contamination on fresh chickens at retail (July 2015 to March 2016)* [online]. Available from: www.food.gov.uk/science/microbiology/campylobacterevidenceprogramme/retail-survey-year-2 [Accessed 28 June 2016].

Fung TT, Hu FB, Wu K, Chiuve SE, Fuchs CS, Giovannucci E. 2010. The Mediterranean and Dietary Approaches to Stop Hypertension (DASH) diets and colorectal cancer. *Am J Clin Nutr.* 92 (6) 1429-1435.

Gabriel HE, Crott JW, Ghandour H, Dallal GE, Choi SW, Keyes MK, Jang H, Liu Z, Nadeau M, Johnston A, Mager D and Mason JB. 2006. Chronic cigarette smoking is associated with diminished folate status, altered folate form distribution, and increased genetic damage in the buccal mucosa of healthy adults. *American Journal of Clinical Nutrition.* 83 (4) 835-841.

Gani A. 2015. *UK shoppers give pork the chop after processed meats linked to cancer*. *Guardian* [online]. Available from: www.theguardian.com/uk-news/2015/nov/23/bacon-sausage-sales-fall-who-report-cancer-risk-processed-meat [Accessed 16 December 2015].

Gathirua-Mwangi WG and Zhang J. 2014. Dietary factors and risk for advanced prostate cancer. *European Journal of Cancer Prevention.* 23 (2) 96-109.

Geissler C and Powers H eds. 2005. *Human Nutrition*. 11th ed. [CD-ROM] London: Elsevier Churchill Livingstone. Figure 12.3.

Gerber M and Corpet D. 1999. Energy balance and cancers. *European Journal of Cancer Prevention.* 8 (2) 77-89.

Gerhardsson-DeVerdier MG, Hagman U, Peters RK, Steineck G and Overvik E. 1991. Meat, cooking methods and colorectal cancer - A case-referent study in Stockholm. *International Journal of Cancer.* 49 520-525.

Gibson S and Ashwell M. 2003. The association between red and processed meat consumption and iron intakes and status among British adults. *Public Health Nutrition.* 6 (4) 341-350.

Gibson RS, Heath AL and Szymlek-Gay EA. 2014. Is iron and zinc nutrition a concern for vegetarian infants and young children in industrialized countries? *American Journal of Clinical Nutrition.* Vol 100 Supplement 1, 459S-4568S.

Gillespie KM, Bain SC, Barnett AH, Bingley PJ, Christie MR, Gill GV and Gale EA. 2004. The rising incidence of childhood type 1 diabetes and reduced contribution of high-risk HLA haplotypes. *Lancet.* 364 (9446) 1699-700.

Gkogkolou P and Böhm M. 2012. Advanced glycation end products: Key players in skin aging? *Dermatoendocrinology.* 4 (3) 259-270.

GLOBOCAN. 2012. Estimated cancer incidence, mortality and prevalence worldwide in 2012 [online]. Available from: <http://globocan.iarc.fr> [Accessed 21 April 2016].

Gomez SL, Quach T, Horn-Ross PL, Pham JT, Cockburn M, Chang ET, Keegan TH, Glaser SL and Clarke CA. 2010. Hidden breast cancer disparities in Asian women: disaggregating incidence rates by ethnicity and migrant status. *American Journal of Public Health.* 100 Suppl 1 S125-31.

Grant WB. 2014. A Multicountry Ecological Study of Cancer Incidence Rates in 2008 with Respect to Various Risk-Modifying Factors. *Nutrients.* 6 (1) 163-189.

Greger M. 2006. *Bird Flu: A Virus of Our Own Hatching*. USA: Lantern Books.

Greger M. 2012. *Which type of vitamin B12 is best—cyanocobalamin, methylcobalamin, or hydroxycobalamin?* [online]. Available from: <http://nutritionfacts.org/questions/which-type-of-b12-is-best/> [Accessed 9 December 2015].

Greger M. 2015. *What Causes Insulin Resistance?* Video [online]. Available from: <http://nutritionfacts.org/video/what-causes-insulin-resistance/> [Accessed 25 October 2016].

Guardian. 2015. *Official advice on low-fat diet and cholesterol is wrong, says health charity* [online]. Available from: www.theguardian.com/society/2016/may/22/official-advice-to-eat-low-fat-diet-is-wrong-says-health-charity [Accessed 28 June 2016].

Hadjirin NF, Lay EM, Paterson GK, Harrison EM, Peacock SJ, Parkhill J, Zadoks RN and Holmes MA. 2015. Detection of livestock-associated methicillin-resistant *Staphylococcus aureus* CC398 in retail pork, United Kingdom. *Euro Surveill.* 20 (24) pii: 21156.

Harcombe Z. 2011. *The Obesity Epidemic What caused it? How can we stop it?* [online]. Available from: www.theobesityepidemic.co.uk/assets/pdfs/TOE-WAPF2011.pdf [Accessed 15 February 2016].

Harcombe Z, Baker JS, Cooper SM, Davies B, Sculthorpe N, DiNicolantonio JJ and Grace F. 2015. Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review and meta-analysis. *Open Heart.* 2 (1) e000196.

Harcombe Z, Baker JS, Cooper SM, Davies B, Sculthorpe N, DiNicolantonio JJ and Grace F. 2015a. Erratum for: Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review and meta-analysis. *Open Heart.* 2 (1) e000196corr1.

- Hawks J, Wang ET, Cochran GM, Harpending HC and Moyzis RK. 2007. Recent acceleration of human adaptive evolution. *Proceeding of the National Academy of Sciences*. 104 (52) 20753-20758.
- Hemalatha, S, Platel K. and Srinivasan K. 2007. Influence of germination and fermentation on bioaccessibility of zinc and iron from food grains. *European Journal of Clinical Nutrition*. 61 (3) 342-348.
- Hemilä H and Paunio M. 1997. Blood donation, body iron stores, and risk of myocardial infarction. Confidence intervals and possible selection bias call study results into question. *British Medical Journal*. 314 (7097) 1830-1831.
- Henderson L, Irving K, Bates CJ, Prentice A, Perks J, Swan G and Farron M. 2003. *The National Diet and Nutrition Survey: Adults Aged 19-64 Years. Volume 3. Vitamin and Mineral Intake and Urinary Analytes*. The Stationery Office; London.
- Henry AG, Brooks AS and Piperno DR. 2014. Plant foods and the dietary ecology of Neanderthals and early modern humans. *Journal of Human Evolution*. 69, 44-54.
- Herbert V. 1994. Staging vitamin B12 (cobalamin) status in vegetarians. *The American Journal of Clinical Nutrition*. 59 (5) 1213S-1222S.
- Hermann R, Knip M, Veijola R, Simell O, Laine AP, Akerblom HK, Groop PH, Forsblom C, Pettersson-Fernholm K, Ilonen J; FinnDiane Study Group. 2003. Temporal changes in the frequencies of HLA genotypes in patients with Type 1 diabetes—indication of an increased environmental pressure? *Diabetologia*. 46 (3) 420-425.
- Hex N, Bartlett C, Wright D, Taylor M and Varley D. 2012. Estimating the current and future costs of Type 1 and Type 2 diabetes in the UK, including direct health costs and indirect societal and productivity costs. *Diabetic Medicine*. 29 (7) 855-862.
- Hoeflich J, Hollenbach B, Behrends T, Hoeg A, Stosnach H and Schomburg L. 2010. The choice of biomarkers determines the selenium status in young German vegans and vegetarians. *British Journal of Nutrition*. 104 (11) 1601-1604.
- Hoeks J, van Herpen NA, Mensink M, Moonen-Kornips E, van Beurden D, Hesselink MK, Schrauwen P. 2010. Prolonged fasting identifies skeletal muscle mitochondrial dysfunction as consequence rather than cause of human insulin resistance. *Diabetes*. 59 (9) 2117-2125.
- Holtrop G, Johnstone AM, Fyfe C and Gratz SW. 2012. Diet composition is associated with endogenous formation of N-nitroso compounds in obese men. *Journal of Nutrition*. 142 (9) 1652-1658.
- Hooda J, Shah A and Zhang L. 2014. Heme, an essential nutrient from dietary proteins, critically impacts diverse physiological and pathological processes. *Nutrients*. 6 (3) 1080-1102.
- Hooper L, Summerbell CD, Higgins JP, Thompson RL, Clements G, Capps N, Davey S, Riemersma RA and Ebrahim S. 2012. Reduced or modified dietary fat for preventing cardiovascular disease. *Cochrane Database of Systematic Reviews*. 5 CD002137.
- Hope J. 2014. Saturated fat 'ISN'T bad for your heart': Major study questions decades of dietary advice. *Daily Mail*. 17 March [online]. Available from: www.dailymail.co.uk/health/article-2582867/Saturated-fat-DOESNT-cause-heart-disease-all.html [Accessed 28 June 2016].
- Hope J. 2015. Butter ISN'T bad for you after all: Major study says 80s advice on dairy fats was flawed *Daily Mail*. 9 February [online]. Available from: www.dailymail.co.uk/health/article-2946617/Butter-ISN-T-bad-Major-study-says-80s-advice-dairy-fats-flawed.html [Accessed 28 June 2016].
- Hosseini M, Naghan PA, Jafari AM, Yousefifard M, Taslimi S, Khodadad K, Mohammadi F, Sadr M, Rezaei M, Mortaz E and Masjedi MR. 2014. Nutrition and lung cancer: a case control study in Iran. *BMC Cancer*. 14, 860.
- Hu FB, Manson JE and Willett WC. 2001. Types of dietary fat and risk of coronary heart disease: a critical review. *Journal of the American College of Nutrition*. 20 5-19.
- Huang L, Yao L, He Z, Zhou C, Li G, Yang B, and Deng X. 2014. Roxarsone and its metabolites in chicken manure significantly enhance the uptake of As species by vegetables. *Chemosphere*. 100, 5762.
- Hughes R, Cross AJ, Pollock JR and Bingham S. 2001. Dose-dependent effect of dietary meat on endogenous colonic N-nitrosation. *Carcinogenesis*. 22 (1) 199-202.
- Hunnicut J, He K and Xun P. 2014. Dietary iron intake and body iron stores are associated with risk of coronary heart disease in a meta-analysis of prospective cohort studies. *Journal of Nutrition*. 144 (3) 359-366.
- Institute Of Medicine (US) Standing Committee On The Scientific Evaluation Of Dietary Reference Intakes And Its Panel On Folate, Other B Vitamins, 1998. *Dietary Reference Intakes: Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline*. Food and Nutrition Board, Institute of Medicine. Washington, DC: National Academy Press.
- InterAct Consortium, Bendinelli B, Palli D, Masala G, Sharp SJ, Schulze MB, Guevara M, van der AD, Sera F, Amiano P, Balkau B, Barricarte A, Boeing H, Crowe FL, Dahm CC, Dalmeijer G, de Lauzon-Guillain B, Egeberg R, Fagherazzi G, Franks PW, Krogh V, Huerta JM, Jakyszyn P, Khaw KT, Li K, Mattiello A, Nilsson PM, Overvad K, Ricceri F, Rolandsson O, Sánchez MJ, Slimani N, Sluijs I, Spijkerman AM, Teucher B, Tjonneland A, Tumino R, van den Berg SW, Forouhi NG, Langeberg C, Feskens EJ, Riboli E and Wareham NJ. 2013. Association between dietary meat consumption and incident type 2 diabetes: the EPIC-InterAct study. *Diabetologia*. 56 (1) 47-59.
- IRI. 2015. *Processed meat cancer scare wipes 10% (£3 million) off sales of sausages and bacon according to IRI Retail Advantage data* [online]. Available from: www.iriworldwide.com/en-GB/insights/pressreleases [Accessed 6 January 2016].
- Jägerstad M and Skog K. 1991. Formation of meat mutagens. *Advances in Experimental Medicine and Biology*. 289 83-105.
- Jakyszyn P, Agudo A, Ibáñez R, García-Closas R, Pera G, Amiano P and González CA. 2004. Development of a food database of nitrosamines, heterocyclic amines, and polycyclic aromatic hydrocarbons. *Journal of Nutrition*. 134 2011-2014.
- Jemal A, Bray F, Center MM, Ferlay J, Ward E and Forman D. 2011. Global cancer statistics. *CA: A Cancer Journal for Clinicians*. 61 69-90.
- Jiang R, Ma J, Ascherio A, Stampfer MJ, Willett WC and Hu FB. 2004. Dietary iron intake and blood donations in relation to risk of type 2 diabetes in men: a prospective cohort study. *American Journal of Clinical Nutrition*. 79 (1) 70-75.
- John EM, Stern MC, Sinha R and Koo J. 2011. Meat consumption, cooking practices, meat mutagens, and risk of prostate cancer. *Nutrition and Cancer*. 63 (4) 525-537.
- Johnson JR, Kuskowski MA, Smith K, O'Bryan TT and Tatini S. 2005. Antimicrobial-resistant and extraintestinal pathogenic *Escherichia coli* in retail foods. *Journal of Infectious Disease*. 191 (7) 1040-1049.
- Johnston J, Prynne CJ, Stephen AM, Wadsworth ME; British Birth Cohort. 2007. Haem and non-haem iron intake through 17 years of adult life of a British Birth Cohort. *British Journal of Nutrition*. 98 (5) 1021-1028.
- Joosen AM, Kuhnle GG, Aspinall SM, Barrow TM, Lecommandeur E, Azqueta A, Collins AR and Bingham SA. 2009. Effect of processed and red meat on endogenous nitrosation and DNA damage. *Carcinogenesis*. 30 1402-1407.
- Joshi AD, Corral R, Catsburg C, Lewinger JP, Koo J, John EM, Ingles SA and Stern MC. 2012. Red meat and poultry, cooking practices, genetic susceptibility and risk of prostate cancer: results from a multiethnic case-control study. *Carcinogenesis*. 33 (11) 2108-2118.
- Kaesler B. 2005. *Vitamins: 9. Vitamin B12 (Cyanocobalamins)*, *Ullmann's Encyclopedia of Industrial Chemistry*. Weinheim: Wiley-VCH.

Kanis JA, Odén A, McCloskey EV, Johansson H, Wahl DA and Cooper C; IOF Working Group on Epidemiology and Quality of Life. 2012. A systematic review of hip fracture incidence and probability of fracture worldwide. *Osteoporosis International*. 23 (9) 2239-2256.

Kassianos AP, Coyle A and Raats MM. 2015. Perceived influences on post-diagnostic dietary change among a group of men with prostate cancer. *European Journal of Cancer Care (Engl)*. 24 (6) 818-826.

Kato I, Koenig KL, Baptiste MS, Lillquist PP, Frizzera G, Burke JS, Watanabe H and Shore RE. 2003. History of antibiotic use and risk of non-Hodgkin's lymphoma (NHL). *International Journal of Cancer*. 107: 99-105.

Katz, D. 2015. An Open Letter to the BMJ Regarding US Dietary Guidance. *Huffington Post*. 25 September [online]. Available from: www.huffingtonpost.com/david-katz-md/an-open-letter-to-the-bmj_b_8195708.html [Accessed 28 June 2016].

Kerstetter JE, O'Brien KO and Insogna KL. 2003. Dietary protein, calcium metabolism, and skeletal homeostasis revisited. *American Journal of Clinical Nutrition*. 78 (3 Suppl) 584S-592S.

Kerstetter JE, O'Brien KO, Caseria DM, Wall DE and Insogna KL. 2005. The impact of dietary protein on calcium absorption and kinetic measures of bone turnover in women. *The Journal of Clinical Endocrinology and Metabolism*. 90 (1) 26-31.

Kerstetter JE. 2009. Dietary protein and bone: a new approach to an old question. *American Journal of Clinical Nutrition*. 90 (6) 1451-1452.

Key TJ, Fraser GE, Thorogood M, Appleby PN, Beral V, Reeves G, Burr ML, Chang-Claude J, Frentzel-Beyme R, Kuzma JW, Mann J and McPherson K. 1999. Mortality in vegetarians and non-vegetarians: detailed findings from a collaborative analysis of 5 prospective studies. *American Journal of Clinical Nutrition*. 70 (3 Suppl) 516S-524S.

Key TJ, Appleby PN, Spencer EA, Travis RC, Roddam AW and Allen NE. 2009. Mortality in British vegetarians: results from the European Prospective Investigation into Cancer and Nutrition (EPIC-Oxford). *American Journal of Clinical Nutrition*. 89 (5) 1613S-1619S.

Key TJ, Appleby PN, Crowe FL, Bradbury KE, Schmidt JA and Travis RC. 2014. Cancer in British vegetarians: updated analyses of 4998 incident cancers in a cohort of 32,491 meat eaters, 8612 fish eaters, 18,298 vegetarians, and 2246 vegans. *American Journal of Clinical Nutrition*. 100 (1 Suppl) 378S-385S.

King JC, Blumberg J, Ingwersen L, Jenab M and Tucker KL. 2008. Tree nuts and peanuts as components of a healthy diet. *Journal of Nutrition*. 138 (9) 1736S-1740S.

Kitessa SM, Abeywardena MY. 2016. Lipid-Induced Insulin Resistance in Skeletal Muscle: The Chase for the Culprit Goes from Total Intramuscular Fat to Lipid Intermediates, and Finally to Species of Lipid Intermediates. *Nutrients*. 8 (8).

Koeth RA, Wang Z, Levison BS, Buffa JA, Org E, Sheehy BT, Britt EB, Fu X, Wu Y, Li L, Smith JD, DiDonato JA, Chen J, Li H, Wu GD, Lewis JD, Warrier M, Brown JM, Krauss RM, Tang WH, Bushman FD, Lusis AJ and Hazen SL. 2013. Intestinal microbiota metabolism of L-carnitine, a nutrient in red meat, promotes atherosclerosis. *Nature Medicine*. 19 (5) 576-585.

Kleinbongard P, Dejam A, Lauer T, Jax T, Kerber S, Gharini P, Balzer J, Zotz RB, Scharf RE, Willers R, Schechter AN, Feelisch M and Kelm M. 2005. Plasma nitrite concentrations reflect the degree of endothelial dysfunction in humans. *Free Radical Biology and Medicine*. 40 (2) 295-302.

Kuhnle GG, Story GW, Reda T, Mani AR, Moore KP, Lunn JC and Bingham SA. 2007. Diet-induced endogenous formation of nitroso compounds in the GI tract. *Free Radical Biology and Medicine*. 43 (7) 1040-1047.

Larsson SC and Wolk A. 2006. Meat consumption and risk of colorectal cancer: a meta-analysis of prospective studies. *International Journal of Cancer*. 119 2657-2664.

Larsson SC, Orsini N and Wolk A. 2006. Processed meat consumption and stomach cancer risk: a meta-analysis. *Journal of the National Cancer Institute*. 98 (15) 1078-1087.

Larsson SC and Wolk A. 2012. Red and processed meat consumption and risk of pancreatic cancer: meta-analysis of prospective studies. *British Journal of Cancer*. 106 (3) 603-607.

Larsson SC and Orsini N. 2014. Red meat and processed meat consumption and all-cause mortality: a meta-analysis. *American Journal of Epidemiology*. 179 (3) 282-289.

Li FC, Choi BC, Sly T and Pak AW. 2008. Finding the real case-fatality rate of H5N1 avian influenza. *Journal of Epidemiology and Community Health*. 62 (6) 555-559.

Lipsitch M, Singer RS and Levin BR. 2002. Antibiotics in agriculture: When is it time to close the barn door? *Proceedings of the National Academy of Sciences*. 99, 5752-5754.

Liu JM, Hankinson SE, Stampfer MJ, Rifai N, Willett WC and Ma J. 2003. Body iron stores and their determinants in healthy postmenopausal US women. *American Journal of Clinical Nutrition*. 2003 78 (6) 1160-1167.

Liu Q, Peng H, Lu X, Zuidhof MJ, Li XF and Le XC. 2016. Arsenic Species in Chicken Breast: Temporal Variations of Metabolites, Elimination Kinetics, and Residual Concentrations. *Environmental Health Perspectives*. 124 (8) 1174-1181.

Liu YY, Wang Y, Walsh TR, Yi LX, Zhang R, Spencer J, Doi Y, Tian G, Dong B, Huang X, Yu LF, Gu D, Ren H, Chen X, Lv L, He D, Zhou H, Liang Z, Liu JH and Shen J. 2016a. Emergence of plasmid-mediated colistin resistance mechanism MCR-1 in animals and human beings in China: a microbiological and molecular biological study. *Lancet Infectious Diseases*. 16 (2) 161-168.

Leitzmann C. 2005. Vegetarian diets: what are the advantages? *Forum on Nutrition*. 57 147-156.

Levine ME, Suarez JA, Brandhorst S, Balasubramanian P, Cheng CW, Madia F, Fontana L, Mirisola MG, Guevara-Aguirre J, Wan J, Passarino G, Kennedy BK, Wei M, Cohen P, Crimmins EM and Longo VD. 2014. Low protein intake is associated with a major reduction in IGF-1, cancer, and overall mortality in the 65 and younger but not older population. *Cell Metabolism*. 19 (3) 407-417.

Lobo V, Patil A, Phatak A and Chandra N. 2010. Free radicals, antioxidants and functional foods: Impact on human health. *Pharmacognosy Reviews*. 4 (8) 118.

Loh YH, Jakszyn P, Luben RN, Mulligan AA, Mitrou PN and Khaw KT. 2011. N-Nitroso compounds and cancer incidence: the European Prospective Investigation into Cancer and Nutrition (EPIC)-Norfolk Study. *American Journal of Clinical Nutrition*. 93 (5) 1053-1061.

Long AN, Atwell CL, Yoo W and Solomon SS. 2012. Vitamin B(12) deficiency associated with concomitant metformin and proton pump inhibitor use. *Diabetes Care*. 35 (12) e84.

Lovati MR, Manzoni C, Gianazza E, Arnoldi A, Kurowska E, Carroll KK and Sirtori CR. 2000. Soy protein peptides regulate cholesterol homeostasis in Hep G2 cells. *Journal of Nutrition*. 130 (10) 2543-2549.

Luca F, Perry GH and Di Rienzo A. 2010. Evolutionary adaptations to dietary changes. *Annual Review of Nutrition*. 30, 291-314.

Lund BM. 2015. Microbiological Food Safety for Vulnerable People. *International Journal of Environmental Research and Public Health*. 12 (8) 10117-10132.

Mangano KM, Walsh SJ, Kenny AM, Insogna KL and Kerstetter JE. 2014. Dietary acid load is associated with lower bone mineral density in men with low intake of dietary calcium. *Journal of Bone Mineral Research*. 29 (2) 500-506.

Mangels R, Messina V and Messina M. 2011. *The dietitian's guide to vegetarian diets: issues and applications*. 3rd ed. Sudbury, MA: Jones and Bartlett Learning.

- Marlow HJ, Hayes WK, Soret S, Carter RL, Schwab ER and Sabaté J. 2009. Diet and the environment: does what you eat matter? *American Journal of Clinical Nutrition*. 89 (5) 1699S-1703S.
- Marrero NM, He FJ, Whincup P and Macgregor GA. 2014. Salt intake of children and adolescents in South London: consumption levels and dietary sources. *Hypertension*. 63 (5) 1026-1032.
- Marsh AG, Sanchez TV, Michelsen O, Chaffee FL and Fagal SM. 1988. Vegetarian lifestyle and bone mineral density. *American Journal of Clinical Nutrition*. 48 (3 Suppl) 837-841.
- Matthews JE, Dickey BW, Miller RD, Felzer JR, Dawson BP, Lee AS, Rocks JJ, Kiel J, Montes JS, Moe CL, Eisenberg JN and Leon JS. 2012. The epidemiology of published norovirus outbreaks: a review of risk factors associated with attack rate and genogroup. *Epidemiology and Infection*. 140 (7) 1161-1172.
- McCarthy K. 2015. The Honourable Member. *Viva!life*. 58 p40.
- McDougall J, Thomas LE, McDougall C, Moloney G, Saul B, Finnell JS, Richardson K and Petersen KM. 2014. Effects of 7 days on an ad libitum low-fat vegan diet: the McDougall Program cohort. *Nutrition Journal*. 13, 99.
- McMahon GM, Hwang SJ, Tanner RM, Jacques PF, Selhub J, Muntner P and Fox CS. 2015. The association between vitamin B12, albuminuria and reduced kidney function: an observational cohort study. *BMC Nephrology*. 16, 7.
- Melkonian SC, Daniel CR, Ye Y, Tannir NM, Karam JA, Matin SF, Wood CG and Wu X. 2016. Gene-environment interaction of genome-wide association study-identified susceptibility loci and meat-cooking mutagens in the etiology of renal cell carcinoma. *Cancer*. 122 (1) 108-115.
- Melnik BC. 2012. Leucine signaling in the pathogenesis of type 2 diabetes and obesity. *World Journal of Diabetes*. 3 (3) 38-53.
- Mendiola J, Torres-Cantero AM, Moreno-Grau JM, Ten J, Roca M, Moreno-Grau S and Bernabeu R. 2009. Food intake and its relationship with semen quality: a case-control study. *Fertility and Sterility*. 91 (3) 812-818.
- Meyers DG, Jensen KC and Menitove JE. 2002. A historical cohort study of the effect of lowering body iron through blood donation on incident cardiac events. *Transfusion*. 42 (9) 1135-1139.
- Micha R, Wallace SK and Mozaffarian D. 2010. Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and meta-analysis. *Circulation*. 121 (21) 2271-2283.
- Micha R, Michas G and Mozaffarian D. 2012. Unprocessed red and processed meats and risk of coronary artery disease and type 2 diabetes—an updated review of the evidence. *Current Atherosclerosis Reports*. 14 (6) 515-524.
- Mintel. 2015. *Number of global vegetarian food and drink product launches doubles between 2009 and 2013* [online]. Available from: www.mintel.com/press-centre/food-and-drink/number-of-global-vegetarian-food-and-drink-product-launches-doubles-between-2009-and-2013 [Accessed 27 October 2015].
- Morino K, Petersen KF, Shulman GI. 2006. Molecular mechanisms of insulin resistance in humans and their potential links with mitochondrial dysfunction. *Diabetes*. 55 (Suppl. 2) S9-S15.
- MRC/BHF. 2006. MRC/BHF Heart Protection Study, questions and answers about cholesterol [online]. Available at: www.hpsinfo.org/fact_chol.shtml [Accessed 17 October 2016].
- Muñoz-Bravo C, Gutiérrez-Bedmar M, Gómez-Aracena J, García-Rodríguez A and Navajas JF. 2013. Iron: protector or risk factor for cardiovascular disease? Still controversial. *Nutrients*. 5 (7) 2384-2404.
- National Creutzfeldt-Jakob Disease Surveillance Unit. 2016. *The National Creutzfeldt-Jakob Disease Surveillance Unit (NCJDSU), University of Edinburgh. Creutzfeldt-Jakob Disease in the UK* [online]. Available from: www.cjd.ed.ac.uk/documents/figs.pdf [Accessed 4 July 2016].
- National Obesity Forum. 2015. *Eat Fat, Cut The Carbs and Avoid Snacking To Reverse Obesity and Type 2 Diabetes* [online]. Available from: <https://phcuk.org/wp-content/uploads/2016/05/Eat-Fat-Cut-The-Carbs-and-Avoid-Snacking-To-Reverse-Obesity-and-Type-2-Diabetes-National-Obesity-Forum-Public-Health-Collaboration.pdf> [Accessed 28 June 2016].
- National Osteoporosis Society. 2016. *What is osteoporosis?* [online]. Available from: www.nos.org.uk/about-osteoporosis [Accessed 16 June 2016].
- Navarrete A, van Schaik CP and Isler K. 2011. Energetics and the evolution of human brain size. *Nature*. 480 (7375) 91-93.
- Nelson M and Poulter J. 2004. Impact of tea drinking on iron status in the UK: a review. *Journal of Human Nutrition and Dietetics*. 17 (1) 43-54.
- NHS Choices. 2014. *Salt – the facts* [online]. Available from: www.nhs.uk/Livewell/Goodfood/Pages/salt.aspx [Accessed 22 February, 2016].
- NHS Choices. 2014a. *The top 5 causes of premature death* [online]. Available from: www.nhs.uk/Livewell/over60s/Pages/The-top-five-causes-of-premature-death.aspx [Accessed 18 April, 2016].
- NHS Choices. 2014b. *Kidney cancer* [online]. Available from: www.nhs.uk/Conditions/cancer-of-the-kidney/Pages/introduction.aspx [Accessed 8 June 2016].
- NHS Choices. 2014c. *Pancreatic cancer* [online]. Available from: www.nhs.uk/conditions/Cancer-of-the-pancreas/Pages/Introduction.aspx [Accessed 9 June 2016].
- NHS Choices. 2015. *Red meat and the risk of bowel cancer* [online]. Available from: www.nhs.uk/Livewell/Goodfood/Pages/red-meat.aspx [Accessed 11 June 2015].
- NHS Choices. 2015a. *Vitamins and minerals – B vitamins and folic acid* [online]. Available from: www.nhs.uk/Conditions/vitamins-minerals/Pages/Vitamin-B.aspx [Accessed 18 January 2015].
- NHS Choices. 2015b. *Lung cancer* [online]. Available from: www.nhs.uk/conditions/Cancer-of-the-lung/Pages/Introduction.aspx [Accessed 19 May 2016].
- NHS Choices. 2015c. *Stomach cancer* [online]. Available from: www.nhs.uk/Conditions/Cancer-of-the-stomach/Pages/Introduction.aspx [Accessed 15 June 2016].
- NHS Choices. 2015d. *Britain: ‘the fat man of Europe’* [online]. Available from: www.nhs.uk/Livewell/loseweight/Pages/statistics-and-causes-of-the-obesity-epidemic-in-the-UK.aspx [Accessed 16 June 2016].
- NHS Choices. 2015e. *Food poisoning* [online]. Available from: www.nhs.uk/conditions/Food-poisoning/Pages/Introduction.aspx [Accessed 22 June 2016].
- NHS Choices. 2015f. *Bird flu (avian flu)* [online]. Available from: www.nhs.uk/conditions/Avian-flu/Pages/Introduction.aspx [Accessed 28 June 2016].
- Niinistö S, Takkinen HM, Uusitalo L, Rautanen J, Vainio N, Ahonen S, Nevalainen J, Kenward MG, Lumia M, Simell O, Veijola R, Ilonen J, Knip M and Virtanen SM. 2015. Maternal intake of fatty acids and their food sources during lactation and the risk of preclinical and clinical type 1 diabetes in the offspring. *Acta Diabetologica*. 52 (4) 763-772.
- Niki E. 2011. Do free radicals play causal role in atherosclerosis? Low density lipoprotein oxidation and vitamin E revisited. *Journal of Clinical Biochemical Nutrition*. 48 (1) 3-7.
- Nunan D. 2016. Important methodological flaws limit the findings from the paper by Ravnskov and colleagues. *BMJ Open*. 6 (6) e010401.

Obeid R, Fedosov SN and Nexo E. 2015. Cobalamin coenzyme forms are not likely to be superior to cyano- and hydroxyl-cobalamin in prevention or treatment of cobalamin deficiency. *Molecular Nutrition and Food Research*. 59 (7) 1364-1372.

Office for National Statistics. 2014. *Cancer Registration Statistics, England: First release: 2014* [online]. Available from: www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/conditionsanddiseases/bulletins/cancerregistrationstatisticsengland/firstrelease2014 [Accessed 11 May 2016].

O'Keefe SJ, Chung D, Mahmoud N, Sepulveda AR, Manafe M, Arch J, Adada H, van der Merwe T. 2007. Why do African Americans get more colon cancer than Native Africans? *J Nutr*. 137 (1 Suppl) 175S-182S.

O'Neill. 2015. Antimicrobials in agriculture and the environment: reducing unnecessary use and waste [online]. Available from: <http://amr-review.org/sites/default/files/Antimicrobials%20in%20agriculture%20and%20the%20environment%20-%20Reducing%20unnecessary%20use%20and%20waste.pdf> [Accessed 4 July 2016].

Orlich MJ, Singh PN, Sabat  J, Jaceldo-Siegl K, Fan J, Knutsen S, Beeson WL and Fraser GE. 2013. Vegetarian dietary patterns and mortality in Adventist Health Study 2. *JAMA Internal Medicine*. 173 (13) 1230-1238.

Ornish D, Weidner G, Fair WR, Marlin R, Pettengill EB, Raisin CJ, Dunn-Emke S, Crutchfield L, Jacobs FN, Barnard RJ, Aronson WJ, McCormac P, McKnight DJ, Fein JD, Dnistrian AM, Weinstein J, Ngo TH, Mendell NR and Carroll PR. 2005. Intensive lifestyle changes may affect the progression of prostate cancer. *Journal of Urology*. 174 (3) 1065-1069.

Ornish D, Lin J, Chan JM, Epel E, Kemp C, Weidner G, Marlin R, Frenda SJ, Magbanua MJ, Daubenmier J, Estay I, Hills NK, Chainani-Wu N, Carroll PR and Blackburn EH. 2013. Effect of comprehensive lifestyle changes on telomerase activity and telomere length in men with biopsy-proven low-risk prostate cancer: 5-year follow-up of a descriptive pilot study. *Lancet Oncology*. 14 (11) 1112-1120.

Pan A, Sun Q, Bernstein AM, Schulze MB, Manson JE, Willett WC and Hu FB. 2011. Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. *American Journal of Clinical Nutrition*. 94 (4) 1088-1096.

Pan A, Sun Q, Bernstein AM, Schulze MB, Manson JE, Stampfer MJ, Willett WC and Hu FB. 2012. Red meat consumption and mortality: results from 2 prospective cohort studies. *Archives of Internal Medicine*. 172 (7) 555-563.

Parslow RC, McKinney PA, Law GR, Staines A, Williams R and Bodansky HJ. 1997. Incidence of childhood diabetes mellitus in Yorkshire, northern England, is associated with nitrate in drinking water: an ecological analysis. *Diabetologia*. 40 (5) 550-556.

Peppas M, Goldberg T, Cai W, Rayfield E, Vlassara H. 2002. Glycotoxins: a missing link in the "relationship of dietary fat and meat intake in relation to risk of type 2 diabetes in men". *Diabetes Care*. 25 (10) 1898-1899.

Petersen KF, Dufour S, Befroy D, Garcia R, Shulman GI. 2004. Impaired mitochondrial activity in the insulin-resistant offspring of patients with type 2 diabetes. *New England Journal of Medicine*. 350 (7) 664-671.

Phillips DH. 1999. Polycyclic aromatic hydrocarbons in the diet. *Mutation Research*. 443 (1-2) 139-147.

Pizzorno J, Frassetto LA, and Katzinger J. 2010. Diet-induced acidosis: is it real and clinically relevant? *British Journal of Nutrition*. 103 (8) 1185-1194.

Public Health England. 2016. PHE response to the National Obesity Forum and Public Health Collaboration opinion paper [online]. Available from: <https://www.gov.uk/government/news/phe-response-to-the-national-obesity-forum-and-public-health-collaboration-opinion-paper>

Public Health England. 2016a. *Health interventions – Cancer* [online]. Available from: <http://healthierlives.phe.org.uk/topic/mortality/health-intervention/cancer#are/E06000023/par/E92000001> [Accessed 18 April 2016].

Public Health England. 2016b. *The Eatwell Guide* [online]. Available from: www.gov.uk/government/publications/the-eatwell-guide [Accessed 24 May 2016].

Purchas RW and Busboom JR. 2005. The effect of production system and age on levels of iron, taurine, carnosine, coenzyme Q(10), and creatine in beef muscles and liver. *Meat Science*. 70 (4) 589-596.

Qi X, Qian YH, Bao CJ, Guo XL, Cui LB, Tang FY, Ji H, Huang Y, Cai PQ, Lu B, Xu K, Shi C, Zhu FC, Zhou MH and Wang H. 2013. Probable person to person transmission of novel avian influenza A (H7N9) virus in Eastern China, 2013: epidemiological investigation. *British Medical Journal*. 347 f4752.

Qi L, van Dam RM, Rexrode K and Hu FB. 2007. Heme iron from diet as a risk factor for coronary heart disease in women with type 2 diabetes. *Diabetes Care*. 30 (1) 101-106.

Ravnskov U, Diamond DM, Hama R, Hamazaki T, Hammarskj ld B, Hynes N, Kendrick M, Langsjoen PH, Malhotra A, Mascitelli L, McCully KS, Ogushi Y, Okuyama H, Rosch PJ, Schersten T, Sultan S and Sundberg R. 2016. Lack of an association or an inverse association between low-density-lipoprotein cholesterol and mortality in the elderly: a systematic review. *BMJ Open*. 6 (6) e010401.

Richman EL, Stampfer MJ, Paoirek A, Broering JM, Carroll PR and Chan JM. 2010. Intakes of meat, fish, poultry, and eggs and risk of prostate cancer progression. *American Journal of Clinical Nutrition*. 91 (3) 712-721.

Richman EL, Kenfield SA, Stampfer MJ, Giovannucci EL and Chan JM. 2011. Egg, red meat, and poultry intake and risk of lethal prostate cancer in the prostate-specific antigen-era: incidence and survival. *Cancer Prevention and Research (Phila)*. 4 (12) 2110-2121.

Richter CK, Skulas-Ray AC, Champagne CM and Kris-Etherton PM. 2015. Plant protein and animal proteins: do they differentially affect cardiovascular disease risk? *Advances in Nutrition*. 6 (6) 712-728.

Roberts WC. 2000. Twenty questions on atherosclerosis. *Proceedings (Bayl Univ Med Cent)*. 13 (2) 139-143.

Roden M, Krssak M, Stingl H, Gruber S, Hofer A, F rnsinn C, Moser E, Waldh usl W. Rapid impairment of skeletal muscle glucose transport/phosphorylation by free fatty acids in humans. 1999. *Diabetes*. 48 (2) 358-364.

Rohrmann S, Linseisen J, Jakobsen MU, Overvad K, Raaschou-Nielsen O, T jnnel nd A, Boutron-Ruault MC, Kaaks R, Becker N, Bergmann M, Boeing H, Khaw KT, Wareham NJ, Key TJ, Travis R, Benetou V, Naska A, Trichopoulou A, Pala V, Tumino R, Masala G, Mattiello A, Brustad M, Lund E, Skeie G, Bueno-de-Mesquita HB, Peeters PH, Vermeulen RC, Jakszyn P, Dorronsoro M, Barricarte A, Tormo MJ, Molina E, Arg uelles M, Melin B, Ericson U, Manjer J, Rinaldi S, Slimani N, Boffetta P, Vergnaud AC, Khan A, Norat T and Vineis P. 2011. Consumption of meat and dairy and lymphoma risk in the European Prospective Investigation into Cancer and Nutrition. *International Journal of Cancer*. 128 (3) 623-634.

Rohrmann S, Overvad K, Bueno-de-Mesquita HB, Jakobsen MU, Egeberg R, T jnnel nd A, Nailler L, Boutron-Ruault MC, Clavel-Chapelon F, Krogh V, Palli D, Panico S, Tumino R, Ricceri F, Bergmann MM, Boeing H, Li K, Kaaks R, Khaw KT, Wareham NJ, Crowe FL, Key TJ, Naska A, Trichopoulou A, Trichopoulos D, Leenders M, Peeters PH, Engeset D, Parr CL, Skeie G, Jakszyn P, S  nchez MJ, Huerta JM, Redondo ML, Barricarte A, Amiano P, Drake I, Sonestedt E, Hallmans G, Johansson I, Fedirko V, Romieu J, Ferrari P, Norat T, Vergnaud AC, Riboli E and Linseisen J. 2013. Meat consumption and mortality—results from the European Prospective Investigation into Cancer and Nutrition. *BMC Medicine*. 11, 63.

Rohrmann S, Linseisen J, N  thlings U, Overvad K, Egeberg R, T jnnel nd A, Boutron-Ruault MC, Clavel-Chapelon F, Cottet V, Pala V, Tumino R, Palli D, Panico S, Vineis P, Boeing H, Pischon T, Grote V, Teucher B, Khaw KT, Wareham NJ, Crowe FL, Goufa I, Orfanos P,

- Trichopoulos A, Jeurnink SM, Siersema PD, Peeters PH, Brustad M, Engeset D, Skeie G, Duell EJ, Amiano P, Barricarte A, Molina-Montes E, Rodríguez L, Tormo MJ, Sund M, Ye W, Lindkvist B, Johansen D, Ferrari P, Jenab M, Slimani N, Ward H, Riboli E, Norat T and Bueno-de-Mesquita HB. 2013a. Meat and fish consumption and risk of pancreatic cancer: results from the European Prospective Investigation into Cancer and Nutrition. *International Journal of Cancer*. 132 (3) 617-624.
- Rohrmann S and Linseisen J. 2015. Processed meat: the real villain? *Proceedings of the Nutrition Society*. 1-9.
- Rohrmann S, Nimptsch K, Sinha R, Willett WC, Giovannucci EL, Platz EA and Wu K. 2015. Intake of Meat Mutagens and Risk of Prostate Cancer in a Cohort of U.S. Health Professionals. *Cancer Epidemiology and Biomarkers Prevention*. 24 (10) 1557-1563.
- Rusher DR and Pawlak R. 2013. A Review of 89 Published Case Studies of Vitamin B12 Deficiency. *Journal of Human Nutrition and Food Science*. 1 (2) 1008.
- Sacks FM, Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Bray GA, Vogt TM, Cutler JA, Windhauser MM, Lin PH and Karanja N. 1999. A dietary approach to prevent hypertension: a review of the Dietary Approaches to Stop Hypertension (DASH) Study. *Clinical Cardiology*. 22 (7 Suppl) III6-10.
- SACN. 2003. *Salt and Health* [online]. Available from: www.gov.uk/government/uploads/system/uploads/attachment_data/file/338782/SACN_Salt_and_Health_report.pdf [Accessed 22 February 2016].
- SACN. 2010. *Iron and Health* [online]. Scientific Advisory Committee on Nutrition. Published by TSO (The Stationery Office) [online]. Available from: www.gov.uk/government/uploads/system/uploads/attachment_data/file/339309/SACN_Iron_and_Health_Report.pdf [Accessed 11 June 2015].
- SACN. 2013. *SACN position statement on selenium and health* [online]. Available from: www.gov.uk/government/uploads/system/uploads/attachment_data/file/339431/SACN_Selenium_and_Health_2013.pdf [Accessed 11 June 2015].
- Salonen JT, Nyyssönen K, Korpela H, Tuomilehto J, Seppänen R, Salonen R. 1992. High stored iron levels are associated with excess risk of myocardial infarction in eastern Finnish men. *Circulation*. 86 (3) 803-811.
- Santarelli RL, Pierre F and Corpet DE. 2008. Processed meat and colorectal cancer: a review of epidemiologic and experimental evidence. *Nutrition and Cancer*. 60 (2) 131-44.
- Saunders AV, Craig WJ, Baines SK and Posen JS. 2013. Iron and vegetarian diets. *Medical Journal of Australia*. 199 (4 Suppl) S11-16.
- Scarborough P, Allender S, Clarke D, Wickramasinghe K and Rayner M. 2012. Modelling the health impact of environmentally sustainable dietary scenarios in the UK. *European Journal of Clinical Nutrition*. 66 (6) 710-715.
- Schüpbach R, Wegmüller R, Berguerand C, Bui M and Herter-Aeberli I. 2015. Micronutrient status and intake in omnivores, vegetarians and vegans in Switzerland. *European Journal of Nutrition*. Oct 26. [Epub ahead of print] p11-11.
- Scialla JJ and Anderson CA. 2013. Dietary acid load: a novel nutritional target in chronic kidney disease? *Advances in Chronic Kidney Disease*. 20 (2) 141-149.
- Sellmeyer DE, Stone KL, Sebastian A and Cummings SR. 2001. A high ratio of dietary animal to vegetable protein increases the rate of bone loss and the risk of fracture in postmenopausal women. *American Journal of Clinical Nutrition*. 73 (1) 118-122.
- Sempos C.T. 2002. Do body iron stores increase the risk of developing coronary heart disease? *American Journal of Clinical Nutrition*. 76, 3, 501-503.
- Sieri S, Krogh V, Pala V, Muti P, Micheli A, Evangelista A, Tagliabue G and Berrino F. 2004. Dietary patterns and risk of breast cancer in the ORDET cohort. *Cancer Epidemiology Biomarkers and Prevention*. 13 (4) 567-572.
- Singh M and Das RR. 2011. Zinc for the common cold. *Cochrane Database Systematic Reviews*. (2) CD001364.
- Sinha R, Rothman N, Brown ED, Salmon CP, Knize MG, Swanson CA, Rossi SC, Mark SD, Levander OA and Felton JS. 1995. High concentrations of the carcinogen 2-amino-1-methyl-6-phenylimidazo-[4,5-b]pyridine (PhIP) occur in chicken but are dependent on the cooking method. *Cancer Research*. 55 (20) 4516-4519.
- Sinha R, Cross AJ, Graubard BI, Leitzmann MF and Schatzkin A. 2009. Meat intake and mortality: a prospective study of over half a million people. *Archives of Internal Medicine*. 169 (6) 562-571.
- Smith DL, Harris AD, Johnson JA, Silbergeld EK and Morris JG Jr. 2002. Animal antibiotic use has an early but important impact on the emergence of antibiotic resistance in human commensal bacteria. *Proceeding of the National Academy of Science*. 99 (9) 6434-649.
- Sparks LM, Xie H, Koza RA, Mynatt R, Hulver MW, Bray GA, Smith SR. 2005. A high-fat diet coordinately downregulates genes required for mitochondrial oxidative phosphorylation in skeletal muscle. *Diabetes*. 54 (7) 1926-1933.
- Springmann M, Godfray HC, Rayner M and Scarborough P. 2016. Analysis and valuation of the health and climate change cobenefits of dietary change. *Proceeding of the National Academy of Sciences*. 113 (15) 4146-4151.
- Spurlock M. 2004. Super size me. New York, NY, Hart Sharp Video.
- Stender S, Dyerberg J, Astrup A. 2007. Fast food: unfriendly and unhealthy. *Int J Obes (Lond)*. 31 (6) 887-890.
- Stern N. 2006. *Stern Review on The Economics of Climate Change Executive Summary* [online]. Available from: www.wwf.se/source.php/1169157/Stern%20Report_Exec%20Summary.pdf [Accessed 6 July 2016].
- Stolzenberg-Solomon RZ, Cross AJ, Silverman DT, Schairer C, Thompson FE, Kipnis V, Subar AF, Hollenbeck A, Schatzkin A and Sinha R. 2007. Meat and meat-mutagen intake and pancreatic cancer risk in the NIH-AARP cohort. *Cancer Epidemiology and Biomarkers Prevention*. 16 (12) 2664-2675.
- Suarez DL. 2000. Evolution of avian influenza viruses. *Veterinary Microbiology* 74 15-27.
- Sullivan JL. 1981. Iron and the sex difference in heart disease risk. *Lancet* 1 (8233) 1293-1294.
- Sun Y, Li Z, Li J, Li Z and Han J. 2016. A Healthy Dietary Pattern Reduces Lung Cancer Risk: A Systematic Review and Meta-Analysis. *Nutrients*. 8 (3).
- Tanabe S, Miyauchi E, Muneshige A, Mio K, Sato C and Sato M. 2007. PCR method of detecting pork in foods for verifying allergen labeling and for identifying hidden pork ingredients in processed foods. *Bioscience, Biotechnology and Biochemistry*. 71 (7) 1663-1667.
- Tantamango-Bartley Y, Jaceldo-Siegl K, Fan J and Fraser G. 2013. Vegetarian diets and the incidence of cancer in a low-risk population. *Cancer Epidemiology and Biomarkers Prevention*. 22 (2) 286-294.
- Tasevska N, Sinha R, Kipnis V, Subar AF, Leitzmann MF, Hollenbeck AR, Caporaso NE, Schatzkin A and Cross AJ. 2009. A prospective study of meat, cooking methods, meat mutagens, heme iron, and lung cancer risks. *American Journal of Clinical Nutrition*. 89 (6) 1884-1894.
- Tasevska N, Cross AJ, Dodd KW, Ziegler RG, Caporaso NE and Sinha R. 2011. No effect of meat, meat cooking preferences, meat mutagens or heme iron on lung cancer risk in the prostate, lung, colorectal and ovarian cancer screening trial. *International Journal of Cancer*. 128, 402-411.

- TEDDY Study Group. 2008. The Environmental Determinants of Diabetes in the Young (TEDDY) Study. *Annals of the New York Academy of Sciences*. 1150 1-13.
- Teicholz N. 2015. The scientific report guiding the US dietary guidelines: is it scientific? *British Medical Journal*. 351 h4962.
- Teicholz N. 2015a. Erratum for: The scientific report guiding the US dietary guidelines: is it scientific? *British Medical Journal*. 351 h4962.
- Temme EH and Van Hooijdonck PG. 2002. Tea consumption and iron. *European Journal of Clinical Nutrition*. 56 (5) 379-386.
- The Carbon Trust. 2016. The Eatwell Guide: a More Sustainable Diet [online]. Available from: www.carbontrust.com/media/672635/phe-sustainable-diets.pdf [Accessed 30 November 2016].
- Thiébaud AC, Jiao L, Silverman DT, Cross AJ, Thompson FE, Subar AF, Hollenbeck AR, Schatzkin A and Stolzenberg-Solomon RZ. 2009. Dietary fatty acids and pancreatic cancer in the NIH-AARP diet and health study. *Journal of the National Cancer Institute*. 101 (14) 1001-1011.
- Thomson CD, Chisholm A, McLachlan SK, Campbell JM. 2008. Brazil nuts: an effective way to improve selenium status. *American Journal of Clinical Nutrition*. 87 (2) 379-384.
- Tran M. 2015. Research criticising 1980s fat guidelines misguided, say scientists. *The Guardian*, 10 February [online]. Available from: www.theguardian.com/lifeandstyle/2015/feb/10/research-criticising-1980s-fat-guidelines-misguided-say-scientists [Accessed 2 December 2015].
- Tucker KL, Rich S, Rosenbergm I, Jacques P, Dallal G, Wilson PW and Selhub J. 2000. Plasma vitamin B-12 concentrations relate to intake source in the Framingham Offspring study. *The American Journal of Clinical Nutrition*. 71 (2) 514-22.
- Tucker KL, Morita K, Qiao N, Hannan MT, Cupples LA and Kiel DP. 2006. Colas, but not other carbonated beverages, are associated with low bone mineral density in older women: The Framingham Osteoporosis Study. *American Journal of Clinical Nutrition*. 84 (4) 936-942.
- Tuomainen TP, Punnonen K, Nyyssönen K and Salonen JT. 1998. Association between body iron stores and the risk of acute myocardial infarction in men. *Circulation*. 97 (15) 1461-1466.
- Turner BL and Thompson AL. 2013. Beyond the Paleolithic prescription: incorporating diversity and flexibility in the study of human diet evolution. *Nutrition Reviews*. 71 (8) 501-510.
- Ungchusak K, Auewarakul P, Dowell SF, Kitphati R, Auwanit W, Puthavathana P, Uprasertkul M, Boonnak K, Pittayawonganon C, Cox NJ, Zaki SR, Thawatsupha P, Chittaganpitch M, Khontong R, Simmerman JM and Chunsutthiwat S. 2005. Probable person-to-person transmission of avian influenza A (H5N1). *New England Journal of Medicine*. 352 (4) 333-340.
- Uribarri J, Woodruff S, Goodman S, Cai W, Chen X, Pyzik R, Yong A, Striker GE and Vlassara H. 2010. *Journal of the American Dietetic Association*. 110 (6) 911-916.
- van der A D, Peeters P, Grobbee D, Marx J and van der Schouw Y. 2004. Dietary haem iron and coronary heart disease in women. *European Heart Journal*. 26 (3) 257-262.
- Vang A, Singh PN, Lee JW, Haddad EH, Brinegar CH. 2008. Meats, Processed Meats, Obesity, Weight Gain and Occurrence of Diabetes among Adults: Findings from Adventist Health Studies. *Annals of Nutrition and Metabolism*. 52 (2) 96-104
- Vehik K, Hamman RF, Lezotte D, Norris JM, Klingensmith GJ, Rewers M and Dabelea D. 2008. Trends in high-risk HLA susceptibility genes among Colorado youth with type 1 diabetes. *Diabetes Care*. 31 (7) 1392-1396.
- Vergnaud AC, Norat T, Romaguera D, Mouw T, May AM, Travier N, Luan J, Wareham N, Slimani N, Rinaldi S, Couto E, Clavel-Chapelon F, Boutron-Ruault MC, Cottet V, Palli D, Agnoli C, Panico S, Tumino R, Vineis P, Agudo A, Rodriguez L, Sanchez MJ, Amiano P, Barricarte A, Huerta JM, Key TJ, Spencer EA, Bueno-de-Mesquita B, Büchner FL, Orfanos P, Naska A, Trichopoulou A, Rohrmann S, Hermann S, Boeing H, Buijsse B, Johansson I, Hellstrom V, Manjer J, Wirfält E, Jakobsen MU, Overvad K, Tjønneland A, Halkjaer J, Lund E, Braaten T, Engeset D, Odysseos A, Riboli E and Peeters PH. 2010. Meat consumption and prospective weight change in participants of the EPIC-PANACEA study. *American Journal of Clinical Nutrition*. 92 (2) 398-407.
- Vergnaud AC, Norat T, Romaguera D, Peeters PHM; EPIC PANACEA. 2010a. Meat intake's influence on body fatness cannot be assessed without measurement of body fat. Reply to A Astrup et al. *Am J Clin Nutr*. 92, 1275-1276.
- Vergnaud AC, Norat T, Mouw T, Romaguera D, May AM, Bueno-de-Mesquita HB, van der A D, Agudo A, Wareham N, Khaw KT, Romieu I, Freisling H, Slimani N, Perquier F, Boutron-Ruault MC, Clavel-Chapelon F, Palli D, Berrino F, Mattiello A, Tumino R, Ricceri F, Rodríguez L, Molina-Montes E, Amiano P, Barricarte A, Chirlaque MD, Crowe FL, Orfanos P, Naska A, Trichopoulou A, Teucher B, Kaaks R, Boeing H, Buijsse B, Johansson I, Hallmans G, Drake I, Sonestedt E, Jakobsen MU, Overvad K, Tjønneland A, Halkjaer J, Skeie G, Braaten T, Lund E, Riboli E and Peeters PH. 2013. Macronutrient composition of the diet and prospective weight change in participants of the EPIC-PANACEA study. *PLoS One*. 8 (3) e57300.
- Virtanen SM, Jaakkola L, Räsänen L, Ylönen K, Aro A, Lounamaa R and Akerblom HK, Tuomilehto J. 1994. Nitrate and nitrite intake and the risk for type 1 diabetes in Finnish children. Childhood Diabetes in Finland Study Group. *Diabetic Medicine*. 11 (7) 656-662.
- Vogiatzoglou A, Smith AD, Nurk E, Berstad P, Drevon CA, Ueland PM, Vollset SE, Tell GS and Refsum H. 2009. Dietary sources of vitamin B-12 and their association with plasma vitamin B-12 concentrations in the general population: the Hordaland Homocysteine Study. *American Journal of Clinical Nutrition*. 89 (4) 1078-1087.
- Walczyk T, Muthayya S, Wegmüller R, Thankachan P, Sierksma A, Frenken LG, Thomas T, Kurpad A, Hurrell RF. 2014. Inhibition of iron absorption by calcium is modest in an iron-fortified, casein- and whey-based drink in Indian children and is easily compensated for by addition of ascorbic acid. *Journal of Nutrition*. 144 (11) 1703-1709.
- Wang H, Feng Z, Shu Y, Yu H, Zhou L, Zu R, Huai Y, Dong J, Bao C, Wen L, Wang H, Yang P, Zhao W, Dong L, Zhou M, Liao Q, Yang H, Wang M, Lu X, Shi Z, Wang W, Gu L, Zhu F, Li Q, Yin W, Yang W, Li D, Uyeki TM and Wang Y. 2008. Probable limited person-to-person transmission of highly pathogenic avian influenza A (H5N1) virus in China. *Lancet*. 371 (9622) 1427-1434.
- Wang Y and Beydoun MA. 2009. Meat consumption is associated with obesity and central obesity among US adults. *International Journal of Obesity*. 33 (6) 621-628.
- Wang Y, Lehane C, Ghebremeskel K and Crawford MA. 2010. Modern organic and broiler chickens sold for human consumption provide more energy from fat than protein. *Public Health Nutrition*. 13 (3) 400-408.
- Wang Z, Klipfell E, Bennett BJ, Koeth R, Levison BS, Dugar B, Feldstein AE, Britt EB, Fu X, Chung YM, Wu Y, Schauer P, Smith JD, Allayee H, Tang WH, DiDonato JA, Lusis AJ and Hazen SL. 2011. Gut flora metabolism of phosphatidylcholine promotes cardiovascular disease. *Nature*. 472 (7341) 57-63.
- Ward MH, Cross AJ, Abnet CC, Sinha R, Markin RS and Weisenburger DD. 2012. Heme iron from meat and risk of adenocarcinoma of the esophagus and stomach. *European Journal of Cancer Prevention*. 21 (2) 134-138.
- Watanabe F, Abe K, Fujita T, Goto M, Hiemori M and Nakano Y. 1998. Effects of microwave heating on the loss of vitamin B(12) in foods. *Journal of Agriculture and Food Chemistry*. 46 206-210.
- Watanabe F. 2007. Vitamin B12 sources and bioavailability. *Experimental Biology and Medicine* (Maywood). 232 (10) 1266-1274.
- Watanabe F, Yabuta Y, Bito T and Teng F. 2014. Vitamin B -containing plant food sources for vegetarians. *Nutrients*. 6 (5) 1861-1873.

- WCRF/AICR. 2007. Second Expert Report, Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective [online]. Available from: <http://wcrf.org/sites/default/files/Second-Expert-Report.pdf> [Accessed 17 October 2016].
- WCRF/AICR. 2016. *Continuous Update Project Report: Diet, Nutrition, Physical Activity and Stomach Cancer* [online]. Available from: www.wcrf.org/sites/default/files/Stomach-Cancer-2016-Report.pdf [Accessed 15 June 2016].
- Weikert C, Walter D, Hoffmann K, Kroke A, Bergmann MM and Boeing H. 2005. The relation between dietary protein, calcium and bone health in women: results from the EPIC-Potsdam cohort. *Annals of Nutrition and Metabolism*. 49 (5) 312-318.
- WHO/FAO. 2002. *Diet, nutrition and the prevention of chronic diseases*. [online] Available from: www.who.int/nutrition/topics/4_dietnutrition_prevention/en/index1.html [Accessed 14 October 2013].
- WHO. 2003. *Global strategy on diet, physical activity and health* [online] Available from: www.who.int/dietphysicalactivity/media/en/gsf_general.pdf [Accessed 16 June 2016].
- WHO/IARC. 2010. IARC Monographs on the evaluation of carcinogenic risks to humans Volume 94. *Ingested nitrate and nitrite and cyanobacterial peptide toxins* [online]. Available from: <http://monographs.iarc.fr/ENG/Monographs/vol94/mono94.pdf> [Accessed 26 April 2016].
- WHO/IARC. 2015. IARC Monographs evaluate consumption of red meat and processed meat [online]. Available from: www.iarc.fr/en/media-centre/pr/2015/pdfs/pr240_E.pdf [Accessed 1 December 2015].
- WHO/IARC. 2015a. Q&A on the carcinogenicity of the consumption of red meat and processed meat [online]. Available from: www.iarc.fr/en/media-centre/iarcnews/pdf/Monographs-Q&A_Vol114.pdf [Accessed 15 June 2016].
- WHO. 2006. H5N1 avian influenza in domestic cats [online]. Available from: www.who.int/csr/don/2006_02_28a/en/ [Accessed 30 June 2016].
- WHO. 2016. *Influenza at the human-animal interface Summary and assessment, 9 May to 13 June 2016* [online]. Available from: www.who.int/influenza/human_animal_interface/Influenza_Summary_IRA_HA_interface_06_13_2016.pdf?ua=1 [Accessed 29 June 2016].
- Willett W, Sacks F and Stampfer M. 2014. *Dietary Fat and Heart Disease Study is Seriously Misleading* [online]. Available from: www.hsph.harvard.edu/nutritionsource/2014/03/19/dietary-fat-and-heart-disease-study-is-seriously-misleading [Accessed 11 June 2015].
- World Bank. 2015. *GDP per capita* [online]. Available from: <http://data.worldbank.org/indicator/NY.GDP.PCAP.CD> [Accessed 2 October 2015].
- Xue XJ, Gao Q, Qiao JH, Zhang J, Xu CP and Liu J. 2014. Red and processed meat consumption and the risk of lung cancer: a dose-response meta-analysis of 33 published studies. *International Journal of Clinical Experimental Medicine*. 7 (6) 1542-1553.
- Yang WS, Wong MY, Vogtmann E, Tang RQ, Xie L, Yang YS, Wu QJ, Zhang W and Xiang YB. 2012. Meat consumption and risk of lung cancer: evidence from observational studies. *Annals of Oncology*. 23 (12) 3163-3170.
- Yao L, Huang L, He Z, Zhou C and Li G. 2013. Occurrence of arsenic impurities in organoarsenics and animal feeds. *Journal of Agriculture and Food Chemistry*. 61, 320-324.
- You and Henneberg. 2016. Meat consumption providing a surplus energy in modern diet contributes to obesity prevalence: an ecological analysis. *BMC Nutrition*. 2 (1).
- You and Henneberg. 2016a. Meat in modern diet, just as bad as sugar, correlates with worldwide obesity: An ecological analysis. *Journal of Nutrition & Food Sciences*. 6 (4) 517.
- Zhang S, Hunter DJ, Rosner BA, Colditz GA, Fuchs CS, Speizer FE and Willett WC. 1999. Dietary fat and protein in relation to risk of non-Hodgkin's lymphoma among women. *Journal of the National Cancer Institute*. 91: 1751-1758.
- Zhang Q, Ma G, Greenfield H, Zhu K, Du X, Foo LH, Hu X and Fraser DR. 2010. The association between dietary protein intake and bone mass accretion in pubertal girls with low calcium intakes. *British Journal of Nutrition*. 103 (5) 714-723.
- Zhang J, Dhakal IB, Zhao Z, Li L. 2012. Trends in mortality from cancers of the breast, colon, prostate, esophagus, and stomach in East Asia: role of nutrition transition. *Eur J Cancer Prev*. 21 480-489.
- Zheng H, Cable R, Spencer B, Votto N, Katz SD. 2005. Iron stores and vascular function in voluntary blood donors. *Arterioscler Thromb Vasc Biol*. 25 (8) 1577-1583.
- Zoncu R, Efeyan A and Sabatini DM. 2011. mTOR: from growth signal integration to cancer, diabetes and ageing. *Nature Reviews Molecular Cell Biology*. 12 (1) 21-35.

This report reveals the ground-breaking scientific evidence that meat offers no health benefits and is linked to heart disease, diabetes, cancer and many other diseases.

It examines the links between red, processed and white meat and disease. It explains why meat causes food poisoning and its links with BSE, bird flu and antibiotic-resistant superbugs – and how they pose a threat we can't afford to ignore.

The meat industry has money and influence and is prepared to cloud the issue in a similar way to the tobacco industry. However, with pressure from groups like Viva! and reports such as this, public health guidelines are slowly beginning to change. The research is unequivocal and set out here so you have no need to wait, see the evidence for yourself.

"This report reveals what the meat industry doesn't tell you. Find out what substances in chicken, beef, pork and lamb are linked to our biggest killers – heart disease, diabetes and cancer. It will leave you in no doubt about the harm meat does and explains why going meat-free is one of the best things you can do if you want to lead a long and healthy life."

Dr Michael Greger, physician, author and international speaker on nutrition, food safety and public health and author of *How Not to Die*.



Published by: Viva!Health, 8 York Court,
Wilder Street, Bristol BS2 8QH
T: 0117 944 1000
E: health@viva.org.uk
W: www.vivahealth.org.uk
©Viva!Health 2017
Registered charity 1037486

Meat the Truth
© Viva! 2017
£10

ISBN 978-0-9571873-5-1



9 780957 187351