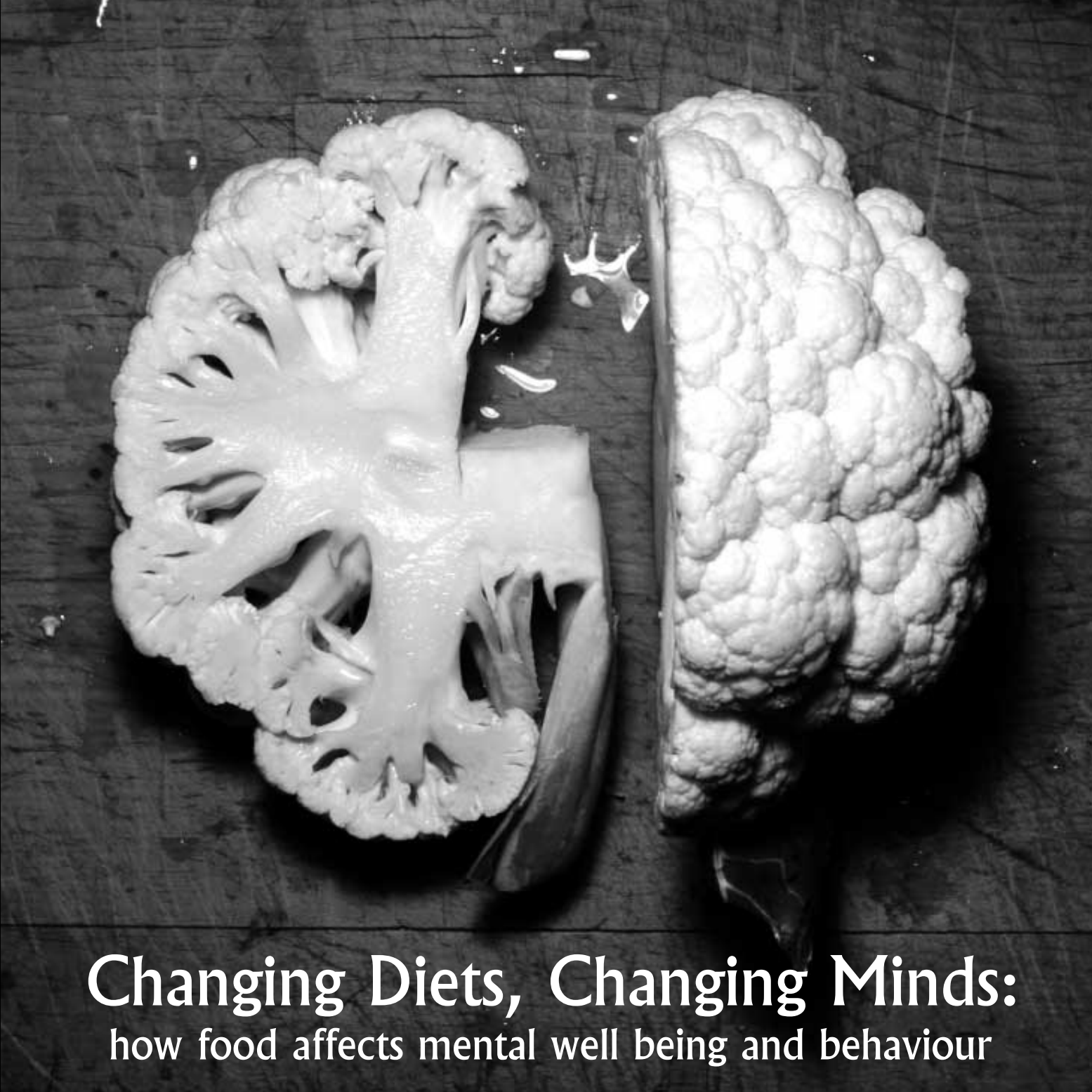

Changing Diets, Changing Minds:

how food affects mental well being and behaviour

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The views expressed in this publication are not necessarily the views of those acknowledged or of Sustain's membership, individually or collectively. All errors and omissions are entirely those of the author.

* There is a separate companion report to this volume, produced by Sustain's partner in the Food and Mental Health Project, the Mental Health Foundation entitled *Feeding Minds: the impact of food on mental health*. The report can be found at www.mentalhealth.org.uk.

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Written by Courtney Van de Weyer

Winter 2005

Contents

An important note on the nature of this report

This report aims to make accessible to a varied audience the breadth and depth of research that appears to demonstrate a link between diet and mental health, and to place that evidence in the context of a changing food system.

As such, it tries to pull together information on a variety of specialist subjects - from foetal brain growth to Alzheimer's disease - into one document, to provide a good overview of the issue. Due to this variety, it has been impossible to go into each subject in as much detail as some readers would like, and some related subjects - such as dyslexia - we have been unable to cover at all.

All the research studies that have been referenced have been accepted for publication in scientific journals. However, this is not a systematic review of all the literature, nor is the report intended primarily for a scientific audience. We accept that there are differences in the quality of studies included, even though they have been published, and have tried to distinguish between those that should be given greater credence and those that should be read with more care.

However, we have not systematically weighted each study according to its methods or size. Our intention is that the document should be assessed as a whole, to demonstrate the breadth and depth of research, and not as a scientific review of particular studies or particular issues. We fully acknowledge that more research is needed, and this is reflected in our recommendations.

We also accept that, while we have aimed for a balance between those studies that have found a connection between diet and mental health, and those that have not, we have focused more on the research that demonstrates a link between diet and mental health.

Scientifically trained readers may take issue with some aspects of the report - including the use of some terms in their common usage, and not their technical medical definition. We emphasise that this is because we hope this report will be widely accessible, particularly to those beyond the scientific community.

Non-scientific readers may not be aware of the reasons why food supplements are referred to so frequently. Because of the nature of controlled studies, with their need for double-blind conditions, it is often difficult or impossible to test food itself. Therefore, supplements are often used as a replacement. Although we happily acknowledge the obvious validity of their use in trials, it is important to state clearly that Sustain does not support the use of food supplements as an alternative to eating a healthy diet.

Foreword

Over thirty years ago, the scientist Michael Crawford warned that the explosion in the prevalence of heart disease seen in the west during the 20th century would soon be followed by a rise in mental health problems. Reasoning that the nutrients necessary for keeping a healthy heart were the same as those needed for a healthy brain, he predicted that the dietary causes of poor heart health would inevitably lead to increased problems in brain and mental health.

Michael was a pioneer of such research in those days, and his work is now finally gaining increasing and well-deserved recognition, both domestically - he is now a Professor of the Institute of Brain Chemistry and Human Nutrition - and internationally. Unfortunately, the warnings from Professor Crawford and the growing number of colleagues working in this important field, are still not being heeded by policy makers, and his predictions are coming true. Depression is now the leading cause of disability worldwide and mental health problems presently cost £100 billion each year in the UK alone.

Mental health is all too often a neglected subject and, until now, it has been completely neglected by those working on food policy issues. Although common sense tells us that what we eat will affect the way we feel, there is resistance to the idea that diet affects mental health. I well remember similar resistance, some 20 years ago, to the idea that our poor quality diets were contributing to our high rates of coronary heart disease.

This reluctance to accept what is now obvious seems bizarre, with the benefit of 20 years of hindsight. But we simply cannot wait another 20 years to reach the same position with food and mental health. If we leave current trends to continue unchecked over that period, the costs of mental illness will probably far exceed the already huge costs of physical illness. Worse still, if we do not address the fact that our food and farming system is environmentally unsustainable, then we may lose the means to prevent much diet-related ill health. In particular, fish stocks in some parts of the world have already been exhausted, and many other are on the brink of collapse.

We can prevent these potentially catastrophic futures. I hope this report marks a point where our efforts to do so begin to accelerate.

Professor Tim Lang
Professor of Food Policy, City University and former Chair of Sustain: the alliance for better food and farming

Summary

The brain is the largest organ in the body and, like our hearts, livers and other organs, it is affected by what we eat and drink. However, unlike with other organs, the links between diet and the brain - and hence how we think and act - are not yet recognised by government and most health professionals. This report - and its companion volume - aims to change that situation.

Sustain: the alliance for better food and farming, working with the Mental Health Foundation, has collected and analysed around 500 research studies, published in peer-reviewed scientific journals, to demonstrate the breadth and depth of the evidence linking what we eat with mental well-being and behaviour.

Despite the large number and generally good quality of the research studies, scientific understanding of these links is far from complete, but it is already clear that our diets affect how our brains are made and how they work, throughout our lives. Some periods, though, are more critical than others for brain development.

For example, 70% of the energy absorbed by a foetus during pregnancy is directed towards brain development. Other research has focused on the impact of diet at different life stages right up to, and including, old age. The significance of diet for mental health and well-being varies, but there appears to be no point at which diet has no effect.

There are some important nutrients for brain development and function, but they can only work properly if a wide range of other nutrients are also available in the right amounts and in proportion to each other. There is no "magic bullet" or single nutrient that holds the key to mental health and well-being.

The combination of nutrients that is most commonly associated with good mental health and well-being is as follows:

- polyunsaturated fatty acids (particularly the omega 3 types found in oily fish and some plants);
- minerals, such as zinc (in whole grains, legumes, meat and milk), magnesium (in green leafy vegetables, nuts and whole grains), and iron (in red meat, green leafy vegetables, eggs and some fruit); and
- vitamins, such as folate (in green leafy vegetables and fortified cereals), a range of B vitamins (whole grain products, yeast and dairy products), and anti-oxidant vitamins such as C and E (in a wide range of fruit and vegetables).

This is, of course, the same type of healthy balanced diet that is widely recommended to reduce our risk of developing coronary heart disease, strokes, a range of cancers, diabetes and a number of digestive disorders and conditions.

People eating diets that lack one or more of this combination of polyunsaturated fats, minerals and vitamins, and/or contain too much saturated fat (or other elements, including sugar and a range of food and agricultural chemicals) seem to be at higher risk of developing the following conditions:

- Attention-deficit/hyperactivity disorder (ADHD)
- A range of depressive conditions
- Schizophrenia
- Dementia, including Alzheimer's disease

The evidence so far does **not** show that these conditions can be prevented or cured by diet alone. However, evidence is accumulating that the combination of polyunsaturated fats, minerals and vitamins may help to:

- relieve the symptoms of some mental illnesses;
- improve the effectiveness of medication for some conditions; and
- reduce the unpleasant side-effects of some medications.

The diet that would give us the right amount and balance of these nutrients would contain lots of different vegetables and fruit, a wide variety of whole grains, nuts, seeds and legumes, and some occasional oily fish, lean meat and dairy products. It makes evolutionary sense that this type of diet would be good for both our physical and mental health.

When food supplies were plentiful, our ancestors would have eaten broadly this kind of diet. Unfortunately that is not what most of us are eating now. The agricultural and industrial revolutions, followed by the globalisation of world food trade mean that most people in rich countries (and growing proportions in poor countries) eat:

- small amounts of a few types of vegetables and fruit;
- very few whole grain products - our carbohydrates are mostly refined (such as sugar and products made from white flour) - and from very narrow range of cereals (90% of the calories from cereals eaten in the UK are from wheat);
- very little oily fish, but large quantities of intensively produced meat, meat products and dairy products;
- unknown (and possibly unknowable) combinations of food and agricultural chemicals, either as intentional additives or accidental residues.

As a result, instead of our diets providing a healthy combination of polyunsaturated fats, minerals and vitamins, we are eating too much saturated fat, sugar and salt and not enough vitamins and minerals. Our analysis of the research indicates that this diet is fuelling not only obesity, cardiovascular disease, diabetes and some cancers, but may also be contributing to rising rates of mental ill-health and anti-social behaviour.

The personal costs of mental ill-health are already too high. The costs to society as a whole are high - the latest estimate is £100 billion in the UK alone - and rising. This report concludes by urging government to tackle, with renewed urgency, our unhealthy and unsustainable food and farming systems. The changes we are calling for will have negligible risks and enormous benefits to physical and mental health and well-being. The consequences of failing to make these changes could be almost too terrible to contemplate.

Introduction

Much attention is currently being paid to the growing crisis of obesity. Throughout the industrialised world and, increasingly, in non-industrialised countries people are eating too much unhealthy food and exercising too little. The result is a staggering rise in the number of individuals, many of them children, whose weight far exceeds what is healthy. In turn, as has been well documented, unhealthy lifestyles are contributing to the increase in diseases such as diabetes and diet-related cancers.

Exercise is an important factor, but recent changes in the amount and types of food eaten are root causes of these sudden and dramatic changes in patterns of disease.

At the same time, the world is also facing another less visible health crisis - this one in the realm of mental health. Like obesity, mental and behavioural disorders are of increasing and serious public health concern. However, unlike obesity, mental health receives very little public attention. There is also very little awareness of the growing evidence that the same factors responsible for obesity, (i.e. too much and the wrong kinds of food), may also be partly responsible for this increase in mental and behavioural problems.

Disorders affecting mental health are very common and affect all societies and all ages. Worldwide, 450 million people suffer from mental or neurological disorders or from psychological problems related to alcohol or drug abuse.[1] These conditions include depressive disorders, bipolar affective disorder ("manic depression"), schizophrenia, Alzheimer's disease and obsessive compulsive disorder.

Representing four of the leading ten causes of disability worldwide, mental health problems affect more than 25 per cent of all people at some point in their lives. At any one time, about 10 per cent of the adult population is suffering from a mental or behavioural disorder. It is also estimated that one family in four is affected by a member with a mental or behavioural problem.[2]

Like obesity, the problem is increasing. Presently, mental health problems account for 12 per cent of the global burden of disease, with depression being the leading cause of disability worldwide. Five other mental health conditions figure in the top 20 causes of disability in the world. At current rates, it is predicted that depression will become the second highest cause of the global disease burden within the next 15 years.[3]

One way to measure the impact of diseases is to use the DALY system – one Disability Adjusted Life Year is one lost year of "healthy" life. This measure is used by bodies such as the World Health Organisation to assess the impact not only of early deaths, but also of lost working years among adult populations – both on human health and on economic health, especially in poorer countries. In 1990, mental and neurological disorders (these include epilepsy) accounted for 10.5 per cent of the total DALYs lost due to all diseases and injuries. In 2000, this had risen to 12.3 per cent and projections indicate that, by 2020, it will rise to 15 per cent.[4]

The cost of mental illness

This has profound implications. Not only does this result in the suffering of individuals and the consequent detrimental effect on families; the economic cost to society is serious. The direct cost of medical treatment is already high, but when the indirect cost of lost productivity is calculated, the price of mental health problems is enormous.[a]

The cost of mental ill-health in the UK, including everything from lost productivity to informal care, is currently approaching £100 billion[5] per year. £28.3 billion of this is due to losses to the economy, as many

a However, the indirect costs calculated are always underestimates, as lost opportunity costs to individuals and families are not quantified.

adults with mental health problems do not hold jobs. Moreover, one study in the UK showed that expenditure on inpatient mental health services was 22 per cent of the total healthcare spending.[6]

The cost to industry is also enormous: over 91 million working days are lost to mental health problems each year, costing companies £4 billion annually[b].[7]

There is no clear consensus as to why such an increase is happening. Suggestions have included increased recognition and diagnosis, the "stress of modern living" and greater isolation from family and other support networks.

The role of food?

However, there is also now a growing canon of research showing a relationship between what a person eats and the way that person feels or acts. Evidence points to links not only between nutrition and day-to-day mood fluctuations, but also more severe mental illness and behavioural problems. Taken in its entirety, our review of the evidence suggests that, similar to obesity, too many people eating the wrong kind of food could be a contributing factor in the rise of mental and behavioural problems.

Anecdotal evidence of the connection between diet and brain function has been present and growing for many years. Beyond grandmotherly advice on 'brain food', a number of health professionals, parents, teachers and patient organisations have repeatedly asserted that changes to diet are mirrored in mental health and behavioural changes. Initially, many of the claims were made about children's behavioural reaction to food additives, but now it is much broader – research into depression, schizophrenia, dementia and anti-social behaviour have all implicated diet as a factor in the progression and treatment of these conditions.

Although it is true that mental and behavioural disorders are highly complicated, involving a combination of biological, psychological and social factors, there is also a growing understanding that mental health has a physical basis in the brain. Indeed, widely accepted and promoted pharmacological treatments work by altering physical attributes of the brain and disorders are often attributed to physical and chemical malfunctions.

This begs the question – what may be causing the physical fault? The brain is an organ, just like the heart, stomach or liver – and whatever affects these organs can also affect the brain. Nutrients consumed in the

b This may also be an underestimate, as employees may prefer to say they are suffering from stress, rather than the more-stigmatised depression.

diet are some of the most consistent physical influences on the body. Whether someone is healthy or not can often (and increasingly) be traced back to the types, amount and nutritional quality of the food they eat.

Resistance to the link

However, there has been fairly consistent resistance from many in the established medical community and government (as well as the manufacturers of nutritionally-denuded products) to the idea that what a person eats affects how he or she feels or behaves. Evidence for this can be seen in the lack of reference to the diet-brain connection, or to groups highlighting the issue, in government publications and policy documents.[c]

Some scepticism about the links between nutrition and mental or behaviour is not surprising, as the evidence quoted has often been based on relatively small studies and individual case histories. For medical and official opinion to shift, larger trials producing consistent data are needed. Fortunately, these are now starting to appear.

Nonetheless, it is important to look at the full body of data that has already accumulated, not only as a basis for further work, but also to indicate what could and should be done now on the basis of existing evidence. After all, the acceptance of causal factors in diet-related health is a gradual process – it is only surprisingly recently that diet was recognised as a factor in coronary heart disease, and only then after many years of research and campaigning.

The purpose of this report

Many research scientists have worked over many years on a wide range of diet-brain related topics. Indeed, some of the first were the very scientists involved in discovering and defining vitamins – more often than not, the very first symptom of a vitamin deficiency is psychological.

Hundreds of studies linking diet to mental health and behaviour have been published in peer-reviewed medical journals. Many of these have tested patients' response to dietary changes in randomised controlled trials, considered to be the 'gold standard' for authoritative clinical evidence. Others have looked more

c For example, the most recent Department of Health directory of mental health organisations, *Contact: a directory for mental health 2005* fails to mention a single organisation that draws attention to the role of diet in mental health. (For a list of these organisations, see page 97).

generally at population levels of mental illness and food intake. It is important to note that the report does not claim that any one study 'proves' anything – but, although not all studies have delivered positive results, the sheer number and quality of those that have found a link between what we eat and how we feel should merit both public and policy attention.

It is also vital to emphasise that this report does not seek to suggest that poor diets are the causal factor in all mental health problems, behavioural disorders or mood fluctuations. However, our review of the evidence suggests that nutrition is a highly plausible and important contributory factor in both the cause and treatment of such conditions. Moreover, it also suggests that the dramatic change in the way food is produced and consumed, witnessed over recent decades, is a plausible explanation for the rise of mental health and behavioural problems over the same period.

The first and second sections of this report present the evidence linking diet throughout a person's life to brain health and general mental well-being. Our review of the evidence begins with the way nutrients influence physical brain development in early life; discusses how day-to-day mood and well-being are affected by what we eat, and concludes with how older brains are affected by a lifetime's nutrition.

The third section examines the evidence linking diet to four specific disorders – Attention Deficit Hyperactivity Disorder (ADHD), depressive affective disorders, schizophrenia and Alzheimer's disease.

The fourth chapter sets out how and why diets have altered so dramatically over recent decades. It presents an overview of how our food has changed beyond recognition over recent years, outlining the changes in agricultural and industrial production that have contributed to reduced availability of the very nutrients that appear to support brain nutrition, and increased the consumption of nutrients that seem likely to undermine mental health.

In the final chapter, we contend that improvements in diet would be beneficial to such a large proportion of the population that action is now needed at a national level to improve the situation. Our review of the evidence of the links between diet and mental health should, at the very least, lend weight to current government and Food Standards Agency initiatives encouraging the food industry to improve the nutritional value of processed foods. We also hope that it will stimulate government into reviewing food and farming policies to ensure that these work in favour of the best possible human nutrition. In the final section, we therefore also recommend what government could be doing to ensure that the nutritional quality of our food supply is improved, to contribute to the health, both physical and mental, of the nation.

The science of nutrition and the brain

In order to understand how food might affect mental health and behaviour, it is useful to understand how food physically affects the brain. The following chapter details the science behind how nutrients affect the brain, aimed at readers with no background in the area. More experienced readers may wish to proceed directly to the next chapter.

The food we eat contains nutrients that are necessary for the human body to grow, repair itself and sustain life. In fact, the human body can be seen as a synthesis of the nutrients it takes in through food, from the moment of conception until death. The healthy growth of a foetus in the womb is highly dependent on whether it receives adequate nutrition during gestation and, when the baby is born and grows towards adulthood, what he or she eats will have a profound impact on his or her growth and health.

What do we need to eat?

Although a cliché, humans literally are what they eat. The composition and chemical processes of the human body are highly dependent on what nutrients a person consumes from food. The following provides only a brief summary of basic human nutrition, in order to set the scene for our discussion of the links between diet and mental health and well-being.

A human body weighing 65 kg comprises:

- 61.6% water,
- 17% protein,
- 13.8% fats,
- 6.1% minerals and
- 1.5% carbohydrates.[4]

It is estimated that the human body requires between 40 and 50 nutrients to function properly.[1] These can be divided into the following categories: water, carbohydrates, fats, proteins, minerals, vitamins, and indigestible nutrients such as fibre[2]. The larger components of the diet are described as macronutrients (carbohydrates, fats and proteins); the smaller components are described as micronutrients (minerals and vitamins).[a] Nutrients provide the building blocks of the body, as well as supplying the energy it needs to function.[3]

Proteins

The type and quality of proteins consumed in the diet emerged as an important factor in our review of evidence for the links between diet and mental health. We therefore draw attention to the "essential" amino acids that must be consumed in the diet, because they cannot be made in the body, in order to inform the later discussion.

a There are also many other substances that exist in food that may be useful to or necessary for the human body, but little is known about them. For example, there are hundreds of chemical compounds in coffee, including caffeine, but only about twenty of them have been studied. It seems likely that "basic" nutritional information will increase as more studies are done.

Proteins, found in a wide range of foods, are necessary for the growth and repair of the body, as they are the main constituents of the body's cells and tissues. Proteins are made up of chains of amino acids most of which are manufactured within the human body. However, there are eight (nine for children) that are called "essential" because the body cannot make them; they can only be found in food.[5] Because there are many types of amino acids, there are many types of proteins – some of which are a better biological "quality" than others, depending on how many of the essential amino acids they contain.

Dietary fats

The type and quality of fats consumed in the diet emerged as an especially important factor in our review of evidence for the links between diet and mental health. We therefore describe the different types of fat here in some detail, to inform the later discussion.

Dietary fats (also called lipids) take two forms: structural fat and storage fat. Structural fats are vital for cell membranes and other cellular tissue, while storage fat stockpiles fuel for the body.[6]

Dietary fats are built from fatty acids, which are either saturated or unsaturated. The unsaturated fats, in their turn, can be categorised further as polyunsaturated and mono-unsaturated fatty acids (see box).

Saturated and unsaturated fats

There are several different types of fat which, depending on their molecular construction, have important differences in their chemical properties. Fats are made from a combination of other molecules, including fatty acids. A fatty acid is a string of carbon atoms, each attached to hydrogen atoms, with a weak acid molecule at one end.

A fatty acid can either be 'saturated' (where each carbon atom's possible bonds are taken up with a hydrogen atom), or 'unsaturated' (where some of those bonds are shared doubly with the adjoining carbon atom). If there are more than two of these 'double bonds' it is described as a polyunsaturated fatty acid (PUFA). If there are more than four, it is described as highly unsaturated (HUFA).

Although these differences appear to be very small, the double bonds change considerably the characteristics and dietary effects of the fats that the fatty acids create. Saturated fatty acids are rigid, while unsaturated fatty acids are more flexible. Most fats will possess a range of fatty acids, and take their properties from whichever acid is more dominant. If a fat has a high proportion of saturated fats, it will be hard and essentially solid (e.g. butter). If there are more unsaturated fats, it will be softer and more liquid (e.g. olive oil). The lower the saturation, the more flexible or fluid the fat.

There is a great deal of evidence that diets high in saturated fat can increase the risk of a range of diseases, particularly coronary heart disease.

All fats have a mixture of saturated and unsaturated fatty acids – in different proportions depending on the source of the fat. Fat from land animals tends to have a higher proportion of saturated fats, while fat from plants and some fish oils have a higher proportion of unsaturated fats.[7]

The polyunsaturated fats (PUFAs) include the "essential fatty acids" (EFAs), linoleic acid and linolenic acid. These are referred to as structural fats because they are the main component in cell walls. Like essential amino acids (protein) they cannot be made by the body but must be derived from the diet.[8]

Carbohydrates

Carbohydrates come in two forms: sugars ('simple' carbohydrates – monosaccharides and disaccharides) and starches ('complex' carbohydrates – polysaccharides). They are burned during metabolism to provide the body with energy.

Micronutrients – vitamins and minerals

In our review of the evidence for the links between diet and mental health, certain minerals and vitamins emerged as making a crucial contribution to brain function.

The micronutrients – minerals and vitamins – perform a number of essential functions. The main minerals in the human body are calcium, chlorine, cobalt, copper, fluorine, iodine, iron, magnesium, manganese, phosphorus, potassium, selenium, sodium, sulphur, and zinc. Some of these are found in large amounts within the body, while some are required only in very small amounts (these are called 'trace elements').[9] Minerals can form the basis of bodily tissue (e.g. calcium for bones), provide the essential elements of hormones (e.g. iodine for the thyroid) and assist with vital bodily functions (e.g. iron for healthy blood).[10]

Some of the more important vitamins include A (or retinol), B1 (thiamine), B2 (riboflavin), B6, B12, C (ascorbic acid), D, E, K, folic acid, niacin (nicotinic acid), biotin, and pantothenic acid.[11] All vitamins, with the exception of vitamin E (which the body is able to synthesise), must be derived from the diet.

How the nutrients are used

When food is eaten, it is broken down by the digestive process into its constituent parts so that nutrients can be absorbed or used for energy. For example, if fat is ingested, it will be broken down into its different fatty acids, which may then be converted (or 'elongated') into the types of fatty acids the body needs. Similarly, protein will be broken down into its different amino acids. These are then available to perform a wide range of processes in the body – including acting as a precursor to other metabolic processes.[12]

A very important point is that, even though every nutrient has a specific function, no nutrient ever works completely alone – this is known as the principle of nutrient interaction.[13] *Anything that a nutrient does, it does with the assistance of a series of other processes, only made possible by the presence of other nutrients. It is vital that this is taken into account particularly in the light of studies, detailed later, which test specific nutrients in isolation.*

How do nutrients physically affect the brain?

In order to understand how diet may affect mood and behaviour, it is first necessary to understand how the nutrients described above physically affect the brain.

How the brain works

The brain, and the rest of the nervous system, is partially constructed from billions of nerve cells, called neurons. Communication between these neurons allows the brain to 'work' – the communication taking the form of electrical or chemical signals between brain cells, using a complex and unique process to facilitate the passing of information throughout the nervous system.

The chemicals that carry the signals are called neurotransmitters. When a neurotransmitter chemical is released, it stimulates the target cell, which then continues the signalling process. Its job done, the neurotransmitter is either absorbed by the releasing cell or destroyed by an enzyme.

There are over one hundred different chemical neurotransmitters in the nervous system, some distributed throughout and some concentrated only in specific neuronal areas. All perform specific roles in the

functioning of the nervous system – including in mood, sleep, motor function, and pain relief.[14] One of the major groups is the monoamines, which includes the neurotransmitters serotonin, dopamine, norepinephrine and acetylcholine.

Most neurotransmitters are made within the brain, derived from a variety of different chemical compounds known as the neurotransmitter's "precursors". If the precursor is not available, the brain will be unable to create the neurotransmitter. The knock-on result may be that the neurons will be unable to communicate correctly.

How the brain is made

Like all cells, neurons are composed of a cellular membrane that is mostly fat (or lipid) surrounding a nucleus and other matter suspended in water. As such, the brain itself is mostly made of water, but with about 60 per cent of its "dry weight" being fat.[15] The spinal cord is similar, being about 70 per cent fat.[16]

The fat in the brain is unique in the body for being predominantly composed of highly unsaturated fatty acids (see above)[17] – the most flexible type of fat. This ensures that the cell membranes, which play a vital role in transferring and incorporating nutrients[18] and are essential to the correct functioning of the cell, are also highly flexible, allowing rapid changes in the membrane shape.[19] This flexibility is vital to the successful communication between the billions of cells in the brain.

Essential nutrients for the brain

The brain is no different from any other organ in its need for proper and adequate nourishment. Brain cells and neurotransmitters are all created from nutrients, and nutrients continue to influence both their growth and their activity.

Nutrients and neurotransmitters

As described above, neurotransmitters are made from chemical precursors – usually from an amino acid (protein). There are several different types of amino acids, many of which are made within the body.

However, as already mentioned, a number of amino acids are defined as 'essential' because they have to be obtained from the diet and cannot be created by the body. Some of these essential amino acids include leucine, phenylalanine, lysine and tryptophan. (Others, such as tyrosine, are considered "conditionally essential" because, although they can be made in the brain, dietary sources may also be necessary to ensure adequate supplies.[20])

Some of the essential amino acids are themselves precursors to neurotransmitters, such as serotonin (from the essential amino acid tryptophan) and catecholamine (from tyrosine and phenylalanine). This means that if the diet does not provide adequate amounts of these amino acids, the neurotransmitters may not be produced adequately – theoretically creating problems in communication between neurons. (However, it should be noted that the likelihood of a diet being deficient in amino acids is quite low, particularly in Western countries.)

The following text highlights a number of neurotransmitter precursors that are relevant to our discussion of the links between diet and mental health.

Serotonin

Perhaps the most widely studied in diet-behaviour research has been the connection between the amino acid tryptophan and the neurotransmitter serotonin. Tryptophan is found in foods such as eggs, meat and beans and is first converted in the body to 5-hydroxytryptophan (5-HTP) before being converted to serotonin. Some of this serotonin is then further metabolised to produce the hormone melatonin – known for its sleep-inducing effects.

The levels of tryptophan available for conversion to the serotonin transmitter is not only affected by the amount contained in the diet. The presence of protein (even though it contains tryptophan) decreases tryptophan's availability in the brain because it introduces amino acids that compete with tryptophan for transport to the brain. Conversely, carbohydrates (even though they contain no tryptophan) actually increase tryptophan's availability, because the insulin released when carbohydrates are digested puts the competing amino acids to a different use, easing tryptophan's entry into the brain.

In addition, a number of different enzymes, minerals and vitamins are essential to convert tryptophan to serotonin.[21]

Catecholamines

Catecholamines are a class of neurotransmitters that include norepinephrine and dopamine. Both norepinephrine and dopamine have a number of roles, including mood regulation.[22] The dietary precursor is the amino acid tyrosine which, although not strictly an 'essential' amino acid, is still partially derived from food.[23]

Tyrosine is converted into these neurotransmitters by several enzymes, some of which are facilitated by other nutrients in the diet – for example copper and vitamin C.[24] Even if the levels of tyrosine are theoretically adequate, a lack of other vital nutrients may impede its functioning.

Acetlycholine

Another important example of dietary effects on neurotransmitters is choline. Choline can be made in the body, but it is also consumed as a component of the plant lipid lecithin (found in, for example, eggs, liver, peanuts and cauliflower). Choline is the precursor to the neurotransmitter acetylcholine, which is important in memory formation.

Thiamine, or other B vitamins, also affects this same neurotransmitter. The presence of such B vitamins is necessary for the body to produce its own choline[25] and to ensure that another amino acid, homocysteine, is properly converted to acetylcholine.

Nutrients and neurons

Essential fatty acids

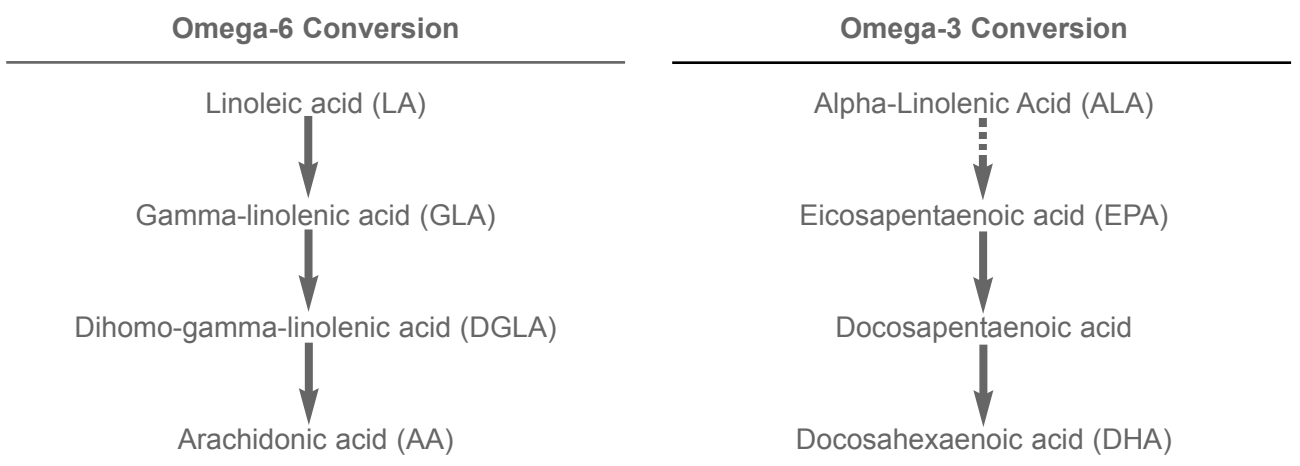
As we have already related, the brain is about 60 per cent fat – mostly in the form of highly unsaturated fatty acids – the most flexible form of fat. Moreover, 20 per cent of the dry weight of the brain – approximately one-third of the amount of total fat – is made from the 'essential' fatty acids.[26] Like the essential amino acids, essential fatty acids must be derived from the food we eat.

All of the essential fatty acids in the brain fall into two categories – omega-3 and omega-6.[27] These names denote families of essential fatty acids – both polyunsaturated. The omega-3 fatty acids include alpha-linolenic acid; the omega-6 fatty acids include linoleic acid. It is generally understood that the balance of omega-3s and omega-6s consumed in the diet is important for brain health. But these essential fatty acids are also important because of the different stages they undergo during their conversion in the body into long-chain fatty acids.[28] Each type – omega-3 and omega-6 – goes through a separate 'conversion sequence', with

each stage of the sequence performing or contributing to important processes for brain health.

When essential fatty acids are eaten by animals and humans, they are digested and then elongated or converted into different types of fatty acids (see above) – the elongation makes them 'long-chain' fatty acids. In the case of linoleic acid (LA, from the omega-6 group), it is first converted to gamma-linolenic acid (GLA); then to dihomogammalionlenic acid (DGLA) before being further elongated to become arachidonic acid (AA). Arachidonic acid comprises approximately eight per cent of the dry weight of the brain.[29]

A simplified illustration of the essential fatty acid conversion sequence



Alpha-linolenic acid (ALA, from the omega-3 group) is also converted through a series of elongations of the fatty acid. After some initial conversions, it is converted to eicosapentaenoic acid (EPA); then to docosahexaenoic acid (DHA). Like arachidonic acid from omega-6 fatty acids, DHA from omega-3 fatty acids makes up about eight per cent of the dry weight of the brain.[30] DHA also makes up 60 per cent of the photoreceptors responsible for receiving light photons and sending image messages to the brain.[31]

Although only very small amounts of EPA are found in the brain, it serves a vital purpose in acting as a precursor to the production of eicosanoids.[32] Eicosanoids are a group of short-lived hormones that perform a number of complex functions in the brain, including regulating inflammation and releasing some neurotransmitters. AA and DGLA (from omega-6 fatty acids) and DHA (from omega-3 fatty acids) are also precursors to eicosanoids.

Both linoleic acid (omega-6) and alpha-linolenic acid (omega-3) are found in plants - linoleic in the seeds and alpha-linolenic in the leaves.[33] Animals that have eaten the plants (which contain only the shorter chain fatty acids) convert these short-chain fatty acids into longer-chain fatty acids in their bodies. When the animals, in turn, are eaten by humans, the longer-chain fatty acids are digested, meaning that fewer conversions or elongations are necessary within the human body. In humans, conversion from LA and ALA is considered extremely slow – all told, less than two per cent of dietary LA and ALA might be converted to AA or EPA.[34] Therefore, it is often argued that it is more efficient for humans to eat already converted fatty acid chains by eating animals.

This is supported by evidence that vegans and vegetarians often have lower levels of omega-3 fatty acids in their bodies than meat or fish eaters.[35, 36] *However, there is no evidence that vegetarians' or vegans' health suffer from these lower levels – in fact, they are widely considered to be healthier than meat eaters. This apparent contradiction warrants further investigation.*

It is not possible for the two essential fatty acids, omega-3 and omega-6, to 'cross-convert' into one another's conversion sequences. So, for the brain to receive adequate amounts of AA, DHA, and EPA, which are vital for many brain functions, the body needs an adequate intake of both. Because of the approximately equal presence of both types of fatty acid in the brain, it is believed that dietary intake of the two should ideally be balanced equally.[37] This is partly because intake of one may affect the conversion rate of the other – higher intakes of LA (omega-6) to ALA (omega-3) tend to reduce the amount of the omega-3 fatty acids converted to EPA and DHA.[38] Vitally, the ratios of both in the brain are regulated and reflected by the ratio of these two fatty acids in the diet.[39]

In discussing the links between the type, quantity and quality of fats in the diet and brain health, it is also important to note that high intakes of saturated fatty acids may also affect the synthesis of the longer-chain unsaturated fatty acids, interfering with their incorporation into the brain.[40]

Micronutrients, oxidation and other factors

Micronutrients have a long and documented history of association with behavioural and mental disorders. Indeed, if one explores the symptoms of micronutrient deficiencies, behavioural or psychological disturbances are almost always listed as symptoms.[41] Often, these mental symptoms appear before any physical signs of a deficiency[42] - for example, scurvy from vitamin C deficiency is often preceded by irritability and a general malaise.

It has already been noted that copper and vitamin C can play a role in the creation of neurotransmitters. Zinc and magnesium also appear to have a role in the stimulation and levels of certain neurotransmitters.[43] Moreover, lipid metabolism is affected by zinc[44] as well as by magnesium and calcium.[45]

Some micronutrients (antioxidants) also play a role in protecting brain tissue from oxidation.[46] Oxidation occurs when free radicals (unstable oxygen molecules) bond to and destroy other compounds. Vitamins C and E are able to bond with free radicals, effectively neutralising these damaging effects. Vitamin E also helps lipid metabolism – because polyunsaturated fats are highly susceptible to oxidation, the presence of vitamin E facilitates successful conversion to long-chain fatty acids.[47]

Conclusion

Scientific knowledge about both the brain and nutrition's effect on the body is growing every day. With this growth in knowledge is coming greater understanding about how nutrients in food physically affect the brain. However, it already seems relatively clear that the food we eat has an impact on both the structure and physical function of the brain - how this might then affect the way our brain operates will be addressed in the following chapters.

Diet, brain development and mental well being throughout the lifecycle

The effects of diet on mental health are not only evident among those who are diagnosed with a mental health condition. Diet affects everyone's mental well-being, throughout their lives. The preceding chapter established that nutrients derived from the diet have an effect on the brain; but how is this reflected in brain and mental development? The following provides a summary of the evidence linking diet to brain development and mental well-being throughout a person's life.

Nutrition in prenatal, postnatal and early life stages

The periods before conception, during pregnancy and throughout the first three years of life are the most critical times for brain growth.[1] This is when neurons are formed and the connections are made where neurotransmitters work. What happens to the brain in this short period will affect a person's mental and emotional well-being for the rest of their life.

Birth weight

Much of the research on the effects of early nutrition on the brain comes from inferences drawn from studies of birth weight. Therefore, information on the importance of birth weight will be presented here first.

A baby that is born at full term and at a healthy weight has many advantages, both physical and mental, over pre-term or low-weight babies. Observation of this phenomenon has led to the "foetal origins hypothesis", which proposes that malnutrition in the womb leads the foetus to adapt its physiology and metabolism to a limited supply of nutrients, causing health problems later in life.[2] Originally linked to physical diseases such as coronary heart disease and diabetes,[3] some evidence now shows that babies born underweight (who often continue to be small for their age) frequently suffer mental health problems.

A systematic review of studies that looked at the mental development of low birth-weight infants confirmed that they are more likely to have poorer levels of mental development than those born at normal weight.[4] Negative effects can eventually be seen in IQ, language and reading ability,[5,6,7] with differences noticeable as early as six months of age.[8] There is also evidence that children born pre-term and with extremely low birth weights have a higher incidence of cognitive problems than children born at full term.[9]

A low birth weight can also affect behaviour and mental well-being. Studies have shown that infants with low birth weights are less co-operative, less active, less alert and less happy than normal-weight infants.[10] As a low birth-weight child grows, it may become more anxious,[11] less cheerful,[12] suffer from a poor attention span,[13] and be assessed as having more behavioural problems than normal birth-weight children.[14]

There is also evidence that low birth weights may lead to increased psychological distress in later life and studies have shown higher rates of depressive disorders in adolescence,[15] early adulthood[16] and later in middle age[17] (see chapter 3 for further evidence on rates of depression).

Moreover, the effect can be intergenerational. The birth weight of a mother and father is linked to the likely birth weight of any child they produce.[18,19] If they themselves had low birth weights, a cycle of low birth weight may develop, accompanied by associated developmental problems.

Preconception

The nutritional status of women and men before they even to try to conceive is likely to have a significant impact on the outcome of any eventual pregnancy. Good nutritional status increases fertility and the chance of giving birth to a healthy baby.[20]

Health before conception influences the body's ability to store and use fat during pregnancy, affecting the eventual nourishment of the embryo. The body's avoidance of this problem can be seen in the inability of very thin or athletic woman to become pregnant.[21] Men's fertility is also affected by diet. A man's sperm count can be decreased through nutrient deficiencies, most notably zinc.[22] Sperm count may also be reduced by pesticide exposure.[23]

Low birth weight

There is some evidence that suggests that the nutritional status of a woman at the time of conception appears to have more of an impact on her future child's birth weight than her nutritional status during her pregnancy.[24,25] It is suggested that this is because poor nutritional status at very early stages can lead to problems in embryo development and DNA replication that cannot be compensated for later in pregnancy or during early life.[26]

Several studies exploring the high prevalence of low birth weights in socially deprived areas have found strong correlations between poor nutrient intake *before* pregnancy and eventual low birth weights.[27,28,29]

Folic acid

Another issue relating to preconception health is the mother's folic acid status. In some women, a low folic acid intake can lead to a higher risk of neural tube defects.[30] The neural tube is the beginning of the brain

and spinal cord, and any defects have very severe neurological consequences. Because it is formed so early in pregnancy, when the mother may not even be aware that she is pregnant, healthy preconception folic acid status is vital. Supplementation reduces the risk significantly[31] and some countries have instituted food fortification to ensure adequate preconception levels in all women.

Maternal nutrition and foetal development

Of all of the organs in the body, the brain and spinal cord are perhaps the most sensitive to prenatal influences (not only nutrition, but stress and other environmental factors).[32] Development of the brain in the womb is extraordinary, with seventy percent of all the brain cells a human will ever have formed before birth.

The brain is particularly susceptible in the first few weeks after conception, when the basic structure of the brain is being formed. After this point, it is relatively well protected until the midpoint of pregnancy at which time there is a massive growth spurt.[33] During all of this period, seventy percent of the energy taken by the foetus is directed towards brain development.[34]

This energy is in the form of nutrients. However, there is a concerning paucity of information on the specific nutrients needed for the optimum growth of a foetus. One researcher expressed a common frustration on this issue: "it is a disgrace that we can't be more precise (other than prescribing a generally healthy diet), but the research hasn't been done".[35]

However, there is a clear connection between generally good maternal nutrition and healthy birth weights[36] and it is commonly accepted that general malnutrition leads to low birth weights.[37,38] By inference, this might mean that there is a connection between maternal nutrition and the mental and emotional problems seen in those with low birth weights (see above). In fact, evidence is growing that malnutrition in the womb does indeed lead to impaired neurodevelopment.[39,40]

Moreover, evidence of the effect of specific nutrients on healthy brain development is also growing. Much of the research in this area has concentrated on the contribution of essential fatty acids, which we discuss below.

Essential Fatty Acids (EFAs)

During pregnancy, over 600 grams of EFAs are transferred from the mother to the foetus – the type of fats that can only be gained from dietary sources – at an average of 2.2 grams per day.[41] Specifically, it is DHA and AA (the major structural EFAs in the brain - see chapter 1) that are selectively transferred by the placenta to the foetus. Apparently, little conversion of essential fatty acids takes place in the womb.[42]

The EFA status of a foetus is intimately related to that of its mother.[43] If the EFAs are not available from the mother's diet, the placenta will take them from the mother's body.[44] Accordingly, the mother's EFA status – particularly her level of DHA - decreases throughout the pregnancy.[45] This transfer of DHA greatly increases during the third trimester, the same time that the foetus' brain is growing rapidly.[46]

Some studies have shown that adequate amounts of AA and DHA in the mother are linked to higher birth weights, and longer gestational periods,[47.48] and hence to the wide range of health benefits associated with these factors (see above). Moreover, results of a number of studies have further suggested that high levels and intakes of essential fatty acids by the mother, particularly DHA, have cognitive and behavioural benefits for the child.[49.50.51] One recent study that measured the mother's total intake of fish (often high in DHA) during pregnancy found benefits from a high intake in the children's eventual language development.[52] Another found benefits for children's attention levels.[53] However, the relationship is not clear-cut, as other studies have found no correlation between measures of intelligence and DHA status at birth.[54]

Micronutrients

Some studies have shown a relationship between maternal intakes of specific vitamins and minerals and birth weight. Adequate levels of zinc, magnesium, and vitamins E and A have all been linked to healthy birth weights[55] and low levels of vitamin B6 have been noted in mothers of low birth-weight infants.[56] A trial that provided pregnant women with a multivitamin and mineral supplement found fewer incidences of low birth weight in the supplemented group.[57] It also found a twofold reduction in the risk of pre-term deliveries.

The iodine status of mothers is another widely recognised factor in foetal brain development. In fact, iodine deficiency is considered to be the most common preventable cause of neurodevelopment problems. Despite the fact that intervention to prevent iodine deficiency is relatively cheap through supplementation,[58] the condition is still widespread in areas where the soil is iodine deficient - most often in mountainous areas of poorer countries.

Although the most familiar result of iodine deficiency in pregnancy is cretinism, a severe neurological disorder, there is also some evidence that less acute deficiencies can result in less severe – but more widespread – problems in cognitive development. Studies comparing populations with differing iodine status have found significant differences in average IQs.[59]

Toxic substances

Alcohol is a well known toxin to developing foetal brains. Foetal Alcohol Syndrome, a result of abusing alcohol during pregnancy, results in damage to the central nervous system, specifically loss of neurons and damage to glial cells (the supporting cells to neurons in the nervous system). The symptoms last until adulthood and can include mental retardation, decreased IQ and hyperactivity.[60]

Exposure to some pesticides while pregnant appears to increase the likelihood of a pre-term birth.[61] A link has also been found between pesticide exposure during pregnancy and a smaller infant head. Because head size has been linked to later cognitive ability, there may be a link between pesticide exposure and subsequent mental development.[62]

Infants and early childhood

In contrast to the gaps in research on maternal nutrition and foetal health, there is a wealth of research on nutrition in early childhood and its effects on later mental and cognitive processes. The importance of providing the correct nourishment for mental development during this time is well accepted and widely promoted.

The brain in the first two to three years of life is particularly vulnerable to malnutrition. The earlier the malnutrition occurs, the greater the impact on the brain. And, the longer it goes on, the worse the result.[63] Studies on the impact of nutrients on the brain, mental development and behaviour during this time often measure what happens later in life, as a child grows. As such, there is a large overlap between studies looking at infant feeding and early childhood nutrition.

The following provides only a selection of some of that research and the findings that are relevant to this review of the evidence for the effects of diet on mental health.

Between the third trimester and two years of age (infancy), the human brain experiences its most critical growth spurt.[64] During this time, brain cells are moving to where they belong and improving their structure for communication. As such, when a human is born, 70 per cent of energy intake is used for brain development.[65]

Following this is the period of early childhood – when the brain is continuing to grow and develop. This is the time of most rapid development in the human lifetime. A time of critical physical, cognitive and emotional growth, it is normally during this period that behavioural and cognitive problems first become apparent.[66]

Breastfeeding and infant formula

Because a young infant is intolerant to normal foods, due to the immaturity of its digestive system, for the first few months of its life it must rely on milk for all of its nutritional energy needs.

Breast milk from the mother, if she is in good health and has an adequate diet, will provide a complete food for the early months, including all of the necessary macro and micronutrients.[67] At 50 to 60 per cent of the total, fat provides the principal form of energy found in breast milk. This fat is mostly unsaturated, with about five percent in the form of the essential fatty acids, some as already pre-formed long-chain polyunsaturated fatty acids.[68]

Specifically, breast milk consistently contains arachidonic acid (AA) and docosahexaenoic acid (DHA),[69] with the levels of each reflected in each woman's milk by the corresponding amount in her diet.[70] The mother's diet also influences other properties of breast milk, such as the presence of immunoglobulin, which helps protect the infant from infections.[71]

Much research has been done comparing the advantages and disadvantages of breastfeeding versus feeding with formula milk. The internationally accepted consensus, due to evidence of the protective physical health benefits for the infant in both the immediate and long-term, is that "breast is best". There is well accepted evidence that health problems in the first few years are lower in infants who have been breastfed, as well as growing evidence of benefits such as decreased chances of developing obesity or diabetes later in life. There is now also evidence that breastfeeding has a cognitive benefit as well.

Cognitive advantages from breastfeeding

Many studies have tested whether infants who are breastfed demonstrate improved cognitive development over those who are fed with formula milk. A systematic review of these studies concluded that breastfeeding does indeed have a beneficial effect on neurodevelopment.[72] A separate analysis reached the same conclusion – those infants who are breastfed have a consistent cognitive advantage over non-breastfed infants.[73]

The studies found that enhanced brain functioning was detectable early in infancy, measured by the child's performance in a number of tests – such as verbal comprehension – and by clinical observation.

The authors also found evidence that increasing the duration of the breastfeeding led to a gradual increase in benefit. Moreover, the research showed that the cognitive benefits of breastfeeding accompanied the infant through childhood and even into adolescence. Although there are a number of factors that could be the cause of this advantage, such as higher education levels amongst women who breastfeed, there is also a case for the role of nutrition

Due to their role in the structure of the brain (see above), many of the studies have suggested that the advantage of breast milk for the brain comes from the presence of AA and DHA.[74,75,76,77,78] Studies testing the blood DHA concentrations of infants confirm that DHA levels are higher in those that have been breastfed.[79] One (rather morbid) study was able to examine the brains of infants who had died early in life, confirming that the DHA levels in the brains of those who had been breastfed were significantly higher than those who had not.[80]

Trials have been conducted to test this theory. Because ethically it is not possible to randomise infants and mothers into breastfeeding or formula-fed groups, tests are usually done on children whose mothers have already chosen to bottle feed. One test found that infants fed on formula milk supplemented with DHA in the first weeks of their lives performed better in cognitive, language and motor ability tests when they had reached 18 months than those fed on standard formula milk.[81] Another, which followed infants on supplemented formula milk until they had reached one year, found cognitive, language and visual benefits at over three years of age.[82]

One consequence of this research has been the recommendation that infant formulas include these long chain polyunsaturated fatty acids in order to try to replicate some of the benefits of breast milk.[83,84] Some infant formulas (although not all) do now include them.[85]

Pre-term Infants

Infants born prematurely often miss out on the third trimester's concentration on brain and nervous system development. During this time, increasing amounts of DHA are transferred from the mother, whose personal levels of DHA correspondingly decrease.[86] Moreover, pre-term infants are born with very little fat reserves, making them fully dependent on their diet for essential fats.[87]

It has been suggested that being born pre-term may be detrimental to a baby's later cognitive abilities. One of the main findings of the review described above was that infants with low birth weights derived a larger benefit from breastfeeding than those of normal weight.[88] Tests with DHA supplementation in pre-term formula milk have found benefits for later cognitive development.[89]

General malnutrition

The weaning of infants onto solid foods is recommended after four to six months of breast milk, but often occurs earlier. If the replacement foods do not supply the level of macro and micronutrients provided by milk, deficiencies can lead to altered behaviour and cognitive abilities.

As the infant grows into early childhood, nutrition remains key to both current and future mental development. Much of the research in this area has focused on severe nutritional deficiencies.

Stunting in children is often caused by poor nutrition. If a stunted child's diet improves dramatically, then the child can often grow to a normal height. It is not entirely agreed which nutrients are vital, but protein, zinc and iron are all thought to be important factors.[90]

Stunting has been shown to have an immediate effect on cognitive and behavioural development. A study of children between nine and 24 months old found that the stunted infants were less active, less adventurous, less enthusiastic and generally less happy than non-stunted infants.[91]

General calorie and protein deficiencies early in life also have an impact on later cognitive development and intelligence.[92] General malnutrition in the very young has been shown to have detrimental effects on later school behaviour,[93] academic attainment[94] and motor skills in adolescence.[95]

One recent study, which compared nutrient intake in nutritionally at-risk 3-5 year olds found that the group that had been supplemented with a healthier diet early in life partook in less violent and anti-social behaviour when teenagers.[96] (However, diet was only one of the manipulated factors – exercise and mental stimulation were also included, so firm conclusions are not possible.)

If malnutrition is treated early, it appears that it might be possible to reverse cognitive problems. Several studies have demonstrated improved mental performance by supplementing malnourished young children's diets with additional calories.[97,98,99]

Anaemia

Iron deficiency is one nutritional problem that has received much research attention. It is well accepted that anaemic young children have impaired levels of cognitive development when compared with non-anaemic children.[100] Young children that have developed anaemia related to low iron intake have also been shown to cling more closely to their primary caregivers, to be more isolated, less attentive and to show less pleasure and playfulness.[101]

Anaemia in infancy has also been shown to have an effect on future development, as children who were anaemic in infancy often continue to suffer poor mental development throughout childhood.[102,103] With one important exception,[104] trials to correct the anaemia have not found consistent benefits for mental development even when iron levels are restored to normal.[105]

Childhood and adolescence

Reflecting a commonly held belief, a recent long-term study has confirmed that adolescent behaviour and mental health may have deteriorated significantly and measurably over the past 25 years.[106] Many researchers (along with parents, teachers and campaigners) have suggested that the changes in nutrition provided in school and at home over that period may be a contributory factor.

Much of the research exploring the connection between diet and mental health and behaviour in the young has centred on hyperactivity, specifically Attention Deficit Hyperactivity Disorder (ADHD). There is also growing concern about the rising levels of depression in the young. These are both explored in depth in

chapter 2. Although clearly interconnected, there is also a body of research relating diet to other aspects of mental well-being and behaviour, including anti-social behaviour, which is discussed below.

There is also a wealth of data on the impact of nutrition on other facets of children's mental and cognitive well-being – amongst others, research has found nutritional links to autism, dyslexia, and dyspraxia. All point to an influence of diet on brain development and hence, mental well-being and behaviour in childhood and beyond. They are also intimately related to the areas discussed both below and in the next chapter. However, these conditions are beyond the scope of this report and so will not be addressed here.

Academic attainment

One possible outcome of poor diets and brain development that has received much attention has been the subject of academic attainment. Similar to research on the impact of diet on an infant's later cognitive development, many studies have examined the link between children's nutritional status and achievement in the classroom. This ranges from the relationship of long-term malnutrition (see above) to the immediate effects of everyday eating habits.

Particularly, several studies have shown that children who eat breakfast can improve their daily and long-term academic performance.[107,108,109,110] However, due to conflicting results from other studies, researchers have been hesitant to draw firm conclusions.[111] Nonetheless, it does seem agreed that missing breakfast is more damaging to children who already suffer from general malnutrition.[112,113]

Notwithstanding the diet and brain relationship, it is hardly surprising that children distracted by hunger and lacking basic energy supplies find it hard to pay attention and learn. Schools around the world have instituted breakfast programmes, with many reporting benefits to test scores and other academic measures.

A number of studies have also suggested that supplementing malnourished children with vitamins and minerals can lead to improved intelligence and academic performance.[114,115,116117] However, the evidence points to benefits only for children who have deficient diets. There is no currently available published evidence that supplementing the diets of healthy and well-fed children will improve their academic prowess.[118]

Anti-Social Behaviour

Schools that have breakfast clubs also report improved behaviour in the classroom. A number of published studies have shown that hungry children behave worse in school, registering reductions in fighting and absence and increased attention when meals are provided.[119,120,121] More specifically, two studies have found that school children who received supplements of essential fatty acids showed less aggression, compared with controls, when they were placed under stress.[122,123]

A number of studies have looked at the impact of nutrition on younger people who engage in anti-social (including criminal) behaviour. As mentioned above, a recent study has suggested that early diet may be a factor in teenage violence. Unfortunately, the study's design makes it difficult to say with authority whether the evidence is strong enough to prove a causal link.

However, a very well-designed study has been published which does implicate diet as a factor in anti-social behaviour.[124] The study in a young offenders prison assessed one set of young offenders, supplementing their diets with vitamins, minerals and essential fatty acids, against a control group on an unsupplemented prison diet. The research design ensured that an improved diet was the only variable factor.

When complete, the researchers reported significant and remarkable reductions in anti-social behaviour in the supplemented prisoners. Reductions in incidents ranged from minor offences of disobedience to more severe violent acts. Moreover, separate studies have found the same results.[125,126]

Adults

Most of the research on the effects of diet on adult mental well-being and behaviour explores specific mental health problems, such as depression. These are addressed in detail in chapter 2. However, there is also a fair amount of research exploring the more subtle effects of diet on everyday mood and behaviour.

Meals and foods

Breakfast has been the subject of a number of studies, most showing that eating in the morning leads to improved mood. Two studies found that individuals who ate something for breakfast every day reported

better mental well being than those who had erratic morning routines.[127,128] Immediate benefits include improved memory[129] and a sense of calm under challenging conditions.[130] Although the type of food that a person eats for breakfast is important, it appears that simply eating something is important in its own right.[131]

General fish consumption has also been reported to have an association with better moods and a higher score for self-reported mental health.[132] This study was adjusted for variables such as income, age and other eating patterns, yet still found a significant improvement in mood in people who ate fish regularly. A recent study has also found fish consumption to be associated with a reduced risk of cognitive impairment (including poor memory) in middle age.[133] Further, the study found an increased risk of impairment associated with the consumption of cholesterol and saturated fat.

The elimination of sucrose (sugar) and caffeine from the diet has been shown to relieve emotional distress.[134] Further investigation by the same researchers has suggested that it was the elimination of sucrose that was the most likely contributing factor.[135]

Macronutrients

Many of the pieces of research on the effects of everyday diets on mood have explored the ratios of macronutrients consumed at mealtimes – for example, carbohydrates and protein. Researchers, noting the craving for carbohydrates experienced by those with seasonal affective disorder (SAD) and pre-menstrual syndrome (PMS), hypothesised that the brain was attempting to relieve negative feelings by encouraging an increase in tryptophan in the diet.[136] This would, in turn, lead to an increase in serotonin and, theoretically, an improvement in mood (see previous chapter).

This theory led to a number of studies looking at the mood-regulating effects of carbohydrates (trials have tested a variety of carbohydrate sources, both simple sugars and complex starches). Research has found higher intakes of carbohydrates amongst subjects who reported that they were feeling 'low'.[137,138] Further studies have also found correlations between high carbohydrate meals and subjects who reported that they had better moods.[139,140]

However, one of the main difficulties with the theory is that even a small amount of protein (as little as 2 to 4 per cent) in a meal would prevent an increase in tryptophan in the brain.[141] Even though such a low-protein diet could be contrived in a clinical setting, it is unlikely to have much relevance in the real world.

Other studies have not found a link between carbohydrates and improved mood.[142] Moreover, further studies have found better correlations between mood and protein intake.[143,144] Due to the varying results and abnormal meals necessary to achieve authoritative results, it has yet to be determined if carbohydrates possess mood altering effects.[145] If they do, they seem likely to be weak.[146]

However, there may be better evidence of a more cumulative effect. Studies that have looked at the effects of diet in the longer term have found correlations between a higher carbohydrate intake over several days or weeks and a better mood.[147,148] (This does not support the tryptophan theory, however, as all the diets had sufficient protein to prevent the theoretical effect.[149])

Tryptophan and tyrosine

Although the carbohydrate/tryptophan theory is not proven, there is still good evidence of a tryptophan effect on mood. A number of studies have found that depleting subjects of tryptophan – usually through a specially prepared drink – has immediate mood-lowering effects. This has been demonstrated in completely healthy individuals,[150,151] patients who have recovered from depression[152] and even relatives of depressed patients (who didn't have depression themselves).[153]

However, it must be noted that studies that have attempted to improve mood by supplementing tryptophan have been unsuccessful.[154] It can, however, have a sedative effect and was widely sold as a sedative until a contaminated batch led to it being removed from sale in 1990. (There is a movement currently for its reintroduction.)

A study that depleted the levels of tyrosine (see chapter 2) also found that depleted subjects became more irritable and experienced decreased energy levels.[155]

Micronutrients

If an individual has a less than adequate intake of a vitamin or mineral, the first symptoms will often be psychological, such as irritability or a depressed mood.[156] This has been observed for vitamins C,[157] B12,[158] B2[159] and others. This observation has led to research on the mood effects of depleting or supplementing various micronutrients.

One double-blind placebo study supplemented subjects with nine separate vitamins, at over ten times the recommended daily level, for one year. At the end of the year, both men and women in the supplemented group reported better mental well being than the control group.[160] The same study also found better performance on a range of cognitive tests in the females taking the supplement.[161]

Thiamine, or vitamin B1, has been the subject of a number of studies. Several controlled trials have demonstrated that subjects experience low mood, irritability and fatigue when they have a low thiamine status – and that their mood improves when their thiamine status is increased.[162,163,164,165]

A low selenium status has also been associated with poor mood. Interestingly, when selenium status is low in the body, the brain is the organ that keeps the tightest hold of the remaining amount, which has been taken as a hint of its importance in brain function.[166] One study found that the lower the selenium in the diet, the higher the frequency of anxious and depressed moods. Supplementing the diet with selenium over five weeks appeared to lead to improvements.[167] Another study, which depleted selenium from some of the study subjects' diets, recorded increased levels of hostility and depressed moods when status was low.[168] Other studies have found similar results.

Iron deficiency has also been implicated in poor mood and attention. This has been noted in anaemic woman, particularly those taking the contraceptive pill.[169] Folate deficiency has also been implicated in both depressed moods and in major depression (see chapter 2).

Finally, a link with cognitive performance has also been noted. The intake of vitamins B6 and B12 have been associated with better memory in middle age[170] and a trial that tested supplementation found benefits to cognition.[171] However, the trial did not find any impact on mood.

Older people

Reasons for the decline in function that occurs as the brain ages are still a matter of scientific uncertainty. Until recently, problems associated with an ageing brain were thought to be a consequence of the loss (or 'death') of neurons. More recently, however, growing evidence suggests that the cause of cognitive decline is probably much more complicated, involving changes to neurotransmission, the brain's ability to alter itself and gene expression. A recent study has even suggested that this process of change can begin in some people during early middle age.[172] Why this is so in some individuals and not in others has not been established.

The research on diet and mental well-being in later life has mostly centred on the loss of memory and other cognitive abilities, particularly in relation to dementia and Alzheimer's disease. These are addressed in the next chapter. However, a number of studies have also looked at the impact of diet on less severe cognitive decline, usually testing word recall and other short-term memory faculties.

The boundary dividing general cognitive decline and dementia is unclear – there is no definite line that sits between general memory loss and the development of dementia. Rather, it is described more accurately as a continuum.[173] This makes it difficult, and possibly inappropriate, to distinguish between those studies looking specifically at dementia and those investigating causes and treatment of less severe memory loss.

Nonetheless, the understanding that diet might have a protective effect on the ageing brain is supported by a growing body of evidence and is becoming more widely recognised. Due to an increasing number of studies showing a preventative effect of diet on memory loss and other cognitive problems, some dementia-related organisations now promote healthy diets as a preventative measure against age-related cognitive decline.[174] However, it is generally agreed that more trials are needed to test whether dietary change can halt, slow or even reverse a decline in brain function in an individual already affected.

Micronutrients

There is a wealth of studies looking at the cognitive effects of the B vitamins, particularly folate, B6 and B12, in the elderly.[175,176,177,178,179,180] The studies have shown varying strengths of association, but have suggested that, as a whole, deficiencies in these vitamins may increase vulnerability to cognitive decline and dementia as a person ages.[181] However, reviews of the trials testing treatment of cognitive decline with B vitamins have not had promising results.[182,183,184]

Several studies have also found cognitive benefits of supplementation with the antioxidant vitamins E and C, for elderly subjects who had a history of taking supplements.[185,186] However, any benefits noted have only been associated with the combination of the two vitamins, and positive effects have not always been found.[187,188]

Fats and vegetables

Several studies have pointed to associations between fat intake and cognitive decline. High intakes of saturated fats (found in, for instance, red meat, cheese and other dairy products) have been linked with higher levels of cognitive impairment.[189,190] Correspondingly, one study found that higher intakes of polyunsaturated fats (specifically from fish) by elderly men was linked to better cognitive function.[191]

Another study that compared meat eaters and vegetarians demonstrated a two-fold increased risk of dementia in the meat eaters.[192] Whether this was due to a lower intake of fat or a higher intake of vegetables is unclear. However, a more recent study has found that women who have eaten more vegetables, specifically leafy green and cruciferous varieties (e.g. broccoli and cauliflower), over their lifetime perform better on tests of cognitive function than woman who ate less.[193]

Parkinson's Disease

Parkinson's Disease is a brain disorder that occurs when the neurons that produce and regulate the neurotransmitter dopamine are damaged. The symptoms are body tremors, stiffness and slow movement. After Alzheimer's disease, it is the most common neurodegenerative disease.[194] Although it is usually associated with the elderly, it can and does affect younger individuals.

Parkinson's is suspected, although not confirmed, to be caused by a combination of genetics and environment – i.e. that some individuals are genetically susceptible to the disease and environmental factors trigger its onset.

Nutrients

A number of researchers have investigated the role that diet may play in the onset of Parkinson's disease. Due to the theory that cell oxidation may be a factor, a number of studies have investigated antioxidants (e.g., vitamins C and E). The results have been mixed, but in general antioxidants appear to be ineffective in preventing the onset of Parkinson's.[195,196,197] However, vitamin E appears more promising as providing a possible protective effect.[198] A recent review of studies highlighted that the most promising form of vitamin E was from food, and not from supplements.[199]

Low dietary iron levels have also been linked to the development of Parkinson's.[200,201] Moreover, a number of studies have fairly consistently found a relationship between animal and saturated fat intake and a higher risk of Parkinson's onset.[202,203,204,205] One study found a correlation with high intakes of dairy products.[206]

Pesticides

Many studies have examined the impact of another food-related factor on the onset of Parkinson's – pesticide exposure. Following a review of over 40 studies that showed an increased risk of the disease following exposure to a number of different pesticides, the UK government's Advisory Committee on Pesticides has recently called for more research in order to confirm the relationship and to clarify which pesticides create the highest risk.[207]

A previous review of many of these studies had also confirmed a relationship.[208] "Exposure" has ranged from those who work in agriculture,[209] those who live in the vicinity of sprayed areas,[210] and even those who have eaten large amounts of sprayed fruit.[211]

Conclusion

A wealth of research studies appear to confirm that the nutrients in food affect the health and well being of the brain – supported by growing knowledge into how they affect its physical structure and process shown in the previous chapter. Everything from proper neurodevelopment to short-term mood fluctuations can be affected by the food a person eats, beginning before their conception and continuing into their old age.

As shown by the range of nutrients implicated in the studies discussed above, it is clear that no single nutrient can be considered the panacea to good brain development and mental well being. Rather, it seems that a generally healthy diet, as recommended for the physical health of the body – containing the necessary range of micro- and macronutrients and minimising the consumption of unhealthy nutrients, such as saturated fat – is the key.

Having established that food is a factor is everyone's brain health and mental well being, the next chapter will explore when food may be a factor in more severe mental health conditions.

The role of diet in specific mental health conditions

The preceding chapter set out the case that there is growing evidence of a connection between the nutritional quality of the food we eat, our brain development and our general well-being. It demonstrated that a wide range of studies show that food, and the nutrients it contains, appear to have both short-term and long-term effects on cognitive development, mood and behaviour in all age groups.

There is also growing evidence that severe mental health problems can be linked to dietary factors. This chapter presents the peer-reviewed published evidence of the links between diet and Attention Deficit Hyperactivity Disorder (ADHD), depression, schizophrenia and Alzheimer's disease. In order to show the range of the research, we present epidemiological, physiological and clinical trial evidence in each case.

We begin our analysis with a caveat: *Our examination of evidence relating to each mental health problem in isolation showed that there is little evidence to date that these conditions can be prevented or cured by diet alone, nor that any are caused solely by the absence or presence of particular elements of the diet.*

Moreover, it must be emphasised that, particularly in the case of physiological evidence, there is an issue of causation. Mental health problems commonly lead to disordered eating and this may account for why differences are found in nutrient levels in the bodies of individuals with mental health problems. Some studies have accounted for this, but not each one cited.

However, we can also say with some confidence that evidence is accumulating that some nutrients may help to:

- Relieve the symptoms of some mental illness;
- Improve the effectiveness of medication in some cases; and
- Reduce the side-effects of some medications.

In addition, a diet lacking in particular nutrients, or poor nutrition in general, may be associated with the development of several types of mental health problems. Low intakes of the following nutrients have been most frequently found in those with a range of mental illnesses:

- Polyunsaturated fatty acids (commonly found in fish and plant oils);
- Minerals, such as zinc (commonly found in meat, grain, legumes and milk), magnesium (green leafy vegetables, nuts and grain) and iron (red meat, green leafy vegetables, eggs and some fruit); and
- Vitamins, such as folate (commonly found in green leafy vegetables and fortified cereals), a range of B vitamins (milk, dairy, yeast and grain products), and anti-oxidant vitamins such as C and E (fruit and vegetables).

Finally, some evidence suggests that diets with high intakes of saturated fats and sugar may be associated with the development of some mental health conditions.

ADHD

> *"Ignoring or denying (or exaggerating) the effect of diet on behaviour is not helpful to children and their families."* [1]
>
> Michael F Jacobsen and David Schardt, Diet, *ADHD and Behaviour: A Quarter Century Review*, Washington DC: Center for Science in the Public Interest, 1999

Attention-deficit/hyperactivity disorder (ADHD) is a highly controversial disorder, both in its diagnosis and its treatment. Questions still arise as to whether it is a disorder at all, with some contending that it is simply an extreme version of temperamental behaviour. However, it is defined in the fourth edition of the Diagnostic & Statistical Manual of Mental Disorders (DSM-IV) as a disorder in its own right and will be treated as such for the purposes of this report. Furthermore, many studies look at children who are generally hyperactive, and not necessarily diagnosed as ADHD. Some of these studies will also be included here.

ADHD is characterised by overactivity, impulsiveness and an inability to sustain attention. Low self-esteem, under-achievement and difficulties in socialising are also often experienced.[2] It is difficult to diagnose, as there is no specific test or marker, and its identification usually relies on a checklist of typical behaviours. Due to such factors, there is no definitive measure of how widespread the condition is – although it is generally thought to occur in 3-5% of all children. The most widely used treatment is medication.[3]

ADHD is most common in childhood or adolescence, but it can persist into adulthood, resulting in ongoing problems of under-achievement and social difficulties. There is a high overlap with other learning disabilities, such as dyslexia, dyspraxia and autistic spectrum disorders. The overlap with dyslexia may be as much as 30-50% of cases.[4]

Food additives

Controversy over the effect of diet on the symptoms of ADHD has been raging for nearly 30 years. This has mostly centred on the effect that food additives may have on behaviour, especially in young children. The focus on food additives is largely as a result of the work of allergist Benjamin Feingold in the 1970s.

The Feingold diet, which eliminates artificial colourings, flavourings and salicylates (a natural chemical found

in some foods), is still recommended and widely implemented by parents and parental support groups, who often report excellent benefits from it or similar types of dietary restriction. However, it has been generally dismissed by most professional medical bodies as ineffective, due to a perceived lack of clinical evidence.[5]

In truth, the controlled studies on the Feingold diet have shown mixed results[6] and are beset by criticisms of the study design from both supporters and critics of Feingold. Although there does not yet seem to be an accepted, definitive answer, a number of studies do appear to confirm that a very small percentage of children (e.g. between 0.03 and 0.15%) are sensitive to food colourings and preservatives. However, it has been suggested that the studies that arrived at these small percentages have underestimated the true scale of the incidence. One review of the studies deduced that the overall incidence among British children would be, at minimum, more than one per cent.[7]

More recent research, funded by MAFF (the now-defunct Ministry of Agriculture, Fisheries and Food) and subsequently overseen by the UK Food Standards Agency, again attempted to determine whether young children experienced hyperactivity when exposed to certain additives.[8] The children were not necessarily diagnosed with ADHD. The findings were again mixed – although the parents certainly noticed changes in behaviour, this was not replicated in the clinical assessment. When the study was presented to the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT), the committee decided that it was not possible to reach firm conclusions from the study. A new study has now been funded by the Food Standards Agency and the results are expected in 2007.

Dietary epidemiological evidence

The epidemiological evidence for the causes of ADHD is limited, due mainly to problems of definition and to changes in diagnostic criteria.[9] However, it is known that ADHD is much more prevalent in boys,[10] and children who develop the disorder are less likely to have been breastfed.[11] There is also evidence of a link between ADHD and cigarette-smoking by the mother during pregnancy.[12]

These findings suggest there may be a link between ADHD and polyunsaturated fatty acids (PUFAs). The superiority of breastfeeding over formula milk is partly due to the presence of essential fatty acids[13,14] in breastmilk, and evidence suggests that males may be more vulnerable than females to deficiencies of PUFAs.[15] (It should also be highlighted that smoking promotes the release of free radicals, which can destroy fatty acids.) The idea of a link between hyperactivity and essential fatty acids was first proposed by the founders of the

Hyperactive Children's Support Group (HACSG, a parental support group for families with children with hyperactive behaviour), over 20 years ago.[16] They had surveyed children known to group members and found similarities between their physical symptoms and the symptoms of essential fatty acid (EFA) deficiency. These included abnormal thirst, eczema, and asthma. Further studies have confirmed that children diagnosed with ADHD commonly share characteristics of EFA deficiency – including dry, rough skin, dull hair and frequent urination.[17,18]

The HACSG study also noted that dietary intake of EFAs was no different than in non-ADHD children. It therefore suggested that the problem might lie in the children's ability to convert EFAs within the body (see chapter 1), rather than in differences in the diets that the children were eating.

Relevant physiology in those with ADHD

Following this suggestion by HACSG, a number of controlled trials have compared the levels of EFAs in children with ADHD. One of the earlier trials compared the blood[a] of 48 hyperactive children (not necessarily diagnosed with ADHD) with 49 ordinary children, finding that the levels of some EFAs (DHA, DGLA and AA – see chapter 1) were significantly lower in the hyperactive children.[19] This finding has been replicated several times in trials of children diagnosed with ADHD[20,21,22,23] as well as of adults with the same condition.[24]

Some of these trials also noted correlations between the levels of EFAs in the body and the degree of the disorder – i.e., the lower the level, the worse the symptoms. One found this particularly associated with deficiencies of total omega-3 fatty acids.[25] Some of these studies have also measured the children's dietary intake, again finding no measurable differences to account for the lower levels of EFA found in children with ADHD.[26,27]

Deficiencies in zinc have also been noted in those diagnosed with ADHD.[28] One study found that a third of those with ADHD showed a marked zinc deficiency, which again could not be accounted for by dietary intake.[29] Another study found that iron levels were significantly lower in those with ADHD when compared to controls – it also found that the lower the iron level, the more severe the symptoms.[30] A non-controlled trial also found evidence of magnesium deficiencies in children with ADHD.[31]

a Measuring levels in the blood gives a realistic measurement of the level found in the brain.

Research trials

Starting with tests using the Feingold diet, there have subsequently been many clinical trials testing different foods, supplements and additives for their effects on the symptoms of hyperactivity and ADHD. Most of these have highlighted a gap between what the parents view as having an effect and what the clinicians observe.

Few foods diets

There is a generally acknowledged prevalence of food sensitivities in those with ADHD, although debate continues over their significance in contributing to the disorder. Acute food allergies are well accepted, but less severe (and harder to measure) sensitivities have less official recognition. Nonetheless, there is still a great deal of anecdotal evidence that for some children with ADHD, behaviour improves when certain foods are eliminated from their diet.

Some clinical studies have shown a benefit of elimination diets under controlled conditions. One study placed 78 children on a diet that restricted foods generally believed to have an effect, and the behaviour of 59 of the children improved. For 19 of those children, it was possible to disguise the foods thought to have an effect and test them under double-blind conditions. When the children ate those foods, there was a significant worsening of behaviour.[32]

Another double-blind study compared the effect of restricted diets with medication, finding that the behaviour of nearly a quarter of the children improved when they were placed on the restricted diet. Although more children showed improvement on the medication, the researchers concluded that the level of improvement in both groups was about the same, suggesting that diet was as effective as medication for some children.[33]

These and other trials have led to the elimination approach being included as an acceptable and recommended option in one overview of ADHD treatment protocol.[34]

Nutrients

Other trials have tested the efficacy of specific supplements on ADHD patients. These have mostly been of minerals and essential fatty acids, following the observations of deficiencies in ADHD patients.

Polyunsaturated fatty acids

A number of trials have tested different essential fatty acids for their effects on hyperactivity and ADHD. Generally, those testing DHA alone[35,36] and those testing omega-6 fatty acids (gamma-linolenic acid – GLA)[37,38] have been unsuccessful – in line with tests on other mental disorders (see other sections in this chapter).

A trial that tested a mix of omega-3 and omega-6 fatty acids did find a significant improvement in behaviour over a placebo, correlating with measurements of red blood cell fatty acid concentrations.[39] Another study tested children diagnosed with dyslexia who displayed symptoms of ADHD – when supplemented with both omega-3 and omega-6 fatty acids, the ADHD behaviours improved.[40] A recent trial, which tested children diagnosed with developmental dyspraxia, also found that the often-accompanying ADHD symptoms decreased significantly when the children were given an omega-3 and omega-6 supplement.[41]

Micronutrients

Following the observation that many ADHD patients are deficient in zinc, several trials have tested the efficacy of zinc supplementation, with generally encouraging results. Two double-blind trials found significant improvements with zinc when compared with a placebo; one alongside normal medication[42] and the other as a stand-alone treatment.[43]

Magnesium supplementation has also been tested in a double-blind controlled trial, resulting in a significant improvement in symptoms over the six months of the trial.[44]

An open trial of iron supplements found that parents noticed a decrease in ADHD symptoms among their children.[45] However, this was not observed by the children's teachers.

Sugar

Sugar has anecdotally been identified as a possible culprit in ADHD and general hyperactivity. Assessing its effect is difficult, as there are several different types of sugar naturally present in, and added to, a wide range of foods. Moreover, foods high in sugar, particularly refined sugar, tend to also contain artificial colourings and preservatives, further complicating any study.[46]

Nonetheless, several studies have been done to test the effect of sugar consumption on children with ADHD. A review was conducted of the controlled studies, reaching the conclusion that there was no clear evidence that sugar had an effect on children's behaviour, but that it still could not be ruled out in some children.[47] However, this analysis has been criticised as flawed, as the studies did not include many children with ADHD, were of brief duration (1-2 days) and involved only small numbers of children.[48] Longer and larger trials of the effects of sugar on children with ADHD would be useful, similar to the trial currently being undertaken on behalf of the Food Standards Agency, to assess the behavioural effects of certain food additives.

Summary

There is a long history of association between ADHD and dietary factors. Despite strong anecdotal support, the published evidence does not clearly point to food being a consistent causal factor in ADHD.

However, most of the trials do appear to show benefit for some children, sometimes only in just a few children, but sometimes more. This may be a reflection of what many advocates of dietary therapy have been suggesting for years – that *some* children (and their parents) may find relief from ADHD through altering diets.

There is clearly room for more clinical trials, longer in duration and with larger numbers of participants. But, at the same time, there is clearly room for listening to those who have children with ADHD under constant, if not clinical, observation – i.e., the parents.

Depression

"Depression seems to be the one disorder that is most responsive to diet." [49]

Larry Christensen, *Diet-Behaviour Relationships - Focus on Depression,*
Washington: American Psychological Association, 1996

In the past century, the prevalence of severe depression in the population has increased dramatically, reportedly by as much as twenty-fold since 1945.[50] Along with a general increase, the age of onset has lowered, with more cases reported in adolescents and young adults.[51] It is not plausible to explain the increases by changes in "reporting…or labeling" [52] – rather, there has been a genuine rise in the numbers of individuals developing and suffering from depression.

Explanations for the rise have included increased life stress and changing social networks. However, evidence also points to the changes in nutrition that have occurred at the same time (see the next chapter).

Dietary epidemiological evidence

A number of cross-country and population-based studies have linked the intake of certain foods or nutrients with the reported prevalence of different types of depression. Although the studies do not clearly prove causation, they are the basis of a growing body of evidence linking dietary intake with depressive disorders.

Several studies have looked at the intake of essential fatty acids, measuring intake by the amount of fish or seafood consumed. Correlations between low intakes of fish by a country and high levels of depression amongst its citizens – and the reverse – has been shown for major depression,[53] post-partum depression[54] and seasonal affective disorder (SAD).[55] A correlation has also been demonstrated for bipolar affective disorder.[56]

Epidemiological research has also been undertaken involving specific populations. A study looking at the changing diets of peoples living in the Arctic and Sub-Arctic regions found that levels of depression, including seasonal affective disorder, were rising at the same time that traditional diets were being abandoned for more processed foods.[57] One study in Finland found that people who frequently eat fish have lower levels of depression than people who consume fish less often.[58] However, another study with similar methods found no connection between the intake of fish and depression.[59]

Separate research has also found a correlation between dietary intake of folate (folic acid) and depression. The study followed over 2,000 people and found that those with low intakes of folate in their diet were significantly more likely to be diagnosed with depression than those with higher intakes.[60] Another study found that folate intake in a Latino population was also correlated with depression (however the association was only found in women).[61] Another study found that higher intakes of Vitamin B12 were associated with significantly better results of treatment in patients with major depression.[62]

Recent research has also found an 11-fold increase in depression amongst females who were born premature or with a low birth weight (see chapter 1).[63]

Relevant physiology in those with depression

Alongside the epidemiological evidence, many studies have shown that patients diagnosed with depression exhibit nutrient-related physiological differences when compared to individuals without depression. (However, as noted above, it is not wholly clear whether these are causative of depression, or a result of disordered eating as a result of the condition.)

Neurotransmitter precursors

A number of studies have looked for differences in the level of the essential amino acid tryptophan (see the first chapter) between depressed and non-depressed subjects. The results have been conflicting, with some results finding differences and some not. However, studies of severely depressed individuals have found consistent differences between the ratios of tryptophan to other competing amino acids, when compared to non-depressed subjects.[64] This ratio is important as it will influence the ability of tryptophan to reach the brain.

One study found that subjects with minor depression had significantly higher ratios than subjects with more severe depression.[65] These findings suggest that tryptophan levels may be a factor in major depression.[66]

Micronutrients

A number of studies have measured the levels of certain vitamins in the bodies of depressed patients. Much of this work arose out of observations of the depressive symptoms of vitamin deficiencies. Often, the first sign of an inadequate intake of micronutrients are psychological, and depression has been associated with low levels of B1, B2 and vitamin C.[67]

Most of these studies have measured the levels of folate in depressed patients.[68] The studies found that substantial percentages of hospitalised depressed patients had low folate levels, whilst only a very small percentage of non-depressed controls were deficient.[69, 70] One study found that subjects diagnosed with major depression had lower folate concentrations in their blood than controls who had never suffered from depression. The study also found that patients with even minor depression had lower levels, but not as low as those with major depression.[71]

Some of the studies of the effects of folate observed that low levels of B12 were also apparent in people with depression.[72] One study found that the blood of depressed patients contained low levels of vitamin B12, even when there was no apparent dietary deficiency.[73] It was also observed that patients with low levels of folate and B12 showed poorer moods than patients who had at least one of the vitamins at a normal level.[74] Deficiencies in vitamin B6 have also been observed in depressed patients.[75]

As for minerals, several studies have shown that the levels of zinc are lower in the blood of patients diagnosed with depression.[76, 77]

Polyunsaturated fatty acids

A number of studies have concentrated on the levels of polyunsaturated fatty acids (PUFAs) found in patients with depression. Although earlier studies found no differences in the levels between depressed and non-depressed patients,[78] more recent research – having better methodology[79] – has consistently found significant variations.

Studies examining the fatty acid composition of the blood of depressed patients have found differences in both the total level of PUFAs and differences in the ratio of omega-3 to omega-6 fatty acids, when compared to individuals without depression.[80, 81, 82, 83] The studies found lower levels of omega-3 fatty acids, specifically

DHA, and higher ratios of omega-6 to omega-3. Other research found a correlation between the severity of the depression and a higher ratio of arachidonic acid (AA – from omega-6) to EPA (from omega-3).[84]

Similar results have been found for bipolar affective disorder. Research into bipolar patients found that their levels of AA and DHA were significantly lower than in non-patients, although the total amount of PUFAs was the same.[85]

Many of the studies have proposed that the differences might be the result of an abnormality in fatty acid metabolism in depressed patients. However, a study that compared the body PUFA level, whilst also measuring patients' dietary intake, found that not only was the severity of depression correlated with the lower levels of omega-3 found, but it was also correlated with lower dietary intake.[86] This may mean that the low level of PUFAs found in depressed patients is not due to any difference in the way the body handles these nutrients, but rather because of the amount eaten (seemingly different to ADHD, see above).

Women with postpartum depression exhibit similar abnormalities in their bodies' levels of PUFAs. Pregnancy depletes the mother's level of PUFAs, due to the transfer of EFAs to the developing foetus (see the previous chapter) and after delivery the levels continue to decline.[87] Studies comparing woman with and without postpartum depression showed that the depressed women had lower levels of omega-3 in their bodies, as well as higher ratios of omega-6 to omega-3.[88]

Researchers have also measured the levels of PUFAs in adipose tissue, or "storage fat". Because this tissue reflects dietary intake over one to three years, these measurements reveal accumulation and levels of essential fatty acids over a longer term than the levels in the blood.[89] The studies found that depressed individuals show lower levels of omega-3 in their adipose tissues, when compared to non-depressed individuals,[90] specifically lower levels of DHA.[91] However, one similar study on adolescents did not show such a relationship.[92]

Oxidation

There is evidence that depressed patients have higher levels of oxidant activity in their bodies than non-depressed people.[93] Patients diagnosed with both obsessive-compulsive disorder (OCD) and depression have been shown to have higher levels of oxidative activity than patients diagnosed with OCD alone.[94]

Higher levels of oxidant activity would mean increased oxidation of fats and could be a possible factor in the levels of PUFAs in the bodies of depressed patients. Separate research has linked greater depressive symptoms with higher levels of fat oxidation.[95]

Research trials

Unipolar depression has been the subject of many clinical trials testing the efficacy of different nutrients in alleviating its symptoms. Effects on bipolar disorder have also been tested, but not to the same extent.

Neurotransmitter precursors

Some of the initial trials done in this area investigated the effects of supplying subjects directly with neurotransmitter precursors, following the theory that depression could be caused by faults in neurotransmission in the brain. The precursor to the neurotransmitter serotonin (the amino acid tryptophan), has been the subject of much testing. There is good evidence showing that consuming tryptophan leads to an increase of brain serotonin, and that removing it from the diet reduces serotonin[96] - one trial found that those who were in remission from depressive symptoms relapsed almost immediately when given a tryptophan-free drink.[97]

However, the results of trials using tryptophan to treat depression have been conflicting. Of the studies comparing tryptophan to a placebo, only a small number showed tryptophan to be superior; comparing it to antidepressants showed tryptophan to be only equivalent or inferior to the medication.[98] However, controlled studies that compared the combined effect of both antidepressants and tryptophan, to antidepressants given in isolation, and to a placebo, consistently found that the addition of the precursor tryptophan gave better results.[99] It has been noted that the subjects with low ratios of tryptophan to competing amino acids are more sensitive to increases or decreases in tryptophan. This might suggest that those with more severe depression are more likely to respond to this sort of treatment.[100]

Tyrosine, the amino acid precursor to the neurotransmitter catecholamine, was tested during the 1980s. However, the results were not promising enough to continue and it has been largely abandoned as a possible treatment.[101]

Vitamins

Because of the widespread incidence of folate deficiency in depressed patients, much of the vitamin research has tested the efficacy of folate on relieving depressive symptoms. Mostly it has been tested as an additional treatment in patients already receiving care.

A recent review of all of the controlled studies that had tested folate concluded that there was evidence that adding the micronutrient to existing treatment did help to improve depressive symptoms. Although it was unclear whether both subjects with normal folate levels and subjects with deficient levels would be helped, the study did confirm that folate may have a useful role as a supplemental treatment.[102]

A recent controlled study also tested the effect of zinc on patients diagnosed with unipolar depression. It found that depressive symptoms were significantly relieved when the patients were supplemented with 25 mg of zinc over two months.[103]

For bipolar disorder, an open trial tested the efficacy of high-dose multivitamin supplementation on already medicated patients.[104] The patients received the supplementation for six months and, for those that completed the trial, more than a 50% reduction in symptoms was reported. The patients' need for medication was also decreased by over 50%, with some people completely replacing their medication with supplements. The authors followed this study with a placebo-controlled trial, but the results were not yet available at time of writing.

Polyunsaturated fatty acids

To date, many studies have investigated the impact of essential fatty acids, particularly omega-3, in treating depression. The studies have been widely reported in the press, doing much to popularise (at least in the public's eye) the use of fish oils as a treatment for depression.

A number of case reports have been published, some showing dramatic effects of omega-3 supplementation on depression symptoms. One of these supplied EPA as an additional treatment to an individual who had previously not shown improvement on antidepressants. Nine months after beginning treatment with EPA, the patient had no depressive symptoms. Although case reports may be unreliable, due to the placebo effect, further weight was given to this trial because the researchers also measured physiological changes through brain imaging. They found that the EPA treatment was accompanied by significant structural changes in the patient's brain, all of which indicated reduced breakdown and increased synthesis of brain phospholipids.[105]

On the heels of promising case reports have come a number of controlled trials. Interestingly, trials testing the efficacy of DHA (which makes up the majority of omega-3 essential fatty acids in the brain, see the first chapter) appear to have been largely unsuccessful. One double-blind placebo trial administered two grams

of pure DHA to patients over a six-week period. The results showed no benefit over the placebo.[106] However, there is considerable debate about the negative results, partly due to the relatively-short length of the trials.

One of the first controlled trials tested for efficacy in bipolar disorder. Over four months, patients were treated with a combination fish oil of EPA and DHA in the ratio of 2:1, in addition to their regular treatment. In every outcome measure, the patients on the fish oil did better than those on the placebo, including experiencing longer periods of remission.[107] A more recent, open study reported that irritability in bipolar patients was also significantly reduced following supplementation with low-dose fish oil.[108]

A four-week trial treated unipolar depression patients with EPA alongside antidepressant therapy. The controlled trial found highly significant benefits of the EPA over placebo by the third week.[109] Another placebo-controlled study, administering a high-dose of fish oil with the patients' usual treatment, also found significant improvement in the fish oil group.[110]

Another three-month study tested three different doses of EPA (1g, 2g, and 4g) against a placebo in patients with persistent depression, despite receiving antidepressant drug therapy. Interestingly, the most significant improvement was shown in the group receiving just the one gram. The group receiving two grams did not show an improvement and the group receiving four showed some, but nothing significant.[111]

Although not a study specifically on depression, a randomised trial comparing EPA and a placebo in women with borderline personality disorder found that EPA was significant in decreasing depressive symptoms, as well as aggression.[112]

Summary

A good deal of evidence appears to point to a role of nutrition in the prevalence and outcome of depressive disorders. Rates of depression have increased dramatically at the same time as the food system has changed beyond recognition (see the next chapter). Epidemiological studies have found significant correlations between what a population eats and the level of depression it suffers. Physiological measurements of patients suffering depression point again and again to deficiencies in one nutrient or another. And, finally, nutrient-based treatment of those who experience depression is showing efficacy.

However, despite reports in the popular press, there is no *clinical* evidence that popping fish oils or multivitamin pills will cure depression. The controlled trials so far have used the nutrients being tested predominantly as add-on treatments to traditional antidepressant drugs. Here the evidence is firmer – improved levels of nutrients may help the antidepressants work better. Further research into why this is so is now necessary.

Harder to prove through controlled trials is whether or not adequate nutrient intake could prevent the development of depression in the first place.

Schizophrenia

> *"Schizophrenia is not caused by a nutritional deficiency... Nutrition is a way of putting the biochemistry of the brain in the best possible shape to allow other treatments to work. Nutrition is a partner in the therapeutic alliance, not the only player."*
>
> David Horrobin, *Nutritional Supplementation in Schizophrenia.*
> Schizophrenia Association of Great Britain, Newsletter no. 27

Schizophrenia is a severe mental disorder that results in hallucinations, delusions, and disordered thinking. It is the only disease that is distributed roughly equally throughout every population in the world – in every country that has been studied, between 0.5 and 1.5 percent of the population – approximately one in every hundred people - will develop schizophrenia during their lifetime.[113] No society, race or culture has ever been found to be free of this particular mental disorder.[114]

Historical data shows that the incidence of schizophrenia increased dramatically during the Industrial Revolution. In England, first-time admissions to asylums doubled between 1869 and 1900,[115] mirroring developments in other industrialising countries. Although it was suggested that this was a result of greater recognition (due to increasing numbers of people living together in cities), it has been suggested more recently that this could not have explained such as sustained growth over many decades. Rather, the increase appeared to reflect a genuine rise in incidence.[116]

Although incidence rates are similar around the globe, there are differences in the outcome of the disease. In measuring factors such as the duration and severity of the illness, along with the degree of life disruption,

researchers have found wide variations in the outcome between different countries.[117] Generally, people with schizophrenia in poorer countries fare better than those in richer, industrialised countries – in the WHO 10-country study at two-year follow-up, for example, the percentage of cases with full remission after a single episode ranged between 3% in the USA and 54% in India.[118] According to the World Health Organisation, "*this undoubtedly means that environment plays a crucial role as an outcome determinant in schizophrenia*".[119] Suggested environmental factors have included better family support and better social tolerance to the disease.

Dietary epidemiological evidence

Another possibility was explored in a Danish study over 20 years ago. The study[120] compared the outcome of schizophrenia with the amount of fat in the average national diet, as measured in the Food and Agriculture Organisation's food balance sheets. The comparison found significant correlations between low intakes of fat from consuming birds and land animals (i.e. saturated fats) and better health outcomes. Higher intakes of these fats correlated with worse health outcomes. Correspondingly, where there were higher percentages of fat from vegetables, fish and seafood, there were also better outcomes.[121]

More recent ecological analysis has found links with another dietary factor – sugar. This analysis found that "*a higher national dietary intake of refined sugar and dairy products predicted a worse two-year outcome of schizophrenia.*"[122]

Breastfeeding and prenatal nutrition

General nutritional deficiency has also been correlated with the development of schizophrenia – one study examined the development of the disease in the children of woman who suffered famine in the Nazi-blockaded Netherlands during World War II. Children who were conceived during the most severe part of the famine were twice more likely to develop schizophrenia than the rest of the population.[123] However, this situation created a general deficiency, so did not point to any particular missing dietary factor. These results have recently been confirmed by a study examining the Chinese famine of 1959-1961 – the researchers again found a correlated increase between pre-conception famine and schizophrenia.[124]

Because of the need for polyunsaturated fatty acids during the development of the foetal and infant brain, coupled with the advantages of breastfeeding over formula feeding in delivering those nutrients (see preceding chapter) – and the suggested link between PUFAs and schizophrenia (see below) – it has been suggested that there may be a link between breastfeeding and a reduced risk of developing schizophrenia. However, even though there have been a number of studies, some quite large, these have not presented firm evidence for such a link.[125] However, one study did find a positive correlation between the duration of breast-feeding and the age at onset of schizophrenia, suggesting that breastfeeding might at least postpone the eventual development of the disease.[126]

Coeliac disease

There is limited evidence to suggest a link between coeliac disease (the inability to digest proteins found in cereals) and schizophrenia. Evidence of the link was first suggested during the 1960s,[127] but subsequent studies failed to replicate those results and there has been little systematic investigation since.[128] However, a recent population-based study found the risk of schizophrenia was three times greater in people with a history of coeliac disease.[129]

However, this study has attracted some criticism. Its measurements included the parents of the schizophrenic patients – without including these, the prevalence was the same as the rest of the population. Another issue was that the study was completed in Denmark. Recent evidence suggests that coeliac disease is more common in Denmark than previously thought, similar to other Scandinavian countries. Higher than expected cases of coeliac disease would have confounded the study's results.[130]

Relevant physiology in those with schizophrenia

This epidemiological evidence chimes with a number of observations made of physiological differences between those with schizophrenia and those without. Several studies have shown that the bodies of people with schizophrenia contain lower levels of polyunsaturated fatty acids (PUFAs) than people who do not have the condition.[131,132] Other studies showed that there is an increased breakdown or low synthesis of phospho-lipids in the cell membranes of people with schizophrenia[133,134] – including that their brains contain a higher than normal levels of the enzyme (phopholipase 2) that actually breaks down membrane phospholipids.[135] Further evidence of a link with essential fatty acids comes from the observation that people with schizo-

phrenia do not experience a deep flushing of the skin when given niacin, or nicotinic acid, as most people do. This flushing is a result of a further metabolism of arachidonic acid (prostaglandin D2), implying that this metabolism is abnormal in those with schizophrenia.[136]

These observations led to the "membrane hypothesis of schizophrenia" – suggesting that abnormalities in phospholipid metabolism may underlie the course and severity of the disease.[137] More recent research suggests that variations in cell membranes would not in themselves lead to schizophrenia, but rather might reflect underlying abnormalities of fatty acid metabolism and interaction with diet and medication.[138]

Further studies into the physiology of schizophrenia indicate that antioxidant enzymes are also lower in the brains of those with schizophrenia – suggesting that their cells may be more vulnerable to oxidation.[139, 140, 141] Another study showed that people with schizophrenia have lower levels of folate in their bodies.[142] Given that PUFAs are highly susceptible to oxidation, it may be the case that there is a link with lower than normal levels of membrane PUFAs and phospholipid breakdown. Destruction of the phospholipid membrane would be likely to affect neuron function – perhaps leading to the distorted information processing present in the brains of those with schizophrenia.[143]

Research trials

The membrane hypothesis and research into oxidation, along with earlier work on treating the disease with nutrients such as vitamin C, have led to a number of research studies and trials testing the efficacy of treating schizophrenia with a number of different nutrients. Essential fatty acids have received the most attention, along with a range of vitamins.

Polyunsaturated fatty acids

The results for essential fatty acids have been mixed, yet promising – in the words of one of the principal researchers, "*a conservative view is that the present evidence regarding the efficacy of EPA in the treatment of schizophrenia is suggestive but not definitive*".[144]

Indeed a systematic review of all of the studies took place in 2003, finding a good suggestion of an effect, but noting that all of the studies were too small to be conclusive, so more trials were needed: "*The use of*

omega-3 polyunsaturated fatty acids for schizophrenia remains experimental and large well designed, conducted and reported studies are indicated and needed." [145]

One earlier study took detailed dietary records of 20 patients with schizophrenia and compared them with each patient's symptoms – finding that the higher the intake of omega-3 fatty acids, the less severe the symptoms.[146] This same study, in an open trial with no control group, supplemented patients' antipsychotic medication with a concentrated fish oil (containing both DHA and EPA) for six weeks. This was followed by a significant improvement in psychotic symptoms, which correlated with the level of omega-3 levels measured in the patients' cell membranes.

Another study administered EPA alone to a single patient with schizophrenia, who was on no other medication, resulting both in changes to the phospholipid structure of the patient's cell membranes and improving the patient's symptoms. The improvement had been sustained over three years.[147]

These earlier observations and case studies have led to larger controlled trials. As of 2002, five double-blind randomised control trials had been published. The first of these attempted to distinguish between the effects of EPA and DHA (both omega-3). Evidence was building of the greater effect of EPA over DHA, which was surprising considering that the brain's essential fatty acids are predominantly in the form of DHA (see the first chapter). The reason that EPA demonstrated this greater effect is not yet understood, but recent evidence points to changes that also occur to the arachidonic acid (AA, omega-6) in the brain when EPA is consumed.[148]

Forty-five patients were divided into three groups and received DHA, EPA or a placebo for three months, in addition to their normal medication. All the patients receiving EPA showed improvement in their symptoms that were statistically significant over any improvement on DHA or the placebo.[149]

The second controlled trial, reported in the same article, used only EPA and a placebo on patients who were previously unmedicated, but given antipsychotics if deemed clinically necessary. Twelve patients were administered the placebo, while 14 were given EPA for three months. At the end of the study all 12 who had received the placebo needed antipsychotics, while only eight of the 14 in the EPA group were medicated, and still showed improvement of symptoms. The researchers drew the conclusion that EPA might be helpful in addition to normal medication, yet further research was required to test its usefulness as a sole therapeutic agent.[150]

Another controlled trial tested EPA and placebo alongside three different types of schizophrenia medication – typical antipsychotics, atypical antipsychotics and clozapine (used as a sedative and for treatment-resistant schizophrenia). Although little difference was noted in the patients taking the two antipsychotics, the clozapine patients assigned to receive EPA showed significant improvement over the corresponding placebo group.[151]

A South African trial randomly assigned EPA or placebo as well as medication to chronically ill and severely schizophrenic patients for three months. The EPA group showed significant improvement in symptoms – although, unlike the above study, patients on clozapine did not show improvement.[152]

Out of the five published trials, only one did not find a benefit. A trial in the United States randomly assigned 87 patients to placebo or EPA in addition to their medication for 16 weeks. The results showed no benefit to symptoms or mood.[153]

Antioxidants

As for controlled studies on antioxidants, one study compared 18 patients with schizophrenia with 15 controls, measuring the levels of a free radical known as superoxide. The patients showed a significantly higher level of superoxide when compared to the controls – moreover, the higher the level of superoxide, the higher the level of negative symptoms.[154]

However, trials testing the efficacy of treating schizophrenia with antioxidants and vitamins has been mixed. A double-blind, randomised controlled trial testing vitamin B6 showed no improvement in schizophrenic symptoms,[155] while another controlled trial testing supplementation with megavitamins showed no improvement in symptoms or behaviour over the five-month trial, in spite of raising vitamin levels in the body.[156]

However, a controlled trial supplementing folate-deficient schizophrenic patients with folate did show improvement in symptoms over the six-month trial.[157] Additionally, a birth cohort study in Finland found an association with vitamin D supplementation during early life and a lower incidence of schizophrenia in males (there was no effect on females).[158]

Tardive dykinesia

Most of the trials that showed benefits from the EPA noted particular benefits to patients suffering from tardive dykinesia (TD – also known as Involuntary Movement Disorder), which is a condition caused by the long-term use of neuroleptic medications, such as those used to treat schizophrenia. TD is characterised by a series of involuntary and repetitive movements of the body. The best treatment is to stop the medication, but this is not always practicable. Some of the "improved symptoms" shown in the above controlled trials were actually improvements in the symptoms of TD, not of schizophrenia itself.

Evidence has suggested that TD may be caused by an overproduction of free radicals.[159] As such, a number of trials have been conducted to test whether antioxidants may be helpful in preventing or treating TD. Vitamin E has been tested several times, with varying success. One trial found no improvement in the group receiving this antioxidant, but did find that the symptoms of the control group worsened, while the vitamin E group stayed the same. The suggestion was that vitamin E might help to prevent deterioration in TD patients.[160] A more recent controlled trial using vitamin E did show improvement in the TD patients who received it, while the placebo group showed no improvement.[161]

Because high doses of vitamin E can actually increase oxidation, another trial tested a combination therapy of vitamins E and C (which would work as an antioxidant to the E vitamin's pro-oxidant effect), noting improvements in all of the patients.[162] The trial was small and had no control group, but merits further investigation. Another study has shown a beneficial effect on TD symptoms from vitamin B6.[163]

Summary

No research suggests that schizophrenia is the result of nutritional deficiency. However, epidemiological evidence points to a link between the course and outcome of the disease and dietary intake. Specifically, it points to a link between "western" industrialised diets high in fat and sugar– which are quickly spreading to non-industrialised countries – with a worse outcome for those with schizophrenia. Historical evidence also points to a rise in the severity of the disease at the very time western diets were experiencing drastic change (see the next chapter).Moreover, growing evidence points to a role for certain nutrients in managing the symptoms of both schizophrenia itself and the side-effects of the standard medication. Controlled trials have shown promising results in improving the symptoms of schizophrenia, but more evidence is still needed.

Patients not taking their medication is a serious problem in schizophrenia treatment – unpleasant side effects, such as tardive dykinesia, are often the reason why. Further research into how to manage these symptoms through nutrients with very few side effects would undoubtedly improve both medical treatment for schizophrenia and the health and well-being outcomes for patients.

Dementia, particularly Alzheimer's disease

Dementia is a term used to describe the symptoms resulting from certain diseases that affect the brain, such as stroke and Alzheimer's disease. Alzheimer's is the most common form of dementia. It is a progressive, physical disease of the brain, in which there is a gradual and continual death of brain cells. Symptoms include loss of memory, confusion, mood swings and withdrawal.

Alzheimer's disease has become much more common in the past 50 years, a trend that is believed to be the result of a combination of factors, including age, genetics and environment.[164] Supporting the environmental factor are studies showing that the incidence of dementia in immigrants becomes similar to the incidence in their adopted country.[165,166]

Growing epidemiological evidence suggests that diet may be one of those environmental factors. Supporting this is evidence that there is a connection between Alzheimer's disease and cardiovascular disease,[167] including that drugs used to treat cardiovascular disease also result in a lower incidence of Alzheimer's.[168] It is well accepted that cardiovascular disease is linked to diet, particularly high intakes of saturated fat. The same may also be true for Alzheimer's.

Moreover, high homocysteine levels (homocysteine being an amino acid found in blood, and a metabolism of an essential amino acid), is known to be a risk factor for cardiovascular disease and, increasingly, being considered a risk factor for Alzheimer's. Homocysteine concentrations in the body are strongly influenced by diet, with B vitamins, such as folate, being particularly effective in reducing levels.

Dietary epidemiological evidence

Much of the evidence linking Alzheimer's disease and diet is either epidemiological or the result of long-term cohort studies. Many of the studies have shown a positive association between saturated fat intake and the

incidence of dementia, whilst also showing an inverse relationship between its incidence and PUFA intake.[169] It has also been shown that individuals who eat fewer calories generally have a decreased risk of developing Alzheimer's.[170] Correspondingly, a recent study has linked long-term obesity to a higher risk of developing dementia.[171]

A study looking at the total fat intake of 11 countries found a correlation between higher levels of fat consumption and higher levels of Alzheimer's amongst the over 65s.[172] It has also been observed that elderly Japanese people, known for their high fish consumption, have very low levels of Alzheimer's.[173] The "Mediterranean diet", with its high intakes of monounsaturated fatty acids, has been linked to low rates of dementia in Italy,[174] while high intake of cereals and fish seems to decrease risk in North American and northern European countries.[175]

The "Rotterdam Study", a prospective population-based study, also found a close link between the intake of total fat, specifically saturated fat, and the development of dementia, as well as a lower risk of developing dementia, particularly Alzheimer's disease, with a high fish intake.[176] Another study measuring people's food frequency found that Alzheimer's was more than twice as likely in people with the highest intakes of saturated and hydrogenated (artificially saturated) fats.[177]

Elderly people who ate fish or seafood at least once a week were shown to have a lower incidence of developing dementia, including Alzheimer's, in a cohort study of over 1600 people over seven years.[178] A separate study found the same result, correlating eating fish once a week with a 60 per cent lower risk of developing Alzheimer's.[179]

A different perspective was shown in an investigation into the relationship between the consumption of animal products and the development of dementia. A study compared two groups of meat eaters and vegetarians, with one group matched for age, sex and residence. In the matched groups, the researchers found that the meat (including fish) eaters were more than twice as likely to develop dementia as their vegetarian counterparts. In both cohorts, the vegetarians that developed dementia showed a delay in its onset when compared to the meat eaters.[180] It is unclear whether this outcome was the result of higher fat consumption by the meat eaters or of higher vegetable consumption by the vegetarians.[181]

The possible protection offered by vegetable consumption has also been explored in studies of antioxidants. The Rotterdam Study (described above) found that high intakes of vitamins C and E were linked to a lower risk of Alzheimer's, particularly amongst smokers.[182] A separate study also found that high vitamin E intake

from food was associated with a decreased risk of developing Alzheimer's, but did not find any significant benefit from intake of vitamin C or from vitamin E from supplements.[183]

However, a recent study has found that intake of supplements containing both vitamins E and C did seem to offer protection against developing Alzheimer's.[184] The study followed several thousand elderly people over five years, measuring their supplement intake and comparing it to the incidence of Alzheimer's. In line with the study above, it did not find any benefit from using vitamin E or C alone, or with the use of multi or B vitamins supplements.

Another recent study has suggested that high dietary intakes of folate may be associated with a reduced risk of developing Alzheimer's. By tracking over 500 patients over 14 years, researchers correlated above-recommended intakes of the B vitamin with an eventual lower risk of developing the disease.[185]

Relevant physiology in those with Alzheimer's

A number of physiological studies have shown nutrient-related differences in the bodies of those with Alzheimer's disease. Again, it is not clear whether these are causative of Alzheimer's, or a result of disordered eating as a result of the condition.

Autopsies of brains from Alzheimer's patients have shown low levels of polyunsaturated fatty acids throughout, while other aged brains (compared to younger brains) do not show differences in fatty acid composition.[186] Also, more than one study has shown that Alzheimer's patients show lower levels of PUFAs in their blood when compared to healthy people of the same age.[187,188]

In respect of antioxidants, one study measured the blood of patients with a range of dementias, including Alzheimer's, and found that all of the antioxidants measured were lower in dementia patients than in healthy people. It also found correspondingly higher levels of oxidation of fats.[189]

Another study has also reported low levels of folate and vitamin B12 in the blood of patients diagnosed with Alzheimer's. The study also found elevated levels of homocysteine.[190]

Research trials

There have been only a few clinical trials testing the efficacy of nutrient treatment on patients diagnosed with Alzheimer's disease. The trials that have been completed have been predominantly unsuccessful.

Recently, several reviews have been carried out looking at any controlled trials using B vitamins as a treatment for dementia and Alzheimer's disease. These included folate,[191] B6[192] and B12.[193] The reviewers concluded in each review that no evidence of a benefit could be found.

Another controlled trial tested vitamin E supplementation along with the drug selegiline on over 300 patients over a two-year period. The researchers reported that the treatment appeared to slow the progression of the disease.[194] However, the results do not resolutely point to the vitamin E for this benefit. More recent research on the effect of vitamin E in rats has proved promising, but further tests on human have not been published. Regardless, the American Psychiatric Association does recommend supplementation with vitamin E in the treatment of patients with dementia.[195]

Probably the most promising controlled trial tested the effect of a mixed PUFA supplement on 100 Alzheimer's patients over a period of four weeks. Of the 60 that received the supplement, improvement in mood, co-operation and short-term memory was reported by the guardians of 49 of the patients.[196]

Summary

Although controlled trials have not found much efficacy in treating already developed dementia or Alzheimer's disease with nutrients, the epidemiological evidence does point to a role in the prevention of this disease through diet. Like other diseases, it seems unlikely that one particular nutrient can be separated out as essential – rather that a generally healthy diet may decrease chances of developing dementia and Alzheimer's.

More research is needed to establish whether there is a role for nutrients to help those already diagnosed, whether as a stand-alone treatment or alongside drug therapy.

Conclusion

The previous chapter presented evidence that a healthy brain, influencing mental well being, can be linked to the quality of a person's diet. This chapter has presented further evidence that this link may be a factor in more severe mental health problems, specifically ADHD, depression, schizophrenia and Alzheimer's disease. Clearly, a large and growing number of studies are demonstrating a connection between what a person eats and their experience of mental illness.

As mentioned in the Introduction and in each of the four preceding sections on specific conditions, the global prevalence of mental health problems is increasing. Entirely satisfactory explanations for this increase have not been found. However, taking into account the evidence linking nutrients to brain development, mental well being and mental illness, a possible explanation could be a parallel change in food intake.

This possibility leads us to examine what, if any, changes have occurred to food intake over the same time of the rise in mental health problems. This will now be explored in the next chapter.

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INGREDIENTS: BLEACHED ENRICHED FLOUR, FERROUS SULFATE, THIAMIN MONONITRATE, RIBOFLAVIN, FOLIC ACID, WATER, PARTIALLY HYDROGENATED VEGETABLE OIL (SOYBEAN, COTTONSEED), CREAM CHEESE (MILK, CREAM CULTURES, SALT, CAROB BEAN GUM), COOKE (CURED WITH WATER, SALT, SUGAR, SMOKE SODIUM PHOSPHATE, SODIUM ERYTHORBAT SODIUM NITRITE), EGGS, EGG YOLKS. CONTA OR LESS OF THE FOLLOWING: GLYCERIN, SA YEAST, CORN STARCH, MILK PROTEIN CONCE SODIUM CASEINATE, DEXTROSE, SODIUM PH

Changing diets and the implication for our mental health

Recent decades have seen a substantial global increase in mental ill health. In the previous chapters, we presented a review of the growing body of evidence pointing to a connection between nutrition, behaviour, brain development and mental health. Connecting diet to a rise in mental illness and disorders raises an important question: has the human diet undergone some change over the same period that may be a causal factor?

Human dietary requirements were summarised in the first chapter. Although nutrition is a relatively new science and we are still making discoveries about all the nutrients we need, how they all work, and their function and complex interplay within the body, it is already clear that we need dozens of different nutrients from a wide variety of different foods. Our need for this variety has evolved over millions of years.

Yet the agricultural revolution and increasing world trade, followed by the Industrial Revolution, brought about profound changes in the amount and types of food grown and reared, and how it was processed. Even more rapid changes in our food supply systems occurred after World War II. The result is that humans are now eating a diet unrecognisable to our prehistoric ancestors, even though our nutritional requirements are much the same.

As well as changes in the nutritional balance of our diets, we are also now consuming much larger quantities of human-made chemicals – both deliberately, as legal additives, and accidentally, as agrichemical contamination - than ever before, and in an infinite variety of combinations.

A wide range of physical illnesses is now clearly linked to the diet of highly processed foods produced from an industrialised agriculture that focuses on just a few monocrops for a large proportion of our dietary intake. We have moved progressively away from healthy whole foods such as leafy vegetables, wholegrains, fruit and lean meat, towards a diet rich in fats, salt and sugar and low in essential micronutrients.

Epidemiologists have already recorded the effects of this transition as rising rates of coronary heart disease, some cancers, and a wide range of conditions linked to obesity, such as diabetes. However, much less research, so far, has examined what this same diet might be doing to our minds, as well as our bodies.

An historical perspective

Diet and evolution

Although scientific knowledge of human nutritional requirements is very recent, and indeed continues to develop, humans' need for these nutrients has evolved over millions of years, being both an adaptation to and a product of the environment. A vital ingredient of that environment is food, but the modern diet has changed beyond recognition from the one eaten by early humans.

Throughout the many millions of years of human development, humans lived in small, constantly moving groups following migrant animals and seasonal vegetation. Sustenance was gained by foraging for wild plants and animals, a process representing over 99% of human dietary history.[1] This diet would have included edible roots and leaves, berries and nuts, wild beans, small animals, fish and shellfish and, occasionally, meat from larger animals such as mammoth and bison.[2]

A wide range of foods were eaten, including hundreds of different species of fruits and vegetables.[3] Using evidence from modern primates and hunter-gatherer groups still living in remote parts of the world, it has been estimated that diets might have been made up of between 100 and 150 different plant species annually[4] and animals would have most likely been eaten in their entirety – i.e. not solely the muscle usually eaten today.[5]

Compared to modern humans, the hunter-gatherers would have eaten more protein and more vitamins and minerals. Most carbohydrates would have been derived from fruit, there would have been low to zero levels of saturated fats[6] and, depending on the season, calorie intake would have been quite low.[7] The ratio of omega-3 and omega-6 essential fatty acids would have been nearly equal.[8]

This nomadic, foraging lifestyle lasted for the vast majority of human existence so far, and it is only very recently that major changes have occurred. The first of these was the switch from wholly wild foods to the beginning of food cultivation.

Agricultural revolution

About 10,000 years ago, the earth – in terms of climate, animals and vegetation – entered the period in which we live today. One result was a disappearance of some of the animals traditionally hunted for food.[9] At the same time, archaeological evidence shows the first evidence of formal agriculture, with humans beginning to cultivate plants and domesticate animals for sources of food.

The development of agriculture led to significant changes in the nutrition of these early humans. One was a growing dependency on cereal grains and a decrease in the breadth of the previously highly varied diet.[10] Cereals became the staple food for humans, accounting for 40-90 per cent of calories consumed[11] and replaced fruits and vegetables as the main source of energy. Consumption of fruits and vegetables dropped to 20 per cent or less of total food intake, at the same time reducing the consumption of vitamins and minerals.

As grain consumption increased, meat consumption – particularly wild meat –also decreased. One result was an alteration of the previously equal ratio of omega-3 and omega-6 fatty acids to a higher proportion of omega-6 fats in the diet.[12]

Unsurprisingly, this shift in food and nutrient consumption patterns had a distinct impact. Studies of skeletal evidence point to a marked decline in health when humans moved from the hunter-gatherer way of life to more settled agriculture. Diseases such as osteoporosis, dental caries and anaemia increased[13] and there was also a distinct reduction in stature.[14]

The change from a hunter-gatherer lifestyle to society based around settled agriculture resulted in a relatively fast shift in the human diet. In the space of a few thousand years, a diet that had developed over many millions of years was essentially replaced. Changes continued to gather pace as the years passed – a second agricultural revolution led to greater productivity and, many years later, seafaring led to an explosion of world trade, essentially swapping food that had been available only locally for millions of years. However, this fast rate of change seems positively leisurely when compared with more recent changes, even within living memory.

The Industrial Revolution

Beginning in the late 18th century, the Industrial Revolution greatly altered the way food was produced and consumed. In Britain, just as the population was being pushed off the land due to the enclosures, the rise of the factories was pulling them into the cities. The arrival of large numbers of workers into towns and cities required reliable quantities of cheap food to sustain them.

This necessity led, in part, to a variety of changes both to the way food was prepared and where it came from. New preservation methods – such as canning and freezing – coupled with the steam and transport revolution, allowed grains and other foods to be transported vast distances more quickly and cheaply.[15] The global food trade had begun in earnest, bringing with it novel foods – such as bananas – previously only known to those travelling abroad.

Along with the new preservation methods, around this time the steel roller mill was introduced. Driven by steam engine, the new system ground out white flour more cheaply and efficiently than ever before. This, coupled with the fall in wheat prices, made white bread and new mass-produced foods, such as biscuits,

available to the wider population. Although it was unknown at the time, this method of grinding wheat into fine white flour was changing its micronutrient and fibre composition. Vitamins normally found in wheat were being discarded in the new process, with white flour containing less than a quarter of the zinc, magnesium, vitamin B6 and vitamin E found in wholegrain flour.[16]

Another new factor in food processing was the development of pressing to extract vegetable oils for use in processed foods. As the consumption of these processed foods began to rise, so too did the consumption of omega-6 fats, further tipping the balance against omega-3 fatty acids.[17]

Concurrently, meat consumption increased dramatically. Whereas the average person in Europe consumed only eight ounces of meat per year prior to 1800, this rose to around 100 pounds per person per year during the 19th century.[18]

As the 20th century dawned, only a couple of hundred years had passed since a wholesale change to the human diet had been adopted in Britain, followed all too soon by other industrialising countries. Not only were previously unknown foods introduced, new processing methods were changing traditional foods into unrecognisable new versions of themselves – often missing the very elements which made them nourishing. These changes to the human diet were themselves changed by further developments, particularly since the end of World War II.

Upheaval in the 20th century

In Britain, the first part of the 20th century was marked by increasing global food trade and, due to concerns about malnourished soldiers, increasing government involvement in ensuring the nutritional value of the nation's food supply. There was a growing interest in nutritional science, and knowledge of the causes of malnutrition improved. Of particular note was the discovery of the role of vitamins, followed by the ability to synthesise them in large quantities.

The realisation that malnutrition was not necessarily the result of hunger, but also of poor food quality, led to the practice of fortifying foods with vitamins and minerals. The first food fortification started early in the 20th century with the addition of iodine to salt, in efforts to prevent goitre (an abnormally enlarged thyroid gland). As vitamins were discovered and synthesised, fortification became more widespread, particularly of milk and flour.[19]

The early 20th century also threw up new challenges to the population's diet and nutritional health. At the time of World War I, Britain was only producing about 25 per cent of its own food, with the rest being imported more cheaply from its colonies.[20] But, with the outbreak of the war and resulting German blockades, this reliance on imports became highly problematic. Rationing was introduced in 1918 and again in 1940 during World War II, to ensure that what food was available was shared fairly among the population.

Rationing resulted in a restricted, yet nutritious, diet for the wider population. Limited as it was, it is often suggested that the British population has never been healthier than it was during those years. Certainly, infant mortality decreased and children's general health improved. The rations provided the poor with more protein and vitamins, and the rest of the population ate less meat, fats and sugar.[21] Wartime necessity also advanced food technology, leading to new products such as dehydrated vegetables, dried eggs and new methods for meat processing (most famously Spam).[22]

The end of World War II, and the experience of rationing, brought about a major reassessment of the state of the British food supply. Policies were developed to ensure that farmers could meet the food needs of the population, including direct payments to farmers and price supports. The founding of the European Economic Community (now the European Union) had food security at its core, with the Common Agricultural Policy developed with the express purpose of keeping farmers in business and increasing and sustaining the food supply.

The food supply did indeed increase and rationing was withdrawn from Britain in 1953. This increase in supply, accompanied by technological and social developments, meant that that next 50 years saw yet another dramatic change in the amount and type of food eaten globally.

What are we eating now?

According to the Food and Agriculture Organisation of the United Nations, production of food within the last 30 years has grown faster than the global population. Counting only the food available to humans (i.e., not livestock feed) the world now produces the equivalent of 2,700 calories per person per day, whereas only 30 years ago the amount available was 2,300 calories.[23] What this means is that the existing food production system provides enough food to meet everyone on earth's calorie requirements every single day.

However, there is a glaring inequality in the way that food is distributed globally, with the result that hunger still persists in many parts of the world, alongside over-consumption in others. In most industrialised

countries, calorie intake is in excess of daily requirements. While, for example, sub-Saharan Africa has an average of 2,100 calories per person per day and Bangladesh has an average of 2,200, Western Europe has an average of 3,500 calories available to every person every day - far above the recommended level.[24] One of the more obvious consequences of this change, coupled with other factors such as reduced physical activity, has been the increase in obesity throughout the world.

But simply having more food has not been the only change. The last 50 or so years have also seen remarkable alterations to the types of food we eat, the way we eat it, the way we prepare it, and the way it is produced.

Since 1942 the British government has collected data on the weekly consumption of food in British households, which is then compiled in the annual National Food Survey (now the Expenditure and Food Survey). This was later complimented by the National Diet and Nutrition Survey, which looks in more depth at the dietary habits and nutritional status of the British population on a regular basis. From these unique records, and other surveys, it is possible to describe in great detail the changes in the types of food eaten by the British population, and compare this to dietary patterns in other parts of the world. From such surveys, the following trends can be observed:

- **Cereals.** Currently, the average calorie intake of cereal grains in Europe and the United States is between 10-29 per cent of the total diet.[25] In the UK, the average calorie intake of cereals per person per day is 849 – or 25% of the total daily diet. The vast majority of this comes from wheat, which provides nearly 90 per cent of those calories.[26]

- **Meat.** Consumption began to rise once rationing was withdrawn in the early 1950s; a pattern repeated in the rest of the world. In 1961, the world ate 71 million metric tonnes of meat, but by 2002 this had risen to over 245 million, more than a three-fold increase.[27] In Europe each person eats, on average, nearly 92 kg of meat per year,[28] with the total amount of animal products making up 20-40% of the European diet. The average is slightly lower in the UK, at nearly 81 kg, or about 13 per cent of the total daily diet.[29] In the United States, consumption is higher with an average of 124 kg.[30]

- **Vegetables and fruit.** Since the first National Food Survey, there has been a 34 per cent decline in UK vegetable consumption.[31] Although the current recommendation is to eat at least five portions of fruit and vegetables per day (equating to 400g), the most recent National Diet and Nutrition Survey found that only 13 per cent of men and 15 per cent of women did so, with most eating barely half of that amount.[32] Compared to other European countries, the United Kingdom has one of the lowest intakes of

fruits and vegetables, with an average of just over 200g eaten per person per day.[33]

- **Fish.** Britons eat 59 per cent less fish than when the National Food Survey first began.[34] Nutritional advice is to eat at least 140g of oily fish per week, but much of the UK population fails to do so. In fact, most fail quite spectacularly. On average, seafood contributes only about one per cent of the nation's daily calories.[35] However, the UK is not alone. Across Europe, less than 10 per cent of countries meet what the WHO regards as a "reasonable" consumption of 200g of seafood per person per week.[36]

- **Eggs and milk.** Egg consumption in Britain has more than halved since the 1950s, while milk consumption has been on the decline since the 1970s – down from 2.75 litres per person per week in 1960 to 1.6 litres in 2004.[37]

- **Sugar.** In 1900, global production of sugar was about 8 million tonnes. By 1970 it was 70 million tonnes, the largest increase in agricultural production in any sector over that period.[38] On average, the UK population eats nearly 44 kg of sugar per person per year, though this is less than in the early 1960s, when the average was nearly 53 kg per person per year.

- **Alcohol.** World-wide consumption averages about four litres of pure alcohol per person per year, a rise of 19 per cent between 1990 and 2000.[39] In the UK, 60 per cent of men and 44 per cent of women exceed the recommended alcohol intake.[40] As well as the normal concerns about high alcohol intake, alcohol can often reduce the absorption of minerals from the diet, such as calcium, magnesium and zinc.[41]

- **Infant feeding.** It is widely accepted that breast milk is by far the best for babies, with advantages for immunity, brain development and long-term growth. However, globally only 35 per cent of infants are exclusively breastfed.[42] In the UK, many babies are not breastfed at all and there is a sharp decrease in the breastfeeding of babies after birth. Although 69 per cent of babies in the UK are breastfed as soon as they are born, this drops to only 42 per cent by the age of six weeks and to 13 per cent by nine months.[43] Fortunately, the last ten years have seen increases in breastfeeding in certain countries, including the UK.

- **Poverty.** Surveys of the diets of low-income consumers show significantly smaller intakes of healthier foods (fruits, vegetables, fish and nuts) and higher intakes of sugar, meat products and white bread, than the diets of their wealthier counterparts.[44] As a result, those on low incomes have lower intakes of both vitamins and minerals, particularly vitamin A and iron.[45]

In nutritional (rather than food) terms, the latter half of the 20th century saw increases in the consumption of saturated fats, sodium (salt) and simple carbohydrates (i.e. refined sugar).[46 47] Conversely, intakes of vitamin A, iron, riboflavin, iodine, magnesium and potassium were generally lower than recommended levels.

Processed food

A number of factors prompted the rapid rise in the volume and variety of processed food in the diet. The steady rise in the numbers of women joining the workforce reduced the number of women able or willing to stay at home and cook meals from scratch. To address this social change, there were new developments in labour-saving equipment, and the introduction of convenience food, ranging from an entire meal needing simple heating to snack foods that can be eaten directly from the packaging.

The introduction of convenience foods was an instant and staggering success. Packaged and processed foods have become the norm in most industrialised countries and increasingly throughout the world. The impact can be seen in vivid numbers:

- Since the 1950s, there has been a 70% decline in the purchase of flour – directly attributed to the availability of ready prepared foods.[48]
- There are now approximately 320,000 different packaged food and drink products in the marketplace[49] with more than 20,000 new ones being introduced in 2002 alone (some of which will fail and be withdrawn).[50]
- In an average supermarket, there are approximately 25,000 different products on the shelves[51] - with fresh food making up a very small percentage.
- At the end of 2004, total sales of the top products in the main processed food categories (such as 'bagged' snacks and canned drinks) accounted for over £62 billion in the UK alone.[52]

Many packaged and processed food products are nutritionally inferior to less processed and whole foods. Processed foods tend to be high in calories, fat, salt and/or sugar. Soft drinks, for example, are high in calories and sugar, yet in the UK in 2000, the average person drank 186 litres of soft drinks.[53] Some 75% of salt in the diet comes not from salt added at the table, but from processed foods, including bread and breakfast cereals,[54] with white bread often the single largest source of salt in an individual's diet.[55] Processed foods can also be missing nutritional elements – one study found that canned chicken soup had only a tenth of the beneficial fats of homemade chicken soup.[56]

Processed foods – including those eaten outside the home, as well as in it - have also introduced a new type of fat into the diet – trans fats, also known as hydrogenated fat. Trans fats have no known nutritional benefit, but mounting evidence suggests that they are much less healthy than even standard saturated fats – leading the UK's Food Standards Agency to advise that consumers should minimise the consumption of hydrogenated fats and trans fats to avoid their damaging health effects. Although they exist in small quantities in some animal products, their availability has increased due to the growth of processed foods. In brief, the process of hydrogenation artificially hardens fat to help products keep their shape at room temperature, and to reduce the risk of the fat turning rancid. This makes trans fats useful in prolonging the shelf-life of a product, making them a popular ingredient in manufactured foods such as cakes, biscuits and pastries. It has been estimated that the average adult in the UK eats between two and three grams of trans fat per day.[57]

Alongside trans fats, the rise of processed foods has been accompanied by a rise in the use of soybean oil. Soybeans are grown throughout the world and have been eaten as food for thousands of years, particularly in China. But, in the 1940s, soybeans began to be commercially pressed for oil and its production now surpasses the production of any other oil.[58] Soya oil is now estimated to account for 83% of the calories from fat in the American diet, representing a thousand-fold increase in its consumption since the beginning of the 20th century.[59] This is significant nutritionally because soya oil is higher in omega-6 fatty acids than omega-3s.

Food additives

There is a long history of adding substances to food to improve tastes or prevent spoilage – including smoke, salt, alcohol, spices and vinegar. But it was not until the development of food science, starting in the Industrial Revolution, that

Dramatic changes to our food supply over recent years

- In the UK, 33% of all food expenditure is on food eaten outside of the home[60] – an increase of 40% between 1980 and 2000.[61]
- Fast food sales totalled £7.4 billion in 2003 in the UK, equivalent to £123.80 per person during that year[62], 3% higher than the previous year. Forecasters predict that sales will reach £8 billion by 2008.
- In the US, 50% of all food expenditure is on food outside the home – up from 30% in 1965.[63]
- Fast food sales in the US rose from $6 billon in 1970[64] to $148.6 billion in 2003,[65] and are still rising.

Processed foods also tend to be lower in micro-nutrients than their fresh equivalents. For example, tomato juice can lose 65% of the original tomato's vitamin C, 17% of its niacin and 40% of its carotene.[66]

chemicals began to be added to the food supply in significant amounts. The 1950s saw an explosion in their use, mirroring the rise of processed foods and, by the beginning of the 1960s, there were 2,500 different chemicals being used in food production.[67] Approximately the same number are being used today, with one source claiming at least 3,850.[68]

Without additives, many modern processed foods simply could not exist. Additives can work to prevent food poisoning, prevent spoilage, add micro-nutrients and increase the range of foods available. However, 90% of additives can only be considered "cosmetic", changing the colour or flavour of the food, not its safety or nutritional quality.[69] The largest number of additives are flavourings, with 4,500 different chemical compounds used to simulate different tastes.[70]

Food sweeteners, such as aspartame and saccharin, are not included in the flavouring category, but rather are classified as foods. Sweeteners have no calories and are used to replace sugar in a processed food, or sold as products themselves. There are currently thirteen different types, with aspartame the most widely used[71] and having a global market of $2.5 billion annually.[72]

The widespread use of food additives means that anyone who eats any amount of processed food will ingest them. It has been estimated that the average person in the UK and other industrialised countries will eat over four kilos of additives every year.[73]

The use of food additives has always been regulated, even during the Middle Ages.[74] Accordingly, the growth in additive use since the 1950s has been accompanied by assessment and regulation by international organisations and national governments. Regulation by national governments is usually based on the advice of the joint FAO/WHO Expert Committee on Food Additives (JECFA).[75] The JECFA provides "acceptable daily intake values" which are estimates of how much of a certain chemical can be ingested safely by the average adult consumer throughout a lifetime.[76] These regulations take the form of bans on certain additives and approval of others, often with labelling requirements and/or limits on the amounts used.

However, despite this body of regulation, concerns remain about the health effects of food additives. First, approvals and safety limits are predominantly based on the results of tests on animals, which are then assumed to apply to humans.[77] Moreover, these animal tests examine whether a certain additive could cause cancer or genetic mutations – other issues (such as effects on behaviour or mood) are not investigated. Most problematic is the difficulty posed by the so-called "cocktail effect" that occurs when additives are inevitably combined in unpredictable mixtures in people's diets.

Industrialised farming

Even unprocessed food has been changing in recent decades. Since the drive to increase agricultural output began in earnest after World War II, there have been a number of changes in crop production and animal farming that influence both the quantity and quality of the food consumed in the 21st century.

The genetic diversity of food crops is declining, with the result that only an estimated ten per cent of the variety of crops eaten over human history are still being farmed today.[78] Only nine crops are responsible for over three-quarters of the plants eaten.[79] Some 97 per cent of the varieties of fruits and vegetables found in America in 1900 are now extinct,[80] along with 83 per cent of the varieties of wheat developed in the Middle East.[81] Only four of the over 5,000 varieties of potatoes are grown commercially[82] and large numbers of animals, including cow and sheep varieties, are now extinct. Relying on such a narrow range of plants and animals has a number of worrying consequences, not least the likely reduction in the range of micro-nutrients and trace elements we eat.

In addition, several studies[83] comparing the nutrient composition tables for certain foods have suggested that continuous replanting and then inadequate re-fertilisation of the soil is leading to poor soil quality, including a loss of minerals. This, in turn, may be leading to a loss of minerals in the food grown in that soil, and the studies show significant losses of copper, magnesium, calcium, zinc and iron, amongst others. However, other studies suggest there is no such loss, arguing that the studies are not comparing like with like[84] and are misinterpreting data by not taking into account different soil and climatic conditions.[85]

This is clearly an area requiring clarification, but a recent study, which compared the measurements over 15 years from the actual soil (as opposed to the resulting crops) did indeed find a decline in the levels of zinc and magnesium in the soil in England and Wales.[86]

Also potentially affecting the micro-nutrient composition of the food we eat is the distance it travels to get to our plate. "Food miles" is the term often used to describe how far a food travels from the place it is grown to the place it is eaten. It is difficult and complex to estimate, but one study calculated that the food purchased in an average UK shopping trip will have travelled over 3,000 kilometres.[87] The environmental toll of so much long-distance transport is of great concern in its own right but, in addition, it could also be reducing the micro-nutrient density of modern diets. More research is required, but it is thought that the shorter the time between harvest and consumption of a food, the higher the nutrient content of that food.[88]

Vitamins, particularly A, C, E, riboflavin, folate and thiamine, can easily be lost through long storage, and exposure to light, oxygen and heat.[89]

Animal fat

The concern over declining nutritional quality is not confined to plant foods. There is also unease about the change in animals reared for food. A number of studies indicate that intensively reared animals have higher ratios of saturated fat to quality protein than wild species, or those less intensively reared.[90]

The poultry industry has grown dramatically: whereas the worldwide production of chicken in 1961 was below 9 million metric tonnes, in 2002 it was over 73 million.[91] The result has been considerably intensified production techniques. Intensively farmed chickens now reach their slaughter weight twice as fast as they did only 30 years ago. One result has been a change in the nutritional profile of chicken meat. Whereas a chicken carcass used to be 2 per cent fat, it is now 22 per cent. The diet fed to chickens is, of course, no longer insects, seeds and plants, but commercially grown cereals and soya-based foods. This new diet has reduced levels of omega-3 fatty acids and increased omega-6 fatty acids in chicken meat.[92]

Over 30 years ago, the same issue was reported in cattle – a comparison of domestic, intensively reared cattle and wild bovids found that the carcass of the domestic animal contained 30 per cent fat, whereas the wild species contained five per cent.[93] A further difference between the meat eaten today and the meat eaten in the past is the presence of marbling, or the presence of veins of fat throughout the muscle tissue of domestically reared animals – wild species do not possess such marbling.[94]

Because of the less intensive methods of production, there is some evidence that organic farming can lead to animals with better nutritional profiles. For example, milk from organic cows is reported to contain higher levels of omega-3 fatty acids and vitamin E than milk from intensively-reared cows.[95] This is attributed to the organic cows' diet of clover and grass.

The story of fish is similar. Due in part to concerns about falling levels of wild fish stocks, modern aquaculture (fish farming) was developed in the 1960s. It is now the fastest growing form of food production in the world.[96] Now, much of the fish found in supermarkets comes from fish farms.

The environmental impact of fish farming is alarming, as are the potential nutritional consequences. Farmed fish are fed fish meal which, alongside fish oil and flesh, may contain soya, wheat, ash and poultry by-products.[97] As with chicken, one result is a change in farmed fish's nutritional profile. Farmed fish, due to confinement and overfeeding, have fattier flesh. And once again, the ratio of omega-3 to omega-6 fatty acids in that fat is altered towards higher levels of omega-6. Wild Atlantic salmon, for example, has a ratio of omega-3 to omega-6 of 3.9 to 1, while in farmed Atlantic salmon the ratio is 1.1 to 1.[98]

There are also concerns about fish contamination. Nearly all fish – wild or farmed - contain traces of mercury[99] and there is a widespread presence of dioxins and polychlorinated biphenyls (PCBs) throughout the entire fish stock. In 2000, it was found that 83 per cent of oily fish were contaminated, with some farmed salmon containing 16 times more PCBs than wild salmon.[100]

The result is confusion among consumers and dietary advisors over how much fish is safe to eat, regardless of its nutritional benefits.

Pesticides

Although, like contaminants, pesticides are not an intentional part of the diet, the use of pesticides in farming has contributed to change in the food we eat. Since the end of World War II, the world has been introduced to a huge arsenal of synthetic chemicals in the battle against agricultural pests. One result has been the enormous increase in agricultural output seen in the past 50 years. Another has been environmental degradation as the chemicals built up in soil, water and animal flesh. This also means new and toxic chemicals are in the food supply.

About 350 different pesticides are presently used in conventional farming,[101] with 31,000 tonnes sprayed every year in the UK alone.[102] Throughout the past few decades of modern pesticide usage, concerns have been raised about toxicity to humans, sometimes resulting in a ban on the use of certain pesticides. However, these bans operate mostly at a national level, meaning they are still sold and used in other areas of the world. For example, the organochlorines used widely in the middle of the 20th century were banned in many countries, including the UK. However, these chemicals are still used in other countries, including in some that export food to Britain. Some of these pesticides still in use are raising concern due to their possible impact on the nervous and reproductive systems.[103]

Like additives, pesticides used in food production are regulated by governments and international agencies. In the UK, this takes the form of the Maximum Residue Level (MRL). However, MRLs are not safety limits; rather they check that the pesticide is being used in the approved way and "*assist international trade in produce treated with pesticides*".[104]

Also like additives, the testing and regulation of pesticides does not take into account the inevitable mixing that occurs in a diet made up of different foods. This again raises concerns about the "cocktail effect" where many different chemicals are ingested together. Recent research undertaken by the UK's Food Standards Agency states that any risk would be small, but admits that the evidence is limited and not easily predictable.[105] Again, as with food additives, pesticides testing assumes an adult consumer, and there is no testing for more vulnerable groups such as children and foetuses.[106]

Although there are concerns about the health effects of pesticides, we are regularly ingesting them in the food we eat. Testing for residues by the Pesticide Residues Committee (PRC) in 2002 found that nearly 43% of all the fruits and vegetables tested contained residues, with some exceeding the approved limit.[107] This included 78% of the apples and 50% of the lettuces tested.[108] This is not confined to fruits and vegetables, as 30% of the cereals and 78% of the bread tested also showed residues.[109] In addition, most of the produce tested by the PRC between 1998 and 2003 showed residues of multiple pesticides, including in 82% of oranges and 42% of strawberries tested.[110]

And the results?

Clearly, the diets that humans eat in the 21st century are dramatically different from the diets of our ancestors, even our very recent ones. After literally millions of years of evolving and adapting to a highly varied diet, containing a wide range of nutrients, the last 200 years have seen the diversity of the food supply shrink drastically.

True, we have more food than our ancestors could ever have wished for. Yet much of the modern diet contains "*too much fat in general, too much hard, saturated fat in particular, too much sugar and salt and not enough fibre.*"[111]

Moreover, much of our food is dosed with synthetic chemicals, few of which have been thoroughly tested individually, much less as a mixture. Soil depletion and other consequences of modern farming methods may

mean some foods are losing the very nutrients that used to make them good to eat.

The result of all these changes has been a rise in serious health problems that are in part attributed to the modern diet, including obesity, coronary heart disease, diabetes, cancer, osteoporosis and dental caries.[112] Other problems, such as rising levels of some cancers, may be linked to some chemicals added to our food, but a causal link is very difficult to establish.

Coupling this with the evidence presented in previous chapters raises the obvious question. Is it possible that the same changes that are contributing to our rising physical health problems are also contributing to our rising mental health problems?

Conclusion and Recommendations

Over recent decades there has been a marked and worrying rise in mental health and behavioural problems. Indeed, by 2020, depression is projected to be the second highest cause of the global burden of disease. The costs are borne by individuals and society - both in personal anguish and in lost working years - and the healthcare bill for psychiatric services continues to rise inexorably.

At the same time, around the world, diet-related physical conditions are also on the increase, such as heart disease, diabetes, certain cancers and obesity. Given that our assembling of the scientific research points to strong links between nutrition and mental health, it seems likely that these two trends – in both mental and physical health – are intimately related.

This report, therefore, raises both questions and challenges. If diet-related diseases are on the increase, then we can conclude that at least some proportion of these diseases is preventable. At the very least, some of their symptoms could be mitigated by improvements in nutrition. But where should we look for the root causes of changes that could have such profound effects on physical and mental health across whole populations?

The trends in physical and mental health have taken place side by side with enormous changes in the food system and people's lifestyles over the past century, particularly since the end of World War II. It can be argued (and indeed it is also widely accepted, including by the World Health Organisation) that our agricultural policies, food marketing, food education and industrial food production (described together as the 'food system'), in conjunction with sedentary lifestyles, all play a role in contributing to growing rates of coronary heart disease, diabetes and certain cancers.

In this report, we have presented the scientific evidence linking nutrition to brain and mental health. Just like other bodily organs, the brain is not impervious to poor diet. An unbalanced diet containing too many unhealthy ingredients, or lacking in essential nutrients is likely to have consequences for brain function and development. The research reviewed in this report suggests that such deficiencies and imbalances can then be reflected in a person's mental health and behaviour – from minor mood changes through to more severe psychiatric conditions. We contend therefore that, just like physical health, mental health is also partly dependent on the food system that feeds it.

Looking at the evidence, what is perhaps most striking is the wide range of nutrients that appear to be important for brain and mental health, with these nutrients working in combination to provide what the brain needs to develop and function effectively. In the field of physical health, it is well understood that health promotion rarely boils down to advice to increase consumption of single nutrients in isolation. Health benefits arise from consumption of a varied and balanced diet based largely on complex carbohydrates, essential fats, high quality protein, and foods rich in micronutrients – in other words, a diet rich in fruits and vegetables, wholegrain cereals, pulses, nuts, seeds and animal products such as lean meat and oily fish. It is widely accepted, based on population studies, that such diets help to build and maintain healthy organs and are protective against certain diseases.

Our examination of the research indicates that this is also likely to be true for brain health. Mental health benefits are also associated with a varied and balanced diet that delivers the full range of nutrients needed for the brain to function properly. Moreover, the same factors that seem to cause damage to the brain are those linked to problems with the rest of the body, e.g. saturated fat, trans fats (from artificially hydrogenated fat in processed foods) and sugar. Consequently, the dietary advice needed to build and maintain a healthy brain is really no different from the advice for a healthy body.

This healthy eating advice might seem straightforward. However, most people do not achieve even the most basic of dietary goals, such as consuming five portions of fruit and vegetables, eating two portions of fish a week, or basing most of their meals on complex carbohydrates from wholegrain sources. There are many factors that affect people's choices: lack of information and education; highly effective marketing of unhealthy foods; cost and availability of healthy options; unhealthy ingredients in everyday processed foods; low-cost and low-quality catering in schools, hospitals and prisons; and a host of other personal, lifestyle and economic reasons well-documented by public health organisations.

Moreover, evidence is growing that even if a healthy diet were to be adopted, the basic ingredients of foodstuffs may be of lesser nutritional value than they were in the past, due to changes in plant and animal varieties, agricultural methods and storage times. These factors may also affect physical and mental health. Micronutrients and essential fats are just some of the vital food components that appear to have declined in many important foods due to production methods – yet these are the very types of nutrients that our review of the research suggests may be most important for mental health. Meanwhile, potentially damaging components (such as artificial trans fats, food additives and pesticides) have been introduced into our food system over recent decades.

We hope that this report will convince policymakers that, unless methods of achieving healthier diets and improving the quality and sustainability of the food supply are given higher priority in government policy, the costs of treating mental, physical and behavioural problems will continue to increase. And as we have shown, those costs are already enormous.

We conclude that our food system holds a key to some of the fundamental causes of the growing physical and mental health problems witnessed in recent decades. Recognition of these links could lead (at the very least) to a new approach to treatment, better advice to patients, and better food in psychiatric wards, to give the best possible chances to some of the most vulnerable people in our society. However, the significance of this research does not stop at the doors of mental health services. We could all benefit from a better

Fish stocks - no more food for thought?

Although the brain appears to work best with a generally varied and healthy diet, one of the nutrients implicated in many studies as affecting mental health is the lack of the essential fatty acid, omega-3. Oily fish are a rich source of these fatty acids and the UK government recommends eating two portions of fish every week (of which one should be oily fish) because omega-3s are also linked to a reduced risk of heart disease. However, most of the population fails to act on this advice.

Many studies on diet and mental health have looked specifically at fish consumption for indications of the connection between omega-3 intake and mental health – for instance finding lower rates of depression amongst fish-eating populations and better behaviour amongst children receiving fish-oil supplements. The result has been a good deal of coverage in the popular press extolling the virtues of fish and fish oils for the brain.

However, as the Royal Commission on Environmental Pollution has recently highlighted,1 there is serious concern about the falling level of fish stocks and with it, the availability of this vital food. Presently, over 52 per cent of the world's fisheries are fully exploited, with 17 per cent being over exploited. Put simply, if trends continue as they are, there may be no more fish in the seas. Furthermore, if the general population was indeed to act on the advice to eat a portion of oily fish a week, without commensurate conservation policies in place, this would simply accelerate the extinction process.

Moreover, as mentioned in the previous chapter, fish can be contaminated with mercury and dioxins, which accumulate through the ecosystem, and become concentrated through the food chain in higher organisms such as fish. Currently, women who are pregnant or may become pregnant are advised by the UK's Food Standards Agency to limit consumption of certain fish in order to avoid contaminants.2 But, as we saw in the second chapter, pregnancy is one of the most important periods for brain development. Without a good source of omega-3 fats from fish during this early stage, what are the consequences for future brain development and mental health?

As such, we are facing a dilemma. How can we recommend increasing fish consumption for brain health, when doing so may destroy future generations' access to this highly beneficial food? And, how can we can we ensure that babies' brains are given the best start in life, when doing so may cause damage through mercury or dioxin poisoning?

There isn't a simple answer. However, much more could be done to help sustain fish stocks and stop the poisoning of the oceans. And policies to do so could find useful support from both environmental and health ministries, working together towards a common and mutually beneficial goal, for the benefit of all.

understanding of how food affects both physical and mental health. This report should, for instance, lend new impetus to food manufacturers to create and market healthier products; and to government initiatives to track these improvements and set standards for food quality – especially for children. It might also prompt us to take a far-reaching look at the way we produce and provide food, and the agricultural and industrial processes that plant the seeds of our mental and physical well-being.

The understanding of the links between food and mental health might also lead us to conclude that threats to natural resources – such as the loss of plant and animal diversity in farming and the imminent collapse of marine fish stocks – are worthy of urgent government attention (see box) not just for historic or conservation value, but also to contribute to the mental well-being of ourselves and our descendants.

What policy makers could do now

The Government as a whole, and all relevant departments and agencies, should officially recognise the links between diet and mental health and incorporate this recognition into all food and mental health-related policy and practice. For instance, general healthy eating campaigns such as five-a-day should always include a mental health component.

Because the diet that is good for the brain is also the same diet that is good for the body, government should increase financial and political support for measures to ensure that sustainable[a] supplies of a wide variety of nutrient-rich foods are available, affordable and attractive for people to obtain both now and in the future.

Specifically:

1. The UK population and particular groups who are at increased risk of mental health problems should be provided with information about foods that promote their mental, emotional and physical well-being.

   Stakeholders: Department of Health (E)[b], Health Scotland (S),Health and Social Care. Department (W), Department of Health, Social Services and Public Safety (NI), Food Standards Agency (UK)

2. United Kingdom Health Departments should review and improve the food and nutrition standards for the mental health and social care sectors, in light of the evidence that a range of nutrients contribute to mental health and wellbeing.

   Stakeholders: Department of Health (E), Health Scotland (S), Health and Social Care Department (W), Department of Health, Social Services and Public Safety (NI)

a We agree with the Brundtland definition of sustainability as 'meeting the needs of the present without compromising the ability of future generations to meet their own needs'. This incorporates all three pillars of sustainable development: environmental, social and economic.
b E=England, NI=Northern Ireland, S=Scotland, W=Wales

3. Organisations that commission mental health services should include within commissioning criteria and service specifications food and nutrition standards for any services that provide food.

   Stakeholders: Primary Care Trusts (E), Local Authorities (E, S, W, NI), Health Boards (S), Local Health Boards (W), Health and Social Services Boards/Trusts (NI)

4. Annual monitoring of food and nutrition standards across the health and social care sector should be incorporated into current performance assessment mechanisms.

   Stakeholders: Healthcare Commission (E, W), Commission for Social Care Inspection (E), Care Commission (S), Quality Insurance Scotland (S), Mental Welfare Commission (S), Health Inspectorate Wales (W), Care Standards Inspectorate (W), Northern Health and Social Services Council (NI), Department of Health, Social Services and Public Safety (NI)

5. Primary care should have ready access to information on the link between diet and mental health as well as a working knowledge of the information and expertise available to support people through dietary change.

   Stakeholders: Primary Care Trusts (E), Health Boards (S), Local Health Boards (W), Health and Social Services Boards/Trusts (NI)

6. Secondary mental health service staff should have ready access to nutrition specialists for liaison and consultation.

   Stakeholders: Mental Health Trusts (E), Health Boards (S), Local Health Boards (W), Health and Social Services Boards/Trusts (NI)

7. All existing NHS and social care facilities that provide meals to service users, including the independent and not for profit sector, should instigate sustainable food policies and practices, so that all service users and staff are encouraged to choose diverse and culturally appropriate meals, snacks and drinks that promote their mental, emotional and physical well-being.

   Stakeholders: Strategic Health Authorities (or their successor) (E), Local Health Boards (W), Health Boards (S), Health and Social Services Boards/Trusts (NI), Local Authorities (E, S, W, NI),

8. All prison facilities should instigate sustainable food policies and practices so that all residents and staff are encouraged to choose culturally diverse and appropriate meals, snacks and drinks that promote their mental, emotional and physical well-being.

   Stakeholders: Home Office (E, W), Scottish Executive (S), Northern Ireland Office (NI)

9. Research funding bodies should co-ordinate their strategies and increase the grants available to investigate the relationship between diet and mental health, particularly the effectiveness of interventions.

   Stakeholders: Department of Health and particularly its Research Funders Group (E), Scottish Executive Health Department and National Programme for Improving Mental Health and Well-being (S) Health and Social Care Department (W), Department of Health, Social Services and Public Safety (NI), Food Standards Agency (UK)

10. Regulations should be introduced to support the promotion of healthy food to children, and to protect them from all forms of broadcast and non-broadcast marketing of unhealthy food.

    Stakeholders: Department for Culture Media and Sport (UK), Ofcom (UK), The Department of Health's Advertising Forum (E), Department of Health (E) Health Scotland (S), Scottish Executive (S) Health and Social Care Department (W), Department of Health, Social Services and Public Safety (NI)

11. Practical food skills, including cooking and growing, should be reintroduced as a compulsory part of the national curriculum.

    Stakeholders: Department for Education and Skills (E, W), Health Promoting Schools (S), Scottish Executive Education Department (S), Department of Education (NI)

12. The progressive approach to ensuring better food in school meals should be continued and in addition access to free water dispensers should be available to all children by 2007.

    Stakeholders: The School Food Trust (E, W), Department for Education and Skills (E, W), Health Promoting Schools (S), Scottish Executive Education Department (S), Department of Education (NI)

13. Targets should be introduced to reduce unhealthy levels of fat, sugar and salt in processed food, and to remove damaging trans fats from food ingredients and food products. As an interim measure, manufacturers should be encouraged to label clearly the nutritional quality of and ingredients in their products.

    Stakeholders: Food Standards Agency (UK)

14. Agricultural policy development should be informed by what is known of its nutritional impact and its subsequent effect upon our mental as well as physical health. Specifically, support must be increased for organic farming, the production and promotion of fruit and vegetables and other micronutrient-rich foods and for alternative sources to oily fish of omega-3 fats. Moreover, government policy on promoting fish consumption needs to change to promoting only sustainable sources of oily fish, with low levels of toxicity.

    Stakeholders: Department for the Environment, Food and Rural Affairs (E, W), Department of Agriculture and Rural Development (NI), Food Standards Agency (UK), Department of Environment and Rural Affairs (S)

Contact Organisations

The following are UK-based organisations that work specifically on the connection between diet and mental health and behaviour. It is not a definitive list and does not include many organisations that may provide information about the connection along with their other work.

Food and Behaviour Research
Box 6066
Nairn
Scotland
IV12 4YN
www.fabresearch.org

The Food and Mood Project
Box 2737
Lewes, East Sussex
BN7 2GN
www.foodandmood.org

The Hyperactive Children's' Support Group (HACSG)
71 Whyke Lane,
Chichester, West Sussex
PO19 7PD
www.hacsg.org.uk

Institute of Brain Chemistry and Human Nutrition
North Campus
London Metropolitan University
166-220 Holloway Rd.
London
N7 8DB
www.north.londonmet.ac.uk/ibchn

Institute of Optimum Nutrition - Brain Bio Centre
13 Blades Court
Deodar Road
Putney
London
SW15 2NU
www.brainbiocentre.com

Natural Justice
University Laboratory of Physiology,
Parks Road
Oxford
OX1 3PT
www.physiol.ox.ac.uk/natural.justice

The Schizophrenia Association of Great Britain
"Bryn Hyfryd", The Crescent,
Bangor. Gwynedd
LL57 2AG
www.sagb.co.uk

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CHANGING DIETS AND THE IMPLICATION FOR OUR MENTAL HEALTH

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CONCLUSION AND RECOMMENDATIONS

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Changing Diets, Changing Minds:
how food affects mental well being and behaviour

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the alliance for better food and farming

Sustain: The alliance for better food and farming is a registered charity which advocates food and agriculture policies and practices that enhance the health and welfare of people and animals, improve the living and working environment, enrich society and culture, and promote equity. We represent around 100 national public interest organisations working at international, national, regional and local level.

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Mental Health Foundation

Founded in 1949, the Mental Health Foundation is the leading UK charity working in mental health and learning disabilities. The Foundation exists to help people survive, recover from and prevent mental health problems.

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