

Food and mood: how do diet and nutrition affect mental wellbeing?

Poor nutrition may be a causal factor in the experience of low mood, and improving diet may help to protect not only the physical health but also the mental health of the population, say **Joseph Firth and colleagues**

Depression and anxiety are the most common mental health conditions worldwide, making them a leading cause of disability.¹ Even beyond diagnosed conditions, subclinical symptoms of depression and anxiety affect the wellbeing and functioning of a large proportion of the population.² Therefore, new approaches to managing both clinically diagnosed and subclinical depression and anxiety are needed.

In recent years, the relationships between nutrition and mental health have gained considerable interest. Indeed, epidemiological research has observed that adherence to healthy or Mediterranean dietary patterns—high consumption of fruits, vegetables, nuts, and legumes; moderate consumption of poultry, eggs, and dairy products; and only occasional consumption of red meat—is associated with a reduced risk of depression.³ However, the nature of these relations is complicated by the clear potential for reverse causality between diet and mental health (fig 1). For example, alterations in food choices or preferences in response to our temporary psychological state—such as “comfort foods” in times of low mood, or changes in appetite from stress—are common human

experiences. In addition, relationships between nutrition and longstanding mental illness are compounded by barriers to maintaining a healthy diet. These barriers disproportionality affect people with mental illness and include the financial and environmental determinants of health, and even the appetite inducing effects of psychiatric medications.⁴

While acknowledging the complex, multidirectional nature of the relationships between diet and mental health (fig 1), in this article we focus on the ways in which certain foods and dietary patterns could affect mental health.

Mood and carbohydrates

Consumption of highly refined carbohydrates can increase the risk of obesity and diabetes.⁵ Glycaemic index is a relative ranking of carbohydrate in foods according to the speed at which they are digested, absorbed, metabolised, and ultimately affect blood glucose and insulin levels. As well as the physical health risks, diets with a high glycaemic index and load (eg, diets containing high amounts of refined carbohydrates and sugars) may also have a detrimental effect on psychological wellbeing; data from longitudinal research show an association between progressively higher dietary glycaemic index and the incidence of depressive symptoms.⁶ Clinical studies have also shown potential causal effects of refined carbohydrates on mood; experimental exposure to diets with a high glycaemic load in controlled settings increases depressive symptoms in healthy volunteers, with a moderately large effect.⁷

Although mood itself can affect our food choices, plausible mechanisms exist by which high consumption of processed carbohydrates could increase the risk of depression and anxiety—for example, through repeated and rapid increases and decreases in blood glucose. Measures of glycaemic index and glycaemic load can be used to estimate glycaemia and insulin demand in healthy individuals after eating.⁸ Thus, high dietary glycaemic

load, and the resultant compensatory responses, could lower plasma glucose to concentrations that trigger the secretion of autonomic counter-regulatory hormones such as cortisol, adrenaline, growth hormone, and glucagon.⁹ The potential effects of this response on mood have been examined in experimental human research of stepped reductions in plasma glucose concentrations conducted under laboratory conditions through glucose perfusion. These findings showed that such counter-regulatory hormones may cause changes in anxiety, irritability, and hunger.¹⁰ In addition, observational research has found that recurrent hypoglycaemia (low blood sugar) is associated with mood disorders.⁹

The hypothesis that repeated and rapid increases and decreases in blood glucose explain how consumption of refined carbohydrate could affect psychological state appears to be a good fit given the relatively fast effect of diets with a high glycaemic index or load on depressive symptoms observed in human studies.⁷ However, other processes may explain the observed relationships. For instance, diets with a high glycaemic index are a risk factor for diabetes,⁵ which is often a comorbid condition with depression.^{4 11} While the main models of disease pathophysiology in diabetes and mental illness are separate, common abnormalities in insulin resistance, brain volume, and neurocognitive performance in both conditions support the hypothesis that these conditions have overlapping pathophysiology.¹² Furthermore, the inflammatory response to foods with a high glycaemic index¹³ raises the possibility that diets with a high glycaemic index are associated with symptoms of depression through the broader connections between mental health and immune activation.

Diet, immune activation, and depression

Studies have found that sustained adherence to Mediterranean dietary patterns can reduce markers of inflammation in humans.¹⁴ On the other hand, high calorie

KEY MESSAGES

- Healthy eating patterns, such as the Mediterranean diet, are associated with better mental health than “unhealthy” eating patterns, such as the Western diet
- The effects of certain foods or dietary patterns on glycaemia, immune activation, and the gut microbiome may play a role in the relationships between food and mood
- More research is needed to understand the mechanisms that link food and mental wellbeing and determine how and when nutrition can be used to improve mental health

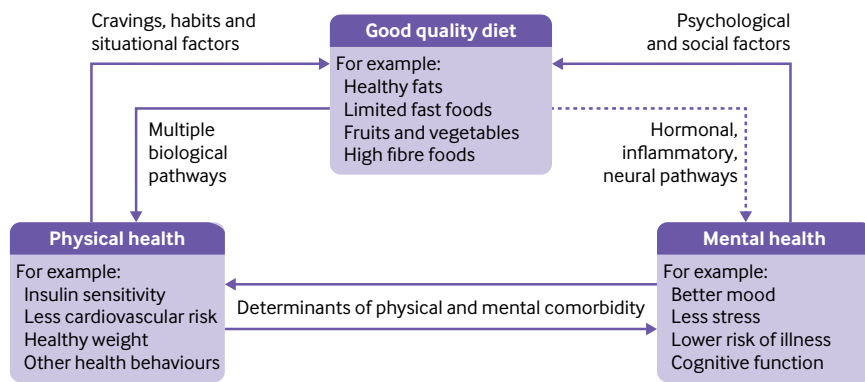


Fig 1 | Hypothesised relationship between diet, physical health, and mental health. The dashed line is the focus of this article.

meals rich in saturated fat appear to stimulate immune activation.^{13 15} Indeed, the inflammatory effects of a diet high in calories and saturated fat have been proposed as one mechanism through which the Western diet may have detrimental effects on brain health, including cognitive decline, hippocampal dysfunction, and damage to the blood-brain barrier.¹⁵ Since various mental health conditions, including mood disorders, have been linked to heightened inflammation,¹⁶ this mechanism also presents a pathway through which poor diet could increase the risk of depression. This hypothesis is supported by observational studies which have shown that people with depression score significantly higher on measures of “dietary inflammation,”^{9 3 17} characterised by a greater consumption of foods that are associated with inflammation (eg, trans fats and refined carbohydrates) and lower intakes of nutritional foods, which are thought to have anti-inflammatory properties (eg, omega-3 fats). However, the causal roles of dietary inflammation in mental health have not yet been established.

Nonetheless, randomised controlled trials of anti-inflammatory agents (eg, cytokine inhibitors and non-steroidal anti-inflammatory drugs) have found that these agents can significantly reduce depressive symptoms.¹⁸ Specific nutritional components (eg, polyphenols and polyunsaturated fats) and general dietary patterns (eg, consumption of a Mediterranean diet) may also have anti-inflammatory effects,^{14 19 20} which raises the possibility that certain foods could relieve or prevent depressive symptoms associated with heightened inflammatory status.²¹ A recent study provides preliminary support for this possibility.²⁰ The study shows that medications that stimulate inflammation typically induce depressive states in

people treated, and that giving omega-3 fatty acids, which have anti-inflammatory properties, before the medication seems to prevent the onset of cytokine induced depression.²⁰

However, the complexity of the hypothesised three way relation between diet, inflammation, and depression is compounded by several important modifiers. For example, recent clinical research has observed that stressors experienced the previous day, or a personal history of major depressive disorders, may cancel out the beneficial effects of healthy food choices on inflammation and mood.²² Furthermore, as heightened inflammation occurs in only some clinically depressed individuals, anti-inflammatory interventions may only benefit certain people characterised by an “inflammatory phenotype,” or those with comorbid inflammatory conditions.¹⁸ Further interventional research is needed to establish if improvements in immune regulation, induced by diet, can reduce depressive symptoms in those affected by inflammatory conditions.

Brain, gut microbiome, and mood

A more recent explanation for the way in which our food may affect our mental well-being is the effect of dietary patterns on the gut microbiome—a broad term that refers to the trillions of microbial organisms, including bacteria, viruses, and archaea, living in the human gut. The gut microbiome interacts with the brain in bidirectional ways using neural, inflammatory, and hormonal signalling pathways.²³ The role of altered interactions between the brain and gut microbiome on mental health has been proposed on the basis of the following evidence: emotion-like behaviour in rodents changes with changes in the gut microbiome,²⁴ major depressive disorder

in humans is associated with alterations of the gut microbiome,²⁵ and transfer of faecal gut microbiota from humans with depression into rodents appears to induce animal behaviours that are hypothesised to indicate depression-like states.^{25 26} Such findings suggest a role of altered neuroactive microbial metabolites in depressive symptoms.

In addition to genetic factors and exposure to antibiotics, diet is a potentially modifiable determinant of the diversity, relative abundance, and functionality of the gut microbiome throughout life. For instance, the neurocognitive effects of the Western diet, and the possible mediating role of low grade systemic immune activation (as discussed above) may result from a compromised mucus layer with or without increased epithelial permeability. Such a decrease in the function of the gut barrier is sometimes referred to as a “leaky gut” and has been linked to an “unhealthy” gut microbiome resulting from a diet low in fibre and high in saturated fats, refined sugars, and artificial sweeteners.^{15 23 27} Conversely, the consumption of a diet high in fibres, polyphenols, and unsaturated fatty acids (as found in a Mediterranean diet) can promote gut microbial taxa which can metabolise these food sources into anti-inflammatory metabolites,^{15 28} such as short chain fatty acids, while lowering the production of secondary bile acids and p-cresol. Moreover, a recent study found that the ingestion of probiotics by healthy individuals, which theoretically target the gut microbiome, can alter the brain’s response to a task that requires emotional attention²⁹ and may even reduce symptoms of depression.³⁰ When viewed together, these studies provide promising evidence supporting a role of the gut microbiome in modulating processes that regulate emotion in the human brain. However, no causal relationship between specific microbes, or their metabolites, and complex human emotions has been established so far. Furthermore, whether changes to the gut microbiome induced by diet can affect depressive symptoms or clinical depressive disorders, and the time in which this could feasibly occur, remains to be shown.

Priorities and next steps

In moving forward within this active field of research, it is firstly important not to lose sight of the wood for the trees—that is, become too focused on the details and not pay attention to the bigger questions. Whereas discovering the anti-inflammatory

properties of a single nutrient or uncovering the subtleties of interactions between the gut and the brain may shed new light on how food may influence mood, it is important not to neglect the existing knowledge on other ways diet may affect mental health. For example, the later consequences of a poor diet include obesity and diabetes, which have already been shown to be associated with poorer mental health.^{11 31-33} A full discussion of the effect of these comorbidities is beyond the scope of our article (see fig 1), but it is important to acknowledge that developing public health initiatives that effectively tackle the established risk factors of physical and mental comorbidities is a priority for improving population health.

Further work is needed to improve our understanding of the complex pathways through which diet and nutrition can influence the brain. Such knowledge could lead to investigations of targeted, even personalised, interventions to improve mood, anxiety, or other symptoms through nutritional approaches. However, these possibilities are speculative at the moment, and more interventional research is needed to establish if, how, and when dietary interventions can be used to prevent mental illness or reduce symptoms in those living with such conditions. Of note, a recent large clinical trial found no significant benefits of a behavioural intervention promoting a Mediterranean diet for adults with subclinical depressive symptoms.³⁴ On the other hand, several recent smaller trials in individuals with current depression observed moderately large improvements from interventions based on the Mediterranean diet.³⁵⁻³⁷ Such results, however, must be considered within the context of the effect of people's expectations, particularly given that individuals' beliefs about the quality of their food or diet may also have a marked effect on their sense of overall health and wellbeing.³⁸ Nonetheless, even aside from psychological effects, consideration of dietary factors within mental healthcare may help improve physical health outcomes, given the higher rates of cardio-metabolic diseases observed in people with mental illness.³³

At the same time, it is important to be remember that the causes of mental illness are many and varied, and they will often present and persist independently of nutrition and diet. Thus, the increased understanding of potential connections between food and mental wellbeing should never be used to support automatic

assumptions, or stigmatisation, about an individual's dietary choices and their mental health. Indeed, such stigmatisation could be itself be a casual pathway to increasing the risk of poorer mental health. Nonetheless, a promising message for public health and clinical settings is emerging from the ongoing research. This message supports the idea that creating environments and developing measures that promote healthy, nutritious diets, while decreasing the consumption of highly processed and refined "junk" foods may provide benefits even beyond the well known effects on physical health, including improved psychological wellbeing.

Contributors and sources: JF has expertise in the interaction between physical and mental health, particularly the role of lifestyle and behavioural health factors in mental health promotion. JEG's area of expertise is the study of the relationship between sleep duration, nutrition, psychiatric disorders, and cardiometabolic diseases. AB leads research investigating the molecular mechanisms underlying the effect of stress and inflammation on human hippocampal neurogenesis, and how nutritional components and their metabolites can prevent changes induced by those conditions. REW has expertise in genetic epidemiology approaches to examining casual relations between health behaviours and mental illness. EAM has expertise in brain and gut interactions and microbiome interactions. All authors contributed to, read, and approved the paper, and all the information was sourced from articles published in peer reviewed research journals. JF is the guarantor.

Competing interests: We have read and understood BMJ policy on declaration of interests and declare the following: JF is supported by a University of Manchester Presidential Fellowship and a UK Research and Innovation Future Leaders Fellowship and has received support from a NICM-Blackmores Institute Fellowship. JEG served on the medical advisory board on insomnia in the cardiovascular patient population for the drug company Eisai. AB has received research funding from Johnson & Johnson for research on depression and inflammation, the UK Medical Research Council, the European Commission Horizon 2020, the National Institute for Health Research (NIHR) Biomedical Research Centre at South London and Maudsley NHS Foundation Trust, and King's College London. REW receives funding from the National Institute for Health Research Biomedical Research Centre at the University of Bristol. EAM has served on the external advisory boards of Danone, Viome, Amare, Axial Biotherapeutics, Pendulum, Ubiome, Bloom Science, Mahana Therapeutics, and APC Microbiome Ireland, and he receives royalties from Harper & Collins for his book *The Mind Gut Connection*. He is supported by grants from the National Institute of Diabetes and Digestive and Kidney Diseases, and the US Department of Defense. The views expressed are those of the authors and not necessarily those of the organisations above.

Provenance and peer review: Commissioned; externally peer reviewed.

This article is part of series commissioned by *The BMJ*. Open access fees are paid by Swiss Re, which had no input into the commissioning or peer review of the articles. *The BMJ* thanks the series advisers,

Nita Forouhi, Dariush Mozaffarian, and Anna Lartey for valuable advice and guiding selection of topics in the series.

Joseph Firth, research fellow^{1,2}

James E Gangwisch, assistant professor^{3,4}

Alessandra Borisini, researcher⁵

Robyn E Wootton, researcher^{6,7,8}

Emeran A Mayer, professor^{9,10}

¹Division of Psychology and Mental Health, Faculty of Biology, Medicine and Health, Oxford Road, University of Manchester, Manchester M13 9PL, UK

²NICM Health Research Institute, Western Sydney University, Westmead, Australia

³Department of Psychiatry, Columbia University Vagelos College of Physicians and Surgeons, New York, USA

⁴New York State Psychiatric Institute, New York, NY, USA

⁵Section of Stress, Psychiatry and Immunology Laboratory, Institute of Psychiatry, Psychology and Neuroscience, Department of Psychological Medicine, King's College London, London, UK

⁶School of Psychological Science, University of Bristol, Bristol, UK

⁷MRC Integrative Epidemiology Unit, Oakfield House, Bristol, UK

⁸NIHR Biomedical Research Centre, University Hospitals Bristol NHS Foundation Trust and University of Bristol, Bristol, UK

⁹G Oppenheimer Center for Neurobiology of Stress and Resilience, UCLA Vatche and Tamar Manoukian Division of Digestive Diseases, UCLA, Los Angeles, CA, USA

¹⁰UCLA Microbiome Center, David Geffen School of Medicine, UCLA, Los Angeles, CA, USA

Correspondence to: J Firth
joseph.firth@manchester.ac.uk



OPEN ACCESS

This is an Open Access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>.



Check for updates

- 1 Friedrich MJ. Depression is the leading cause of disability around the world. *JAMA* 2017;317:1517. doi:10.1001/jama.2017.3826
- 2 Johnson J, Weissman MM, Klerman GL. Service utilization and social morbidity associated with depressive symptoms in the community. *JAMA* 1992;267:1478-83. doi:10.1001/jama.1992.03480110054033
- 3 Lassale C, Batty GD, Baghdadli A, et al. Healthy dietary indices and risk of depressive outcomes: a systematic review and meta-analysis of observational studies. *Mol Psychiatry* 2019;24:965-86. doi:10.1038/s41380-018-0237-8
- 4 Firth J, Siddiqi N, Koyanagi A, et al. The Lancet Psychiatry Commission: a blueprint for protecting physical health in people with mental illness. *Lancet Psychiatry* 2019;6:675-712. doi:10.1016/S2215-0366(19)30132-4
- 5 Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA* 2002;287:2414-23. doi:10.1001/jama.287.18.2414

- 6 Gangwisch JE, Hale L, Garcia L, et al. High glycemic index diet as a risk factor for depression: analyses from the Women's Health Initiative. *Am J Clin Nutr* 2015;102:454-63. doi:10.3945/ajcn.114.103846
- 7 Salari-Moghaddam A, Saneei P, Larijani B, Esmailzadeh A. Glycemic index, glycemic load, and depression: a systematic review and meta-analysis. *Eur J Clin Nutr* 2019;73:356-65. doi:10.1038/s41430-018-0258-z
- 8 Bao J, de Jong V, Atkinson F, Petocz P, Brand-Miller JC. Food insulin index: physiologic basis for predicting insulin demand evoked by composite meals. *Am J Clin Nutr* 2009;90:986-92. doi:10.3945/ajcn.2009.27720
- 9 Seaquist ER, Anderson J, Childs B, et al, American Diabetes Association, Endocrine Society. Hypoglycemia and diabetes: a report of a workgroup of the American Diabetes Association and the Endocrine Society. *J Clin Endocrinol Metab* 2013;98:1845-59. doi:10.1210/jc.2012-4127
- 10 Towler DA, Havlin CE, Craft S, Cryer P. Mechanism of awareness of hypoglycemia. Perception of neurogenic (predominantly cholinergic) rather than neuroglycopenic symptoms. *Diabetes* 1993;42:1791-8. doi:10.2337/diab.42.12.1791
- 11 Salvi V, Hajek T. Brain-metabolic crossroads in severe mental disorders. *Front Psychiatry* 2019;10:492. doi:10.3389/fpsy.2019.00492
- 12 McIntyre RS, Kenna HA, Nguyen HT, et al. Brain volume abnormalities and neurocognitive deficits in diabetes mellitus: points of pathophysiological commonality with mood disorders? *Adv Ther* 2010;27:63-80. doi:10.1007/s12325-010-0011-z
- 13 O'Keefe JH, Gheewala NM, O'Keefe JO. Dietary strategies for improving post-prandial glucose, lipids, inflammation, and cardiovascular health. *J Am Coll Cardiol* 2008;51:249-55. doi:10.1016/j.jacc.2007.10.016
- 14 Kastorini C-M, Milionis HJ, Esposito K, Giugliano D, Goudevenos JA, Panagiotakos DB. The effect of Mediterranean diet on metabolic syndrome and its components: a meta-analysis of 50 studies and 534 906 individuals. *J Am Coll Cardiol* 2011;57:1299-313. doi:10.1016/j.jacc.2010.09.073
- 15 Noble EE, Hsu TM, Kanoski SE. Gut to brain dysbiosis: mechanisms linking western diet consumption, the microbiome, and cognitive impairment. *Front Behav Neurosci* 2017;11:9. doi:10.3389/fnbeh.2017.00009
- 16 Yuan N, Chen Y, Xia Y, Dai J, Liu C. Inflammation-related biomarkers in major psychiatric disorders: a cross-disorder assessment of reproducibility and specificity in 43 meta-analyses. *Transl Psychiatry* 2019;9:233. doi:10.1038/s41398-019-0570-y
- 17 Firth J, Stubbs B, Teasdale SB, et al. Diet as a hot topic in psychiatry: a population-scale study of nutritional intake and inflammatory potential in severe mental illness. *World Psychiatry* 2018;17:365-7. doi:10.1002/wps.20571
- 18 Köhler-Forsberg O, N Lydholm C, Hjorthøj C, Nordentoft M, Mors O, Benros ME. Efficacy of anti-inflammatory treatment on major depressive disorder or depressive symptoms: meta-analysis of clinical trials. *Acta Psychiatr Scand* 2019;139:404-19. doi:10.1111/acps.13016
- 19 Yahfoufi N, Alsadi N, Jambi M, Matar C. The immunomodulatory and anti-inflammatory role of polyphenols. *Nutrients* 2018;10:E1618. doi:10.3390/nu10111618
- 20 Su K-P, Lai H-C, Yang H-T, et al. Omega-3 fatty acids in the prevention of interferon-alpha-induced depression: results from a randomized, controlled trial. *Biol Psychiatry* 2014;76:559-66. doi:10.1016/j.biopsych.2014.01.008
- 21 Borsini A, Alboni S, Horowitz MA, et al. Rescue of IL-1 β -induced reduction of human neurogenesis by omega-3 fatty acids and antidepressants. *Brain Behav Immun* 2017;65:230-8. doi:10.1016/j.bbi.2017.05.006
- 22 Kiecolt-Glaser JK, Fagundes CP, Andridge R, et al. Depression, daily stressors and inflammatory responses to high-fat meals: when stress overrides healthier food choices. *Mol Psychiatry* 2017;22:476-82. doi:10.1038/mp.2016.149
- 23 Osadchiv V, Martin CR, Mayer EA. Gut microbiome and modulation of CNS function. *Compr Physiol* 2019;10:57-72. doi:10.1002/cphy.c180031
- 24 Cryan JF, Dinan TG. Mind-altering microorganisms: the impact of the gut microbiota on brain and behaviour. *Nat Rev Neurosci* 2012;13:701-12. doi:10.1038/nrn3346
- 25 Kelly JR, Borre Y, O'Brien C, et al. Transferring the blues: depression-associated gut microbiota induces neurobehavioural changes in the rat. *J Psychiatr Res* 2016;82:109-18. doi:10.1016/j.jpsychires.2016.07.019
- 26 Kelly JR, Keane VO, Cryan JF, Clarke G, Dinan TG. Mood and microbes: gut to brain communication in depression. *Gastroenterol Clin North Am* 2019;48:389-405. doi:10.1016/j.gtc.2019.04.006
- 27 Sonnenburg ED, Sonnenburg JL. The ancestral and industrialized gut microbiota and implications for human health. *Nat Rev Microbiol* 2019;17:383-90. doi:10.1038/s41579-019-0191-8
- 28 Ghosh TS, Rampelli S, Jeffery IB, et al. Mediterranean diet intervention alters the gut microbiome in older people reducing frailty and improving health status: the NU-AGE 1-year dietary intervention across five European countries. *Gut* 2020;69:1218-28. doi:10.1136/gutjnl-2019-319654
- 29 Tillisch K, Labus J, Kilpatrick L, et al. Consumption of fermented milk product with probiotic modulates brain activity. *Gastroenterology* 2013;144:1394-401, 1401.e1-4. doi:10.1053/j.gastro.2013.02.043
- 30 Liu RT, Walsh RFL, Sheehan AE. Prebiotics and probiotics for depression and anxiety: a systematic review and meta-analysis of controlled clinical trials. *Neurosci Biobehav Rev* 2019;102:13-23. doi:10.1016/j.neubiorev.2019.03.023
- 31 Wootton RE, Lawn RB, Millard LAC, et al. Evaluation of the causal effects between subjective wellbeing and cardiometabolic health: Mendelian randomisation study. *BMJ* 2018;362:k3788. doi:10.1136/bmj.k3788
- 32 Jebeile H, Gow ML, Baur LA, Garnett SP, Paxton SJ, Lister NB. Association of pediatric obesity treatment, including a dietary component, with change in depression and anxiety: a systematic review and meta-analysis. *JAMA Pediatr* 2019;173:e192841. doi:10.1001/jamapediatrics.2019.2841
- 33 Ma J, Rosas LG, Lv N, et al. Effect of integrated behavioral weight loss treatment and problem-solving therapy on body mass index and depressive symptoms among patients with obesity and depression: the RAINBOW randomized clinical trial. *JAMA* 2019;321:869-79. doi:10.1001/jama.2019.0557
- 34 Bot M, Brouwer IA, Roca M, et al, MoodFOOD Prevention Trial Investigators. Effect of multivitamin supplementation and food-related behavioral activation therapy on prevention of major depressive disorder among overweight or obese adults with subsyndromal depressive symptoms: the MoodFOOD randomized clinical trial. *JAMA* 2019;321:858-68. doi:10.1001/jama.2019.0556
- 35 Francis HM, Stevenson RJ, Chambers JR, Gupta D, Newey B, Lim CK. A brief diet intervention can reduce symptoms of depression in young adults – a randomised controlled trial. *PLoS One* 2019;14:e0222768. doi:10.1371/journal.pone.0222768
- 36 Jacka FN, O'Neil A, Opie R, et al. A randomised controlled trial of dietary improvement for adults with major depression (the 'SMILES' trial). *BMC Med* 2017;15:23. doi:10.1186/s12916-017-0791-y
- 37 Parletta N, Zarnowiecki D, Cho J, et al. A Mediterranean-style dietary intervention supplemented with fish oil improves diet quality and mental health in people with depression: A randomized controlled trial (HELFI-MED). *Nutr Neurosci* 2019;22:474-87. doi:10.1080/1028415X.2017.1411320
- 38 Rozin P, Fischler C, Imada S, Sarubin A, Wrzesniewski A. Attitudes to food and the role of food in life in the USA, Japan, Flemish Belgium and France: possible implications for the diet-health debate. *Appetite* 1999;33:163-80. doi:10.1006/appe.1999.0244
- 39 Hunt A, Harrington D, Robinson S. Vitamin B12 deficiency. *BMJ* 2014;349:g5226. doi:10.1136/bmj.g5226

Cite this as: *BMJ* 2020;369:m2440
<http://dx.doi.org/10.1136/bmj.m2440>