Noncommunicable diseases (NCDs) globally account for the largest burden of early mortality and are predicted to cost more than US$30 trillion over the next 20 years (Bloom et al., 2011). However, when the global burden of disease is viewed in terms of disability rather than mortality, mental and substance use disorders account for the leading cause of health-related disability worldwide, with unipolar depression alone accounting for the second highest number of years lost to disability (Murray et al., 2013). Although methodological challenges complicate attempts to assess possible increases in the prevalence of mental disorders, there are data to support such increases from the United States (Twenge et al., 2010), Britain (Collishaw et al., 2004), Taiwan (Fu et al., 2013), and Australia (O’Donnell et al., 2013), although upward trends in young people may be plateauing (Maughan et al., 2008).

Although three of the “Big Four” NCDs—cardiovascular disease (CVD), type 2 diabetes mellitus, and cancer—are well known to be directly influenced by unhealthy diet (WHO, 2011; Swinburn et al., 2011), there is now highly consistent evidence across age groups, cultures, and countries to suggest that unhealthy diet is also a key risk factor for common mental disorders, particularly depression. The following presents a discussion of the change to global eating patterns and the recent literature highlighting the relationships between diet and mental health.

**CHANGES TO THE FOOD SUPPLY AND GLOBAL IMPACT ON HEALTH**

Substantial changes in efficiencies of production, marketing, transport, and sale of food have had a highly detrimental impact on dietary patterns across the globe, with a widespread shift toward increased intake of fast foods and sugar-sweetened beverages (Adair and Popkin, 2005). In the West, dietary patterns are commonly high in saturated fats and refined sugar, with nutrient-poor and energy-dense foods contributing approximately 30% of the daily intakes of US adults (Kant, 2000). A comprehensive review of data from the recurrent National Health and Nutrition Examination
Surveys in the United States concluded that only one in 10 Americans has a “good” diet (Briefel and Johnson, 2004). Although slight improvements have been detected in the dietary intakes of sugar and fats in recent years, intakes of nutrient- and fiber-rich vegetables and whole grains remain far lower than recommended (Bowman et al., 2014). Moreover, compared with wealthier areas, the urban built environment of those living in low socioeconomic status neighborhoods promotes unhealthy food consumption, with more fast food outlets and convenience stores and associated advertising of unhealthy food products than wealthier areas (Larson et al., 2009), leading to pronounced health inequalities.

Equally, changes in economic conditions in developing economies, including increased income, trade liberalization, and globalization, as well as increased penetration of processed food industries, have led to profound shifts in habitual dietary patterns away from traditional diets higher in plant foods rich in fiber and nutrients toward patterns characterized by manufactured food products higher in saturated fat, sugar, salt, and refined carbohydrates and foods of animal origin. As a result, there is now a far greater burden of poor health and mortality from NCDs than from undernutrition and infectious diseases in Latin America, North Africa, the Middle East, South East and East Asia, and the Pacific Rim (Popkin, 2002). Indeed, more than three-quarters of all nutrition-related chronic diseases occur in developing countries (Popkin and Du, 2003). As one example, consumption of whole-grain cereals and vegetables decreased in China between 1989 and 1997 whereas the consumption of meat, meat products, and plant oils increased (Popkin and Du, 2003). This represents a shift away from carbohydrates to fats as the dominant energy source and was reflected in a concurrent increase in the prevalence of overweight individuals and obesity in China (Popkin and Du, 2003). Likewise, Indian data from 1987 to 1988 and from 1999 to 2000 showed a decrease in the consumption of rice and wheat, while whole milk and egg consumption increased, as did consumption of biscuits, salted snacks, prepared sweets, edible oils, and sugar (Food and Agriculture Organization, 2004). Alongside these changes were declines in the intake of fruit and vegetables. In particular, consumption of processed foods and ready-to-eat foods were observed to increase with income. Concordant with these changes, in 1999 one-third of men and half of women in the middle classes in India were obese.

When considering changes in dietary patterns over recent decades, it is also worth making a comparison with the composition of our ancestors’ diets. It is estimated that dietary intakes of micronutrients for early humans may have been up to 10 times that of modern humans because of the composition of wild plant foods known to be consumed by hunter-gatherers (Brand-Miller and Holt, 1998); carbohydrate consumption was almost exclusively derived from fruits and vegetables (Eaton and Eaton, 2000). In comparison, contemporary Americans obtain 72% of their total dietary energy from dairy products, cereals, refined sugars, refined vegetable oils, and alcohol (Cordain et al., 2005). Furthermore, the high potassium-to-sodium ratios and high fiber intakes known to characterize Paleolithic diets are the inverse of contemporary Western diets (Cordain et al., 2005).
Although there was wide variation in dietary regimens for Stone Agers, preagricultural humans consumed more animal protein than current Westerners (Eaton and Eaton, 2000). A meta-analysis of dietary studies of hunter-gatherers reported that animal foods provided approximately 65% of energy intakes for early humans (Cordain et al., 2002). However, this did not appear to adversely affect CVD risk factors because of the different lipid profiles of the foods consumed; in contrast to intensively farmed cattle, wild game contain high levels of monounsaturated fatty acids as well as long-chain polyunsaturated fatty acids (PUFAs). In concert with higher intakes of plant foods and lower intakes of sodium, as well as much higher levels of physical activity, this pattern of consumption served to inhibit the development of CVD (Cordain et al., 2002). In support of this, intervention studies performed in indigenous Australian populations have reported a pronounced reduction in risk factors for CVD, as well as metabolic abnormalities associated with diabetes, after reversion to a traditional hunter-gatherer diet containing substantial quantities of red meat from wild animals (O’Dea, 1984; O’Dea and Sinclair, 1985). Reflecting these findings, nutritional studies of contemporary hunter-gathers have reported high plasma concentrations of folate and vitamin B\(_{12}\) (Metz et al., 1971).

Another important aspect of the shift in habitual diets globally is that of an increase in refined carbohydrate consumption. The consumption of caloric sweeteners, which include high fructose corn syrup and a wide range of other monosaccharides (glucose and fructose) and disaccharides (sucrose and saccharose), has increased significantly across the globe (Popkin and Nielsen, 2003), largely because of the proliferation of processed foods that have these sweeteners added to them. An analysis of exposure data from 52 countries, performed at Harvard University, demonstrated that raised blood glucose accounts for 21% of ischemic heart disease and 13% of stroke mortality worldwide (Danaei et al., 2006). Of note, higher-than-optimal blood glucose concentrations were associated with a higher risk of mortality than full-blown diabetes (Danaei et al., 2006). This underscores the deleterious impact of these dietary changes on a global scale.

**NUTRIENTS AND MENTAL HEALTH**

Although there has long been interest in the idea that diet is related to mental health, before 2009, scientific data were scarce and the existing literature focused primarily on individual nutrients or foods—particularly fish and the long-chain omega-3 polyunsaturated fatty acids (n-3 PUFAs). Estimates from studies examining Paleolithic nutrition and extant hunter-gatherer populations suggest that humans evolved to consume a diet consisting of an omega-6 to omega-3 ratio of 1–2:1 (Eaton and Konner, 1985). This compares to Western ratios of between 10 and 20:1 (Simopoulos, 2001). Several lines of evidence indicate that these changes in fatty acid consumption may have had a detrimental impact on mental health.

In one of the first studies in this field, Hibbeln (1998) suggested that substantial cross-national variation in prevalence rates of depression may be, in part, a function
of a demonstrated, strong, inverse correlation between national levels of fish consumption and national depression prevalence rates across nine countries. In a similar study (Hibbeln, 2002), postpartum depression was inversely associated with docosahexaenoic acid (DHA) concentrations in breast milk in a multinational comparison of 16 countries, whereas a strong inverse relationship also existed between postpartum depression and fish consumption across 22 countries. Likewise, in another cross-national comparison (Noaghiul and Hibbeln, 2003), higher national seafood consumption was associated with lower prevalence rates of bipolar spectrum disorders. To test the hypothesis according to its proposed biological mechanisms, schizophrenia data were used as a control condition. Seafood consumption was not associated with national prevalence rates of schizophrenia in any analysis, supporting a specific relationship between n-3 PUFAs and mood disorders. Although these studies present findings that are supportive of the hypothesis of a relationship between n-3 PUFA consumption and depressive illness, the results must be viewed with caution. Clearly, a correlation between factors at the national level may be explained by a multitude of cultural, social, and economic factors that could not be controlled for in appropriate multivariate analyses. On the other hand, cross-national studies may provide a valuable perspective because the habitual consumption of a particular food group may be more stable over time at a population level than in that of individuals.

In several population studies, which allow for an assessment of possible confounding, low levels of fish and/or n-3 PUFA consumption are associated with increased depression (Appleton et al., 2007; Bountziouka et al., 2009; Colangelo et al., 2009; Silvers and Scott, 2002; Tanskanen et al., 2001; Timonen, et al., 2004), although some of these studies suggest nonlinear relationships (Jacka et al., 2013a; Sanchez-Villegas et al., 2007). However, trials of n-3 PUFA supplementation in depression have yielded equivocal results. One meta-analysis examining the effect of n-3 PUFA supplementation for depressed mood concluded that there was a small beneficial effect of treatment with n-3 PUFA compared with placebo, but that this benefit was restricted to those with major depressive disorder (MDD; Appleton et al., 2010). This may reflect an increased need for the long-chain omega-three fatty acids for those suffering major depression, in which the increased inflammation and accompanying oxidative stress commonly observed in major depression (Maes et al., 2011) results in increased lipid peroxidation and a reduction in lipid levels in neuronal membranes. On the other hand, another meta-analysis suggests that supplementation with n-3 PUFA formulations that are relatively higher in eicosapentaenoic acid (>60%) compared with DHA may be efficacious in treating depression (Lin et al., 2012). Another meta-analysis also concluded that n-3 PUFA as an adjunctive supplement improves bipolar depression but not mania (Sarris et al., 2012).

There are many other nutrients found in healthy foods that have also been related to mental health. Folate is found in abundance in vegetables, fruits, and salads, yet the intake of these foods is commonly less than recommended dietary intakes within populations. Clinical studies have long observed folate deficiency and low folate status in those with clinical depression, and low folate is also associated with depression.
in population studies (Morris et al., 2003), even in the presence of folate fortification (Ramos et al., 2004). Indeed, population studies are plentiful. In a study of middle-aged Finnish men, the odds of self-reported depression for those in the lowest tertile of folate intake were increased by nearly 50% after all adjustments (Tolmunen et al., 2003), whereas another prospective study in Finnish men reported that intakes of folate below the median were associated with a threefold increased risk of MDD over more than 10 years of follow-up (Tolmunen et al., 2004). Likewise, a study of 732 elderly Korean men reported that lower serum levels of folate and vitamin B₁₂ and higher levels of homocysteine were all associated with an increased risk of clinically significant depression over the follow-up period (Kim et al., 2008), whereas a Japanese study reported that serum folate levels predicted depression risk over 3 years of follow-up in office workers (Nanri et al., 2012). Two cross-sectional, population-based studies also examined this relationship and found a significant, linear association of folate intake with depressive symptoms in Japanese men, but not women (Murakami et al., 2008), and an inverse relationship between folate intake and MDD in Australian women (Jacka et al., 2012b). A meta-analysis of case-control, population, and cohort studies confirmed the association between low folate status and depression (Gilbody et al., 2007), whereas a Cochrane review concluded that folate may be useful as an adjunctive treatment for depression, although it is still unclear as to whether supplementation will benefit those with low and normal levels of folate (Taylor et al., 2004).

Magnesium is another micronutrient that is found in abundance in healthy foods, such as fruits, vegetables, fish, legumes, and whole grains, and it is inversely associated with depression. A recent meta-analysis including data from nearly 20,000 participants reported a 1.34-fold increased risk of hypomagnesemia in those with depression (Cheungpasitporn et al., 2015); however, data from population studies focusing on dietary intakes are somewhat equivocal. Jacka et al. (2009) observed an inverse relationship between dietary magnesium intake and depression in a large sample of community-dwelling men and women in Norway that was independent of potentially confounding variables and reported similar inverse and independent relationships between magnesium intake and clinical depressive disorders in Australian women (Jacka et al., 2012b). In contrast, Derom et al. reported no relationship between magnesium intake and depression risk in the SUN Cohort study (Derom et al., 2012). Likewise, an inverse relationship between magnesium intake and depression in a large US study was only observed in younger people (Tarleton and Littenberg, 2015), with a positive relationship seen in older people. Barragan-Rodriguez et al. reported that magnesium supplementation was as effective as pharmacotherapy in treating depression in elderly diabetic patients with hypomagnesemia (Barragan-Rodriguez et al., 2008). However, magnesium was not found to alleviate symptoms of depression or anxiety in premenstrual women (Walker et al., 1998). Certainly, experimental studies support a role for magnesium in depression; in animals, a magnesium-deficient diet increases depression and anxiety-related behavior (Singewald et al., 2004), whereas magnesium treatment appears to improve such behaviors (Poleszak et al., 2004, 2005).
Zinc is another nutrient of particular interest in depressive illness. There have now been cross-sectional studies showing inverse relationships between the dietary intake of zinc and self-reported depression in pregnant women (Roy et al., 2011), female students (Amani et al., 2011), and a large representative sample of Australian women (Jacka et al., 2012b). In support of this finding, a recent study has also reported that women habitually consuming less than the recommended intake of red meat, a food rich in zinc, were more likely to be diagnosed with clinical depressive and/or anxiety disorders (Jacka et al., 2012a), although higher than recommended levels of intake were also associated with an increased likelihood of these illnesses. Low dietary zinc intake was also associated with increased risk of depression in older Australian men and women over time (Vashum et al., 2014). In a clinical context, low concentrations of zinc are commonly observed in patients with major depression (Swardfager et al., 2013) whereas zinc supplementation has been shown to enhance the efficacy of antidepressant therapy (Nowak et al., 2003a), with a systematic review supporting its use as an adjunctive therapy (Lai et al., 2011). There are also supportive data from animal studies suggesting that zinc may exert antidepressant effects (Nowak et al., 2003b). Thus further studies of zinc as a mono- and adjunctive therapy in depression may be warranted.

**Diet Quality**

It is important to recognize that magnesium, folate, zinc, and long-chain fatty acids are all components of a healthy diet, found primarily in foods such as leafy green vegetables, legumes, whole grains, lean red meat, and fish. As such it is likely that the apparent relationship between the consumption of these individual components of food and mental health is explained by the overall quality of an individual’s habitual diet. As a response to this understanding, the new field of nutritional psychiatry has now, in concert with the wider field of nutrition research, moved away from examining individual nutrients toward an examination of the importance of whole diet in mental health. In this context there have now been many observational studies published in the last several years demonstrating cross-sectional and prospective relationships between diet quality and the common mental disorders in adults, adolescents, and children. In the first study to examine the relationship between dietary patterns and clinical mood and anxiety disorders, a dietary pattern comprising vegetables, fruit, beef, lamb, fish, and whole-grain foods was associated with a reduced likelihood of clinically diagnosed depressive and anxiety disorders, whereas a Western dietary pattern comprising processed and unhealthy foods was associated with an increased likelihood of psychological symptoms as well as MDD and dysthymia in Australian women. Increased a priori diet quality scores were also associated with reduced psychological symptoms (Jacka et al., 2010b). In this same cohort of women, increased scores on the same healthy dietary pattern were also associated with a halving in the odds for bipolar disorder whereas those with higher scores on the Western dietary pattern and glycemic load measures were more likely to have bipolar disorder (Jacka et al., 2011a). Associations between
diet quality and mental health outcomes have also been reported in a study of more than 7000 adults in western Norway (Jacka et al., 2011c). A healthier diet, measured with an a priori diet quality score, was associated with a reduction in the odds ratios for depression and anxiety in women and with reduced odds for depression, but not anxiety, in men. Nanri et al. (2010) have also reported that middle-aged Japanese municipal employees who were in the highest tertile of healthy Japanese dietary pattern scores, characterized by higher intakes of vegetables, fruit, soy products, and mushrooms, were significantly less likely to be depressed than those in the lowest tertile, although there was no discernible relationship between unhealthy food intake and depression. In the United States, a healthier diet was associated with reduced depressive symptoms cross-sectionally, including after adjustment for race, gender, age, education, and income (Kuczmarski et al., 2010). Conversely, an increased consumption of high-calorie sweet foods was associated with increased depressive symptoms in more than 4500 middle-aged US women (Jeffery et al., 2009). In the same study, an increased intake of low-calorie foods (e.g., green salads, roast chicken, baked fish, low-fat milk, and cold cereals) was associated with reduced depressive symptoms, independent of age, race, body mass index (BMI), and education.

There are many prospective studies that have also shed light on this topic. In Spain, Sanchez-Villegas et al. (2009) demonstrated an inverse association between the level of adherence to a Mediterranean dietary pattern (MDP) and the risk for incident depression over time in more than 10,000 middle-aged university graduates. This association existed before and after controlling for a comprehensive range of potentially confounding factors, including sociodemographic, anthropometric, and lifestyle factors; other health behaviors; and medical history. Of course, depression itself may cause poor dietary choices, which may in turn worsen an existing condition. Thus many of the prospective studies have conducted sensitivity analyses to investigate the “reverse causality” hypothesis. In the SUN Cohort study the authors attempted to refute reverse causality as an explanatory factor by repeating analyses after excluding participants who reported depression in the first 2 years of follow-up, as well as examining depression at or before the baseline assessment as an exposure, with adherence to the MDP as the outcome variable (Sanchez-Villegas et al., 2009). In fact, the relationship of diet to MDP adherence was strengthened rather than diminished after removing participants with incipient depression whereas there was no observable relationship between earlier depression and adherence to the MDP. A study undertaken in the ongoing Whitehall II cohort study also found an increased risk for incident depression over 5 years in people consuming a Western-style diet pattern as well as a reduced risk for those eating a “whole-foods” diet pattern (Akbaraly et al., 2009). These authors also excluded those identified with depression at baseline and reanalyzed the data, examining depression at an early time point as a predictor of diet quality at the next follow-up. Once again, results of these analyses did not support depression as a predictor of poor dietary behavior. Indeed, a recent Australian study explicitly addressed the reverse causality hypothesis and found—contrary to expectations—that individuals who had previously experienced
depression had better diets on average than those who had no such history of depression (Jacka et al., 2015). Such data suggest that the relationship between depressive symptoms and dietary changes may not be straightforward.

Another important consideration in these observational studies is the possibility that other factors explain the association between diet and mental health. Given that socioeconomic position is consistently related to both poorer quality diet (Galobardes et al., 2001; Henderson et al., 2002) and risk for depression (Butterworth et al., 2012), there is no doubt that factors such as educational level, social disadvantage, and occupation are plausible explanations for the relationships observed between diet and depression. Although most previous studies in this field have included appropriate measures in their analyses and largely excluded them as explanatory variables, it is inevitable that the tools used to measure such factors will be imperfect and unable to fully capture the construct of socioeconomic circumstances. One recent study did attempt to address this issue and constructed a comprehensive variable that encompassed labor force status, occupation, educational attainment, income, welfare dependency, financial hardship, and childhood disadvantage. Although this construct did explain the cross-sectional relationship between unhealthy diet and depression observed in older Australian adults in this study, it did not fully explain the prospective associations seen between dietary patterns and mental health over time in this group (Jacka et al., 2014).

Thus the hypothesis that diet is related to common mental disorders, particularly depression, is supported by studies in a wide range of countries and cultures as diverse as Spain, Norway, China, the United States, Japan, Australia, and many others. Reflecting this, a recent systematic review and meta-analysis, including results from 13 observational studies, concluded that a healthy diet is significantly associated with a reduced odds for depression (odds ratio: 0.84; 95% confidence interval [CI]: 0.76, 0.92; \( P < 0.001 \)) (Lai et al., 2013). Likewise, a meta-analysis of 22 studies investigating the protective effects of adherence to a Mediterranean-style diet on brain diseases demonstrated that higher adherence was associated with a reduced risk for depression (relative risk = 0.68, 95% CI: 0.54–0.86) as well as cognitive decline (Psaltopoulou et al., 2013).

At the other end of the age spectrum, diet quality is also associated with mental health in adolescents and children. In an Australian study, a lower consumption of a healthy diet and increased consumption of unhealthy and processed foods were independently associated with increased odds for self-reported symptomatic depression in more than 7000 young adolescents (Jacka et al., 2010a). For those adolescents in the highest category of “healthy” diet scores, the likelihood of depression was nearly halved compared with those in the lowest category, whereas for those in the highest quintile of “unhealthy” diet score, the likelihood of depression was increased by nearly 80% compared with the lowest quintile. These relationships demonstrated a dietary intake-response pattern observed before and after adjustment for a wide range of potential confounding factors, including sociodemographic factors, health and dieting behaviors, and familial environment (Jacka et al., 2010a). Jacka et al. (2011b) also examined approximately 3000 Australian adolescents and found that
diet quality was cross-sectionally and prospectively associated with adolescent mental health. In this study, improvements in diet quality were mirrored by improvements in mental health whereas reductions in diet quality were associated with declining psychological functioning. Another Australian study reported that adolescents with a dietary pattern higher in take-away foods, red meat, and sweets exhibited higher levels of internalizing and externalizing behaviors, which are markers of mental health status (Oddy et al., 2009). However, there was no relationship observed between a healthy dietary pattern and such behaviors. Another study, in Chinese adolescents, showed inverse relationships between higher scores on a traditional dietary pattern, comprising whole grains, vegetables, fruit, rice, and soya products, and depression and anxiety as well as positive relationships between both an unhealthy snacking dietary pattern and a high meat dietary pattern and depression and anxiety (Weng et al., 2012). In Norway a high consumption of unhealthy foods was associated with increased odds for behavioral problems in adolescents whereas both fruit and fish consumption were associated with fewer behavioral problems (Overby and Hoigaard, 2012), although there was no relationship between vegetable consumption and behavior. Likewise, a German study reported that an increased intake of confectionery was associated with increased emotional symptoms in children compared with low intake whereas a higher diet quality score was associated with lower odds for emotional symptoms after adjustment for variables such as sociodemographic characteristics, BMI, physical activity, television viewing, and computer use (Kohlboeck et al., 2012).

Another important consideration is that of the possible impact of nutrition in very early life and its relationship to the risk for mental health problems in children. Early-life exposures appear to potently influence child behavioral, emotional, and learning outcomes (Lewis et al., 2014). Therefore a better understanding of this critical developmental period is of immense importance in identifying early-life risk factors that are modifiable, such as diet, to support prevention efforts. In this context there are important data from a very large Norwegian cohort study indicating that unhealthy maternal diet during pregnancy, as well as both healthy and unhealthy dietary patterns during the first years of life, are associated with the risk for mental health problems in young children (Jacka et al., 2013b). In this study there was evidence suggesting consistently great effects of unhealthy diets on children’s behavioral and emotional outcomes compared with the possible effects of insufficient healthy food intake. This study has now received support from two further gold standard cohort studies. In the Generation R study in the Netherlands, a lower level of adherence to a healthy MDP and higher adherence to an unhealthy Dutch dietary pattern in pregnancy were independently related to emotional-behavioral dysregulation in children in their early years (Steenweg-de Graaff et al., 2014). Likewise, studies conducted using data from the Avon Longitudinal Study of Parents and Children in the United Kingdom have reported relationships between maternal dietary patterns and both cognition and emotional-behavioral dysregulation in children (Barker et al., 2013; Pina-Camacho et al., 2014).
Reflecting the new data in this field, a systematic review concluded that an increased consumption of unhealthy, sugary, and fat-rich foods is related to increased risk of psychological symptomatology in children and adolescents (O’Neil et al., 2014). Given that most mental health problems develop by age 25, and that diet is a modifiable environmental exposure for the entire population, these new data in children and adolescents have important public health implications. Taken together with the studies in adults, this nascent but compelling evidence base indicates that the global changes in dietary habits are likely to be influencing the prevalence of common mental disorders.

INTERVENTION STUDIES
This new body of observational data is notable for the relative consistency of the reported relationships and the observed effect sizes. Inverse relationships between diet quality and mental health have now been reported across a multitude of countries, from children through to the elderly, in men and women, and utilizing a wide range of mental health and dietary measures. However, although there is one trial currently underway (O’Neil et al., 2013), so far there have been no published studies that have specifically sought to answer the question “If I improve my diet, will I feel less depressed?” This is an increasingly common question in clinical practice and the general community, and it remains unanswered to date, representing a serious gap in our knowledge base. A systematic review examined the data from dietary interventions that have examined mental health outcomes in various populations and concluded that, although data from depressed samples are currently lacking, there is some evidence suggesting a positive impact of dietary improvement on depression (Opie et al., 2014).

It is also worth noting the new data from two intervention studies that support dietary improvement as a means to prevent the incidence of depression. In the large PREDIMED study, older individuals randomized to an MDP compared with a low-fat diet tended to be less likely to develop depression over the period of the intervention, and this relationship was particularly pronounced for those individuals with type 2 diabetes (Sanchez-Villegas et al., 2013). Those in the Mediterranean diet groups also demonstrated improved cognition compared with controls (Martinez-Lapiscina et al., 2013). Another US study reported that dietary counseling was as effective as psychotherapy in reducing the rate of transition from subsyndromal to clinical depression in older adults (Stahl et al., 2014). Although these two studies must be regarded as preliminary evidence given the lack of statistical power in the PREDIMED study and the lack of a control group in the US intervention, they do give rise to some optimism regarding the potential of dietary interventions to prevent depression.

CLINICAL APPLICATIONS
This new literature provides face validity for the role of nutritional factors in the genesis and management of depression. Although the nascent evidence base consists primarily of reports from observational studies, the data largely fulfill the Bradford
Hill criteria for causality (Jacka et al., 2012c) and are consistent and compelling. Moreover, although there is currently a dearth of evidence regarding the efficacy of dietary modulation to treat depression, it is clear that diet has a major impact on comorbid physical disorders that are disproportionally more common in people with depression, such as cardiovascular disorders and diabetes. This suggests that policy, public health, and clinical actions taken to improve diet should have benefits for mental health (Jacka et al., 2012c), with the precautionary principle supporting such actions.

It is critical that we now gain a detailed understanding of the pathophysiological pathways that mediate this relationship to develop targeted interventions. These are thus far unknown. Chronic low-grade inflammation, with accompanying oxidative stress, is a common feature of virtually all mental disorders, as well as the somatic disorders with which mental disorders are so commonly comorbid. This indicates a central role for immune system dysfunction in mental illness (Berk et al., 2013). Related to this is the new knowledge regarding the human gut microbiome as a core driver of immune functioning and the development of the brain and the metabolic and innate immune system during early life. Indeed, emerging data from experimental and human studies now suggest that the gut is a key pathway by which environmental factors, such as poor diet, sedentary behavior, and stress, influence the immune system and host health, with downstream effects on the risk for mental, as well as physical, disorders. This highlights the urgent need to elucidate the role of diet in the bidirectional communication within this axis and the development of new preventive and therapeutic interventions for these disorders based on modification of diet, the gut microbiome, and immune function.

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