



Diet, exercise, and mental disorders – public health challenges of the future

| Review

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Abstract: A role of socio-medical causation, comprising social, environmental and lifestyle factors, in the etiology and prevention of diseases was proposed by the German public health pioneer Rudolf Virchow. In China, Sun Yat-sen regarded sanitation and health as indices of a country's prosperity and social civilization. Today, diet and physical activity are increasingly recognized as potentially modifiable lifestyle factors influencing the onset and outcomes of mental disorders. The life expectancy of many groups of people with mental illness is markedly reduced compared to the general population. This mortality gap is due partly to high rates of obesity and low rates of exercise. The present short review evaluates the evidence of the role of diet and exercise in the prevention and treatment of mental disorders. Associated public mental health issues are also discussed.

The effects of single nutrients in the treatment of mental disorders appear to be modest at best, while the investigation of dietary patterns seems more promising. Clinical studies using sound methodology are largely lacking, and rigorous trials assessing long-term outcomes of dietary approaches should be conducted. The associations between dietary patterns, adiposity, inflammation and mental health are a potentially valuable field of research. Epidemiological studies have shown that physical activity and exercise can prevent or delay the onset of various mental disorders and may have therapeutic benefits when used as sole or adjunct therapy in psychiatry. While findings of controlled studies are sparse, preliminary evidence suggests that physical activity can improve physical, subjective, and disorder-specific clinical outcomes.

Mental health is vital to public health. It is protective against unhealthy lifestyles, physical disease, and social inequalities. Emerging evidence demonstrates the effectiveness of lifestyle changes in the promotion of mental health and in the primary and secondary prevention of mental illness. The promotion of an active lifestyle and physical exercise is likely to be a highly cost-effective intervention in improving population mental health and wellbeing. Physical exercise appears to be a promising alternative or additional treatment option for individuals with mental disorders. The initiating and financing of high-quality studies examining the effects of physical exercise on mental disorders should be undertaken; such measures would have additional health benefits, such as positive cardiovascular and metabolic effects. Financial returns from large-scale investigations into exercise as a treatment for mental disorders cannot be expected. Support through public funding and non-profit organizations is needed in order to help counter the challenges posed by public mental health. Public health efforts may also include school-based interventions and the creation of environments conducive to physical activity and improved nutrition. Political interventions may be necessary.

In conclusion, public health should prioritize dietary factors and exercise in the combatting of mental disorders. The effectiveness of lifestyle changes involving diet and exercise in improving mental health should be evaluated in the treatment of individuals and in population-based public health programs.

Key words: Lifestyle; diet; exercise; mental disorders; attention-deficit/hyperactivity disorder; dementia; depression; schizophrenia; mortality gap; prevention; public mental health.

1. Introduction

Mental disorders have become increasingly important public health issues. Mental health is vital to public health. It is protective against unhealthy lifestyles, physical disease, and social inequalities. Emerging evidence demonstrates the effectiveness of lifestyle changes in the promotion of mental health. Lifestyle factors adversely affecting the physical and mental health of people with psychiatric illness include higher rates of obesity [1] and relatively low rates of exercise [2]. In recent decades, major shifts in lifestyle have occurred globally in regard to dietary intake and physical activity levels. While the consumption of nutrient-dense foods has progressively diminished, the intake of high-energy foods, processed foods, and sugar has increased significantly [3]. Physical activity has changed markedly as a result of industrialization and urban living, and the physical activity levels of more than 30% of the global population are now described as insufficient [4].

Nutrition has been linked to behavior, cognition, and mood, and considerable evidence points to the importance of nutrition for mental health. Nutritional deficiencies are today recognized as a risk factor for mental disorders, appearing to play a significant role in their etiology and management [5,6]. A review of over 160 studies highlights the importance of dietary factors, suggesting that the mental health of countries may be linked to them [7]. The role of exercise in physical health and wellbeing has been long established. The identification of the possible beneficial effects of physical exercise on mental health and psychological wellbeing has become a focus of psychiatric research [8].

While lifestyle-based approaches in several chronic medical diseases, such as cardiovascular disease, have been successful, preventive measures have not been adopted in psychiatry. Lifestyle and chronic medical conditions are largely ignored as contributing factors to the development, course, and outcomes of mental disorders. Growing evidence suggests that nutrition, diet, physical activity, and sports are some of the most important modifiable lifestyle factors in the prevention and therapy of mental disorders. Given the enormous medical, social, and economic costs of contemporary lifestyles, therapeutic lifestyle changes may be of paramount importance.

The present review evaluates the evidence regarding the role of diet and exercise in the prevention and

treatment of mental disorders, such as depression, dementia, schizophrenia, and attention-deficit/hyperactivity disorder (ADHD). Associated public health issues are also presented.

2. Diet and depression

Depression is a common and important cause of morbidity and mortality worldwide; it has been predicted to become the second-leading health burden on society among all diseases worldwide by 2020 [9]. In middle- and high-income countries today, depression is the leading cause of disease burden [10].

Although antidepressants are commonly prescribed to people with depression, many questions regarding the use of these drugs remain unanswered, including their efficacy in milder forms of depression, their effects beyond eight weeks of treatment, the harms associated with individual antidepressant drugs as well as the long term adverse effects of antidepressants [11].

The quality of diet as a factor in depression has increasingly been recognized in recent years. Extensive observational evidence across age groups and geographical regions supports the contention that dietary quality is a possible risk or protective factor for depression [5,12–15]. For example, some evidence suggests that adherence to a high-quality diet (healthy/prudent or Mediterranean) is associated with a lower risk for the onset of depressive symptoms [16]. Cross-sectional studies showed that depression was associated with the consumption of ready-made and snack foods among college students [17], of sweet foods among middle-aged women [18], and of high-glycemic index foods among homebound elderly people [19]. However, the relationship between depression and carbohydrate consumption cannot be interpreted causally and is probably bidirectional. A small number of experimental studies with small sample sizes reported that random assignment to diets with a higher carbohydrate content and glycemic load had detrimental effects on mood [20–22],

Longitudinal studies with larger sample sizes have shown an association between an increased risk of depression and consumption of sweetened beverages [23], refined foods, such as fried foods, processed meats, refined grains, and sweetened desserts [24], and pastries (e.g. doughnuts, muffins, other baked goods) [25]. While a ‘Western’ dietary pattern, characterized by high intake

of non-whole grains, white potatoes, cheese, meat, discretionary oil and fat, and added sugar, was not significantly associated with depression, 'healthy' dietary pattern scores (high intake of whole grains, vegetables, fruits, fish, nuts and seeds) were inversely associated with depression scores and odd ratios of depression [26]. Individuals adhering more closely to a Mediterranean-style diet were shown to have a decreased risk of depression over 10 years of follow-up [27]. This association could not be explained by socioeconomic or other lifestyle factors, and there was no evidence of reverse causality. Over a 5-year follow-up period, adults scoring higher on a 'whole food' dietary pattern were shown to have a reduced risk of developing depression, while an increased risk was seen for those scoring higher on a 'processed food' dietary pattern [24]. These relationships remained robust after adjusting for a range of confounding variables and could not be explained by reverse causality. A dietary pattern characterized by consumption of vegetables, fruit, beef, lamb, fish and wholegrain foods was associated with a reduced likelihood of major depressive disorder, while a dietary pattern with a higher intake of processed and 'unhealthy' foods was associated with an increased likelihood of higher psychological symptomatology and clinical depression [28]. In Australian adolescents, inverse relationships between measures of diet quality and the likelihood of depression were found [29]. A 'Western' dietary pattern was also related to an increased risk of depressive symptoms in adolescents [30]. Plausible biological pathways mediating this relationship include adiposity and inflammation, while a 'healthy' dietary pattern appears to be protective in these pathways [30]. The complex associations of dietary patterns, adiposity, inflammation and mental health problems, including depression, require confirmation in longitudinal studies. Other studies have also shown inverse associations between measures of diet quality and depression (e.g. [34–36]). In a randomized clinical trial (RCT), the efficacy of a dietary improvement program for the treatment of major depressive episodes was examined [34]. Despite the preliminary nature of this RCT, the results indicate that a more nutritious diet leads to improved mental health, with dietary improvement, guided by a clinical dietician, possibly providing an efficacious treatment strategy for the management of depression [34]. Replication using more sophisticated study designs in larger samples is necessary to confirm these effects.

High-glycemic index diets could be a risk factor for depression in postmenopausal women. In a prospective

cohort study, a progressively higher dietary glycemic index and higher consumption of added sugars were associated with an increasing likelihood of incident depression, while higher consumption of lactose, fiber, fresh fruit, and vegetables was significantly associated with a lower risk of incident depression [35]. The generalizability of these findings to other populations with depression should be examined.

At present, the available evidence across different nutrients, food types and dietary patterns does not allow firm conclusions regarding an etiological role of nutritional factors in depression [36]. The adherence to certain dietary patterns may provide better predictors of disease risk than the analysis of single nutrients or foods [37]. For example, the Mediterranean pattern was found to be associated with a lower risk of depression [27]. A possible explanation for this relationship is that high levels of antioxidants in fruits, vegetables and olive oil may account for protective effects since depressive symptoms are associated with lipid peroxidation [38]. The findings of cohort studies suggested that diets containing folate, ω -3 fatty acids, olive oil, fish, fruits, vegetables, legumes, and nuts and vegetables may have a protective effect against depression, while diets consisting of processed foods, such as refined grains, processed meat, whole-fat dairy products, fried foods, and chocolate, were associated with a higher risk of depression [36].

In summary, diet may influence the risk of depression, although the available evidence is inconclusive. The promotion of healthy-eating patterns at the public health level may have potential benefits. Methodologically sound prospective cohort studies examining the association between diet and depression risk as well as randomized controlled prevention and treatment trials are needed.

3. Exercise and depression

Adults with major depressive disorder have been found to engage in low levels of physical activity and high levels of sedentary behavior [39]. Since physical activity and sedentary behavior are independent predictors of mortality, lifestyle interventions targeting both the prevention of sedentary behavior and adoption and maintenance of physical activity are warranted [39].

A prospective RCT in patients with major depressive disorder over four months revealed that the efficacy of both supervised exercise in a group setting and home-based exercise seems to be comparable to antidepressant medication; exercise and drug treatments

tended to be better than placebo, i.e. they showed higher remission rates and lower scores on the Hamilton Depression Rating Scale [40]. The findings of a 1-year follow-up of this study suggested that exercise during the follow-up period seems to extend the short-term benefits of exercise and may augment the benefits of antidepressant use [41].

The results of various meta-analyses indicated that exercise has a moderate to large antidepressant effect [42–49]. A Cochrane review determined the effectiveness of exercise in the treatment of depression in adults compared with no treatment or a comparator intervention [50]. Exercise was moderately more effective than a control intervention in reducing symptoms of depression. However, the analysis of methodologically robust trials showed a smaller effect in favor of exercise. When compared to pharmacological or psychological therapies, exercise appeared to be no more effective than either of these approaches, although this conclusion was based on a limited number of small trials. Another systematic review with meta-analysis assessed the benefits and harms of exercise in individuals with depression [51]. The findings of trials with less than a high risk of bias suggested that exercise interventions produced small or negligible antidepressant effects, and there were no significant effects on severity of depression or lack of remission during follow-up [51]. Exercise probably needs to be continued in the longer-term in order to maintain beneficial effects on mood [46].

A narrative review of physical activity and exercise in a depression intervention in young people concluded that these factors are promising in protecting against the development of symptoms of depression [52] and demonstrated bidirectional relationships between physical activity, exercise and adolescent mental health [53]. Physical activity and exercise interventions may be particularly helpful in young people since they are non-stigmatizing and have few side-effects [54].

Depression in elderly people, with its high prevalence and inadequate therapy, creates a substantial societal burden and is a public health priority. Exercise has been proposed as a treatment strategy for elderly sufferers of depression. In a meta-analysis of RCTs, the pooled effect of exercise on depression severity in older people (standardized mean difference (SMD): -0.34) [55] was comparable to the range of effects estimated for different antidepressant medications (SMD = 0.2 – 0.5) [56] and psychotherapy (SMD = 0.18 – 0.34) [57]. In elderly individuals with clinically meaningful symptoms of

depression, structured exercise (with mixed elements of endurance and strength training tailored to individual ability) is likely to reduce the severity of depression. Furthermore, individualized mixed exercise has very few risks, is easy to access and may improve a wide range of additional health outcomes.

Preliminary results of studies with various limitations suggest that exercise could also be potentially beneficial as a treatment or augmentation strategy for impaired cognition in major depressive disorder [58].

Observational data have demonstrated that regular exercise is protective against depression, while physical inactivity is a risk factor for developing depressive symptoms. For example, American women who were more physically active had a reduced risk of clinical depression during 10 years of follow-up [59]. In British birth cohort studies, increased leisure-time physical activity in adolescence was associated with elevated wellbeing in adulthood [60]. Furthermore, regular physical activity in childhood was related to a reduced likelihood of depression in adulthood [61]. A systematic review of prospective studies examined whether physical activity is protective against the onset of depression [62]. It was shown that baseline physical activity was negatively associated with a risk of subsequent depression, providing evidence that physical activity may prevent future depression [62].

In summary, little evidence is available showing exercise to be effective in the treatment of individuals with depression. The antidepressant effect of exercise could possibly be larger if exercise-based programs were tailored to specific populations of depressed individuals. However, there is promising evidence indicating that physical activity can prevent future depression.

4. Diet and dementia

The global aging of populations is accompanied by increased rates of cognitive decline and dementia. The most common form of dementia, occurring in more than half of affected individuals, is Alzheimer's disease (AD) [63]. AD is a progressive neurodegenerative disorder characterized by severe impairments of memory, language, and behavior. The multi-factorial pathology of AD is controlled by various molecular events, including oxidative stress, protein aggregation, mitochondrial dysfunction and neuro-inflammation. Effective treatments for dementia are not, as yet, available. In view of the immense social and economic impact of dementia, preventive measures are urgently needed.

Several strategies in relation to the effects of diet and nutrition in dementia have been investigated. The early decline in brain glucose metabolism in AD has led to investigations assessing the supplementation of the normal glucose supply with ketone bodies (ketogenic diets), since ketone bodies produced during glucose deprivation can be metabolized by the brain when glucose utilization is impaired [64]. Most published observational studies reported an inverse relationship between vitamins, ω -3 fatty acids and AD. The majority of intervention studies suggest beneficial effects of combined vitamins and ω -3 fatty acids when administered in the early stages of the disease [65]. Well designed, high-quality studies need to be conducted to confirm the clinical relevance of these findings.

Polyphenolic compounds, such as resveratrol, have been associated with protection against dementia syndromes such as AD and vascular dementia. The neuroprotective activity of these compounds demonstrated in in-vitro and in-vivo studies suggests a promising role for these compounds in the prevention and treatment of dementia [66,67]. However, the findings of available intervention trials of resveratrol in individuals with mild cognitive impairment or AD do not provide evidence of neuroprotective or therapeutic effects. Pterostilbene, an analog of resveratrol, appears to be more effective than resveratrol in ameliorating brain alterations associated with aging and may be a more promising compound for future research [66].

Consumption of fruit and vegetables has been reported to be inversely associated with the risk of cognitive disorders [68]. Some evidence, mainly derived from observational studies, suggests a protective association between certain nutrients (e.g. folate, flavonoids, vitamin D, and certain lipids) or food groups (e.g. seafood, vegetables, fruits, and potentially moderate caffeine consumption) and cognitive outcomes in older people [69]. A systematic review of recent RCTs explored nutritional intervention efficacy in preventing the onset of late-life cognitive disorders and dementia in cognitively healthy subjects aged 60 years and older [70]. The evidence provided from the 35 included RCTs showed that intervention through dietary pattern changes and medical food/nutraceutical supplementation improved specific cognitive domains or cognitive-related blood biomarkers [70].

An association between nutrition and cognitive outcomes appears to be stronger for healthy dietary patterns (e.g. Mediterranean) than for individual nutrients [69,71]. The Mediterranean diet has been

linked to better cognitive function in the elderly population and may help reduce the risk of developing dementia in a cost effective and sustainable manner [72]. Cohort studies and RCTs have shown beneficial effects of the Mediterranean dietary pattern on cognitive function [73]. The Mediterranean diet appears to reduce the risk of cognitive decline by decreasing the risk of cardiovascular diseases and by exerting anti-oxidative and anti-inflammatory effects [74]. Five RCTs investigating the Mediterranean diet and cognition have so far been published. Most data from these studies were non-significant, with small effect sizes [75]. However, significant improvements in cognitive domain composites in the study using the most robust design [76] warrant additional research. The findings of this study suggested that Mediterranean diet attenuated cognitive decline or improved cognition during a study period of four years [76]. Further RCTs need to be conducted in order to examine long-term effects of the Mediterranean diet in larger samples.

In summary, nutrition is a modifiable risk factor involved in preventing and delaying the onset of age-related cognitive impairment and dementia. Neuroprotective dietary patterns on cognition should therefore be explored in RCTs [77].

5. Exercise and dementia

Certain lifestyle activities, including physical exercise, have the potential to contribute to cognitive reserve capacity and thereby to reduce the incidence of dementia in older adults [78]. Some evidence suggests that exercise may help improve memory function in individuals with mild cognitive impairment [79]. However, evidence from RCTs regarding a role of exercise training in preventing mild cognitive impairment or dementia is sparse and does not support the premise that exercise reduces the risk of developing clinically relevant cognitive outcomes [80]. In a recent multicenter RCT, a moderate to high intensity aerobic and strength exercise training program did not slow cognitive impairment in individuals with mild to moderate dementia [81]. While the exercise training program improved physical fitness, no noticeable improvements in other clinical outcomes were found [81]. A systematic review summarized the effects of physical exercise in older adults with AD on executive function deficits, i.e. disruptions of attentional control, memory, cognitive flexibility, planning, and reasoning [82]. Trends toward improvement in executive function scores were seen across all six studies included, with significant

improvements found in four of them [82]. The small number of available studies call for further long-term exercise RCTs.

Exercise has been demonstrated to preserve white and gray matter, induce changes in the hippocampus, including neurogenesis, and improve cognitive function [83]. Most animal and human studies have suggested that physical activity attenuates neuropathological changes and beneficially affects cognitive function in AD [84]. In elderly people with mild cognitive impairment or dementia, meta-analysis showed a significant effect of aerobic exercise on IL-6 and TNF- α decrease and positive effects on the expression of brain-derived neurotrophic factor (BDNF), while the effects of physical exercise on oxidative processes remain unclear [85].

Although precise physical activity guidelines cannot be provided, the available evidence suggests that the incorporation of regular physical activity into daily routines may mitigate AD-related symptoms, especially in early stages of the disease. In summary, exercise can reduce the risk of cognitive decline and AD and may have beneficial effects on cognitive function [86]. Further investigations to confirm this are needed.

6. Diet and schizophrenia

Schizophrenia is among the most disabling and costly long-term conditions globally [87]. Antipsychotic medications are the mainstay of treatment, with long-term outcomes being poor [88]. In particular, interventions targeting negative symptoms and cognitive dysfunction are needed to reduce residual symptoms. Cognitive impairments, which contribute significantly to poor functional outcomes and long-term disability, are common in patients with schizophrenia. Treatment options are limited [89].

Among all severe mental disorders, individuals with schizophrenia may have the poorest metabolic health and greatest premature mortality [90–92]. The increase in mortality is primarily due to elevated cardiovascular and metabolic risks, since patients with schizophrenia have higher rates of obesity, diabetes mellitus, and metabolic syndrome compared to the general population [93]. Evidence for intrinsic disease links between schizophrenia and diabetes has been provided. Many individuals with first-episode psychosis have been shown to present with impaired glucose tolerance and insulin resistance [94]. A causal relationship could not be established; the two disorders may share intrinsic inflammatory disease links [94]. Antipsychotic

medications are also associated with an increased risk of diabetes, obesity, and metabolic syndrome [93].

Meta-analyses assessing nutrient levels have shown deficits in vitamins B, C, D and E in individuals with long-term schizophrenia [95–98]. Vitamin B9 (folate) and B12 deficits have been shown to be associated with symptom severity [99], and B-vitamin supplementation can markedly reduce symptoms of schizophrenia [100]. In a study assessing serum nutrient status in first-episode psychosis, deficits in vitamin D and folate were shown to be present from the onset of illness and to be associated with worse symptomology [101]. Future studies should examine the direction of these relationships. Certain vitamins and minerals have been suggested to be potentially effective in improving symptomatic outcomes of schizophrenia when administered as an adjunctive to antipsychotics. A meta-analysis of RCTs provided preliminary evidence that vitamin B supplementation may reduce psychiatric symptoms in some patients with schizophrenia, while no symptomatic effects of antioxidant vitamins or dietary minerals were found [100]. These effects may be modulated by inflammatory, glutamatergic, and neurotropic pathways as well as an upregulation of glutathione. The bioavailable amino acid N-acetyl cysteine may have effects as an adjunctive treatment in schizophrenia [102].

In summary, preliminary evidence suggests that certain vitamin and mineral supplements may reduce psychiatric symptoms in some people with schizophrenia. Further nutrient trials should also assess effects on other outcomes such as neurocognition and metabolic health in schizophrenia.

7. Exercise and schizophrenia

The influence of aerobic exercise on symptoms and cognition in schizophrenia has recently attracted increasing attention. Exercise examined as sole or adjunctive treatment in schizophrenia ranges from yoga and light stretching to moderately intense walking, bike riding, or team sports. These interventions suggest beneficial effects of moderately intense exercise [103]. Several studies have shown effects of aerobic exercise on positive, negative and global symptoms [104], quality of life and cognitive functions in individuals with schizophrenia. The findings of a number of published studies point to an effect on cognitive functioning, including global and working memory, attention and vigilance as well as social cognition [105], while other studies could not confirm these results [106].

The impact of aerobic exercise on brain structure, connectivity, and function in patients with schizophrenia has been examined in several studies. The majority of studies investigating the neural effects of exercise in individuals with schizophrenia focused on the hippocampus and reported beneficial effects of exercise (for review see [107]). However, due to differences in duration, frequency, and intensity of exercise, the results were inconsistent. Preliminary evidence indicates that aerobic exercise can increase hippocampal volume and cortical thickness, exerts a neuroprotective effect against hippocampal volume decrease and cortical thinning and markedly increases serum BDNF levels, which are related to neurogenesis, neuroplasticity, and cognitive improvement (for review see [108]). The cognitive benefits of exercise in schizophrenia may be due to neurogenesis stimulated by exercise. However, the available evidence is insufficient to draw definitive conclusions.

8. Diet and ADHD

ADHD is one of the most common psychiatric diagnoses in childhood and adolescence and may persist into adulthood [109]. A variety of environmental and social factors moderating genetic susceptibility may be involved in the etiology of ADHD. Medication and behavior therapy have been shown to yield short-term symptom reduction and other beneficial outcomes in many individuals with ADHD. However, the long-term administration of common ADHD medications has been shown to have no proven efficacy and to be associated with adverse effects [110–112]. This highlights the need to find alternative treatment approaches.

Various nutrients have been linked to brain development and functioning, and dietary improvement may be able to assist in the prevention and therapy of ADHD [113]. Various dietary approaches have been suggested as potentially useful in the treatment of ADHD. The main dietary compounds proposed to be involved in the etiology of ADHD and to have therapeutic efficacy include polyunsaturated fatty acids (PUFAs) and micronutrients such as vitamins and minerals. Evidence of therapeutic efficacy in ADHD of dietary administration of ω -3 PUFAs is marginal or non-existent [114–117]. Pre-treatment PUFA status may influence the efficacy of supplementation, and clinically relevant effects may be confined to individuals with PUFA deficiency. It is unclear whether vitamin and mineral deficits play a role in the etiology of ADHD and whether the respective micronutrient supplementation could have therapeutic

efficacy [113,115]. In view of the considerable controversy surrounding the clinical entity of ADHD [109] and the phenotypic and etiopathophysiological heterogeneity of ADHD, potential therapeutic effects of nutritional supplements could be confined to ADHD subgroups as yet unidentified.

Individuals with ADHD may benefit from improved lifestyle choices [117]. The interrelationship between diet and other lifestyle factors including physical activity should therefore play a more prominent role in the investigation of the etiology and therapy of ADHD.

9. Exercise and ADHD

Acute physical activity has been shown to improve executive functions [118,119]. Children with ADHD have also demonstrated improved executive function following acute exercise [120,121]. Tentative explanations for the exercise effects may be the allocation of attention resources, influences on the dorsolateral prefrontal cortex, and induction of exercise-induced dopamine release.

A limited number of small, open clinical trials have evaluated the impact of physical exercise on behavioral and cognitive functioning in children with ADHD. The findings from these preliminary studies provide some support for the hypothesis that sustained programs of physical exercise are beneficial to children with ADHD [122]. In a systematic review and meta-analysis examining the effectiveness of exercise interventions on ADHD-related symptoms, the main cumulative evidence indicated that short-term aerobic exercise over 6–10 weeks, based on several aerobic intervention formats, was effective in mitigating symptoms such as attention, hyperactivity, impulsivity, anxiety, executive function and social disorders, in children with ADHD [123]. Another review presented the evidence for management of ADHD with long term aerobic physical activity, based on the findings from widely varying studies, research designs and outcome measures [124]. Preliminary evidence suggests that physical activity can be beneficial for ADHD symptoms, executive function, and motor abilities. These findings support the use of physical activity as an adjunctive treatment for ADHD, but present evidence is insufficient to recommend physical activity as a monotherapy [124]. A systematic review of short-term and long-term studies supported the clinical benefits of physical activity on behavioral, cognitive and physical symptoms in individuals with ADHD [125]. Adverse effects of physical exercise were not reported in any study. Another systematic review revealed that physical

activity of 20–30 min (intensity 40–75%) produced positive acute effects on processing speed, working memory, planning and problem solving in young people with ADHD, while data on the effects on behavior were contradictory [126]. Longer term physical activity (≥ 30 min/d, $\geq 40\%$ intensity, ≥ 3 d/week, ≥ 5 weeks) improved attention, inhibition, emotional control, behavior, and motor control [126].

The effect of physical activity during late adolescence on ADHD symptoms in early adulthood was investigated in 232 monozygotic twin pairs [127]. The results revealed that greater weekly energy expenditure in adolescence was significantly associated with reduced ADHD symptom levels in early adulthood, even when controlling for unmeasured confounding (all genetic and shared environmental factors shared within monozygotic twin pairs), and ADHD symptoms and body-mass-index at baseline [127]. In conclusion, physical activity in adolescence might decrease ADHD symptoms in early adulthood.

In summary, preliminary evidence suggests that physical activity may be a protective factor in ADHD and may have positive effects in children with ADHD. However, common limitations of the studies investigating the impact of physical activity on ADHD symptoms are small sample size, lack of randomization, heterogeneity of outcome measures, and the retrospective nature of the studies [128].

10. Diet, exercise, and other mental disorders

Numerous nutritional interventions in other mental disorders have been conducted. For example, a role of gluten and casein in the etiology of autism spectrum disorder together with the therapeutic efficacy of gluten-free and casein-free diets have been proposed (for review see [129]). The nutritional proteins gluten (from wheat) and casein (from dairy products) are metabolized to peptides that have been demonstrated to bind to opiate receptors in the central nervous system and to mimic the effects of opiate drugs. It has been speculated that these opioid peptides, formed during digestion, lead to increased activity in the endogenous opioid system, which is linked to the symptoms of autism ('opioid excess theory') [130]. These peptides might be metabolized insufficiently and may enter the blood circulation as a result of increased permeability of the intestinal membrane ('leaky gut'). After crossing the blood-brain barrier, they may exert direct effects on the brain [131]. However, the scientific basis underlying claims of the efficacy of gluten-free and casein-free diets in the

treatment of autism is weak. A relatively large number of seriously flawed studies and a few methodologically acceptable studies conducted to date do not allow firm conclusions concerning the diet's efficacy. Despite the popularity of the diet, its widespread use and the belief of parents in its benefits, most scientific evaluations have failed to confirm therapeutic effects (for review see [129,132]). Evidence in support of the hypothesis of a role of gluten in schizophrenia is also lacking [133].

The current evidence of a therapeutic effect of exercise in a range of mental disorders can be found in published literature reviews [134,135]. Despite promising findings, adequately powered, high quality RCTs are required to prove the efficacy of exercise in the treatment of mental disorders.

11. Diet, exercise, and the mortality gap in mental illness

Severe mental illness, including schizophrenia, schizoaffective disorder, bipolar affective disorder and depression with psychotic features, is associated with premature mortality [136]. People with severe mental illness have a mortality gap of 15–20 years compared to the general population, and this gap has widened in recent decades [137]. Major driving factors in the markedly increased death rates include preventable cardiometabolic complications due to high rates of overweight and obesity [1,138–139], the iatrogenic effects of some psychiatric medications [140–141], high rates of smoking and a relatively bad diet [142]. High levels of sedentary behavior have been shown to be an independent risk factor for cardiometabolic disease in people with schizophrenia [2,143].

Further lifestyle research may contribute to improved management of the factors associated with premature mortality in individuals with mental illness. For example, lifestyle programs including nutrition interventions have been shown to prevent weight gain in early psychosis [144,145] and reduce weight in the course of the disease [146,147]. However, a major factor in reducing the mortality in people with psychosis is the choice of neuroleptics, since some second generation antipsychotic drugs contribute differentially to metabolic liabilities [148], to raised risks of weight gain [149] and diabetes [150] as well as to increased mortality [151].

12. Diet, exercise, and public health

The German pathologist and anthropologist Rudolf Virchow (1821–1902), father of the public health movement, proposed a role of socio-medical causation,

comprising social, environmental and lifestyle factors, in the etiology and prevention of diseases [152]. In 1848, as a young doctor, Virchow conducted an official investigation into the causes of an epidemic of typhus in Upper Silesia. He posited a clear causal link between the disease, poverty, and the lack of political rights [153]. He proposed radical social reform and became increasingly politically active, including in the 1848 revolution in Prussia.

Following his experiences with the epidemic of bubonic plague in Guangzhou and Hong Kong at the end of the 19th century and his observation of the deplorable sanitary condition of these cities, the Chinese physician and statesman Sun Yat-sen (1866–1925) came to regard sanitation and health as indices of a country's prosperity and degree of social civilization [154]. Sun's recognition of the significance of social conditions in health inequities may have influenced his decision to forego his medical career in favor of revolutionary politics. Sun took a leading role in the Chinese revolution of 1911 and subsequently became the first president of China. While Virchow claimed that a public health doctor must also be an "attorney for the poor" and that "medicine is a social science, and politics nothing but medicine on a large scale" [155], Sun pursued a career in politics rather than medicine in order to prioritize "healing a nation" over "healing the sick".

The views of Virchow and Sun on medical matters informed their political and social convictions. They stressed the importance of health inequities determined by the social conditions under which people live. Both men lived during a time in which major socio-medical issues included starvation and infection, which they believed to be driven by immiseration. Today, the problem of social inequalities may be seen in rates of obesity, metabolic diseases, and mental disorders.

The distribution of income has been identified as the decisive factor in health inequalities [156], with greater income differences associated with lower standards of population health. Health status, health-related behavior and the acceptance of preventive measures are influenced primarily by socio-economic conditions, standard of living, social security, education, work conditions and psychosocial stress rather than by medical care. It has been argued that social injustice remains a major cause of death today [157,158]. While considerable financial resources have been devoted to research and action, the health gradient has remained largely unchanged or has even worsened. At the same time, inequalities in income, wealth and life chances

have also widened [158]. A meta-analysis of studies concluded that rates of mental illness were higher in societies with larger income differences, with the United States and the United Kingdom leading the world in both mental illness and income inequality [159]. In light of this, a significantly greater emphasis on the reduction of health inequalities is of prime importance.

Between 1850 and 2000, a major shift in the leading causes of death from infectious diseases (e.g. tuberculosis, dysentery, cholera, typhoid fever, pneumonia) to other causes, such as heart disease, cancer, stroke, lung disease, and diabetes, took place [160]. This change was largely due to scientifically based public health advances, such as improved sanitation and hygiene, improved nutrition, and the introduction of vaccinations. The average lifespan in humans has doubled since 1900. However, the decrease in mortality due to certain diseases has paved the way for other conditions, such as diabetes, dementia, and chronic kidney disease. It is therefore important today to delay age-related health issues and to increase healthspan, i.e. the length of the time people remain in good health, free of significant long-term conditions, such as diabetes or serious mental illness, frailty, or severe physical disability.

Chronic physical and mental ill health has become an increasing public health concern. In the case of Britain, by the age of 50, most people will have at least one long-term health condition, such as serious mental illness, asthma or diabetes [161]. Psychiatric disorders create a huge social and economic burden for health care systems globally [162]. Mental, neurological, and substance-use disorders, such as schizophrenia, depression, epilepsy, dementia, and alcohol dependence, have been estimated to account for 13% of the global burden of disease, with mental illness causing the greatest burden [163]. The world's health systems are unable to bear, either socially or economically, this extraordinary burden. If health care continues to focus primarily on treating the consequences rather than the causes of ill health, societies will ultimately be unable to afford their health systems. The focus of public health efforts needs to shift to the prevention and delaying of health problems. Evidence-based disease prevention programs reducing modifiable risk factors will lead to both improvements in health and cost reductions. The inclusion of nutrition, diet, physical activity, and sports in psychiatric research and, to a much lesser degree, clinical practice is an emerging field of mental health science. Public policy changes are likely to be needed to translate the findings

of efficacy of dietary and nutraceutical interventions into population-wide changes in eating behavior and related benefits [164]. Similar changes regarding exercise and a physically active lifestyle should also be introduced. Both health improvements and cost reductions could be achieved by providing targeted, evidence-based disease prevention programs that reduce modifiable risk factors.

At the population level, significant cross-sectional correlations between physical activity and mental health have been demonstrated. Regular physical activity was shown in the United States to be associated with a significantly decreased prevalence of current major depression, panic disorder, agoraphobia, social phobia, and specific phobia [165]. An association between leisure-time physical activity of any intensity and depression was also found in Norway [166]. A Dutch study reported lower rates of any affective, anxiety, or substance use disorder in individuals who exercised at least one hour per week [167]. In prospective studies, physical activity reduced both the overall incidence of mental disorders and the incidence of anxiety, somatoform, and dysthymic disorder [168]. A four-year prospective study demonstrated that physical activity decreased the incidence of depressive and anxiety disorders in older adults [169]. There is promising evidence that any level of physical activity, including low levels (e.g. walking <150 min/week), can prevent future depression [62]. From a population health perspective, the promotion of physical activity may serve as a valuable public mental health strategy in reducing the risk of developing depression [62]. Regular physical activity may also have therapeutic implications, since people engaging in regular physical activity were more likely to recover from mental illness at a three-year follow-up [167]. In view of such findings, an increase in the United States health plan coverage for exercise programming in community mental health settings for people with serious mental disorders has been called for [170].

The epidemiological evidence presented above provides the rationale for the initiation of public health measures supporting physical activity and exercise at the population level. Such public disease preventive programs should ideally be introduced in childhood and adolescence, and school-based interventions may be useful and feasible measures. The available literature, based on RCTs, supports the relationship between physical activity, cognition, and academic performance [171]. For example, moderate-to-vigorous physical activity has been shown to be associated with improved

reading and arithmetic skills in children [172]. No evidence exists to support the premise that an increase in curricular physical activity during the school day leads to a deterioration in academic performance. The classroom provides a useful location for interventions designed to increase physical activity since this is where students spend the greater part of the school day. Classroom-based physical activity has been shown to provide a viable approach to improving fitness, body mass index, cognitive function, and academic achievement [171,173]. School lessons incorporating physical activity are inexpensive, easily implemented and may improve both academic achievement and several health issues. Time allocated to physical education has been reduced in many schools. In view of the health, mental, and academic benefits of physical activity, the amount of physical education in schools should be substantially increased. The feasibility of an after-school exercise program for children with ADHD living in an urban poor community was examined in the United States [174]. This study demonstrated that, with proper training and support, after-school physical activity programs can be implemented for children in a high poverty community.

The initiating and financing of large-scale, well-designed studies investigating the effects of exercise on mental disorders should be advocated, particularly since physical exercise brings additional health benefits, including positive cardiovascular and metabolic effects. Adequate funding for this kind of approach may not be readily forthcoming, since financial returns from large-scale investigations of exercise treatment of mental disorders cannot be expected. Support through public funding and non-profit organizations is required to help meet the public mental health challenges.

Physical activity as a therapeutic target is commonly neglected in medical practice, and the available evidence of efficacy of physical activity has not been translated into treatment guidelines. In respect of the practical application of research findings, several questions remain to be answered, including the amount of exercise required to obtain preventive or therapeutic benefits and the extent to which benefits continue following cessation. In comparison to other therapies, changes in physical activity levels are low cost, are easily scalable and can be introduced quickly and easily.

In developed countries, the burden of obesity contributes to increasing health inequality [175]. The prevention and treatment of obesity using behavioral, educational, and pharmacological interventions has

yielded limited success. Evidence supporting the existence of obesogenic environments has emerged [176]. This is a novel approach in obesity research focusing on the investigation of environments that promote high energy intake and sedentary behavior. The obesogenicity of an environment has been defined as “the sum of influences that the surroundings, opportunities, or conditions of life have on promoting obesity in individuals or populations” [177]. The development of effective environmental interventions in relation to obesity requires an understanding of how groups of individuals interact with their environments in terms of physical activity and food intake.

The built environment is a factor that influences lifestyles, body weight (obesogenic environments), and, in light of the importance of physical activity and healthy eating illustrated in this review, mental health. The built environment includes land use (e.g. buildings, parks), transportation systems (roads, public transport), and infrastructure (people’s homes, schools, workplaces, shopping areas) [178]. People’s places of residence provide them with opportunities to be physically active during their day to day activities and leisure time. They provide the potential for outdoor activities and socializing with others. Spending time outdoors has been shown to be positively associated with physical activity (e.g. [179]). Regular physical activity and healthy eating can be either facilitated or impeded by the built environment. Built environments that are attractive and safe and that provide access to healthy food and varied opportunities for physical activity are considered conducive to the adoption of healthy lifestyles. Examples of factors in the built environment that promote physical activity include walkable neighborhoods (presence of sidewalks, walking paths), non-motorized transportation infrastructure (bike paths) as well as recreational and sports infrastructure (playgrounds, parks, pools, sports clubs). Healthy eating can be promoted by providing easy access to food retailers offering healthy, diverse, and affordable foods.

Unhealthy environments are major determinants of childhood obesity. The food industry exposes children to commercial influences that may encourage unhealthy dietary behavior, contributing to obesity. Obesogenic environments influenced by the food industry include schools, retailers, mass media (television, internet), and promotional campaigns [180]. The importance of monitoring the obesogenic environment and identifying policy tools to protect children from food marketing both inside schools and in school surroundings is supported by

recent findings [181]. A new lifestyle intervention, performed in a real-life setting, aims to improve cardiometabolic health in severe mentally ill residential patients by adjusting the obesogenic environment; the effectiveness of the intervention requires further evaluation [182].

Findings in this research area cannot readily be generalized across countries with distinct environments and cultures (e.g. [183]). Most studies on the obesogenic effects of environments stem from North America and Australia as well as Europe and Japan. Country-specific studies are needed to inform local decision making in city planning, transportation, and parks [184]. Coordinated international studies would be helpful in guiding international actions.

In conclusion, the built environment is an important target in addressing weight-related and mental health problems in modern societies. Recommendations include the promotion of active transportation and mixed land use, the improvement of access to sports facilities and infrastructure as well as increased availability of quality foods offered in under-served neighborhoods (‘food deserts’) [185]. The shaping of environmental measures, including urban design and planning, could potentially contribute to successful obesity prevention interventions. This requires further investigation, since the currently available research findings do not allow a clear identification of the manner in which physical environment influences adult weight status [186].

The prevention of dementia will necessarily become a central public health issue in view of the large number of people affected, the continuing demographic change, and the lack of an effective therapy. The available evidence of modifiable risk factors in the primary prevention of dementia could inform a public health agenda [187]. The total impact of a set of risk factors, comprising diabetes, midlife hypertension, midlife obesity, physical inactivity, depression, smoking, and low educational attainment, on the worldwide prevalence of AD has been estimated to be 28.2% [188].

Over-consumption of foods and beverages high in sugar, fat, and salt is associated with an elevated risk of obesity and diet-related non-communicable diseases. The consumption of sugary beverages, in particular, has steadily increased in many countries. The raising of the price of unhealthy foods and beverages through taxation is a potential policy measure to discourage over consumption of these products [189]. An excise tax based on the sugar content of soft drinks has been found to be the most effective tax in reducing the consumption

of these beverages [190]. In the United States, state soft drink sales and excise tax information from 1989 to 2006 suggest that soft drink taxation leads to a reduction in soft drink consumption by children and adolescents [191]. The sugar-sweetened beverage industry, whose effective marketing strategies contribute greatly to obesogenic environments, oppose legislative efforts to pass sugary beverage taxes [192].

Both Rudolf Virchow and Sun Yat-sen clearly understood the relationship between social inequities and health outcomes [193]. Medicine, as a social science, has an obligation to draw attention to health-related problems and to attempt to provide theoretical and practical solutions. Calls for governments to address the challenge of health inequities determined by social conditions are not new [194]. Today, non-communicable diseases cannot be addressed effectively without action on social determinants of health. The problem of increased consumption of energy-dense foods high in sugars, fat, and salt but low in vitamins, minerals, and other micronutrients is rooted not only in people's lifestyle choices and eating habits but also in the lack of availability of healthy, affordable food, especially in urban poor areas [194]. Thus, a social gradient in health is accompanied by a food gradient.

In summary, mental health protects against unhealthy lifestyles, physical disease, and social inequalities. Public health measures should address diet and physical exercise-related issues in preventing mental health problems. Emerging evidence demonstrates the effectiveness of lifestyle changes in the promotion of mental health and in the primary and secondary prevention of mental illness. The encouraging of healthy eating and of an active lifestyle involving physical exercise would contribute to the improvement of population mental health and wellbeing. Public health efforts in regard to lifestyle changes may include school-based interventions and the creation of environments conducive to physical activity and improved nutrition. Political interventions, such as health-related taxes on food and beverages, may also be necessary. Both politicians and clinicians should place a greater focus on the persisting mortality gap and related lifestyle factors in people with schizophrenia.

13. Conclusions

The current bias towards biological and pharmacological approaches to mental disorders, at the cost of social and lifestyle interventions, is heavily promoted by the pharmaceutical industry. Public health interventions may

therefore be needed to facilitate the investigation of lifestyle changes in mental health, independent of the influence of lobbying interest groups.

The consideration of diet and physical exercise in the treatment of mental disorders is warranted for the following reasons: (1) diet and exercise may help improve symptoms of mental disorders; (2) diet and exercise may mitigate the increase in body weight and sedentary behavior induced by lifestyle and psychotropic medications; and (3) diet and exercise can beneficially influence cardiovascular and metabolic problems that may lead to a reduced life expectancy in many serious mental disorders [136,137].

Observational research studies have demonstrated relationships between diet quality and common mental disorders. Biological pathways implicated in mental illness can be modulated by diet. These pathways, including inflammation, oxidative stress, mitochondrial dysfunction, brain plasticity, and gastrointestinal microbiota, are likely to overlap and interact with each other. For some nutrients and food groups, therapeutic efficacy may be greater in individuals with deficiencies in certain nutrients. Furthermore, beneficial effects associated with overall diet composition may be more relevant in nutritional approaches to mental health than single nutrients. In regard to age-related cognitive impairment, current evidence of an association between nutrition and cognitive outcomes is stronger for healthy dietary patterns (e.g. Mediterranean diet) than for individual nutrients, possibly due to the cumulative beneficial effects of the many components of these diets [69].

In regard to dietary influences on mental health, the gut microbiome has recently become a focus of research. The bidirectional communication between gut microbiota and the central nervous system ('gut-brain axis') has attracted increasing interest in recent years. Gut microbiota may affect cognitive function and mental health-related behaviors through multiple pathways [195], and it has even been suggested that the human gut microflora may act as a 'second brain', involved in neurodegenerative disorders such as AD [196]. A Western diet high in saturated fat and added sugars significantly alters the proportions of commensal bacteria in the gastrointestinal tract, thereby impairing cognitive functions [197]. Dysbiosis and inflammation of the gut have been linked to several mental disorders including anxiety and depression [198,199]. Dysbiotic and poorly diversified microbiota may interfere with the synthesis and secretion of neurotrophic factors, such as

BDNF, γ -aminobutyric acid and N-methyl-D-aspartate receptors, which are widely associated with cognitive decline and dementia [200]. Normalizing microbial balance may therefore have a role in psychiatric prevention and therapy.

Numerous nutraceutical interventions have been conducted in populations with various mental disorders. Supplementation has included ω -3 fatty acids, vitamins, minerals, and amino acids. The clinical efficacy of these interventions has not, thus far, been adequately established. Large, long-term trials using sufficiently powered RCT designs are therefore needed to investigate their efficacy and safety. In addition, the effects of different dietary patterns should be examined since the effects of single nutrients may be modest. Food supplements should be administered with caution as they may have unwanted side effects, which may become apparent many years after administration and thus elude detection.

That there are many overlaps and interactions between nutrition and other lifestyle factors, including physical activity, should not be overlooked. For example, the robustly designed RCT demonstrating significant cognitive benefits in people on a Mediterranean diet [76] was conducted in highly active people living in a Mediterranean culture. Thus, the external generalizability of these findings needs to be proven.

Epidemiological studies have found that physical activity and exercise can prevent or delay the onset of various mental disorders and may have therapeutic benefits when used as sole or adjunct therapy in psychiatry. For example, physical exercise appears to be an efficacious therapy for depression and may also be used in the treatment of anxiety disorders. Exercise has been demonstrated to be at least as effective as pharmacotherapy for depressive and anxiety disorders, and preliminary evidence suggests that it is equally as effective as cognitive behavior therapy (for review see [8,201]). Neurobiological mechanisms underlying the effects of physical exercise in mental disorders include neuroplasticity-related synaptogenesis and neurogenesis, which have been identified in animal studies; these could possibly mediate effects of aerobic exercise on brain structure and function (e.g. [202]).

Despite the large number of published trials assessing the effects of physical exercise on mental disorders, further investigations with more robust methodology analyzing the efficacy of interventions in large cohorts and clinically relevant populations are required. In addition, type of exercise, form of execution as well as

duration and intensity of exercise need to be examined. Optimal exercise dosage and dose-response relationships need to be established according to the severity of specific disorders. The effects of the sole administration of exercise versus the combination of exercise and medication should also be studied. Long-term effects beyond the intervention are of importance. Trials in countries outside Europe and North America should be conducted in order to assess the validity of study findings for populations in Africa, Asia, and South America. Furthermore, possible adverse effects, such as exercise addiction [203], should be examined. In addition to the efficacy of exercise in psychiatric symptoms, future studies should also examine possible beneficial effects on metabolism and cardiovascular fitness in people with mental disorders. Given the benefits of exercise for psychiatric symptoms, physical health, and social functioning, feasible methods for delivering exercise in clinical practice should be explored [204].

It is likely that only a small proportion of those with mental disorders use physical activity, exercise or sports as a complementary intervention [205], and an even smaller proportion engages in sufficient exercise to improve their condition [206]. In the same way that too low a dosage of pharmacotherapy or psychotherapy may be ineffective, the right dosage of exercise needs to be determined in the management of mental disorders [207].

A number of factors have been found to be relevant in the success or failure of exercise interventions in mental disorders. Studies that followed public health recommendations [208] concerning the intensity and duration of exercise interventions were more likely to reveal significant clinical improvements than those that did not. Patients' compliance during an exercise program and continuation after program termination has been shown to produce greater benefits in terms of treatment outcomes than general fitness [209,210]. Social support appears to be essential to successful completion of exercise programs and to positive effects derived from them [166,211]. Exercise interventions should be tailored to a person's health, age, social situation, and physical activity interests. Outdoor versus indoor activities may have differential effects on mood states [212].

Obesogenic environments have become a focus of research and may be targets of public health initiatives. A physically active lifestyle is facilitated by societal factors. Politicians should consider how the built environment may influence the physical activity level of the population. Potential areas for policy intervention to

create supportive environments for physical activity and healthy eating at local government level may include the walking environment and food policy [213]. Health systems need to create the necessary environments and infrastructure to ensure that physical activity and supervised exercise can be prescribed as medicine [214]. Policies introducing taxation of sugary drinks and other unhealthy foods can reduce the consumption of these products [189].

Diet and exercise as measures in the prevention and management of mental disorders are widely ignored in academic psychiatry. Nutrition education is largely omitted from medical school and during postgraduate training [215]. Future research needs to go beyond the focus on calories and energy balance and should address the interaction between psychoactive drugs and diet, in regard, for example, to weight gain and metabolic problems.

Individuals with schizophrenia and other mental disorders in high-income countries experience an average mortality gap of 20 years for men and 15 years for women; this is due to lifestyle-related factors, iatrogenic effects of some psychoactive drugs, increased rates of unnatural deaths, and less effective physical healthcare. This “scandal of premature mortality” [136] has been claimed to imply a “failure of social policy and health promotion, illness prevention and care provision” [216], and, further, that the “unacceptable apathy of governments and funders of global health must be overcome to mitigate the human, social, and economic costs of mental illness” [163]. Lifestyle research may contribute to countering the dramatically reduced life expectancy in those with mental illness. Furthermore, antipsychotic medications with a lower metabolic risk should be prescribed, and sufferers from schizophrenia should be able to fully benefit from improvements in health care available to the general population.

14. The bottom line

Major lifestyle factors involved in mental ill health today include modern diet and a sedentary lifestyle. Nutrition, diet and physical activity have long been underestimated as contributing factors to the development and therapy of mental disorders. A disregard of these factors constitutes a failure of clinical practice and of public health policy in the promotion of mental health and the prevention of mental disorders. Lifestyle interventions should be routinely provided to all individuals with mental disorders and should be included in treatment guidelines. The effectiveness of lifestyle changes related

to diet and exercise in improving mental health should be evaluated in the treatment of individuals and in population-based public health programs.

Conflict of interest

The author declares no conflict of interest.

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