The failure to commit to a lifestyle wherein physical exercise constitutes an important aspect of the daily routine is accommodated generally by various expressions of poor health prognosis. For example, among medical students, insufficiency and poor quality of sleep combined with excessive daytime sleepiness influences cognitive ability, with detrimental outcomes, and psychological distress as well as exerting a negative impact on the academic performance of those involved [1]. Remarkably, in conjunction with physical exercise and dietary habits, sleep parameters are construed as the “third pillar of good health”. In a study of European, Hispanic and Afro Americans (N=206, 049) covering anthropometric features (height and weight), sociodemographic factors, health behaviors (smoking and physical activity), emotional distress, and physician-diagnosed health conditions, including hypertension, coronary heart disease, diabetes, heart attack, stroke, kidney disease, and cancer, the associations between short-disturbed sleep and cardiovascular disease and risk factors, body mass index, emotional distress, and lack of physical activity/sedentary behavior were significant even after adjustment for covariates, that included age, ethnicity, gender, marital status, and income: duration of sleep and cardiovascular disease, body mass index, emotional distress, and physical activity all made important contributions to poor health [2]. Sleep disturbance may be associated with cardio-metabolic health issues [3,4]; thus, among adolescents and children guidelines comprise an assortment of combinations relating to sleep, sedentary behaviours, light-, moderate-, and vigorous-intensity physical activity and exercise [5,6]. The concept of ‘movement behaviours’, that involve physical activity, sedentary behaviour and sleep duration, and cardiovascular-metabolic is emerging as an important consideration in health issues both among children and adults [7,8]. Physical exercise affects the quality and duration of sleep over a wide range of age-groups and laboratory animals [9] thereby prompting the notion of ‘exercise-sleep reciprocity’ [10]. Nevertheless, in a study of adolescents concerning the effects of physical exercise and fitness upon sleep-timing parameters no support was obtained for the association between lower levels of exercise and fitness with shorter sleep duration and higher body mass index [11].

Sleep and physical exercise exert reciprocal interrelationships upon each other as a function complicated, multi-layered bilateral interactions bringing into the equation a plethora of multitudinous physiological, psychological and epigenetic pathways. Physical exercise, beneficial in promoting sleep quality and reducing sleep problems, appears also to be associated with several behavioural and somatic attributes involving a multitude of moderating factors that include gender, age-level, overall fitness levels, parameters of sleep and several properties of the exercise type, including intensity, duration, repetitiveness, time of day, before-or-after meals and environment. The necessity of persisting with current notions pertaining to the fundamental physio psychological aspects of the exercise sleep interaction in order to understand the benefits of exercise upon the quantity and quality of sleep among healthy subjects and patients ought to be pursued to further enhancement [9]. In a study of laboratory rodents comparing exercised and non-exercised (sedentary) rats, it was shown that in the sedentary control rats, hippocampal and cortical IL-1β mRNA (interleukin-1β mRNA) expressions among the latter sleep-deprived rats were up-regulated compared to the former, and at the protein levels, hippocampal IL-1β and TNF-α and cortical IL-6 contents were higher among these animals. At peripheral sites, TNF-α (tumour necrosis factor-α), IL-6 and noradrenaline concentrations were greater among the sedentary sleep-deprived rats than among the sedentary control animals. Physical exercise schedules blocked the elevated levels of hippocampal IL-1β mRNA expression and protein content, as well as TNF-α content among the animals experiencing sleep deprivation induction. At the periphery, exercise reduced sleep deprivation-induced increase of IL-6 concentration without affecting the TNF-α and noradrenaline levels. Thus, the seven-week period of an exercise training schedule prior to acute total sleep deprivation prevented the pro-inflammatory reactions that had arisen in the rat hippocampus among the sedentary rats, with particular regard the IL-1β cytokine at both the gene expression and protein content levels [10].

Despite the well-documented health benefits of physical exercise (), it has been described that, among North Americans,
aerobic, endurance, and resistance, muscle-strengthening, exercise is partaken by only 21.7% of individuals (Centres for Disease Control and Prevention, CDC, 2016), a situation presents even in most European countries [12-14], most often due to the reported pressures of job-stress [15]. Sleep health properties are recognized increasingly to constitute important aspects of physical and mental health by both the medical profession and the general public as sleep and its disorders exert intimate influences also with male sexual dysfunction [16]. Remarkably, a News report on the BBC (British Broadcasting Company) related that longitudinal study showed that even weekend exercise enhanced marked health variables [17,18]. Leisure-time physical exercise is linked positively with elevated Positive Affect (PA) and life satisfaction, but not Negative Affect (NA), as has been repeatedly demonstrated [19-21]. Through the application of a weekly diary method from 70 employees across four weekends, employing repeated pre- and post-weekend estimations, such as psychological detachment, relaxation, mastery, and control, weekend physical activity/exercise was linked to lower Monday’s state (NA), using PANAS, only in the cases wherein the workers achieved a high level of psychological detachment from their work schedules during the weekend break. In cases where they had not achieved, their weekend physical exercise/activity was associated with higher levels of NA. Furthermore, the eventual benefits of weekend physical activity/exercise were observed solely in those cases wherein the workers had slept for longer periods during the weekend nights. In a study of 70 employees over four consecutive weekends, a major effect of slightly, not significant, reduced ‘Monday NA’ was moderated by psychological detachment, relaxation, mastery and control, and sleep quality [22]; physical exercise was associated with sleep duration. Taken together, these results imply that physical activity/exercise presents necessary boundary conditions for psychological detachment and sleep duration and quality that influence the presumed recovery influences upon NA and health issues [22,23].

Both late-life sleep disturbances and midlife insomnia-nightmares were associated with impaired cognition, as assessed by the mini-mental state examination, with lowered score levels after 3-11 years [24]. Furthermore, self-reported abnormalities in short-long sleep duration may be related to incident stroke with links between incident stroke and sleep apnea, rapid eye ‘movement sleep behavior’ (see above) disorder, restless legs syndrome, periodic limb movements of sleep, insomnia, and shift work [25]. Both moderate endurance exercise and high-intensity-interval-training exercise incorporated during the early life phase of the lifespan were shown to be effective in reverting or preventing certain metabolic-cardiovascular issues [26]. In depressive and related states, chronic stress (bereavement/sorrow) seems to prime individuals to experience an exaggerated inflammatory response to episodes of acute stress thereby underlining the association between self-reported sleep disturbances and inflammation linked to depressive symptoms. It has been observed that depression in the form of bereavement moderates the association between self-reported sleep disturbances and pro-inflammatory conditions [27]. In both clinical and animal laboratory models of depression, as well as in the brains of suicide victims, brain-derived neurotrophic factor levels are reduced [28], and in some cases increased through treatments with antidepressant compounds [29].

Positive Affect (PA), Body Mass Index (BMI) and Obstructive Sleep Apnoea (OSA), all presenting features of chronic cardiovascular disorders among older adult individuals, with somewhat higher severity among manual workers, were examined in a cross-sectional study that assessed the relationship between PA, BMI and OSA severity in manual workers. Fifty-five individuals participated, 23 females, 32 males, with mean ages 55.2, were examined for OSA and completed a PA and anthropometric assessment. Taken together at mean levels, the incidence of OSA severity was mild, the PA levels were moderate and 32% of the sample was classified as obese yet it was found that the levels of PA were associated negatively with the levels of OSA severity, whereas BMI strongly and independently predicted levels of OSA severity, despite the lack of evidence for mediatory effects. As both PA and BMI were significantly associated with OSA in older manual workers, increasing PA should also be a focus of treatment for OSA [30]. Exercise delivers a unique non-pharmacologic, non-invasive, hermetic intervention that incorporates a range of regimes whether dynamic or static, endurance, aerobic or resistance [18,19]. In a study involving both adolescent and adult populations (N = 280, 144 male and 136 female participants), it was shown that the propensity and compliance for physical exercise, measured as the “Archer-Garcia ratio”, was predicted overwhelmingly by PA and was associated with health, well-being, positive dimensions with affect and age-level [31]. The associations between sleep duration and quality seem well-grounded: thus among adolescents, sleep quality showed stronger links with all measures of emotions/affectivity as compared to sleep duration whereby shorter sleep duration was associated more specifically with lower positive emotions (PA: happiness, followed by positive affect), whereas poorer sleep quality displayed stronger associations with Negative Affect (NA). The protecting of sleep duration appears promote positive emotions and the enhancement sleep quality induces reductions of mood disturbances [32].

Various studies incorporating cross-national data indicate that extremes of inactivity/activity can significantly influence insomnia risk independent of country. Insomnia risk associated with very low levels of activity may be mediated by poorer health and disadvantageous social status. However, while very high levels of activity increase insomnia risk independent of health and demographic factors, they may also confound with personally and occupationally demanding lifestyles [33]. Intuitively, time spent outdoors is related to physical exercise/activity and time spent...
indoors is related to sedentary behaviour and deficits in, amongst other necessities, sleep [34]. In this context, it is noteworthy that the variation of symptoms among insomniacs presenting comorbidity for depressive disorders is modulated by BDNF gene polymorphisms as indicated by the greater prevalence of the heterozygous (A/G) VAL/MET polymorphism compared with healthy controls [35]. Furthermore, it was observed also that both the serum BDNF concentrations and the BDNF Val66Met polymorphisms among a population of healthy young adults, 45 male and 34 female participants, aged 20 to 29 years, were related to their specific sleep patterns during weekends but not with those observed during the weekdays, implying that the systems involved in the BDNF availability control may be linked to certain endogenous sleep characteristics rather than the socially-constrained sleep schedules among these healthy young adults [36,37]. Also, different phases of the sleep-waking cycle, such as sleep-wake activity and the homeostatic regulation of rapid eye movement sleep of both male and female rats demonstrate unequivocally that an intact BDNF system presented a critical modulator describing the baseline activity and homeostatic regulation of rapid eye movement sleep [38]. Suffice it to say, the influence of physical exercise in promulgating expressions of BDNF benefits to ameliorate a plethora of affective and neurodegenerative markers over the lifespan, but predominantly among ageing individuals, has been well-documented [39-43]. Nevertheless, although the advantages of exercise in promoting sleep properties provide health bonuses, the detrimental effects of ‘over-exercise’ ought not to be neglected: thus, among female youth athletes, decreased sleep duration and increased training-load were associated independently with impairments of individual subjective well-being [44].

Conclusions

Despite certain notable exceptions, the general consensus seems to be that physical exercise, through enhancing PA, reducing NA, promoting BDNF, reducing obstructive sleep apnoea, improving metabolic and cardiovascular status, alleviates/boosts both the duration and quality of sleep among those afflicted by sleep problems and healthy individuals. It is necessary to maintain an awareness of the reciprocity of sleep-exercise-sleep in considerations of the health determinants of these conditions. Finally, the hormesis characteristics of exercise schedules, independent of type and properties, would appear to affect the psychological and somatic resilience to sleep disturbance as found to be influential in other instances of poor health, not least those observed during ageing [45].

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