Obesity Medicine 7 (2017) 4–14

Contents lists available at ScienceDirect

Obesity Medicine

journal homepage: http://www.journals.elsevier.com/obesity-medicine

Effects of sleep deprivation on neurocognitive capacities in obese individuals not qualifying for sleep treatment: A systematic review

Harunya Sivanesan [a], Junaid Ahmad Bhatti [b, c, d, *]

[a] Sleep Lab, Toronto Rehabilitation Institute, Toronto, Canada
[b] Sunnybrook Research Institute, Evaluative Clinical Sciences, Toronto, Canada
[c] University of Toronto, Department of Surgery, Toronto, Canada
[d] Institute for Clinical Evaluative Sciences, Toronto, Canada

ARTICLE INFO

Article history:
Received 6 January 2017
Accepted 16 June 2017

Keywords:
Anthropometric
Sleep apnea
Daytime sleepiness
Reaction times
Vigilance

ABSTRACT

Background: Obesity is an independent predictor of sleep deprivation (SD) and neurocognition. The effects of obesity on neurocognitive performance have mostly been studied for SD conditions requiring treatment, e.g., severe obstructive sleep apnea (OSA). We reviewed studies assessing the effects of SD on neurocognitive capacities in obese individuals not qualifying for an SD treatment.
Methods: We conducted a descriptive systematic review. We searched for studies using three electronic databases: MEDLINE®, Psych Info®, and EMBASE® (1980–2016). We included studies that compared the effects of SD in obese participants to that of obese participants without SD condition or non-obese participants under SD condition. We excluded studies on participants qualifying for SD treatment, such as treatment for severe OSA, or obesity-associated comorbidities.
Results: Of 859 studies, 17 met the inclusion criteria. These studies assessed the effects of eight SD conditions on 22 neurocognitive tasks in obese participants. Obese individuals with mild to moderate OSA show significantly lower neurocognitive performance compared to obese individuals without OSA, on tasks assessing vigilance (four studies), phonemic fluency (one study), executive function (one study), and verbal memory (one study). Two studies found that obese adolescents with shorter sleep hours performed worse on a global cognition scale and for fine motor skills. Studies noted that extending sleep hours resulted in better performance on tasks relating to attention, intelligence, and decision-making.
Conclusion: Available studies suggest that SD might worsen neurocognitive capacities in obese individuals. Such impairments in obese individuals might have clinical implications, such as increased injury risks.

1. Background

Sleep is an essential survival necessity (Bernier et al., 2013; Ferrara et al., 2012; Jackson et al., 2013). While sleep patterns vary individually, there is a consensus that adults require about eight-hours of sleep to function optimally (Ferrara et al., 2012; Jackson et al., 2013; Esposito and Carotenuto, 2010). However, sleep deprivation (SD) has become increasingly common (Bernier et al., 2013; Esposito and Carotenuto, 2010; Born and Wilhelm, 2012; Kallestad et al., 2012; Kopasz et al., 2010). For instance, a recent survey reported that about one in nine Canadians slept six hours or less per night (Gilmour et al., 2013). Chronic SD is associated with conditions such as glucose intolerance, cardiovascular diseases, hypertension, and impaired immune system functioning (St-Onge et al., 2012; St-Onge and Schechter, 2014). SD bears negative effects on neurocognitive performance, which can lead to impairments in procedural memory and academic performance (St-Onge et al., 2012; St-Onge and Schechter, 2014; Manber et al., 1995; Smith, 2001).

Sleep disorders, i.e., sleep-associated medical conditions, are not uncommon (Karacan et al., 1983). It is found that 15–35% of the adult population have some symptoms of poor sleep quality, such as difficulty falling or maintaining sleep (Bixler et al., 1979; Karacan et al., 1983). Obstructive sleep apnea (OSA) is the most prevalent sleep disorder (Boeka and Lokken, 2008). OSA leads to repetitive upper airway collapse during sleep (Ayas et al., 2014a; Fleetham

* Corresponding author. Sunnybrook Health Sciences Centre, 2075 Bayview Avenue, G106, Toronto, ON, M4N 3M5, Canada.
E-mail address: junaid.bhatti@ices.on.ca (J.A. Bhatti).

http://dx.doi.org/10.1016/j.obmed.2017.06.002
2451-8476/

et al., 2011). OSA can cause poor sleep quality and sleep disruption that results in excessive daytime sleepiness (Boeka and Lokken, 2008). In the United States (US), it is estimated that about 9% of men and 4% of women suffer from OSA (Nabil et al., 2009). Another estimate suggests that about 80% of OSA cases are undiagnosed (Lee et al., 2008). OSA severity is often defined by the apnea-hypopnea index (AHI) and by excessive daytime sleepiness that is assessed clinically by the Epworth Sleepiness Scale (ESS) (Boari et al., 2004). Patients with an AHI index of 30 or higher, or an ESS of 15 or higher, are considered for a medical treatment such as Continuous Positive Airway Pressure (CPAP) (Boari et al., 2004). CPAP treatment in severe OSA has shown to improve daytime functioning as well as reduce the risks of OSA-associated comorbidities such as cerebrovascular events, myocardial infarction, hypertension, and diabetes mellitus (Boari et al., 2004; Golbin et al., 2008; Jean-Louis et al., 2008, 2010).

Literature consistently indicates correlations between SD conditions and obesity (Resta et al., 2001). For instance, OSA is found to be most prevalent in overweight or obese individuals (Resta et al., 2001). Resta et al. found that OSA is present in more than 50% of obese individuals with a mean Body Mass Index (BMI) greater than 40 kg/m (Ferrara et al., 2012) (Resta et al., 2001). Over the last four decades, the prevalence of obesity, i.e., individuals with a BMI of 30 kg/m^2 or more, has tripled in North America, Western Europe, and Australia (Collaboration NCDRF, 2016). Studies have shown that obesity trends are directly associated with increasing OSA diagnosis (Resta et al., 2001). One reason is increased airway collapse because of adipose tissue leading to sleep disordered breathing (Resta et al., 2001). Foster et al. (2009) found that a one unit increase in BMI increases the risk of having OSA by almost ten percent (Foster et al., 2009).

Nonetheless, there is evidence that obese individuals may suffer from SD by mechanisms other than the OSA (Ohayon and Vecchierini, 2005; Vorona et al., 2005). Studies have shown that neuro-inflammatory responses as a result of excessive adipose tissue increases sleepiness in asymptomatic obese individuals (Gaines et al., 2016; Kritikou et al., 2015). Gaines et al. (2016) found that the association of visceral fat and sleep-disordered breathing was mediated by two neuroinflammatory cytokines, C-reactive protein (CRP) and Interleukin-6 (IL-6) (Gaines et al., 2016). The effects of these markers on these associations were about 82% and 42% respectively. Moreover, the study also found that individuals with mild to moderate OSA had higher levels of visceral fat, IL-6, CRP, and leptin (Gaines et al., 2016). Another study showed that OSA is associated with HPA (hypothalamic-pituitary-adrenal) activation in both obese and non-obese men (Kritikou et al., 2015). It has been reported that this excessive autonomic arousal could dysregulate the sleep cycle and lead to SD (Henley et al., 2009). Taken together, the literature suggests that obese individuals might be prone to SD though they might not have severe OSA qualifying them for CPAP.

Neurocognitive task performances are sensitive markers of the detrimental effects of SD on physical and psychosocial functioning (Van Dongen et al., 2003). Literature is consistent that restricting sleep duration to four to six hours per day over two weeks has detrimental effects on neurocognitive performance (Van Dongen et al., 2003). For instance, reaction times in the Psychomotor Vigilance Task (PVT) and the processing speeds in the Digit Symbol Substitution task (DSST) are prolonged in SD individuals, as compared to those who are not sleep-deprived (Van Dongen et al., 2003). The evidence about the neurocognitive effects of SD in otherwise healthy obese individuals has not been reviewed (Van Dongen et al., 2003). In particular, literature is lacking on sleep conditions that do not require immediate treatment such as CPAP. Some evidence does suggest that obese individuals might be more likely to have suboptimal physical performance and are likely to suffer from traffic injuries and falls as a consequence (Wiegand et al., 2009; Osborne et al., 2014). Both SD and obesity are pervasive, and understanding their combined impact on neurocognitive capacities could help in explaining health risks in obese individuals (Jackson et al., 2013; Karimi et al., 2015). We reviewed studies comparing the SD effects on neurocognitive capacities in obese participants to that of obese participants without SD or non-obese participants under SD conditions. We excluded studies on patients qualifying for an SD treatment or with OSA comorbidities.

2. Methods

2.1. Study design

We conducted a descriptive systematic review of peer-reviewed manuscripts indexed in three most commonly used databases, namely, MEDLINE®, EMBASE®, and PSYCINFO®. We used the OVID® platform to retrieve titles of published manuscripts published between January 1, 1980 and June 30, 2016. This review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines.

The search was limited to journal articles published in English and studies conducted in humans. We entered terms related to obesity, neurocognitive tasks, and SD used in previous systematic reviews (Astill et al., 2012; Kohler et al., 2010; Lal et al., 2012). For example, the following combinations were used to extract study abstracts from the OVID® platform:

((Obes* OR Obesity OR Obese OR body mass index OR BMI) AND (sleep deprivation/apnea (sleep restrict* OR sleep depriv* OR sleep apnea OR obstructive sleep apnea OR OSA)) AND (cognitive domains (Neurocognition' OR 'Neurocognitive' OR 'Neuropsychology' OR 'Neuropsychological' OR 'Cognitive' OR 'Cognition' OR 'Intelligence quotient' OR 'IQ' OR 'VIQ' OR 'PIQ' OR 'North American Adult Reading Test' OR 'NAART' OR 'wide Range Achievement Test' OR 'WRAT' OR 'Wechsler Adult Intelligence Scale' OR 'WAIS' OR 'Mental speed' OR 'Digit Symbol Substitution Test' OR 'DSST' OR 'Trail Making Test' OR 'TMT' OR 'Reaction time' OR 'Attention' OR 'Attentional' OR 'Vigilance' OR 'Concentration' OR 'Continuous Performance Test' OR 'CPT' OR 'Digits Forward' OR 'Learning' OR 'Memory' OR 'Working memory' OR 'Declarative memory' OR 'Verbal memory' OR 'Non-verbal memory' OR 'Visual memory' OR 'Logical memory' OR 'Autobiographical memory' OR 'Prospective memory' OR 'Immediate memory' OR 'Delayed memory' OR 'Verbal learning' OR 'Digits Backward' OR 'California Verbal Learning Test' OR 'CVLT' OR 'Rey Auditory Verbal Learning Test' OR 'RAVLT' OR 'Wechsler Memory Scale' OR 'WMS' OR 'Free recall' OR 'Rey Complex Figure Test' OR 'RCFT' OR 'Verbal skills' OR 'Verbal fluency' OR 'Category fluency' OR 'Letter fluency' OR 'Controlled Oral Word Association Test' OR 'COWA-FAS' OR 'Animal Naming' OR 'Visuospatial' OR 'Constructional' OR 'Block design' OR 'Rey Complex Figure Test' OR 'RCFT' OR 'Clock test' OR 'Executive function' OR 'Reasoning' OR 'Inhibitory control' OR 'Executive control' OR 'Concept formation' OR 'Wisconsin Card Sorting Test' OR 'WCST' OR 'Stroop Color Word Test' OR 'Stroop' OR 'SCWT' OR 'Theory of Mind' OR 'ToM' OR 'Emotion processing' OR 'Emotional decision-making' OR 'Benton Facial Recognition Test' OR 'BFRT' OR 'Faces Test' OR 'Eyes Test' OR 'Hinting Task' OR 'False belief and deception' OR 'Picture sequencing' OR 'Character intention' OR 'Faux Pas'))

2.2. Study selection

The study selection was done in following steps. *Identification*: After retrieving the potential studies, the first author read the titles and abstracts. The reviewer selected, without prejudice, the