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Undiagnosed Obstructive Sleep Apnoea and Physical Activity in Older Manual Workers.

Abstract

Cardiovascular disease (CVD) is a negative health outcome of Obstructive Sleep Apnoea (OSA). Risk factors associated with OSA development include low physical activity (PA), high body mass index (BMI), and increasing age (>50 years) and weight loss is usually recommended as treatment. This cross-sectional study examined the association between PA, BMI and OSA severity in manual workers. Fifty-five participants, (23 females, 32 males) mean age 55.2, were examined for OSA and completed a PA and anthropometric assessment. On average, OSA severity was mild, PA levels were moderate and 32% of the sample was classified as obese. PA was negatively associated with OSA severity, but BMI strongly independently predicted OSA severity, with no evidence of mediation. 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.

Keywords: Sleep disordered breathing; exercise; aging workforce; increased BMI; sleep apnoea.

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Sleep is essential for life and integral to health and wellbeing (Roehrs, 2000). However, during the ageing process sleep patterns become disrupted, which leads to sleep deprivation (Stanley, 2005). Inadequate sleep has adverse effects on the body, including the production of higher cortisol (stress hormone) levels, which causes wear and tear physiologically, and further contributes to wakefulness (Stanley, 2005).

Although it is uncertain whether age-related sleep changes predict the onset of sleep complaints, a common condition responsible for age-related sleep disturbance, is a respiratory sleep disorder called obstructive sleep apnoea (OSA) (Al Lawati, Patel, & Ayas, 2009; Banno & Kryger, 2005). OSA is caused by upper airway collapse resulting in repeated incidence of airflow cessation (apnoea) or airflow reduction (hypopnoea), and consequently leads to arousals from sleep, alongside increased sympathetic nervous system activation, hypoventilation and hypoxemia (Al Lawati et al., 2009). The severity of OSA is measured as the number of respiratory interruptions that coincide with a decrease in oxygen saturation (apnoea-hypopnoea index) (AHI). The AHI indicates the number of apnoea and hypopnoea episodes detected in one hour of sleep (Laitinen, Anttalainen, Pietinalho, Hamalainen, Koskela, & Group., 2003)

It is estimated that 1.5 million adults in the UK have OSA, although only approximately 330,000 are actually diagnosed and treated (Rejon-Parrilla, Garau, & Sussex, 2014), thus, many adults may unknowingly have undiagnosed OSA. Recently, Webber, Lee, Soo, Gustave, Hall, Kelly, and Prezant (2011) illustrated the incidence of undiagnosed OSA in a group of male rescue workers (firefighters and emergency medical staff) (n = 13,330) with a mean age of 44.2 years and found that only 13.9% who were deemed at risk for OSA were actually diagnosed. Owing to an increasingly older population (Government Office for Science, 2016) and rising obesity levels (NHS, 2013), the prevalence of OSA is likely to

worsen over time (Rejon-Parrilla et al., 2014). This is of enormous concern as OSA has serious implications for health, increasing the risk of conditions such as cardiovascular disease (CVD) (Floras, 2014), type-2 diabetes (Pamidi & Tasali, 2012), and hypertension (Zhang & Si, 2012). Thus, early diagnosis is essential if health and wellbeing are to be preserved over the course of working life and into retirement.

As many companies now need to retain older workers beyond retirement age, it is important to bear in mind the impact of ageing-related diseases on health and wellbeing and ability to work, particularly in respect to OSA (Billett, Dymock, Johnson, & Martin, 2011). Laitinen et al. (2003) suggest that moderate to severe OSA is prevalent in males below retirement age and that the incidence of OSA peaks around the age of 50-59 years. Age-related physiological changes that are believed to contribute to the development of OSA include tissue and systemic alterations. For instance, one suggested mechanism of OSA, a greater stiffness of the soft palate found in middle-aged sleep apnoea patients, is thought to be caused by the pharyngeal fatty infiltration brought about by ageing (Veldi, Vasar, Vain, & Kull, 2004). Growing older also triggers significant changes within the cardiovascular system, which predispose an individual to CVD and as such adds additional CVD risk to that presented by OSA (McEniery, Wilkinson, & Avolio, 2007). In addition to the above naturally occurring changes, poor health behaviours such as excessive alcohol consumption, smoking, obesity and lack of physical activity (PA) may amplify the likelihood of developing OSA (Koyama, Esteves, Oliveira e Silva, Lira, Bittencourt, Tufik, & de Mello, 2012; Simpson, McArdle, Eastwood, Ward, Cooper, Wilson, Hillman, Palmer, & Mukherjee, 2015). Thus, the physiological effects of growing older together with poor health behaviours are likely co-contributors to the development of OSA.

The health implications of OSA have enormous impact on quality of life and the ability to function effectively at work. For example, certain features of OSA such as

excessive daytime sleepiness and morning headaches may present their own problems during the course of the working day (Banno & Kryger, 2005), such that work efficiency may be compromised and could result in sickness presenteeism (attending work whilst ill) (Guertler, Vandelanotte, Short, Alley, Schoeppe, & Duncan, 2015). Sleep loss owing to OSA becomes a real problem in the workplace in terms of production and even health and safety (Gaultney & Collins-McNeil, 2009). This is particularly true of older workers in manual occupations, where non-health related outcomes of OSA such as difficulty in staying awake, may lead to an increased risk of workplace injury (Heaton, Azuero, & Reed, 2010). Occupations that involve driving carry a high risk of road traffic accidents where OSA is concerned (Lemos, Marqueze, Sachi, Lorenzi-Filho, & de Castro Moreno, 2008). Road traffic accidents relating to OSA are costly to industry and to lives and have been evidenced to warrant that American commercial drivers in Philadelphia are screened before being deemed fit for work (Gurubhagavatula, Nkwuo, Maislin, & Pack, 2008).

OSA-related sleepiness might be even more of a problem in older manual workers, who are already likely to experience age-related fatigue (Kiss, De Meester, & Braeckman, 2008). As a result of daytime sleepiness, adults aged over 50 years old with OSA are unlikely to practice good health behaviours such as regular PA, a lack of which has been shown to be associated with moderate to severe OSA (Peppard & Young, 2004; Simpson et al., 2015). Chennaoui, Arnal, Sauvet, and Leger (2015) suggest that OSA limits exercise, however, Butner, Hargens, Kaleth, Miller, Zedalis, and Herbert (2013) disagree and conclude that capacity to exercise is more likely related to age and body weight. Either way, previous research suggests that the severity of OSA increases with age and weight, and that regular PA may alleviate age-related worsening of cardiorespiratory function, promote weight loss, and potentially reduce the severity of OSA (Butner et al., 2013; Vanhecke, Franklin, Ajluni, Sangal, & McCullough, 2008). Recent evidence highlights the potential of PA in reducing

the severity of OSA (Aiello, Caughey, Nelluri, Sharma, Mookadam, & Mookadam, 2016), however, there is limited evidence regarding the association between OSA and PA in those older than 50 years. The available literature generally suggests that low levels of daily PA is associated with increased severity of OSA independent of BMI in those who are older than 50 years old (Peppard & Young, 2004; Simpson et al., 2015; Stanley, 2005; Verwimp, Ameye, & Bruyneel, 2013). Further, research is sparse in respect of PA and the occurrence of undiagnosed OSA in older manual workers.

It is also important to bear in mind that the ability to perform manual work competently without negative effect to health requires cardiorespiratory fitness, which may be achieved through regular leisure-time PA (Leino-Arjas, Solovieva, Riihimaki, Kirjonen, & Telama, 2004). As such, strenuous workloads that regularly exceed a worker's ability due to lack of fitness will increase the risk of long-term sickness absence and the development of CVD. On the contrary, increased leisure-time PA may decrease these risks (Holtermann, Burr, Hansen, Krause, Sogaard, & Mortensen, 2012; Holtermann, Hansen, Burr, Sogaard, & Sjogaard, 2012). Notably, manual workers are reported to have lower levels of leisure time PA than those of white-collar workers (e.g. managers) (Gram, Westgate, Karstad, Holtermann, Sogaard, Brage, & Sjogaard, 2016; Leino-Arjas et al., 2004). There is also evidence to suggest that increased BMI is common among manual workers (Fransen, Wilsmore, Winstanley, Woodward, Grunstein, Ameratunga, & Norton, 2006). Thus, together with high physical workload, low levels of PA and high BMI, manual workers are at increased risk of OSA and CVD development.

Consequently, the aim of the present study was to explore whether PA is associated with undiagnosed OSA as measured by an AHI score of ≥ 5, in a sample of manual workers aged over 50 years old (Ejaz, Khawaja, Bhatia, & Hurwitz, 2011). It was hypothesized that

those with an undiagnosed presence of OSA would report low levels of PA, independent of BMI.

Method

Participants and Design

Participants were recruited by the researcher through advertising in an original population of 225 manual workers. The group was comprised of healthcare workers (nurses, paramedics and healthcare assistants), Physical Education teachers, and craft/labourers aged over 50 years old (mean = 57.11, standard deviation (SD) = 5.62 years) who were recruited in 2015 as part of a study on presenteeism in manual workers from various industries (Thogersen-Ntoumani, Black, Lindwall, Whittaker, & Balanos, 2017). Those who already had a diagnosis of and were receiving treatment for OSA, or were under current investigations for OSA, or had a history of cardiorespiratory conditions were excluded. Participants who met the inclusion criteria i.e. had no previous diagnosis of OSA, who had no history of cardiorespiratory conditions, were otherwise healthy and were not receiving treatment for OSA were included and invited for testing. Fifty-five participants (healthcare workers, PE teachers and craft/labourers) willing to undertake further investigation were invited to complete a questionnaire to measure daily levels of physical activity, and to wear a home sleep screening device overnight to identify the presence of obstructive sleep apnea. Participants were told that the ApneaLink would indicate the degree of OSA but was not an official diagnosis. Results of the screening were given to the participants in confidence via a telephone call or in person. Participants were encouraged to see their GP if our screening was suggesting that OSA was present. The University of Birmingham STEM ethics committee approved the study and written informed consent was given by all participants.

Measures

Physical Activity Questionnaire. The Baecke Questionnaire (Baecke, Burema, & Frijters, 1982) was used to measure habitual levels of physical activity. The Baecke Questionnaire measures physical activity across three contexts: sport/exercise, occupational (referred to as work), and leisure-time physical activity in the past month, and provides total and domain-specific scores. The questionnaire includes a rating of amount (hours per week, months per year) and type (swimming, jogging etc.) of activity. It has been tested in a group of workers over one month with a test-retest reliability of 0.71 (Philippaerts & Lefevre, 1998). Congruent validity and concurrent validity have been supported across three levels of professional status (manual workers, clerks and managers (Philippaerts & Lefevre, 1998). More recently, (Hertogh, Monninkhof, Schouten, Peeters, & Schuit, 2008) reported in a validation study that the scale can correctly classify low (0-8) and high (>15) activity, but is less accurate in identifying moderate (9-15) activity. The Cronbach's alpha for internal consistency in the present study was 0.75.

Sleep Testing. To assess the presence of OSA, participants wore an ApneaLink™ (ResMed, Sydney, Australia) for one night. ApneaLink™ is a home sleep monitoring device and has been widely used in research as a tool for detecting sleep-related breathing conditions. The device is a two-channel monitor that provides ventilatory data via nasal flow, and oxygen saturation via pulse oximetry, which are analyzed to give an apnea/hypopnea index (AHI) score indicating OSA (ResMED, 2006). A minimum wear-time of at least two hours, but ideally four hours, is required for a valid evaluation (Erman, Stewart, Einhorn, Gordon, & Casal, 2007). The device has 100% sensitivity and is 87.5% specific in identifying apnea, together with a 95% success and compliance rate (Patel, Alexander, & Davidson, 2007). It thus, provides a reliable and cost effective method of screening and diagnosing OSA.

Participants wore the device at home and were scored for OSA based on an apnoea/hypopnoea index (AHI). As per the ApneaLink™ default settings, apnoea was

defined as a reduction in flow by 20% of normal for at least 10 seconds and hypopnoea as a reduction in flow by 70% of normal for at least 10 seconds (Crowley, Rajaratnam, Shea, Epstein, Czeisler, Lockley & Safety, 2013). Pulse oximetry data became indicative for the presence of OSA when during apnoeas and hypopnoeas saturation dropped by more than 5% below normal. The severity of OSA is defined by AHI as follows: an AHI of 5-15 per hour was defined as mild OSA, AHI of 15-30 per hour as moderate OSA and AHI > 30 per hour as severe OSA (AASM Task Force, 1999).

Anthropometric Measures. Prior to testing, height and weight were measured to calculate body mass index (BMI) (kg/m^2). Salter Ultra Slim Glass Analyser scales (Model no.9141, HoMedics Group Ltd., Kent, UK) was used to measure weight, body fat percentage (through bioelectrical impedance) and BMI (kg/m^2). A Bosch PLR 30c Laser Measure (Robert Bosch GmbH, Germany) was used to measure height.

Procedures

A participant information sheet with details on the use of the ApneaLink™ device was provided alongside a demonstration on how to wear it. Participants were shown by the researcher how to operate the device and given a paper illustration for reference. Participants were required to wear the device on a "normal/regular" night and to go to sleep at their habitual bedtime. Wear time was required overnight for one night and the device was collected the following day. All participants wore the ApneaLink™ for the minimum required time of four hours (Erman et al., 2007). ApneaLink™ software produced a computer generated summary report with automatic scoring for interpretation of AHI score and presence of OSA. Graphical readouts from the ApneaLink™ were checked by trained researchers to ensure that they reflected the AHI score given.

Data Analysis

Data analysis was conducted using IBM Statistical Package for the Social Sciences (SPSS) version 22. Firstly, correlations and ANOVAs were performed to explore potential associations between OSA severity, PA, socio-demographic variables and BMI. Further exploration of the associations between OSA, PA and BMI was carried out using linear regression analysis. Change in R-squared is reported as the effect size from the regression analyses. Mediation analyses were conducted using the PROCESS macro (Hayes, 2013) based on simple linear regression modelling.

Results

Descriptive Statistics

Descriptive statistics for all participants are shown in Table 1. The mean (SD) age of the participants was 55.2 (4.21) years, and the sample included slightly more males (58.2%) than females. The mean AHI score was 8.9 (11.75) indicating undiagnosed levels of mild OSA. Mean Physical Activity (PA) levels were reported as moderate, and 32% of the sample was classified as obese with a BMI $\geq$ 30 kg/m^2. PA levels were further broken down to reveal high levels of work PA (heavy lifting and walking), moderate leisure PA and lower levels of sport and exercise (e.g. running, swimming, cycling) (Table 1). OSA severity groups were grouped as follows: 'No OSA' = 25, 'Mild OSA' = 19, 'Moderate OSA' = 7, 'Severe OSA' = 4. Of those with mild OSA, 14 participants had a lower BMI than the group mean of 28.50 kg/m^2. Further, the lowest BMI for those with mild OSA was 21 kg/m^2. There were no significant gender differences for OSA severity or PA. Age was also unrelated to OSA severity and PA.

OSA severity and physical activity. Correlation revealed that PA was significantly and negatively associated with OSA severity, r(54) = -0.29, p = 0.04, such that those who engaged in less physical activity had greater OSA severity. A follow-up ANOVA between

the four OSA severity groups (no, mild, moderate, severe) revealed no significant overall main effect of group, $F(3,53) = 1.98$, $p = 0.13$, however, pairwise post-hoc comparisons demonstrated significantly lower levels of PA in those with moderate OSA compared to those without OSA, $p = 0.037$ (Figure 1). There were no other significant group differences for PA.

OSA severity, physical activity and BMI. The correlation between OSA severity and BMI was significant, $r(54) = 0.51$, $p < 0.001$, such that those with higher BMI had higher OSA severity. BMI, as would be expected, was also significantly different between the OSA severity groups, $F(3,50) = 7.7$, $p < 0.001$, $\eta^2 = 0.316$. Post-hoc analysis demonstrated significant differences between the OSA severity groups for BMI (Figure 2). Further, BMI and PA were significantly positively associated, $r(54) = 0.29$, $p = 0.03$. Interestingly, when BMI was added into a regression model predicting OSA severity from PA, the significant negative association between PA and OSA severity ($\beta = -0.29$, $p = 0.04$, $\Delta R^2 = 0.08$) became non-significant, $\beta = -0.15$, $p = 0.23$, $\Delta R^2 = 0.193$ (Figure 3). The PROCESS macro was performed within SPSS to explore whether BMI was mediating the association between PA and OSA severity, however, showed there was no indication of a significant indirect effect, 95% CI [- 0.4233, 0.0022], Effect - 0.1677, $p = 0.069$, suggesting no evidence of mediation.

Discussion

The occupations of the participant sample of manual workers in the present study included healthcare workers, PE teachers and craft/labourers. Previous evidence of the lack of leisure-time PA in manual workers, suggests that low levels of PA in the present study alongside raised BMI may increase the risk of OSA development (Gram et al., 2016; Leino-Arjas et al., 2004). Preliminary results supported the hypothesis that undiagnosed levels of OSA were associated with low levels of PA. This is in keeping with previous studies which found that lower levels of PA are associated with increased severity of OSA (Peppard & Young, 2004;

Verwimp et al., 2013) and in contrast to Tan, Alen, Cheng, Mikkola, Tenhunen, Lyytikainen, Wiklund, Cong, Saarinen, Tarkka, Partinen, and Cheng (2015) who recently found that low levels of PA were not associated with OSA in individuals aged over 50 years old. However, the present study also showed BMI to be a stronger indictor of undiagnosed OSA than PA, implying that BMI may be a higher risk for OSA than low PA. That BMI was the stronger (and only significant) predictor in the model that included PA could be due to two possibilities. First, there was reduced power to find effects given the moderate sample size and the correlation between these variables, or second BMI is a multi-faceted variable, and contains variance not determined by PA, which may contribute through different (non-PA related) mechanisms to OSA severity. In observational research it is difficult to separate these aspects. However, this finding corresponds with Webber et al. (2011) who found evidence of undiagnosed OSA in those with BMI >30 kg/m^2. However, the present study also found levels of currently undiagnosed mild OSA in those with a lower BMI than that classified as obese (>30 kg/m^2) suggesting that BMI may not be the sole trigger for the development of OSA.

Additionally, participants in the present study without OSA reported significantly higher levels of PA than those with moderate OSA. Arguably, there is evidence to suggest that BMI is a risk factor for OSA (Koyama et al., 2012) and previous research has shown that OSA severity increases with body weight (Butner et al., 2013). Additionally, it has been suggested that the only effective method of reducing OSA severity is through weight-loss (Young, Peppard, & Gottlieb, 2002). Interestingly, the capacity to exercise is thought to be associated with age and weight and therefore, those who are older and have higher BMI may find it harder to exercise (Butner et al., 2013). In contrast, it is also logical that those who do not engage in much physical activity might also develop a higher BMI (Hankinson, Daviglus, Bouchard, Carnethon, Lewis, Schreiner, Liu, & Sidney, 2010). The present study partially

confirms the theory of Butner et al. (2013) in that BMI was found to be associated with levels of PA, although the direction of causality between these variables obviously cannot be inferred. It might be assumed that low PA may play a part in the development of OSA, but its role in the development or prevention of OSA is unclear. Previous evidence has demonstrated a lack of clarity in the ability of PA to reduce OSA severity in those aged over 50 years old with or without weight-loss (Schobersberger, 2013; Sengul, Ozalevli, Oztura, Itil, & Baklan, 2011). The question as to whether PA or BMI form the strongest risk factor of OSA severity is difficult to entangle given the expected and observed correlation between the two, and this is illustrated by a recent review, which explored the capacity of PA to reduce OSA independent of BMI (Aiello et al., 2016). The review authors concluded that current evidence is inconclusive and that the benefits of PA to health are worth considering it a modifiable risk factor (Aiello et al., 2016). Encouragingly, there is evidence that PA may well protect or reduce the severity of OSA independently of weight loss, although it is generally agreed that the mechanisms by which this occurs are unclear (Kline, Crowley, Ewing, Burch, Blair, Durstine, Davis, & Youngstedt, 2011; Peppard & Young, 2004; Simpson et al., 2015; Verwimp et al., 2013).

Given the low adherence to the present gold standard treatment of OSA (continuous positive airway pressure), it is necessary to consider alternative or additional methods of management that may alleviate OSA and reduce CVD risk (Butner et al., 2013). Spencer, Heidecker, and Ganz (2016) support this consideration, noting the benefit of PA to reduce risk factors associated with OSA development (including higher BMI) and reduce negative health conditions as a consequence of OSA. Thus, it is possible that PA is associated with the development of OSA and is at least a factor to be explored with regard to promoting weight-loss and improving overall health in the prevention and management of OSA (Aiello et al., 2016; Katzmarzyk & Lear, 2012).

It is possible that other health behaviours such as diet, smoking, and alcohol consumption that were not included in the present study may have had a bearing on the results. However, Franklin and Lindberg (2015) suggest that smoking is not an established risk factor of OSA and the role of alcohol is unclear. Thus, with the exception of BMI, there was little evidence to suggest that confounding variables were an important consideration in measuring the association of OSA and PA in the participant group.

The present study is not without limitations. First, there is the obvious issue of reverse causation, making it difficult to infer whether low PA predicts OSA or vice versa, similarly with the link between BMI and PA, however, given that PA engagement is self-selected, only PA intervention randomized trials could start to shed light on the potential causal direction of effects, which is a future direction for this research. Second, the sample size is relatively small, making it possible that the association between PA and OSA severity was attenuated by BMI due to low power and reduced degrees of freedom. Indeed, the lack of evidence of mediation suggests this, and in fact makes it probable that both PA and BMI are independent contributors to OSA severity, although this would need to be confirmed in a larger study. Third, the majority of the participants demonstrated moderate levels of PA and mostly none or mild levels of OSA severity. Thus, it is possible that there were not sufficient participants with moderate to severe OSA in the group to provide a clearer outcome of the association between PA and OSA. However, a strength of the study is that all participants were in manual occupations (classed as manual or involving a substantial physical component e.g. PE teaching) and all were older than 50 years old, which is a neglected group; given the growing age of the workforce, older workers' health is becoming of paramount importance (Billett et al., 2011). Fourth, self-report measures of PA are limited due to risk of over-reporting and recall bias. However, the instrument used to assess PA in the present study is validated to be one of the best methods of self-report PA measures and provides

detailed information on three areas of PA. Future research may benefit from using objective measures of PA.

Evidence in respect of the association between undiagnosed OSA severity and PA is limited. Only a few studies have used participants aged over 50 years old and of those, OSA has already been diagnosed or the sample size is very small (Redolfi, 2015; Simpson et al., 2015; Verwimp et al., 2013). Findings from the present study add to current research into OSA and PA and extend it by examining the presence of undiagnosed OSA and levels of PA in older manual workers. The results highlight the potential existence of OSA that may reside in those without a formal OSA diagnosis in older manual workers and its potential for further development if left untreated. Further, it emphasizes the role of PA and BMI in undiagnosed OSA in this participant group. Findings from the present study support previous research illustrating the high incidence of undiagnosed OSA in manual workers and in the UK (Rejon-Parrilla et al., 2014; Webber et al., 2011).

Conclusion

It is clear from the results that undiagnosed OSA exists in the group of older manual workers examined in the present study. Indeed, low levels of PA were found to be associated with increased severity of OSA. It should also be noted that BMI in the present study was a stronger indicator of OSA severity than PA and therefore future intervention guidance may prove confusing. However, given the negative health outcomes associated with OSA and the benefits that regular PA offers to physical health and mental wellbeing, as well as a method of reducing weight and thereby BMI, it is worthwhile considering low PA as a risk factor for OSA in older workers. Further, the outcome of the present study warrants the consideration of PA as a cost effective management treatment of OSA through aiding weight loss and promoting healthy cardiovascular function in this age group.

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