Other investigators have also found that daytime sleepiness was related more strongly to the presence of respiratory symptoms than to a diagnosis of lung disease or the degree of airway obstruction [116].

We saw significant gender differences after correction for age, BMI, smoking status, country of origin, GOLD and MMRC. Men had worse sleep quality measures and spent more time sleeping during the day than women. There is currently only limited information regarding the influence of gender on sleep in COPD. In a previous study using actigraphy no significant gender-related differences in sleep quality were found [98]. Other studies have documented that women appear to report more sleep-related complaints [117]. However, objective and subjective assessments of sleep seem to reflect different aspects and may therefore not be necessarily in agreement [118].

During weekends patients had significant more fragmented sleep and spent significantly more time awake after the first sleep onset than during weekdays. Nonetheless, our results showed that impaired sleep during weekends may be compensated by sleeping longer.

This study showed that nights of lower sleep quality were followed by days of lower levels of physical activity in people with COPD. The robustness of the results was verified by controlling for several confounding factors such as severity of airflow limitation and severity of dyspnoea. The association of impaired sleep with worse daytime performance is consistent with findings showing that it could play a substantial role in daytime symptoms and chronic fatigue [119]. Moreover these findings are consistent with other correlational evidence drawn from older adults suggesting that objectively measured poor sleep was associated with worse daytime physical function, yet none of these studies demonstrates a direct effect of sleep on subsequent physical activity [100, 101]. Although inferring a causal relationship was out of the scope of this study, our finding provided a good illustration of the sequential association as it incorporated a clear chronological order of the predictor (sleep quality measure) and the predicted variable (physical activity). However, the possibility that poor daytime performance may be a causative factor for poor sleep quality should be considered.

The deficit in good sleep, in association with changes of sleep architecture, may make sleep not as restorative as needed and may cause a significant sleep deficit [120]. Accordingly, our study provides evidence to suggest that poor sleep efficiency and short sleeping bouts during the night are possibly compensated by additional daytime naps.

The fact that poor sleep quality was associated with reduced physical activity levels may have important consequences with regard to current clinical practice. First, our findings suggest the possibility of increasing spontaneous engagement in physical activity through improving sleep since, in the absence of any intervention, patients having had a better night of sleep spontaneously engaged in more physical activity the following day. Second, existing strategies for promoting physical activity tend to focus on actions during the day. Additional efforts in promoting quality sleep among physically inactive subgroups may increase the overall impact of these interventions. We may speculate that poor sleep quality could make the motivation to exercise by day worse and contribute to the vicious cycle of deconditioning that affects COPD sufferers (see Figure 36). Finally, sleep should be assessed and taken into account when analysing physical activity in COPD, although recent literature is often-overlooking the continuity between night-time sleep and daytime physical activity [121].

This study has some limitations which should be considered when interpreting the results. Our participants were not screened for sleep-related disorders, such as obstructive sleep apnoea (OSA). However, because COPD and OSA are both common chronic conditions, these should be