

## Research: Epidemiology

# Obstructive sleep apnoea in Type 2 diabetes mellitus: increased risk for overweight as well as obese people included in a national primary care database analysis

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### Abstract

**Aims** To determine obstructive sleep apnoea prevalence in people with Type 2 or Type 1 diabetes in a national primary care setting, stratified by BMI category, and to explore the relationship between patient characteristics and obstructive sleep apnoea.

**Methods** Using the Royal College of General Practitioners Research and Surveillance Centre database, a cross-sectional analysis was conducted. Diabetes type was identified using a seven-step algorithm and was grouped by Type 2 diabetes, Type 1 diabetes and no diabetes. The clinical characteristics of these groups were analysed, BMI-stratified obstructive sleep apnoea prevalence rates were calculated, and a multilevel logistic regression analysis was completed on the Type 2 diabetes group.

**Results** Analysis of 1 275 461 adult records in the Royal College of General Practitioners Research and Surveillance Centre network showed that obstructive sleep apnoea was prevalent in 0.7%. In people with Type 2 diabetes, obstructive sleep apnoea prevalence increased with each increasing BMI category, from 0.5% in those of normal weight to 9.6% in those in the highest obesity class. By comparison, obstructive sleep apnoea prevalence rates for these BMI categories in Type 1 diabetes were 0.3% and 4.3%, and in those without diabetes 1.2% and 3.9%, respectively. Obstructive sleep apnoea was more prevalent in men than women in both diabetes types. When known risk factors were adjusted for, there were increased odds ratios for obstructive sleep apnoea in people with Type 2 diabetes in the overweight and higher BMI categories.

**Conclusions** Obstructive sleep apnoea was reported in people with both types of diabetes across the range of overweight categories and not simply in the highest obesity class.

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### Introduction

Obstructive sleep apnoea (OSA) is associated with higher morbidity and mortality rates among people with diabetes [1–3]. It is also associated with hypertension, heart failure, pulmonary arterial hypertension, acute cardiovascular events and cardiac arrhythmias [1]. Its presence in people with diabetes already at increased risk of micro- and macrovascular complications [4] is likely to place them at even greater cardiovascular risk [5,6].

Obesity is a well-established risk factor for OSA [7], and may link OSA and Type 2 diabetes mellitus through systemic inflammation [5,6]. In people with Type 1 diabetes but without obesity, the link may be through the development of autonomic neuropathy [8].

OSA data in Type 2 diabetes have usually been reported from specialist clinic observations, with a limited number of reports having a primary care perspective. OSA has been reported in randomized controlled clinical trials, e.g. Sleep AHEAD [9], and also in secondary care specialist clinics, e.g. Amin *et al.* [10]. The few studies that have assessed OSA in a primary care setting include a UK-based study which showed that 4% of men with Type 2 diabetes had pre-existing diagnosed OSA, but 57% were at high risk of OSA [11]. Of those at high risk, a subset was studied overnight using

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**What's new?**

- Links among Type 2 diabetes mellitus, obesity and obstructive sleep apnoea (OSA) have been established in secondary care clinic studies, but primary care population data are limited.
- From a national primary care network, we ascertained that people with Type 2 diabetes had an increased OSA prevalence in every excessive weight category, from overweight to obesity class 3. People with Type 1 diabetes had similar OSA prevalence rates as those without diabetes.
- Strategies to diagnose and manage OSA need to consider its increased prevalence in people with Type 2 diabetes who are overweight as well as those with obesity.

oximetry, and 22% of this group were found to have OSA [11]. Another primary care study in the USA found the prevalence of OSA to be 18% [12]. That study also found that men were two times more likely to have OSA than women [12].

The relationship between OSA and Type 1 diabetes has received less attention than Type 2 diabetes, but a meta-analysis of four studies found a significant correlation between OSA and Type 1 diabetes [13]. OSA at any level was found in 51.9% of 186 people with Type 1 diabetes (mean BMI range 22.9–25.8 kg/m<sup>2</sup>), with moderate to severe OSA in 16.7% of this population [13]. This reinforces the argument that processes independent of BMI contribute to the high OSA rates seen in Type 1 diabetes.

The most recent UK primary care study in this area was published more than a decade ago [11]. As obesity and Type 2 diabetes prevalence rates have increased [4,14] during this period, it is timely to reassess prevalence rates. The aims of the present study were: (1) to determine the prevalence of OSA in people with Type 2 and Type 1 diabetes in a national primary care setting; (2) to stratify these prevalence rates according to BMI categories; and (3) to explore the relationship between gender and OSA in people with Type 2 and Type 1 diabetes.

**Methods**

A cross-sectional analysis of the Royal College of General Practitioners (RCGP) Research and Surveillance Centre (RSC) network was conducted, identifying people with diabetes as previously described [15]. Only those alive and registered with a primary care practice on 31 December 2016 were included. In brief, the RCGP RSC is a nationally representative primary care sentinel network, which consists of data from more than 200 primary care practices across England, including more than 2 000 000 people [16,17].

Diabetes was identified using Read codes (5-byte version 2), which comprise all commonly recorded major diagnostic codes, investigation results and medication codes [15,18]. Types of diabetes were categorized through a seven-step algorithm, which included insulin use, age at insulin initiation, oral hypoglycaemic agent use and BMI [15]. This categorization resulted in three groups for analysis: people with Type 2 diabetes, people with Type 1 diabetes and those with no diabetes. OSA was identified using the specific Read code Fy03. OSA and diabetes data were stratified by BMI category [19].

We reported the clinical characteristics of each group according to age, sex, ethnicity, BMI, socio-economic status (using the Index of Multiple Deprivation [20]), smoking status, alcohol use and blood pressure. Age was calculated as of 31 December 2016, while BMI, smoking status, alcohol use and blood pressure were taken from the latest code entered into the patients' record. Missing data were included in the analysis; multiple imputation and a sensitivity analysis were used to account for missing data in regression analysis.

We then compared the prevalence of OSA in each group according to BMI category/obesity class. The categories/classes used were as follows: underweight (<18.5 kg/m<sup>2</sup>), normal (18.5–24.9 kg/m<sup>2</sup>) and overweight (25.0–29.9 kg/m<sup>2</sup>), and obesity class 1 (30.0–34.9 kg/m<sup>2</sup>), class 2 (35.0–39.9 kg/m<sup>2</sup>) and class 3 (≥40.0 kg/m<sup>2</sup>) [19]. The relationship between gender and OSA was explored in Type 2 and Type 1 diabetes, and logistic regression was used to identify other potential predictors of OSA in the Type 2 diabetes group.

**Statistical analyses**

Descriptive statistics (means and SD values for continuous variables; proportions for categorical data) were used to characterize the three groups. When investigating differences between groups, 95% CIs were calculated.

Multilevel logistic regression was performed (using clustering to account for variability at the practice level) to identify predictors of OSA in the Type 2 diabetes group. Missing data were imputed using multiple imputation with chained equations (predictive mean matching and 10 imputed datasets). Sensitivity analysis was also used to account for any substantial amount of missing data in predictor variables. The following variables were adjusted for in the model: age, gender, socio-economic status, duration of diabetes, BMI, smoking status, alcohol use, and asthma. Odds ratios with 95% CIs and *P* values were calculated for each model variable.

**Results**

A total of 1 275 461 adult records from the RCGP RSC database were analysed. Of these, 1 179 730 (92.5%) people did not have diabetes, 84 394 (6.6%) had Type 2

**Table 1** Characteristics of the adult general practice population with and without diabetes in the Royal College of General Practitioners Research and Surveillance Centre database

Characteristic	Type 2 diabetes N = 84 394	Type 1 diabetes N = 5443	No diabetes N = 1 179 730
Mean (SD) age, years	66.5 (13.7)	42.6 (15.9)	46.2 (18.5)
Median (IQR) age, years	68.0 (20.0)	41.0 (24.0)	44.0 (29.0)
Men, n (%)	46 894 (55.6)	2984 (54.8)	573 620 (48.6)
Mean (SD) BMI, kg/m <sup>2</sup>	30.8 (6.5)	26.8 (5.5)	26.1 (5.6)
Ethnicity, n (%)			
White	58 641 (69.5)	4020 (73.9)	762 809 (64.7)
Asian	7445 (8.8)	198 (3.6)	70 228 (6.0)
Black	3607 (4.3)	107 (2.0)	35 815 (3.0)
Mixed	698 (0.8)	63 (1.2)	14 090 (1.2)
Other	685 (0.8)	32 (0.6)	12 866 (1.1)
Recorded data, %	84.2	81.2	75.9
IMD, n (%)			
Quintile 5 (least deprived)	19 738 (23.4)	1390 (25.5)	314 772 (26.7)
Quintile 4	17 892 (21.2)	1162 (21.3)	257 139 (21.8)
Quintile 3	15 894 (18.8)	1022 (18.8)	214 548 (18.2)
Quintile 2	14 658 (17.4)	934 (17.2)	199 908 (16.9)
Quintile 1 (most deprived)	16 029 (19.0)	915 (16.8)	190 174 (16.1)
Recorded data, %	99.8	99.6	99.7
Smoking status, n (%)			
Never	21 984 (26.0)	2170 (39.9)	487 953 (41.4)
Current	12 571 (14.9)	1050 (19.3)	227 986 (19.3)
Ex-smoker	47 405 (56.2)	2067 (38.0)	389 906 (33.1)
Recorded data, %	97.1	97.1	93.7
Alcohol use, n (%)			
None	22 012 (26.1)	1015 (18.6)	181 102 (15.4)
Within limits	24 965 (29.6)	1981 (36.4)	437 003 (37.0)
Excess	35 114 (41.6)	2041 (37.5)	315 694 (26.8)
Recorded data, %	97.3	92.5	79.2
Blood pressure measured, n (%)	84 166 (99.7)	5383 (98.9)	1 003 506 (85.1)
Mean (SD) SBP, mmHg	131.9 (14.6)	126.5 (14.6)	126.1 (15.5)
Mean (SD) DBP, mmHg	75.0 (9.6)	74.0 (9.1)	76.1 (9.8)

DBP, diastolic blood pressure; IMD, index of multiple deprivation; IQR, interquartile range; SBP, systolic blood pressure.

**Table 2** BMI category of the adult general practice population with and without diabetes in the Royal College of General Practitioners Research and Surveillance Centre database

Characteristic	Type 2 diabetes N = 84 394	Type 1 diabetes N = 5443	No diabetes N = 1 179 730
BMI category, n (%)			
Underweight	574 (0.7)	109 (2.0)	41 533 (3.5)
Normal	13 346 (15.8)	2085 (38.3)	433 935 (36.8)
Overweight	28 275 (33.5)	1877 (34.5)	328 947 (27.9)
Obesity class 1	22 780 (27.0)	866 (15.9)	134 809 (11.4)
Obesity class 2	11 089 (13.1)	266 (4.9)	44 908 (3.8)
Obesity class 3	7162 (8.5)	117 (2.1)	22 919 (1.9)
Recorded data, %	98.6	97.7	85.4

BMI categories: underweight, <18.5 kg/m<sup>2</sup>; normal, 18.5–24.9 kg/m<sup>2</sup>; overweight, 25.0–29.9 kg/m<sup>2</sup>. Obesity classes: class 1, 30.0–34.9 kg/m<sup>2</sup>; class 2, 35.0–39.9 kg/m<sup>2</sup>; class 3, ≥40.0 kg/m<sup>2</sup>.

diabetes and 5443 (0.4%) had Type 1 diabetes (Table 1). The remaining people had other types of diabetes and were not included in this analysis. Across the three groups, the majority of people were white and the largest ethnic minority was Asian. People with Type 2 diabetes were older than those with Type 1 diabetes or without diabetes. More people with Type 2 diabetes were ex-smokers or had obesity compared with the other two groups (Tables 1 and 2).

Within the total adult RCGP RSC population, OSA was present in 8443 people, resulting in a prevalence of 0.7%. While the prevalence of OSA was ≤0.5% for people of normal BMI or who were underweight in each of the three groups, this increased with each increasing BMI category, even in those who were overweight (25.0–29.9 kg/m<sup>2</sup>; Table 3). Within the overweight category, OSA prevalence was highest in those with Type 2 diabetes at 1.2%, compared with the two other groups (Type 1 diabetes: 0.7%, no

**Table 3** Prevalence of OSA according to BMI category and obesity class

	OSA prevalence, % (95% CI)		
	Type 2 diabetes N = 84 394	Type 1 diabetes N = 5443	No diabetes N = 1 179 730
BMI category			
Underweight	0.2 (0.00–0.52)	0.0 (0.00–0.00)	0.1 (0.09–0.16)
Normal	0.5 (0.35–0.58)	0.3 (0.10–0.62)	0.1 (0.14–0.16)
Overweight	1.2 (1.08–1.33)	0.7 (0.37–1.17)	0.5 (0.47–0.51)
Obesity (all classes)	4.7 (4.48–4.89)	1.6 (0.96–2.32)	1.7 (1.62–1.73)
Obesity class			
1	2.8 (2.59–3.02)	1.4 (0.69–2.19)	1.2 (1.13–1.25)
2	5.3 (4.93–5.77)	1.1 (0.00–2.63)	2.0 (1.85–2.10)
3	9.6 (8.95–10.32)	4.3 (0.85–8.55)	3.9 (3.66–4.16)

OSA, obstructive sleep apnoea.

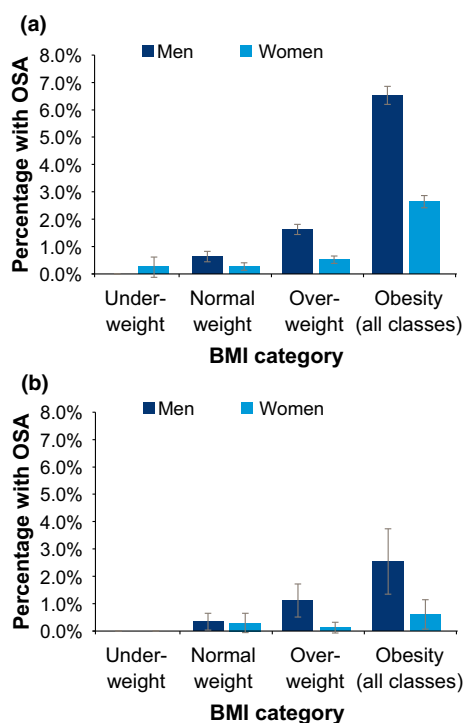
BMI categories: underweight, <18.5 kg/m<sup>2</sup>; normal, 18.5–24.9 kg/m<sup>2</sup>; overweight, 25.0–29.9 kg/m<sup>2</sup>. Obesity classes: class 1, 30.0–34.9 kg/m<sup>2</sup>; class 2, 35.0–39.9 kg/m<sup>2</sup>; class 3, ≥40.0 kg/m<sup>2</sup>.

diabetes: 0.5%; Table 3). A similar pattern was evident when all obesity classes were combined, with the highest OSA prevalence of 4.7% in people with Type 2 diabetes (Table 3).

When obesity was analysed by class, we found that OSA increased as class increased for the groups no diabetes and Type 2 diabetes. People with Type 2 diabetes in obesity class 3 (≥40 kg/m<sup>2</sup>) had the highest OSA rate (9.6%) of any group analysed in this study (Table 3).

Analysing absolute numbers of people with Type 2 diabetes, it was apparent that more men than women had OSA in the overweight and obese categories (Fig. 1a). This difference was largest in the obese categories [men vs women: 6.5% (95% CI 6.20–6.86) vs 2.6% (95% CI 2.42–2.87)]. A similar trend was noted in people with Type 1 diabetes (Fig. 1b).

Multilevel regression analysis of the group with Type 2 diabetes showed that OSA was more likely in those aged 35–54 and 55–74 years vs those aged 18–34 years (Table 4). Even when the data were adjusted for multiple variables, men with Type 2 diabetes were still more likely to have OSA than women with Type 2 diabetes. This was also the case for increasing BMI, with people within obesity class 3 more likely to have OSA than those of normal weight; however, again, a higher odds ratio was apparent in people within the overweight category. Smoking and asthma were also associated with an increased OSA odds ratio in the adjusted model, whilst people with diabetes for ≥10 years had greater odds of OSA than those with a duration of diabetes of between 5 and 10 years. (Table 4). Socio-economic status and alcohol consumption were not associated with OSA.



**FIGURE 1** Prevalence of obstructive sleep apnoea (OSA) according to BMI category and gender in people with (a) Type 2 diabetes and (b) Type 1 diabetes. BMI categories: underweight, <18.5 kg/m<sup>2</sup>; normal, 18.5–24.9 kg/m<sup>2</sup>; overweight, 25.0–29.9 kg/m<sup>2</sup>. Obesity (all classes), ≥30.0 kg/m<sup>2</sup>. Error bars represent the 95% CI.

## Discussion

Approximately 1% of the adult RCGP RSC population was diagnosed with OSA. It is of interest that higher OSA prevalence rates were recorded in people who were in the lowest overweight category compared with those of normal weight, across the Type 2 diabetes, Type 1 diabetes and no diabetes groups; however, as expected, the prevalence of OSA continued to increase as BMI increased into the obesity categories. The prevalence of OSA was higher in people with Type 2 diabetes compared with Type 1 diabetes, but rates of OSA were similar between the Type 1 diabetes and no diabetes groups across all weight categories. Men with Type 2 diabetes had a higher OSA prevalence than women across all grades of overweight and obesity, which was similar to findings reported in previous studies [10,12,21].

**Table 4** Obstructive sleep apnoea in Type 2 diabetes mellitus: results of a multilevel logistic regression model, clustered at the practice level

Characteristic	OR (95% CI)	P
Age		
18–34 years	1.00 (reference)	
35–54 years	1.68 (1.17–2.58)	0.011
55–74 years	1.71 (1.23–2.70)	0.008
≥75 years	0.85 (0.61–1.39)	0.438
Gender		
Female	1.00 (reference)	
Male	3.07 (2.76–3.38)	<0.001
IMD quintile		
Quintile 5 (least deprived)	1.00 (reference)	
Quintile 4	1.00 (0.68–1.48)	0.970
Quintile 3	1.01 (0.88–1.15)	0.911
Quintile 2	0.97 (0.67–1.30)	0.706
Quintile 1 (most deprived)	0.94 (0.80–1.10)	0.419
Duration of diabetes		
<1 year	0.92 (0.77–1.09)	0.333
1–5 years	1.00 (reference)	
5–10 years	1.09 (0.97–1.21)	0.154
≥10 years	1.17 (1.05–1.30)	0.006
BMI category		
Underweight	0.51 (0.07–3.57)	0.499
Normal	1.00 (reference)	
Overweight	2.22 (1.71–2.93)	<0.001
Obesity class 1	5.16 (3.99–6.74)	<0.001
Obesity class 2	10.52 (8.12–13.78)	<0.001
Obesity class 3	21.80 (16.71–28.39)	<0.001
Smoking status		
Non-smoker	1.00 (reference)	
Current	1.24 (1.06–1.42)	0.003
Ex-smoker	1.24 (1.10–1.38)	<0.001
Alcohol consumption		
Alcoholism	1.00 (reference)	
Hazardous	1.08 (0.90–1.30)	0.872
Safe	1.00 (0.83–1.22)	0.344
Non-drinker	1.00 (0.82–1.23)	0.834
Other risk factors		
Asthma	1.00 (reference)	
No asthma	0.59 (0.53–0.64)	<0.001

OR, odds ratio. BMI categories: underweight, <18.5 kg/m<sup>2</sup>; normal, 18.5–24.9 kg/m<sup>2</sup>; overweight, 25.0–29.9 kg/m<sup>2</sup>. Obesity classes: class 1, 30.0–34.9 kg/m<sup>2</sup>; class 2, 35.0–39.9 kg/m<sup>2</sup>; class 3, ≥40.0 kg/m<sup>2</sup>.

The highest OSA prevalence measured in the present study (9.6% in people with Type 2 diabetes and in obesity class 3, ≥40.0 kg/m<sup>2</sup>) was below previously published values in people with Type 2 diabetes. In an analysis of a large US primary care database, Heffner *et al.* [12] reported OSA in 18% of all people with Type 2 diabetes, and in 23% of people with obesity and Type 2 diabetes, while the Sleep AHEAD study reported a prevalence of 86.6% in people with obesity and Type 2 diabetes in a trial population who underwent polysomnography testing [9]. It is possible that the differences in clinical practice between the USA and UK led to better detection rates of OSA in the US study, and therefore apparently higher prevalence rates. Another possibility is that these differences may reflect the study design, with all patients in the Sleep AHEAD study undergoing diagnostic tests for OSA, but other studies, including the

present RCGP RSC study, examining large databases at specific time points. The values reported here are more similar to the 2–8% reported for the general population [21]. Not only were the OSA prevalence rates of the Type 2 diabetes group lower than those reported in other populations, but so was its prevalence within those with Type 1 diabetes [13]. The reasons for these apparent discrepancies between studies include the study population and diagnostic criteria, with clinical trials and studies of people recruited from secondary care centres being likely to record higher prevalence rates than primary care studies [12]; however, the pathophysiological mechanisms underlying the links between OSA and these two types of diabetes may also impact on these prevalence rates.

Obstructive sleep apnoea is a multifactorial disease, with variation in its pathophysiology highly likely among individuals [22]. OSA occurs when the upper airway collapses repeatedly during sleep, resulting in disrupted sleep and oxygen desaturation [3,22]. The precise basis of the association between diabetes and OSA is unknown and could be driven by mechanical (secondary to obesity), metabolic (hyperglycaemia-induced neuropathy) and/or inflammatory processes [5,6]. OSA, obesity and Type 2 diabetes are probably linked by increased systemic inflammation [5,6], while Type 1 diabetes is linked to OSA through autonomic neuropathy [8]. Aurora and Punjabi [6] suggested the inflammatory effects of OSA may result in glucose metabolism dysfunction and eventually lead to Type 2 diabetes. This is not the case in every individual, as OSA can develop secondarily to or worsen with the increased metabolic dysfunction associated with Type 2 diabetes [6]. People with Type 2 diabetes may develop neuropathy as a diabetes-related complication, which can also put them at risk of OSA [6]. The links among insulin resistance, glucose intolerance, obesity, mechanical changes to the functioning of anatomical structures, Type 2 diabetes and OSA make it difficult to establish which of these events happens first and how directly causal any one factor is of another [5,6].

A possible reason for the large difference between previously reported OSA prevalence rates in people with Type 1 diabetes (~52%) [13] and the present study (4.3% in obesity class 3) could be that general practitioners within the UK do not consider a diagnosis of OSA in people of normal weight. As the 52% prevalence rate was estimated within a meta-analysis, which used data from studies of selected populations [13], it may have been an overestimate. Nevertheless, there is a large difference between these two values. OSA is known as an underdiagnosed condition [9], and as people with Type 1 diabetes are less likely to be overweight, physicians may not consider the possible presence of OSA; however, an alternative reason for this underdiagnosis could be related to the mechanisms underlying the pathophysiology of OSA in people with Type 1 diabetes vs those with Type 2 diabetes. If people with Type 1 diabetes have rates of OSA similar to those without diabetes (vs those with Type 2



diabetes), it implies that perhaps aberrant glucose levels are only loosely correlated to the presence of OSA. Further analyses of populations containing people with Type 1 diabetes and Type 2 diabetes are needed to determine the exact role of abnormal glucose control in the aetiology of this complex sleeping condition.

The main limitation of the present study is that the precise methods used by general practitioners to diagnose OSA within the RCGP RSC database remain unknown. Self-reporting of OSA, rather than systematic completion of sleep questionnaires followed by polysomnographic studies, is likely to yield very different prevalence rates. Alternatively, some people included in the database were probably diagnosed with OSA after a specialist visit, through the use of hospital letters. Within the RCGP RSC database a variety of diagnostic approaches are likely to have been taken, which may have led to an under-representation of the true prevalence of the condition. In addition, Read codes do not allow researchers to distinguish between absence of a condition (OSA in this case) and missing information on presence of a condition, which may contribute to this underestimate. For asthma, a sensitivity analysis was used to account for this lack of distinction, and the prevalence rate of ~17% indicated high sensitivity and specificity (estimates at ~0.9) and closely agreed with the output from the multilevel model. For the remaining variables, the largest missing data category was <3%, so bias attributable to missing data was likely to be small. Another limitation is the low number of people with Type 1 diabetes which precluded regression analysis in this group. In addition, whether people were receiving treatment for OSA was not determined.

To help people with OSA, the condition first needs to be diagnosed correctly. Prior to participating in a sleep study or undergoing overnight polysomnography for a full diagnosis, individuals presenting with sleep problems should provide their sleep history as part of a routine examination, then undergo evaluation of symptoms specific to OSA [23]. The results of the present study suggest that people with obesity, and particularly those with obesity and Type 2 diabetes, should be considered for OSA screening at regular intervals, as they are more likely to have the condition than people without these diseases. The Epworth Sleepiness Scale provides a measure of daytime sleepiness and may indicate that OSA is present if a person has a score  $\geq 10$  [24]. Other studies investigating the risk of OSA use the easy-to-apply Berlin questionnaire or another type of questionnaire [25,26]. These relatively straightforward methods should be simple to use within the primary care setting, as a first step to diagnosing OSA. Given the high prevalence of OSA reported in some studies of people with Type 1 diabetes [13] and its relative under-detection in routine clinical practice, a case could also be made for application of such questionnaires in the care of those with Type 1 diabetes, irrespective of their BMI [8].

Once a diagnosis of OSA has been made in a person who is overweight or obese, strategies need to be put in place to help with weight loss [27]. Our data showed that the majority of the Type 2 diabetes group were in the overweight or obese categories. Low-calorie diets and lifestyle modification programmes that focus on increasing exercise and eating healthily have proven successful, but not for every person [24,27,28]. Pharmacotherapies for weight loss as adjuncts to lifestyle alterations have also reduced OSA symptoms [29]. Newer classes of diabetes medications, including sodium-glucose co-transporter-2 inhibitors and glucagon-like peptide-1 receptor agonists, do offer a weight reduction benefit in addition to their glucose-lowering properties [30]. Other treatment methods for OSA include mandibular advancement splints, continuous positive airway pressure and bariatric surgery [24]. Any programme designed to address OSA also needs to take into account the increased prevalence of OSA in people with Type 2 diabetes.

In conclusion, with increased prevalence in all classes of obesity within diabetes likely contributing to increased OSA prevalence [6], healthcare professionals need to be aware of the increased risk of OSA in people within all overweight categories and not just those in the high obesity range. Further work is needed to fully understand the aetiology and underlying mechanisms of OSA.

Management options need to include weight-reduction treatments for a large proportion of the population with diabetes. Men and those with Type 2 diabetes, who are at high risk of OSA, need to be actively questioned about the possibility of OSA and then managed according to local guidelines, as appropriate.

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#### Competing interests

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### Data accessibility statement

The RCGP RSC data set can be accessed by researchers on a case-by-case basis, the application process can be found at [www.rcgp.org.uk/rsc](http://www.rcgp.org.uk/rsc). Depending on the nature of the application further research ethics approval application may be required.

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