



# Spousal cardiometabolic risk factors and incidence of type 2 diabetes: a prospective analysis from the English Longitudinal Study of Ageing

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## Abstract

**Aims/hypothesis** In the UK, more than one million people have undiagnosed diabetes and an additional five million are at high risk of developing the disease. Given that early identification of these people is key for both primary and secondary prevention, new screening approaches are needed. Since spouses resemble each other in cardiometabolic risk factors related to type 2 diabetes, we aimed to investigate whether diabetes and cardiometabolic risk factors in one spouse can be used as an indicator of incident type 2 diabetes in the other spouse.

**Methods** We analysed data from 3649 men and 3478 women from the English Longitudinal Study of Ageing with information on their own and their spouse's diabetes status and cardiometabolic risk factors. We modelled incidence rates and incidence rate ratios with Poisson regression, using spousal diabetes status or cardiometabolic risk factors (i.e. BMI, waist circumference, systolic and diastolic BP, HDL- and LDL-cholesterol and triacylglycerols) as exposures and type 2 diabetes incidence in the index individual as the outcome. Models were adjusted for two nested sets of covariates.

**Results** Spousal BMI and waist circumference were associated with incident type 2 diabetes, but with different patterns for men and women. A man's risk of type 2 diabetes increased more steeply with his wife's obesity level, and the association remained statistically significant even after adjustment for the man's own obesity level. Having a wife with a 5 kg/m<sup>2</sup> higher BMI (30 kg/m<sup>2</sup> vs 25 kg/m<sup>2</sup>) was associated with a 21% (95% CI 11%, 33%) increased risk of type 2 diabetes. In contrast, the association between incident type 2 diabetes in a woman and her husband's BMI was attenuated after adjusting for the woman's own obesity level. Findings for waist circumference were similar to those for BMI. Regarding other risk factors, we found a statistically significant association only between the risk of type 2 diabetes in women and their husbands' triacylglycerol levels.

**Conclusions/interpretation** The main finding of this study is the sex-specific effect of spousal obesity on the risk of type 2 diabetes. Having an obese spouse increases an individual's risk of type 2 diabetes over and above the effect of the individual's own obesity level among men, but not among women. Our results suggest that a couples-focused approach may be beneficial for the early detection of type 2 diabetes and individuals at high risk of developing type 2 diabetes, especially in men, who are less likely than women to attend health checks.

**Data availability** Data were accessed via the UK Data Service under the data-sharing agreement no. 91400 (<https://discover.ukdataservice.ac.uk/catalogue/?sn=5050&type=Data%20catalogue>).

**Keywords** Cardiometabolic risk factors · Obesity · Primary prevention · Screening · Secondary prevention · Spouse · Type 2 diabetes · Undiagnosed diabetes

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Jannie Nielsen and Adam Hulman contributed equally to this study.

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## Research in context

### What is already known about this subject?

- Early identification of people at high risk of developing, or with undiagnosed, type 2 diabetes is key for primary and secondary prevention. However, in the UK more than one million people have undiagnosed diabetes and five million are at high risk of developing the disease
- Current screening programmes have non-response rates of 25–70% or higher, and non-participation tends to be greater in people with the highest levels of background risk
- Spouses resemble each other in their cardiometabolic risk factors

### What is the key question?

- Can the risk of incident type 2 diabetes in an individual be predicted based on the cardiometabolic risk factors of their spouse?

### What are the new findings?

- Obesity levels in one spouse are associated with incident type 2 diabetes in the other spouse
- For men, the risk of type 2 diabetes as an outcome of his wife's obesity level is over and above that associated with his own obesity level

### How might this impact on clinical practice in the foreseeable future?

- Our results suggest that health practitioners may improve the early detection of diabetes risk by using a couples-based approach, rather than an exclusively individual-based approach

## Abbreviations

DBP	Diastolic BP
ELSA	English Longitudinal Study of Ageing
IR	Incidence rate
IRR	Incidence rate ratio
SBP	Systolic BP
SES	Socioeconomic status

## Introduction

Early identification of individuals at high risk of developing type 2 diabetes or with undiagnosed type 2 diabetes is key for the primary and secondary prevention of type 2 diabetes and related morbidity. Randomised controlled trials have provided proof of concept that type 2 diabetes can be prevented or postponed in individuals with impaired glucose tolerance through a healthy diet, increased physical activity and weight loss [1, 2]. In individuals with recently diagnosed diabetes, early glycaemic control [3] and attention to cardiovascular risk factors [4, 5] can reduce diabetes-related complications and comorbidities, such as retinopathy, neuropathy and cardiovascular disease. In the UK, approximately 1.1 million people are thought to have undiagnosed diabetes, and a further five

million impaired glucose regulation [6, 7]. However, a relatively low proportion (7.5%) of those with impaired glucose regulation are aware of their risk status [8].

Current screening recommendations for type 2 diabetes in the UK focus predominantly on an individual-level risk assessment based on age, sex, ethnicity, weight, hypertension and family history of type 2 diabetes [9], often implemented as part of a more general health check [10]. Diabetes screening programmes recruiting people based on such individual risk levels have non-response rates of 25–70% or higher [10, 11], and non-participation tends to be higher in people at the highest levels of background risk such as men, overweight individuals and people from lower strata of socioeconomic status (SES) [11, 12]. Although the English National Health Service ‘Health Check’ programme has improved overall attendance rates and participation among older people and those from more deprived socioeconomic groups [10, 12], participation is still lower among men compared with women, and in lower socioeconomic groups as compared with higher socioeconomic groups [12]. In Denmark, it has been suggested that responding to and participating in a health check is a powerful marker of healthy self-selection. Thus, 10 year all-cause mortality among participants in a health check was 44% lower/27% lower than in people who were either invited but did not respond or not invited at all, respectively [13].

These findings clearly show the need for alternative approaches for the early identification of people at high risk of type 2 diabetes or with undiagnosed type 2 diabetes who might not be identified by or willing to participate in the current individual-focused screening programmes. Given that studies have shown that spouses resemble each other in cardiometabolic risk factors such as plasma glucose [14], lipids [15], BP [15, 16], measures of obesity [17, 18] and, to some extent, type 2 diabetes [16, 19–21], spousal cardiometabolic risk factor levels could be a novel entry point to identify individuals at risk of type 2 diabetes or with undiagnosed diabetes, especially among those who are unlikely to attend regular screening or health checks. Therefore, the overall aim of this study was to investigate whether an individual's risk of developing type 2 diabetes is affected by the cardiometabolic risk factor levels in their spouse and, thus, if individuals at high risk of developing type 2 diabetes can be identified by their spouse's cardiometabolic risk factor levels.

## Methods

We analysed data from the English Longitudinal Study of Ageing (ELSA) cohort. ELSA has collected economic, social, health and biological data from eight waves of participants between 1999 and 2015. Participants were sampled from 3 years of the Health Survey for England (1998, 1999 and 2001) to form wave 0 in ELSA. Details of the original sampling framework in ELSA have been published previously [22, 23]. Ethical consent was obtained for all waves of ELSA and all participants gave informed consent to take part in the study [22, 23].

For the present study, ELSA wave 0 served as baseline for the main analysis. To be included in the present study, the index individual was required to fulfil two criteria at baseline: (1) to be a core member of ELSA (in ELSA, participants born before 1 March 1952 were considered as core members); and (2) to share a household with a spouse (same ELSA household ID). We defined a spouse as: (1) the opposite-sex person the index individual reported to be their spouse; or (2) a non-sibling, opposite-sex cohabitant with an age difference of less than 18 years living in the same household. According to this definition, out of 11,205 ELSA wave 0 core members, 7909 index individuals lived together with a spouse. We extracted information on our main outcome, type 2 diabetes incidence of the index individual, up to ELSA wave 7 (data collected in 2015). Type 2 diabetes status was either self-reported (waves 0–7) or screen-detected based on fasting plasma glucose ( $\geq 7.0$  mmol/l) or HbA<sub>1c</sub> ( $\geq 48$  mmol/mol [6.5%]) at waves 2, 4 and 6. Participants leaving the cohort (death or non-attendance) were censored at their last visit. Owing to the ELSA inclusion criteria regarding age, incident diabetes was

diagnosed after age 45 years. Therefore, we used the term 'type 2 diabetes'.

Baseline information on age, sex, ethnicity (white/non-white), employment grade as a proxy for SES (the highest reported grade at the couple level was used for both spouses) and spouse's diabetes status were obtained through questionnaires, while body height and weight (used to calculate BMI), waist circumference, and systolic (SBP) and diastolic BP (DBP) were measured during a clinical examination (see electronic supplementary material [ESM] [Methods](#) and ESM Table 1). We excluded index individuals with prevalent diabetes at baseline ( $n = 372$ ; 230 men and 142 women) and those with missing data on age, SES at couple level or race/ethnicity ( $n = 16$ ; eight men and eight women), or with missing information about their spouse's diabetes status or related risk factors ( $n = 394$ ; 111 men and 283 women). Consequently, our final baseline sample included 7127 index individuals (3649 [51%] men and 3478 women) (ESM Fig. 1), of whom 5740 (81%) were included as both an index individual and spouse in the respective sex-stratified analyses. Spouses born after 1 March 1952 did not have follow-up data and thus were not included as index individuals in our analysis. However, these spouses might have contributed exposure data, because we only used spousal risk factor levels from baseline.

For analyses of LDL-cholesterol, HDL-cholesterol and triacylglycerol levels in the spouse as exposures for incident type 2 diabetes in the index individual, we used a subsample consisting of 4300 individuals (2174 men [51%] and 2126 women) as lipid measures were not included in ELSA before wave 2 (year 2004–2005).

**Statistical analysis** We used Spearman's Rank correlation coefficient to test the statistical dependence between the same cardiometabolic risk factor in the two spouses. We used Poisson regression models with log person-years as offset and the follow-up period cut into 1 year age bands to model incidence rates (IRs) and to assess incidence rate ratios (IRRs) of type 2 diabetes in a sex-stratified analysis [24]. Our main exposures of interest were the following cardiometabolic risk factors in the spouse at baseline: diabetes status (type not specified), obesity (BMI and waist circumference), SBP and DBP, and lipid levels (LDL-cholesterol, HDL-cholesterol, triacylglycerols). All exposures, except diabetes, were considered as continuous measures. We explored non-linear associations between the continuous exposures and the outcome by including cubic spline terms. If this did not improve the model fit, we retained only the linear term.

All models were adjusted for age, ethnicity and SES (model 1). As a next step, models with continuous exposures were further adjusted for the index individual's own value of the respective spousal exposure variable (model 2). Models including BP or lipid levels were also adjusted

for BP- and lipid-lowering medication, respectively. To compare spousal cardiometabolic risk factors, we fitted the models after standardising the exposure variables. Statistical significance was inferred at  $p < 0.05$ . All statistical analyses were conducted using R version 3.3.1 (R Foundation for Statistical Computing, Vienna, Austria) and the Epi R package (version 2.24) [25].

## Results

Baseline characteristics of the index individuals stratified by sex are summarised in Table 1. The cohort was predominantly of white race/ethnicity, of middle and high SES, overweight and with a median SBP close to 140 mmHg. In general, men (index individuals) were slightly more obese (median difference 0.6 kg/m<sup>2</sup>;  $p = 0.05$ ) and 2 years older than their wives ( $p < 0.001$ ). Four per cent of the couples were not married.

At baseline, Spearman correlations between the same risk factor in the wife and the husband were: BMI 0.17; waist circumference 0.24; SBP 0.21; DBP 0.12; LDL-cholesterol 0.11; HDL-cholesterol 0.17; TG 0.20 ( $p < 0.001$  for all tests).

In the main analysis, the median follow-up time was 11.5 years (Q1–Q3 5.5–15.0 years). During follow-up, 452 men and 315 women were diagnosed with type 2 diabetes. The incidence of type 2 diabetes was 12.6 per 1000 person-years (95% CI 11.5, 13.8) in men and 8.6 per 1000 person-years (95% CI 7.7, 9.6) in women. The proportion of incident cases based on self-reported diabetes (i.e. participants who reported that their diabetes had been diagnosed by a doctor) was 68%. Having a spouse with diabetes was not statistically significantly associated with an increased risk of type 2 diabetes for either men (IRR 1.02 [95% CI 0.64, 1.65],  $p = 0.92$ ) or women (IRR 1.40 [95% CI 0.95, 2.08],  $p = 0.09$ ).

Associations between obesity in the spouse and the incidence of type 2 diabetes in the index individual are displayed in Figs 1 and 2. The use of spline terms did not improve any of

**Table 1** Baseline characteristics of index individuals stratified by sex

Characteristic	Men	Women
Baseline wave 0 (1998, 1999 and 2001)		
<i>n</i>	3649	3478
Spousal diabetes	134 (3.7)	218 (6.3)
Spousal overweight/obesity <sup>a,b</sup>	2203 (66)	2423 (76)
Age, years	60 (53–68)	59 (53–66)
SES <sup>c</sup>		
Professional/managerial	1529 (42)	907 (26)
Skilled non-manual	328 (9)	1297 (37)
Skilled manual	1187 (33)	298 (9)
Unskilled/semi-skilled manual	573 (16)	898 (26)
Non-white race/ethnicity	83 (2.3)	52 (1.5)
BMI <sup>d</sup> , kg/m <sup>2</sup>	27.2 (25.1–29.7)	26.8 (24.1–30.4)
Waist circumference <sup>e</sup> , cm	99 (92–106)	86 (79–95)
SBP <sup>f</sup> , mmHg	139 (128–152)	138 (125–152)
DBP <sup>f</sup> , mmHg	80 (73–88)	74 (67–82)
Wave 2 (2004–2005) <sup>g</sup>		
<i>n</i>	2174	2126
Age, years	64 (58–71)	62 (57–69)
LDL-cholesterol (mmol/l)	3.5 (2.9–4.1)	3.8 (3.2–4.4)
HDL-cholesterol (mmol/l)	1.3 (1.2–1.6)	1.6 (1.4–1.9)
Triacylglycerol (mmol/l)	1.6 (1.1–2.3)	1.4 (1.1–2.1)

Values are *n* (%) or medians (interquartile range)

<sup>a</sup> Defined as BMI  $\geq 25$  kg/m<sup>2</sup>

<sup>b</sup> Data missing for 322 men and 287 women

<sup>c</sup> Individual-level data missing for 32 men and 78 women

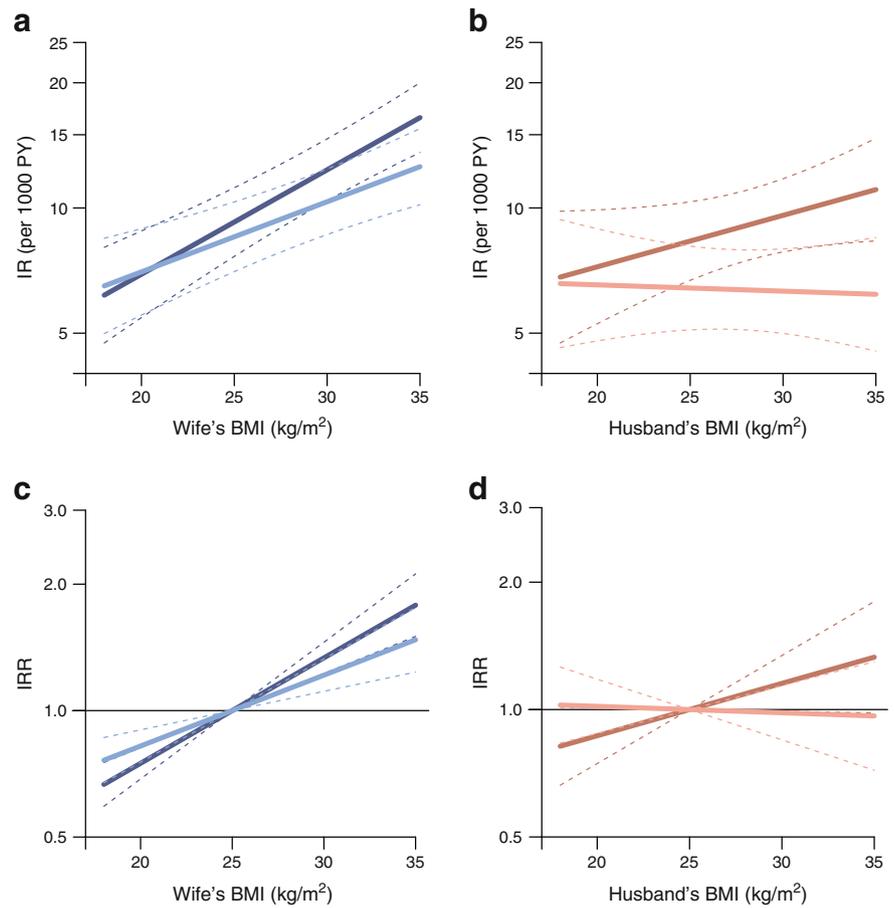
<sup>d</sup> Data missing for 276 men and 286 women

<sup>e</sup> Data missing for 823 men and 758 women

<sup>f</sup> Data missing for 1073 men and 972 women

<sup>g</sup> Wave 2 served as baseline for the blood lipid measures, as these were not included in waves 0 and 1

**Fig. 1** IRs and IRRs of type 2 diabetes (solid lines) with 95% CIs (dashed lines) among men (**a**, **c**) and women (**b**, **d**) by spousal BMI. The vertical axis was  $\log_e$  transformed for each figure. Dark lines: adjusted for age (60 years), SES (skilled) and ethnicity (white); light lines: adjusted for age, SES, ethnicity and the index individual's median risk factor level. PY, person-years



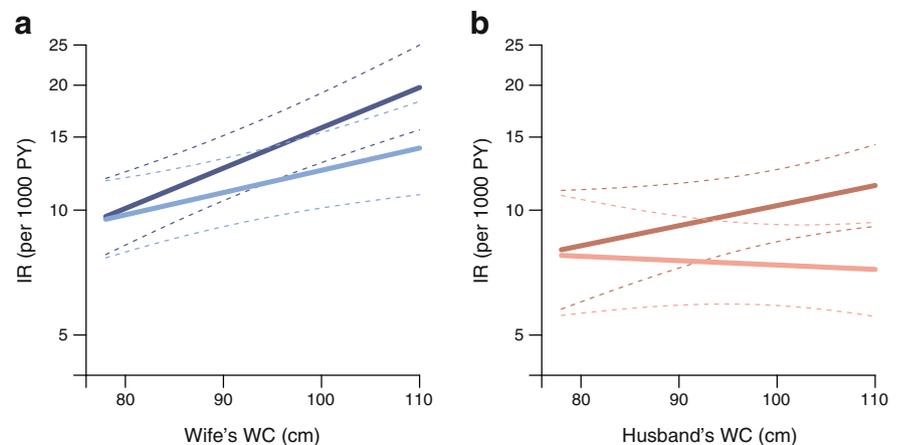
the models; therefore, only linear terms were retained in the models. IRRs are presented in Table 2.

Regarding overweight/obesity, the associations exhibited different patterns for men and women. Among men, the risk of type 2 diabetes increased steeply with their wife's BMI, and the association remained statistically significant even after adjustment for the man's own BMI level (Fig. 1a). For example, the risk of a man developing type 2 diabetes increased by 6% for every 1  $\text{kg}/\text{m}^2$  increase in his wife's BMI (Table 2).

Furthermore, a man whose wife had a BMI of 30  $\text{kg}/\text{m}^2$  had a 21% increased risk (IRR 1.21 [95% CI 1.11, 1.33]) of diabetes compared with a man whose wife's BMI was 25  $\text{kg}/\text{m}^2$ , regardless of the man's own BMI (Fig. 1c). The same 21% risk increase was associated with a 1.6  $\text{kg}/\text{m}^2$  difference in the man's own BMI (equivalent to a 5  $\text{kg}/\text{m}^2$  difference in the wife's BMI).

Among women, there was an initial indication of increased type 2 diabetes risk in relation to the husband's BMI, but the

**Fig. 2** IRs of type 2 diabetes (solid lines) with 95% CIs (dashed lines) among men (**a**) and women (**b**) by spousal waist circumference (WC). The vertical axis was  $\log_e$  transformed for each figure. Dark lines: adjusted for age (60 years), SES (skilled), ethnicity (white); light lines: adjusted for age, SES, ethnicity and the index individual's median risk factor level. PY, person-years



**Table 2** IRRs for developing type 2 diabetes for every unit increase in spousal risk factor

Spousal risk factor	Men			Women		
	<i>n</i> <sup>a</sup>	Model 1	Model 2	<i>n</i> <sup>a</sup>	Model 1	Model 2
BMI index (1 kg/m <sup>2</sup> )	3222	1.06 (1.04, 1.08)	1.04 (1.02, 1.06)	3073	1.03 (1.00, 1.06)	1.00 (0.97, 1.03)
Waist circumference (5 cm)	2732	1.12 (1.07, 1.17)	1.06 (1.02, 1.11)	2600	1.06 (1.00, 1.12)	0.99 (0.93, 1.05)
SBP (10 mmHg)	2357	1.02 (0.96, 1.08)	0.99 (0.93, 1.05)	2226	0.99 (0.92, 1.07)	0.96 (0.89, 1.03)
DBP (10 mmHg)	2357	1.04 (0.94, 1.15)	1.02 (0.92, 1.12)	2226	0.94 (0.83, 1.06)	0.92 (0.81, 1.03)
LDL-cholesterol (1 mmol/l)	1043	1.01 (0.81, 1.26)	1.04 (0.84, 1.30)	1109	0.93 (0.73, 1.18)	0.94 (0.74, 1.20)
HDL-cholesterol (1 mmol/l)	1090	1.24 (0.69, 2.24)	1.37 (0.75, 2.50)	1163	0.55 (0.26, 1.18)	0.74 (0.34, 1.59)
Triacylglycerol (1 mmol/l)	1090	1.06 (0.86, 1.32)	1.03 (0.82, 1.29)	1164	1.28 (1.13, 1.44)	1.26 (1.11, 1.44)

Values are presented as IRRs (95% CI) per unit difference in the analysed spousal risk factor

Model 1: adjusted for age, ethnicity and SES

Model 2: adjusted for age, ethnicity, SES and the index individual's risk factor level

Models including BP or lipids were also adjusted for any related medical treatment (yes/no)

<sup>a</sup> *n* varies across risk factors as not all individuals had all measurements

incidence curve flattened out after adjustment for the woman's own BMI (Fig. 1b). These findings were consistent with the associations between a spouse's waist circumference and the incidence of type 2 diabetes in the index individual (Fig. 2a, b). The standardised analysis comparing the effect of a 1 SD difference in the wife's BMI or waist circumference showed that the magnitude of the effect on the incidence of type 2 diabetes in men was similar for overall and central obesity, both in the model without (IRR 1.24 [95% CI 1.16, 1.32] for BMI; IRR 1.26 [95% CI 1.16, 1.37] for waist circumference) and with (IRR 1.15 [95% CI 1.08, 1.23] for BMI; IRR 1.14 [95% CI 1.04, 1.24] for waist circumference) adjustment for the man's own obesity.

We did not find an association between the spouse's BP, HDL-cholesterol or LDL-cholesterol level and the risk of type 2 diabetes in the index individual for either sex (Table 2, ESM Figs 2, 3). However, we observed an increased risk of type 2 diabetes in women as a function of higher triacylglycerol levels in their husbands (Table 2, ESM Fig. 3f), even after adjustment for the woman's own triacylglycerol level.

## Discussion

The results of our study show that spousal BMI and waist circumference are associated with type 2 diabetes risk in the index individual. For men, the type 2 diabetes risk was only partly explained by the man's own obesity level; for women, the relationship disappeared when the woman's own obesity was accounted for. Except for the triacylglycerol levels in the husband, similar associations with diabetes risk in the index individual were not found for spousal diabetes, BP or cholesterol.

In line with a retrospective study from the Framingham cohort in the USA [20], we did not find that diabetes in the spouse was associated with a clearly increased risk of type 2 diabetes in the index individual. In contrast, a Swedish prospective study including more than 3.5 million families and 3296 incident cases of type 2 diabetes that found a 32% higher risk of developing type 2 diabetes in individuals with a spouse with type 2 diabetes [19]. In addition, a US insurance-based study with more than 200,000 individuals found an 11 times higher risk of diabetes in individuals with a spouse diagnosed with the same disease within the last 3 years [26]. A meta-analysis of five studies, including the Swedish one, found similar results [21]. However, the results were statistically non-significant after excluding the Swedish study, although the point estimate still indicated a 33% higher risk of diabetes. The much lower number of individuals with new type 2 diabetes in the Framingham cohort [20] and in our own study (both  $n < 770$ ) may partly explain why neither was able to replicate the findings from the much larger Swedish and US studies [19, 26]. In addition, however, cross-sectional studies have reported inconsistent findings with respect to the statistical significance of the risk of type 2 diabetes among individuals whose spouses have the disease [15, 16, 27, 28].

The relationship between spousal obesity and the development of type 2 diabetes is supported by two studies from the USA that found that spousal overweight [26] or obesity [20, 26] (defined based on BMI) increased the odds of type 2 diabetes in the index individual. These studies did not look at the association by sex. Our finding that spousal obesity but not spousal type 2 diabetes is a risk factor for the development of type 2 diabetes may be explained by a combination of the aetiology of type 2 diabetes and theories of spousal similarities in risk factors for type 2 diabetes. Theories of spousal similarities in risk factors for type 2 diabetes can be classified into two

main components: non-random mating and convergence over time [29]. The first theory refers to individuals seeking a partner based on phenotype similarity or phenotypic preference; for example, people finding a partner with a similar body composition or activity pattern, or partners preferentially meeting in socioeconomically stratified environments such as the workplace or educational institutions. The second theory suggests that, over the course of the relationship, spouses converge and become similar due to ‘social contagion’; for example, a couple will eat the same diet or jointly engage in similar physical or sedentary activities. Thus, two potentially phenotypically similar people may meet and converge further in health-related behaviours over time, leading both individuals to develop risk factors for type 2 diabetes, such as obesity. However, they may not both develop type 2 diabetes as they might have different underlying physiological predispositions for the disease. Spousal resemblance in obesity is supported by our study and other studies [18, 30], and by results from a US study in which having a spouse who became obese almost doubled an individual’s risk of also becoming obese [31]. Consequently, spousal obesity as a risk factor for the development of type 2 diabetes may simply reflect such spousal obesity resemblance, which seems to be the case among the women in our study: the association between spousal BMI and type 2 diabetes risk diminished in women after adjusting for their own BMI. In contrast, for men, the wife’s obesity status was associated with type 2 diabetes incidence beyond the effect of the man’s own obesity level. The same risk increment of incident type 2 diabetes was associated with a threefold larger difference in the spouse’s BMI than in his own. It is conceivable that the wife’s obesity level might be a proxy for other shared unhealthy behaviours, such as physical inactivity, which is a strong risk factor for type 2 diabetes [32, 33] even in absence of overweight/obesity [34]. Studies are inconsistent in terms of spousal similarities in physical activity [16, 29, 35]. Nevertheless, one study showed that inactive women are more likely to have an inactive spouse compared with inactive men [16].

Another factor that might explain why the wife’s obesity status is a stronger risk factor for incident type 2 diabetes than the husband’s is that, in this cohort, women’s dietary preferences may exert a higher degree of influence over the spouse’s diet than vice versa. In a US study, women were up to five times more likely than men to be responsible for planning and preparing meals and shopping for food [36]. Thus, the wife’s food choices may influence what the husband eats to a higher degree than vice versa, at least in the home setting. If this is an unhealthy or unbalanced diet, both the wife’s and the husband’s risk of type 2 diabetes may be influenced, and thus the joint risk of developing type 2 diabetes. Trials of dietary or general lifestyle intervention have shown that the spouses of people randomised to lifestyle modification also benefit in terms of improved diet and weight loss [37, 38]. However, dietary intake or composition in the home setting may differ

from food intake outside the home. Given a woman’s higher likelihood of being the food decision-maker in the home setting, it could be speculated that a woman’s eating patterns outside the home setting might mirror the pattern at home, whereas a husband’s food preferences outside the home might be very different. Thus, if a husband’s unhealthy food intake originates from outside the home setting, these choices might not influence the wife’s risk of type 2 diabetes. Couples with a higher resemblance in poor food diet score have been shown to also have a stronger resemblance in BMI changes over time than couples with discordant diet scores [31]. Unfortunately, the data available in ELSA did not allow us to examine these explanatory mechanisms directly.

It could be argued that a spousal resemblance in dietary patterns might, to a certain extent, be mirrored in a spousal resemblance in blood lipid levels and BP. Although the present study showed a consistent spousal resemblance of moderate strength in cardiovascular risk factors, only triacylglycerol levels in the husband were associated with an increased risk of type 2 diabetes in the wife. However, the relationship between triacylglycerols and insulin resistance and diabetes risk is currently classed as ‘likely non-causal’ [39].

It is possible that sex differences in the metabolic phenotype most closely associated with diabetes risk explain this sex-specific finding [40], but we have not been able to examine this in further detail in the current study.

The novelty and main strength of our study was that we examined the sex-specific effects of spousal risk factors along their entire continuum. We also investigated to what extent an association is explained by a person’s own risk factor level. In the present study, diabetes status at baseline was self-reported, which may have influenced the weak relationship between spousal diabetes and incident type 2 diabetes in the index individuals, as a meta-analysis has reported that spousal diabetes concordance is lower when diabetes status is self-reported [21]. Furthermore, we only analysed spousal diabetes and cardiometabolic risk factors measured at baseline. These factors might have changed during the follow-up, and couples might have separated. However, according to the theory of non-random mating [29], the increased type 2 diabetes risk linked to spousal obesity is likely to be carried forward even after divorce or widowhood.

In conclusion, the risk of incident type 2 diabetes among older English residents is associated with their spouse’s obesity level. This suggests that a couples-focused approach may be beneficial for the early detection of individuals with type 2 diabetes and those at high risk of developing type 2 diabetes, especially men (who are less likely than women to attend health checks). Thus, when healthcare professionals encounter an overweight or obese middle-aged to older person, even without knowing the spouse’s weight, attention should be given to the spouse as he/she may be at elevated risk of type 2 diabetes.

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**Data availability** Data were accessed via the UK Data Service under the data-sharing agreement no. 91400. ELSA data are available for all registered users at the UK Data Service website (<https://discover.ukdataservice.ac.uk/catalogue/?sn=5050&type=Data%20catalogue>; accessed October 27, 2017).

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**Duality of interest** The authors declare that there is no duality of interest associated with this manuscript.

**Contribution statement** JN developed the conception of the study, contributed to the statistical analyses and the interpretation of data, and drafted, revised and finalised the article. AH contributed to the study design, was responsible for the statistical analyses, and contributed to the interpretation of data and development, revision and finalisation of the manuscript. DRW developed the conception of the study, supervised the statistical analysis, and contributed to the interpretation of the data and development, revision and finalisation of the manuscript. All authors approved the final version of the manuscript. JN and AH are the guarantors of this work.

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