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Increasing body mass index at diagnosis of diabetes in young adult people during 1983–1999 in the Diabetes Incidence Study in Sweden (DISS)

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Abstract. Littorin B, Nyström L, Gullberg B, Råstam L, Östman J, Arnqvist HJ, Björk E, Blohmé G, Bolinder J, Eriksson JW, Scherstén B, Sundkvist G (University of Malmö/Lund, Malmö/Lund; Umeå University, Umeå; University Hospital, Uppsala; Söder Hospital, Stockholm; Huddinge Hospital, Huddinge; Faculty of Health Sciences, University of Linköping, Linköping and University Hospital, Umeå; Malmö University Hospital, Malmö, Sweden). Increasing body mass index at diagnosis of diabetes in young adult people during 1983–1999 in the Diabetes Incidence Study in Sweden (DISS). *J Intern Med* 2003; **254**: 251–256.

Objective. To study trends in body mass index (BMI) at diagnosis of diabetes in all young Swedish adults in the age range of 15–34 years registered in a nation-based registry.

Design. The BMI was assessed at diagnosis in diabetic patients 15–34 years of age at diagnosis, for a period of 17 years (1983–1999). Islet cell antibodies (ICA) were measured during three periods (1987–1988, 1992–1993 and 1998–1999).

Setting. A nationwide study (Diabetes Incidence Study in Sweden).

Subjects. A total of 4727 type 1 and 1083 type 2 diabetic patients.

Main outcome measures. Incidence-year specific BMI adjusted for age, gender and time of diagnosis (month).

Results. Body mass index at diagnosis increased significantly both in type 1 (21.4 ± 3.6 to 22.5 ± 4.0 ; $P < 0.0001$) and in type 2 (27.4 ± 6.8 to 32.0 ± 6.0 ; $P < 0.0001$) diabetic patients, also when adjusted for age, gender and month of diagnosis. A similar significant increase in BMI was found in type 1 diabetic patients and in type 2 diabetic patients in the periods 1987–1988, 1992–1993 and 1998–1999; years when ICA were assessed and considered in the classification of diabetes. Despite this increase in BMI, there was no increase in the incidence of diabetes in young-adult people in Sweden.

Conclusion. Body mass index at diagnosis of diabetes in subjects 15–34 years of age has substantially increased during 1983–1999 in Sweden when adjusted for age, gender and month of diagnosis.

Keywords: Body Mass Index, incidence, islet cell antibodies, type 1 diabetes, type 2 diabetes, year of diagnosis.

Abbreviations: BMI, Body Mass Index; ICA, islet cell antibodies.

Introduction

Available data suggest that the prevalence of obesity is increasing, both in UK [1] and in the US [2]. In

concordance, studies in Sweden have shown that body mass index (BMI) and the prevalence of obesity have increased amongst 18-year-old men in Sweden during the last 24 years [3]. In agreement, other

Swedish studies have shown that the increase in BMI and obesity has been particularly pronounced in young adult people during the 1980s [4, 5]. The role of obesity in the pathogenesis of diabetes type 2 [6] is evident and is supported by the recent observation that increased BMI in young age groups may explain a corresponding increase in the prevalence of diabetes [7]. Indeed, obesity has been suggested to precipitate the postpubertal onset of type 1 diabetes [8]. Thus, increased BMI in young-adults may not only affect the risk of developing type 2 diabetes but also the male excess of autoimmune type 1 diabetes, as it has been reported in a similar study in 15–34-year age group [9]. To further explore the putative impact of obesity on the development of diabetes in young adults, the development in BMI during 17 years amongst incident cases of diabetes in young-adult (15–34 years old) people in Sweden as recorded in the Diabetes Incidence Study in Sweden (DISS) were examined. The aims of this report were to evaluate whether there was an increase in BMI amongst incident cases of diabetes in young-adults during 1983–1999, and, if so, whether this affected the incidence of diabetes.

Materials and methods

Since 1983, there is a population-based prospective registration of all newly diagnosed cases of diabetes mellitus in the age range of 15–34 years in Sweden, the Diabetes Incidence Study in Sweden (DISS) [10]. In this study, on a standardized form, following data are recorded: day of birth, name, gender, maximum blood glucose during the first 2 weeks of diabetes (until 1992), height, body weight at diagnosis, and the type of diabetes as classified by the reporting physician [insulin-dependent diabetes (IDDM), non-insulin-dependent diabetes (NIDDM), diabetes secondary to other diseases, gestational, or unclassifiable diabetes].

To increase the strength of the study, the registration form was changed in 1992. In the new version, the diagnostic blood glucose value is noted, i.e. either as fasting, random, or 120 min after a 75-g oral glucose tolerance test. From 1992, type 1 and type 2 diabetes are used synonymously with the terms IDDM and NIDDM, respectively. Patients with gestational diabetes are not reported to DISS.

The objectives of DISS are to follow the trend in the incidence of diabetes in young adults and to find

clues to the aetiology and pathogenesis of diabetes and its complications. Therefore, during 1987–1988 [11], 1992–1993 [12] and during 1998–1999, islet cell antibodies (ICA) were measured at diagnosis. The prevalence of ICA amongst patients considered to have type 1 or type 2 diabetes was constant during the years.

Validity

The DISS is ongoing, and all Swedish diabetologists are repeatedly asked to check their records for new patients with diabetes amongst young-adults. Thus, as diabetes is a chronic disease, patients who might have been initially overlooked will be detected and reported later. Using a routine-based computer register (the Patient Administrative System Inpatient care), the registration was validated during 1983–1987 in two counties in southern Sweden covering 9.2% of subjects aged 15–34 years in Sweden [13]. The level of ascertainment of type 1 diabetes was 0.86, i.e. almost identical with that obtained in 1986–1997, 0.91, when local hospital registers of diabetic patients in one county in northern Sweden were used for validation. With regard to type 2 diabetes, the level of ascertainment is difficult to validate. Patient administrative systems are based on inpatients and type 2 diabetic patients are mostly diagnosed and treated as outpatients. Therefore, the ascertainment in type 2 diabetic patients is less than in type 1 diabetic patients. The ascertainment for type 2 diabetes seems, however, to have been constant during the years. The ascertainment study in northern Sweden 1986–1997 gave the same degree of ascertainment (0.49) as the 1983–1987 ascertainment study (0.54).

This study was confined to the period 1983–1999 and included incident cases registered with type 1 or type 2 diabetes. To avoid the risk for misclassification, cases classified as with secondary or unclassifiable diabetes were excluded. The frequencies of secondary (1–2%) and unclassifiable (5–10%) diabetes were constant during the observation period.

As reported in the Tables BMI was calculated as kg m^{-2} according to data from the standardized registration form. Main outcome measures were year-specific BMI in type 1 and type 2 diabetes adjusted for age, gender and time of diagnosis

(month). Obesity was defined in accordance with WHO [14], i.e. BMI ≥ 30 was considered as obesity.

The ICA were determined by a prolonged immunofluorescence assay [15, 16] and the results were expressed in Juvenile Diabetes Foundation (JDF) units. The detection limit (cut-off limit for abnormality) was 2 JDF (1987–1988), 6 JDF (1992–1993), and >0 JDF (1998–1999), respectively. The sensitivity was 100% and the specificity 100% in the last (no. 13) International Diabetes Workshop proficiency test for ICA.

Statistics

The relationship between the year at diagnosis and BMI was evaluated with regression analysis and adjusted for age, gender and month of the year. Differences between groups were evaluated by analysis of variance (Kruskal–Wallis test) and, if significant, differences between two groups were evaluated with the Mann–Whitney *U*-test. Differences in frequencies were evaluated with chi-square test. A *P*-value <0.05 was considered significant.

Results

Physical characteristics

Table 1 shows the complete material comprising 5810 patients registered during the 17 years, 4727 were classified as type 1 and 1083 as type 2 diabetic patients. There was predominance for men amongst type 1 diabetic patients (male : female ratio 1.9). As expected, age and BMI were significantly ($P < 0.0001$) higher in type 2 than in type 1 diabetic patients, both for women and men.

BMI and age according to year at diagnosis

Figure 1 shows that mean BMI at diagnosis significantly increased from 1983 to 1999, both in type 1 (from 21.4 ± 3.6 to 22.5 ± 4.0 [$P < 0.0001$]) and in type 2 diabetic patients (from 27.4 ± 6.8 to 32.0 ± 6.0 [$P < 0.0001$]). Indeed, during the years, BMI continuously increased with 0.085 year^{-1} for type 1 and 0.309 year^{-1} for type 2 diabetic patients. Amongst type 1 female diabetic patients, the increase was similar for all ages, whereas in males, the increase was found in patients 22 years of age or older. No similar age-dependent difference was discovered amongst type 2 diabetic patients. BMI varied with the season ($P = 0.0211$). We had therefore to adjust for month at diagnosis and not only for age and gender (BMI higher in men than in women) when we finally related BMI to the year of diagnosis. Nevertheless, when these factors were considered,

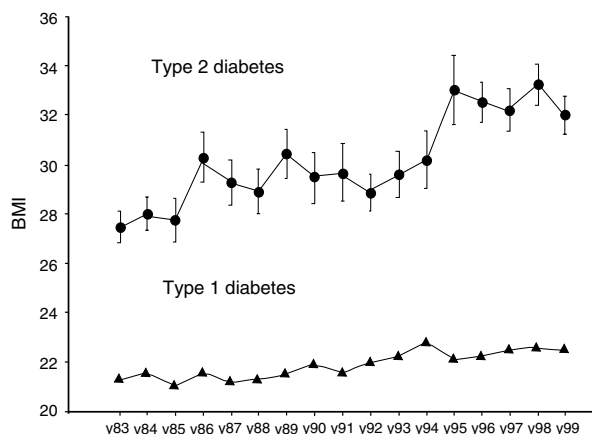


Fig. 1 Body mass index (BMI) according to year at diagnosis in Diabetes Incidence Study in Sweden (DISS) in type 1 and type 2 diabetic patients in Sweden during 1983–1999. Data are mean \pm SEM.

Table 1 Male–female ratio, age and body mass index (BMI) according to gender and type of diabetes in 5810 patients reported to the Diabetes Incidence Study in Sweden (DISS) 1983–1999

Characteristics	Type 1 diabetes		Type 2 diabetes		All	
	Male	Female	Male	Female	Male	Female
No. of cases	3073	1654	599	484	3672	2138
Male : female ratio	1.9		1.2		1.7	
Mean age (years \pm SD)	24.0 \pm 5.7	23.4 \pm 5.7	28.5 \pm 4.9	27.7 \pm 4.8	24.7 \pm 5.8	24.4 \pm 5.8
Mean BMI (kg m^{-2} \pm SD)	22.0 \pm 3.7	21.5 \pm 3.8	30.0 \pm 6.8*	29.9 \pm 8.1*	23.3 \pm 5.3	23.4 \pm 6.2

* $P < 0.0001$ versus corresponding type 1 diabetic patients.

still we found a significant increase in BMI during the observation period of 17 years, both for type 1 ($P < 0.0001$) and type 2 ($P < 0.0001$) diabetic patients.

Prevalence of obesity

The prevalence of obesity at diagnosis ($\text{BMI} \geq 30$) increased significantly ($P < 0.0020$) from 36% in 1983, to 58% in 1999 amongst type 2 diabetic patients whereas the prevalence was unchanged and low amongst type 1 diabetic patients (3.5% vs. 4.7%).

BMI related to ICA

Owing to problems with a classification based on clinical judgement [17], the material was subdivided into two groups during the years when ICA were assessed: (i) Patients with type 1 diabetes combined with ICA ('autoimmune positive type 1 diabetes') and (ii) Patients with type 2 diabetes without ICA. Table 2 shows that BMI significantly increased amongst patients with autoimmune positive type 1 diabetes and amongst patients classified as type 2 diabetes during the periods 1987–1988, 1992–1993 and 1998–1999. Indeed, it appeared as the increase in BMI amongst patients classified as type 2 diabetic had recently occurred (i.e. after 1993) whereas the increase amongst autoimmune positive type 1 diabetic patients seemed to have started earlier (i.e. after 1988).

Table 2 The development of mean body mass index (BMI) (\pm SD) in autoimmune positive type 1 and classical type 2 diabetic patients in Diabetes Incidence Study in Sweden (DISS)

Year of diagnosis	Autoimmune positive Type 1 diabetes	Classical Type 2 diabetes
1987–1988	$n = 220$ 20.8 ± 3.0	$n = 32$ 28.9 ± 7.4
1992–1993	$n = 215$ $21.5 \pm 3.1^*$	$n = 79$ 29.1 ± 7.1
1998–1999	$n = 227$ 22.0 ± 3.5	$n = 93$ $33.1 \pm 7.1^{**}$
Kruskal–Wallis	$P = 0.0009$	$P = 0.0005$

* $P < 0.0301$ versus 1987–1988; ** $P < 0.0006$ versus 1992–1993.

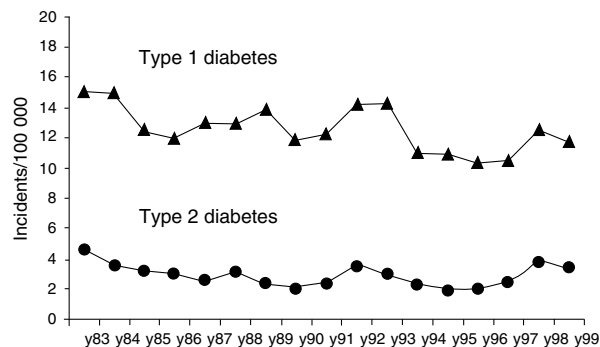


Fig. 2 Incidence of type 1 and type 2 diabetes amongst young adult people aged 15–34 years in Sweden during 1983–1999.

Incidence of diabetes

Figure 2 shows that the incidence of diabetes tended to decrease during the 17 years: for type 1 diabetes the decrease was $1.4\% \text{ year}^{-1}$ and statistically significant ($P = 0.008$), for type 2 diabetes the decrease was not statistically significant ($P = 0.17$).

Discussion

The present study shows that there has been a clear increase in BMI during the last 17 years in young-adult people developing diabetes in Sweden. The increase seems to be continuous and not stepwise in type 1 diabetic patients whereas it may have been a phenomenon of the last years in type 2 diabetic patients.

Despite this increase in BMI, it appears as the incidence of diabetes in Sweden amongst young-adult people 15–34 years of age had not increased during the years.

Our observation of an increase in BMI amongst young-adult diabetic patients during the last 17 years fits well with recent observations in Norway where obesity ($\text{BMI} \geq 30$) increased from 3–6% to 8–12% in 20–39-year-old diabetic patients during the periods 1984–1986 to 1995–1997 [7]. Although the prevalence later almost doubled in our newly diagnosed diabetic patients (11–18%), the prevalence of obesity was clearly higher already in 1983 in our study compared with the Norwegian. As BMI is increasing in the general young population [1, 2, 3], our finding may reflect this general trend of BMI in the general population. The observation that BMI increased in both type 1 and type 2 diabetic

patients also when ICA were considered, underlines the strength of our study.

Albeit the significant increase in BMI, the incidence of diabetes did not increase during the years in our study. In fact, the incidence of type 1 diabetes decreased. Recently, when DISS was merged with the Swedish Childhood Diabetes Study, we found that this decrease in incidence of type 1 diabetes amongst young-adults corresponded with an increase in the incidence amongst children below 6 years of age [18]. In children, it is difficult to separate the impact of linear growth from increment in BMI. Although a high and rapid linear growth is associated with the development of type 1 diabetes in children [19, 20], obesity on its own is also associated with a development of type 1 diabetes [21]. The lack of association between BMI and the incidence of type 1 diabetes in our study supports the idea that growth rather than BMI is important for the development of type 1 diabetes. In type 2 diabetes, the incidence was unchanged and no increase was noted, despite the increase in BMI. In this context, we have to consider that the validity of the registration of type 2 diabetic patients is less than in type 1 diabetic patients. Nevertheless, as the ascertainment was constant during the years for type 2 diabetic patients, it seems unlikely that differences in ascertainment explain the constant incidence of type 2 diabetes. Although almost half of the subjects with type 2 diabetes in young adults may have been missed, it is unlikely that active screening in obese groups or differences of unknown cases amongst the adult population contributed to the increase of BMI. In addition, lean subjects with type 2 diabetes should not be preferentially missed and thereby increasing the prevalence of obesity. Lean subjects with recent-onset diabetes will be suspected to have type 1 diabetes and therefore referred to diabetes clinics and reported to DISS. The proportion of cases classified as type 1, type 2 and unclassifiable diabetes, was stable during the study suggesting that, despite the increase in BMI, no major shift in the classification of diabetes had occurred. Indeed, our observation of an increase in BMI without an increase in incidence of type 2 diabetes fits well with two recent reports from Scandinavia [22, 23].

Increased BMI in young-adult people may, however, not necessarily be associated with an increased incidence of type 2 diabetes before 34 years of age.

Long-term increments of BMI in the young-adults may affect the incidence of diabetes later as noted in senior individuals [24]. It has been calculated that each kilogram of weight gained annually over 10 years increases the risk for developing diabetes with 49% in obese [25]. The frequency of obesity is increasing in children [26]. This may increase the incidence of diabetes in the future, emphasizing that it is important to continue with the prospective registration of incident diabetic patients in DISS. In this context, it could be mentioned that other factors like habitual snoring [27] might aggravate the risk of BMI for developing type 2 diabetes. Similarly, lifestyle factors such as tobacco and alcohol consumption increase the risk for type 2 diabetes in obese subjects [28]. Indeed, a sedentary lifestyle [29] favoured by TV watching [30] may be more important for the development of diabetes than increments in BMI.

In conclusion, BMI increased in incident cases of type 1 and type 2 diabetic patients in young-adult people in Sweden during a period of 17 years. So far, no increase in incidence of diabetes is noticed. The DISS Registry will reveal whether the increasing degree of obesity in children will lead to an increased incidence of diabetes in young adults.

Conflict of interest statement

No conflict of interest was declared.

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References

- 1 Seidell JC. Obesity in Europe: scaling an epidemic. *Int J Obes Relat Metab Disord* 1995; 19: 1–4.

- 2 Flegal KM. Trends in body weight and overweight in the U.S. population. *Nutr Rev* 1996; **54**: 97–100.
- 3 Rasmussen F, Johansson M, Hansen HO. Trends in overweight and obesity among 18-year-old males in Sweden between 1971 and 1995. *Acta Paediatr* 1999; **8**: 431–7.
- 4 Kuskowska-Wolk A, Bergström R. Trends in body mass index and prevalence of obesity in Swedish men 1980–89. *J Epidemiol Community Health* 1993; **47**: 103–8.
- 5 Kuskowska-Wolk A, Bergström R. Trends in body mass index and prevalence of obesity in Swedish women 1980–89. *J Epidemiol Community Health* 1993; **47**: 195–9.
- 6 Zimmet PZ. Kelly West Lecture 1991. Challenges in diabetes epidemiology – from West to the rest. *Diabetes Care* 1992; **15**: 232–52.
- 7 Midthjell K, Krüger Ö, Holmen J, Tverdal A, Claudi T, Björndal A, Magnus P. Rapid changes in the prevalence of obesity and known diabetes in an adult Norwegian population. The Nord-Trøndelag health surveys 1984–1986 and 1995–1997. *Diabetes Care* 1999; **22**: 1813–20.
- 8 Weets I, Van Autreve J, Van derAuwera BJ *et al.* and the Belgian Diabetes Registry. Male-to-female excess in diabetes diagnosed in early adulthood is not specific for the immune-mediated form nor is it HLA-DQ restricted: possible relation to increased body mass index. *Diabetologia* 2001; **44**: 40–7.
- 9 Blohmé G, Nyström L, Arnquist HJ *et al.* Male predominance of type 1 (insulin-dependent) diabetes mellitus in young adults: result from a 5-year prospective nationwide study of the 15–34-year age group in Sweden. *Diabetologia* 1992; **35**: 55–62.
- 10 Östman J, Arnquist HJ, Blohmé G *et al.* Epidemiology of diabetes mellitus in Sweden. Results of the first year of a prospective study in the population age group 15–34 years. *Acta Med Scand* 1986; **220**: 437–45.
- 11 Landin-Olsson M, Karlsson FA, Lernmark Å, Sundkvist G, and the Diabetes Incidence Study in Sweden Group. Islet cell and thyrogastic antibodies in 633 consecutive 15- to 34-yr-old patients in the Diabetes Incidence Study in Sweden. *Diabetes* 1992; **41**: 1022–7.
- 12 Törn C, Landin-Olsson M, Lernmark Å *et al.* Prognostic factors for the course of cell function in autoimmune diabetes. *J Clin Endocrinol Metab* 2000; **85**: 4619–23.
- 13 Littorin B, Sundkvist G, Scherstén B *et al.* Patient administrative system as a tool to validate the ascertainment in the diabetes incidence study in Sweden (DISS). *Diabetes Res Clin Pract* 1996; **33**: 129–33.
- 14 World Health Organization. *Obesity: Preventing and Managing the Global Epidemic*. Geneva: World Health Organization, 1998.
- 15 Landin-Olsson M, Sundkvist G, Lernmark Å. Prolonged incubation in the two-colour immunofluorescence test increases the prevalence and titres of islet cell antibodies in type 1 (insulin-dependent) diabetes mellitus. *Diabetologia* 1987; **30**: 327–32.
- 16 Borg H, Fernlund P, Sundkvist G. Protein tyrosine phosphatase-like protein IA2-antibodies plus glutamic acid decarboxylase 65 antibodies (GADA) indicates autoimmunity as frequently as islet cell antibodies assay in children with recently diagnosed diabetes mellitus. *Clin Chem* 1997; **43**: 2358–63.
- 17 Littorin B, Sundkvist G, Hagopian W *et al.* Islet cell and Glutamic acid decarboxylase antibodies at diagnosis of diabetes predict the subsequent need for insulin treatment – a cohort study in young adults whose disease was initially labeled as type 2 or unclassifiable diabetes. *Diabetes Care* 1999; **22**: 409–12.
- 18 Pundziute-Lyckå A, Dahlquist G, Nyström L *et al.* The Incidence of Type 1 diabetes has not increased but shifted to a younger age at diagnosis in the 0–34 years group in Sweden 1983 to 1998. *Diabetologia* 2002; **45**: 783–91.
- 19 Blom L, Persson LÅ, Dahlquist G. A high linear growth is associated with an increased risk of childhood diabetes. *Diabetologia* 1992; **35**: 528–33.
- 20 EURODIAS Substudy 2 Study Group. Rapid early growth is associated with increased risk of childhood type 1 diabetes in various European populations. *Diabetes Care* 2002; **25**: 1755–60.
- 21 Hyppönen E, Virtanen SM, Kenward MG, Knip M, Åkerblom HK. Obesity, increased linear growth, and risk of type 1 diabetes in children. *Diabetes Care* 2000; **23**: 1755–60.
- 22 Jacobsen BK, Njolstad I, Thune I, Wilsgaard T, Lochen M-L, Schirmer H. Increase in weight in all birth cohorts in a general population. The Tromsø study, 1974–1994. *Arch Intern Med* 2001; **161**: 466–72.
- 23 Eliasson M, Lindahl B, Lundbergh V, Stegmyr B. No increase in the prevalence of known diabetes between 1986 and 1999 in subjects 25–64 years of age in northern Sweden. *Diab Med* 2002; **19**: 874–80.
- 24 Sakurai Y, Teuya K, Shimada N *et al.* Relationship between weight change in young adulthood and the risk of NIDDM. *Diabetes Care* 1997; **20**: 978–82.
- 25 Resnick HE, Valsania P, Halter JB, Lin X. Differential effects of BMI on diabetes risk among black and white Americans. *J Epidemiol Community Health* 2000; **54**: 596–602.
- 26 Reilly JJ, Dorosty AR. Epidemic of obesity in UK children. *Lancet* 1999; **354**: 1874–5.
- 27 Elmasry A, Janson C, Lindberg E, Gislason T, Tageldin MA, Boman G. The role of habitual snoring and obesity in the development of diabetes: a 10-year follow-up in a male population. *J Intern Med* 2000; **248**: 13–20.
- 28 Persson PG, Carlsson S, Svanström L, Ostenson CG, Efendic S, Grill V. Cigarette smoking, oral moist snuff use and glucose intolerance. *J Intern Med* 2000; **248**: 103–10.
- 29 Perry IJ, Wannamethee SG, Walker MK, Thomson AG, Whincup PH, Shaper AG. Prospective study of riskfactors for development of non-insulin dependent diabetes in middle aged British men. *BMJ* 1995; **310**: 560–4.
- 30 Hu FB, Leitzmann MF, Stampfer MJ, Colditz GA, Willett WC, Rimm EB. Physical activity and television watching in relation to risk for type 2 diabetes mellitus in men. *Arch Intern Med* 2001; **161**: 1542–8.

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