Short-Term Weight Change and the Incidence of Diabetes in Midlife

Results from the Australian Longitudinal Study on Women's Health

GITA D. MISHRA, PHD[1]
GRETCHEN CARRIGAN, BSC, BECON[1]
WENDY J. BROWN, PHD[2]
ADRIAN G. BARNETT, PHD[1]
ANNETTE J. DOBSON, PHD[1]

OBJECTIVE — Although there is consensus that excess adiposity is strongly associated with type 2 diabetes, its relationship with weight change is less clear. This study investigates the relative impact of BMI at baseline and short-term (2- or 3-year) weight changes on the incidence of diabetes.

RESEARCH DESIGN AND METHODS — Prospective data were collected from a population-based cohort of middle-aged women participating in the Australian Longitudinal Study on Women's Health ($n = 7{,}239$ for this report). To date, participants have completed four mailed surveys (S1, 1996; S2, 1998; S3, 2001; and S4, 2004). Generalized estimating equations were used to model binary repeated-measures data to assess the impact of BMI at S1 and weight change (S1 to S2; S2 to S3) on 3-year incidence of diabetes at S3 and S4, respectively, adjusting for sociodemographic and lifestyle factors.

RESULTS — BMI at S1 was strongly associated with the development of diabetes by S3 or S4. Compared with women who had a BMI <25 kg/m^2, those with BMI ≥ 25 kg/m^2 had higher incidence of diabetes ($P < 0.0001$), with odd ratios reaching 12.1 (95% CI 7.6–19.3) for women in the very obese group (BMI ≥ 35 kg/m^2). There was no association between shorter-term weight gain or weight loss on first-reported diagnosis of diabetes ($P = 0.08$).

CONCLUSIONS — Because women's risk of developing type 2 diabetes in midlife is more closely related to their initial BMI (when aged 45–50 years) than to subsequent short-term weight change, public health initiatives should target the prevention of weight gain before and during early adulthood.

Diabetes Care 30:1418–1424, 2007

Over recent decades, the number of cases of type 2 diabetes worldwide has increased rapidly from ~30 million in 1985 to 194 million in 2003. By the year 2025, the figure is predicted to rise to 330 million (1). In Australia it was estimated that 854,400 adults, or 6.3% of the population, had diabetes in 2003 (1). This is expected to rise to almost 1.3 million by the year 2025, or an estimated prevalence of 7.6% (1). The consequent complications such as coronary artery and peripheral vascular disease, stroke, diabetic neuropathy, amputation, renal failure, and blindness will result in excess morbidity and mortality, as well as having a significant economic cost (1).

This epidemic of diabetes closely parallels the worldwide obesity epidemic (2), with Australia being one of the worst-affected nations (3). There are major concerns about the health consequences of obesity, including the growing burden of chronic diseases and an escalation of future costs of health services (4). From 1980 to 2000, the prevalence of obesity in the Australian urban population has risen from 7.1 to 18.4% (5). This represents a 2.5-fold increase over the last 20 years (5). Absolute weight gain in the last two decades has also been substantial. Australian prospective studies have consistently demonstrated that, on average, middle-aged women are currently gaining approximately one-half kilogram per year (6,7).

There are well-established associations between being overweight or obese and type 2 diabetes (2,8). The risk appears to increase with the duration of obesity (9,10). There is also evidence for an independent effect of weight change, particularly weight gain, on an increased risk of diabetes, whereas weight loss is followed by a reduced risk of type 2 diabetes over periods of 5–10 years (10–12). In the U.S. Nurses' Health Study, weight gain during adulthood, even at modest levels (e.g., <5 kg), was associated with increased risk of diabetes, independent of initial body weight. In contrast, women who lost >5 kg reduced their risk for diabetes by 50% (11).

Far less is known about short-term weight change and the time lags between incremental weight gain during midlife and its impact on the risk of diabetes. Prospective cohort studies can provide valuable evidence on the time course of changes in weight and the onset of weight-related health conditions. In this article, we use data from the first four surveys of the midage cohort of the Australian Longitudinal Study on Women's Health. The objective is to examine the relative importance of initial BMI and of short-term weight change (over 2 or 3 years) on the incidence of diabetes.

RESEARCH DESIGN AND METHODS

The Australian Longitudinal Study on Women's Health

The Australian Longitudinal Study on Women's Health (ALSWH) is a prospective study of factors affecting the health and well-being of three cohorts of women aged 18–23 years (young), 45–50 years

From the [1]School of Population Health, University of Queensland, Queensland, Australia; and the [2]School of Human Movement Studies, University of Queensland, Queensland, Australia.

Address correspondence and reprint requests to Gita Mishra, PhD, School of Population Health, University of Queensland, Herston 4006, Australia. E-mail: g.mishra@uq.edu.au.

Received for publication 24 October 2006 and accepted in revised form 15 February 2007.

Published ahead of print at http://care.diabetesjournals.org on 10 March 2007. DOI: 10.2337/dc06-2187.

Abbreviations: ALSWH, Australian Longitudinal Study on Women's Health.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

(middle aged), and 70–75 years (older) at the time of the initial surveys in 1996. Women were selected randomly from the National Medicare Health Insurance Database (which includes all permanent residents of Australia regardless of age, including immigrants and refugees) with intentional overrepresentation of women living in rural and remote areas. Further details of the recruitment methods and response rates have been described elsewhere (13). The study collects self-reported data using mailed surveys at 2- to 3-year intervals from ~40,000 women living in all states and territories of Australia. The surveys include questions about health conditions, symptoms, and diagnoses; use of health services; health-related quality of life; social circumstances, including work and time use; demographic factors; and health behaviors. Complete details of each survey are on the study website (available at http://www.alswh.org.au).

Informed consent was obtained from all participants in 1996, with ethical clearance obtained from the University of Newcastle. This article only includes data from the middle-aged cohort. There were four waves of data collection from 1996 to 2004 (S1, 1996; S2, 1998; S3, 2001; and S4, 2004).

A total of 13,716 middle-aged women agreed to participate at S1, and by S4 10,905 women remained in the study (79.5%), 204 (1.5%) had died, 636 (4.6%) had withdrawn from the study, and the remainder did not participate in at least one of S2, S3, and S4, generally because they could not be contacted.

Diabetes

At each survey women were asked if they had been told by a doctor that they had diabetes. At S1 they were asked if they had ever had a diagnosis of diabetes. At S2, S3, and S4 they were asked whether they had been diagnosed with diabetes in the time period that had elapsed since the previous survey. For the analysis, there was a definitional issue that had to be considered: diagnosis of diabetes was not differentiated into type 1 or type 2 at S1 and S4. Because the number of new cases of type 1 diabetes was small ($n = 5$ at S2 and $n = 12$ at S3), these were combined with the type 2 diabetic cases, so that the data reported here are for both type 1 and type 2 diabetes and referred to simply as "diabetes." Diabetes status could be determined at all four surveys for 10,629 women. Prevalence of diabetes at each survey was defined as the proportion of these women who reported at this survey or a previous survey that they had been told they had diabetes.

BMI and weight change

Women were asked to report their height and weight at each survey. Self-reported heights from the first three surveys were used to obtain a single estimated value for each woman by averaging the available data. BMI for each woman at S1 was calculated as self-reported weight (kg) at S1 divided by the square of estimated height (m^2). BMI was categorized as underweight (<18.5 kg/m^2), healthy weight (18.5 to <25 kg/m^2), overweight (25 to <30 kg/m^2), obese (30 to <35 kg/m^2), or very obese (≥ 35 kg/m^2) according to the World Health Organization classification (4). Fewer than 2% of women were classified as underweight at S1, so this category was combined with the healthy weight group for the analysis. The average percentage of annual weight change was calculated by subtracting self-reported weight at successive surveys and dividing by weight at the earlier survey and the number of years between the surveys. (S1 and S2 were 2 years apart, whereas S2 and S3 were 3 years apart). Average percentage of annual weight change was categorized as high loss ($\leq -5\%$), moderate loss (-5 to $\leq -2.5\%$), small loss (-2.5 to $\leq -1.5\%$), stable (-1.5 to $\leq +1.5\%$), small gain ($+1.5$ to $\leq +2.5\%$), moderate gain ($+2.5$ to $\leq +5\%$.), or high gain ($> +5\%$). As an indication of absolute weight gain, for a woman in the healthy weight range with BMI 22 kg/m^2 and height 1.65 m, 5% weight gain equates to 3 kg per year, whereas for a very obese woman with BMI 37 kg/m^2 and height 1.65 m, 5% weight gain equates to 5 kg per year.

Data needed to calculate BMI at S1 were missing for 696 women. In addition, data needed to calculate average percentage of annual weight change between S1 and S2 or between S2 and S3 were missing for 2,503 women. Thus, we had data on diabetes, BMI, and weight change for 7,875 women.

Menopause status

Menopause status at S1 was defined on the basis of self-reported menstrual bleeding. Women who reported no menstrual bleeding in the last 12 months were classified as postmenopausal. Those with menstrual bleeding in the last 12 months but not in the last 3 months or with different menstrual frequency compared with the previous year were classified as perimenopausal. Those with menstrual bleeding in the last 3 months and in the last 12 months and with the same frequency as in the year before were classified as premenopausal. Women who had a hysterectomy were classified as having surgical menopause.

Social and lifestyle factors

Social and behavioral factors were based on information collected at S1. Educational qualifications were categorized as "no formal qualifications;" "school certificate;" "higher school certificate;" "trade apprenticeship, certificate, or diploma;" or "university degree or higher degree." Area of residence was categorized as "urban," "large rural center," "small rural center," or "other rural or remote area" (14).

Participants were asked to report frequency of engaging in vigorous (e.g., aerobics, jogging) and less vigorous (e.g., walking and swimming) exercise lasting for ≥ 20 min in a normal week (15). Responses were scored using approximate weekly frequencies of exercise (never = 0, once a week = 1, 2 or 3 times per week = 2.5, 4–6 times per week = 5, every day = 7, and more than once a day = 10) and then weighted to reflect the intensity of the activity (vigorous = 5 and less vigorous = 3). The resulting physical activity scores ranged from 0 to 80 and were categorized as "none (<5)," "low (5 to <15)," "moderate (15 to <25)," or "high (≥ 25)." A score of 15 is commensurate with the current recommendation of moderate intensity activity on most days of the week. This measure is described in more detail elsewhere (7,16) and has previously been shown to have acceptable test-retest reliability (17). Standard questions were used to categorize respondents as never-smoker, ex-smoker, or current smoker.

Statistical analysis

Predictors of 3-year incidence of diabetes were examined using repeated-measures data, with weight change between S1 and S2 used to predict incidence between S2 and S3 and weight change between S2 and S3 used to predict incidence between S3 and S4. These time lags were used to minimize the possibility of reverse causality from weight loss due to behavioral intervention or treatment as a consequence of the diabetes diagnosis or due to weight changes induced by the disease (18).

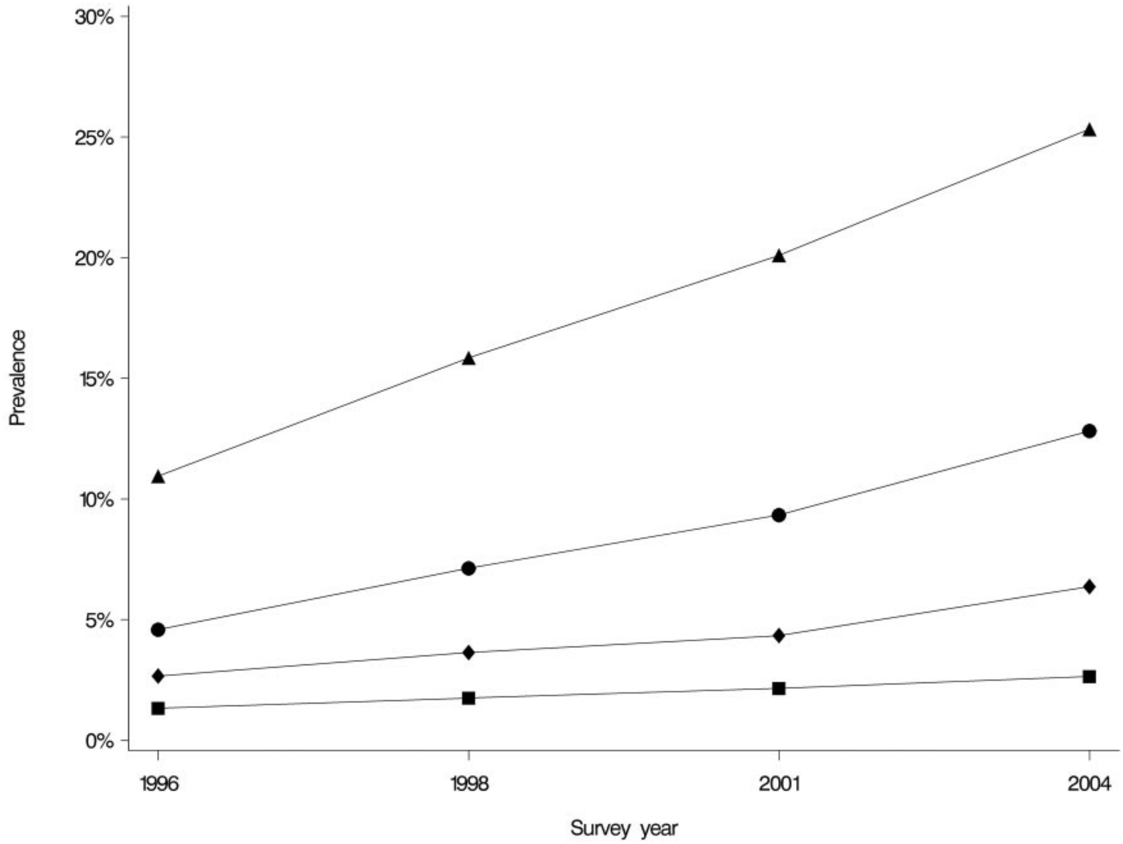

Figure 1—*Trends in prevalence of diabetes in middle-aged women grouped according to BMI at the first survey of the ALSWH.* ■, *healthy* (n = 5,252); ♦, *overweight* (n = 2,858); ●, *obese* (n = 1,178); ▲, *very obese* (n = 612).

Women who first reported a diagnosis of diabetes at S1 or S2 were excluded from this analysis (n = 406). Data for women who first reported a diagnosis of diabetes at S3 were not included for the subsequent time period (n = 119). Data from S1 were used for all other predictor variables: BMI, menopausal status, area of residence, education level, physical activity level, and smoking status.

Incidence of diabetes from S2 to S3 or from S3 to S4 was modeled using generalized estimating equations for binary repeated-measures data (19). The regression coefficients obtained from generalized estimating equations reflect the relationship between the incidence of diabetes and the corresponding predictor variables using all longitudinal data available (20). The within-subject correlation structures were assumed to be exchangeable; that is, the correlations between repeated measurements were assigned as being equal. The analyses were performed using SAS, version 9.1.3 (21) using PROC GENMOD with the options TYPE = EXCH and D = Binomial and Link = logit. For univariate analyses, separate models were fitted for each of the predictor variables. For multivariable analysis a model was fitted with all the predictors, then those that were not statistically significant at the 5% level were omitted, and a final parsimonious model was fitted.

RESULTS — The prevalence of diabetes at each survey among women categorized by BMI at S1 is shown in Fig. 1. At S1, prevalence ranged from 1.3% in the healthy weight category to 10.9% among the very obese women. Over time, the prevalence increased in all four BMI groups with greater increases in the more overweight groups.

Three-year incidence rates from S2 (1998) to S3 (2001) and from S3 to S4 (2004) and crude odds ratios for developing diabetes, by each of the weight and other variables, are shown in Table 1. Incidence rates were highest for women who were obese at S1, were oldest (age 50 at S1), reported none or low levels of physical activity, or were ex-smokers. In the univariate (unadjusted) models, BMI, age, smoking status, educational qualifications, and menopausal status were all statistically significantly associated with the development of diabetes. Annual percent weight change was only a statistically significant risk factor for those with moderate weight loss (−5 to ≤−2.5%) (P = 0.02) and for those in the high-gain (>+5%) group (P = 0.05) compared with the stable group. Similarly, physical activity was only associated with a lower incidence of diabetes for those in the high-activity group (P = 0.05) compared with the least active group (Table 1).

In the fully adjusted model (Table 2), BMI at S1 remained most strongly associated with incidence of diabetes. There was a significant increase in the incidence of reported diabetes across BMI groups at S1 compared with women in the healthy BMI group ($P < 0.0001$ for linear trend). Age was positively associated with the incidence of diabetes, with an odds ratio of 1.11 per year increase in age. Ex-smokers were 1.5 times more likely to develop diabetes than never-smokers. Annual weight change and activity level were not statistically significant factors in the fully adjusted model, nor were there any significant interactions between weight change and BMI groups (results not shown).

Table 1—*Number of women and 3-year incidence (%) of diabetes at S3 (2001) and S4 (2004), with odds ratios for developing diabetes from S2 (1998) to S3 (2001) and from S3 to S4 (2004) for 7,239 middle-aged women: results from univariate analyses*

Characteristics at S1 in 1996	S3 Number of women (diabetes incidence %)	S4 Number of women (diabetes incidence %)	Odds ratio (95% CI)	*P* value (for the variable and for each category)
BMI (kg/m^2)				*<0.0001*
Healthy (<25)*	3,985 (0.5)	3,965 (0.5)	1.00	
Overweight (25 ≤ BMI < 30)	2,064 (1.0)	2,044 (2.1)	3.16 (2.12–4.72)	<0.0001
Obese (30≤ BMI < 35)	815 (3.1)	790 (4.4)	7.88 (5.25–11.83)	<0.0001
Very obese (≥35)	375 (6.1)	352 (5.7)	12.75 (8.21–19.81)	<0.0001
Annual percent weight change				*0.3*
High loss (≤−5%)	246 (0.8)	108 (2.8)	1.11 (0.45–2.76)	0.82
Moderate loss (−5 to ≤−2.5%)	565 (1.2)	412 (3.6)	1.79 (1.12–2.87)	0.02
Small loss (−2.5 to ≤−1.5%)	503 (1.2)	475 (0.8)	0.80 (0.42–1.55)	0.51
Stable (−1.5 to ≤+1.5%)	3,151 (1.3)	3,778 (1.2)	1.00	
Small gain (+1.5 to ≤+2.5%)	993 (1.2)	1,017 (2.0)	1.26 (0.84–1.89)	0.27
Moderate gain (+2.5 to ≤+5%)	1,167 (0.9)	1,059 (1.7)	1.03 (0.67–1.57)	0.90
High gain (>+5%)	614 (1.5)	302 (3.3)	1.65 (1.00–2.72)	0.05
Age (per year)			1.14 (1.03–1.25)	*0.01*
Physical activity level				*0.09*
None	1,804 (1.4)	1,778 (2.0)	1.00	
Low	2,258 (1.3)	2,228 (1.8)	0.93 (0.66–1.31)	0.7
Moderate	1,967 (1.1)	1,945 (1.3)	0.70 (0.48–1.03)	0.07
High	1,210 (0.8)	1,200 (1.3)	0.63 (0.40–1.00)	0.05
Smoking status				*0.04*
Never-smoker	4,015 (1.0)	3,974 (1.4)	1.00	
Ex-smoker	2,133 (1.7)	2,097 (2.0)	1.51 (1.12–2.04)	0.007
Current smoker	1,091 (1.0)	1,080 (1.9)	1.18 (0.79–1.77)	0.4
Education				*0.04*
No formal	1,048 (1.2)	1,035 (2.3)	1.00	
School certificate	2,327 (1.6)	2,290 (1.6)	0.90 (0.61–1.34)	0.6
Higher school certificate	1,217 (1.2)	1,203 (2.1)	0.91 (0.58–1.42)	0.7
Trade apprentice/certificate diploma	1,501 (1.1)	1,485 (1.1)	0.62 (0.39–0.99)	0.05
University/higher degree	1,146 (0.7)	1,138 (1.2)	0.54 (0.32–0.91)	0.02
Menopause status				*0.009*
Premenopausal	3,159 (0.9)	3,130 (1.4)	1.00	
Perimenopausal	2,097 (1.5)	2,065 (1.0)	1.06 (0.74–1.52)	0.7
Postmenopausal	415 (0.7)	412 (2.2)	1.24 (0.67–2.28)	0.5
Surgical menopause	1,568 (1.5)	1,544 (2.8)	1.85 (1.33–2.58)	0.0003
Area of residence				*0.5*
Urban	2,256 (1.3)	2,524 (1.4)	1.00	
Large rural center	1,073 (1.2)	1,060 (1.4)	0.98 (0.63–1.53)	0.9
Small rural center	1,042 (1.0)	1,032 (1.6)	0.94 (0.59–1.47)	0.8
Other rural/remote	2,568 (1.3)	2,535 (2.0)	1.22 (0.88–1.68)	0.3

*As only 118 women had BMI <18.5 kg/m^2, their data are included in the healthy weight category. Italicized *P* values are for test for linear trend across each variable.

CONCLUSIONS — Overweight and obesity are becoming increasingly serious problems in Australia and throughout the world (4,5). Although there is consensus that excess adiposity is strongly associated with the development of type 2 diabetes, the association between incidence of diabetes with weight change is less clear. Prospective data from middle-aged women participating in this longitudinal study were used to investigate this issue. Our findings indicate that initial BMI at S1 was much more strongly associated with the development of diabetes than shorter-term weight changes (losses or gains) over the 2–3 years before the diagnosis.

This study shows that compared with women in the healthy weight range, those who were overweight or obese at S1 had substantially higher incidence of diabetes in subsequent surveys, with odds ratios in the obese group reaching >12. These findings support those from a number of other studies, including the U.S. Nurses' Health Study, in which 61% of type 2 diabetes cases could be attributed to overweight and obesity (22). Similarly in the Women's Health Study, the relative risk for developing type 2 diabetes was 9 for obese women compared with healthy weight women (8). The magnitude of the association with BMI was much stronger than with physical activity, confirming a finding among Finnish men and women (23).

Table 2—*Odds ratios for incidence of diabetes: results from a fully adjusted model and a parsimonious model that includes only those variables that were statistically significant at the 5% level in the fully adjusted model*

	Fully adjusted model*		Parsimonious model†	
	OR (95%CI)	*P* value	OR (95%CI)	*P* value
BMI (kg/m^2)		*<0.0001*		*<0.0001*
Healthy (<25)‡	1.00		1.00	
Overweight (25 ≤ BMI < 30)	3.00 (2.00–4.50)	<0.0001	3.08 (2.06–4.60)	<0.0001
Obese (30 ≤ BMI < 35)	7.32 (4.81–11.14)	<0.0001	7.77 (5.18–11.66)	<0.0001
Very obese (≥35)	12.48 (7.84–19.88)	<0.0001	12.59 (8.10–19.55)	<0.0001
Percent weight change per year		*0.08*		
High loss (≤−5%)	0.56 (0.22–1.41)	0.2		
Moderate loss (−5 to ≤−2.5%)	1.32 (0.82–2.14)	0.3		
Small loss (−2.5 to ≤−1.5%)	0.63 (0.32–1.21)	0.2		
Stable (−1.5 to ≤+1.5%)	1.00			
Small gain (+1.5 to ≤+2.5%)	1.24 (0.82–1.87)	0.3		
Moderate gain (+2.5 to ≤+5%)	0.93 (0.60–1.42)	0.7		
High gain (>+5%)	1.54 (0.92–2.56)	0.1		
Age	1.11 (1.00–1.23)	*0.04*	1.11 (1.01–1.23)	*0.03*
Physical activity level		*0.6*		
None	1.00			
Low	1.12 (0.79–1.59)	0.5		
Moderate	0.88 (0.59–1.31)	0.5		
High	0.90 (0.56–1.45)	0.7		
Smoking status		*0.04*		*0.04*
Never-smoker	1.00		1.00	
Ex-smoker	1.50 (1.11–2.03)	0.009	1.48 (1.09–2.01)	0.01
Current smoker	1.25 (0.83–1.90)	0.3	1.31 (0.87–1.97)	0.2
Education		*0.4*		
No formal qualifications	1.00			
School certificate	1.13 (0.74–1.70)	0.6		
Higher school certificate	1.31 (0.81–2.10)	0.3		
Trade apprentice/certificate/diploma	0.88 (0.53–1.44)	0.6		
University/higher degree	0.86 (0.49–1.49)	0.6		
Menopause status		*0.3*		
Premenopausal	1.00			
Perimenopausal	0.96 (0.67–1.37)	0.8		
Postmenopausal	1.03 (0.55–1.93)	0.9		
Surgical menopause	1.37 (0.97–1.94)	0.08		
Area of residence		*0.7*		
Urban	1.00			
Large rural center	0.92 (0.59–1.44)	0.7		
Small rural center	0.83 (0.52–1.33)	0.4		
Other rural/remote	1.06 (0.76–1.48)	0.8		

*Adjusted for all factors listed. †Only including significant variables ($P \leq 0.05$) from the fully adjusted model. ‡Only 118 women had BMI <18.5 kg/m^2 so we have included them in the healthy weight category. Italicized *P* values are for test for linear trend across each variable.

The strong association with initial BMI at S1 found in this study may reflect at least three underlying issues: the role of duration of obesity as a risk factor for diabetes; the timing of weight change during a critical period before S1, such as during early adulthood; and the latency of the effect of weight gain on diabetes. Previous studies have found that weight change during adulthood over longer periods than those considered in this study is associated with incidence of type 2 diabetes (12,11,24). For example, in the U.S. Nurses' Health Study, there was a strong independent effect of initial BMI on predicting risk of type 2 diabetes; a weight gain of 8–10.9 kg (from age 18 until 1976 when the women were aged between 30 and 55 years) was associated with a relative risk of type 2 diabetes of 2.7, whereas those who gained ≥20 kg had a relative risk of 12.3 (11). Results from the Health Professionals Follow-Up Study have also shown that among men the relative risks of type 2 diabetes were 1.4, 1.6, and 2.1 for those who gained 3–5 kg, 6–8 kg, and ≥9 kg, respectively, in the 10 years before the incidence of diabetes (12).

In the U.S. Diabetes Prevention Program, weight loss was associated with a 16% reduction in risk of diabetes. As the participants were in the intensive lifestyle intervention arm of the program, they experienced much greater weight loss than women in our study (mean change of 5.8% per year compared with 0.7% per year in our study) (25).

A Danish longitudinal study followed-up two groups of men, one with juvenile-onset diabetes and obesity and the other a randomly selected comparison group, who were weighed at average ages of 20, 33, 44, and 51 years. The study found that the risk of type 2 diabetes increased with weight gain in early adult life but not with weight changes in the late 30s and the 40s (18). This finding supports our results of no association between percentage of change in weight during middle age and the development of diabetes.

A reduced risk of developing diabetes with increased activity has been demonstrated in several prospective studies (2). In most of these, a significant inverse association between physical activity and diabetes remained even after adjustment for BMI. The few studies that have investigated the impact of weight change on the incidence of diabetes have not focused on physical activity and diabetes in their major hypotheses or have not reported the results (12). We found that physical activity level at S1 was not associated with the incidence of diabetes over surveys 2–4. This may be explained by lack of an effect or by inadequate measurement of physical activity, as we did not ask the women to report duration of activities in S1.

Our results revealed that ex-smokers were at a greater risk of developing diabetes than never-smokers, whereas there was no statistically significant effect for smokers. One reason for this increased risk among ex-smokers could be the higher rate of weight gain in this group before S1. Previous research with this cohort has shown that the odds of gaining weight in the 5-year period from S1 to S3 were much higher among women who quit smoking than in current smokers or never-smokers (7).

The attenuation of the effects of menopause status (primarily hysterectomy) in the adjusted model may be due to diabetes and hysterectomy having a number of risk factors in common. For instance, although being overweight and having a higher waist-to-hip ratio are risk factors for diabetes, they are also associated with increased risk of fibroids. Therefore it might be expected that there would be a higher hysterectomy rate among overweight women (26). Furthermore, hysterectomy at baseline could be indicative of weight gain before S1, as previous research has shown that women who had a hysterectomy were more likely to gain weight (>5 kg in 5 years) than those who did not (7,26).

The most obvious limitation of this study is that all the data are self-reported. Previous studies have shown that women are likely to understate their weight and overstate their height (27), so that BMI is more likely to be underestimated than overestimated. However, in a validity study with 200 women from the ALSWH, we found 82% agreement on BMI categories from self-reported and measured BMI ($\kappa = 0.72$), with approximately equal proportions of women with underestimated (9.4%) and overestimated (8.8%) BMI, so this may not be a major bias in the study as a whole. On the other hand, undiagnosed cases of diabetes would not be identified, and the effect would be to reduce the estimated incidence rates and their associations with weight.

This study has a number of strengths. It has a community-based sample rather than clinical groups of women. The large sample size means that it is possible to obtain reasonably stable estimates of incidence rates between successive surveys. In a previous article, we have shown that weight gain in this cohort was associated with quitting tobacco smoking and with menopause, so it was important to include these factors in the analyses (7).

In general, strategies for weight loss as a treatment for adult obesity have met with limited success, and lost weight is usually regained within 2 years (28). The results presented here and supported by previous studies (8,18,22) seem to indicate that the risk of developing type 2 diabetes in midlife is more closely related to initial BMI than to subsequent short-term weight change. Moreover, our previous work with both the middle-aged and younger cohorts of the ALSWH indicates that women who are overweight or obese are more likely to gain weight over subsequent surveys than their counterparts who remain in the healthy weight range (7,29). As there is no cure for diabetes, the results presented here point to the paramount importance of primary prevention strategies in the fight against overweight and obesity.

Previous work suggests that only small changes in energy balance (which can result from increasing physical activity and reducing total energy intake) are necessary to prevent the development of overweight and obesity (6,7,30). We concur with Hill et al. (30) that it is now particularly important to "inspire people to make behavior changes within the current environment that are sufficient to resist the push of environmental factors toward weight gain." These changes are required during early adulthood, so that young adults do not develop overweight or obesity as they approach middle age and increase their risk of early development of diabetes and other chronic illness.

Acknowledgments— The ALSWH, which was conceived and developed by groups of interdisciplinary researchers at the Universities of Newcastle and Queensland, is funded by the Australian Government Department of Health and Ageing.

We thank all the participants for their valuable contribution to this project.

References

1. International Diabetes Federation: Prevalence of diabetes. [article online], 2006. Available from http://www.eatlas.idf.org/prevalence/. Accessed 18 July 2006
2. Schulze MB, Hu FB: Primary prevention of diabetes: what can be done and how much can be prevented? *Annu Rev Public Health* 26:445–467, 2005
3. Dixon T, Waters AM: A Growing Problem: *Trends and Patterns in Overweight and Obesity Among Adults in Australia*. Canberra, Australia, Australian Institute of Health and Welfare, 2003
4. World Health Organization: *Obesity: Preventing and Managing the Global Epidemic*. Geneva, World Health Org., 2000
5. Cameron AJ, Welborn TA, Zimmet PZ, Dunstan DW, Owen N, Salmon J, Dalton M, Jolley D, Shaw JE: Overweight and obesity in Australia: the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Med J Aust* 178:427–432, 2003
6. Ball K, Crawford D, Ireland P, Hodge A: Patterns and demographic predictors of 5-year weight change in a multi-ethnic cohort of men and women in Australia. *Public Health Nutr* 6:269–281, 2003
7. Brown WJ, Williams L, Ford JH, Ball K, Dobson AJ: Identifying the energy gap: magnitude and determinants of 5-year weight gain in midage women. *Obes Res* 13:1431–1441, 2005
8. Weinstein AR, Sesso HD, Lee IM, Cook NR, Manson JE, Buring JE, Gaziano JM: Relationship of physical activity vs body mass index with type 2 diabetes in women. *JAMA* 292:1188–1194, 2004
9. Pontiroli AE, Galli L: Duration of obesity is a risk factor for non-insulin-dependent diabetes mellitus, not for arterial hypertension or for hyperlipidaemia. *Acta Diabetol* 35:130–136, 1998
10. Wannamethee SG, Shaper AG: Weight change and duration of overweight and obesity in the incidence of type 2 diabetes. *Diabetes Care* 22:1266–1272, 1999

11. Colditz GA, Willett WC, Rotnitzky A, Manson JE: Weight gain as a risk factor for clinical diabetes mellitus in women. *Ann Intern Med* 122:481–486, 1995
12. Koh-Banerjee P, Wang Y, Hu FB, Spiegelman D, Willett WC, Rimm EB: Changes in body weight and body fat distribution as risk factors for clinical diabetes in US men. *Am J Epidemiol* 159:1150–1159, 2004
13. Lee C, Dobson AJ, Brown WJ, Bryson L, Byles J, Warner-Smith P, Young AF: Cohort profile: the Australian Longitudinal Study on Women's Health. *Int J Epidemiol* 34:987–991, 2005
14. Australian Institute of Health and Welfare: *Rural, Regional and Remote Health: A Guide to Remoteness Classifications*. Canberra, Australia, Australian Institute of Health and Welfare, 2004
15. Risk Factor Prevalence Study Management Committee: *Risk Factor Prevalence Study: Survey Number 3*. Canberra, Australia, National Heart Foundation of Australia and Australian Institute of Health, 1990
16. Brown WJ, Mishra G, Lee C, Bauman A: Leisure time physical activity in Australian women: relationship with well being and symptoms. *Res Q Exerc Sport* 71:206–216, 2000
17. Booth ML, Owen N, Bauman AE, Gore CJ: Retest reliability of recall measures of leisure-time physical activity in Australian adults. *Int J Epidemiol* 25:153–159, 1996
18. Black E, Holst C, Astrup A, Toubro S, Echwald S, Pedersen O, Sorensen TI: Long-term influences of body-weight changes, independent of the attained weight, on risk of impaired glucose tolerance and type 2 diabetes. *Diabet Med* 22: 1199–1205, 2005
19. Dobson AJ: *An Introduction to Generalized Linear Models*. Boca Raton, FL, Chapman & Hall/CRC Press, 2002
20. Twisk JWR: *Applied Longitudinal Data Analysis for Epidemiology*. Cambridge, Cambridge University Press, 2003
21. SAS Institute: *SAS/STAT Users Guide*. Cary, NC, SAS Institute, 1999
22. Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, Willett WC: Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. *N Engl J Med* 345: 790–797, 2001
23. Hu G, Lindstrom J, Valle TT, Eriksson JG, Jousilahti P, Silventoinen K, Qiao Q, Tuomilehto J: Physical activity, body mass index, and risk of type 2 diabetes in patients with normal or impaired glucose regulation. *Arch Intern Med* 164:892–896, 2004
24. Field AE, Manson JE, Laird N, Williamson DF, Willett WC, Colditz GA: Weight cycling and the risk of developing type 2 diabetes among adult women in the United States. *Obes Res* 12:267–274, 2004
25. Hamman RF, Wing RR, Edelstein SL, Lachin JM, Bray GA, Delahanty L, Hoskin M, Kriska AM, Mayer-Davis EJ, Pi-Sunyer X, Regensteiner J, Venditti B, Wylie-Rosett J: Effect of weight loss with lifestyle intervention on risk of diabetes. *Diabetes Care* 29:2102–2107, 2006
26. Ceausu I, Shakir YA, Lidfeldt J, Samsioe G, Nerbrand C: The hysterectomized woman: is she special? The women's health in the Lund area (WHILA) study. *Maturitas* 53:201–209, 2006
27. Niedhammer I, Bugel I, Bonenfant S, Goldberg M, Leclerc A: Validity of self-reported weight and height in the French GAZEL cohort. *Int J Obes Relat Metab Disord* 24:1111–1118, 2000
28. Gill T, Bauman A, Rychetnik L, Hector, Miller Y, King L: Detailed review of intervention studies: how do we best address the issues of overweight, obesity and cardiovascular disease? [article online], 2004. Available from http://www.heartfoundation.com.au/downloads/Ov_Ob_interventions_report.pdf. Accessed 18 July 2006
29. Ball K, Brown W, Crawford D: Who does not gain weight? Prevalence and predictors of weight maintenance in young women. *Int J Obes Relat Metab Disord* 26: 1570–1578, 2002
30. Hill JO, Wyatt HR, Reed GW, Peters JC: Obesity and the environment: where do we go from here? *Science* 299:853–855, 2003