Long-Term Obesity and Cardiovascular, Inflammatory, and Metabolic Risk in U.S. Adults

Jennifer B. Dowd, PhD, Anna Zajacova, PhD

This activity is available for CME credit. See page A4 for information.

Background: People worldwide are becoming obese at earlier ages, increasing exposure to long-term obesity.

Purpose: To examine how BMI at age 25 years predicts later obesity and test the importance of long-term obesity beyond obesity severity for adult cardiovascular, inflammatory, and metabolic risk.

Methods: Data from adults aged 35–64 years from the 1999–2010 U.S. National Health and Nutrition Examination Survey were analyzed in 2013 to test how BMI at age 25 years predicts later adult BMI. Next, logistic regression models predicted the odds of elevated risk for blood pressure (BP); high-density lipoprotein cholesterol; total cholesterol; triglycerides; C-reactive protein (CRP); and glycosylated hemoglobin (HbA1c) by BMI at age 25 years and current BMI.

Results: Men obese at age 25 years had a 23.1% estimated probability of Class III obesity after age 35 years, compared to a 1.1% probability for men of normal weight at this age. For women, these probabilities were 46.9% and 4.8%, respectively. Those obese in both periods had higher odds of elevated BP, CRP, and HbA1c compared to those of normal weight at age 25 years, with no effects for lipids. After adjustment for current BMI, these associations were either eliminated (for BP and CRP) or greatly reduced (HbA1c).

Conclusions: The biological risks of long-term obesity are primarily due to the risk of more severe obesity later in life among those obese early in life, rather than obesity duration. Current body weight rather than duration may be the best reflection of clinical cardiovascular and metabolic risk.
(Am J Prev Med 2014;46(6):578–584)

Introduction

The striking rise in obesity rates in the U.S. over the past several decades has led to a parallel rise in the duration of obesity over the average individual's lifetime. According to the most recent 2009–2010 estimates, 35.9% of American adults aged ≥20 years are categorized as obese, a number that has risen from 13.3% in 1960–1962,[1,2] and the probability of being obese at age 25 years has increased by 30% for those born between 1955 and 1975.[3] The trend toward increasing lifelong obesity in recent cohorts has been projected to decrease life expectancy and increase disability rates in the U.S.[2,3] Although current obesity has well-known associations with cardiovascular and metabolic risk factors,[4,5] less is known about the health consequences of longer-term obesity. If the duration of obesity exposure increases health risks analogously to the risks of pack-years of smoking,[6] the implications for population aging and the medical costs of the high number of U.S. children and young adults who are currently obese could be dramatic.[7]

Longer obesity duration may impact biological risk through a variety of pathways. The impact of obesity on peripheral insulin resistance may eventually exhaust pancreatic β cells, leading to decreased insulin production and subsequent diabetes.[8] Obesity is associated with altered cardiac hemodynamics, which over time may impact cardiac muscle performance and increase blood pressure.[9] Adipose tissue is also associated with increased expression of inflammatory cytokines such as interleukin (IL)-6 and tumor necrosis factor (TNF)-α,[9] and long-term obesity may inhibit regulatory responses resulting in a systemic proinflammatory state. Alternatively, longer

From the Department of Epidemiology and Biostatistics, City University of New York School of Public Health, Hunter College, City University of New York Institute for Demographic Research (Dowd), City University of New York, New York, New York; and the Department of Sociology (Zajacova), University of Wyoming, Laramie, Wyoming

Address correspondence to: Jennifer B. Dowd, PhD, City University New York School of Public Health, Hunter College, 2180 Third Avenue, 5th Floor, New York NY 10035. E-mail: jdowd@hunter.cuny.edu.

0749-3797/$36.00
http://dx.doi.org/10.1016/j.amepre.2014.01.016

obesity duration may be associated with health risks primarily through its association with more severe obesity, which has known associations with a higher prevalence of hypertension, dyslipidemia, diabetes, and inflammation.[5,10] Despite these potential links, few studies have explicitly examined the health effects of long-term obesity. The current study analyzed the association of obesity at age 25 years with severity of obesity and biomarkers of cardiovascular, metabolic, and inflammatory risk later in adulthood in a nationally representative U.S. sample.

Methods

Data

Data were analyzed from the National Health and Nutrition Examination Survey (NHANES), 1999–2010, a representative survey of the non-institutionalized U.S. population.[11] These continuous cross-sectional surveys conducted by the National Center for Health Statistics collect extensive demographic and health data, including biological markers, through a household interview and separate medical examination. Additional details of the NHANES survey design have been published previously.[12] NHANES data are de-identified, publicly available, and thus exempt from human subjects approval. The analysis was conducted in 2013.

Analytic Sample

For analysis of the association of BMI category at age 25 years with obesity class after age 35 years, all respondents aged 35–64 years with non-missing data for BMI at age 25 years and current BMI were included (N=13,887). For the biomarker analysis, the sample was restricted to adults aged 35–64 years who participated in the medical examination and whose BMI placed them in the obese category (n=5,626). From this group, 269 adults did not report their weight at age 25 years, 33 had no valid biomarkers, and an additional three had no education information, resulting in a final sample size of 5,321.

Measures

Exposures. BMI at age 25 years and current BMI were the key exposure variables of interest. The NHANES interview component included a weight history questionnaire asking, *How much did you weigh at age 25 years?* Recalled weight is highly correlated with measured weight across long recall periods.[13,14] Current height and weight were measured by a trained technician during the medical examination, and both current and BMI at age 25 years were calculated as BMI=weight [kg]/height [m^2]. BMI at both time points (both calculated using current height) was then categorized according to CDC guidelines ($<$18.5=underweight, 18.5–24.9=normal, 25.0–29.9=overweight, and $\geq$30=obese). For analysis, respondents were classified into three BMI trajectory groups based on their BMI at age 25 years: normal−obese, overweight−obese, and obese−obese. There were 111 respondents who reported their weight in the underweight range at age 25 years; they were included in the normal−obese group.

Outcomes. Several markers of cardiovascular and metabolic risk were considered, which were dichotomized according to conventional clinical cut-offs for high risk[15–18]: (1) systolic blood pressure $>$140 mm/Hg or diastolic blood pressure $>$90 mm/Hg; (2) high-density lipoprotein (HDL) cholesterol ($<$40 mg/dL); (3) total cholesterol $>$200 mg/dL; (4) serum triglycerides ($\geq$150 mg/dL); (5) glycosylated hemoglobin (HbA1c) $\geq$6.5% as a measure the average blood glucose over the past 2–3 months[19]; and (6) high-sensitivity C-reactive protein (hsCRP) ($>$3.0 mg/L) as a marker of inflammation. Blood pressure was taken from the mean of three readings, and serum lipids were analyzed from the fasting subsample. Details of NHANES laboratory procedures are available at cdc.gov/nchs/nhanes.htm. Modeling the biomarkers as continuous rather than dichotomous outcomes yielded similar substantive results, which are available online in the supplementary table.

Covariates. All models controlled for age (years); race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, and other); education (less than high school, high school completion, some college, or completed college or higher); smoking status (current, former, or never); and NHANES interview year.

Statistical Analysis

First, the association of BMI category at age 25 years with obesity class after age 35 years was examined, as one direct way in which early obesity can influence later health is through increasing the likelihood of more severe obesity at later ages. Ordered logistic regression was used to predict the probability of current Class I obesity (BMI$\geq$30 but $<$35); Class II obesity (BMI$\geq$35 but $<$40); and Class III obesity (BMI$\geq$40) based on BMI categories at age 25 years, adjusting for continuous age and race/ethnicity. The next aim was to examine how duration and current level of obesity jointly influence the biological risk profiles of middle-aged adults. For this aim, logistic regression models were used to predict the relative odds of high biological risk as a function of the different BMI trajectories and control variables. The normal−obese category was used as the reference in all models, so that direct comparisons could be made between those who were obese in both periods and those who are only obese in the current period. Model 1 adjusted for sociodemographic covariates, and Model 2 additionally controlled for current BMI (continuous) to account for the more severe obesity among those obese at age 25 years and currently obese. Because of observed gender differences in risk factors and possible differences in the relation of obesity to risk factors by gender,[20] all models were run separately for men and women, conducted using Stata, version 11.2 (StataCorp LP, Cary NC) and adjusted for the NHANES complex survey design. Identical models that included additional comparisons of BMI trajectories for those not currently obese to the normal−obese reference group were also conducted and are available upon request.

Results

Table 1 shows descriptive statistics for the currently obese analytic sample by weight status at age 25 years. Consistent with secular obesity trends, those who were obese at age 25 years were slightly younger (45.4 years) compared to those with normal weight at age 25 years (50.4 years). Figures 1 and 2 demonstrate the striking

Table 1. Descriptive statistics, U.S. National Health and Nutrition Examination Survey, 1999–2010 (adults aged 35–64 years, currently obese, *n*=5,321)

	BMI category at age 25 years		
	Normal weight	Overweight	Obese
BMI at age 25 years (M [SE])	22.2 (0.05)	27.2 (0.03)	34.8 (0.18)
BMI at interview (M [SE])	34.3 (0.10)	35.2 (0.13)	39.8 (0.28)
Age (M [SE])	50.4 (0.23)	48.0 (0.25)	45.4 (0.33)
Female gender	70.7	35.6	46.9
Race			
Non-Hispanic white	68.8	72.5	71.1
Non-Hispanic black	15.0	12.3	15.5
Hispanic/Mexican American	12.2	11.9	9.7
Other	4.1	3.3	3.8
Education			
Less than high school	18.7	16.4	16.5
High school	26.9	25.3	28.7
Some college	32.1	34.1	36.7
Bachelor's degree or more	22.3	24.3	18.2
Smoking			
Never smoker	49.6	46.6	52.7
Past smoker	26.4	22.8	20.7
Current smoker	20.8	28.5	25.1
Biomarkers of risk			
High blood pressure	21.4	21.3	22.8
Low HDL cholesterol	24.1	36.2	33.6
High total cholesterol	58.4	52.2	46.4
High triglycerides	45.0	47.6	41.1
High C-reactive protein	61.4	54.9	65.2
High HbA1c	10.8	12.6	17.4
n	2,424	1,898	999

Note: Values are percentages, unless otherwise noted.
HbA1c, glycosylated hemoglobin; HDL, high-density lipoprotein

association between obesity at age 25 years and the probability of severe obesity after age 35 years. Men who were already obese at age 25 years (of whom only 6.5% were Class III) had a 23.1% probability of Class III obesity after age 35 years, compared to a 5.3% probability for those who were overweight at age 25 years, and a 1.1% probability for those who were normal weight. Although more obese women already fell into Class III obesity at age 25 years (15.9%), the upward trajectories were even more dramatic; a woman who was obese at age 25 years had a 46.9% probability of Class III obesity after age 35 years compared to a 22.3% probability for a woman who was overweight at age 25 years, and 4.8% probability for a woman who was normal weight at age 25 years. Thus, individuals who are obese at age 25 years have a substantial risk of morbid obesity later in life, with health effects that may be distinct from obesity duration.

Table 2 shows results for the joint effect of long-term and severity of obesity on biological risk factors. Model 1 for each risk factor does not adjust for the severity of

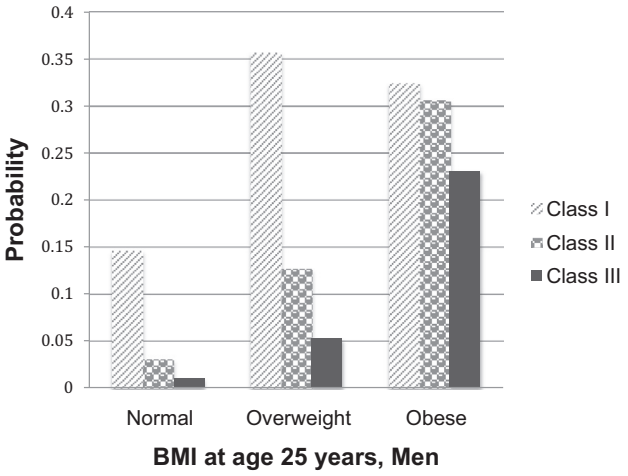

Figure 1. Probability of Class I, II, and III obesity after age 35 years by BMI at age 25 years, men
Data from the National Health and Nutrition Examination Survey (1999–2010).

current obesity. Overall, being in the obese−obese category was associated with higher odds of elevated blood pressure (OR=1.40, 95% CI=1.01, 1.95); CRP (OR=1.58, 95% CI=1.19, 2.10); and HbA1c (OR=2.20, 95% CI=1.47, 3.27) for men, compared to those in the normal−obese category (Table 2, Model 1). The odds of dyslipidemia were lower for those in the obese−obese category, but these differences were not statistically significant in men. This pattern was similar for women, with the odds for both elevated CRP and HbA1c significantly higher for the obese−obese category compared to normal−obese, but no significant risk of increased blood pressure. Women in the obese−obese category had reduced odds of high total cholesterol (OR=0.58, 95% CI=0.45, 0.75) compared to those who were in the normal−obese category. There were no elevated risks for men in the overweight−obese category relative to the normal−obese, whereas for women there was an elevated risk in this category of low HDL (OR=1.34, 95% CI=1.01, 1.79) and high HbA1c (OR=1.57, 95% CI=1.12, 2.20), but a lower risk of elevated total cholesterol (OR=0.74, 95% CI=0.59, 0.94) and CRP (OR=0.75, 95% CI=0.57, 0.99).

Model 2 added controls for current continuous BMI to account for the fact that individuals obese at age 25 years are likely to be heavier than their currently obese counterparts who were not obese at this age. The results strongly suggest that the increased odds of high CRP in Model 1 were due to the more severe current obesity of those who were obese at 25 years rather than long-term obesity. Once the severity of current obesity was controlled for, the odds of high CRP associated with current obesity were similar for those who were obese at age 25 years compared to normal weight at this age (men: OR=0.79, 95% CI=0.59, 1.06; women: OR=0.77, 95% CI=0.53, 1.13). The higher odds for elevated HbA1c were also greatly reduced following adjustment for obesity severity, remaining significantly elevated only for men in the obese−obese category compared to normal−obese (OR=1.55, 95% CI=1.01, 2.37), with a similar OR not reaching statistical significance for women (OR=1.57, 95% CI=0.98, 2.50). Of note, after controlling for the severity of current obesity, women who became obese more recently remained at higher risk for both elevated total cholesterol and triglycerides compared to those who were obese in both periods. Although certain associations were statistically significant for women versus men or vice versa, the overall pattern and magnitude of coefficients were similar for men and women (Table 2), suggesting that the association between long-term obesity and biological risk does not differ substantively by gender.

Discussion

The health implications of long-term obesity are of increasing medical and public health importance. The current findings suggest that the biological risks of longer-term obesity are primarily due to the risk of more severe obesity later in life among those obese early in life, rather than the impact of long-term obesity per se. This is good news in some respects, as overweight and obese young adults who can prevent additional weight gain can expect their biological risk factors to be no worse than those who reach the same level of BMI later in life. The bad news, in turn, is that maintaining a stable level of

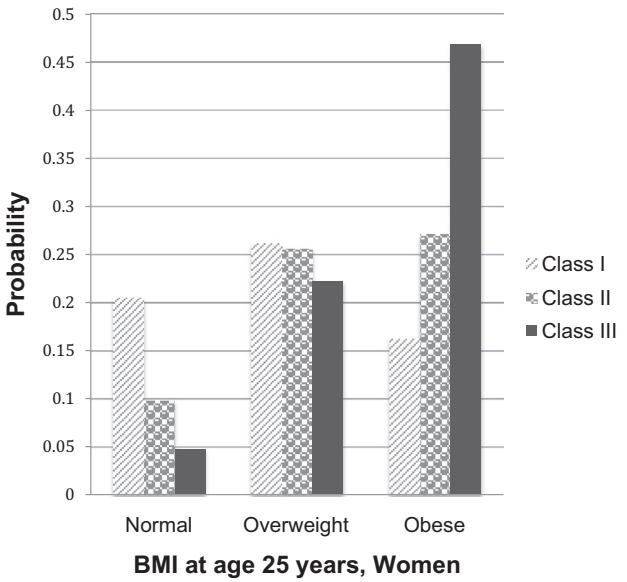

Figure 2. Probability of Class I, II, and III obesity after age 35 years by BMI at age 25 years, women
Data from the National Health and Nutrition Examination Survey (1999–2010).

Table 2. Logistic models of elevated biomarker risk, exponentiated coefficients (95% CIs)

	Blood pressure		(Low) HDL cholesterol		Total cholesterol		Triglycerides		C-reactive protein		HbA1c%	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Men												
Normal—obese	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Over—obese	1.06	1.00	0.90	0.87	0.86	0.89	1.05	1.01	1.22	1.04	1.15	1.05
	(0.81, 1.40)	(0.76, 1.32)	(0.70, 1.16)	(0.67, 1.13)	(0.67, 1.10)	(0.69, 1.14)	(0.70, 1.56)	(0.67, 1.51)	(0.96, 1.56)	(0.81, 1.33)	(0.80, 1.65)	(0.72, 1.54)
Obese—obese	**1.40***	1.10	0.92	0.80	0.71	0.82	0.85	0.73	**1.58****	0.79	**2.20*****	**1.55***
	(1.01, 1.95)	(0.75, 1.60)	(0.67, 1.26)	(0.58, 1.13)	(0.50, 1.00)	(0.58, 1.16)	(0.56, 1.27)	(0.47, 1.14)	**(1.19, 2.10)**	(0.59, 1.06)	**(1.47, 3.27)**	**(1.01, 2.37)**
BMI, cont.		**1.05****		**1.03***		**0.97***		1.03		**1.16*****		**1.07*****
		(1.02, 1.08)		**(1.00, 1.05)**		**(0.95, 1.00)**		(0.99, 1.06)		**(1.13, 1.20)**		**(1.04, 1.10)**
Women												
Normal—obese	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Over—obese	1.22	1.07	**1.34***	1.27	**0.74***	**0.76***	0.93	0.87	1.03	**0.75***	**1.57****	1.25
	(0.90, 1.64)	(0.77, 1.47)	**(1.01, 1.79)**	(0.94, 1.71)	**(0.59, 0.94)**	**(0.59, 0.98)**	(0.67, 1.29)	(0.62, 1.22)	(0.80, 1.32)	**(0.57, 0.99)**	**(1.12, 2.20)**	(0.87,1.80)
Obese—obese	1.29	0.93	1.24	1.06	**0.58*****	**0.61*****	0.74	**0.61***	**1.76****	0.77	**2.59*****	1.57
	(0.87, 1.92)	(0.62, 1.39)	(0.86, 1.80)	(0.69, 1.63)	**(0.45, 0.75)**	**(0.47, 0.80)**	(0.48, 1.15)	**(0.38, 0.97)**	**(1.23, 2.52)**	(0.52, 1.12)	**(1.81, 3.73)**	(0.98, 2.50)
BMI, cont.		**1.05*****		1.02		0.99		**1.03***		**1.16*****		**1.08*****
		(1.02, 1.08)		(1.00, 1.05)		(0.97, 1.01)		**(1.00, 1.06)**		**(1.13, 1.19)**		**(1.05, 1.11)**

Note: Boldface indicates statistical significance. All models control for age, race/ethnicity, educational attainment, smoking, and National Health and Nutrition Examination Survey wave.
*$p<0.05$, **$p<0.01$, ***$p<0.001$.
HbA1c, glycosylated hemoglobin; HDL, high-density lipoprotein

obesity from a young age is not the norm, and being obese at age 25 years places individuals at risk of much more severe levels of obesity later in life compared to those who are normal weight at age 25 years.

Only a few previous papers have examined the association of longer-term obesity and biomarkers of cardiovascular and metabolic risk, and to our knowledge the present study is the first using the most recent nationally representative NHANES surveys. Using NHANES III (1988–1994) data, Janssen et al.[21] found that overweight duration >10 years compared to ≤10 years was associated with increased risk of various markers of metabolic syndrome in both men and women in the U.S. Consistent with the current findings, the associations were largely eliminated with controls for the severity of current overweight. Recent work has also identified an association between obesity duration and incident type II diabetes in both the Framingham cohort and Coronary Artery Risk Development in Young Adults (CARDIA) cohorts.[22,23] This is partially consistent with the current findings for HbA1c, in which much of the association of duration and HbA1c was attenuated with controls for the severity of BMI, but the odds of elevated HbA1c remained slightly higher for longer-term obese men. Recent results from CARDIA indicated a significant association between duration of obesity and coronary calcification in middle age.[24] Like most previous studies of obesity duration, the CARDIA study used 0 years of duration as the reference category, making it difficult to determine whether different durations among obese individuals are statistically distinguishable from one another, and in most cases the confidence intervals for 1–5 years to >20 years of duration overlapped. To the extent that these results reflect a real association of duration and coronary calcification, it may suggest that duration of obesity could affect the development of atherosclerosis through pathways beyond those measured by traditional cardiovascular risk factors.

The results found here for lipids differed from the other risk factors, with obesity at age 25 years associated with lower levels of total cholesterol and triglycerides for women, a result that remained with adjustment for obesity severity. As one possibility is that individuals who became obese earlier in their lives were more likely to be identified to be at risk and prescribed lipid-lowering medications, identical models with controls for lipid-lowering medication use were run as a sensitivity analysis, which did not alter these findings. The findings are consistent with a recent study of the 1958 British Birth Cohort, which found no evidence of worse lipid profiles with longer duration of obesity, and that the association of current BMI with lipids was stronger for those with the lowest childhood BMI.[25] Recent examination of four childhood cohorts from the U.S. and Europe also reported that associations of childhood obesity with type 2 diabetes, dyslipidemia, and high-risk intima-media thickness were not significant once adult obesity was taken into account, although an elevated risk of hypertension remained.[26] The current results that longer-term obesity is associated with lower risk of elevated lipids compared to the same level of obesity acquired more recently are also consistent with earlier findings from NHANES, which showed that weight gain in adulthood compared to weight stability was a significant risk factor for metabolic syndrome in U.S. adults.[27] Taken together, these findings may suggest that more recent weight gain is a more salient risk factor for dyslipidemia than long-term obesity.

To our knowledge, this study is the first to examine the association between long-term obesity and inflammation. A significant portion of circulating CRP in obese individuals is produced by adipose tissue,[28] but whether long-term obesity induces higher circulating levels above current adiposity has not been tested. This study found strong associations between obesity at age 25 years and risk of elevated CRP, although the association was entirely accounted for by adjustment for severity of current obesity. This confirms strong associations between current adiposity and high CRP but does not support the idea that long-term obesity itself contributes to systemic inflammation through dysregulation of inflammatory mechanisms.

A recent study[29] found a dose-response association between duration of obesity and mortality using data from the Framingham cohort. In contrast to the current results, the association between obesity duration and mortality increased after controlling for current BMI, a result that the authors speculate is due to the nonlinear relationship between BMI and mortality, and particularly the fact that in their sample higher current BMI was associated with lower mortality.[29] The average age of obesity onset in the Framingham sample was 50 years, whereas more recent cohorts are experiencing much earlier onset of obesity. By examining the impact of obesity that arises by age 25 years, the current study may better characterize the biological risk of these more recent cohorts.

There are several limitations to the current analysis. Weight at age 25 years was self-reported, and thus may have been subject to recall bias, particularly if those with higher risks were more likely to distort or misremember their weight at this age. Nonetheless, recalled weight is highly correlated with measured weight.[13,14] Another limitation is that the interval between the weight at age 25 years and current weight captured in this cross-sectional survey varied by current age of the respondent. Sensitivity analyses were run, stratifying by ages 35–49

years and 50–64 years to look for evidence of different associations of BMI trajectories on risk by age, and found similar substantive results (available on request).

In summary, this study adds to growing evidence that in terms of traditional cardiovascular, inflammatory, and metabolic risk, obesity duration confers little additional risk beyond the current level of attained weight. Although this finding could be considered good news in light of the dramatic increase in the probability of being obese by age 25 years in recent U.S. cohorts,[3] it should be remembered that obesity at this age remains hazardous because of its tracking with even more severe obesity later in life. Moreover, duration of obesity may still have important implications for mobility and musculoskeletal disease; research questions that should be investigated. Prevention of weight gain at all ages should thus be a clinical and public health priority, but current attained weight rather than duration may be the best reflection of current metabolic and cardiovascular risk.[15-18]

The NHANES data used in this study are publicly available from the National Center for Health Statistics.

No financial disclosures were reported by the authors of this paper.

References

1. Flegal KM, Carroll MD, Kit BK, Ogden CL. Prevalence of obesity and trends in the distribution of body mass index among us adults, 1999–2010. JAMA 2012;307(5):491–7.
2. Alley DE, Chang VW. The changing relationship of obesity and disability, 1988–2004. JAMA 2007;298(17):2020–7.
3. Reither EN, Hauser RM, Yang Y. Do birth cohorts matter? Age-period-cohort analyses of the obesity epidemic in the U.S. Soc Sci Med 2009;69(10):1439–48.
4. Gregg EW, Cheng YJ, Cadwell BL, et al. Secular trends in cardiovascular disease risk factors according to body mass index in U.S. adults. JAMA 2005;293(15):1868–74.
5. Zajacova A, Dowd JB, Burgard SA. Overweight adults may have the lowest mortality, do they have the best health? Am J Epidemiol 2011;173(4):430–7.
6. Abdullah A, Wolfe R, Mannan H, Stoelwinder JU, Stevenson C, Peeters A. Epidemiologic merit of obese-years, the combination of degree and duration of obesity. Am J Epidemiol 2012;176(2):99–107.
7. Alley D, Lloyd J, Shaffer T, Stuart B. Changes in the association between body mass index and medicare costs, 1997—2006. Arch Intern Med 2012;172(3):277–8.
8. Felber JP. Pathways from obesity to diabetes. Int J Obes Relat Metab Disord 2002;26(2S):S39–S45.
9. Marinou K, Tousoulis D, Antonopoulos AS, Stefanadi E, Stefanadis C. Obesity and cardiovascular disease: from pathophysiology to risk stratification. Int J Cardiol 2010;138(1):3–8.
10. Nguyen NT, Magno CP, Lane KT, Hinojosa MW, Lane JS. Association of hypertension, diabetes, dyslipidemia, and metabolic syndrome with obesity: findings from the National Health and Nutrition Examination Survey, 1999 to 2004. J Am Coll Surg 2008;207(6):928–34.
11. NHANES 2009–2010 public release file documentation. Atlanta GA: CDC, 2011.
12. CDC. National Center for Health Statistics. NHANES 1999—2000 public release file documentation. Atlanta GA: CDC, National Center for Health Statistics, 2002.
13. Norgan NG, Cameron N. The accuracy of body weight and height recall in middle-aged men. Int J Obes Relat Metab Disord 2000;24(12):1695–8.
14. Perry GS, Byers TE, Mokdad AH, Serdula MK, Williamson DF. The validity of self-reports of past body weights by U.S. adults. Epidemiology 1995;6(1):61–6.
15. Third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III): final peport. Circulation 2002;106(25):3143.
16. Lenfant C, Chobanian AV, Jones DW, Roccella EJ. Seventh report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7): resetting the hypertension sails. Circulation 2003;107(24):2993–4.
17. American Diabetes Association. Standards of medical care in diabetes, 2010. Diabetes Care 2010;33(1S):S11–S61.
18. Ridker PM. C-reactive protein and the prediction of cardiovascular events among those at intermediate risk: moving an inflammatory hypothesis toward consensus. J Am Coll Cardiol 2007;49(21):2129–38.
19. Selvin E, Steffes MW, Zhu H, et al. Glycated hemoglobin, diabetes, and cardiovascular risk in nondiabetic adults. N Engl J Med 2010;362(9):800–11.
20. Vaccarino V, Badimon L, Corti R, et al. Ischaemic heart disease in women: are there sex differences in pathophysiology and risk factors?: Position paper from the Working Group on Coronary Pathophysiology and Microcirculation of the European Society of Cardiology. Cardiovasc Res 2011;90(1):9–17.
21. Janssen I, Katzmarzyk PT, Ross R. Duration of overweight and metabolic health risk in American men and women. Ann Epidemiol 2004;14(8):585–91.
22. Abdullah A, Stoelwinder J, Shortreed S, et al. The duration of obesity and the risk of type 2 diabetes. Public Health Nut 2011;14(01):119–26.
23. Reis JP, Hankinson AL, Loria CM, et al. Duration of abdominal obesity beginning in young adulthood and incident diabetes through middle age: the CARDIA study. Diabetes Care 2013;36(5):1241–7.
24. Reis JP, Loria CM, Lewis CE, et al. Association between duration of overall and abdominal obesity beginning in young adulthood and coronary artery calcification in middle age. JAMA 2013;310(3):280–8.
25. Pinto Pereira SM, Power C. Life course body mass index, birthweight and lipid levels in mid-adulthood: a nationwide birth cohort study. Eur Heart J 2013;34(16):1215–24.
26. Juonala M, Magnussen CG, Berenson GS, et al. Childhood adiposity, adult adiposity, and cardiovascular risk factors. N Engl J Med 2011;365(20):1876–85.
27. Alley DE, Chang VW. Metabolic syndrome and weight gain in adulthood. J Gerontol A Biol Sci Med Sci 2010;65A(1):111–7.
28. Eiriksdottir G, Smith AV, Aspelund T, et al. The interaction of adiposity with the CRP gene affects CRP levels: Age, Gene/Environment Susceptibilty–Reykjavik study. Int J Obes 2009;33(2):267–72.
29. Abdullah A, Wolfe R, Stoelwinder JU, et al. The number of years lived with obesity and the risk of all-cause and cause-specific mortality. Int J Epidemiol 2011;40(4):985–96.

Appendix

Supplementary data

Supplementary data associated with this article can be found at http://dx.doi.org/10.1016/j.amepre.2014.01.016.