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Number of children and the risk of obesity in older women

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Abstract

Background. The purpose of this study is to examine number of live births, other reproductive factors, and health behaviors in relation to obesity risk among older women.

Methods. Data were collected during in-person and telephone-based interviews from a population-based cohort in Utah.

Results. A total of 2,035 women aged 66–102 are included in this report. Overall, 403 (20%) older women were determined to be obese. The rates of obesity were significantly higher with increasing numbers of children, demonstrating a dose–response relationship ($P < 0.05$). After adjustment for age, education, marital status, BMI at age 18, use of oral contraceptives, hysterectomy status, physical activity, current use of hormone therapy, and age at menarche, the risk of obesity increased 11% with each additional live birth. In additional analyses that excluded nulliparous women, after adjusting for cumulative months of breast-feeding, the risk of obesity increased 7% with each live birth. In this cohort of older women, we found higher rates of obesity with increasing number of children that was independent of socioeconomic status and other confounding factors.

Conclusions. In a cohort of older women, higher rates of obesity were associated with increasing number of children that was independent of socioeconomic status and other confounding factors.

Keywords: Obesity; Parity; Older women

Introduction

Obesity is a significant health problem among adult women in the United States [1]. In older women, obesity has been linked to the development of type 2 diabetes mellitus [2], cardiovascular disease [3], dementia [4], urinary incontinence [5], and cancers [6,7]. Understanding the role reproductive history plays in the development of weight gain has implications for preventing obesity in later life.

Having children is frequently cited by younger women as a cause of obesity [8]. Several mechanisms have been proposed to explain the association of number of children and obesity among women, such as insulin resistance associated with pregnancy [3,9,10], hormonal alterations secondary to fewer ovulatory cycles [11], increased glucocorticoid activity [12], and the excess deposit of fat tissue that accumulates, preferentially in the femoral area, during pregnancy [13]. Many of these physiological changes associated with pregnancy have been shown to persist years after childbearing [14]. In addition to these physiological changes, motherhood may also be associated with changes in diet and physical activity to accommodate living with small children.

Several investigators have found an association between reproductive history and obesity among older women [15–18]. A population study from Finland found that number of children among women aged 25–84 was closely related to the prevalence of obesity independent of marital status, occupation, and smoking habits [15]. In a more recent study from Sweden, number of children was also associated with

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obesity among 5,464 women ages 45–73 [16]. In the Nurse's Health Study, an increase in BMI was found with increasing number of children among women aged 42–67 [17]. The Rancho Bernardo Study also found an association with number of children and obesity among women many years after childbearing [18].

Investigators have proposed socioeconomic status as a significant confounder in the number of children and obesity relationship [19]. Women with less education or of lower social class are at higher risk for obesity [20], and this effect may be related to personal habits such as excessive caloric intake as well as number of children.

We aimed to test the hypothesis that increasing number of children is associated with increasing risk for obesity among older women independent of socioeconomic status, other reproductive factors, and important health behaviors. The Cache County Study on Memory, Health, and Aging provides an excellent cohort to examine this research question.

Methods

Sampling frame and subjects

Cache County lies in a large mountain valley in northern Utah that extends to the Idaho border. The genetic structure of this Utah population is broadly representative of other U.S. populations of northern European ancestry. More than 90% of the elderly members of this population are members of The Church of Jesus Christ of the Latter-day Saints (LDS), and unique features of this group include low prevalence of alcohol use and smoking and high rates of parity. According to the 1990 census, Cache County had the greatest mean female life expectancy of all U.S. counties [21].

We used a Medicare enrollee list provided by the Health Care Financing Administration to identify permanent county residents 65 years and older as of January 1, 1995. We invited 3,260 women to participate and enrolled 2,928 women (90%) over an 18-month period. Of these, 145 resided in a nursing home or assisted living facility at the time of enrollment.

Participants received a baseline cognitive screening examination and completed a face-to-face interview that included an assessment of lifetime and current medical conditions. The present analyses excluded 275 women with prevalent or possible dementia and another 393 women who died, refused to continue participation, or permanently moved away from the area. We also excluded women with proxy-reported baseline information (n = 38) and those lacking data on height and weight (n = 103). Participants were also administered a woman's health questionnaire over the telephone (n = 2,090) or as a face-to-face interview (n = 132), which inquired about reproductive factors. An additional 84 women were excluded because of incomplete women's health information. A total of 2,035 women are included in this report. These subjects were 99% Caucasian.

Interview procedure

All contact with participants employed procedures previously approved by the institutional review boards of Duke University Medical Center, Durham, NC, and Utah State University, Logan. Each participant was visited at her residence, and after providing written, informed consent, was given an extended interview that included sections on demographic variables, medical history (including a detailed accounting of medication history), and history of smoking and alcohol use.

During the in-person interview, all subjects reported their current height and weight and were asked to recall their height and weight at age 18. Since measures of weight alone do not consider the amount of body tissue per unit of height, we calculated a BMI, a better measure of body habitus. We used the criteria of BMI 30 kg/m^2 or greater to define obesity [22].

Because social support has been shown to be associated with other serious health conditions [23], we asked "Do you feel that you have enough contacts with other people or do you wish that you had more?"

Subsequent to the in-person baseline interview, we conducted a woman's health telephone interview to obtain a detailed reproductive history. Interviewers inquired about the number of pregnancies and the number of live births. We focused our analyses on number of live births because pregnancy losses are less likely to be fully recalled than are number of live births [24]. In particular, many pregnancy losses are clinically unrecognized and therefore not recalled. We recorded age at first live birth and categorized age at first live birth as less than 20 years or 20 years and older. We collected information on whether mothers breast-fed their infant and calculated the cumulative number of months that they breast-fed their children. We asked about ever use of oral contraceptives (yes/no), ever use of hormone therapy (HT), and current use of HT (yes/no). We asked about the age of menarche, the age at menopause, and whether they had a hysterectomy, oophorectomy, or both.

Using a supplemental mailed questionnaire, we asked about physical activity. We asked "About how often do you take part in vigorous physical activity including jogging, tennis, racquetball or squash, lap swimming, aerobics, vigorous bicycling, skiing, hiking, hunting, or other vigorous sports." The response categories were categorized to yes ("usually every day" or "2–6 times a week") and no ("about once a week", "few times a month", or "rarely or never").

Statistical analysis

Prevalence and risks factors for obesity were compared to number of live births. We used multiple logistic regression models to estimate the probability of obesity as a function of age and number of live births and several other important covariates. Likelihood ratio chi^2 tests were used in these models to evaluate the significance of improved fit with introduction of additional terms.

Results

The sample consisted of 2,035 women with an average age of 77 years (range from 66 to 102 years) and a mean BMI of 27. Overall, 20% of women were obese (BMI ≥ 30). The number of live births per woman ranged from 0 to 14, with a mean of four live births. Both the mean BMI as well as the proportion who were obese increased with increasing number of children ($P < 0.05$).

As shown in Table 1, the women who were obese were significantly younger (74.5 vs. 77.2 years), more likely to have started menarche before age 12 (18% vs. 10%), more likely to have been obese at age 18 (2% vs. 0%), more likely to have ever used oral contraceptives (18% vs. 13%), less likely to have participated in vigorous physical activity (7% vs. 13%), and less likely to have reported drinking any alcohol (1% vs. 3%) ($P < 0.05$). Of note, women who breast-fed or reported more cumulative months of breast-feeding were not significantly different with regards to rates of obesity.

Table 2 demonstrates the linear dose–response effect of increasing number of children on obesity. In comparison to the reference group of no live births, women with three through eight or more live births were significantly more likely to be obese. As shown in Table 3, after adjustment for age, education, marital status, BMI at age 18, use of oral contraceptives, hysterectomy status, physical activity, current use of HT, and age at menarche in multivariate logistic regression models, the risk of obesity increased 11% with each additional live birth. In additional analyses that excluded nulliparous women (see Table 4), after adjusting for cumulative months of breast-feeding, the risk of obesity increased 7% with each live birth.

Table 1
Characteristics associated with obesity among older women

Variable	Percentage obese, $N = 403$	Percentage not obese, $N = 1632$	P value
Sociodemographic			
Age (years) [SD]	74.5 [5.4]	77.2 [6.8]	<0.001
12 or more years education	86	87	0.5
Enough social support	87	89	0.2
Ever worked outside of home	86	85	0.6
Currently married	58	56	0.3
Reproductive			
Menarche <12 years of age	18	10	<0.001
Menopause <45 years of age	25	26	0.8
Hysterectomy	47	44	0.3
Bilateral oophorectomy	24	24	0.9
Ever use of HT	62	60	0.4
Current use HT	31	34	0.2
Ever use of oral contraceptives	18	13	0.02
Age at first live birth <20 years	18	15	0.2
Parity (number of live births)	4.6 [2.1]	3.9 [2.2]	<0.001
Breast-fed ever	75	76	0.7
Cumulative months breast-feeding	14.7 [17]	13.4 [16]	0.2
Health behaviors			
Vigorous physical activity	7	13	0.002
Smoking, ever	8	7	0.5
Regular alcohol use	1	3	0.01
Obesity at age 18 (BMI ≥ 30)	2	0	<0.001

Table 2
Logistic regression with unadjusted odds ratios for number of live births as a nominal variable with obesity as the outcome variable[a]

Independent variable	Odds ratio	95% Confidence interval
No live births	1.00	
1 live birth	2.2	0.75–6.4
2 live births	2.3	0.92–5.7
3 live births	2.6	1.1–6.4
4 live births	3.4	1.4–8.2
5 live births	2.8	1.2–6.8
6 live births	3.2	1.3–7.8
7 live births	5.2	2.1–13.4
8 or more live births	7.1	2.8–17.8

[a] $n = 1634$ because we were missing physical activity for 401 women who did not complete the supplemental mailed questionnaire.

Discussion

The major new finding in this study was that a dose–response relationship exists between the number of children and obesity among older women. Adjustment for sociodemographic variables, reproductive factors, and other predictors of obesity had little effect on the estimates of the odds ratios. Our study supports the observation of Sheldon (1949) that "maternal obesity", the development of obesity in women after having a baby, especially among multiparous women, may have a long-lasting and detrimental effect on health of older women [25].

Our results suggest that pregnant women, especially those who have already had several children, are an impor-

Table 3
Logistic regression model with obesity as outcome[a]

Independent variable	Odds ratio	95% Confidence interval
Age, years	0.92	0.90–0.94
Parity, number of live births	1.11	1.05–1.18
Education (12 or more years vs. less)	0.93	0.62–1.39
Currently married	0.68	0.52–0.91
BMI at age 18	1.20	1.14–1.27
Use of oral contraceptives	1.21	0.86–1.7
Current use of HRT	0.64	0.47–0.86
Age at menarche <12	0.69	0.48–0.99
Had hysterectomy	1.18	0.90–1.54
Moderate physical activity	0.46	0.28–0.74

[a] n = 1634 because we were missing physical activity for 401 women who did not complete the supplemental mailed questionnaire.

tant high-risk population for obesity in later life. On average, women with seven or more children were 4 kg/m^2 heavier than women with no children (data are not shown). This translates to approximately 18 kg or 40 lb excess weight [26]. Prevention of weight gain among women with multiple children may have a significant public health impact. Specific counseling concerning the primary prevention of weight gain and obesity—promoting physical activity, healthy eating, and the maintenance of appropriate body weight—may be especially prudent for women during their first pregnancy.

The current study differs from previous studies in several ways. First, this unique cohort of older women is fairly homogeneous with regards to religious beliefs, education, and socioeconomic status, yet had a high mean number of children with considerable variation. Second, we controlled for other reproductive factors that have been reported to be related to obesity such as age at menarche, hysterectomy status, oral contraceptive use, and current use of HT. Third, we collected information on breast-feeding, which has not been studied adequately [24]. Fourth, this multiparous cohort provided more power to detect small associations, particularly in women with seven or more pregnancies (n = 210), than did previous studies. Finally, this cohort provides an opportunity to study this issue over a broad range of elderly women.

It remains unclear whether the association between number of live births and obesity is primarily mediated through physiological or behavioral mechanisms, or both. Pregnancy may affect the risk of obesity through insulin resistance. Insulin resistance is greater in multigravid women than in women with fewer pregnancies, perhaps as a result of altered glucose metabolism during pregnancy or post-pregnancy alterations in the amount and distribution of body fat [10]. In our study, we adjusted our analyses for both BMI at age 18 and physical activity, which are related to insulin resistance. If insulin resistance is thought to mediate the multigravid effect on obesity, future research might investigate the role of agents that decrease insulin resistance, for example, biguanide medications in reducing both obesity and type 2 diabetes mellitus risk among multigravid older women.

One potential mechanism for the development of obesity is that pregnancy and child rearing have an impact on levels of stress and social support. Stress has been shown to lead to poor health outcomes, but higher social support may counteract the effect of stress [27]. The specific role of many children in the social network is not known. Harris et al. [12] found mothers with high levels of social support had significantly higher mean BMI. We measured social support in our cohort and found a positive association between parity and social support (data are not shown) but no relationship between social support and obesity.

Previous studies have focused on the number of pregnancies that have included spontaneous abortions. If the physiological mechanism mediating the relationship between pregnancy and obesity is hormone- or insulin-resistance-related, then a physiological effect should be more prominent in full-term pregnancies (live births) compared to short-term spontaneous abortions. Although the correlation between gravidity and parity is high, the two factors may not be interchangeable if pregnancy losses affect obesity risk. We found that the number of pregnancies was also related to obesity (data are not shown).

Another potential mechanism is the effect of parity on number of ovulatory cycles. The more pregnancies and breast-feeding a woman experiences, the fewer ovulatory cycles she will have. A decrease in ovulatory cycles may result in hormone alterations (e.g., a decrease in endogenous estrogen) causing an increase in BMI and obesity, and changes in body fat distribution [8]. If this is the main physiological mechanism of obesity, we would expect to see an independent effect of oral contraceptive use, because these medications function by inhibiting ovulation. We observed a higher rate of oral contraceptive use in obese women compared to non-obese women. Since oral contraceptives were not available until the mid 1960s, many women in this age cohort had little opportunity to use oral

Table 4
Logistic regression model with obesity as outcome[a]

Independent variable	Odds ratio	95% Confidence interval
Age, years	0.92	0.90–0.94
Parity, number of live births	1.07	1.01–1.15
Cumulative months breast-feeding	1.00	1.00–1.01
Education (12 or more years vs. less)	0.94	0.63–1.41
Currently married	0.68	0.51–0.91
BMI at age 18	1.20	1.14–1.26
Use of oral contraceptives	1.22	0.86–1.72
Current use of HRT	0.60	0.44–0.81
Age at menarche <12	0.74	0.51–1.07
Had hysterectomy	1.21	0.92–1.59
Moderate physical activity	0.46	0.28–0.75

[a] n = 1547 because we excluded 87 women with no live births and 401 women with incomplete information on physical activity from the supplemental mailed questionnaire.

contraceptive during their reproductive years. Presumably, the younger women in our cohort, aged 66–74 years, may have had access to oral contraceptives during the end of their reproductive career. Oral contraceptive users tended to have larger families in this population. Their religion may support oral contraceptive use if they already had a large number of children or there was a medical reason to prevent future pregnancies.

Several investigators have proposed that educational level confounds the relationship of high parity and early age at first live birth and risk for obesity [3]. Women with less education may be poorer and at higher risk for obesity because of their health behaviors (e.g., excessive caloric intake and less physical activity). In this homogenous population of older women, education was not associated with obesity.

We did not find a relationship between a history of breast-feeding and obesity in older women. Although in analyses that include women with at least one live birth, we did find cumulative months of breast-feeding attenuated the parity effect on obesity. Overall, we noted a positive relationship between cumulative months of breast-feeding and rates of obesity. Although it is physiologically reasonable that lactating women should experience greater weight loss than nonlactating women because of the increase in energy expenditure required for breast milk production, most studies have not found a difference in weight loss among lactating and nonlactating women [28].

We also found that a younger age at menarche was associated with obesity. One would expect obesity among teenagers to be associated with a younger age at menarche because of the higher estrogen levels observed in obesity, but it is somewhat surprising to observe these effects among older women. In another study, the relationship of body composition measures (e.g., percent of lean mass) has been linked to timing of menarche and menstrual cycle length [29].

Some limitations of this study should be noted. We used self-reported weight and height data. Among women in the Nurse's Health Study, self-reported and measured weight were highly correlated (0.96) [30]. Future prospective studies should include actual measures of body composition such as waist-to-hip ratio. The population is not diverse, 99% are white and 93% are of LDS faith. Women may not accurately report alcohol and smoking behaviors because of social norms. There may have been some misclassification in age at menarche and BMI at age 18 because of inaccurate recall among some women of an event that occurred 50 years earlier.

Future research among older women should explore the effect of number of children on cardiovascular disease, type 2 diabetes mellitus, and cancers adjusting for obesity to further understand the mechanism by which an association between number of children and health risk may be mediated. In summary, the mechanism by which an association between number of children and obesity may be mediated remains unclear. Repeated full-term pregnancies may result in physiological changes that increase the risk for obesity. On the other hand, the mechanism for obesity may be related to behavioral changes in diet and physical activity. Although it is doubtful whether knowledge of this increased risk for obesity could influence the number of children women choose to have, these findings may be helpful in understanding the physiological mechanism of obesity in women. Multiparous women may need additional information on how to modify their risk for obesity through medications and health behaviors. Future research should explore behavioral effects of having children on the risk for obesity in both mothers and fathers.

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References

[1] Flegal LM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among U.S. adults, 1999–2000. JAMA 2002;288: 1723–7.

[2] Carey VJ, Walters EE, Colditz GA, et al. Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women. Am J Epidemiol 1997;145:614–9.

[3] Ness RB, Harris T, Cobb J, et al. Number of pregnancies and the subsequent risk of cardiovascular disease. N Engl J Med 1993; 328:1528–33.

[4] Gustafson D, Rothenberg E, Blennow K, et al. An 18-year follow-up of overweight and risk for Alzheimer disease. Arch Intern Med 2003;163:1524–8.

[5] Sustersic O, Kralj B. The influence of obesity, constitution and physical work on the phenomenon of urinary incontinence in women. Int Urogynecol J Pelvic Floor Dysfunct 1998;9:140–4.

[6] Shoff SM, Newcomb PA. Diabetes, body size, and risk of endometrial cancer. Am J Epidemiol 1998;148:234–40.

[7] Stoll BA. Timing of weight gain in relation to breast cancer risk. Ann Oncol 1995;6:245–8.

[8] Rossner S. Pregnancy, weight cycling and weight gain in obesity. Int J Obes Relat Metab Disord 1992;16:145–7.

[9] Godsland IF. The influence of female sex steroids on glucose metabolism and insulin action. J Intern Med 1996;738:1–60.

[10] Kritz-Silverstein D, Barrett-Connor E, Wingard DL. The effect of parity on the later development of non-insulin dependent diabetes mellitus or impaired glucose tolerance. N Engl J Med 1989;321: 1214–9.

[11] Gunderson EP, Abrams B. Epidemiology of gestational weight gain and body weight changes after pregnancy. Epidemiol Rev 2000; 22:261–74.

[12] Harris HE, Ellison GT, Richter LM, et al. Are overweight women at increased risk of obesity following pregnancy? Br J Nutr 1998;79: 489–94.

[13] Kaye SA, Folsom AR, Prineas RJ, et al. The association of body fat distribution with lifestyle and reproductive factors in a population study of postmenopausal women. Int J Obes 1990;14:583–91.

[14] den Tonkelaar I, Seidell JC, van Noord PA, et al. Fat distribution in relation to age, degree of obesity, smoking habits, parity and estrogen use: a cross-sectional study in 11,825 Dutch women participating in the DOM-project. Int J Obes 1990;14:753–61.

[15] Heliovaara M, Aromaa A. Parity and obesity. J Epidemiol Community Health 1981;35:197–9.

[16] Lahmann PH, Lissner L, Gullberg B, Berglund G. Sociodemographic factors associated with long-term weight gain, current body fatness and central adiposity in Swedish women. Int J Obes Relat Metab Disord 2000;24:685–94.

[17] Manson JE, Rimm EB, Colditz GA, et al. Parity and incidence of non-insulin dependent diabetes mellitus. Am J Med 1992;93:13–8.

[18] Barrett-Connor E. Sex differences in coronary heart disease. Why are women so superior? The 1995 Ancel keys lecture. Circulation 1997;95:252–64.

[19] Grinblatt JA. Number of pregnancies and risk of cardiovascular disease. N Engl J Med 1993;329:1893 [discussion 1894–5].

[20] Wardle J, Waller J, Jarvis MJ. Sex differences in the association of socioeconomic status with obesity. Am J Public Health 2002;92: 1299–304.

[21] Norton MC, Breitner JC, Welsh KA, et al. Characteristics of non-responders in a community survey of the elderly. J Am Geriatr Soc 1994;42:1252–6.

[22] NHLBI Obesity Education Initiative Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—The Evidence Report. Obes Res 1998;6:51s–209s.

[23] Broadhead WE, Kaplan BH, James SA, et al. The epidemiologic evidence for a relationship between social support and health. Am J Epidemiol 1983;117:521–37.

[24] Ness RB, Schotland HM, Flegal KM, Shofer FS. Reproductive history and coronary heart disease risk in women. Epidemiol Rev 1994;16:298–314.

[25] Sheldon JH. Maternal obesity. Lancet 1949;6585:869–73.

[26] Ley C, Lees B, Stevenson J. Sex- and menopause-associated changes in body-fat distribution. Am J Clin Nutr 1992;55:950–4.

[27] D'Elio MA, Ness RB, Matthews KA, Kuller LH. Are life stress and social support related to parity in women? Behav Med 1997;23:87–94.

[28] Potter S, Hannum S, McFarlin B, et al. Does infant feeding method influence maternal postpartum weight loss? J Am Diet Assoc 1991;91:441–6.

[29] Symons JP, Sowers MF, Harlow SD. Relationship of body composition measures and menstrual cycle length. Ann Hum Biol 1997; 24:107–16.

[30] Rimm EB, Stampfer MJ, Colditz GA, et al. Validity of self-reported waist and hip circumferences in men and women. Epidemiology 1990;1:466–73.