

Association between atopic dermatitis and obesity in adulthood

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Summary

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Background Obesity in early childhood is associated with increased risk for and severity of atopic dermatitis (AD).

Objective To determine whether obesity in adulthood is associated with risk of AD. **Methods** This was a retrospective case-control study of 2090 adults using questionnaire, height and weight, and skin-prick testing between January 1994 and December 2003.

Results Obesity in adults was associated with increased AD [multinomial logistic regression: adjusted odds ratio (aOR) 1.43, 95% confidence interval (CI) 1.08–1.89; $P = 0.01$], but not nonatopic dermatitis (aOR 0.59, 95% CI 0.21–1.68; $P = 0.32$). Obesity was also associated with increased atopic asthma (aOR 1.98, 95% CI 1.47–2.66, $P < 0.0001$), but not associated with nonatopic asthma ($P = 0.20$), atopic or nonatopic rhinoconjunctivitis ($P = 0.08$ and 0.31 , respectively), food allergies ($P = 0.67$ and 0.35 , respectively) or atopy ($P = 0.40$). The association between obesity and AD remained significant even when controlling for history of asthma, rhinoconjunctivitis and food allergies (aOR 1.40, 95% CI 1.05–1.86; $P = 0.02$) or in subset analyses of subjects with AD alone (aOR 1.96, 95% CI 1.02–3.75; $P = 0.04$) and with comorbid asthma, rhinoconjunctivitis and/or food allergies (aOR 1.40, 95% CI 1.03–1.91; $P = 0.03$).

Conclusion Obesity in adulthood is associated with AD. Further studies are warranted to determine if weight loss may prevent or mitigate AD in adults.

Atopic dermatitis (AD) is a chronic inflammatory disease of skin that affects 1–3% of U.S. adults.^{1,2} AD is often intensely pruritic and can be a significant source of morbidity, including decreased performance at school³ and work,⁴ impaired social relationships^{5,6} and increased risk of infections.^{7,8} The identification of modifiable risk factors for AD may enable the development of novel strategies for prevention and management of the disease. Several modifiable risk factors for AD have previously been identified, including stress, irritants, food, inhalant and/or contact allergens.⁹

A recent study by Silverberg et al.¹⁰ identified prolonged obesity (> 2.5 years) in early childhood as a modifiable risk factor for AD in children. Short-term obesity was not associated with increased AD, suggesting that rapid correction of obesity with weight loss may prevent the disease.¹⁰

Obesity has been reported to produce numerous effects on the immune system, including altered leucocyte distribution, activation and trafficking^{11–15} and proinflammatory immune responses^{12,13,16} that may contribute to the pathogenesis of AD. However, it is not known if the association between obesity and increased AD is limited to early childhood, when

the immune system is most plastic, or also exists during adulthood.

In the present study, we examined an adult cohort to determine if there is an association between obesity in adulthood and increased AD.

Methods

This study was approved by the institutional review board (IRB) for human subjects of the Beth Israel Medical Center, New York, NY. All patients who presented to the allergy clinic at the Beth Israel Medical Center were given paper-based self-administered questionnaires as part of routine clinical practice from 1994 to 2003. The *a priori* aims of the questionnaire were to identify determinants of AD and other disorders, including obesity, race/ethnicity and other demographic information. The data were retrospectively collected from 2003 to 2006 and IRB approval was for retrospective data collection and chart review. Exclusion criteria were age ≤ 18 years, presence of dermatographism and incomplete body mass index (BMI) or questionnaire information. No personal identifiers

were used during the study. Written informed consent was obtained as part of the questionnaire. Data were collected and entered into a computerized database by an observer who was neither aware of the *a priori* hypothesis nor involved in the statistical analysis. AD and nonatopic dermatitis were defined by self-report of eczema with or without a family history of atopic disorders and one or more positive skin-prick tests, respectively.

Questionnaire

Patients completed a one-page questionnaire that included self-reported age (years), sex (M/F), and race ethnicity (African-American, Hispanic-American, Asian-Pacific American or Caucasian). Patients were asked to circle yes or no for personal or family history of 'eczema', 'asthma', 'hay fever (itchy eyes or nose)' for allergic rhinoconjunctivitis, and/or 'food allergy'. Answer choices were encoded as 0/1 for no/yes and M/F, respectively, and 1–4 for race ethnicity. Data entry was verified by screening for nonpredefined responses for multiple choice questions and for noninteger or implausible (> 100 year) values for age.

Growth parameters

Weight (lb) and upright height (in) were measured for all patients upon enrolment. BMI was calculated by $[\text{weight (lb)} \times 703] / \text{height (in)}^2$. Obese categorization was taken as $\text{BMI} \geq 30$ and encoded as a binary variable where 1 = obesity (exposure) and 0 = nonobesity (no exposure). This variable was used for logistic regression analysis (see below).

Skin-prick testing

No patients had taken oral antihistamines or corticosteroids for at least 1 week, or topical antihistamines or corticosteroids for at least 72 h, prior to skin-prick testing. All skin tests were standardized with > 10 mm spacing between specific test sites. The skin-prick testing panel included the following standardized allergens: trees, grasses, weeds, moulds, dog, cat, dust mites, mouse, feathers and cockroach, in 1 : 10 and 1 : 20 w/v prick-testing concentrations (Greer Laboratories, Inc., Lenoir, NC, U.S.A.; Hollister Stier, Spokane, WA, U.S.A.). Normal saline was used as a negative control, where any response (≥ 1 mm) was considered dermatographism and therefore excluded from the study.¹⁷ Histamine dihydrochloride (10 mg mL⁻¹; Hollister Stier) was used as a positive control. All skin tests were performed via a prick technique using one-time use, disposable, 25 g 5/8 precision glide needles (Becton Dickinson, Franklin Lakes, NJ, U.S.A.) to the volar surface of the arm. Wheal size (mm) was evaluated 10 and 15 min later and documented. A positive skin test was judged by a wheal size 3 mm greater than that with normal saline and/or greater than or equal to the histamine control. Flare size was not routinely measured because of occasional difficulty appreciating flares in skin of colour and questionable reliability.¹⁸

Atopic dermatitis

AD (cases) and nonatopic dermatitis were defined by self-report of eczema with or without a family history of atopic disorders and one or more positive skin-prick tests, respectively. This definition was selected at the conclusion of the study in order to minimize recall bias and to distinguish AD from other eczematous disorders, analogous to the major AD criteria of personal and family history of cutaneous atopy according to Hanifin and Rajka.¹⁹ Controls included all other subjects who responded 'no' to personal history of 'eczema'.

Statistical analysis

The baseline characteristics of the patients were reviewed. Age at presentation was found to be not normally distributed as judged by analysis of residuals. Therefore, age was encoded as an ordinal variable (19–39, 40–59, ≥ 60) for all analyses. $P < 0.05$ was taken to indicate statistical significance for all estimates. However, the multiple dependent tests performed in this study increase the risk of falsely rejecting the null hypothesis. Therefore P -values near 0.05 should be interpreted with caution. All tests were two-sided, and data processing and statistics were done in SAS version 9.2 (SAS Institute, Cary, NC, U.S.A.).

Multinomial logistic regression was used to determine whether obesity was associated with AD (eczema), asthma, rhinoconjunctivitis, atopy (as defined above) and food allergies. The dependent variables were encoded as categorical, where 1 = absence of the respective clinical diagnosis by self-report, 2 = presence of the respective clinical diagnosis by self-report without positive skin-prick testing (defined above) or family history of any atopic disorders (eczema, asthma, rhinoconjunctivitis or food allergies), and 3 = presence of the respective clinical diagnosis by self-report with positive skin-prick testing (defined above) and family history of any atopic disorders (eczema, asthma, rhinoconjunctivitis or food allergies). Sex, race ethnicity, age and season of visit were considered potential confounders or effect modifiers; adjusted odds ratios (ORs) were calculated by including them in the full models. Linear interaction terms were tested between obesity and sex, race ethnicity, age and season of visit, and were included in the final models if significant ($P < 0.1$).

Results

Population characteristics

A total of 2439 charts were reviewed, of which 2258 adult patients completed the questionnaire; 27 were excluded based on dermatographism, and 141 were excluded for incomplete BMI or questionnaire data, yielding a total of 2090 subjects with no missing data included in the analysis (Fig. 1). Out of the subjects, 1446 patients were female (69.3%), 1362 were < 40 years old (65.2%) and 1069 were Caucasian (51.2%) (Table 1). First visits were most common in Spring

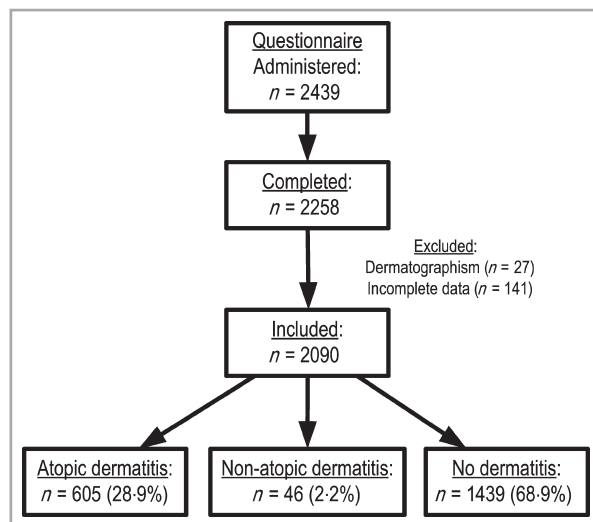


Fig 1. Flow chart for study.

(46.4%), with an even distribution in the other three seasons. Patient-reported rates of disorders included eczema (45.5%), asthma (26.0%), hay fever (61.1%) and food allergies (28.0%). Family history of atopic disorders included eczema (27.6%), asthma (32.9%), hay fever (42.5%) and food allergies (19.6%). A positive skin-prick test was found in 1688 subjects (80.8%), with the exclusion of dermatographism. Seven hundred and seventy-one subjects (36.9%) had both a history of eczema and positive skin-prick test, 733 (35.1%) had both a personal history of eczema and a family history of atopic disorders, and 604 (28.9%) had a combination of a history of eczema, family history of atopic disorders and positive skin-prick test. AD and nonatopic dermatitis were defined by self-report of eczema with or without family history of atopic disorders and one or more positive skin-prick tests, respectively.

Patients with AD had a median of eight positive skin-prick tests (interquartile range six, maximum 13). Five hundred and forty-nine of 604 (90.9%) were positive for dust mite, 481 (79.6%) for trees, 474 (78.5%) for weeds, 419 (69.4%) for cat, 412 (68.2%) for grasses, 382 (63.3%) for mould, 367 (60.8%) for cockroach, 265 (43.9%) for dog, 257 (42.6%) for mouse and 216 (35.8%) for feathers.

Obesity was associated with increased odds of atopic dermatitis

A total of 1258 subjects (60.2%) were normal weight (BMI < 25), 555 (26.6%) were overweight (25 ≤ BMI < 30) and 277 (13.3%) were obese (BMI ≥ 30). Obesity prevalence was higher at ages 40–59 and ≥ 60 years when compared with 18–39 years [age 40–59: OR 2.47, 95% confidence interval (CI) 1.88–3.23, $P < 0.0001$; age ≥ 60: OR 1.96, 95% CI 1.24–3.10, $P = 0.004$]. Moreover, obesity prevalence was higher in Hispanic subjects (OR 1.67, 95% CI 1.25–2.21, $P = 0.0004$), but lower in Asian/Pacific-Americans (OR 0.40, 95% CI 0.25–0.65, $P = 0.0002$) compared with Caucasians

Table 1 Characteristics of the subjects (n = 2090)

Variable	n (%)
Female sex	1446 (69.3)
Age (years)	
19–39	1362 (65.2)
40–59	577 (27.6)
≥ 60	151 (7.2)
Season of visit	
Winter	530 (25.4)
Spring	969 (46.4)
Summer	301 (14.4)
Autumn	290 (13.9)
Race/ethnicity	
African-American	144 (6.9)
Hispanic	510 (24.1)
Asian	366 (17.5)
Caucasian	1069 (51.2)
Personal history	
Eczema	949 (45.5)
Asthma	543 (26.0)
Hay fever	1273 (61.1)
Food allergies	583 (28.0)
Family history	1433 (68.6)
Eczema	571 (27.6)
Asthma	671 (32.9)
Hay fever	881 (42.5)
Food allergies	407 (19.6)
Positive skin-prick test	1688 (80.8)
Personal history of eczema + positive skin prick test	771 (36.9)
Personal history of eczema + family history of atopic disorders	733 (35.1)
Personal history of eczema + family history of atopic disorders + positive skin prick test (i.e. atopic dermatitis)	604 (28.9)

(Table 2). Therefore, age and race/ethnicity were included in the multivariate models. Sex and season of visit were also included due to effect modification.

Multinomial regression models were constructed with obesity as a binary variable, and included age, sex, race/ethnicity and season of visit. Obesity was associated with increased odds of AD (multinomial logistic regression; OR 1.30, 95% CI 1.01–1.70, $P = 0.049$), but not nonatopic dermatitis (OR 0.66, 95% CI 0.23–1.85, $P = 0.42$) (Table 3). This association remained significant when controlling for sex, race/ethnicity and age [AD: adjusted OR (aOR) 1.43, 95% CI 1.08–1.89, $P = 0.01$; nonatopic dermatitis: aOR 0.59, 95% CI 0.21–1.68, $P = 0.32$] (Table 3). The association between obesity and AD remained significant even when controlling for history of asthma, rhinoconjunctivitis and food allergies in multivariate models (aOR 1.40, 95% CI 1.05–1.86, $P = 0.02$) or in subset analyses of subjects with AD alone (aOR 1.96, 95% CI 1.02–3.75, $P = 0.04$) and with comorbid asthma, rhinoconjunctivitis and/or food allergies (aOR 1.40, 95% CI 1.03–1.91, $P = 0.03$). However, there was no significant association between overweight and AD (aOR 0.84, 95% CI 0.65–1.17, $P = 0.15$).

Table 2 Association of demographics with obesity

Variable	Not obese (n = 1813)	Obese (n = 277)	OR (95% CI)	P ^a
Female sex	1243 (86.0)	203 (14.0)	1.25 (0.94–1.66)	0.12
Age (years)				
19–39	1231 (90.4)	131 (9.6)	1.00	–
40–59	457 (79.2)	120 (20.8)	2.47 (1.88–3.23)	< 0.0001
≥ 60	125 (82.8)	26 (17.2)	1.96 (1.24–3.10)	0.004
Season of visit				
Winter	453 (85.5)	77 (14.5)	1.00	–
Spring	843 (87.0)	126 (13.0)	0.76 (0.49–1.17)	0.41
Summer	260 (86.4)	41 (13.6)	0.88 (0.65–1.19)	0.72
Autumn	257 (88.6)	33 (11.4)	0.93 (0.62–1.40)	0.21
Race/ethnicity				
Caucasian	934 (87.4)	135 (12.6)	1.00	–
African–American	121 (84.0)	23 (16.0)	1.32 (0.81–2.13)	0.26
Hispanic	411 (80.6)	99 (19.4)	1.67 (1.25–2.21)	0.0004
Asian	346 (94.5)	20 (5.5)	0.40 (0.25–0.65)	0.0002

Values are n (%). ^aBinomial logistic regression was used with obesity modelled as the binary dependent variable and sex, race/ethnicity, season of visit and age as independent variables.

Table 3 Association of obesity in adults with increased atopic dermatitis and asthma

Atopic disorder	Not obese (n = 1813)	Obese (n = 277)	OR (95% CI)	P	aOR (95% CI)	P ^a
Dermatitis ^b						
None	1259 (69.4)	180 (65.0)	1.00	–	1.00	–
Nonatopic	43 (2.4)	3 (1.1)	0.66 (0.23–1.85)	0.42	0.59 (0.21–1.68)	0.32
Atopic	511 (28.2)	94 (33.9)	1.30 (1.01–1.70)	0.049	1.43 (1.08–1.89)	0.01
Asthma ^b						
None	1462 (80.6)	192 (69.3)	1.00	–	1.00	–
Nonatopic	43 (2.4)	3 (1.1)	0.71 (0.25–2.00)	0.52	0.46 (0.14–1.52)	0.20
Atopic	308 (17.0)	82 (29.6)	2.05 (1.54–2.73)	< 0.0001	1.98 (1.47–2.66)	< 0.0001
Rhinoconjunctivitis ^b						
None	1050 (57.9)	149 (53.8)	1.00	–	1.00	–
Nonatopic	25 (1.4)	2 (0.7)	0.56 (0.13–2.39)	0.43	0.47 (0.11–2.04)	0.31
Atopic	738 (40.7)	126 (45.5)	1.19 (0.92–1.53)	0.19	1.26 (0.97–1.64)	0.08
Food allergies ^b						
None	1464 (80.8)	223 (80.5)	1.00	–	1.00	–
Nonatopic	34 (1.9)	3 (1.1)	0.58 (0.18–1.90)	0.37	0.56 (0.17–1.87)	0.35
Atopic	315 (17.4)	51 (18.4)	1.06 (0.77–1.48)	0.72	1.08 (0.77–1.50)	0.67
Atopy ^c	1463 (80.7)	225 (81.2)	1.04 (0.75–1.43)	0.84	1.15 (0.83–1.61)	0.40

Values are n (%). ^aMultinomial logistic regression was used with individual atopic disorders, and binary logistic regression was used with atopy modelled as the categorical dependent variable, and obesity (body mass index ≥ 30) modelled as an independent variable, including sex, race/ethnicity and age as covariates. ^bEach disorder was modelled as a categorical variable where 1 = absence of the diagnosis, 2 = presence of the diagnosis without family history of atopic disorders or ≥ 1 positive skin-prick test, and 3 = presence of the diagnosis with family history of atopic disorders and ≥ 1 positive skin-prick test. ^cAtopy is defined by at least one positive skin-prick test. OR, odds ratio; CI, confidence interval; aOR, adjusted OR.

Obesity was associated with increased odds of atopic asthma, but not rhinoconjunctivitis, food allergies or atopy

Obesity was associated with increased odds of atopic asthma (multinomial logistic regression; OR 2.05, 95% CI 1.54–2.73, $P < 0.0001$), but not nonatopic asthma (OR 0.71, 95% CI

0.25–2.00, $P = 0.52$) (Table 3). This association remained significant when controlling for sex, race, age (atopic asthma: aOR 1.98, 95% CI 1.47–2.66, $P < 0.0001$; nonatopic asthma: aOR 0.46, 95% CI 0.14–1.52, $P = 0.20$) (Table 3) and diagnosis of AD (atopic asthma: aOR 1.89, 95% CI 1.39–2.57, $P < 0.0001$; nonatopic asthma: aOR 1.10, 95% CI 0.40–3.00, $P = 0.99$). However, there was no significant

association between overweight and atopic asthma (aOR 0.88, 95% CI 0.67–1.17, $P = 0.38$).

Overweight and obesity were not associated with atopic or nonatopic rhinoconjunctivitis (overweight: $P = 0.74$ and 0.72 ; obesity: $P = 0.08$ and 0.31 , respectively) or food allergies (overweight: $P = 0.84$ and 0.73 ; obesity: $P = 0.67$ or 0.35 , respectively) (Table 3). Moreover, overweight and obesity were not associated with atopy as judged by at least one positive skin-prick test ($P = 0.89$ and 0.40 , respectively).

There were no significant linear interaction terms between obesity and race/ethnicity, sex, age and season of visit ($P = 0.21$ – 0.84) for any of the outcomes; therefore, interaction terms were not included in the final models. Race/ethnicity, sex, age and season of visit were retained in the final models because of significant effects or effect modification of obesity estimates.

Discussion

The present study demonstrates an association between obesity and increased AD and asthma in adults. Numerous studies have reported associations between obesity and asthma^{20–27} and obesity and atopy.^{28,29} However, few studies have set forth to examine the association between obesity and AD. A recent study by Silverberg *et al.*¹⁰ found an association between prolonged obesity (> 2.5 years) in early childhood and increased odds of AD and severity of AD. An association between obesity and AD in children was also demonstrated in other clinical and population-based studies.^{30–32} In contrast, earlier studies found no association between obesity and AD.^{23,33,34} However, none of the latter studies was powered primarily to examine the association between obesity and AD. Further, the latter studies used self-report of 'doctor diagnosed' eczema as the outcome for analysis, which probably does not accurately distinguish between atopic and other dermatitides, e.g. irritant or allergic contact, and dyshidrotic, xerotic or seborrheic eczema. The use of a composite definition of AD including self-report, positive skin-prick testing and family history of allergic disorders better distinguishes AD from other eczematous disorders.

The present study compared the effects of obesity in adults with AD and other allergic disorders, including asthma, rhinoconjunctivitis, etc. That obesity was associated with AD or asthma, but not atopy, probably stems from the substantial number of subjects with rhinoconjunctivitis, food allergies or other unspecified disorders, e.g. chronic urticaria or angio-oedema, none of which were associated with obesity. This suggests that obesity has a specific relationship with AD and asthma, but not atopy or other allergic disorders, in general.

AD is a common disorder of childhood, and is a leading cause for visits to a paediatric dermatology clinic. AD is generally mild, and recurrent disease flares usually stop occurring by adulthood. AD that is persistently active into adulthood tends to be more severe and has more recurrent flares.³⁵ The aetiology of AD is multifactorial, including modifiable risk factors, e.g. infectious exposures³⁶ and diet, and nonmodifiable

risk factors, e.g. genetics. The identification and elimination of additional modifiable risk or exacerbating factors are important for the prevention and management of AD.

The demonstration of an association between obesity and AD in adults suggests that the correction of obesity, i.e. weight loss, may improve the prognosis of AD. Previous studies found that weight loss resulted in significant improvement of lung function in asthmatics and decreased asthma symptoms.^{37–39} However, none of these studies examined the role of weight loss on AD. Further studies are needed to determine whether weight loss can similarly improve AD in children and/or adults.

The present study found no association between obesity and atopy. These results are consistent with several other studies that also found no significant association between obesity and atopy.^{23,24,28,40–43} However, some studies have found a significant association. Data from the 2005–2006 NHANES reports show a higher total serum IgE level and an increased OR of atopy in obese children and adolescents.²⁹ Yoo *et al.*⁴⁴ demonstrated an increased OR of atopy in obese subjects compared with those of normal weight, even after adjusting for sex, age and other covariates. In contrast, a case-control study of 457 Swedish adolescents found no significant association between being overweight or obese and atopy.²⁶ Several factors may explain the differences in results, including different regions and environmental exposures, racial and ethnic populations, socioeconomic status, etc. Nevertheless, the present study found no association between obesity and atopy.

It is well established that obesity is a risk and exacerbating factor for psoriasis,^{45–48} another chronic inflammatory skin disorder. However, the present study in adults, together with the results of Silverberg *et al.*¹⁰ in children, suggests that obesity could potentially be a risk or exacerbating factor for AD, as well. Further, psoriasis has been shown to be an independent risk factor for cardiovascular disease⁴⁹ and myocardial infarction,⁵⁰ even when controlling for obesity. It is not yet known if AD is similarly a risk factor for worsened cardiac and vascular disease. Future studies are warranted to elucidate these important questions.

The mechanism behind the association of obesity in adulthood and AD is unknown. Several studies found an association between obesity and increased atopy,^{24,28,29} suggesting a common mechanism. Obesity has a number of effects on the immune system, including altered leucocyte distribution and function in blood^{51,52} and adipose tissue,^{53,54} promotion of the production of proinflammatory cytokines⁵⁴ and altered profile of adipokines.⁵⁵ Low levels of the adipokine adiponectin were previously found in German schoolchildren with AD, while high leptin levels were found in asthma.⁵⁶ Altered adipokine levels in AD may play a causative role in or be secondary to chronic allergic inflammation, but do not appear to be related to use of topical steroid therapy for AD.⁵⁷ Future studies should be performed to elucidate the mechanism of association between obesity, inflammation and AD in both children and adults.

In the present study, obesity prevalence was 13.3% overall, with a significantly higher prevalence in older patients

(20.8%) and Hispanic individuals (19.4%). The prevalence is lower than the age-adjusted prevalence estimates of 22.9% and 30.5% in the NHANES 1988–1994 and 1999–2000 studies, respectively.⁵⁸ Several factors may explain these differences. The majority of subjects in the present study were younger (18–40 years), which was associated with lower obesity prevalence. Moreover, differences of environmental, socioeconomic and/or other lifestyle factors in this cohort may be a contributing factor.

This study has several strengths, including a large sample size and controlling for potentially confounding factors in the statistical models. However, this study also has potential limitations. Subjects were enrolled from an academic referral-based allergy clinic, which allowed for comparison of the specific effects of obesity on AD compared with other allergic disorders. AD was found in 28.9% of patients, which is considerably higher than the previously reported 1–3% U.S. prevalence.^{1,2} The high prevalence of AD in our population is probably due to a combination of (i) selection bias, in that referred patients are more likely to have a severe phenotype of allergic disease, (ii) a large referral pool from in-house dermatology practices and clinics for patients with a high degree of suspicion for allergic disease, and (iii) specific environmental, socioeconomic and lifestyle issues. This may limit the generalizability of the results to the adult atopic population and perhaps not the general population. An inherent limitation to a cross-sectional study is the inability to assess the temporal relationship between the exposure and outcome. Thus, an association between obesity and AD could mean that obesity triggers or worsens AD, or, conversely, that AD causes obesity perhaps secondary to altered lifestyle and topical and/or oral corticosteroid usage. Future research will be aimed at elucidating the temporal relationship between obesity and AD. A limitation of questionnaire-based studies is a potential reporting and misunderstanding bias and a lack of clinical information, such as severity and distribution of AD lesions. Self-reports of eczema by patients are not specific and probably include multiple aetiologies, e.g. xerosis, seborrheic dermatitis, irritant or allergic contact dermatitis. Moreover, there is a remote possibility of recall bias by obese patients to self-report eczema. These were addressed by using a more stringent definition for AD, including self-report of eczema, family history of atopic disorders and at least one positive skin-prick test indicative of atopy. These criteria are analogous to the major AD criteria of personal and family history of cutaneous atopy according to Hanifin and Rajka.¹⁹ Finally, there was a small number of patients with nonatopic dermatitis, which might limit the ability to reach conclusions about a lack of association with obesity.

In conclusion, this study provides evidence of an association between obesity and increased AD, suggesting AD as yet another harmful consequence of obesity. Thus, it is possible that weight loss in adults may prevent or improve AD. Further research aimed at elucidating the temporal relationship between obesity and AD is required.

What's already known about this topic?

- Obesity in early childhood is associated with atopic dermatitis.

What does this study add?

- Obesity in adulthood is associated with atopic dermatitis but not nonatopic dermatitis or atopy alone.

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