A longitudinal cohort study examining determinants of overweight and obesity in adulthood

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ABSTRACT

OBJECTIVES: Adulthood overweight and obesity are multifaceted conditions influenced by a combination of biological, environmental and socio-cultural factors across the lifespan. Using a longitudinal study design, we aimed to identify determinants of adulthood overweight and obesity, in relation to: 1) childhood and life course factors, 2) geographical differences in air quality, and 3) gender-specific factors, in a cohort followed from childhood into adulthood.

METHODS: Childhood data were acquired (1978–1986) from children residing in four distinct Hamilton neighbourhoods (Ontario, Canada), including air-quality assessments. Adulthood data were obtained (2006–2007) from successfully retraced participants ($n = 315$) using comprehensive self-administered questionnaires. Multivariate logistic regressions were used to evaluate determinants of adulthood overweight (BMI: 25–29.9 kg/m²) and obesity (BMI: ≥ 30).

RESULTS: The prevalence of normal weight decreased drastically at follow-up in adulthood, while that of overweight and obesity increased. Both overweight and obesity in adulthood were associated with male gender and occupational exposures to contaminants. Childhood residence in Hamilton neighbourhoods with better air quality was associated with lesser odds of adulthood overweight, whereas adulthood obesity was strongly linked to childhood weight gain (overweight or obesity). Among females, childhood weight status predicted overweight and obesity in adulthood, with always living in Hamilton, lack of additional health insurance, negative self-appraisal and high blood pressure during adulthood identified as other significant predictors. Among males, prolonged occupational exposures to contaminants emerged as a unique determinant of adulthood weight gain.

CONCLUSION: Adulthood overweight and obesity are associated with childhood and life course determinants, including childhood weight status, residential air quality and occupational contaminant exposures, in a gender-specific manner.

KEY WORDS: Overweight; obesity; body mass index; air pollution; longitudinal studies

La traduction du résumé se trouve à la fin de l'article.

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According to the World Health Organization (WHO), the global prevalence of obesity, defined as body mass index (BMI) of ≥ 30 kg/m², has doubled in the last three decades.¹ Similar trends were observed in the Canadian population, with nearly 14.3 million aged 18 years and older (61.8% men, 46.2% women) categorized as either overweight or obese in 2014 – an upsurge from 2003.² Although provincial and territorial variations exist due to region-specific characteristics,³ a predictive analysis anticipated that by 2019, nearly half of all Canadian provinces would have more overweight or obese adults relative to normal weight.⁴ Adulthood obesity is a global health concern, which has consistently been associated with insulin resistance, type 2 diabetes, cardiovascular diseases, asthma, stroke, osteoarthritis, and various types of cancers.^{5,6} These co-morbidities adversely impact the quality of life, longevity, as well as educational and occupational prospects of overweight and obese individuals.^{7,8} Social and psychological correlates of excessive weight, such as low socio-economic status, depression, low self-esteem and discrimination, are also well documented.^{9,10} While the precise aetiology of adulthood overweight and obesity is multifactorial, childhood factors may play vital contributory roles due to tremendous physical and psychosocial development associated with early life stages.

Previous investigations have highlighted early determinants of high BMI in adulthood, which include high birthweight, high pre-pregnancy maternal and post-birth childhood BMI, early menarche, high dietary intake, low socio-economic status and low levels of physical activity.^{10,11} Feeding and parenting styles have also been suggested as determinants.¹² Additionally, investigations have implicated air quality to be an important modulator of long-term weight status.¹³ For example, a recent animal study in pregnant rats exposed to Beijing's unfiltered air showed evidence of weight gain, and offspring were heavier pre- and postnatally with accompanying lung inflammation, systemic oxidative stress and enhanced proinflammatory markers.¹³ Daily inhalation rates tend to be higher in overweight and obese human

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adults,¹⁴ which may result in a greater intake of air contaminants. However, it remains unclear whether chronic exposures to air pollutants early in life have any association with BMI or psychosocial well-being in adulthood. Chronic exposures to air contaminants can have significant long-term health consequences, especially in children due to their developmental stage, underdeveloped immune system, proportion of time spent outdoors, and relatively smaller and immature lungs.¹⁵

Using a 30-year exposure and health data initially collected from a cohort of children residing in Hamilton (Ontario, Canada) during 1978–1986 and from follow-up research in 2006, we aimed to assess childhood and life-course determinants of weight status in adulthood. Complete details of the original program are described elsewhere.¹⁶ Specifically, the original study took place in four geographically-distinct neighbourhoods in Hamilton – a city with a legacy of air-quality issues due to the industrial nature of its economy. The city experienced an air-quality gradient across the four neighbourhoods, with East Lower (EL) bearing the highest levels of air pollution due to its proximity to the industrial core relative to Hamilton's other three neighbourhoods: West Lower (WL), East Upper (EU) and West Upper (WU) (Figure 1).^{16,17} WU region experienced relatively lower levels of air pollution.¹⁷ This natural arrangement provided a unique opportunity to study the role of early air-contaminant exposures on weight status in adulthood. Furthermore, a combination of macro- and micro-environmental factors were examined in relation to weight status in adulthood, using the Life Course Health Development (LCHD) model as a guiding framework.¹⁸ The LCHD model conceptualizes these macro- and micro-environmental contexts along with mechanistic and regulatory processes into an overall integrative framework (Figure 2). Using a

longitudinal design with participants from distinct neighbourhoods of Hamilton, this study also incorporates the time and place elements of LCHD to identify predictors of weight status in adulthood.

Our study objectives were to: 1) apply the LCHD framework to assess childhood and life course determinants of weight status in adulthood, 2) evaluate associations between adulthood weight status and geographical differences in air quality, and 3) assess gender-specific factors associated with weight status in adulthood.

METHODS

Data source

Data from childhood were acquired from the original research program (1978–1986), which included a cohort of 3202 children residing in four neighbourhoods of Hamilton.^{16,19} In 2006, 929 cohort members were successfully retraced, 598 consented to participate and 395 returned completed surveys (response rate 66%), forming the basis of data from adulthood.¹⁹ Of the 395 participants, 80 were excluded due to missing childhood and/or adulthood weight and/or height information required to calculate BMI. This resulted in a final sample size of 315. The McMaster University Research Ethics Board approved the survey and study.

Body mass index categorization at childhood and adulthood

BMI was calculated using self-reported weight and height information as: weight (kg)/height (m²). Each participant's childhood BMI was converted into BMI-for-age percentile score according to WHO Growth Charts adapted for Canada for boys (2–19 years) and girls (2–19 years).²⁰ These BMI-for-age percentile scores were then used to categorize participants into weight categories: wasted (<3rd percentile), normal weight (3rd–<85th), overweight (85th–97th) and obese (>97th). At adulthood, the calculated BMI values were used to classify participants as underweight (BMI: <18.5), normal weight (18.5–24.9), overweight (25–29.9) or obese (≥30). Weight status at adulthood was our primary outcome variable.

Assessment of ambient air quality in childhood

To assess environmental exposures to air pollutants, each participant was assigned annual exposure values for total suspended particulates (TSP) in 1980/1981, 1983/1984 and 1985/1986; particulate matter with aerodynamic diameter <3.3 µm (PM_{3.3}) in 1980/1981, 1981/1982, 1983/1984 and 1985/1986; and sulphur dioxide (SO₂) in 1978/1979, 1980/1981, 1981/1982 and 1983/1984. Exposure estimates were modelled using location of schools the children attended during 1978–1986, and a large network of monitoring sites in Hamilton (details published previously).^{16,19} Second, exposure was assessed by dichotomizing participants based on whether they were exposed to each air pollutant above or at/below median per sampling period (Table 1). Finally, exposure indices were created for TSP, PM_{3.3} and SO₂ to indicate frequency of exposure above or at/below median across all sampling periods. As environmental exposures to air pollutants have previously been linked to respiratory,¹⁹ non-respiratory,^{17,19} and inflammatory disorders,²¹ these air-quality indicators were analyzed as independent variables.

Figure 1. Geographical map of the study area showing the four residential neighbourhoods in Hamilton, Ontario (Source: Barakat-Haddad et al.¹⁷)

Figure 2. Graphical representation of the Life Course Health Development (LCHD) framework as conceptualized in this study (*Modified from: Halfon and Hochstein¹⁸*). Macro-environmental context includes health determinants related to demographic, residential, environmental, occupational, health care, family and social environments (1); whereas, micro-environmental context includes biobehavioural determinants associated with functioning of behavioural, physiological and psychological systems, such as the immune and respiratory systems (2A). Mechanistic and regulatory processes depict developmentally programmed processes that can modulate micro-environmental context (2B). Macro-environmental factors interact with each other and with micro-environmental factors – which are in turn influenced by mechanistic and regulatory processes. These dynamic and multi-contextual interactions are modulated by environmental exposures and determine health outcomes over the life course (3).

Macro-environmental variables at childhood and adulthood

In addition to ambient air-quality assessments, data related to demographic and residential domains were included as part of participants' macro-environment in childhood. Demographic variables included age, gender and household income dichotomized as below versus at/above low-income cut-off. Residential variables comprised neighbourhoods of residence in Hamilton, and whether participants were exposed to second-hand smoke. These variables captured early macro-environmental effects, which may impact adulthood BMI.

Several macro-environmental variables from adulthood were also included. Four dichotomized variables captured the adulthood demographical data: education, current household income, ethnicity and housing tenure; whereas, occupational information included employment status, type of occupation, and length of occupational exposures to gas/dust/contaminants. Residence in Hamilton (always or not) was dichotomized under residential history domain. Residential indoor air quality was surveyed using four binary variables: use of air conditioner/humidifier/filter, type of heating, air-duct cleaning and cooking method. One variable measured duration of residence (< versus ≥ 5 years) in buildings

Table 1. Distribution and prevalence of variables assessed at childhood and adulthood in this longitudinal cohort study

Variables	Weight status at adulthood			Total sample (n = 315)
	Normal weight (n = 103)	Overweight (n = 117)	Obese (n = 95)	
Macro-environmental variables in childhood and adulthood				
Demographic				
Gender, % male***	37	62	56	52
Mean age in childhood (years) [†]	8	8	8	8
Household income in childhood (% below low-income cut-off) [†]	14	11	17	14
Mean age in adulthood (years)	36	36	36	36
Current household income (% below low-income cut-off)	2	3	8	4
Education (% completed high school)	99	99	98	99
Ethnicity (% Canadian)	65	56	67	62
Housing tenure (% homeowners)	85	89	81	85
Residential				
Neighbourhood of residence in Hamilton (%)*, [†]				
East Lower	14	32	17	22
West Lower	19	20	18	19
East Upper	30	22	35	29
West Upper	37	26	31	31
Second-hand smoking in childhood (% exposed) [†]	54	43	46	48
Always resided in Hamilton (%)	57	55	64	58
Resided in property built before 1950 (% for ≥5 years)	19	29	27	25
Residential exposure to gas/dust/contaminants (%)	91	92	94	92
Type of heating (% gas/oil)	96	97	97	96
Frequency of air-duct cleaning (% rarely/do not remember)	70	69	69	69
Type of cooking method (% gas)	38	35	38	37
Residential usage of air conditioner (%)	89	82	91	87
Residential usage of air humidifier (%)	52	41	36	43
Residential usage of air filter (%)	41	33	32	35
Environmental				
Total suspended particulate (TSP) (% >median) [†]				
1980/1981	55	50	48	51
1983/1984	55	49	58	54
1985/1986	58	50	50	53
1980–1986	37	32	33	34
Particulate matter under 3.3 µm diameter (PM _{3.3}) (% >median) [†]				
1980/1981	59	49	47	52
1981/1982	51	46	49	48
1983/1984	41	44	53	46
1985/1986	45	43	51	46
1980–1986	46	36	37	40
Sulphur dioxide (SO ₂) (% >median) [†]				
1978/1979	49	42	46	45
1980/1981	51	49	49	50
1981/1982	47	54	48	50
1983/1984	57	41	50	49
1978–1984	40	29	30	33
Current exposure to second-hand smoking at home/work (%)*	39	38	55	44
Occupational				
Employment (% full time)	71	72	74	72
Type of occupation (%)*				
Clerical	13	7	12	10
Manual	16	20	28	21
Managerial	17	13	14	14
Professional	54	55	38	50
Others	0	5	8	4
Occupational exposures to gas/dust/contaminants (% >5 years)**	42	60	67	56
Health care				
Access to regular family doctor (%)	93	89	94	92
Additional health insurance coverage (%)**	74	79	60	71
Family				
Marital status (% married/common-law)	82	74	71	76
Parental record of asthma or respiratory problems (%)	28	27	39	31
Social				
Social contact subscale (% score of ≤ 5)	14	9	12	11
Group participation subscale (% score of ≤ 4)	65	74	61	67
Micro-environmental variables in childhood and adulthood				
Behavioural				
Experienced smoking in childhood (% ≥ 1 cigarette/day for >6 months) [†]	16	15	15	15
Current smoker (%)	19	25	20	21
Ever smoked on a daily basis (%)	28	33	41	34
Regular alcohol consumption (%)	10	15	9	11
Physical activity (% ≥ 3 times/week for >30 minutes)	46	46	55	49
Physiological				
Asthma in childhood (%)*, [†]	4	15	13	10
Persistent morning cough in childhood (%) [†]	9	8	9	9
Persistent day/night cough in childhood (%) [†]	11	8	18	12
Persistent wheeze in childhood (%) [†]	9	23	14	16

Continued

Table 1. (Continued)

Variables	Weight status at adulthood			Total sample (<i>n</i> = 315)
	Normal weight (<i>n</i> = 103)	Overweight (<i>n</i> = 117)	Obese (<i>n</i> = 95)	
Chest illnesses/colds before age 2 (% pneumonia or bronchitis) [†]	4	7	3	5
Index for pulmonary fraction (% always above the median) [†]	19	23	38	26
Weight status in childhood (%)* ^{***,†}				
Normal weight	87	75	51	72
Overweight	11	19	24	18
Obese	2	6	25	10
High blood pressure in adulthood (% diagnosed)**	6	7	20	10
Psychological				
Self-rated health (% fair/poor)**	6	10	29	15
Concern over air pollution (% moderate or extreme)	54	50	53	52
Emotional distress (% score ≥ 4 on GHQ scale)	6	14	16	12
Stressful life events (% ≥ 2)	7	9	14	10
Feelings about income (% negative)*	7	11	20	12
Feelings about health (% negative)**	0	8	14	7
Feelings about life (% negative)*	1	2	6	3

Note: TSP = total suspended particulates; PM_{3.3} = particulate matter with aerodynamic diameter <3.3 µm; SO₂ = sulphur dioxide; GHQ = general health questionnaire.

* *p* < 0.05; ** *p* < 0.01; *** *p* < 0.001.

[†] Data collected at childhood.

constructed pre-1950 as an exposure indicator to harmful chemicals, such as lead and asbestos, commonly used in construction material in that era. Lead and asbestos exposures have previously been linked to changes in inflammatory markers.^{22,23} Indicators of health care accessibility were evaluated in terms of whether access to regular family doctor and whether additional health insurance beyond provincially-funded coverage were available. Marital status and parental record of asthma or respiratory problems (at least one versus none) were evaluated as part of family environment. Finally, information regarding the social environment was gathered using social contact and group participation scales of Social Health Battery.¹⁹ Further details are presented in Table 1.

Micro-environmental variables in childhood and adulthood

Variables comprising participants' micro-environment at childhood were derived from behavioural and physiological domains. Smoking one or more cigarettes per day for more than 6 months captured the behavioural context; whereas, physiological domain included childhood weight status and the following binary determinants: presence of asthma in childhood, persistent morning or day/night cough, persistent wheeze and chest illnesses/colds before age 2. Airway obstruction was assessed as ratio of forced expiratory volume in first second (FEV₁) over forced vital capacity (FVC) at/below median.

At follow-up, behavioural context was further explored by surveying whether the participant: was a current smoker, was ever a daily smoker, engaged in regular alcohol consumption and performed physical exercise exceeding 30 minutes for ≥3 days/week. Weight status and a diagnosis of high blood pressure were measured as physiological variables. Adulthood psychological context was captured by several factors that gathered binary data on stressful life events, concern over air pollution, emotional distress (scoring ≥4 on General Health Questionnaire), self-rated health, and general feelings about income, health and life. These early and life course determinants were analyzed as independent variables. Further details are presented in Table 1.

Statistical analyses

Data were analyzed using SPSS v20. Descriptive statistics of outcome (dependent) and explanatory (independent) variables were calculated and analyzed. Chi-squared (χ^2) tests were used to compare weight categories at adulthood with each of the categorical independent variables. Those variables meeting a threshold of *p* < 0.10 at this bivariate analysis were entered into a series of multivariate logistic regression models, using forward-stepwise entry procedure. Our first model generated best predictors of adulthood weight status in the entire sample. To examine relative roles of ambient air quality, exposure indices of TSP, PM_{3.3} and SO₂ were forced into this "best-fit" model to generate a second "exposure" model. Interactions of interest were examined. Finally, multivariate logistic regression models were created to analyze gender-specific determinants of adulthood weight status by stratifying the sample on gender. For each variable in logistic regression models, one category was selected as reference and other categories were compared against this reference. Significance, odds ratios (OR), 95% confidence intervals (CI) and pseudo-*R*² values were noted.

RESULTS

In childhood (mean age, 8 years), 72% of participants enjoyed a healthy weight; this declined to 33% in adulthood (mean age, 36 years) in the same cohort. Bivariate analyses indicated significant differences between weight status in adulthood and the following macro-environmental variables: gender, neighbourhood of residence in Hamilton during childhood, current exposure to smoking at home/work, type of occupation, duration >5 years of occupational exposures to gas/dust/contaminants, and additional health insurance coverage (χ^2 , *p* < 0.05). Likewise, significant micro-environmental determinants included childhood weight status, asthma, high blood pressure in adulthood, self-rated health, and negative feelings about income, health and life (χ^2 , *p* < 0.05) (Table 1). Childhood weight categories predicted BMI values in adulthood ($F_{2,312} = 39.3$, *p* < 0.001) (See Supplementary Figure 1, in the ARTICLE TOOLS section on the journal site).

Predictors of overweight and obesity in adulthood

The “best-fit” model showed that being overweight in adulthood was predicted by male gender (OR = 2.88) and duration >5 years of occupational exposures to gas/dust/contaminants (OR = 1.93). Additionally, residence in WU (OR = 0.33), EU (OR = 0.37) and WL (OR = 0.40) neighbourhoods in Hamilton during childhood was negatively associated with adulthood overweight, versus living in EL neighbourhood. Obesity in adulthood was associated with male gender (OR = 2.65), self-rated health at adulthood (OR = 7.23) as well as duration >5 years of occupational exposures to gas/dust/contaminants (OR = 3.06). Being overweight (OR = 3.39) or obese (OR = 31.22) during childhood predicted obesity in adulthood (Table 2).

The “exposure” model explained additional variance relative to “best-fit” model, as assessed by an increase in pseudo- R^2 . Exposure indices of TSP, PM_{3.3} and SO₂ were, however, non-significant and not retained in the “best-fit” model (Table 2).

Gender-specific predictors of adulthood overweight and obesity

Females

Among females, adulthood overweight was negatively associated with childhood residence in WU (OR = 0.17), EU (OR = 0.18) and WL (OR = 0.11) neighbourhoods compared to EL, but positively predicted by childhood overweight (OR = 4.64) and obesity (OR = 16.08). In comparison, adulthood obesity in females was not linked to residential neighbourhoods, but rather, associated strongly with childhood weight categories [overweight (OR = 4.11); obese

(OR = 22.53)]. Other variables predictive of adulthood obesity in females were self-rated health at adulthood (OR = 10.54), having no additional health insurance coverage (OR = 0.27), having high blood pressure at adulthood (OR = 4.99), and always residing in Hamilton region (OR = 4.11) (Table 3).

Males

Adulthood overweight in males was predicted by having no additional health insurance coverage (OR = 2.70), and duration >5 years of occupational exposures to gas/dust/contaminants (OR = 2.57); the latter variable was also associated with male obesity in adulthood (OR = 2.79) (Table 3).

DISCUSSION

As per our objectives, the study revealed global and gender-specific determinants of adulthood weight gain in a cohort longitudinally followed for approximately 30 years. The associations of overweight and obesity in adulthood with childhood residential neighbourhoods and occupational contaminant exposures are novel findings of this study.

Male gender and prolonged occupational exposures to harmful contaminants were associated with adulthood overweight and obesity. This gender disparity of male overweight and obesity is consistent with Canadian statistics as well as the existing literature that shows higher male prevalence of overweight in high-income countries belonging to the Organisation for Economic Cooperation and Development group.^{1,24} The association of overweight and obesity with occupational exposures has rarely been reported.

Table 2. Multivariate logistic regression models showing the associations of overweight and obesity in adulthood with childhood and life-course determinants in the entire cohort

Outcome [†]	Variable (reference)	Classification	Best-fit model (n = 314) [‡]		Exposure model (n = 312) [§]	
			OR	95% CI	OR	95% CI
Overweight in adulthood	Gender (female)	Male	2.88***	1.61–5.16	2.72**	1.50–4.91
	Residence in childhood (East Lower)	West Upper	0.33**	0.15–0.74	0.31**	0.14–0.73
		East Upper	0.37*	0.16–0.88	0.34*	0.14–0.81
		West Lower	0.40*	0.17–0.99	0.34*	0.14–0.86
		Overweight	2.29	1.00–5.27	2.14	0.92–5.01
	Weight status at childhood (normal)	Obese	4.54	0.87–23.66	4.92	0.92–26.10
	Self-rated health (good)	Fair/poor	1.80	0.62–5.24	1.69	0.58–4.95
	Occupational exposures to gas/dust/contaminants (≤5 years)	>5 years	1.93*	1.09–3.43	1.99*	1.11–3.56
	Index for TSP exposure (≤median)	>median	–	–	1.28	0.50–3.24
	Index for PM _{3.3} exposure (≤median)	>median	–	–	0.59	0.25–1.42
	Index for SO ₂ exposure (≤median)	>median	–	–	0.74	0.36–1.50
Obese in adulthood	Gender (female)	Male	2.65**	1.36–5.16	2.46**	1.25–4.83
	Residence in childhood (East Upper)	West Upper	0.80	0.30–2.09	0.82	0.30–2.20
		East Upper	1.09	0.40–2.93	0.99	0.36–2.73
		West Lower	0.67	0.23–1.98	0.60	0.20–1.82
		Overweight	3.39**	1.41–8.16	3.18*	1.31–7.75
	Weight status at childhood (normal)	Obese	31.22***	6.61–147.54	34.58***	7.19–166.40
	Self-rated health (good)	Fair/poor	7.23***	2.61–20.05	6.53***	2.34–18.24
	Occupational exposures to gas/dust/contaminants (≤5 years)	>5 years	3.06**	1.57–5.99	3.18**	1.62–6.24
	Index for TSP exposure (≤median)	>Median	–	–	1.37	0.48–3.93
	Index for PM _{3.3} exposure (≤median)	>Median	–	–	0.75	0.28–2.01
	Index for SO ₂ exposure (≤median)	>Median	–	–	0.57	0.25–1.29
Pseudo R ² (Cox & Snell; Nagelkerke; McFadden)		–	0.285; 0.321; 0.154		0.292; 0.329; 0.158	

Note: * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

[†] Relative to “normal weight in adulthood” category.

[‡] n for adulthood weight categories (best-fit model): normal = 102, overweight = 117, and obese = 95.

[§] n for adulthood weight categories (exposure model): normal = 102, overweight = 116, and obese = 94.

Table 3. Multivariate logistic regression models showing the gender-specific associations of overweight and obesity in adulthood with childhood and life course determinants

Outcome [†]	Variable (reference)	Classification	Females (n = 139) [‡]		Males (n = 163) [§]	
			OR	95% CI	OR	95% CI
Overweight in adulthood	Residence in childhood (East Lower)	West Upper	0.17**	0.05–0.63	–	–
		East Upper	0.18**	0.05–0.62	–	–
		West Lower	0.11*	0.02–0.59	–	–
	Weight status at childhood (normal)	Overweight	4.64*	1.39–15.51	–	–
		Obese	16.08*	1.63–158.37	–	–
	Self-rated health (good)	Fair/poor	4.41	0.79–24.72	–	–
	Occupational exposures to gas/dust/contaminants (≤5 years)	>5 years	–	–	2.57*	1.13–5.86
	Additional health insurance coverage (no)	Yes	1.05	0.33–3.33	2.70*	1.11–6.53
	High blood pressure in adulthood (no)	Yes	0.36	0.05–2.83	–	–
	Always resided in Hamilton (no)	Yes	1.03	0.40–2.69	–	–
Obese in adulthood	Residence in childhood (East Lower)	West Upper	0.86	0.14–5.18	–	–
		East Upper	0.98	0.17–5.49	–	–
		West Lower	1.65	0.23–12.19	–	–
	Weight status at childhood (normal)	Overweight	4.11*	1.14–14.87	–	–
		Obese	22.53**	2.40–211.23	–	–
	Self-rated health (good)	Fair/poor	10.54**	1.93–57.60	–	–
	Occupational exposures to gas/dust/contaminants (≤5 years)	>5 years	–	–	2.79*	1.17–6.67
	Additional health insurance coverage (no)	Yes	0.27*	0.09–0.83	1.25	0.52–3.02
	High blood pressure in adulthood (no)	Yes	4.99*	1.06–23.43	–	–
	Always resided in Hamilton (no)	Yes	4.11*	1.26–13.46	–	–
Pseudo R ² (Cox & Snell; Nagelkerke; McFadden)			0.417; 0.471; 0.249		0.075; 0.085; 0.037	

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

† Relative to “normal weight in adulthood” category.

‡ n for adulthood weight categories (females model): normal = 58, overweight = 41, and obese = 40.§ n for adulthood weight categories (males model): normal = 37, overweight = 73, and obese = 53.

Suadicani et al. found higher prevalence of obesity among men with blood phenotype “O” who had occupational exposures to airborne pollutants for at least 5 years²⁵ – a finding consistent with our study. This effect, however, was not examined among those who were overweight.²⁵ Jerrett and colleagues recently found traffic sources of air pollution positively associated with elevated BMI in children.²⁶ Other studies link higher BMI with a range of occupational variables, such as working extended hours per week,²⁷ and transport and production types of employments,²⁸ which may exacerbate workplace exposures to airborne contaminants. Although, we found a higher prevalence of overweight and obesity in relation to “manual” type of employment in this cohort, it is unclear whether manual employment exacerbated occupational exposures or contributed to elevated BMI by other mechanisms. As obesity is generally considered a low-grade inflammatory state,²⁹ it is possible that occupational exposures to pollutants interact synergistically with environmental and genetic risk factors to influence BMI via inflammatory pathways.

Undoubtedly, adulthood overweight and obesity are multifaceted conditions determined by complex biological and socio-cultural factors;²⁴ though some variables may show weight-class-specific associations, as observed in this study. In addition to male gender and duration of occupational exposures, childhood residential regions in Hamilton were significantly associated with adulthood overweight; whereas, childhood weight status was predictive of adulthood obesity (with no regional associations). Specifically, overweight and obese children showed ~3 and ~31 times greater odds of obesity in adulthood respectively. This finding suggests a high heritability index of obesity, estimated at greater than 0.70 – a value comparable with other

polygenic disorders, such as autism (0.91) and schizophrenia (0.81).³⁰ As adulthood overweight was not predictable by childhood weight status, but rather associated with residential neighbourhoods in our models, these findings highlight a multifactorial aetiology for adulthood overweight. This may suggest genetic determinants to be less or just as deterministic of adulthood overweight as regional or residence-based characteristics. Accordingly, WU, EU and WL regions were all found to be associated with lesser odds of adulthood overweight in comparison with the EL region, which sustained the poorest air quality. This implicates some role of regional air quality in influencing adulthood overweight, even though our “exposure model” did not highlight significant associations of TSP, PM_{3.3} or SO₂ exposures. It is likely that living in a certain Hamilton neighbourhood may influence weight status indirectly, by affecting physical activity levels, food choices or region-specific factors; or directly via air quality-related inflammatory mechanisms. Our results support the notion that adulthood obesity carries a stronger genetic disposition, whereas overweight appears to be relatively heterogeneous with genetic as well as possibly modifiable environmental contributors. A recent study strengthens this complex multifactorial notion as McConnell et al. reported a synergistic effect of early exposure to tobacco smoke and near-roadway pollution with childhood obesity,³¹ which in turn is a strong determinant of adulthood weight gain.

Gender-specific analysis revealed distinct factors among males and females. Prolonged occupational exposures in males and childhood weight status in females consistently predicted adulthood overweight and obesity. Association of childhood weight status among females may reflect strong biological and

socio-cultural factors. It is recognized that females tend to have greater fat mass, smaller height, and lighter overall body weight, which vary based on gender-specific physiological conditions, such as pregnancy, menopause and overall metabolism.³² Hence, it is plausible to find early weight status, determined in part by biological differences in fat metabolism, distribution and storage, to be associated with adulthood BMI among females. In addition to finding residential influences of air quality, females permanently residing in Hamilton since childhood were also four times more likely to be obese. Whether this implies a greater gender-specific susceptibility warrants further research. Higher blood pressure was also associated with female obesity. Interestingly, obese females and overweight males were less likely to own additional health insurance beyond provincially-funded coverage. In this cohort, a lower proportion of obese worked a “professional” type of occupation, but instead held more “manual” occupations, which may explain poorer health coverage as it is more often offered in professional positions. Additionally, duration of occupational exposures as a prime determinant of BMI among males may also be associated with job-related nature and/or tasks performed. A recent health report from Montreal found higher workplace exposures among males to motor vehicle exhaust, petroleum fractions, polycyclic aromatic hydrocarbons as well as building material and abrasive dust.³³

Finally, overweight and obese individuals commonly reported negative feelings about health, income and life. However, the only factor that entered into our model was self-rated health reported as “fair or poor” among the obese adults, especially obese females. Such negative self-appraisals and psychosocial burden of obesity are confirmatory, which negatively influence quality of life, satisfaction, and overall psychological health of affected individuals.⁹

CONCLUSIONS, LIMITATIONS AND FUTURE RECOMMENDATIONS

Adulthood overweight and obesity are associated with childhood and life-course factors, including residential and occupational contaminant exposures, in a gender-specific manner. Longitudinal nature and inclusion of a multitude of variables guided by LCHD model are major strengths of this study. Certain limitations are noteworthy. Self-reported data have limitations, importantly the recall bias related to the recollection of contaminant exposures. Absence of air-quality data past childhood restricted our examination of chronic air-pollution exposures and prevented analyses on whether improved air quality has a restorative health effect. Data on physical activity, food intake levels, and physiological inflammatory indicators were unavailable. Finally, our reconstructed cohort had a reduced sample size. This loss to follow-up, however, was demonstrated to be unrelated to the health outcomes, residential neighbourhoods, or particulate exposures.³⁴ Moreover, as our findings are in line with existing literature, we believe that the participant selection process occurred fairly randomly and is unlikely to influence our results. Future studies with larger samples are warranted to replicate the findings and address limitations.

REFERENCES

1. World Health Organization: *Global Health Observatory Data*. Available at: http://www.who.int/gho/ncd/risk_factors/obesity_text/en/ (Accessed March 24, 2016).
2. Statistics Canada: *Overweight and Obese Adults (Self-Reported)*, 2014. Available at: <http://www.statcan.gc.ca/pub/82-625-x/2015001/article/14185-eng.htm#n1> (Accessed March 24, 2016).
3. Dutton DJ, McLaren L. Explained and unexplained regional variation in Canadian obesity prevalence. *Obesity (Silver Spring)* 2011;19(7):1460–68. PMID: 21253004. doi: 10.1038/oby.2010.339.
4. Twells LK, Gregory DM, Reddigan J, Midodzi WK. Current and predicted prevalence of obesity in Canada: A trend analysis. *CMAJ Open* 2014;2(1):E18–26. PMID: 25077121. doi: 10.9778/cmajo.20130016.
5. Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH. The incidence of co-morbidities related to obesity and overweight: A systematic review and meta-analysis. *BMC Public Health* 2009;9:88. PMID: 19320986. doi: 10.1186/1471-2458-9-88.
6. Luo W, Morrison H, de Groh M, Waters C, DesMeules M, Jones-McLean E, et al. The burden of adult obesity in Canada. *Chronic Dis Can* 2007;27(4):135–44. PMID: 17623559.
7. McLaren L. Socioeconomic status and obesity. *Epidemiol Rev* 2007;29:29–48. PMID: 17478442. doi: 10.1093/epirev/mxm001.
8. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: Systematic review. *Int J Obes (Lond)* 2011;35(7):891–98. PMID: 20975725. doi: 10.1038/ijo.2010.222.
9. Carr D, Friedman MA. Is obesity stigmatizing? Body weight, perceived discrimination, and psychological well-being in the United States. *J Health Soc Behav* 2005;46(3):244–59. PMID: 16259147. doi: 10.1177/002214650504600303.
10. Laitinen J, Power C, Järvelin MR. Family social class, maternal body mass index, childhood body mass index, and age at menarche as predictors of adult obesity. *Am J Clin Nutr* 2001;74(3):287–94. PMID: 11522550.
11. Shumei SG, Wei W, William CC, Alex FR. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. *Am J Clin Nutr* 2002;76(3):653–58. PMID: 12198014.
12. Shloim N, Edelson LR, Martin N, Hetherington MM. Parenting styles, feeding styles, feeding practices, and weight status in 4–12 year-old children: A systematic review of the literature. *Front Psychol* 2015;6:1849. PMID: 26696920. doi: 10.3389/fpsyg.2015.01849.
13. Wei Y, Zhang JJ, Li Z, Gow A, Chung KF, Hu M, et al. Chronic exposure to air pollution particles increases the risk of obesity and metabolic syndrome: Findings from a natural experiment in Beijing. *FASEB J* 2016;30(6):2115–22. PMID: 26891735. doi: 10.1096/fj.201500142.
14. Brochu P, Bouchard M, Haddad S. Physiological daily inhalation rates for health risk assessment in overweight/obese children, adults, and elderly. *Risk Anal* 2014;34(3):567–82. PMID: 24147611. doi: 10.1111/risa.12125.
15. Schwartz J. Air pollution and children's health. *Pediatrics* 2004;113(4 Suppl):1037–43. PMID: 15060197.
16. Kerigan AT, Goldsmith CH, Pengelly LD. A three-year cohort study of the role of environmental factors in the respiratory health of children in Hamilton, Ontario. Epidemiologic survey design, methods, and description of cohort. *Am Rev Respir Dis* 1986;133:987–93.
17. Barakat-Haddad C, Elliott SJ, Pengelly D. Health impacts of air pollution: A life course approach for examining predictors of respiratory health in adulthood. *Ann Epidemiol* 2012;22(4):239–49. PMID: 22463842. doi: 10.1016/j.annepidem.2012.02.010.
18. Halfon N, Hochstein M. Life course health development: An integrated framework for developing health, policy, and research. *Milbank Q* 2002;80(3):433–79. PMID: 12233246. doi:10.1111/1468-0009.00019.
19. Barakat-Haddad C, Elliott SJ, Pengelly D. Does chronic exposure to air pollution in childhood impact long-term respiratory health? *Prof Geogr* 2012;64(3):446–63. doi:10.1080/00330124.2011.609775.
20. Dietitians of Canada: *WHO Growth Charts Set 1*. 2016. Available at: <http://www.dietitians.ca/Dietitians-Views/Prenatal-and-Infant/WHO-Growth-Charts/WHO-Growth-Charts-Set-1.aspx> (Accessed March 26, 2016).
21. Barakat-Haddad C, Elliott SJ, Pengelly D. Childhood exposure to air pollution as a potential contributor of chronic non-respiratory inflammatory disorders: A longitudinal prospective cohort study in Hamilton, Canada. *J Environ Protect* 2013;4:779–88. doi: 10.4236/jep.2013.48091.
22. Sirivarasai J, Wananukul W, Kaojare S, Chanprasertyothin S, Thongmung N, Ratanachaiwong W, et al. Association between inflammatory marker, environmental lead exposure, and glutathione S-transferase gene. *Biomed Res Int* 2013;2013:1–6. PMID: 23484121. doi: 10.1155/2013/474963.
23. Haegens A, van der Vliet A, Butnor KJ, Heintz N, Taatjes D, Hemenway D, et al. Asbestos-induced lung inflammation and epithelial cell proliferation are altered in myeloperoxidase-null mice. *Cancer Res* 2005;65(21):9670–77. PMID: 16266986. doi: 10.1158/0008-5472.CAN-05-1751.
24. Kanter R, Caballero B. Global gender disparities in obesity: A review. *Adv Nutr* 2012;3(4):491–98. PMID: 22797984. doi: 10.3945/an.112.002063.

25. Suadicani P, Hein HO, Gyntelberg F. Airborne occupational exposure, ABO phenotype, and risk of obesity. *Int J Obes (Lond)* 2005;29(6):689–96. doi: 10.1038/sj.ijo.0802956.
26. Jerrett M, McConnell R, Wolch J, Chang R, Lam C, Dunton G, et al. Traffic-related air pollution and obesity formation in children: A longitudinal, multilevel analysis. *Environ Health* 2014;13:49. PMID: 24913018. doi: 10.1186/1476-069X-13-49.
27. Shields M. Long working hours and health. *Health Rep* 1999;11(2):33–48. PMID: 10618741.
28. Allman-Farinelli MA, Chey T, Merom D, Bauman AE. Occupational risk of overweight and obesity: An analysis of the Australian Health Survey. *J Occup Med Toxicol* 2010;5:14. PMID: 20550716. doi:10.1186/1745-6673-5-14.
29. Johnson AR, Milner JJ, Makowski L. The inflammation highway: Metabolism accelerates inflammatory traffic in obesity. *Immunol Rev* 2012;249(1):218–38. PMID: 22889225. doi: 10.1111/j.1600-065X.2012.01151.x.
30. Walley AJ, Blakemore AI, Froguel P. Genetics of obesity and the prediction of risk for health. *Hum Mol Genet* 2006;15(2 Suppl):R124–30. PMID: 16987875. doi: 10.1093/hmg/ddl215.
31. McConnell R, Shen E, Gilliland FD, Jerrett M, Wolch J, Chang CC, et al. A longitudinal cohort study of body mass index and childhood exposure to secondhand tobacco smoke and air pollution: The Southern California Children's Health Study. *Environ Health Perspect* 2015;123(4):360–66. PMID: 25389275.
32. Lovejoy JC, Sainsbury A; Stock Conference 2008 Working Group. Sex differences in obesity and the regulation of energy homeostasis. *Obes Rev* 2009;10(2):154–67. PMID: 19021872. doi: 10.1111/j.1467-789X.2008.00529.x.
33. Labrèche F, Lacourt A, Lavoué J. *Occupational Exposure to Chemical and Physical Contaminants: Sex-Differentiated Analysis*. 2015. Available at: <http://www.irsst.qc.ca/media/documents/PubIRSST/R-895.pdf> (Accessed April 1, 2016).
34. Barakat-Haddad CP, Elliott SJ, Eyles J, Pengelly D. Predictors of locating children participants in epidemiological studies 20 years after last contact: Internet resources and longitudinal research. *Eur J Epidemiol* 2009;24(8):397–405. PMID: 19562490. doi: 10.1007/s10654-009-9364-5.

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RÉSUMÉ

OBJECTIFS : Le surpoids et l'obésité à l'âge adulte sont des conditions multidimensionnelles influencées par une combinaison de facteurs biologiques, environnementaux et socioculturels au long de la vie. À l'aide d'un protocole d'étude longitudinale, nous avons cherché à identifier dans une cohorte suivie de l'enfance à l'âge adulte les déterminants du surpoids et de l'obésité à l'âge adulte par rapport : 1) aux facteurs durant l'enfance et dans le parcours de vie, 2) aux écarts géographiques dans la qualité de l'air et 3) à des facteurs sexospécifiques.

MÉTHODE : Les données sur l'enfance, y compris des analyses de qualité de l'air, ont été acquises (1978–1986) pour des enfants vivant dans quatre quartiers de Hamilton (Ontario, Canada). Les données sur l'âge adulte ont été obtenues (2006–2007) auprès de participants retracés avec succès ($n = 315$) à l'aide de questionnaires exhaustifs à remplir soi-même. Nous avons procédé par régression logistique multivariée pour évaluer les déterminants du surpoids (IMC : 25–29,9 kg/m²) et de l'obésité (IMC : ≥ 30) à l'âge adulte.

RÉSULTATS : La prévalence du poids normal a baissé de façon radicale lors du suivi à l'âge adulte, tandis que celles du surpoids et de l'obésité ont augmenté. Le surpoids et l'obésité à l'âge adulte étaient tous deux associés au sexe masculin et aux expositions professionnelles aux contaminants. Le fait d'avoir habité pendant l'enfance dans un quartier de Hamilton où la qualité de l'air était meilleure était associé à une moindre probabilité de surpoids à l'âge adulte, tandis que l'obésité à l'âge adulte était fortement liée au gain de poids (surpoids ou obésité) durant l'enfance. Chez les femmes, le statut pondéral durant l'enfance a prédit le surpoids et l'obésité à l'âge adulte, les autres variables prédictives significatives étant le fait d'avoir toujours vécu à Hamilton, l'absence d'assurance-santé complémentaire, l'appréciation négative de sa propre valeur et l'hypertension artérielle à l'âge adulte. Chez les hommes, l'exposition professionnelle prolongée à des contaminants s'est révélée être un déterminant unique du gain de poids à l'âge adulte.

CONCLUSION : Le surpoids et l'obésité à l'âge adulte sont associés, de façon sexospécifique, avec des déterminants de l'enfance et du parcours de vie, dont le statut pondéral durant l'enfance, la qualité de l'air dans le quartier résidentiel et l'exposition professionnelle aux contaminants.

MOTS CLÉS : surpoids; obésité; indice de masse corporelle; pollution de l'air; études longitudinales