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Family History of Obesity and Risk of Childhood Overweight and Obesity: A Meta-Analysis

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***Abstract*—**The purpose of this study was to determine the significance of history of obesity for the development of childhood overweight and/or obesity. Accordingly, a systematic literature review of English-language studies published from 1980 to 2012 using the following data bases: MEDLINE, PsychINFO, Cochrane Database of Systematic Reviews, and Dissertation Abstracts International was conducted. The following terms were used in the search: pregnancy, overweight, obesity, family history, parents, childhood, risk factors. Eleven studies of family history and obesity conducted in Europe, Asia, North America, and South America met the inclusion criteria. A meta-analysis of these studies indicated that family history of obesity is a significant risk factor of overweight and /or obesity in offspring; risk for offspring overweight and/or obesity associated with family history varies depending of the family members included in the analysis; and when family history of obesity is present, the offspring are at greater risk for developing obesity or overweight. In addition, the results from moderator analyses suggest that part of the heterogeneity discovered between the studies can be explained by the region of world that the study occurred in and the age of the child at the time of weight assessment.

***Keywords*—**Childhood obesity, overweight, family history, risk factors, meta-analysis.

I. Introduction

FAMILY history of obesity can be considered to be both environmental and biological in nature. In terms of environmental risk, parents, in particular mothers, tend to ingrain their personal eating habits as well as their perceptions of weight to their children [1]. For example, overweight/obese mothers may have poor dietary habits, such as excessive eating; furthermore, they may not view overweight status as a potential risk factor for subsequent health complications [2], [3]. In terms of its biological nature, the genetic influence of overweight/obesity may predispose children to developing this condition. This is especially true when both parents are overweight/obese [4].

The research examining family history of obesity and offspring overweight and/or obesity has yielded some contradictory findings, particularly with regards to the degree of association. One specific factor which may at least partially explain the differing degrees of association is the particular family member that is included in the analyses, namely mothers, father, or both mothers and fathers. In general, a higher degree of salience exists between family history of obesity and offspring overweight and/or obesity when mothers are examined, rather than when fathers are the focus [1], [5]-[9].

Exceptions to these findings, however, do exist. Danielzik and colleagues [10] provide evidence to support the idea that, in some instances, the weight status of fathers appears to have more of an influence on offspring obesity than the weight status of mothers does. For example, while a higher degree of association was discovered between obese mothers and overweight offspring, as well as overweight mothers and overweight offspring, the association between parental obesity and offspring obesity was highest among fathers. Krahnstoever and colleagues [2] also provide contradictory findings to the hypothesis that maternal BMI is more predictive of offspring overweight and/or obesity than paternal BMI. Indeed, these researchers examined the weight status of children at ages seven, nine, and eleven. In each age period, paternal history of obesity produced a higher degree of association with offspring overweight than did maternal obesity. These findings on their own cannot be explained simply due to the small sample sizes. Indeed, while the Krahnstoever and colleagues [2] study included only 197 participants, the study by Danielzik and colleagues [10] included 2,631. An amalgamation of this research is therefore needed to truly understand the association between family history of obesity and offspring overweight and/or obesity.

II. Purpose of the Study

The purpose of this current study is to provide a synthesis of the research with respect to family history as a risk factor for childhood overweight and obesity. The specific questions to be addressed are: (a) Is family history of obesity a significant risk factor of overweight and/or obesity in offspring?, (b) Does the risk for offspring overweight and/or obesity associated with family history vary depending on the family members included in the analyses?, and (c) When family history of obesity is present, are offspring at a greater risk for developing obesity or overweight?

III. Method

Meta-analysis is the research design for this study. Within the context of this study, childhood overweight was operationalized as BMI between the 85th and the 95th percentile, while BMI greater than the 95th percentile was used to signify obesity. Within the context of this study, childhood overweight was operationalized as BMI between the 85th and the 95th percentile, while BMI greater than the 95th percentile will be used to signify obesity. Within the

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context of this meta-analysis, family history of obesity was operationalized as either a mother or father with a BMI greater than or equal to 25.

A. Criteria for Study Inclusion

Studies for this meta-analysis were selected based on a set of detailed criterion that were based on age of participants, date of publication, demographics of the participants, research design, and measurement techniques. They are as follows:

1) The study must involve at least one follow-up visit where the child's weight is measured.
2) Obesity and/or overweight must be measured by a medically acceptable measurement technique, including BMI for age or sex growth charts (11).
3) Studies must focus only on singleton births.
4) At the time of the final follow-up visit, children must be no more than 12 years of age. According to Barlow [11] BMI growth charts categorize children at several distinct at children from 2-5 and then 6-12 thus making the age selection conducive with current growth charts [11]-[12].
5) Children must have been born greater than 28 weeks gestation, with no known birth defects or abnormalities.
6) Studies must have been published within the last 30 years.
7) Studies must report sufficient data, including means, standard deviations, odds ratios, and confidence intervals.

B. Search Strategies

In order to avoid publication bias, the literature search included both published and non-published studies. A publication bias may over-estimate the total effect size since negative results or results that failed to reach statistical significance are often not published. As indicated above, a key way to mitigate this issue is to include both published and unpublished studies in the meta-analysis [13]-[14]. To be comprehensive it is important to search multiple electronic databases when doing meta-analyses [15]. As such, searches were performed on the computerized databases Psych INFO (1967 to March 2012), MedLine (1946 to March 2012), Cochrane Database of Systematic Reviews, and Dissertation Abstracts International. Key words for search were identified by examining existing literature in childhood obesity. All computer searches were conducted using the following key words and phrases, in various combinations: 'pregnancy', 'overweight or obesity', 'parents', 'risk factors', and 'childhood'. In an effort to be comprehensive, the tables of contents for journals that commonly publish articles in this area, including Obesity, International Journal of Obesity, Obesity Research, International Journal of Obesity and Related Metabolic Disorders, The Journal of Nutrition, Pediatrics, and International Journal of Epidemiology were reviewed for relevant studies, along with the reference sections of all identified articles, past reviews, and books in this area.

In all, the search yielded 82 articles relating to family history of obesity; 71 were excluded based on the previously established inclusion criteria, which resulted in a total of 11 studies.

C. Coding of Included Studies

Each study was coded with respect to a variety of study features and statistical findings. Primary categories of study features that were coded included: 1) study design, 2) research question(s), 3) independent variables, 4) dependent variables, 5) population targeted, 6) overweight or obesity measurement, 7) moderator variables, and 8) statistical analyses. To ensure that the coding of the studies was accurate and consistent, the primary author initially coded all eligible studies using a detailed coding scheme. From there, a second, independent coder coded all eligible studies using the same scheme. When a difference between the two coders arose surrounding the inclusion or exclusion of a particular study, the difference was reconciled by reviewing the study together.

D. Quality Assessment of Included Studies

The Center for Evidence Based Medicine, Levels of Evidence [16], provides "levels of evidence" criteria which includes five different levels of study quality (The Oxford 2011 Levels of Evidence). Within the five different levels, levels one and two also have an additional three sub-levels, while level three has two sublevels. Level one suggests the highest quality of study and includes systematic reviews, case-controlled studies, and cohort studies with 80 percent follow-up. Level two includes cohort studies, including retrospective cohort study or follow-up of untreated control patients in a randomized controlled trial, as well as ecological studies. Level three includes case controlled studies, while level four focusing on case series research or poor quality cohort or case controlled studies. Finally, level five involves expert opinion type reports. The CEBM "levels of evidence" were first introduced in 1998, as a means of providing researchers with a tool to help them determine the most appropriate forms of research and evidence to include in their research. Currently the "levels" are essentially a heuristic, or short-cut to finding the likely best evidence (The Oxford 2011 Levels of Evidence).

The CEBM "levels of evidence" were employed in the present meta-analysis to assess the overall quality of the studies included (The Oxford 2011 Levels of Evidence). Studies were initially reviewed based on the design tree as proposed by Bennett and Emberson [17]. This design tree provided a guide as to where on the CEBM "levels of evidence each study would be categorized. Studies are originally classified as either descriptive or analytic. Analytic studies, which were the studies included in the present meta-analysis, attempt to quantify the relationship between two factors, while descriptive studies on the other hand provide a picture of what is occurring in a given population. Once the studies were properly categorized, each was then assigned a level based on the CEMB "levels of evidence".

E. Analyses

Data analysis in this meta-analysis involved a sequence of steps. The first step was descriptive statistics. Specifically, the studies included in the meta-analysis were described with regards to participant characteristics (e.g., age, sex), study

characteristics (e.g., research design, sample size), and outcome constructs (e.g., individual effect sizes). Whatever the set of effect sizes under investigation, it is often of interest to examine the characteristics of those studies that yield statistically significant results. Descriptive analysis essentially describes the key results and various important attributes of the studies under investigation. Indeed, it can be argued that providing a broad description and appraisal of the nature and quality of the body of research under examination is fundamental to all other analyses [18]. Descriptive analyses were conducted by creating participant characteristic and study characteristic tables to look for trends and access ranges. Furthermore, the means for participants' age, sample size, and overweight status were calculated.

The next step involved expressing individual results in a standardized format. According to Egger and colleagues [14], in order to synthesize the studies included in a meta-analysis, results from individual studies must be expressed in a standardized format. In general, results from a meta-analysis are analyzed in terms of effect sizes. An effect size is a measure of the strength (magnitude) and direction of the relationship between two variables, and necessitates either a control group or pre- and post-test for comparison [19]. While there are numerous different types of effect sizes, one particular example is odds ratio (OR). OR is an effect size that is used when the outcome is binary (e.g., obesity versus average weight). This particular effect size facilitates ease in the combination of data, and in testing overall significance [20]. Closely tied with OR is relative risk (RR). While OR examines the number of participants who fulfill the criteria for a given endpoint, divided by the total number of participants who do not, RR, on the other hand, calculates the number of participants who fulfill the criteria for the outcome, divided by all participants [14].

In the present meta-analysis, the effect size of focus will be OR but the RR will also be provided to assist with overall interpretation. ORs will be calculated through the use of the Comprehensive Meta-analysis program [21]. Raw data from each study on the prevalence of overweight and/or obese, as well as non-overweight offspring were compared for family history of obesity and offspring overweight and/or obesity. Results from each trial were graphically displayed with their confidence intervals in a forest plot. The 95% confidence interval would contain the true underlying effect in 95% of the instances, if the study was to be repeated multiple times.

The effect sizes calculated in the present meta-analysis will be used to answer the research questions. Specifically, with regards to family history of obesity, the effect sizes will determine whether or not family history of obesity was a significant risk factor for the development of offspring overweight and/or obesity, if the family member assessed impacted the development of overweight and/or obesity, and when family history of obesity was present, were offspring more likely to become overweight or obese.

The third step involved examining the potential homogeneity or heterogeneity between study results. The Q statistic test for heterogeneity was calculated to assess whether the individual study results were likely to reflect a single underlying effect, as opposed to a distribution of effects. In other words, the Q statistic was used to explore whether the observed variability in the distribution of effect size estimates was greater than would be expected from sampling error. If the Q statistic fails to detect heterogeneity among results, then it is assumed that the differences observed between individual studies are a result of the sampling variation and chance. If the Q statistic is significant, further analyses are then warranted to identify the sources of this observed variability. The forest plots of the effect sizes will be examined to identify potential moderating variables.

In the final step, results were then interpreted by estimating an overall effect by combining the data from all studies. Although different statistical methods exist for combining the data, the method employed for this meta-analysis was a weighted average of the results. In a weighted average, the results from large sample studies tend to have more weight than smaller ones; inversely, results from smaller studies are more susceptible to chance and should be given less weight [14]. The statistical technique of calculating the weighted average can be generally classified into fixed-effect model, and random-effect model. Fixed-effect model considers the variability of the results as a random variation, and individual studies are simply weighted by their precision, meaning that the fixed-effect model only looks at within-study variations. Random-effect model, on the other hand, assumes a different underlying effect for each study, and accounts for this as an additional source of variation, being randomly distributed. In other words, random-effect model takes into account both between-study and within-study variations. Overall, the random-effect model leads to more weight being given to smaller studies and also to wider confidence intervals than the fixed-effect model [14]. In fact, the use of the random-effect model has been advocated for when there is heterogeneity between study results. Furthermore, the random-effects model allows for increased generalization of the findings to other studies with differing characteristics [22]. This meta-analysis calculated the weighted average of the studies using a random-effect model.

F. Moderator Analysis

A moderator variable is essentially an independent variable that interacts with another independent variable in predicting scores on and accounting for variance in a dependent variable [23]. Several factors including age of the participants and place of study were hypothesized to have potential effects on the homogeneity of studies. Indeed, individual studies where the weight of children has been examined at multiple ages have demonstrated higher odds ratios for obesity at younger ages than at older ages [1], [24]. Further, in the examination of individual studies, varying odds ratios have been discovered depending on the country where the study was conducted. Should heterogeneity among studies be discovered, then moderator analyses for both age of the child and location of the study will be conducted.

These two factors (age of children and location of study)

were chosen at the beginning of the study as potential moderator variables as previous research had shown varying findings in individual studies. For example, research examining offspring at age four, five, and six discovered that six year old offspring were at an greater risk of being classified as overweight and/obese as four year old offspring [25]. In terms of location of the study, research indicates that the cultural values of a given region may influence factors such lifestyle choices, including smoking-related behaviour, dietary choices, and physical activity intake which may ultimately influence the results of a given study. When statistical tests suggest heterogeneity in results, moderator analyses are justified (23).

Moderator analyses were conducted by grouping the studies into categories related to each moderator variable. Specifically, in examining age of the offspring, studies were grouped as either including participants over six or six and under. In terms of location of the study, each study was grouped into one of four categories, North America, South America, Europe, or Asia. In the case of moderator analyses, the Q statistic, instead of the OR will be examined. A significant Q statistic suggests that the variable in question appears be moderating the overall effect sizes, thus partially explaining the heterogeneity in the findings.

IV. Results

A. Prevalence

The literature review yielded 11 studies eligible for inclusion in the meta-analysis of family history of obesity and risk for overweight and obesity in childhood. The studies included 39,386 children, and represent pregnancies that occurred from 1980 to 2003 in Europe, Asia, North America, and South America. The prevalence of maternal overweight status ranged from 12% to 43%, while the prevalence of paternal overweight ranged from 16% to 55%. On average, 22% of mothers were overweight, compared to 37% of fathers. With the exception of one study, the prevalence of overweight was higher in fathers than it was in mothers [26]. The prevalence of childhood overweight ranged from 11% to 36%, with lower rates being discovered for childhood obesity (2%-17%). 18% of the entire population of children were classified as overweight, and 6% met the criteria for obesity. The children were between the ages of 3 and 12 at the time their BMI status was assessed.

B. Quality Assessment

As per the CEBM “levels of evidence” (2011) all of the included studies were coded based their assessed quality. As all of the studies were cohort or retrospective studies, they were classified under the second level of quality. Variations in the sublevels, however, were discovered. Of the 11 studies included in the meta-analysis, two were classified as having sub-level ‘a’ quality, while the remaining nine were assessed with sublevel ‘b’ quality. The principal difference between the two sub-levels was that three studies in sublevel ‘b’ had homogeneity in the results.

C. Effect Size Results

The Comprehensive Meta-Analysis [21] software program was used to assist in the calculation, storage and analysis of effect size estimates. The effect size estimates for family history of obesity were computed, including family history of obesity and offspring overweight and obesity, maternal weight and offspring overweight and obesity, paternal weight and offspring overweight and obesity, as well as family history of obesity and offspring obesity versus overweight status.

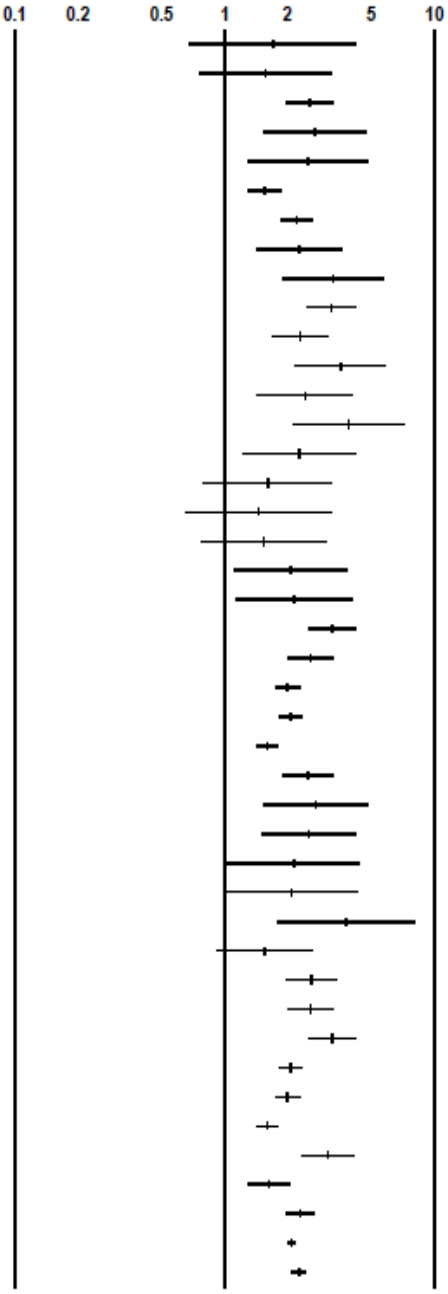
D. Is Family History of Obesity a Significant Risk Factor of Overweight and/or Obesity in Offspring?

With regards to family history of obesity and offspring overweight and/or obesity, the initial research question queried whether or not family history of obesity was s significant risk factor for the development of overweight and/or obesity in offspring. A total of 11 studies were included in the analysis. Children with a family history of obesity were at an elevated risk for overweight and obesity (pooled adjusted odds ratio (OR) 2.27, 95% CI: 2.09-2.46, P for heterogeneity < 0.001 compared with children who did not have a family history of obesity (see Fig. 1). The pooled relative risk (RR) was 1.63, 95% CI: 1.54-1.73, P for heterogeneity < 0.001. This means that children with a family history of obesity are over one and a half times more likely to become overweight and/or obese than offspring with no family history of obesity. The study by Hui and colleagues [6] yielded the greatest effect (OR) 3.92, 95% CI: 2.12-7.25, but excluding that study, only slightly influenced the pooled odds ratio (OR) 2.23, 95% CI: 2.06-2.40, P for heterogeneity < 0.001.

E. Does the Risk for Offspring Overweight and/or Obesity Associated with Family History vary Depending on the Family Members Included in the Analyses?

As it was discovered that family history of obesity was indeed a significant risk factor for the development of overweight and/obesity in offspring, the next question examined whether the risk for overweight and /or obesity varied depending upon the family member examined. Specifically, where the weight status of mothers more salient in terms of increasing the odds of offspring overweight and/or obesity than father, or vice versa? When the risk for offspring overweight or obesity was examined focusing solely on maternal history of overweight or obesity status, the pooled odds ratio were higher (OR) 2.39, 95% 2.05-2.80, P for heterogeneity < 0.001 than when paternal history of overweight or obesity was examined (OR) 2.08, 95% CI: 1.91-2.26, P for heterogeneity < 0.001 (see Figs. 2 and 3). The relative risk was higher when maternal obesity was examined (RR) 1.62, 95% 1.48-1.77, P for heterogeneity < 0.001 than when paternal history of overweight or obesity was examined (RR) 1.41, 95% CI: 1.35-1.46, P for heterogeneity < 0.001.This data provided support that the weight increases the odds of offspring overweight and/or obesity more than the weight status of father does. Still, when fathers were obese, children were at a significantly greater risk for becoming overweight and/or obese than if fathers were not obese.

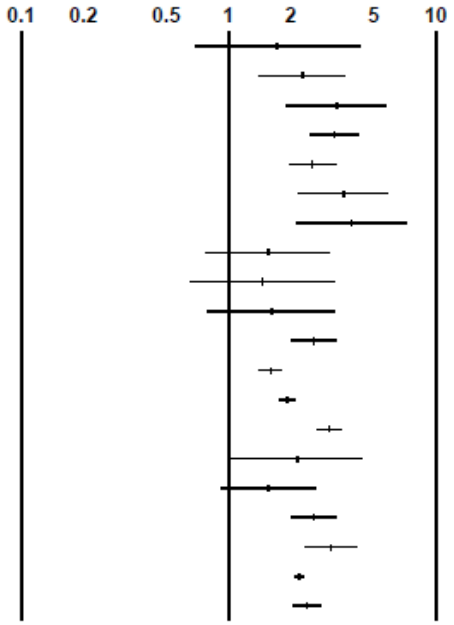
Study Citation	Year	RR (Random) 95% CI
[1]		
[1]		
[10]		1.71 (0.68 – 4.26)
[10]	2005	1.56 (0.77 – 3.23)
[10]	2005	2.54 (1.96 – 3.29)
[10]	2004	2.69 (1.54 – 4.71)
[10]	2004	2.50 (1.30 – 4.81)
[10]	2004	1.55 (1.29 – 1.86)
[10]	2004	2.21 (1.86 – 2.63)
[10]	2004	2.27 (1.42 – 3.65)
[5]	2004	3.31 (1.89 – 5.79)
[5]	2004	3.25 (2.49 – 4.25)
[6]	2004	2.30 (1.70 – 3.13)
[6]	2000	3.58 (2.18 – 5.89)
[2]	2000	2.43 (1.44 – 4.09)
[2]	2003	3.92 (2.12 – 7.23)
[2]	2003	2.27 (1.21 – 4.24)
[2]	2005	1.61 (0.80 – 3.27)
[2]	2005	1.45 (0.65 – 3.23)
[2]	2005	1.55 (0.79 – 3.06)
[2]	2005	2.07 (1.11 – 3.87)
[30]	2005	2.16 (1.14 – 4.11)
[30]	2005	3.28 (2.54 – 4.22)
[30]	2007	2.58 (2.02 – 3.31)
[30]	2010	2.00 (1.75 – 2.29)
[30]	2007	2.07 (1.83 – 2.35)
[30]	2007	1.60 (1.41 – 1,81)
[30]	2007	2.50 (1.90 – 3.29)
[7]	2010	2.74 (1.54 – 4.87)
[7]	2007	2.53 (1.50 – 4.26)
[7]	2007	2.14 (1.04 – 4.41)
[7]	2007	2.09 (1.00 – 4.32)
[7]	2007	3.81(1.79 – 8.13)
[7]	2007	1.56 (0.93 – 2.63)
[7]	2007	2.60 (1.97 – 3.43)
[8]	2005	2.58 (2.02 – 3.31)
[8]	2005	3.28 (2.54 – 4.22)
[8]	2005	2.07 (1.83 – 2.34)
[8]	2005	2.00 (1.75 – 2.29)
[8]	2005	1.60 (1.42 – 1.82)
[8]	2005	3.13 (2.34 – 4.12)
[9]	1999	1.63 (1.29 – 2.07)
[9]	1999	2.30 (3.57 – 5.06)
[26]	2005	**2.09 (2.01 – 2.17)**
		2.27 (2.09 -2.46)



Fixed Combined (41)
Random Combined (41)
Test for heterogeneity: x^2 = 1.26.62,*df* = 40 (*P*<0.0001)
Test for overall effect: Z = 19.69 (*P* <0.0001)

Fig. 1 Meta-analysis of random combined ORs – family history of obesity and risk for offspring overweight and obesity

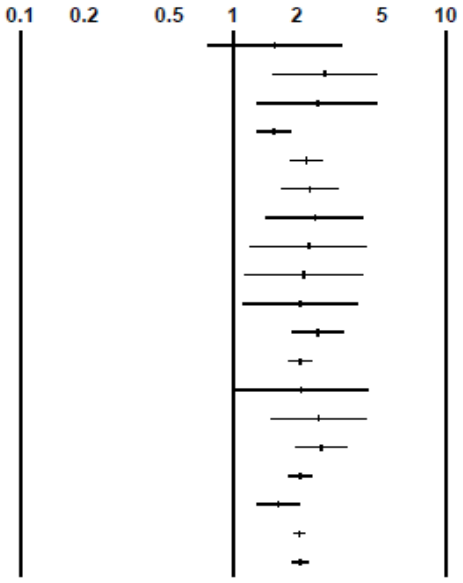
Study Citation	Year	RR (Random) 95% CI
[11]	2005	1.71 (0.66 – 4.26)
[10]	2004	2.27 (1.42 – 3.65)
[10]	2004	3.31 (1.89 – 5.79)
[10]	2004	3.25 (2.49 – 4.25)
[10]	2004	2.54 (1.96 – 3.29)
[5]	2000	3.58 (2.18 – 5.89)
[6]	2003	3.92 (2.12 – 7.25)
[2]	2005	1.55 (0.79 – 3.06)
[2]	2005	1.45 (0.65 – 3.25)
[2]	2005	1.61 (0.78 – 3.27)
[30]	2010	2.58 (2.20 – 3.31)
[30]	2010	1.60 (1.42 – 1.82)
[66]	2007	1.91 (1.75 – 2.10)
[66]	2007	3.07 (2.67 – 3.53)
[7]	2007	2.14 (1.04 – 4.41)
[7]	2007	1.56 (0.93 – 2.63)
[8]	2005	2.58 (2.02 – 3.31)
[9]	1999	3.13 (2.34 – 4.17)
Fixed Combined (18)		**2.19 (2.08 – 2.32)**
Random Combined (18)		**2.40 (2.05 – 2.80)**



Test for heterogeneity: x^2 = 87.18,*df* = 17 (*P*<0.0001)
Test for overall effect: Z = 10.99 (*P* <0.0001)

Fig. 2 Meta-analysis of random combined ORs –maternal history of obesity and risk for offspring overweight and obesity

Study Citation	Year	RR (Random) 95% CI
[1]	2005	1.57 (0.77 – 3.23)
[10]	2004	2.70 (1.54 – 4.71)
[10]	2004	2.50 (1.30 – 4.81)
[10]	2004	1.55 (1.29 – 1.86)
[10]	2004	2.21 (1.86 – 2.63)
[5]	2000	2.30 (1.70 – 3.13)
[6]	2003	2.43 (1.44 – 4.09)
[2]	2005	2.27 (1.21 – 4.24)
[2]	2005	2.16 (1.14 – 4.11)
[2]	2005	2.07 (1.11 – 3.87)
[30]	2010	2.50 (1.90 – 3.29)
[30]	2010	2.07 (1.82 – 2.35)
[7]	2007	2.09 (1.00 – 4.32)
[7]	2007	2.53 (1.50 – 4.26)
[8]	2005	2.60 (1.97 – 3.43)
[8]	2005	2.07 (1.83 – 2.36)
[9]	1999	1.63 (1.29 – 2.07)
Fixed Combined (17)		**2.06 (1.94 – 2.19)**
Random Combined (17)		**2.08 (1.91 – 2.23)**



Test for heterogeneity: x^2 = 21.52.70,*df* = 16 (*P*<.0001)
Test for overall effect: Z = 17.47 (*P* <.0001)

Fig. 3 Meta-analysis of random combined ORs –paternal history of obesity and risk for offspring overweight and obesity

F. When Family History of Obesity is Present, are Offspring at a Greater Risk for Developing Obesity or Overweight?

The purpose of the final research question was to investigate whether when family history of obesity was present, were the offspring more likely to become overweight or obese. Of the 11 studies included in the meta-analysis, eight provided specific effect sizes for family history of obesity and the risk of overweight in offspring, while six studies provided specific effect sizes for family history of obesity and risk for obesity in offspring. As a result, a subgroup analysis was performed to compare the risk for overweight versus the risk for obesity. Data from the subgroup analysis provide support for the final hypothesis. The pooled odds ratio for family history of obesity and risk for obesity (OR) 2.55, 95% CI: 2.20- 2.96, P for heterogeneity < 0.001 (see Fig. 4) was greater than the pooled odds ratio for family history of obesity and risk for overweight in offspring (OR) 2.04, 95% CI: 1.88-2.21, P for heterogeneity < 0.001. The pooled relative risk for family history of obesity and risk for obesity (RR) was 1.75, 95% CI: 1.60-1.94, P for heterogeneity < 0.001 was greater than the pooled odds ratio for family history of obesity and risk for overweight in offspring (RR) 1.55, 95% CI: 1.46-1.65, P for heterogeneity < 0.001. When family history of obesity is present, children are more likely to become obese than overweight. The pooled odds ratio for studies where maternal history of obesity was reported showed that offspring was at risk for overweight or obesity (OR) 2.40, 95% CI: 2.05-2.80, P for heterogeneity < 0.001 (see Fig. 5). It is important to note that children with a family history of obesity (either maternal or paternal) were still over two times at risk of becoming overweight/obese than children with no family history of obesity.

G.Moderator Analyses and Heterogeneity

The effect sizes calculated suggested that family history of obesity increased the odds of children being classified as overweight and/or obese. Despite these noteworthy findings, the Q test suggested heterogeneity of results for family history of obesity. A significant Q test suggests that the differences observed between studies were due to something other than chance. As a result of the detection of heterogeneity, moderator analyses for age at the time of overweight and/or obesity measurement and region of the study were conducted. It is important to note that while variables such as SES status, level of parental education, and dietary patterns were investigated in some studies, due to inconsistencies between the studies; those variables were unable to be investigated as potential moderator variables. The following sections will explain the results of the moderator analyses.

H.Age of the Child at Overweight and/or Obesity Measurement

An analysis of variance was calculated to determine whether there was a difference between the odds of offspring who also had a family history of obesity being classified as overweight and/or obese at six years of age and under versus over six years of age. Again, results suggest a statistically significant difference in the odds of offspring being identified as overweight and/or obesity when they were assessed at age six and under versus when the assessment was done after the age of six ($Q = 0.58$, $p < 0.05$). These results would suggest that the effects sizes for children six and under were larger than those of children older than six.

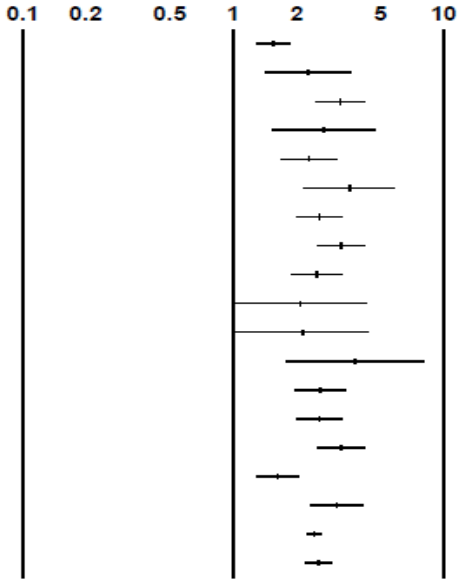
I. Region of the Study

An analysis of variance was calculated to determine whether there was a difference between the odds of offspring of children with a family history of obesity being classified as overweight and/or obese depending on the region of the world where the study was conducted. The same regions used in examining maternal smoking during pregnancy were applied with family history of obesity. There was a significant difference in the odds ratio of offspring being identified as overweight and/or obesity depending on the region where the study took place ($Q = 52.83$, $p < .0001$). This information suggests that the region where the study took place impacted the odds that offspring with a family history of obesity would develop overweight and/or obesity. Specifically, the largest effect sizes were discovered in the Asia region; offspring with a family history of obesity were almost two and a half times more likely to become overweight and/or obese than offspring with no family history of obesity. The effect sizes discovered in the regions of North America, South America and Europe were almost identical, with offspring with a family history of obesity being approximately two times more likely than offspring with no family history of obesity to be classified as overweight and/or obese.

J. Publication Bias

As previously stated, the purpose of a meta-analysis is to consolidate all of the existing research within a given area, not simply the statistically significant research. Despite this overall goal, to ensure credibility of the present meta-analysis, the effect size of each study was plotted to assess for publication bias. Plotting the natural logarithms of the odds ratio of studies with smaller sample sizes (which biases are more likely to occur) against their standard error is common practice [14]. The funnel plot analyses used to test for asymmetry between main and subgroup effect size calculations suggests that studies with smaller sample sizes scattering more widely in the lower portion of the graph reflecting less precision in the estimation of odds ratio thus supporting a non-publication bias in the development of offspring overweight and/or obesity as it pertains to maternal smoking during pregnancy and family history of obesity.

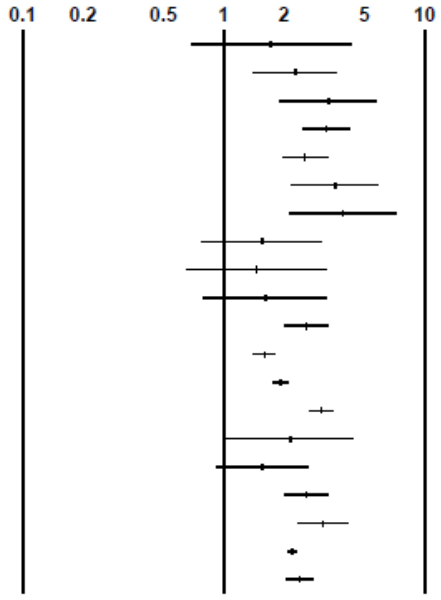
Study Citation	Year	RR (Random) 95% CI
[10]	2004	1.55 (1.29 – 1.86)
[10]	2004	2.27 (1.42 – 3.65)
[10]	2004	3.25 (2.49 – 4.25)
[10]	2004	2.69 (1.54 – 4.71)
[5]	2000	2.30 (1.70 – 3.13)
[5]	2000	3.58 (2.18 – 5.89)
[30]	2010	2.58 (2.02 – 3.31)
[30]	2007	3.28 (2.54 – 4.22)
[30]	2010	2.50 (1.90 – 3.29)
[7]	2007	2.09 (1.00 – 4.32)
[7]	2007	2.14 (1.04 – 4.41)
[7]	2007	3.81 (1.79 – 8.13)
[8]	2005	2.60 (1.97 – 3.43)
[8]	2005	2.58 (2.02 – 3.31)
[8]	2005	3.23 (1.75 – 2.29)
[9]	1999	1.63 (1.29 – 2.07)
[9]	1999	3.13 (2.34 – 4.12)
Fixed Combined (17)		**2.43 (2.24 – 2.59)**
Random Combined (17)		**2.55 (2.20 – 2.96)**



Test for heterogeneity: x^2 = 57.46,*df* = 16 (*P*<0.0001)
Test for overall effect: Z = 12.51 (*P* <0.0001)

Fig. 4 Meta-analysis of random combined ORs – Family History of Obesity and Risk for Offspring Obesity.

Study Citation	Year	RR (Random) 95% CI
[1]	2005	1.71 (.66 – 4.26)
[10]	2004	2.27 (1.42 – 3.65)
[10]	2004	3.31 (1.89 – 5.79)
[10]	2004	3.25 (2.49 – 4.25)
[10]	2004	2.54 (1.96 – 3.29)
[5]	2000	3.58 (2.18 – 5.89)
[6]	2003	3.92 (2.12 – 7.25)
[2]	2005	1.55 (.79 – 3.06)
[2]	2005	1.45 (.65 – 3.25)
[2]	2005	1.61 (.78 – 3.27)
[30]	2010	2.58 (2.20 – 3.31)
[30]	2010	1.60 (1.42 – 1.82)
[66]	2007	1.91 (1.75 – 2.10)
[66]	2007	3.07 (2.67 – 3.53)
[7]	2007	2.14 (1.04 – 4.41)
[7]	2007	1.56 (.93 – 2.63)
[8]	2005	2.58 (2.02 – 3.31)
[9]	1999	3.13 (2.34 – 4.17)
Fixed Combined (18)		**2.19 (2.08 – 2.32)**
Random Combined (18)		**2.40 (2.05 – 2.80)**



Test for heterogeneity: x^2 = 87.18,*df* = 17 (*P*<0.0001)
Test for overall effect: Z = 10.99 (*P* <0.0001)

Fig. 5 Meta-Analysis of Random Combined ORs –Maternal History of Obesity and Risk for Offspring Overweight and Obesity

V. Discussion

A. Prevalence

Based on the participant characteristic, and the means calculated, it was discovered that the prevalence rates of maternal overweight status ranged from 12% to 43% while the prevalence of paternal overweight ranged from 16% to 55%. Higher prevalence rates of parental overweight status tended to be discovered in North American studies. This finding is supported by research that suggests that overweight and/or obesity rates tend to be the highest in North America (e.g., 34.4. percent in United States [27], on average, 22% of mothers were overweight, compared to 37% of fathers. This finding is comparable to Canadian prevalence rates that suggest that approximately 24% of adults are overweight [28]. This difference in maternal and paternal overweight prevalence is inconsistent with worldwide obesity trends, which suggest that the prevalence of overweight and/or obesity in men and women is similar or in some case higher for women than it is men [29]. This gender difference is often explained by the difference in body fat distribution seen in men and women. However, behavioural and socio-cultural factors also appear to play an important role. As such, the differences in the findings from the present research and existing research may rest in term of behavioural and socio-cultural factors. Further the overall prevalence of paternal overweight appears to be inflated by a few extreme prevalence rates from single studies [30].

The prevalence of childhood overweight ranged from 11% to 36%, with lower rates being discovered for childhood obesity (2%-17%). 18% of the entire population of children were classified as overweight, and 6% met the criteria for obesity. Again, these prevalence rates of childhood overweight and/or obesity are similar to both Canadian and worldwide rates [31]-[32]. The younger the children were at the time of the overweight and/or obesity assessment, the higher prevalence rates that were discovered.

B. Quality of Studies

As previously mentioned, the CEBM levels of evidence were used as a means of assessing the overall quality of the individual studies included in the meta-analysis. The CEBM levels of evidence provide researchers with a guideline to help them determine the most appropriate forms of evidence to include in their research. In terms of family history of obesity, two studies were classified as having sublevel ‘a’ of level two quality, while the remaining nine were assessed with sublevel ‘b’ quality.

From a research and practical standpoint, the studies included in the present meta-analysis, were each conducted using sound research procedures and methodology, such as adequate sample sizes and parallel cohort groups where those with the condition in the first group were compared to those without the condition in the second group. The distinguishing feature that impacted the sublevel assigned to the included studies was the degree of follow-up that the study had. Those studies classified as having sublevel ‘b’ quality had less than 80% follow-up, which had the potential to somewhat altered the overall findings of these studies Overall, however, based on the CEBM criteria each of the studies included would be given an overall grade of B in terms of their quality. Indicating that they have reasonable internal and external validity to be generalized to the population of interest, namely, offspring of mothers who smoke during pregnancy.

C. Is Family History of Obesity a Significant Risk Factor of Overweight and/or Obesity in Offspring?

This is the first meta-analysis to investigate family history of obesity and offspring overweight and/or obesity, and consistent findings were discovered. The literature review yielded 11 studies eligible for inclusion in the meta-analysis of family history of obesity and subsequent offspring overweight and/or obesity. The studies included 39,386 children and represent pregnancies that occurred from 1980 to 2003 from various socioeconomic backgrounds in Europe, Asia, North America, and South America. The prevalence of maternal overweight status ranged from 12% to 43%, while the prevalence of paternal overweight ranged from 16% to 55%.On average, it was discovered that 22 percent of mothers and 37 percent of fathers were overweight and/or obese. These findings are consistent with overall prevalence rates across the general population of Canada, which suggests that a higher rate of overweight and/or obesity exists amongst men than women. Studies involving children over the age of 12 were not included in the analyses.

The results of this meta-analysis answered the first research question posed in this study. Specifically, family history of obesity was proven as a significant risk factor for the subsequent development of childhood overweight and/or obesity. While individual studies throughout the obesity risk factor literature have often highlighted the risk family history of obesity poses to the development of overweight and/or obesity in offspring, this is first meta-analysis of its kind. While individual studies included in this meta-analysis reported varying effect sizes, all studies included in the meta-analysis found a positive association between family history of obesity and subsequent childhood overweight and/or obesity. For the individual studies included in this meta-analysis of offspring overweight and/obesity in which a family history of obesity was present, the odds ranged from one-and-a-half times more likely to just under four times more likely. Even when the study producing the largest effect size was removed from the equation [6], the odds of a child born into a family with a history of overweight and/or obesity developing to become overweight and/or obese were over one and a half times greater than for children with no family history of overweight and/or obesity. These finding suggest that family history of obesity is consistently associated with the development of overweight and/or obesity in offspring.

Two principal explanations can account for why a family history of obesity significantly increases the odds for the development of overweight and/or obesity in children: nature and nurture. In terms of nature, the significant role genetic and biological factors play in the development of eating pathology

is becoming increasingly clear [33]. Indeed, a positive association between maternal pre-pregnancy or early pregnancy BMI and offspring BMI in later life has been discovered [34]. Familial transmission of risk for overweight and/or obesity can be observed early in a child's development, and has been hypothesized to arise from an inheritance of non-modifiable genes [35]. Indeed, obesity may be classified into three main categories on the basis of genetic etiology: monogenic, syndromic and polygenic, with monogenic and syndromic forms most often evident in early life [36].

Studies involving heritability of weight first emerged in the early 20th century, with research conducted by Davenport [37] demonstrating a clear association between offspring and parental weight. This early research, however, failed to distinguish the role of genetics versus environmental factors in the determination of weight status in offspring. Over the next seventy years, research emerged specifically investigating the effect of genes on the development of overweight and/or obesity in children. Heritability is defined as the proportion of within-population phenotypic variance attributable to within-population genetic variance [38]. Heritability values range from 0.0, where genes do not contribute at all to the phenotypic variance between individuals, to 1.0, where genes are the sole contributors to phenotypic differences between individuals. Heritability is information at the population level, not at the individual level. Specifically, a heritability of 0.60 informs us that approximately 60 percent of the individual difference (i.e., body weight) that we observe may be in some way attributable to genetic individual difference. It does not, however, signify that 60 percent of the individual's body weight is the result of genetic factors, while the remaining 40 percent is due to environmental factors [39]. In the case of overweight and/or obesity, heritability cannot be viewed as a fixed entity, as the proportion of the phenotype that can be attributed to or by the genotype will depend upon the exposure to environmental factors (e.g., dietary patterns, lifestyles choices) that vary significantly depending on the individual.

Familial studies provide robust evidence for the heritability of body weight [40]. Twin studies in which monozygotic and dizygotic twin pairs are compared have provided evidence to support the contribution of genetics to body weight. Several different twin cohort studies have discovered large heritability coefficients, ranging from 0.6 to 0.8 [41]-[43]. Furthermore, a recent systematic review of twin and adoptive studies examining the genetic and environmental influences on childhood obesity found that while adoption studies supported the influence of the family environment on the development of obesity in children, correlations were substantially stronger between parents and their biological offspring [44]. Taken together, such evidence suggests that genetics plays a vital role in the development of obesity.

With regards to nurture, parents strongly influence the dietary patterns of their children [45]. In fact, parents have been hypothesized to be the central agents in fostering either healthy or unhealthy food intake [46]. The idea that parents influence what and how much food their children consume is not a novel idea. Ultimately, in any given household (especially when children are younger than 12), it is parents who determine what food will be available to their children, both through the purchasing of this food as well as its preparation [47]. If parents are regularly cooking or purchasing high caloric, low-nutrient foods as opposed to healthy foods high in nutritional value, their children have no choice but to eat these foods. In addition to providing their children with food, parents also act as role models, modeling food choices and eating habits [46]. Research suggests that parents' own healthy eating habits are related to a healthy diet in their children [48].

D.Does the Risk for Offspring Overweight and/or Obesity Associated with Family History Vary Depending on the Family Members Included in the Analyses?

Results from the present meta-analysis answered the second research question as to whether the risk for offspring overweight and/or obesity associated with family history vary depending on the family members included in the analyses. Interestingly, when the weight status of mothers and fathers are investigated separately, results consistently suggest that a history of maternal obesity is more salient than paternal obesity with regards to future offspring obesity [4], [6], [49]. One possible explanation for why maternal history of obesity has been shown to increase the risk for future offspring obesity more than that of paternal history of obesity lies within the amount of time mothers spend with their children compared to fathers. Although fathers are increasingly taking a more active role in the daily care of their children, mothers still report doing the majority of the housework, including meal preparation [50], [51]. As such, if the primary caregiver of a child makes unhealthy lifestyle choices themselves (e.g., lack of physical activity, excessive eating, consumption of sugary, high-fat foods, etc.), it is not surprising that their children will also adopt similar lifestyle choices, thus potentially perpetuating the overweight and/or obese weight status.

Intrauterine mechanisms provide another possible explanation as to why maternal history of obesity increases the odds of overweight and/or obesity in offspring more than paternal obesity does. For example, the developmental over-nutrition, also known as the fetal teratogenesis hypothesis, posits that the greater delivery of glucose to the fetus during pregnancy results in fetal hyperinsulinemis, which as a consequence increases insulin-mediated growth. This theory was originally proposed in the 1950s to explain the association between maternal diabetes during pregnancy and excessive growth in the developing fetus [52]. In the 1980s, this theory was broadened to include the possibility that other fuels in addition to glucose but also related to maternal diabetes, such as fatty acids and amino acids, contributed to increased fetal growth [53]. While this original hypothesis was specific to intrauterine growth, recent research has suggested a strong positive correlation between fetal growth and later BMI [54]. In further support of the developmental

hypothesis, a high concentration of maternal glucose has been shown to increase nutrient (fatty acids, amino acids) transfer to the fetus, which results in fetal hyperinsulinemia and increased fetal growth [53]. Hyperinsulinemia has been found to not only be associated with fetal growth, but with the subsequent development of obesity [54], [55].

The developmental over-nutrition hypothesis provides an explanation as to why the offspring of mothers with diabetes are at an increased risk for becoming overweight and/or obese. Association between excessive weight gain during pregnancy in healthy, non-diabetic mothers and overweight and/or obesity in their offspring has been receiving increasing interest in the literature [56], [57]. In this respect, the developmental over-nutrition hypothesis has been expanded to include not only diabetes in pregnancy, but also greater maternal adiposity during pregnancy as a key risk factor in the development of offspring overweight and/or obesity.

Overall, the results from this meta-analysis are important as they demonstrate the necessity of including the family (both mothers and fathers) into overweight/obesity prevention plans, suggesting a family approach to prevention is more effective than an individualized approach. Indeed, although the weight status of mothers increases the likelihood of obesity in offspring by just under two-and-a-half times, the weight status of fathers (while not as salient as mothers) still increases the risk for offspring overweight and/or obesity by over two times compared to children who do not have fathers who are obese. Thus, the results from this meta-analysis support the implementation of family-based intervention and treatment programs.

E. When Family History of Obesity is Present, are Offspring at a Greater Risk for Developing Obesity or Overweight?

The final research question examined whether when family history of obesity was present in either mothers or fathers, were offspring at a greater risk for becoming obese or overweight. Results from this meta-analysis suggest that the odds of an offspring becoming obese when there is a family history of obesity is greater than the odds of becoming overweight. Still, the odds of becoming overweight remained statistically significant. As was the case with maternal smoking during pregnancy and offspring overweight and/or obesity, overweight and obesity are not identical constructs and therefore must be analyzed individually to understand as they each have significant negative health outcomes associated with them [28], as well as varying prevalence rates. Indeed, as mentioned above the prevalence rates of overweight for both adults and children have been found to be consistently higher than the prevalence rates of obesity have consistently been higher than for obesity [58]. This meta-analysis thus provided the relative risk not only for obesity but also for overweight status.

F. Age of the Child at Overweight and/or Obesity Measurement

Based on a visual inspection of the descriptive statistics, the age of the child at the time of the overweight and/obesity assessment appeared to produce some difference in the effects sizes of the studies. Specifically, in studies that examined children six years or age and under, larger effects sizes tended to be reported than in studies that assessed children for overweight and/or obesity after the age of six. As a result, a moderator analysis, using an analysis of variance was calculated to determine whether or not a statistically significant difference existed. Results from the moderator analysis suggest a significant difference; offspring under the age of six who had a family history of obesity were at an increased risk of developing overweight and/or obesity than offspring six years and over with a family history of obesity.

Dietary and lifestyle choices such as amount of physical activity may partially explain the difference the higher effect size for overweight and/or obesity in children under the age of six. The overwhelming majority of the studies included in the meta-analysis did not focus on physical activity or dietary choices or parents, thus preventing their analyses. However, it has been hypothesized that when parents engage in poor dietary or lifestyle choices, these behaviours will be modeled to their children [47]. Further, at a young age, parents are the one that often make diet and lifestyle choices for their children, which increases the likelihood that children will eat the same foods and receive similar amounts of physical activity as their parents [46].

The increasing number of socialization agents that children come into contact with as they age may provide an explanation for why the effect sizes are not as high for children older than six. As children enter school, numerous socialization sources, including their peers or even the social climate of their school begin to influence the lifestyle decision they make [59]. While parents may still largely determine the types of food that children consume, children's opportunity for physical activity increase significantly once they are in the school setting. In fact, many of the current prevention efforts are targeted at school-aged children, with some programs even occurring directly in the school setting [60]. Thus as children get older, they begin to have more opportunities to make their own healthy lifestyle choices, thus suggesting that the influence of prenatal or early childhood risk factors for overweight and/or obesity may decline over time.

G. Region of the Study

Another variable thought to influence the homogeneity of results was the region of the study. Specifically, studies included in the meta-analysis were conducted in the regions of North America, South America, Asia, and Europe. An analysis of variance was calculated to determine whether there was a difference between the risk of offspring of mothers who had a family history of obesity being classified as overweight and/or obese depending on the region of the world where the study was conducted. Results from the moderator analysis suggest that the region where the study took place impacted the odds that offspring of mothers who had a family history of obesity would develop overweight and/or obesity.

This finding may at least be partially explained by the specific cultural norms and expectations, as well as the

attitudes towards obesity for the various regions where the studies took place. For example, dietary patterns and lifestyle choices often depend upon the culture that a given population is living in. For example, the United Kingdom, very few schools have athletic or sports programs [61]. As lifestyle patterns vary among the varying regions across the world, it should therefore come as no surprise that the region in which a study takes place influence the effect sizes of overweight/ and obesity in children whose mothers have a family history of obesity.

VI. Conclusion

One particular area that warrants further research is the investigation of the interplay between genetic and socioeconomic factors on the development of overweight and/or obesity in children. In has been suggested that in developed countries children from lower SES backgrounds are at an increased risk for overweight and/or obesity [62], while in less developed countries, children from higher SES backgrounds are at an increased risk for overweight and/or obesity [63]. Indeed, as obesity moves away from a first world condition to more of a global concern, we must understand not only how genetics impacts weight, but also the overall environment, including SES factors, in which we live. Increasingly children with no prior family history of obesity are increasingly becoming overweight and/or obese [64]. Future research is thus needed to examine the effects of socioeconomic factors on the development of obesity.

A sound body of evidence exists supporting the hypothesis that breastfeeding serves as a protective factor against the development of offspring overweight and/or obesity [65], Limited information, however, currently exists regarding how breastfeeding may interact with family history of obesity for overweight and/or obesity in children. Future research is thus needed to examine if protective factors such as breastfeeding can counteract the negative effects of risk factors such as family history of obesity.

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