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Trajectoires développementales de l'IMC durant l'enfance : Une étude longitudinale sur 8 ans.

par
Laura Elizabeth Pryor

Départment de médecine sociale et préventive
Faculté de médecine

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Ce mémoire intitulé :
Trajectoires développementales de l'IMC durant l'enfance :
Une étude longitudinale sur 8 ans.

présentée par :
Laura Elizabeth Pryor

a été évaluée par un jury composé des personnes suivantes :

Louise Séguin
président-rapporteur

Sylvana Côté
directeur de recherche

Tracie A. Barnett
membre du jury

Résumé de recherche

Trajectoires développementales de l'IMC durant l'enfance :
Une étude longitudinale sur 8 ans.

Introduction : L'obésité infantile, origine de nombreux problèmes de santé, représente un grand défi en santé publique. Récemment, l'importance d'étudier l'évolution du surpoids durant l'enfance ainsi que les facteurs de risques précoces pour l'obésité a été reconnue. Les trajectoires développementales d'indice de masse corporelle (IMC) chez les jeunes représentent une approche innovatrice qui nous permet de mieux comprendre cette problématique importante.

Objectifs: 1) Identifier des trajectoires développementales distinctes de groupes d'enfants selon leur IMC durant l'enfance, et 2) Explorer les facteurs de risques précoces qui prédisent l'appartenance de l'enfant à la trajectoire d'IMC le plus élevé

Hypothèses: 1) On s'attend à retrouver un groupe d'enfants qui suit une trajectoire d'IMC élevée durant l'enfance. 2) On s'attend à ce que certaines caractéristiques de la mère (ex : tabac pendant la grossesse et IMC élevé), soient associées à l'appartenance de l'enfant au groupe ayant la trajectoire «IMC élevé ».

Méthodes: Estimation des trajectoires développementales d'IMC d'enfants, dans un échantillon populationnel (n=1957) au Québec (ELDEQ). Les IMC ont été calculés à partir de données fournies par les mères des enfants et recueillis chaque année sur une durée de 8 ans. Des données propres à l'enfant sa mère, ainsi que socioéconomiques, ont étés recueillies. Une régression logistique multinomiale a été utilisée pour distinguer les enfants avec un IMC élevé des autres enfants, selon les facteurs de risques précoces.

Les programmes PROC TRAJ (extension de SAS), SPSS (version 16), et SAS (version 9.1.3) ont été utilisés pour ces analyses.

Résultats:

Trois trajectoires d'IMC ont étés identifiées : IMC « bas-stable » (54,5%), IMC « modéré » (41,0%) et IMC « élevé et en hausse » (4,5%). Le groupe « élevé et en hausse » incluait des enfants pour qui l'IMC à 8 ans dépassait la valeur limite pour l'obésité. Les analyses de régression logistique ont révélé que deux facteurs de risques maternels étaient significativement associés avec la trajectoire "en hausse" par rapport aux deux autres groupes : le tabac durant la grossesse et le surpoids maternel.

Conclusions:

Des risques d'obésité infantile peuvent êtres identifiés dès la grossesse. Des études d'intervention sont requises pour identifier la possibilité de réduire le risque d'obésité chez l'enfant en ciblant le tabac et le surpoids maternelle durant la grossesse.

Mots clés: Indice de masse corporelle (IMC), obésité infantile, trajectoires développementales de groupe, facteurs de risque précoce, étude populationnelle, tabac pendant la grossesse, obésité maternelle.

Abstract

Developmental Trajectories of Body Mass Index in Early Childhood:
An 8-Year Longitudinal Study.

Introduction: Childhood obesity has become one of the greatest Public Health challenges this century, affecting not only developed nations, but increasingly low- and middle-income countries as well. Estimating developmental trajectories of Body Mass Index (BMI) during early childhood represents an innovative approach towards a better understanding of the development of this health problem.

Objective: To identify groups of children with distinct developmental trajectories of Body Mass Index (BMI) between the ages of five months and eight years, and to identify early-life risk factors that distinguish children in an atypically elevated BMI trajectory group.

Methods: Group-based developmental trajectories of BMI were estimated from annual maternal assessments (5 months to 8 years) in a large population sample (n=1957). Measures of height and weight, as well as family and child characteristics were obtained yearly from mothers. Multivariate logistic regression was used to distinguish children with elevated BMI from other children, using pre and early post-natal risk factors.

Results: Three trajectories of BMI were identified: low-stable BMI (54.5%), moderate BMI (41.0%) and high-rising BMI (4.5%). The high-rising group included children whose BMI, at eight years of age, exceeded the cut-off value for obesity. Multinomial logit regression analyses revealed that two maternal risk factors were significantly associated with the high-rising BMI trajectory group as compared to both the low and moderate groups: smoking during pregnancy and maternal overweight.

Conclusions: Antecedents of childhood obesity can be identified during pregnancy. Intervention studies are needed in order to test the possibility that targeting maternal smoking and maternal obesity during pregnancy would reduce the risk of childhood obesity in the offspring.

Keywords: Body Mass Index (BMI), child obesity, Group-based developmental trajectories, early life predictors, population-based study, maternal smoking, maternal obesity.

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List of Abbreviations

AR : Adiposity Rebound

BIC : Bayesian Information Criterion

BMI : Body Mass Index

CDC: Center for Disease Control

CI: Confidence Interval

CVD: Cardiovascular Disease

DEXA: Dual energy x-ray absorptiometry

ÉLDEQ : Étude longitudinale du développement des enfants du Québec

GDM : Gestational Diabetes Mellitus

IMC : Indice de masse corporelle

IOTF: International Obesity Task Force

ISQ: Institut de la Statistique du Québec

NLSY79: National Longitudinal Study of Youth ‘79

QLSCD : Québec Longitudinal Study of Child Development

OR: Odds Ratio

SES : Socioeconomic Status

SD: Standard Deviation

SE: Standard Error

SGA: Small for Gestational Age

WC: Waist Circumference

WHO : World Health Organization

WHR: Waist-to-hip ratio

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CHAPTER 1: Introduction

1.1 Background

Several epidemiologic studies show a drastic increase in the prevalence of obesity among children over the past decades (Ebbeling, 2002, Wang & Lobstein, 2006). As much as 20-30% of children in North America, Europe and parts of the Western Pacific are overweight or obese (Wang & Lobstein, 2006). Initially touching mostly on industrialized nations, this problem is increasingly prevalent in developing countries (WHO, 2004), creating an odd paradox, wherein the conditions of undernutrition and obesity often co-exist within the same household (Caballero, 2005). It has even been hypothesized that obesity may replace undernutrition and infectious disease as the most significant contributor to ill health worldwide (Kopelman, 2000).

Overweight and obesity can be broadly defined as an abnormal or excessive accumulation of fat that presents a health risk (WHO, 2006). The Body Mass Index (BMI), a measure associating an individual's weight to their height, is commonly used to assess overweight and obesity. Overweight is considered as a BMI equal to or greater than 25 kg/m^2. A BMI equal or greater than 30 kg/m^2 is considered to be indicative of obesity in an individual, and further classifications exist for increasing gradations of obesity severity (James, 2001, WHO, 2004). It is well established that overweight and obese individuals are at an increased risk for several chronic diseases; in particular, cardiovascular disease (CVD), type 2 diabetes mellitus, certain forms of cancer (Samanic, 2006), respiratory complications, and osteoarthritis of large and small joints, and that these risks, as well as the risk for decreased longevity, increase according to the severity of the weight problem (Kopelman, 2000, Walley, 2006, WHO, 2006).

Of particular concern is the growing number of overweight and obese children. According to the 2004 Canadian Community Health Survey, approximately 26% of youth aged 2-17 are overweight or obese, up from 15% in 1979 (Statistics Canada, 2005). A startling by-product of this is the dramatic increase in type 2 diabetes diagnoses among adolescents, a phenomenon once rare in youth (Ebbeling, 2002, Fagot-Campagna, 2000). There is also evidence of early coronary atherosclerosis among obese male adolescents (Malcolm, 1997). In addition, an overweight child may suffer a considerable negative psychosocial impact (Erickson, 2000, Strauss, 2000), numerous endocrine disorders affecting both insulin (insulin resistance, diabetes) and pubertal hormones (precocious puberty, polystic ovarian syndrome (girls), hypogonadism (boys) (Ebbeling, 2002), as well as orthopaedic and/or pulmonary complications (ie: sleep apnoea, asthma, exercise intolerance) that may inhibit participation in games and sports and further contribute to their weight problem (Deckelbaum & Williams, 2001, Ebbeling, 2002, Li, 2003, Speiser, 2004).

Child obesity may persist into adolescence (Nader, 2006) and adulthood (Freedman, 2005). A study tracking overweight from early childhood into adolescence, indicated that children who were overweight at least one time during the preschool period had five times more chance of being overweight in adolescence, as compared to children who were never overweight during the pre-school years (Nader, 2006). Another longitudinal study showed that 53% of overweight girls ages 2-5 years became adults with excess adiposity (Freedman, 2005). Furthermore, the risks for chronic disease associated with obesity may persist into adulthood, even when the excess weight is lost (Deckelbaum & Williams, 2001).

These findings led researchers to search for the early courses of obesity and some found evidence that the perinatal period is a critical period for obesity development (Dietz, 1994, Breir, 2001, Vickers, 2000). Certain adversities in the fetal and early infancy environment may have a significant effect on an individual's likelihood of developing overweight or obesity later on (Huang, 2007, Oken & Gilman, 2003). Increasingly, early life risk factors as well as the distinct developmental paths that a child's level of adiposity may follow are being considered important areas of study (Adair, 2008, Li, 2007).

In a large cohort study of 8234 children in the UK, 25 early-life risk factors for obesity at 7 years of age were examined. Eight of the 25 putative early-life risk factors were associated with an increased risk for obesity at age seven: parental obesity, early adiposity rebound, greater time spent watching television at age 3, the occurrence of "catch-up growth"[1], greater weight gain in the child's first year, high birth weight, and short sleep duration (Reilly, 2005). Other studies have identified maternal smoking during pregnancy (Oken, 2008), and the absence or reduced length of breastfeeding (Armstrong, 2002, Von Kries, 1999) as being associated with child obesity.

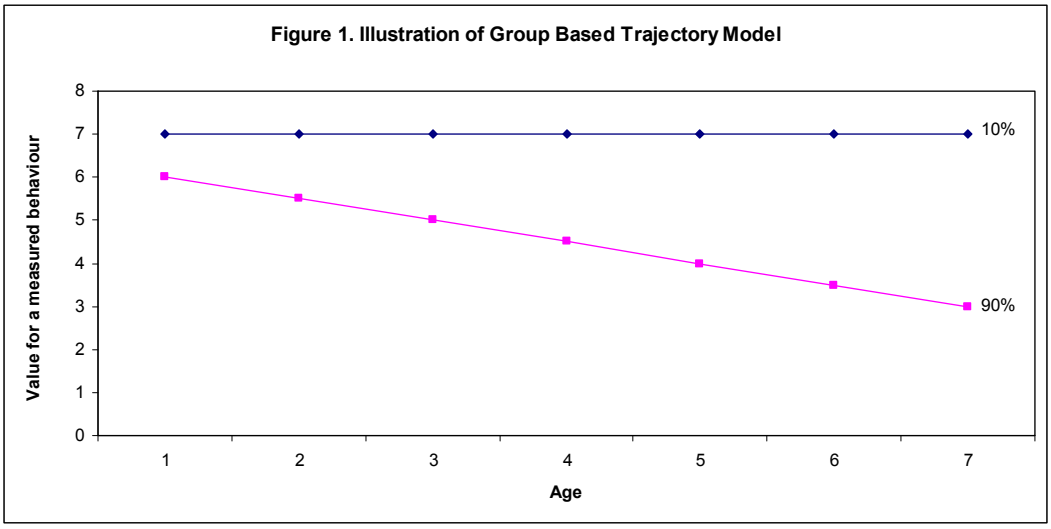
The prevention of the negative health and psychosocial consequences of obesity requires a comprehensive understanding of its development and early risk factors. The frequently used cross-sectional design for studies on childhood obesity does not allow for the identification of distinct developmental patterns (Mustillo, 2003), nor does it aid in our understanding of the specific early life characteristics that may result in such patterns. Growing evidence suggests the importance of using developmental trajectories for this purpose (Adair, 2008). In

[1] defined as an increase in weight Z-score exceeding 0.67 SD between birth and two years, representing the upward crossing of major percentile lines of a usual growth reference (Ekelund, 2006, Ong, 2000)

the present study, we use repeated measures of height and weight from 5 months to 8 years, in order to identify distinct patterns of BMI among a population sample of children. Specifically, we estimated group-based developmental trajectory models of BMIs.

A developmental trajectory depicts the course of a trait or behaviour across time (Nagin, 1999). Very few studies have applied this technique to the progression over time, of child overweight and obesity. In fact, to our knowledge, only one other study has examined early life predictors for particular BMI developmental trajectories in childhood (Li, 2007).

Figure 1 illustrates a hypothetical example of a trajectory.



Thus, a trajectory illustrates the relationship between a behaviour (Y axis) and time (X axis) for distinct groups of individuals having different developmental levels and patterns that distinguish them (Nagin, 1999). In this example, two sub-groups are identified: 1) 10% of children follow an elevated, stable trajectory and; 2) 90% follow trajectories that are elevated at approximately 6 years, but that decline over time.

1.2 Purpose

The purpose of this project is to further our understanding of the development and early-life risk factors for child obesity, to explore a novel statistical technique for the study of child BMI, and to contribute additional knowledge regarding the development of overweight and obesity among children.

1.3 Objectives

The objectives of the present study are to: 1) Identify children in a large Canadian (Québec) population sample with distinct developmental patterns of BMI between 5 months and 8 years and 2) Identify early (perinatal) risk factors for membership in the atypically elevated group, as well as the between-group differences that may exist.

1.4 Hypotheses

We expect to identify a group of children with an atypically elevated BMI. We expect that certain early maternal health characteristics and behaviours, such as maternal smoking and elevated maternal BMI will be associated with membership in this atypically elevated group.

CHAPTER 2: Literature Review

Childhood overweight and obesity is a multifactorial condition, with numerous predisposing risk factors (Reilly, 2005). Over time, a greater caloric intake than expenditure causes weight gain in humans, and may lead to overweight or obesity. Genetic predisposition contributes to an individuals' susceptibility for weight gain. However, as worldwide obesity levels have risen to epidemic proportions over the past two to three decades- a period wherein the gene pool has remained relatively stable (Kopelman, 2000)- it is increasingly believed that environmental and perinatal risk factors are more likely than genetic factors to underlie the current child obesity epidemic (Ebbeling, 2002).

This section focuses on an epidemiological and developmental perspective to the development of childhood obesity and will provide an overview of the numerous early potential risk factors for obesity.

2.1 Developmental Theories and Fetal Origins Hypotheses of Adiposity

In the prevention of obesity, it may be important to identify and to target specific "critical periods" of obesity development in childhood (Rolland-Cachera, 2006). These refer to the developmental stages in which physiological alterations increase the risk of later obesity (Dietz, 1997).

Increases in body fat occur at various life stages. In childhood, these include the perinatal period (from 22 completed weeks of gestation to seven days after birth, WHO, 1999), the adiposity rebound (approximately age 5-7), as well as adolescence/onset of puberty (Dietz, 1994, Zafon, 2007). Different theories

exist as to why body fat increases during these specified periods. An evolutionary approach points to a mechanism in place which stores fat just before periods where the body will undergo stress in order to promote survival (Adair, 2008, Zafon, 2007). For example, as an infants' food supply undergoes two transitions- from placental nutrition to lactation, and then from lactation to solid food- the fat that is accumulated late in gestation may serve to protect the infant from an interrupted supply of energy to the brain (Zafon, 2007). In terms of obesity development, these periods of fat accumulation may be considered problematic in today's obesogenic environments which provide a reduced opportunity for losing this extra body fat. These periods thus create a positive energy balance and may promote overweight/obesity among youth (Adair, 2008).

The "fetal origins" hypothesis suggests that shifts in the nutrition and endocrine status of a fetus will bring about changes in its' development which may cause permanent structural, physiological and metabolic changes for the individual, possibly increasing disease risks in adulthood (Barker, 1995). "Programming" is the term used to designate this process, whereby a stimulus occurring at a critical period in development entails a long-term effect on an individual's health (Barker, 2001, Breir, 2001). It is the systems' plasticity and sensitivity to its' environment during these particular periods which allow it to adapt in this way (Barker, 2001). An alteration in gene expression is thought to occur as a result of this adaptation (Breier, 2001). Recent evidence points to the leptin and insulin endocrine systems as being most influential in obesity and metabolic disorder programming. This may be due to the programming of appetite regulation and hyperphagia, but further research is needed to determine the accuracy of this hypothesis (Breir, 2001). The "critical period" is said to end when

this plasticity is lost, for the system is no longer able to make such adaptations (Barker, 2001).

2.1.1 The Barker Hypothesis

A growing body of both epidemiological and experimental evidence suggests that the prenatal period is a particularly sensitive period for later health outcomes. The "Barker hypothesis" (or "thrifty phenotype" hypothesis) is a theory suggesting that early-life metabolic adaptations may aid in survival by selecting an appropriate trajectory of growth in response to environmental cues (Hales & Barker, 2001). In the 1970's, Barker and colleagues observed that, in areas with a high prevalence of low birth weight babies, there was also an increase in the incidence of cardiovascular disease (CVD). Most of Dr. Barkers' studies examined maternal undernutrition during pregnancy. It was thought that the baby's development changed as a consequence of poor maternal nutrition, increasing their CVD risks (Barker, 1995). Subsequent studies have found similar associations with metabolic disorders and related conditions, and have revealed that the increased risks for diseases such as type 2 diabetes and hypertension in adulthood due to small size at birth may only be seen, or at least intensified, for those who become obese (De Boo & Harding, 2006, Kinra, 2005, Kopelman, 2000, Ong, 2000).

The Barker hypothesis is now widely accepted, and causes concern for societies in transition from poor to better nutrition (Hales & Barker, 2001, Robinson, 2001). This is because those who undergo pre-natal and early life adversities may develop this thrifty phenotype, and in the case where these individuals actually go on to develop in an affluent environment; their risk for developing metabolic disorders, such as obesity and type 2 diabetes may be

enhanced. Conversely, those who developed amid positive perinatal conditions will likely be better able to cope with heavier diets (Robinson, 2001). In Western societies, where severe maternal undernutrition is more rare, it has been observed that the offspring of women who had been previously diagnosed with an eating disorder may have an increased risk for preterm, low birthweight and small for gestational age (SGA) births. These findings suggest a possible thrifty phenotype in Western societies as well (De Boo & Harding, 2006, Conti, 1998, Thame, 1997, Godfrey, 1994).

2.2 Measuring Overweight and Obesity using the BMI

Body Mass Index (BMI), calculated as an individual's weight in kilograms divided by the square of their height (BMI=kg/m²), is a measurement tool used to assess one's body composition. It is a proxy measurement for more sophisticated and reliable tools, such as Dual Energy X-ray absorptiometry (DEXA) and Bioelectrical Impedance. Currently the best available anthropometric estimate for public health purposes (Hall & Cole 2006), it has been instrumental in documenting the recent worldwide increase in the prevalence of obesity (Schousboe, 2003). This is because these measures can be readily obtained, with reasonable precision in a variety of settings and for large numbers of individuals (Troiano & Flegal, 1998). Among adults, BMI has been estimated to correlate at 0.9 with body fat measured by bioelectrical impedance (Schousboe, 2003, *unpublished doctoral thesis*).

In childhood, BMI changes substantially with age, from a median as low as 13 kg/m² at birth, increasing to 17 at age 1, and decreasing to 15.5 at age 6. (Cole, 2000). Different rates of change occur at different ages and these changes

are age and sex specific (Troiano & Flegal, 1998, Wang, 2004). Biological differences in body composition also exist between ethnic groups and populations that may complicate the interpretation of BMI (Wang, 2004).

BMI has been shown to predict morbidity and mortality in a strong, graded relationship (Willet, 1999, Manson, 1995). However, other measures of adiposity should also be considered as indicators of risk. For instance, the waist-to-hip ratio (WHR) may be an important indicator of central obesity (Esmaillzadeh, 2004), which has been linked to increased cardiovascular disease risk. The measurement of waist circumference (WC) is also a useful, although possibly crude, measure of increased central obesity (James, 2001) and its' associated health risks. A Canadian study by Dobbelsteyn and colleagues (2001) used receiver operating characteristic (ROC) curves as well as sensitivity, specificity and positive and negative predictive values with respect to predicting the presence/absence of CVD risk factors, in order to compare WC, WHR, and BMI for their ability to identify individuals with these risk factors. They in fact concluded that WC is most likely the best single anthropometric measurement to use when identifying individuals with cardiovascular disease risk factors.

On the following page are the WHO classifications for BMI and waist circumference with cut-off points to delineate different levels of risks. When taken together, these two measures may provide a more global estimation of an individual level of health risks than either of the indices alone.

Table I. WHO Classification of Obesity, Waist Circumference and Associated Comorbidity Risks

Classification	BMI (kg/m^2)	Risk of comorbidities
Underweight	< 18.5	Low (but risk of other clinical problems increased)
Normal range	18.5 to 24.9	Average
Overweight	≥ 25	
Pre-obese	25.0 to 29.9	Increased
Obese class 1	30.0 to 34.9	Moderate
Obese class 2	35.0 to 39.9	Severe
Obese class 3	≥ 40.0	Very severe

	Waist circumference (cm)	
Comorbidity risk	**Women**	**Men**
Above action level 1	≥80	≥94
Above action level 2	≥88	≥102

2.3 BMI Growth Curves

For adults, the cut-off points used to distinguish between "normal", "overweight", and "obese" have been defined as a function of their association with disease risk. In children, the relationship between BMI and increased disease risk is less clear. The BMI values of children are lower than that of adults and change with age as the child is developing. As these adult cut-off values are not appropriate for children, percentile growth curves have thus been developed in order to track overweight trends throughout childhood. These were initially

developed based on National Survey data, such as the widely used CDC growth charts based on the US National Health and Nutrition Examination Survey (NHANES) data.

In order to increase generalizability, the IOTF (International Obesity Task Force) has created age and sex-specific curves using data from 6 large, nationally representative surveys. The curves were extrapolated from the adult cut-off BMI values for increased disease risk, and percentiles have been assigned, in order to represent an increased risk among children as well (Cole, 2000). However, these may lack precision, as the relationship between adult and childhood risk is yet unclear (Cole, 2000, Wang 2004).

Similar to other existing growth curves, the IOTF curves have been developed from cross-sectional data, which is less useful for explaining growth and development (Cole, 2000). Nevertheless, the curves developed by Cole et. al of the IOTF are now widely cited and will facilitate international comparisons (Wang, 2004).

2.4 BMI Trajectories

A developmental trajectory depicts the course of a trait or behaviour across time (Nagin, 1999). Growing evidence suggests the importance of tracking the development of overweight and obesity, as well as detecting early life risk factors. Developmental trajectories of overweight among youth represent an innovative approach towards our understanding of this epidemic (Adair, 2008). Very few studies have examined developmental trajectories of BMI beginning in early childhood thus far. In 2007, Li et al identified three distinct overweight trajectories for children between the ages of 2 and 12 years: early onset

overweight, late onset overweight, and never overweight. In addition, they were the only researchers thus far to examine early life predictors in association with atypical BMI trajectory groups (Li, 2007). Their results indicate that male gender, black ethnicity, elevated maternal BMI or maternal weight gain during pregnancy, and child birthweight are significantly associated with the early-onset overweight trajectory. All of these factors, except maternal weight gain, were also associated with late onset overweight, as well as maternal smoking and birth order (3rd or more). Breastfeeding more than 4 months was identified as a protective factor against both overweight categories.

However, the trajectory method used in the Li (2007) study was not group-based, as was ours. Rather, it utilized a latent growth mixture model and data from the *National Longitudinal Study of Youth* (NLSY79) in the United States. Our study will contribute new information on the development of BMI among a representative, non-US sample.

2.5 Early-Life Risk Factors for Obesity

2.5.1 Child Risk Factors

Birth Weight

The *in utero* environment is thought to play an important role in the development of obesity (Dietz, 1997, Rogers, 2003, Oken & Gillman 2003). (Hales & Barker, 2001), and birth weight can be an important consideration when studying illnesses related to the "critical periods" or "thrifty phenotype" hypotheses.

Birth weight is most often found to be positively correlated with later overweight and obesity (Adair 2008, Baird, 2005, Eriksson, 2001, Hirschler, 2008, Rogers 2003, Oken & Gillman, 2003). However, a review by Rogers and colleagues (2003) suggests that, while an independent association may exist between birth weight and child overweight, the association with adult overweight/obesity has most often been attributable to parental BMI, raising the question of whether it is genetic factors or the intrauterine environment that is responsible for the link. Authors of a review in that same year (Oken & Gillman, 2003) come to a similar conclusion, and state that it is most likely a combination of both genetic and early pre- and post-natal factors mediating the link between birth weight and future obesity.

Low birth weight is defined by the WHO as a birth weight less than 2500 g (WHO, 2001), and may be due to being born pre-term (<37 weeks gestation) or to being small for gestational age (SGA), or both (Euser, 2008). Certain studies identify low birth weight as also being associated with an increased risk of obesity (Ong, 2002). Others have recently investigated the hypothesis that birth weight may not only be linked to later overweight/obesity status or BMI, but also to body composition. Singhal et al (2002) found that low birth weight, representing poor fetal growth, may program a reduction in lean body mass. A review study by Oken & Gillman (2003) concluded that, while high birth weight is most often associated with an increased BMI in child and adulthood, many studies have found an important association between low birth weight and future central obesity, a condition that increases one's risk for cardiovascular disease. Insulin, as well as neurological endocrine and vascular changes are all considered possible mechanisms that may mediate the associations between either low birthweight and

future central obesity *or* high birthweight and future high BMI; however, future studies are needed to clarify how these factors come to play in each case (Oken, 2003).

It is clear that several confounding factors need to be considered when examining the association between birth weight and obesity. For example, low birth weight may occur due to extreme intrauterine conditions that constrain growth, such as maternal smoking during pregnancy (Kramer, 1987), which in turn may be followed by rapid growth in early infancy. Both the latter two events have also been linked to obesity in later life and will be discussed further on. Twin studies have been used to examine some of the factors confounding low birth weight, since many of these factors are similar between identical twins (eg. gestational age, genetics). These studies tend to report a significant association between birthweight and future weight or height, but not necessarily BMI (Oken, 2003). However, this may possibly be explained by a lack of variability between twin subjects to detect any differences in the variables being examined (Oken, 2003).

It is important to keep in mind that low birth weight is not a perfect measure of fetal development. There are pre-natal factors that can impact on the health of the fetus without affecting birthweight. In addition, birthweight does not distinguish between a shortened gestation (pre-term birth) and adversities in fetal growth (i.e.: a baby may weigh less than 2500 g at birth either because it is born too soon, or because it is small for its gestational age) (WHO, 2001).

Rapid Growth in Infancy

As mentioned previously, rapid growth in infancy (also known as "catch-up growth") can be defined as the increase in weight Z-score exceeding 0.67 SD, between birth and two years. This represents the upward crossing of major percentile lines of a usual growth reference (Ekelund, 2006, Ong, 2000). It may, but not necessarily, occur to compensate for intrauterine growth restriction (Adair, 2008), but by two years of age, growth will normally begin to follow the genetic trajectory (Ong, 2000).

While the effects of rapid growth are difficult to isolate, due to the fact that infancy weight gain is related to both intrauterine and post-infancy growth (Adair 2008), numerous studies report significant positive associations between rapid growth in early life and overweight in later child or adulthood, with seemingly greater risks the longer a child is exposed to rapid weight gain (Baird, 2005, Ong & Loos, 2006). For example, a recent review study found an overall 60% increase in obesity risk if the duration of rapid weight gain is increased from 1 to 2 years (Ong & Loos, 2006). Results from a prospective cohort study of 848 full-term singletons (10% random sample of the *Avon longitudinal study of pregnancy and childhood*, a geographically defined birth cohort in Bristol county, England) suggest that factors which may predict the rapid growth of the child include children having a lower weight, length or ponderal index at birth, primiparous pregnancies, maternal low birth weight, as well as maternal smoking during pregnancy (Ong, 2000). In addition, 30.7% of the sample of children who demonstrated clinically significant catch-up growth (as defined by a gain of greater than 0.67 SD score between birth and two years) was found to have

significantly higher values for BMI, percent body fat, and waist circumference at five years (Ong, 2000).

It has been postulated that certain factors which signal and regulate postnatal catch-up growth may also play a role in the mechanisms underlying the fetal origins hypothesis (Ong, 2000, Singhal, 2003). The programming of later insulin resistance is an example. After finding that rapid growth in infancy was associated with later insulin resistance (after controlling for birth size), Singhal and colleagues (2003) emphasize the importance of postnatal, as compared to intrauterine growth.

Adiposity Rebound

Adiposity rebound is a term used to designate the time when a child's body mass index (BMI) begins to increase after the nadir that normally occurs at 5 or 6 years of age, when a child's body fatness is at a minimum (Whitaker, 1998). Early AR is associated with a faster rate of gain of fat mass and skeletal maturity (Rolland-Cachera, 2006). An early adiposity rebound (AR) is a risk factor for overweight because it indicates that the child's BMI centile is high and/or crossing upwards, and for these children, an elevated BMI in later childhood and adulthood is likely (Cole, 2004). It is perhaps a much better predictor of adult overweight and obesity than childhood BMI, which may poorly predict adult adiposity before the occurrence of the AR (Rolland-Cachera, 2006). In a retrospective cohort study by Whittaker and colleagues (1998), adult obesity was significantly associated with early AR (OR=6.0) after adjusting for parental BMI and BMI at AR. A study of obese children showed that the mean age of AR in obese individuals is at 3 years old compared to 6 years old among the general

population (Rolland-Cachera, 2006). An association between an early age of AR and an elevated BMI in adolescence has also been demonstrated (Prokopec, 1993).

Overall, the role of the timing of the AR remains controversial. According to Dietz (2000), it may account for up to 30% of the proportion of adult obesity that begins in childhood, yet several concerns remain to be investigated. In terms of obesity prevention, it is not yet clear how our knowledge of the AR will help public health efforts, as it may be genetically programmed-an inherited susceptibility to obesity- or it may be due to environmental influence, and therefore modifiable (Whitaker, 1998)

Sleep Duration in Early Childhood

The recent increases in overweight and obesity prevalence are occurring simultaneously with a decrease in individuals' sleep time (Capuccio, 2008). Over the past 50 years, average sleep time in the United States is estimated to have been reduced by 1.5-2 hours (National Sleep Foundation, 2005).

Recently, the association between short sleep duration in childhood and the development of obesity from child to adulthood has been shown in several epidemiological studies. Indeed, a meta-analysis of cross-sectional concluded that an increased risk of obesity exists among short sleepers in both children (pooled OR= 1.89) and adults (pooled OR= 1.55) (Capuccio, 2008). Due to the cross-sectional nature of these studies and the lack of control for confounding factors, these results do not allow the inference of causality. However, a longitudinal study performed in Quebec using QLSCD data also found that children who slept less than 10 hours nightly during early childhood had a significantly increased risk

for developing overweight or obesity by school entry (6 years). Many potentially confounding factors, such as parental obesity, were controlled for in this study. (Touchette, 2008).

Possible mechanisms for the association between short sleep and obesity include: 1) the activation of hormonal responses, such as changes in leptin and ghrelin levels, which may lead to an increase in appetite and caloric intake (Vgontzas, 2003); 2) The activation of inflammatory pathways which may also be implicated in the development of obesity (Miller, 2007); and 3) the possibility that short sleep occurs preferentially among individuals with unfavourable health status and lifestyle characteristics (Patel, 2006).

2.5.2 Maternal Behaviours and Health Characteristics

Infant Feeding

Studies suggest that infants are often overfed (Adair, 2008) or that they are fed solid foods earlier and in greater amounts when the child is perceived as having a **difficult temperament** (Bentley, 2006). Both of these activities may contribute to overweight or obesity when the amount fed to the child is consistently greater than that child's energy needs.

Breastfeeding is a potential protective factor against overweight. However the studies examining breast feeding as a protective factor are presently inconclusive, due in part to methodological concerns such as the lack of control for confounding risk factors (Adair, 2008, Araujo, 2006, Armstrong, 2002).

Possible biological mechanisms by which breastfeeding may protect against obesity include: i) the learned self-regulation of energy intake, because in

comparison to a bottle-fed baby, a breast-fed baby is better able to control the amount of milk consumed based on internal satiety clues, ii) metabolic imprinting. For example, the properties of breast milk (low in protein and high in fat), may protect against future obesity, for high protein intakes in early infancy are thought to stimulate the secretion of insulin and insulin-like growth factor 1, two substances that may be involved in accelerating growth and enhancing adipogenic activity and adipocyte differentiation (Koletzko, 2005), potentially resulting in a greater than normal weight gain (Rolland-Cachera, 1995). In addition, infants fed a low-fat diet may program thrifty genotypes that predispose them to later weight gain (Rolland-Cachera, 2006).

Generally, reviews find a weak to moderate, dose-response protective effect of breastfeeding on later overweight/obesity (Adair, 2008). Owen et al (2005) examined sixty-one studies and concluded that breastfeeding does protect against obesity, but the author stressed the importance of future studies that will more adequately consider important confounding factors, such as maternal obesity and low socioeconomic status.

An interesting finding to note is that, according to a recent study by Buyken et al. (2008), breastfeeding may act as a moderating factor which offsets the strong influence of maternal overweight on her child. However this result was only applicable to males.

Smoking During Pregnancy

Several studies have linked smoking during pregnancy with an increased risk of child (Leary, 2006, Mizutani, 2007, Reilly 2005, Toschke, 2002) and adult (Power & Jefferis, 2002) obesity. It seems that a dose-response relationship exists,

with increased maternal cigarette smoking during pregnancy leading to a greater risk for obesity in children and adults (Power & Jefferis, 2002). Maternal smoking is said to restrict fetal growth (Adair, 2008), leading to a lower birth weight, which has also been associated with the development of obesity. This may be due to the compensatory rapid postnatal catch up growth that occurs (Ong, 2006). Another possible mechanism by which smoking may affect fetal growth is in the programming of appetite regulation (Von Kries, 2002).

A longitudinal study of the 16 766 individuals born in 1958 in England, Ireland and Whales (British Birth Cohort) for whom smoking information was available found that infants whose mothers smoked during pregnancy had a lower birth weight yet a greater chance of being in the highest weight decile at ages 11 and 16 years (Power & Jefferis, 2002). Dubois & Girard (2006) demonstrated a similar association with the children of the QLSCD cohort. Their analyses however revealed that it is not necessarily the combination of smoking with low birth weight and rapid early weight gain that has an effect on future obesity. In this study, the children born to mothers who smoked during pregnancy, had an increased risk of obesity at 4.5 years, but they were not of low birth weight. They were rather of "normal" birth weight with a rapid weight gain from birth to 5 months, or of "high" birthweight, with slower weight gain from birth to 5 months.

Maternal Overweight and Obesity

The link between maternal and child overweight or obesity may be attributable to genetics or shared familial characteristics. With age, parental influence on lifestyle characteristics, such as food choices and physical activity, become increasingly important (Adair, 2008). As our study is looking at very

early child risk factors, we will review some of the evidence regarding genetic factors in the present section.

An important genetic component seems to exist in the variation in BMI; however the extent of this influence is unclear (Haworth, 2008). Results from twin studies range from 50-90% heritability of BMI (Maes, 1997). There is also evidence to support that a common etiology exists between what are considered normal variations in BMI and clinical obesity levels (Haworth, 2008).

The "thrifty genotype" hypothesis, originally proposed by Neel (1962), states that populations who were once frequently threatened by starvation may have developed genes to protect against this threat and promote survival. In today's obesogenic environment- that is, an environment favouring positive energy balance- these same genes may predispose an individual to obesity (Bell, 2005). Certain populations, such as the well-studied Pima Indians of Arizona, are disproportionately affected by obesity and type 2 diabetes. It was proposed that this may be explained by such an adaptation of their genes (Joffe & Zimmet, 1998).

Certain studies have identified that correlations between parental and child overweight are not significant in infancy (Stunkard, 1999, Safer, 2001). In a prospective longitudinal study of infants with mothers who were obese, no correlation was found between the body weights of mothers and their offspring before two years of age (Stunkard, 1999). The authors believe this result corroborates the literature, and that overall, the relationship between maternal and child weight is greater at birth than in the first two years of life. Furthermore, Safer and colleagues (2001) did not observe a correlation between parental and

child overweight until the age of seven years, however it was impossible to determine whether this was due to genetic or environmental factors.

Some evidence relates maternal obesity to increased adult risk for cardiovascular disease through mechanisms related to the fetal origins hypothesis (Godfrey & Barker, 2000). The fetal nutrient intake may be unbalanced due to excess sugar provided by overweight mothers (Barker, 2001). Men who were born to women who were obese during pregnancy were found to have a greater incidence of CVD in adulthood (Forsen, 1997). Furthermore, the offspring of short mothers who were obese showed disproportionately greater incidence of CVD, suggesting that a restrained fetal environment may have played a part in this link (Forsen, 1997, Godfrey & Barker, 2000).

Maternal Diabetes

A mother with Gestational Diabetes Mellitus (GDM) or Type 2 Diabetes has increased glucose, lipid and amino acid concentrations in her blood, which is then delivered to the fetus (Catalano, 2003). This leads to fetal hyperinsulinemia, increased production of growth factors, higher birth weight, and a larger fat mass of infant (Catalano, 2003). Due to confounding, it is difficult for studies to determine whether infants of diabetic mothers have a higher risk of obesity or not. For example, when controlling for maternal BMI, the association between maternal diabetes and child overweight is often attenuated (Gillman, 2003).

Maternal Depression

Among adolescents and adults, links between obesity and depression within the same individual have been established (Dong, 2004, Dragan, 2007,

Stunkard, 2003), suggesting that a common etiology or genetic vulnerability may exist. The association between maternal depression and obesity in her child is less clear, for few studies have been done on this topic. In a cross-sectional study of 589 pairs of mother and child from low-income urban communities in northeast Brazil, Surkan et al. (2008), found a significant association between maternal depression and offspring obesity. However, while the study did control for numerous SES factors, and factors related to child growth, they failed to control for maternal BMI, which could represent a confounder in the association found. The cross-sectional nature of the study also leaves us far from being able to infer causality. Another cross-sectional study (Gibson, 2007) examined 329 children aged 6–13 years (192 healthy weight, 97 overweight and 40 obese), and 265 mothers. No association between maternal depression and child obesity was found. Similarly, in the longitudinal, ecological study of children's home environments (O'Brien, 2007), no association between maternal depression and child overweight/obesity was found, however it is interesting to note that the mothers sensitivity and opportunities for productive activity at home were found to be significant in this study, two factors wherein maternal depression could be thought to have an impact. The O'Brien (2007) study also failed to control for maternal BMI. It is evident that more longitudinal studies are needed to examine the link between maternal depression and child BMI, which control for maternal BMI in addition to other potentially confounding variables.

2.5.3 Family Risk Factors

Socioeconomic Status as a Risk Factor

Socioeconomic Status (SES) is often defined in terms of a combination of three factors: occupation, education and income. In 1989, Sobal and Stunkard (1989) performed a vast review of the literature on the obesity-SES association, from approximately 1960 to 1985. Their main findings included the existence of an inverse relationship between obesity and SES among women in developed societies, with increasing SES being associated with a decreased prevalence of obesity among women. They also found a strong direct relationship between these two factors for men, women and children in developing societies, with higher SES being associated with higher obesity rates. An updated review of the obesity-SES relationship (Maclaren, 2007) which set out to build on Sobal and Stunkards' previous influential work sheds light on the present-day association between obesity and SES. In general, it appears that, in lesser developed countries, there are increased positive associations between SES and obesity. In highly developed countries, this association is reversed, particularly among women. That is, women of higher SES tend to be thinner in developed countries. These results were similar but less striking than the conclusions obtained by Sobal and Stunkard in 1989; they have perhaps been attenuated due to the effects that economic growth, modernization and globalization have had on individual societies (Maclaren, 2007).

For children in industrialized, economically developed countries, being in a family of low SES increases the risk of obesity (Sobal & Stunkard, 1989, Wang, 2001) however this association may also be weakening with time (Adair, 2008). A cross-national comparison of the relationship between child obesity and SES

identified distinct differences between countries. For example, children of higher SES in China and Russia were found to be more likely to be obese, whereas children of lower SES in the US seem more likely to be obese (Wang, 2001). In certain developing countries undergoing the nutrition transition[2] children are often suffering from malnutrition and underweight alongside adults suffering from obesity, within the same household (Caballero, 2005).

Family functioning

O'Brien and colleagues (2007) emphasize the need for further research into how family processes may be related to child obesity. Results from this longitudinal study indicate that parenting style, including mothers' sensitivity and degree of control, are important predictors of child obesity, with less sensitive and more controlling behavior positively associated with child overweight. Similar results were previously found in a study by Rhee et al. (2006). However, the cross-sectional study by Gibson (2007) found no association between poor general family functioning, as reported by the mother, and later child overweight/obesity.

2.6 Summary of Previous Findings

Although awareness has increased regarding the importance of understanding perinatal risk factors for the prevention of obesity (Ebbeling, 2002, Reilly, 2005), there remain numerous gaps in the literature in this regard, and most recognized risk factors are potential rather than confirmed (Reilly, 2005). Many of the existing studies on early risk factors for obesity have been cross-

[2] Shift from under- to over-nutrition problems in developing countries (Wang & Lobstein, 2006) alongside shift in dietary and exercise patterns over the past 20 years (Popkin & Gorden-Larsen, 2004).

sectional, underpowered or unable to consider numerous risk factors simultaneously or to account for confounding variables (Parsons, 1999, Dietz, 2001). For example, we have seen that the numerous studies on the protective role of breastfeeding have not adequately considered confounding risk factors (Adair, 2008, Araujo, 2006, Armstrong, 2002), and there exists a need to disentangle the effects of low birthweight from maternal smoking during pregnancy and rapid growth in infancy, all interrelated and confounding factors. Other potential risk factors have not received enough attention to date, such as the association between maternal depression and child obesity, as well as how family processes may be related to child obesity (O'Brien, 2007).

Another important yet understudied area is the distinct developmental path that a child's level of adiposity may follow (Adair, 2008, Li, 2007). The frequently used cross-sectional design for studies on childhood obesity has not allowed for the identification of distinct developmental patterns (Mustillo, 2003). The Cole curves to delineate obesity are useful; however, as the relationship between BMI and increased disease risk is unclear (Cole, 2000, Wang 2004), it is important that new methods for examining the development of child obesity be investigated.

According to the "Barker hypothesis", early-life metabolic adaptations may aid in survival by selecting an appropriate trajectory of growth in response to environmental cues (Hales & Barker, 2001). In the present study, we take a closer look at many early life factors, as well as the trajectories of BMI development which follow. Since this study is a non-US birth cohort, longitudinal in nature, and able to investigate numerous perinatal risk factors simultaneously, it has the potential to make an important contribution to the existing literature.

CHAPTER 3: Methodology

3.1 Study Design and Participants

The present study is a secondary analysis of data drawn from the Quebec Longitudinal Study of Child Development (QLSCD). A random population sample of families with a 5-month old infant in the year 1998 was recruited in the Canadian province of Quebec (n=2120). The participants were recruited via the Quebec Master Birth registry managed by the Ministry of Health and Social Services. Trained interviewers conducted yearly interviews in the home with the mother or "Person Most Knowledgeable" about the child (PMK), the mother in 98% of cases. The first interview was conducted in 1998, when the baby was approximately 5 months old. Interviews were then conducted yearly until the child reached the age of 8 years. Information was gathered regarding family characteristics, parental, and child behaviors. At every data collection, informed written consent was obtained from all participating parents (Appendix A & B: letter to parents and consent form). The larger QLSCD study has been previously approved by the Ethics committee of Santé Québec (Appendix C). The present study was approved by the Ethics Committee of the Faculty of Medicine of the University of Montreal (Appendix D).

3.2 Attrition Analyses

Longitudinal height and weight data (over 5 time points) was available for 1957 of the 2120 children (Attrition Rate: 7.69 %) to be included in the group-based developmental trajectory estimations. Therefore, the analyses sample included 1957 participants. Socioeconomic Status differed between the children

included and not included in the trajectory models. Specifically, mothers with a lower level of education and with lower income were less well represented in the analyses sample. However, all analyses used weighted scores to ensure that the analysis sample was representative of the target population even though attrition occurred. Table II represents the demographic characteristics of the participating families.

Table II. Demographic Characteristics of Sample at 5 months (n=1957)

		%
Sex of child (girl)		49.7
Ethnic Origins		
Canadian		65
French		26.5
Irish		4.5
Indian		2.7
British		2.6
Italian		2.6
Scottish		1.7
Other*		7,5
Mother Obtained High School Diploma		82.8
Family Income		
<$15,000		11.6
$15,000-30,000		17.6
$30,000-60,000		40.5
>60,000		30.2
	Mean	SD
Mother's Age (years)	26.69	8.906

* "Other" includes all categories of ethnic origin with less than 1% membership

Table III examines differences between the included and excluded families for a variety of variables deemed pertinent by our literature review. In doing so, we see that, not only are families of lower SES less well represented in the trajectory groups, but also those with decreased levels of family functioning, and

with mothers who experience greater levels of depressive symptoms. On the other hand, children who experience less sleep at night and faster early weight gain (two of the identified potential risk factors for later obesity) are more well-represented in our trajectory groups than in the group that was excluded due to a lack of longitudinal data.

Table III. Attrition Analyses: Comparison of Characteristics-Included and Excluded Families

	Included (n=1957)	Excluded (n=163)	x^2 Test of Significance
	%	%	
Child Characteristics			
Sex (female)	49.7	41.1	p= 0.035
Low Birthweight	3.5	2.5	NS
High Birthweight	11.6	9.3	NS
Short Gestational Age	4.8	3.7	NS
Perceived Difficult Temperament (top 25th percentile)	29.7	24.2	NS
Sleep at 5months (< 6 consecutive hours nightly)	79.5	66.2	p<0.001
Fastest average monthly weight gain (highest quintile from birth to 5 months)	19.9	15.7	p<0.007
Maternal Characteristics/Behaviours			
Overweight	28.9	32.3	NS
Smoked during pregnancy	25.2	26.4	NS
Did not Breastfeed Baby	28.3	27.6	NS
Severe Depressive symptoms (top 25th percentile)	21.2	30.9	p=0.004
Family Variables			
Insufficient total family income	22.1	55.6	p<0.001
Mother did not have high school diploma	17.1	30.9	p<0.001
Poor Family Functioning (top 25th percentile)	20.1	30.9	p=0.002

*NS: not statistically significant based on a p=0.05 alpha level cut-off value level .

3.3 Measures

Group based trajectories of BMI (Dependant Variable)

Measures of height and weight were obtained yearly during the interview with the PMK. The data collected when the child was 5 months of age through to 5 years was by maternal report, and is found in the *Computerized Questionnaire Completed by the Interviewer* (ELDEQ, 2000). Specifically, mothers were asked "What is his/her height in feet and inches or in metres/centimetres (without shoes on)?" and "What is his/her weight in kilograms (and grams) or pounds? (ELDEQ, 2000). At 5 years, this information was obtained through a telephone interview with the PMK, as part of the *Paper Questionnaire Completed by the Interviewer* (ISQ, 2007). At 6, 7 and 8 years, this data was obtained during the *Physical Health Evaluation*, through anthropometric measurement of the child by the trained interviewer using standard procedures adapted by the ELDEQ team from the ESSEA (*l'Enquête sociale et de santé auprès des enfants et des adolescents québécois*, 1999) study (ISQ, 2007).

The height and weight variables were validated using the CDC growth charts (NCHS, 2000), and aberrant data was removed. For example, QLSCD statisticians verified whether the responses for height and weight were appropriate given the minimum and maximum cut-offs defined by the CDC curves (ISQ, 2001).

BMI was calculated using the equation BMI=kg/m². Group-based trajectories of BMI were then modelled using the SAS Proc Traj program (SAS version 9.1.3) and the data from the resulting trajectories was used to form the 3-category dependant variable indicating group membership.

Starting in 2002 (age four), the exact age of the child at data collection became less precise because all children began to be measured at roughly the same time of year in order for the data collection to be synchronized with the school system. Children's ages could therefore potentially vary up to one year from one another (ISQ, 2003), however it was not possible to take this into account in trajectory group modelling.

Risk Factors (Independent Variables)

Potential risk factors were chosen based on evidence of associations in the existing literature as well as plausible hypotheses. We have chosen to analyze early life risk factors, including relevant factors in the prenatal period and infancy (before two years of age). This is a logical cut-off for the present study, not only because it aids in providing clarity to the research question, but also because a distinct parting in children's BMI is observed at approximately this time point (~2.5 years). That is, the atypically elevated BMI trajectory group and the other two trajectory groups (obtained in the first phase of analysis) became highly distinct (i.e. raising versus flat) after 2 ½ years. Unless otherwise specified, all variables used were obtained during the home interview with the PMK when the child was 5 months of age.

Child Characteristics

Sex: *The sex of the child was* obtained from hospital records and coded as a dummy variable (boy= 1, girl=0).

Birthweight: obtained from hospital records and transformed into a 3-category variable representing low (<2500 g), normal (2500-4000 g) and high

birthweight (>4000 g). This categorisation represents a common way to characterize birth weight in newborns (Hirschler, 2008, WHO, 2001).

Birth order*:* Birth order at 5 months was obtained via maternal questionnaires. The mother was asked how many children she had had before the birth of the child participating in the study. A 3-category variable was created, indicating if the child was 1st born (=0), 2nd born (=1), or 3rd or later (=2) (Li, 2007, Reilly, 2005).

Gestational Age was obtained from hospital records. A variable contrasting preterm births *(*<37 weeks = 0) from the rest of sample was created (Reilly, 2005, WHO, 2001*).*

Difficult temperament*:* Difficult temperament was assessed via a questionnaire to the PMK. Seven items, taken from the widely used and well-validated *Infant Characteristics Questionnaire* (John Bates, University of Indiana) formed the scale at age 5 months (1998). It is a renowned and widely used scale, considered to be the best measurement tool for this variable in population studies (ISQ, 2003).Scores were afterwards standardized to a 10 point scale, with higher values representing a more difficult temperament. For this study, a variable contrasting the top 25th percentile (=1) from the rest of the sample (=0) was created. *Sleep duration:* The child's consecutive sleep duration at 5 months was divided into 2 categories (< 6 consecutive hours nightly=0, > 6 hours = 1) (Touchette, 2005, 2008).

Average weight gain from birth-5 months*:* There has been a recent narrowing of focus from growth acceleration in the first two years to the first few weeks of life (Kinra, 2009). This variable is intended to assess rapid early life weight gain, and was calculated using the equation: (weight at 5 months)/(weight at birth) /(Age at

5 months-Age at birth (0)). The variable was then divided into quintiles and the two highest quintiles (=1) were compared to the rest (=0).

Maternal Health Behaviours and Characteristics

Smoking during pregnancy*: Smoking during pregnancy* was treated as a dummy variable indicating whether the mother reported smoking (=1) or not (=0) during pregnancy.

Maternal BMI: Mothers reported their height and weight when their baby was 17 months old. BMI was calculated using the equation BMI=kg/m². A dummy variable was created to distinguish mothers with a BMI ≥25 kg/m² (=1), representing overweight or obesity, versus those with a BMI <25 kg/m² (=0), which includes individuals of normal or low weight (Cole, 2000).

Depressive symptoms (when the child was 30 months olds): A reduced version (12 questions) of the CES-D depression scale was used (Radloff, 1977).This scale measures both the frequency of depression in a population, as well as the occurrence and severity of depression in an individual the week prior to the interview. A variable contrasting mothers with more severe depressive symptoms (top 25th percentile=1) from the rest of the sample (=0) was created (O'Brien, 2007).

Breastfeeding (assessed when the child was 17 months old*)*: A 3-category breastfeeding variable was created (did not breastfeed=2, less than 3 months=1, and more than 3 months=0), as a dose-response relationship between breastfeeding and reduced levels of overweight has been observed in the literature (Von Kries, 1999, Harder, 2005),

Initiation of Solid Foods*:* Mothers were asked at what age their child started receiving solid foods. The variable was divided into <4 months (=1), >4 months (=0) (Bronner, 1999).

Young Mother*:* Mothers were asked their age at the birth of their first child. A "young mother" was defined as a mother who was 21 years of age or less at the birth of her first child, and was coded as 1=young mother and 0=not.

Family Characteristics

Socioeconomic Status (when the child was 5 months)*: Maternal Education* and *Insufficient Income Status* were used as measures of Socioeconomic Status. *Maternal education* was treated as a dummy variable indicating if the mother had (=0) or not (=1) a high-school diploma. In the province of Québec, this corresponds to the completion of mandatory schooling. *Insufficient household income* was calculated according to Statistics Canada's guidelines taking into account the family income in the past year, the number of people in the household, and the family zone of residence (urban versus rural, population density). Income was coded as sufficient (0) or not (1).

Family functioning (when the child was 5 months old): Family functioning was assessed with an 8-item scale, developed by researchers of the Chedoke-McMaster hospital (McMaster University) and widely used and validated in Canada and abroad. This scale measures the functionality of the family (eg.: "there are lots of bad feelings in our family"; "in times of crises, we can turn to each other for support"; "we don't get along well together"). Mothers answered on a scale from 1 (strongly agree) to 4 (strongly disagree). Higher values indicate less functional households. The variable was re-coded into two categories

separating the top quartile (poor functioning) from the rest (below 75th percentile=0, 75th percentile and above =1).

Maternal immigrant status: *Maternal immigrant status* was treated as a dummy variable (immigrant=0, non-immigrant=1).

3.4 Summary of Data Analysis

The objectives of the data analysis were two-fold: 1) To identify distinctive groups of developmental trajectories of BMI from 5 months to 8 years of age and; 2) To determine risk factors that predict a child's membership in the atypically elevated trajectory group.

The analysis proceeded in two phases. First, the distinctive clusters of developmental trajectories were identified using a semi-parametric, mixture model (Nagin, 1999), and confirmed by a non-parametric algorithm, KML (Genolini & Falissard, 2009, submitted).Group-based trajectories were modeled using the SAS Proc Traj program and the procedure described below. Second, logistic regression analyses were used to examine the capacity of the risk factors to distinguish membership in the atypically elevated trajectory group as opposed to each of the other two trajectory groups, controlling for the levels of the other risk factors. Factors associated with membership in the moderate group were also explored.

3.5 Data Analysis, Phase 1: Trajectory Modeling

3.5.1 Trajectory Model Selection

A semi-parametric, group-based approach was used for modeling developmental trajectories in the Proc Traj extension of SAS (version 9.1.3). The

semi-parametric group-based method allows for the identification of population heterogeneity in the developmental patterns of the BMI over time (Jones, 2001, Nagin, 1999). Specifically, the procedure allows the identification of groups of children with distinct developmental patterns on a given characteristics. For instance, the procedure has often been used to distinguish children with high levels of problem behaviours such as physical aggression or hyperactivity (Côté, 2007, Romano, 2006). This specific group-based developmental trajectory approach has not to date been used to model the development of children on their BMI.

Following the computation of the BMI variables from the height and weight data at each time point (BMI=kg/m²), these values were entered into the program. In order to ensure that each child included in the trajectories had sufficient data points to allow a robust estimation of trajectories, only those children with 5 or more (out of the 9) available data points were included.

A key step in model estimation was the selection of the number of trajectory groups that best fit the data. Model selection was based on the Bayesian Information Criterion (BIC), which identifies the optimal number of groups in finite mixture models (Keribin, 1998). The BIC rewards parsimony, favouring fewer groups, and is considered to be consistent (Bongers, 2004, Kass & Raftery, 1995, Keribin, 1998). Models with 2 to 4 groups were estimated and the model with the maximum BIC was selected as the optimal model. The BIC criterion for model selection is calculated as: BIC = log(L) -0.5*logOz)*(/t), where *L* is the value of the model's maximized likelihood, *n* is the sample size, and *k* is the number of parameters in the model (Nagin, 1999). When prior information on the

correct model is limited, it is suggested that the largest BIC be selected (Kass & Raftery,1995, Raftery,1995).

Each possible combination of trajectory shapes (curvilinear, quadratic, cubic) in a 2, 3 and 4-trajectory model was tried, in order to determine the model with the maximum BIC, while still maintaining parsimony and not allowing for a group of too small a size.

The outcome data from the selected model in the Proc Traj program was then used to create three variables relevant for further analyses: 1) a posterior probability of membership in each trajectory group (continuous variable varying from 0 to 1); 2) a three-category variable indicating group membership, and 3) a binary variable contrasting the atypically elevated trajectory from the other two trajectories.

3.5.2 Checking Classifications

The posterior probability calculations that were provided by the Proc Traj program provide us with an objective basis for determining whether each individual has been classified into the developmental trajectory group that best matches their BMI data. Individuals were assigned to the group for which their posterior membership probability was the largest (Nagin, 1999). For example, consider a child whose BMI is consistently above average. For this individual, the posterior probability estimate of the child's belonging to a low trajectory group would be near 0, whereas the probability estimate of the child's belonging to the "high rising" group would be high. This is the group that best conforms to the child's BMI development over time.

3.5.3 Verifying an Association with Age

In order to account for the fact that the ages of the children varied at a given data collection period, particularly during the school-age years, independent sample t-tests were performed using SPSS (version 17; SPSS Inc, Chicago, ILL). The purpose of this test was to verify whether the fact that a child was found to be a member of the high-rising group was associated with his or her age at the time of measurement. The mean ages of the children at distinct data collection time points (ages 6, 7 and 8) in the "high-rising" trajectory group were compared to the mean ages of the rest of the children in the other two trajectory groups combined ("moderate" and "low-stable"). The dichotomous variable, which was created to distinguish membership in the high-rising group from the rest of the sample, was used for this purpose, as well as the continuous variables indicating the exact age (in months) of each child at data collection (for ages 6, 7 and 8).

3.5.4 KML Cluster Modeling

Two families of statistical methods exist to determine homogeneous groups of individuals' trajectories: group- based modelling methods and cluster analysis (Genolini & Falissard, submitted). Following the above work in SAS Proc Traj, a model-based method, our same BMI data was input into the KML (K-means longitudinal) program, a non-parametric algorithm for cluster analysis in the R statistical software. It has been specifically designed to work with longitudinal data and is able to deal with missing values; re-running the algorithm several times while varying the starting conditions and/or the number of clusters, in order to determine the appropriate number of clusters and avoid a local maximum (Genolini & Falissard, 2009). In this procedure, the Calinski criterion is

used to choose the best model. KML's non- parametric method works well with trajectories having a non-polynomial shape and is valuable in supporting the robustness of results obtained from models based on more well-known procedures, such as SAS Proc Traj (Genolini & Falissard, 2009).

3.5.5 Variance Component Analyses

To assess the impact of using two different methodologies for weight and height measurements (maternal report from 5 months- 5 years, anthropometric measurement from 6 years-8 years), we performed three variance component analyses in SPSS. The first utilized BMI measurements at 4.5 and 5 years (same method each year), the second with BMI measurements at 5 and 6 years (different methods), and finally with BMI measurements at six and seven years (same method).

3.5.6 Trajectory Modeling by Sex

Before proceeding to the second stage of analysis, we modelled the group-based trajectories separately by sex in order to examine if significant sex differences could be detected in the developmental patterns of boys and girls. This was done in order to determine whether separate analyses of the risk factors for membership in the trajectory groups would be required for each sex.

3.6 Data Analysis, Phase 2: Risk Factors

3.6.1 Descriptive Statistics

We initially explored each variable in the data set separately, looking at the range and central tendencies of the values, both graphically (histogram, box-

plot) and textually. For each continuous variable in the database, we examined the frequency, number of missing values, mean, median, standard deviation, minimum, maximum, and quartiles. We also explored extreme values in box plot and in table form. After the BMI variables were calculated, some of these extreme values (outliers) were removed, as well certain values that were evidently incorrect (e.g. : if the value of an individual's height had *decreased* with age, this decreased value was removed). However, as the ELDEQ database has been previously cleaned up by a set of qualified statisticians, very few data points were removed. For each categorical variable in the database, we examined the frequencies (n and %) and missing values. All non-response values were coded as missing values.

3.6.2 Bivariate Analyses and Treatment of Continuous Variables

We examined the bivariate associations between all independent variables (Risk Factors) and the dependent variable (BMI trajectory groups). Using SPSS for Windows (version 16; SPSS Inc, Chicago, ILL), a chi-square test of joint significance was used to examine the associations between categorical values ($p>0.05$), while an ANOVA test (F stat and p for significance ($p>0.05$)) was used to look at associations between the categorical dependant variable and continuous risk factors.

All continuous variables that were associated with the dependent variable in bivariate analysis were then converted into two- or three-category variables. This was done in order to simplify the interpretation of our results. Cut-off points were chosen either based on clinical cut-off points in the literature or, when this was not possible, using the upper quartile vs. the rest of the sample to distinguish

those more severely affected by the variable in question. The latter technique was only used for the variables *maternal depression*, *difficult temperament*, and *family functioning,* which had been made available to us already in the form of a standardized scale, and were therefore difficult to compare to the literature

Finally, chi-square of joint significance (alpha< .05) tests were once again used to examine the proportion of each risk factor variable to be used in the study (now all categorical) in each of the three trajectory groups (DV). This constitutes the first series of analyses conducted to identify the maternal, child and family characteristics that distinguished trajectory group membership, as only those variables in which a significant (p> 0.05) bivariate association was found in this step were included in the final logistic model.

3.6.3 Correlation Analyses

Collinearity occurs when two or more independent variables are correlated, making it difficult to determine which of the variables is more important based on the regression calculation (Miles, 2001). Collinearity and multicollinearity (more than two covariates are highly correlated), increases uncertainty around the parameter, increases inaccuracy, and can seriously distort the interpretation of the results (Miles, 2001, Tu, 2005).

According to the literature, possible correlations may exist between certain variables in our study. *Smoking during pregnancy* has been shown to be correlated with *low SES* (Mathews 2001, Zimmer & Zimmer, 1998), *young mother* (Cnattingus, 2004), *maternal depression* (Breslau, Kilbey, & Andreski, 1993), and *family functioning* (Brook, Brook, & Whiteman, 2000). *Rapid growth* in early infancy may be related to *low birthweight*, primiparity (*birth order* being 1st in our study), *mother's low BMI*, or *mothers smoking in pregnancy* (Ong, 2000). Also,

socioeconomic status may be correlated with *depression* (Lorant, 2002) and the socioeconomic variables themselves (*high school diploma* and *insufficient income*) may be thought to represent a potential collinearity problem.

Using SPSS, we explored the degree to which the independent variables in our initial model (the ones tested in bivariate analysis) were correlated with each other. A table of zero-order correlations can be found in Appendix G. For this analysis, the continuous form of the variable was used wherever possible, and the Pearson moment product correlation was used, which is obtained by dividing the covariance of the two variables by the product of their standard deviations (Kleinbaum & Klein, 2002). When one variable did not follow the normal law (e.g.: categorical variables), the non-parametric Spearman correlation was instead utilized.

3.6.4 Final Model: Multivariate Analysis

In the final stage of analysis, an examination of the risk factors associated with trajectory group membership was conducted. A polytomous logistic regression model (multinomial logit analysis) enabled us to compare the relationships between the nine risk factor variables included in the final model with our three-level outcome variable (Kleinbaum & Klein, 2002). These analyses were performed in SAS (version 9.1.3) under the "generalized logit" procedure. In turn, both the low-stable and moderate trajectory groups of our 3-level outcome variable were used as reference groups in these analyses. Specifically, we looked at the high-rising trajectory as compared to each of the moderate and low-stable trajectories, as well as the moderate trajectory, as compared to the low-stable trajectory. For each reference group, two odds ratios (OR) were obtained for each of the independent variable values. For example, if

the reference group was the "low" trajectory, then, for each of the independent variables in the model, we obtained:

Pr("High")/Pr("Low") and Pr("Medium")/Pr("Low")

(Kleinbaum & Klein, 2002).

In polytomous logistic regression, with three outcome variables, these two expressions are actually “odds-like”, and not true odds; however, they may be interpreted like odds ratios if only two categories are considered at a time (Kleinbaum & Klein, 2002).

CHAPTER 4: Results

4.5 Data Analysis, Phase 1: Trajectory Modeling

4.5.1 Trajectory Model Selection

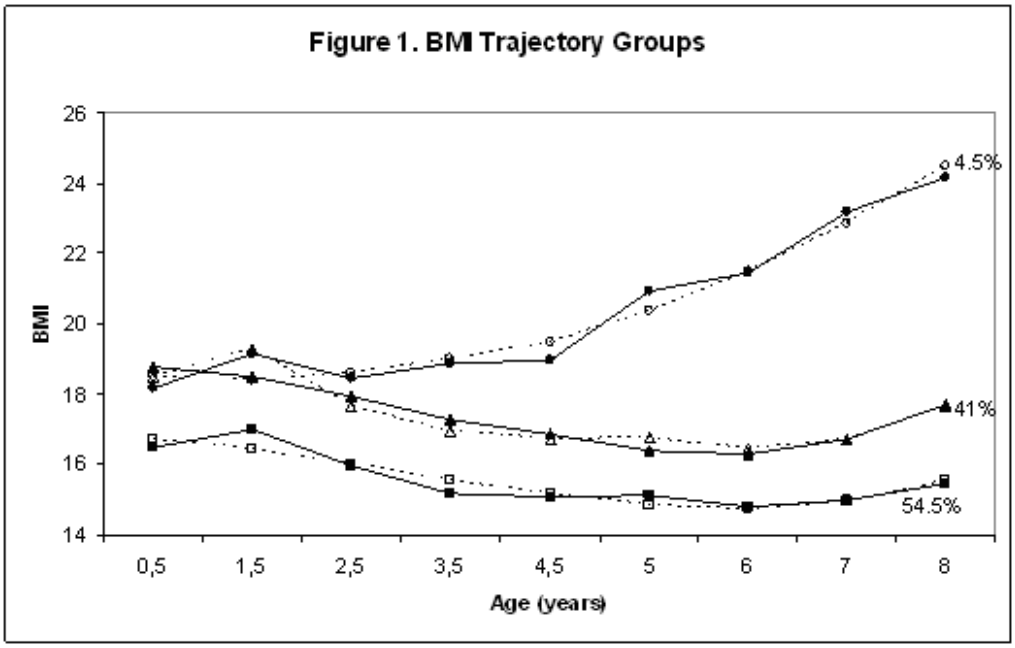
Results from this step can be seen in the tables found in Appendix E. Although the maximized BIC was obtained in the 4-trajectory model, it included a trajectory with a very low number of individuals. The 3-trajectory model with maximized BIC was therefore chosen in order to improve our ability to analyze the data. Note that the values in these tables are absolute values, and that the maximized BIC is that with the *least* negative value.

4.5.2 Checking Classifications

Using the posterior probabilities calculations, we examined the accuracy of our classifications in SPSS. Each trajectory group variable was selected, in turn, and the mean posterior probability for being in that trajectory was noted. A probability of 0.80 is considered to be a good cut-off (Nagin, 1999).We obtained mean posterior probabilities of 0.91, 0.87, and .95 for trajectory groups 1, 2, and 3, respectively; indicating that indeed, our classifications into BMI trajectory groups represent a good fit.

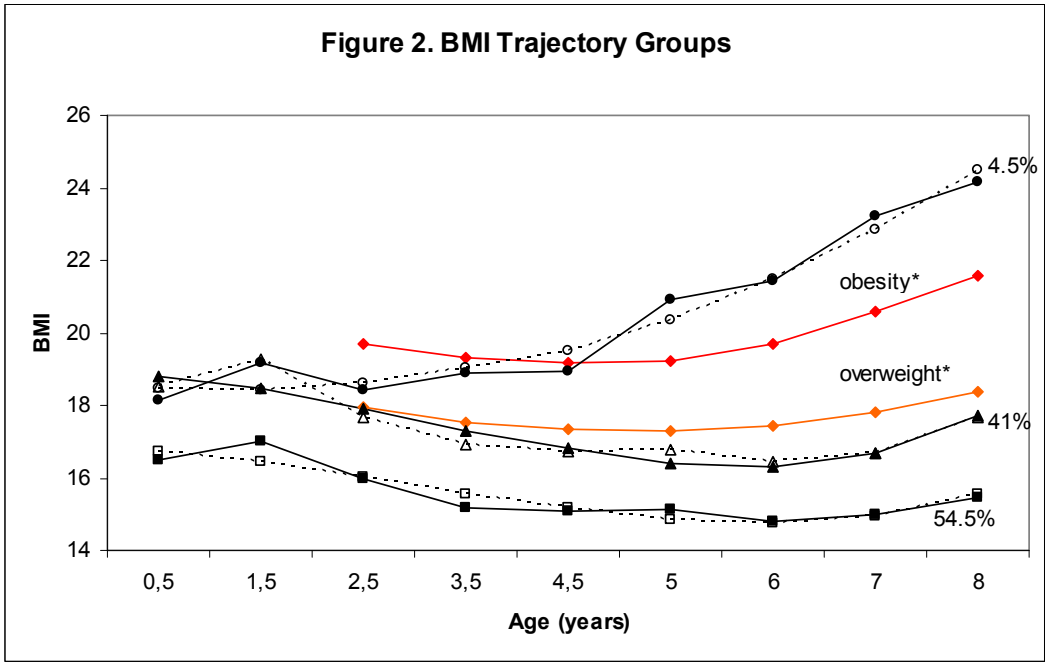
In the first phase of analysis, three distinct groups of BMI trajectories were identified, which we have labeled “low stable BMI”, “moderate BMI” and “high-rising BMI”. These results are illustrated in Figure 1 below. The low stable BMI trajectory represents 54.5% of our sample population (n=1057 children), the moderate trajectory is composed of 41% (n=802 children), and the high-rising

trajectory includes 4.5% (n=88 children). The dashed lines represent the expected values, the solid lines represent the observed values[3].



The orange and red lines in figure 2 (following page) have been added to the graph for comparison purposes. They indicate cut-off values (boys and girls values averaged together) for overweight and obesity in childhood, as defined by the International Obesity Task Force (IOTF) (Cole, 2000).

[3] Predicted values are calculated as the expected value of the random variable depicting each group's BMI values. Expected values are computed based on model coefficient estimates. Actual values are computed as the mean values for all persons assigned to the various groups identified in estimation (Nagin, 1999).



*according to IOTF (International Obesity Task Force) Age-Specific Cut-off Values

The high rising group included children who's BMI, at eight years of age, exceeded the IOTF cut-off value for obesity (boys: 21.6 kg/m², girls: 21.57 kg/m²) (Cole, 2000). The "moderate" and "low-stable" trajectory groups showed similarly shaped developmental patterns, with BMI values of the "moderate" group being higher than that of the low group at each time point. At eight years of age, most children in these two groups had BMI values inferior to the IOTF cut-off for overweight, although the upper 10th percentile of the moderate group is made up of children who fall into the "overweight" category, as defined by Cole. Table IV illustrates these variations in BMI values across the three trajectories. The right side of the table provides a comparison with the aforementioned IOTF cut-off points for boys and girls.

Table IV. Mean BMI, Standard Deviation and 10th and 90th Percentiles for Each Data Point in Each of the Three BMI Trajectory Groups

	Low-stable		Moderate		High-rising		BMI Cut-Off Points*				
								Overweight		Obesity	
5 months								**Boys**	**Girls**	**Boys**	**Girls**
Mean (SD)	16.45	(1.83)	18.55	(2.56)	19.16	(2.13)					
10th-90th percentile	[14.5	18.6]	[15.89	21.26]	[15.46	20.84]					
1.5 years											
Mean (SD)	16.93	(1.83)	19.38	(2.57)	19.03	(0.32)					
10th-90th percentile	[14.85	19.27]	[16.64	22.34]	[15.76	22.23]					
							2 yrs	18.13	17.76	19.8	19.55
2.5 years											
Mean (SD)	15.9	(1.54)	17.74	(1.74)	18.4	(4.18)					
10th-90th percentile	[14.14	17.96]	[15.72	20.07	[15.66	21.32]					
							3 yrs	17.69	17.4	19.39	19.23
3.5 years											
Mean (SD)	15.11	(1.34)	17.03	(1.63)	18.9	(2.3)					
10th-90th percentile	[13.5	16.8]	[15.2	19.1]	[16.22	21.97]					
							4 yrs	17.47	17.19	19.26	19.12
4.5 years											
Mean (SD)	15.05	(1.36)	16.81	(1.49)	18.96	(2.36)					
10th-90th percentile	[13.37	16.72]	[15.07	18.68]	[15.7	22.49]					
5 years							*5 yrs*	17.42	17.15	19.3	19.17
Mean (SD)	15.11	(1.45)	16.81	(1.88)	21.06	(2.31)					
10th-90th percentile	[13.42	16.83]	[14.74	18.99]	[18.13	24.05]					
6 years							*6 yrs*	17.55	17.34	19.78	19.65
Mean (SD)	14.77	(1.09)	16.5	(1.35)	21.49	(2.43)					
10th-90th percentile	[13.42	16.19]	[14.82	18.22]	[18.96	24.85]					
7 years							*7 yrs*	17.92	17.75	20.63	20.51
Mean (SD)	14.94	(1.23)	16.73	(1.46)	23.28	(2.51)					
10th-90th percentile	[13.45	16.52]	[14.86	18.65]	[20.49	27.91]					
8 years							*8 yrs*	18.44	18.35	21.6	21.57
Mean (SD)	15.41	(1.5)	17.69	(1.89)	24.2	(2.44)					
10th-90th percentile	[13.65	17.29]	[15.47	20.43]	[21.38	28.37]					

*IOTF percentile growth charts (Cole et al. 2000)

4.5.3 Verifying an Association with Age

Results from the student T-test verifying whether age is associated with membership in the high-rising trajectory group indicate that:

At age 6, there is a non-significant difference (p=0.228) between the mean age of 74.25 months (member of high-rising group) and 73.78 months (not member of high rising group).

At age 7, there is a non-significant difference (p=0.202) between the mean ages of 86.28 months (high-rising group) and 85.80 months (not member of high-rising group).

At age 8, there is a non-significant difference (p=0.747) between the mean ages of 98.21 months (member of high-rising group) and 97.04 months (not member of high-rising group).

While a higher mean age in months is found for the high-rising group, this difference is not statistically significant. We therefore remain confident that membership in the high-rising trajectory is not explained by an advanced age.

4.5.4 KML Cluster Modeling

The trajectory model with the best fit according to the Calinski criterion is found in Appendix F. While the proportions in the given trajectories are slightly different, it can still be seen that the KML method confirms the trajectories that were found in SAS Proc Traj, particularly in terms of the shape of the trajectories.

4.5.5 Variance Component Analyses

For the three variance component analyses, we observed Var (e1) = 2.0, Var (e2)= 1.7, and Var (e3)=1.0. If the difference in measurement methods had been a substantial problem, we would expect the residual variance of the second model (Var (e2)), which includes different measurement methods, to be substantially higher than the other two variances (Falissard, 2005). This was not the case, as Var (e2) is of a lesser value than Var (e1).

4.5.6 Trajectory Modeling by Sex

When BMI trajectories were modeled by sex (Figures 3 and 4), it became apparent that a slight sex difference existed in the proportions, but not in the patterns of the trajectories. Specifically, the progression of BMI over time followed the same shape for boys and girls.

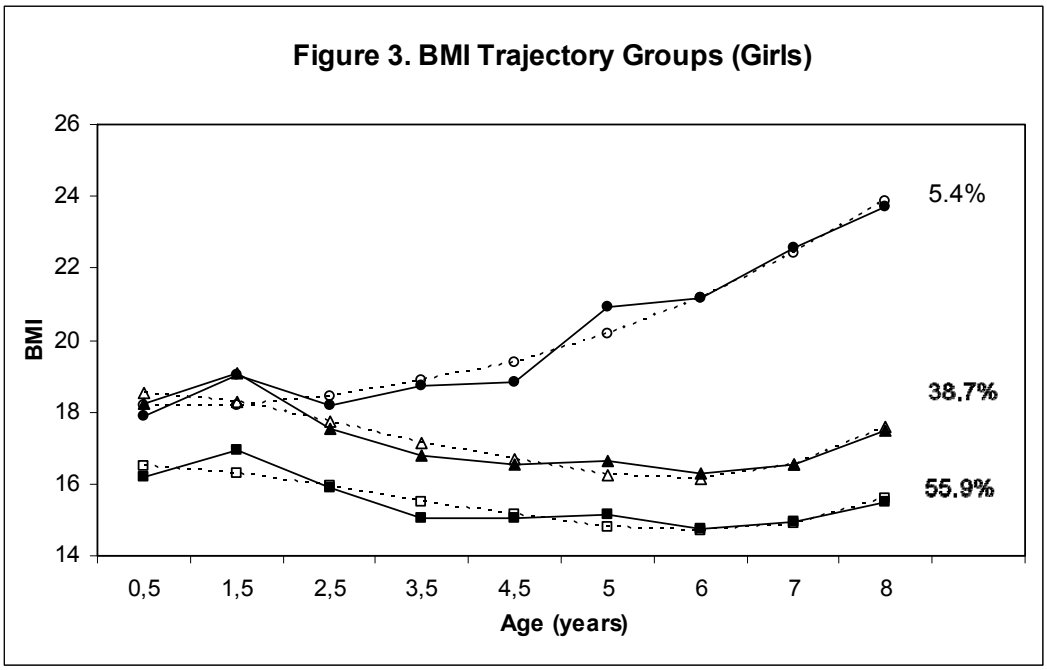


Figure 4. BMI Trajectory Groups (Boys)

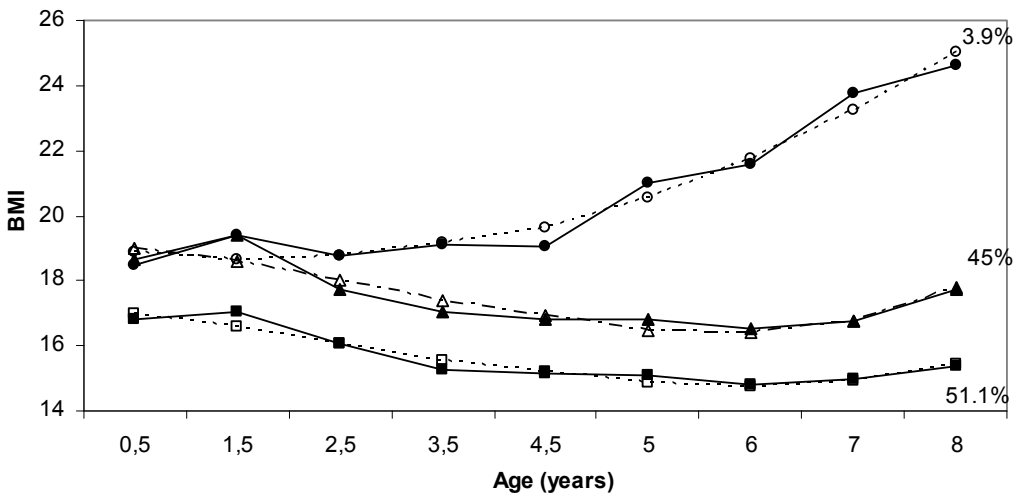


Table V illustrates the sex differences in trajectory group membership, and also indicates that these are significant at the alpha= 0.05 significance level. Specifically, the proportion of boys in the moderate trajectory was greater than that of girls. Conversely, the proportion of boys in the high trajectory was lower than that of girls.

Table V. Proportion of Boys and Girls in BMI Trajectory Groups

	Trajectory Group			Chi-square test
	Low-stable	Moderate	High-Rising	of significance
	n, (%)	n, (%)	n, (%)	
Boys	485(45.5)	461(56.8)	38(47.5)	p< 0.001
Girls	581(54.5)	350(43.2)	42(52.5)	

Pearson x^2 statistic= 23.977

We see however, in Table VI, that the underlying shapes of the curves (parameters) are very similar.

Table VI. Parameters (intercept, linear, curvilinear) for Models by Sex

Trajectory	Girls			Boys			All		
	Parameter	Estimate	SE	Parameter	Estimate	SE	Parameter	Estimate	SE
Low-stable	Intercept	16,4586	0,1268	Intercept	17,1069	0,12335	Intercept	16,7741	0,0878
	Linear	0,0089	0,0102	Linear	-0,02	0,01067	Linear	-0,0048	0,0073
	Quadratic	-0,0013	0,0002	Quadratic	-0,0008	0,00025	Quadratic	-0,001	0,0002
	Cubic	<0,0001	<0,0001	Cubic	<0,0001	0	Cubic	<0,0001	0
Moderate	Intercept	18,5307	0,16027	Intercept	19,0207	0,138	Intercept	18,8296	0,1064
	Linear	0,0083	0,0127	Linear	-0,0033	0,0114	Linear	0,0018	0,0087
	Quadratic	-0,0017	0,00029	Quadratic	-0,0015	0,0003	Quadratic	-0,0018	0,0002
	Cubic	<0,0001	<0,0001	Cubic	<0,0001	<0,0001	Cubic	-0,0016	<0,0001
High-Rising	Intercept	18,2293	0,31154	Intercept	19,0239	0,3319	Intercept	18,5821	0,2294
	Linear	-0,0139	0,01332	Linear	-0,0378	0,0154	Linear	-0,0251	0,0102
	Quadratic	0,0007	0,00012	Quadratic	0,001	0,0002	Quadratic	0,0008	0,0001
n	973			984			1957		
BIC	BIC=-15095.74			BIC= -14636.37			BIC= -29767.80		

The lack of differences between the sexes in terms of the shape of the trajectories indicated that the development of BMI for both sexes could be confidently estimated jointly. Yet, differences in proportions suggested that the sex of the child should be controlled in the prediction analyses.

4.6 Data Analysis, Phase 2: Risk Factors

4.6.1 Bivariate Analyses

Table VII reports the proportion of the individual risk factors associated with each of the three trajectory groups in bivariate analyses, as well as the significance of the association. It can be seen that the sex of the child ($p<0.001$), insufficient family income ($p=0.014$), lack of maternal high school diploma ($p= 0.006$), high maternal BMI ($p<0.001$), smoking during pregnancy ($p <.001$), mother's young age at childbirth ($p=0.002$), as well as membership in the highest quintiles of weight gain between birth and 5 months ($p<0.001$) were significantly

associated with a child's membership in the atypically elevated group. These variables were thereby included in multivariate analyses.

Table VII. Bivariate analyses:proportion of children in BMI trajectory groups according to each risk factor

Variables [%,(n)]		Trajectory Group			p Value on x^2 Test of Significance
		Low-stable (n=1 066)	Moderate (n=811)	High-rising (n=80)	
Child Characteristics					
Sex (girls)		54.5(581)	43.2(350)	52.5(42)	<0.001
Birthweight(g)	<2500	3.8(40)	3.5(28)	1.2(1)	<0.001
	2500-4000	88.8(943)	79.2(637)	90.0(72)	
	>4000	7.4(79)	17.3(139)	8.8(7)	
Birth Order	1st born	44.7(476)	45.6(370)	41.2(33)	0.542
	2nd	38.6(412)	40.1(325)	45.0(36)	
	3+	16.7(178)	14.3(116)	13.8(11)	
Gestational Age (<37 weeks)		3.6(38)	6.3(51)	5.0(4)	0.023
Difficult Temperament*		29.8(317)	29.3(236)	31.2(25)	0.920
Short Sleeper*		20.9(215)	20.3(160)	19.5(15)	0.926
Weight gain*	1 (lowest)	22.6(238)	14.4(115)	18.4(14)	<0.001
(quintiles)	2	22.4(235)	18.9(151)	10.5(8)	
	3	20.6(216)	20.7(165)	23.7(18)	
	4	19.3(203)	20.5(164)	19.7(15)	
	5 (highest)	15.1(159)	25.5(204)	27.6(21)	
Maternal Characteristics/Behaviours					
BMI (>25 kg/m²)**		24.5(256)	31.6(250)	60.8(48)	<0.001
Smoked during pregnancy		23.7(253)	25.3(205)	43.8(35)	<0.001
Breastfed Baby	not at all	27.8(294)	28.7(232)	30.4(24)	0.826
	<3 months	30.2(320)	29.7(240)	34.2(27)	
	>3 months	42(444)	41.7(337)	35.4(28)	
Young Mom*		20.3(206)	21.3(167)	37.3(28)	0.002
Severe Depressive Symptoms*		20.0(213)	22.1(178)	27.5(22)	0.202
Immigrant		8.9(95)	10.5 (85)	13.8 (11)	0,248
Family Variables					
Socioeconomic Status:					
Insufficient family income*		21.0(220)	22.2(178)	35.0(28)	0.014
Mother lacks HS diploma*		17.1(182)	15.9(129)	30.0(24)	0.006
Poor Family Functioning*		21.2(224)	18.4(148)	24.1(19)	0.216

*at time 1 (baby was 5 months).
**at time 2 (baby was 17 months).

4.6.2 Correlation Analyses

The results from the measures of correlation suggest significant correlations in the order of approximately *r*=0.01 to r=0.5, with the highest correlation being between birthweight and gestational age (r=0.49; significant at the p=0.01 level).

When looking at the above-mentioned variables identified in the literature as being potentially correlated, we do indeed find significant, albeit relatively small, correlations (at the p=0.01 level). *Smoking during pregnancy* is significantly correlated with low SES (r=0.133 for *insufficient income*, r= 0.236 for *high school diploma*), *young mother* (r= 0.25), *maternal depression* (r=0.035) and *family functioning* (0.091). *Average early infancy weight gain* (in quintiles) was significantly correlated with *low birthweight* (r=-0.41), *birth order* (r=-0.13), *mother's BMI* (r=-0.08), or *mothers smoking in pregnancy* (r=-0.12). *High school diploma* and *insufficient income* showed a correlation of 0.34.

Given that the zero-order correlations obtained were not very strong, and that we wished to control for the variables chosen for our model, no variables were removed or combined together based on these analyses.

4.6.3 Final Model: Multivariate Analysis

In the next step, Table VIII presents the results of the multinomial logit regression analysis aimed at identifying risk factors distinguishing the relatively small group of children (4.5%) in the atypically elevated trajectory from the other two groups in the context of a multivariate model. Variables were organized into "blocks" based on their type of risk factor: child characteristics, maternal and characteristics, and family characteristics. This choice of blocks corresponds to the factors that are most proximal and personal to the child (e.g. sex and weight gain) to those which are more distal (socioeconomic factors).
(Please note that an enlarged version of table VIII can be found in Appendix I.)

Table VIII. Multinomial logit regression analyses of the association between early childhood risk factors and BMI trajectories (between 1 ½ and 8 years) (n=1957).

	High-Rising vs. Moderate			High-Rising vs. Low-Stable			Moderate vs. Low-Stable		
Variables	**OR**	**95% CI**		**OR**	**95% CI**		**OR**	**95% CI**	
Child									
Average Monthly Weight Gain (1=upper two quintiles)**	1.250	[0.664	2.350]	2.570*	[1.373	4.813]	2.057*	[1.578	2.680]
Gestational Age (1= <37 wks)	0.750	[0.235	2.390]	2.010	[0.615	6.566]	2.680*	[1.474	4.871]
Birthweight (1=underweight)	2.302	[0.623	8.499]	1.002	[0.277	3.631]	0.435*	[0.209	0.909]
Birthweight (2=overweight)	0.591	[0.202	1.725]	1.648	[0.558	4.868]	2.790*	[1.869	4.164]
Sex of Child (1=female)	0.839	[0.463	1.520]	1.057	[0.586	1.908]	1.260	[0.984	1.612]
Maternal									
Smoking during pregnancy (1=yes)	2.238*	[1.212	4.134]	2.479*	[1.347	4.561]	1.108	[0.827	1.483]
Maternal BMI (1=overweight)	1.929*	[1.062	3.503]	2.565*	[1.416	4.647]	1.330*	[1.011	1.748]
Young Mom (1= <21 yrs)	0.960	[0.466	1.976]	0.978	[0.477	2.005]	1.019	[0.733	1.416]
SES									
Sufficient Income (1=no)	1.317	[0.684	2.538]	1.452	[0.756	2.788]	1.102	[0.813	1.494]
Maternal HS Diploma (1=no)	1.317	[0.618	2.806]	1.029	[0.487	2.178]	0.782	[0.541	1.130]

***statistically significant predictor (at the p ≤ 0.05 level)**

****The complementary category refers to the reference category (0).**

When examining the magnitude of the differences in risks for membership in the trajectories, the differences are larger when comparing the high-rising with the low-stable trajectory than when comparing the high-rising with the moderate trajectory. The most important risk distinguishing the high versus the low group was the average monthly weight gain of the child between birth and 5months. Being in the 4th and 5th quintiles of weight gain increased the odds of membership in the high-rising BMI group by an odds ratio of 2.5. Two maternal characteristics were also associated with large and significant increases in the risk for membership in the high-rising group vs. the low-stable group. Maternal overweight or obesity was associated with a 2.6-fold increase in the odds ratio (OR) for membership in the high-rising BMI group. The OR for maternal smoking during pregnancy was nearly 2.5.

Because there are no significant interactions in the model (see Appendix H: Analysis of Interactions) we may consider the OR's to be multiplicative. Therefore, for a child having a mother with both "at-risk" characteristics – maternal smoking and overweight/obesity- the OR for membership in the high BMI trajectory is increased by an OR of 6.4. A child exposed to all three of the significant risk factors for membership in the high-rising vs. low-stable group

would have an increased risk of 16.4. However, results combining risk factors must be interpreted with caution, given that unknown less-than significant interactions may possibly affect their validity (Falissard, 2005).When comparing the high-rising vs. moderate group, the same maternal health characteristics were also found to be significant (OR for smoking during pregnancy= 2.2, OR for maternal overweight=1.9, combined risk of 4.3). Lastly, when comparing membership in the moderate vs. the low-stable group, four risk factors were found to be positively associated with membership in the moderate group: high birthweight (OR=2.79), low gestational age(<37 weeks) (OR=2.68), greater average monthly weight gain from birth to 5 months (OR=2.06), and maternal overweight/obesity (OR=1.3). Low birth weight reduced the risk of membership in the moderate group as compared to the low-stable group (OR=0.435).

CHAPTER 5: DISCUSSION

5.1 Summary and Interpretation of Main Findings

This study had two main objectives. The first was to model the developmental trajectories of BMI beginning in early childhood among a representative sample. Our group-based developmental trajectory model, spanning 9 time points within the first 8 years of children's lives, allowed for the identification of a group of children with a rising BMI trajectory, and two groups of children with relatively stable BMI's over the childhood years. The second objective was to identify early family and child characteristics associated with the subsequent BMI development. We discuss each objective in the following section.

Developmental trajectories of BMI

Our findings indicate that three distinct developmental trajectories of BMI exist for this population sample of Quebec children: low-stable (54.5%), moderate (41.0%), and high-rising (4.5%). The 3-group model was found to be robust and replicable across two estimation methods. Specifically, the trajectories obtained were identical with a semi-parametric mixture model as well as a non-parametric algorithm. Moreover, the model was determined without a priori hypotheses regarding the existence of distinct trajectories, their number or shape, and is able to quantify the uncertainty about an individuals' group membership. This uncertainty in group membership is quantified in terms of posterior probabilities (of group membership) with associated confidence intervals (Nagin, 1999).

Interestingly, past research has identified the existence of a component distribution of BMI within the population. In a study of 3,577 Danish adoptees,

Price et al. (1989) determined that, after removing age and sex effects, three components make up the BMI distribution, causing the skewness that is observed. This cross-sectional study parallels our longitudinal data which points to the existence of three distinct paths an individual's BMI may take, and this starting very early on in life.

Family and child characteristics associated with BMI developmental trajectories

High-Rising vs. Moderate and Low Stable Trajectories

The group-based method of modeling developmental trajectories allows us to identify characteristics of individuals who are members of a particular group (Jones, 2001). Our findings show that maternal smoking during pregnancy, maternal overweight, as well as the child being in the two highest quintiles of weight gain in the first 5 months of life are associated with membership in the high-rising trajectory of BMI. These results are in line with the growing evidence that the perinatal environment has an important influence on later obesity (Breir, 2001, Huang, 2007, Power & Jefferis 2002, Vickers, 2000). These perinatal factors may not only be associated with future obesity, but, as our results suggest, are associated with a distinct trajectory of obesity development beginning in the preschool years.

We found that having a mother who smoked during pregnancy was associated with an over two-fold risk for membership in the high-rising group versus either the low or moderate groups. Several studies have also linked smoking during pregnancy with an increased risk of child (Leary, 2006, Mizutani,

2007, Reilly, 2005, Toschke, 2002) and adult (Power & Jefferis, 2002) obesity. Maternal smoking is said to restrict fetal growth (Adair, 2008), leading to a lower birthweight, which has also been associated with the development of obesity. This may be due to the compensatory rapid postnatal catch up growth (Ong, 2006) or the programming of appetite regulation (Von Kries, 2002) that may occur. An alternative explanation is that smoking and obesity are problems that have a common genetic vulnerability. In line with this idea, recent studies have shown that behaviour problems in the offspring of mothers who smoked during pregnancy have a common genetic risk (D'Onofrio, 2008, Rice, 2009). This is in contrast to the hypothesis that behaviour problems in the offspring of smoking mothers can result from the teratological effects of smoking. In order to demonstrate the genetic causal effect, researchers have used a sophisticated cross-fostering design among women seeking fertility assistance (Rice, 2009). Such a design could shed light on the genetic versus environmental etiology of childhood obesity.

The odds of being in the high-rising group were 1.93 and 2.57 times higher than the moderate and low-stable groups respectively, when the child's mother was considered overweight. The link between maternal and child overweight or obesity may be attributable to genetics or shared familial characteristics. An important genetic component seems to exist in the variation in BMI; though the extent of this influence is not known (Haworth, 2008). Results from twin studies range from 50-90% heritability of BMI (Maes, 1997), yet there is also evidence to support that a common etiology exists between what are considered normal variations in BMI and clinical obesity levels (Haworth, 2008). Certain studies have identified that correlations between parental and child overweight are not

significant in infancy (Stunkard, 1999) or even until 7 years of age (Safer 2001). However, as our high-rising trajectory separated itself at approximately 2.5 years, and there is a significant association with maternal BMI, we have reason to believe that the association between maternal and child BMI is important from at least that time (~2.5 years).

While the effects of rapid growth in infancy are difficult to isolate due to the fact that infancy weight gain is related to both intrauterine and post-infancy factors (Adair, 2008), numerous studies report significant positive associations between rapid growth in the first few weeks and up to the first two years of life with overweight in later childhood and/or adulthood (Ong & Loos, 2006). Our study corroborates this literature, as we observed the average monthly weight gain in the first 5 months of life and identified that children in the upper two quintiles of weight gain during this time presented a 2.6-fold increase in risk for being in the elevated vs. low-stable trajectory.

Our results agree in part with the previous study examining developmental trajectories of BMI in childhood in association with very early risk factors (Li, 2007). Using a latent growth mixture model and data from the *National Longitudinal Study of Youth* (NLSY79) (n=1739 children, aged 2 to 12 years), this study identified three trajectories, representing early onset (10.9%), late-onset (5.2%) and never overweight (83.9%). In consideration of the fact that our study ends at 8 years, the "early onset" trajectory is most comparable to our "high-rising" trajectory. The percentage of children forming part of their "early-onset" trajectory was 10.9%, compared to the 4.5% membership in our "high-rising" trajectory. There are two major explanations for this difference. First, inherent differences in risk factors for obesity development that may exist between the two

populations studied (United States circa 1986 vs. Quebec circa 1998). Second, our two studies differ in the methodology used for trajectory modeling. Authors Li et al. utilized a latent growth mixture model and defined "overweight" in terms of a dichotomous variable (overweight vs. not) using the CDC 95th percentile cut-off, whereas we used a semi-parametric mixture model and modeled brut BMI values across time.

Both our results as well as those of the NLSY79 indicate that maternal BMI is one of the most important risk factor for early onset obesity. The Li (2007) study further finds that the sex of the child (being male), black ethnicity, first-born children, high birthweight and mothers with high school education (out of a 4-category variable, from no high school diploma to more than one college degree) and lower middle-class income were all factors associated with the "early-onset" trajectory. Furthermore, smoking during pregnancy was not significantly associated to this trajectory, as it was to ours.

Certain differences in the predictors found to be associated with the high BMI trajectory in each study may be related to the differences in the proportion of the population with a given risk factor. For instance, the QLSCD study has only 10.2% of *all* types of ethnic minorities (includes Asian, African and Hispanic descents); it would therefore be unlikely to find an association with BMI and a certain ethnicity. This is in contrast to 15.3% of African Americans in the NLSY79 study, which provides the statistical power for a meaningful analysis of this risk factor. The same reasoning cannot, however, explain the difference for the maternal smoking during pregnancy risk factor. With 25.2% of the mothers in our sample having smoked, for at least part of their pregnancy, we detected an association with the overweight trajectory, whereas for the Li study, which

included 31% mothers who smoked during pregnancy, no association was found with their early-onset trajectory.

Moderate vs. Low-Stable Trajectory

While we were particularly interested in looking at the factors associated with membership in the high-rising trajectory, we also looked at the factors distinguishing the moderate from the low-stable trajectory. These potential risk factors were found to be significant during this analysis: high early average monthly weight gain, high birthweight, short gestational age (pre-term infants), and high maternal BMI. Note that both the low-stable and moderate trajectories include predominately "normal" weight children, with the moderate group including a small proportion of overweight children (see Table IV: moderate trajectory column, 90th percentile). Thus, the predictors distinguishing the moderate and low trajectory groups would seem to explain some of the variance between the groups in body shape and composition that is found within the general population (Price, 1989).

Of the four factors, two of these were non-specific risk factors, in that they were also associated with the high-rising to the low-stable comparison. These were early average monthly weight gain (upper two quintiles) (OR=2.06, CI=1.578; 2.68) and maternal BMI (OR=1.33, CI=1.01; 1.75). We may view these risk factors as possibly acting on a continuum, with similar mechanisms underlying the link between both risk factors (rapid weight gain in childhood and maternal overweight) and both the low and moderate trajectory groups. This would be in line with the notion that a common etiology between what are

considered normal variations in BMI and clinical obesity levels does exist (Haworth, 2008).

High birthweight and pre-term births were specific risk factors for the moderate vs. low-stable trajectory. The literature does indicate a positive correlation between birthweight and childhood BMI (i.e. higher birthweight associated with higher BMI; Adair, 2008, Eriksson 2001, Hirschler, 2008, Rogers 2003, Oken & Gillman, 2003). Our study supports this notion for the general population, but not when it comes to distinguishing the smaller percentage of children who will reach pathological levels of obesity.

For the short gestational age risk factor designating a pre-term infant (WHO, 2001), it is perhaps not the fact that the infant is born pre-term, but rather the subsequent rapid growth (80% of pre-term infants undergo "catch-up growth" (Euser, 2008)) that may be associated with higher weight in childhood, in our study and others (Ong & Loos, 2006).

5.2 Limitations and Methodological Issues

BMI as a measurement tool

There is a general questioning in the literature as to the validity of using BMI to measure adiposity. It is considered a proxy measurement for more sophisticated and reliable tools, such as Dual Energy X-ray Absorptiometry (DEXA) and Bioelectrical Impedance, and may misclassify certain children (Wang, 2004). However, it is a very useful tool for epidemiological studies, when it is not feasible to administer the more sophisticated measures. As such, it has

been an essential index to document the recent worldwide increase in the prevalence of obesity (Schousboe, 2003).

For our study, BMI has been calculated using height and weight data which was made available through maternal report for the first six time points, and from anthropometric measurements using standard measurement procedures when the children were 6, 7 and 8 years old. Maternal report data is not considered as reliable as measured data and we acknowledge this as a limitation in our study. In modeling BMI using developmental trajectories, however, measurement error has been reduced, since the BMI trajectory variable reflects a general level of BMI over several time points.

Differing Measurement Methods

The fact that we used two different methods for measuring BMI may potentially impact the interpretation of our results. Three variance component analyses were performed, however, to assess the impact of this difference in methods used: one with measurements at 4.5 and 5 years (same method), one with BMI measurements at 5 and 6 years (different methods), one with measurements at six and seven years (same method). As was previously mentioned, if the difference in measurement methods was a substantial problem, we would have expected the residual variance of the second model to be substantially higher than the other two (Falissard, 2005). This was not found to be the case; we therefore remain confident in our results.

Maternal Report of Child BMI data

In 2002, when the children in the ELDEQ study were approximately 4 years of age, a subset of the population sample had their height and weight measured by a trained interviewer and this data was used to study the accuracy of the previously reported maternal report data (Dubois & Girard, 2007). A 3% overestimation of overweight prevalence was found, due to misreported values for the child's weight, but not height. This misreporting was also found to occur disproportionately among boys, as well as children of lower socioeconomic status.

As the observed trajectories are not stable throughout time, and only one of the trajectories significantly increases, we can be confident that our results are not attributable to a general overestimation of weight. Furthermore, the fact that our overweight trajectory includes a greater percentage of girls than boys does not fit with the observation that a greater overestimation was present for boys BMI at 4.5 years. For these reasons we remain confident in our results; however the possibility that mothers of low SES may be more likely to misreport their child's weight is a methodological concern.

Trajectories and measurement error

Every measurement has an associated error. Our method allows us to profit from numerous measures over time to estimate the pattern of BMI development in children, and in doing so, error is decreased (Nagin, 1999). However, trajectories estimate general tendencies, as patterns in time. They allow us to compare individuals and determine who is higher in terms of a 'rank order'. They do not represent an exact path followed by an individual; but rather, a general tendency. Therefore, while error is decreased because we use many

measurements, the groups that were identified are simply estimations of a developmental pattern.

Additional Limits

Finally, we must also acknowledge that our study represents bidirectional links only, and causality cannot be conferred from these results. The identified early life risk factors are not causal of obesity; rather they increase one's probability of following a developmental pattern resembling the high-rising trajectory. It is possible that a third unexamined variable may explain one or more of these links. Due to the quality of the available data, we were not able to analyze certain risk factors of interest, such as alcohol consumption and postpartum depression. The study also lacks genetic biomarkers which could separate early life environmental influences from genetic predisposition (Li, 2007).

5.3 Implications of Study

As for all research, replications using similar methods are needed before major definitive conclusions are drawn. Still, together with previous findings, the present results point to some implications for the field of public health. We have identified a group of Quebec children representing approximately 4.5% of our sample, who, at approximately 2.5 years of age, began to follow a rising developmental trajectory of BMI. In itself, this has important implications, for we are provided with an innovative method for looking at children's BMI development. The group-based developmental trajectories allow us to analyze BMI development in a simple, natural fashion and to group together children who tend to follow a similar pattern. This is in contrast to the existing approach using the percentile growth curve charts (Cole, 2000), which does not allow us to view

children's BMI development across time. Rather, the growth charts assume that children will generally maintain the same level of adiposity relative to the population throughout childhood (Willms, 2004).

The ability to target very early risk factors is important. It may reduce the likelihood that a child will follow an atypically elevated track of BMI development, which may eventually lead to adolescent and adult obesity, and increased risks for numerous physical and mental health problems. The identification of maternal smoking and BMI, as well as early childhood weight gain, as being associated with the high-rising trajectory, provide us with important considerations in the planning of public health programs aimed at reducing child obesity. This study supports the need to target these early-life risk factors, such as through preventive interventions aimed at women who are pregnant or of childbearing age in order to promote healthy weights and smoking cessation, and to keep a watchful eye on those infants who gain weight rapidly in early infancy.

Finally, the economic burden on our health care system due to obesity is large. A recent analytical review suggests that $4.3 billion, or about 2.2% of total health care costs in Canada were attributable to obesity in 2001 (Katzmarzyk and Janssen, 2004). The authors estimate that, of this figure, $1.6 billion can be attributed to direct costs and $2.7 billion to indirect expenditures. Early prevention is ideal for minimising this burden in the future.

5.4 Further Research

Based on the implications of this and previous studies, intervention studies are now required in order to determine the best methods for targeting the risk

factors of smoking and overweight/obesity in pregnancy. In addition, we are interested in exploring the following areas of research:

Exploring risk factors in middle childhood

We would like to follow-up this study with one that will explore risk factors becoming more prominent after 2.5 years. The study in question would include the same trajectories of BMI that were previously modeled, but the risk factors explored would include variables related more to the child's eating and physical activity habits, home and day-care/pre-school environment. For example, television watching (Reilly, 2005) and cognitive stimulation/opportunities for productive activity at home (OBrien, 2007) will be explored. In addition, the age of adiposity rebound would be taken into consideration at this time (Singhal & Lucas, 2004). Such a study would provide us with interesting insight into the middle childhood factors that distinguish children in the high-rising trajectory, and the relative importance of these factors, as compared to those that were significant during the perinatal period.

Increasing accuracy

The use of repeated and objective anthropometric measurements would allow for increased accuracy of assessment of the BMI variable, thereby increasing the validity of the study. Variables that we were not able to look at could also be examined. For example, maternal alcohol consumption, maternal diabetes, variables related to the father, and maternal nutrient intake during pregnancy.While it was once a common Western belief that maternal nutrition during pregnancy was not so important due to regulatory mechanisms in place that

ensure proper nourishment of the fetus, increasing evidence indicates how this is not the case, and that the mothers body composition and dietary intakes during pregnancy do play a significant role (Godfrey & Barker, 2000). It would therefore be of interest to explore this more thoroughly in future research.

Advanced Research Designs

More sophisticated research designs that could separate the genetic effects from the perinatal environment may be very useful in future research. As was previously mentioned, the cross-fostering design among women seeking fertility assistance to examine child obesity may present a good way to disentangle the genetic versus environmental etiology of this phenomenon. This information would allow us to better understand the mechanisms behind the maternal risk factors that were identified as being significant in our study. Some studies of this nature have been done to explore the genetics of birthweight (Morton, 1955, Brooks, 1995), however it would be useful to now look more closely at maternal overweight. Indeed, maternal overweight may be associated with either genetic and/or perinatal environment risk factors and act via a potentially imbalanced fetal nutrient supply with excess sugar (Barker, 2001). One could also explore whether the effects of smoking occur due to restrained fetal growth or a common genetic vulnerability (Rice, 2009) in this way.

Future research into epigenetic mechanisms is also of great interest in order to determine if these may play a role in the link between the perinatal risk factors and later obesity. Epigenetics, "the study of stable alterations in gene expression potential that arise during development and cell proliferation" (Jaenish & Bird, 2003), is thought to potentially underlie the fetal origins hypothesis

(Waterland, 2009). This presumes the occurrence of "programming" (or "metabolic imprinting") by way of early influences on epigenetic mechanisms.

Validation of Group-based Trajectories for the study of Child Obesity

As the procedure of modeling group-based developmental trajectories of BMI has rarely been used, future studies are also needed to distinguish between the results examining trajectories and those examining obesity at specific time points, in order to determine just how valuable this technique may be. For example, it would be useful to compare whether additional information be obtained regarding the risk factors associated with specific trajectories versus obesity status at a single point in time.

Joint Development of Obesity and Depression

Finally, the important mental health correlates of overweight and obesity have been less often considered than those related to physical health. The literature to date points to a link between obesity and depression, particularly among women (Dong, 2004, Dragan 2007, Stunkard, 2003,). The most consistent findings have been found for the adult population (Erickson, 2000). However, a few studies in children and adolescents have noted increased levels of depressive symptoms, particularly a decrease in self-esteem, and an increase in emotional distress and weight related distress in girls presenting an elevated BMI (Erickson, 2000, Hesketh, 2004, Strauss, 2000). Research is needed in order to clarify the role of psychological distress, which may not only stem from, but could also perpetuate disordered eating behaviours into adolescence and adulthood.

In future studies, we intend to examine the etiology of obesity and depression by studying the developmental origins of these two conditions. The modelling of joint trajectories of BMI and levels of depressive and anxiety symptoms in childhood will be utilized for this purpose.

CHAPTER 6: CONCLUSION

This study allows for an enhanced understanding of the development of adiposity throughout early childhood, by identifying trajectories of BMI development and early-life risk factors associated with these trajectories. Antecedents of childhood obesity can be identified during pregnancy. Intervention studies are needed in order to test the possibility that targeting maternal smoking and maternal obesity during pregnancy would reduce the risk of childhood obesity in the offspring.

Given the pervasiveness and complex nature of obesity in present-day society, our ability to target early-life risk factors that are associated with this problem may be key in reducing future obesity prevalence rates.

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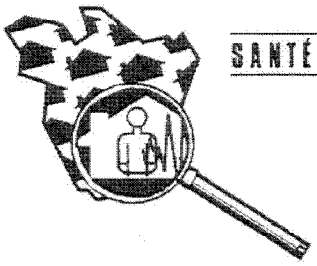
Appendix A: Letter to the Parents



En 2002... J'aurai 5 ans !

Voici une belle occasion de nous parler de vos enfants!

Bonjour chers parents,

Comme plus de 2 000 parents dont l'enfant le plus jeune est âgé d'environ 5 mois, vous avez été choisis au hasard pour participer à une grande première québécoise, soit la réalisation d'une étude qui vise spécialement les très jeunes enfants.

En effet, Santé Québec, en collaboration avec des spécialistes qui ont particulièrement à coeur la santé et le bien-être des enfants d'âge préscolaire, entreprend une étude annuelle qui permettra de mieux comprendre le développement des jeunes enfants québécois. La brochure ci-jointe présente en détail les objectifs de l'étude et le rôle important que les parents tiennent dans ce projet. Nous vous encourageons à lire cette brochure qui mentionne, entre autres, l'objectif global de notre projet, qui est «de mieux comprendre pourquoi certains enfants semblent plus prêts que d'autres à entrer à l'école ».

Une intervieweure du Bureau des intervieweurs professionnels (BIP) vous téléphonera d'ici 2 semaines et se fera un plaisir de vous expliquer les grandes lignes de l'étude et de répondre à vos questions.

Il est également important de mentionner que Santé Québec et le Bureau des intervieweurs professionnels assureront de manière très stricte la confidentialité de toutes les informations que vous partagerez avec nous dans le cadre de cette étude.

D'ailleurs, sachez que nous demeurons disponibles en tout temps pour vous rassurer quant au sérieux de cette étude et quant à la nécessité de votre participation.

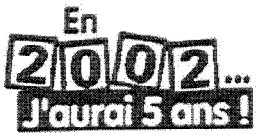
Merci à l'avance de votre attention et... de votre collaboration.

Mireille Jetté
Coordonnateur du projet, Santé Québec

Daniel Tremblay
Directeur, Santé Québec

600 ouest, boul. René-Lévesque, 10ième étage, Montréal (Québec) H3B 1N4
Téléphone: (514) 873-4749 Télécopieur : (514) 864-9919

Appendix B: Parent Consent Form



2 – 9 8 – – – – – – – – – – – – – –
1 2 3 4 5 6 7 8 9 10 11 12 13 14

« En 2002... J'aurai 5 ans! »
Étude expérimentale sur le développement des enfants du Québec
ÉLDEQ - Volet 1998

FORMULAIRE DE «CONSENTEMENT LIBRE ET ÉCLAIRÉ»
Je comprends que ce formulaire fait partie de l'Étude «En 2002... J'aurai 5 ans!». Un groupe de chercheurs de 5 universités québécoises (soit Université Concordia, Université de Montréal, Université de Sherbrooke, Université Laval, Université McGill) mène cette étude en collaboration avec Santé Québec, ses partenaires et le ministère de la Santé et des Services sociaux (MSSS) du Québec.

On m'a expliqué que le BUT de cette étude est de recueillir des renseignements qui aideront à mieux connaître les facteurs qui peuvent influencer le développement des enfants du Québec.

Je reconnais que ma participation à cette étude est VOLONTAIRE, que je suis LIBRE d'y participer et que les renseignements que je donnerai seront traités de manière CONFIDENTIELLE et ANONYME. Tous les renseignements NOMINATIFS que je divulguerai ou dont j'autoriserai l'utilisation seront traités et protégés selon les normes de la LOI DE LA COMMISSION D'ACCÈS À L'INFORMATION du Québec.

Je comprends qu'une personne identifiée par Santé Québec/BIP se présentera à mon domicile, complètera avec moi des questionnaires et laissera des instruments que moi et mon/ma conjoint/e devrons compléter et retourner par la poste. L'intervieweure m'a informé/e qu'en moyenne l'entrevue à la maison durait 1 heure 30 minutes.

Je comprends aussi que pour assurer ma participation aux autres volets de cette étude annuelle, Santé Québec me contactera au cours des quatre prochaines années.

Je, soussigné/e, consens à participer de plein gré à cette enquête longitudinale. Je certifie qu'on me l'a expliqué/e verbalement, qu'on a répondu à toutes mes questions et qu'on m'a laissé le temps nécessaire pour prendre une décision.

Je, soussigné/e, reconnais être libre de me retirer en tout temps sans que cela ne me nuise ou ne m'occasionne des préjudices.

_____ _____
Signature du/de la répondant/e Date

RÉSERVÉ À LA SIGNATURE DE L'INTERVIEWEURE
J'ai expliqué du mieux que j'ai pu l'objet et la nature du projet au/à la signataire. Je lui ai demandé s'il/elle avait des questions à me poser et, le cas échéant, j'y ai répondu. À mon avis, le/la signataire est parfaitement au courant des méthodes de l'étude, des implications de sa participation ainsi que du caractère VOLONTAIRE du présent consentement. J'ai remis un original de ce formulaire au/à la répondant/e et je ramène le second original que je remettrai aux autorités de l'Étude «En 2002... J'aurai 5 ans!».

_____ _____
Signature de l'intervieweure Date

Appendix C: ELDEQ Ethics Approval Response Letter

Phase I

Montréal, le 10 mars 1998

Monsieur Richard Tremblay
Université de Montréal - GRIP
350, Édouard-Montpetit
C.P. 6128
Montréal (Québec) H3C 3J7

Objet: Enquête Santé Québec «En 2002 j'aurai 5 ans»

Cher monsieur Tremblay,

Lors de sa dernière réunion tenue le 12 février dernier, le comité d'éthique de Santé Québec a étudié le projet en titre.

Le comité, après discussion, réserve pour le moment sa décision quant à l'approbation du dit projet et désirerait obtenir des précisions supplémentaires. Le comité approuvera le projet dès que les précisions et correctifs seront apportés à sa satisfaction.

Les éléments suivants pourraient être précisés davantage :

- Simplifier la lettre de consentement éclairé dont les termes apparaissent comme hermétiques et peu accessibles au commun des mortels d'y inclure de l'information simplifiée expliquant la nature du projet.
- Présenter une procédure claire et finale de transfert et de garde des données.

Veuillez agréer, Cher Monsieur Tremblay, l'expression de nos sentiments distingués.

Pierre Durand
Président du comité d'éthique

c.c. Daniel Tremblay, directeur de Santé Québec
Mireille Jetté, coordonnateure du projet

Appendix D: Ethics Approval: University of Montreal Faculty of Medicine

Université de Montréal

CERTIFICAT D'APPROBATION DU COMITÉ D'ÉTHIQUE DE LA RECHERCHE DE LA FACULTÉ DE MÉDECINE (CERFM)

Le Comité d'éthique a étudié le projet intitulé :

Trajectoires développementales de l'IMC durant l'enfance

présenté par : Mme Laura Pryor et Dre Sylvana Côté

et considère que la recherche proposée sur des humains est conforme à l'éthique.

Isabelle B-Ganache, présidente

Date de soumission ou d'étude : 27 Mai 2009

Date d'approbation : **Modifié et approuvé le 22 juin 2009**

Numéro de référence : **CERFM 104 (09)4#360**

N.B. Veuillez utiliser le numéro de référence dans toute correspondance avec le Comité d'éthique relativement à ce projet.

OBLIGATIONS DU CHERCHEUR :

SE CONFORMER À L'ARTICLE 19 DE LA LOI SUR LES SERVICES DE SANTÉ ET SERVICES SOCIAUX, CONCERNANT LA CONFIDENTIALITÉ DES DOSSIERS DE RECHERCHE ET LA TRANSMISSION DE DONNÉES CONFIDENTIELLES EN LIEN AVEC LA RECHERCHE.

SOLLICITER LE CERFM POUR TOUTES MODIFICATIONS ULTÉRIEURES AU PROTOCOLE OU AU FORMULAIRE DE CONSENTEMENT.

TRANSMETTRE IMMÉDIATEMENT AU CERFM TOUT ÉVÉNEMENT INATTENDU OU EFFET INDÉSIRABLE RENCONTRÉS EN COURS DE PROJET.

COMPLÉTER ANNUELLEMENT UN FORMULAIRE DE SUIVI.

Appendix E: Table of BIC Values

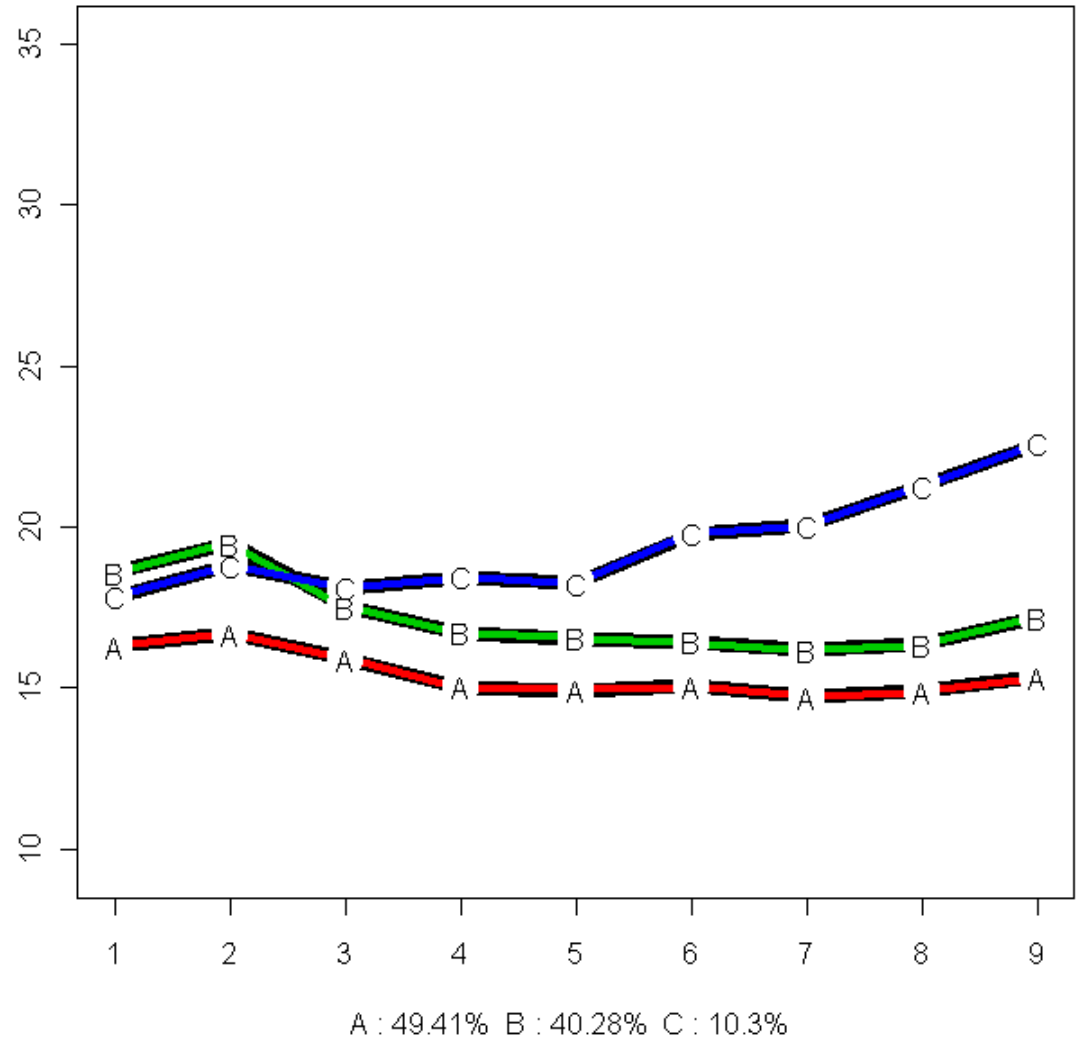
4 traj	Parameter	BIC	4 traj	Parameter	BIC
[0,0,0,0]	0	30932.30	[1,1,1,1]	4	30131.57
[1,0,0,0]	1	30744.87	[0,1,2,1]	4	30003.44
[0,1,0,0]	1	30472.96	[1,0,2,1]	4	29996.15
[0,0,0,1]	1	30472.96	[0,1,1,2]	4	30191.12
[0,0,1,0]	1	30472.96	[1,0,1,2]	4	30094.39
[0,2,0,0]	2	30197.03	[1,1,0,2]	4	30094.39
[2,0,0,0]	2	30310.59	[0,0,1,3]	4	30094.39
[0,0,0,2]	2	30727.56	[2,1,0,1]	4	30094.39
[0,1,1,0]	2	30458.65	[0,2,2,0]	4	30029.22
[0,0,1,1]	2	30269.44	[2,0,0,2]	4	29960.87
[0,1,0,1]	2	30458.65	[1,2,0,1]	4	29879.98
[1,0,0,1]	2	30648.17	[1,1,2,0]	4	30222.54
[0,0,2,0]	2	30197.03	[3,0,0,1]	4	30126.3
[1,0,1,0]	2	30350.86	[0,1,3,0]	4	30044.95
[1,1,0,0]	2	30358.71	[1,0,3,0]	4	30044.95
[1,2,0,0]	3	30101.67	[3,1,0,0]	4	30126.30
[0,0,0,3]	3	30137.36	[2,0,2,0]	4	30029.22
[2,1,0,0]	3	30177.86	[1,3,0,0]	4	30044.95
[0,2,1,0]	3	30248.17	[2,0,1,2]	5	29955.59
[0,0,1,2]	3	30248.17	[2,2,1,0]	5	30003.3
[0,0,2,1]	3	29983.31	[0,3,1,1]	5	29924.81
[1,1,1,0]	3	30126.95	[3,0,1,1]	5	29930.75
[0,1,0,2]	3	30248.17	[3,1,0,1]	5	30009.76
[0,0,3,0]	3	30137.36	[0,3,2,0]	5	30055.51
[1,0,0,2]	3	30322.77	[3,2,0,0]	5	29978.92
[0,1,1,1]	3	30229.19	[1,3,1,0]	5	29821.58
[1,0,1,1]	3	30126.95	[3,1,1,0]	5	30095.07
[1,1,0,1]	3	30126.95	[1,2,1,1]	5	29864.81
[0,2,0,1]	3	30162.79	[2,1,1,1]	5	30266.4
[2,0,0,1]	3	30248.17	[0,0,2,3]	5	29964.62
[0,1,2,0]	3	30101.67	[2,1,2,0]	5	29940.48
[1,0,2,0]	3	30101.67	[0,0,3,2]	5	30083.54
[0,3,0,0]	3	30137.36	[1,0,1,3]	5	30134.79
[3,0,0,0]	3	30137.36	[0,1,1,3]	5	29930.75
[2,0,1,0]	3	30651.95	[1,1,0,3]	5	30162.18
[0,3,1,0]	4	30089.45	[1,3,0,1]	5	29821.58
[3,0,1,0]	4	30089.45	[0,1,3,1]	5	29924.81
[0,2,1,1]	4	29996.15	[2,2,0,1]	5	29793.54
[2,0,1,1]	4	30486.64	[1,1,1,2]	5	30092.59
[2,2,0,0]	4	30029.22	[0,2,0,3]	5	30058.32
[1,2,1,0]	4	29879.98	[1,1,2,1]	5	29984.35
[2,1,1,0]	4	30482.48	[0,1,2,2]	5	29959.92
[0,0,2,2]	4	29960.87	[0,2,1,2]	5	29955.59

[0,2,0,2]	4	29960.87	[1,0,3,1]	5	29821.58
[2,2,1,1]	4	30482.48	[1,2,2,0]	5	29940.48
[0,0,3,1]	4	29921.95	[0,2,2,1]	5	29865.32
[0,3,0,1]	4	29921.95	[2,0,2,1]	5	29793.54
[0,1,0,3]	4	30161.49	[1,0,2,2]	5	29857.28
[1,0,0,3]	4	30044.95	[1,1,3,0]	5	29821.58
[2,3,0,0]	5	29983.5	[3,3,1,0]	7	29956.73
[0,2,3,0]	5	30058.32	[2,3,1,1]	7	29707.58
[2,0,3,0]	5	29983.57	[3,2,0,2]	7	29709.67
[1,2,0,2]	5	29857.28	[3,0,3,1]	7	29724.89
[2,1,0,2]	5	29917.74	[2,3,2,0]	7	29773.69
[0,3,0,2]	5	29899.75	[3,1,0,3]	7	29870.89
[3,0,0,2]	5	29899.75	[3,2,1,1]	7	29756.51
[3,0,2,0]	5	29978.92	[1,3,2,1]	7	29759.50
[2,0,1,3]	6	29921.51	[3,1,3,0]	7	29956.73
[1,1,3,1]	6	29927.56	[1,2,1,3]	7	29849.01
[2,3,1,0]	6	29755.88	[2,3,0,1]	7	29755.88
[3,2,1,0]	6	29742.35	[0,1,3,3]	7	29896.74
[3,0,1,2]	6	30445.77	[1,3,0,3]	7	29803.03
[0,3,2,1]	6	29801.26	[3,0,1,3]	7	29870.89
[3,2,0,1]	6	29742.35	[1,1,2,3]	7	29953.52
[2,1,0,3]	6	29921.51	[1,2,3,1]	7	29707.58
[3,0,3,0]	6	30022.36	[2,1,3,1]	7	29707.58
[1,3,1,1]	6	29799.85	[1,0,3,3]	7	29803.03
[3,1,1,1]	6	30270.25	[1,2,2,2]	7	29803.04
[0,3,0,3]	6	29903.49	[2,1,2,2]	7	29749.91
[1,1,1,3]	6	30096.34	[1,1,3,2]	7	29890.12
[1,0,2,3]	6	29861.04	[1,3,1,2]	7	29781.38
[0,3,1,2]	6	29892.50	[3,1,1,2]	7	30231.11
[1,2,2,1]	6	29833.34	[2,2,1,2]	7	29800.18
[0,1,2,3]	6	29861.04	[2,2,3,0]	7	29847.36
[1,1,2,2]	6	29845.44	[1,3,3,0]	7	29873.83
[1,2,3,0]	6	29944.39	[2,2,0,3]	7	29763.18
[2,2,0,2]	6	29759.39	[2,2,2,1]	7	29727.13
[0,2,3,1]	6	29806.16	[0,2,2,3]	7	29836.40
[0,1,3,2]	6	29799.28	[2,0,2,3]	7	29763.18
[1,0,3,2]	6	29892.99	[2,3,0,2]	7	29733.01
[1,3,0,2]	6	29799.28	[0,2,3,2]	7	29770.31
[3,1,0,2]	6	30378.94	[2,0,3,2]	7	29730.02
[2,1,1,2]	6	29949.73	[0,3,3,1]	7	29764.67
[2,0,3,1]	6	29755.88	[3,3,0,1]	7	29724.89
[0,2,1,3]	6	29959.31	[2,1,1,3]	7	29916.01
[2,2,2,0]	6	29759.39	[0,3,2,2]	7	29769.91
[1,2,0,3]	6	29861.04	[3,0,2,2]	7	29769.91
[2,0,2,2]	6	29759.39	[3,1,2,1]	7	29759.50
[0,2,2,2]	6	29759.39	[0,3,1,3]	7	29896.20

[2,1,2,1]	6	29769.77	[3,3,1,1]	8	29727.59	
[0,0,3,3]	6	30087.30	[3,3,2,0]	8	29743.10	
[3,0,0,3]	6	29903.49	[3,2,3,0]	8	29803.65	
[3,3,0,0]	6	29959.63	[2,3,2,1]	8	29617.92	
[3,0,2,1]	6	29742.35	[3,1,3,1]	8	29675.85	
[2,1,3,0]	6	29943.92	[1,3,2,2]	8	29685.31	
[0,3,3,0]	6	30022.36	[3,2,1,2]	8	29718.26	
[1,2,1,2]	6	29845.44	[1,1,3,3]	8	29893.86	
[1,3,2,0]	6	29870.09	[1,3,1,3]	8	29810.82	
[3,1,2,0]	6	29870.09	[3,1,1,3]	8	29810.82	
[2,2,2,2]	8	29668.70	[3,3,1,3]	10	29660.45	
[2,2,3,1]	8	29617.92	[3,2,3,3]	11	29552.11	
[2,3,3,0]	8	29777.48	[3,3,3,2]	11	29608.77	
[1,2,3,2]	8	29757.08	[2,3,3,3]	11	29542.68	maximized BIC
[1,3,3,1]	8	29741.13	[3,3,2,3]	11	29552.11	
[0,2,3,3]	8	29773.99	[3,3,3,3]	12	29546.35	
[2,0,3,3]	8	29733.81				
[2,3,0,3]	8	29736.76				
[3,2,0,3]	8	29713.46				
[0,3,2,3]	8	29773.70				
[1,2,2,3]	8	29806.83				
[2,1,2,3]	8	29753.47				
[2,2,1,3]	8	29753.47				
[2,3,1,2]	8	29718.26				
[2,1,3,2]	8	29688.97				
[3,2,2,1]	8	29680.38				
[0,3,3,2]	8	29729.97				
[3,0,3,2]	8	29729.97				
[3,0,2,3]	8	29713.46				
[3,3,0,2]	8	29690.85				
[3,1,2,2]	8	29810.16				
[3,3,2,1]	9	29630.39				
[3,2,3,1]	9	29612.51				
[2,3,2,2]	9	29578.34				
[3,1,2,3]	9	29706.36				
[2,3,3,1]	9	29630.39				
[1,3,3,2]	9	29708.96				
[2,3,1,3]	9	29692.43				
[3,3,3,0]	9	29694.64				
[2,2,2,3]	9	29672.49				
[2,2,3,2]	9	29665.68				
[1,2,3,3]	9	29692.43				
[2,1,3,3]	9	29665.68				
[0,3,3,3]	9	29733.64				
[3,0,3,3]	9	29694.64				

[3,3,0,3]	9	29694.64
[3,2,2,2]	9	29578.34
[3,1,3,2]	9	29708.96
[3,3,1,2]	9	29690.49
[1,3,2,3]	9	29689.08
[3,2,1,3]	9	29761.97
[3,3,2,2]	10	29613.49
[3,2,2,3]	10	29582.03
[2,2,3,3]	10	29582.03
[2,3,2,3]	10	29582.03
[1,3,3,3]	10	29660.45
[2,3,3,2]	10	29573.07
[3,2,3,2]	10	29573.07
[3,3,3,1]	10	29581.28
[3,1,3,3]	10	29581.28

Appendix F: Trajectory Model in KML



Appendix G: Zero-Order Correlation Table

Appendix B: Zero-Order Correlation Table

Variables	1*	2	3	4	5	6	7	8	9	10	11*	12	13*	14*	15*	16
Child																
1.Sex*		0,086	0,005	-0,03	0,015	-0,09	0,143	0,003	-0,02	0,01	0,026	0,014	0,019	0,039	-0,01	0,008
2.Birthweight(g)			0,117	0,499	-0,02	-0,04	-0,41	0,119	-0,18	0,092	-0,09	-0,02	0,006	-0,06	-0,08	-0,03
3.Birth Order				-0,01	-0,06	-0,07	-0,13	0,054	0	0,096	0,119	0,057	0,066	0,084	0,054	0,076
4.Gestational Age					-0,03	-0,05	-0,03	-0,01	-0,07	-0,04	-0,04	-0,02	0,026	-0,04	-0,06	-0,04
5.Difficult Temperament						-0,16	0,011	-0,03	0,015	0,036	-0,01	0,119	-0,01	0,007	-0,02	0,075
6.Short Sleeper							0,035	0,03	0,103	-0,27	0,021	-0,05	-0,2	-0,11	0,028	-0,04
7.Weight gain quintiles								-0,08	-0,12	-0,05	0,038	-0,02	-0,02	-0,01	0,008	-0,05
Maternal																
8.BMI									-0,05	-0,08	0,024	0,035	-0,02	0,041	0,043	0,025
9.Smoked during pregnancy*										-0,23	0,25	0,092	-0,14	0,133	0,236	0,091
10.Breastfeed Time											-0,18	-0,08	0,17	-0,06	-0,21	-0,05
11.Young Mom*												0,122	-0,04	0,342	0,408	0,079
12.Depressive Symptoms													0,096	0,195	0,128	0,452
13.Immigrant*														0,294	-0	0,106
Family																
14.Insufficient family income*															0,335	0,194
15.Mother lacks HS diploma*																0,147
16.Poor Family Functioning																

* variable catégorielle
blue: Statistically significant at 0.01 alpha level
yellow: Statistically significant at 0.05 alpha level

Appendix H: Analysis of Interactions

We explored potential interactions among our predictor variables. Given the large number of potential interactions that were tested (36), the Bonferroni correction was used in order to avoid finding statistically significant interactions by chance (Falissard, 2005). It is a criteria that corrects for the number of tests done by dividing the p=0.05 significance level by that number.

Bonferroni correction= alpha level (0.05) /*k* number of tests done

In our case 36 tests were done; we therefore only considered highly significant interactions: $p < 0.0014$. Our most significant interaction was p=0.014 (young mom x mother smoked during pregnancy). Therefore the analyses proceeded without the inclusion of interactions.

Appendix I: Multinomial Logit Regression Analyses (enlarged)

Table VII. Multinomial logit regression analyses of the association between early childhood risk factors and BMI trajectories (between 1 ½ and 8 years) (n=1957).

	High-Rising vs. Moderate			High-Rising vs. Low-Stable			Moderate vs. Low-Stable		
Variables	**OR**	**95% CI**		**OR**	**95% CI**		**OR**	**95% CI**	
Child									
Average Monthly Weight Gain (1=upper two quintiles)**	1.250	[0.664	2.350]	2.570*	[1.373	4.813]	2.057*	[1.578	2.680]
Gestational Age (1= <37 wks)	0.750	[0.235	2.390]	2.010	[0.615	6.566]	2.680*	[1.474	4.871]
Birthweight (1=underweight)	2.302	[0.623	8.499]	1.002	[0.277	3.631]	0.435*	[0.209	0.909]
Birthweight (2=overweight)	0.591	[0.202	1.725]	1.648	[0.558	4.868]	2.790*	[1.869	4.164]
Sex of Child (1=female)	0.839	[0.463	1.520]	1.057	[0.586	1.908]	1.260	[0.984	1.612]
Maternal									
Smoking during pregnancy (1=yes)	2.238*	[1.212	4.134]	2.479*	[1.347	4.561]	1.108	[0.827	1.483]
Maternal BMI (1=overweight)	1.929*	[1.062	3.503]	2.565*	[1.416	4.647]	1.330*	[1.011	1.748]
Young Mom (1= <21 yrs)	0.960	[0.466	1.976]	0.978	[0.477	2.005]	1.019	[0.733	1.416]
SES									
Sufficient Income (1=no)	1.317	[0.684	2.538]	1.452	[0.756	2.788]	1.102	[0.813	1.494]
Maternal HS Diploma (1=no)	1.317	[0.618	2.806]	1.029	[0.487	2.178]	0.782	[0.541	1.130]

***statistically significant predictor (at the p ≤ 0.05 level)**

****The complementary category refers to the reference category (0).**