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Intersections between adolescent fertility and obesity—pathways and research gaps focusing on Latin America populations

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Abstract

Latin America has notably elevated rates of adolescent fertility and obesity in women. Although numerous studies document associations between adolescent fertility and obesity across the life course, the pathways explaining their association are insufficiently theorized, especially regarding the factors in Latin America that may underpin both. Additionally, much of the existing research is from high income countries where fertility and obesity are trending down. In this paper, we review various complex pathways linking adolescent fertility and obesity, highlighting research gaps and priorities, with a particular focus on Latin American populations. We carefully consider pregnancy's distinct impact on growth trajectories during the critical period of adolescence, as well as the cumulative effect that adolescent fertility may have over the life course. We also articulate a pathway through obesity as it may contribute to early puberty and thus, to adolescent fertility. If obesity is a cause of adolescent fertility, not a result of it, or if it is a mediator of early-life exposures to adulthood obesity, these are critical distinctions for policy aiming to prevent

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both obesity and early fertility. Research to better understand these pathways is essential for prevention efforts against obesity and undesired adolescent fertility in Latin America.

Keywords

Adolescence; life course epidemiology; obesity; maternal and child health; global health

INTRODUCTION

High levels of adolescent fertility, typically defined as any pregnancy-related experience before age 20, including live birth, abortion, stillbirth, or miscariage,[1] is still a public health concern worldwide. Some middle-income settings, such as Latin America, appear resistant to the reductions in adolescent fertility associated with economic development elsewhere, with rates remaining relatively stable despite declines in other age groups.[2] For instance, while the fertility rates among those aged 25–29 years in Latin America dropped almost by half, from 198.5 births per 1,000 women in 1980–1985, to 101.9 in 2015–2020, a slower downward trend was observed among those aged 15–19 years, from 89.5 to 63.0 in the same period[3]. Data show that Latin American countries have presented the slowest decline in adolescent fertility rates for those aged 15–19 years compared to all regions of the world and it is the only region with an increasing trend in fertility among those younger than 15 years.[2] Indeed, an estimated 15% of all pregnancies in Latin America occur among girls 19 years old or younger.[2] These statistics are possibly a consequence of the high social and gender inequality found in these settings that interplay with known drivers of adolescent fertility at individual, relational, community, and societal level.[2] These include: lack of knowledge about sexuality and reproduction, early or forced sexual initiation and union, lack of supportive and empowering cultural and gender norms and values, tolerance for or practice of sexual violence, social norms and policies that do not acknowledge adolescents' sexuality and need for sexual health education and reproductive health services, including contraceptives, limited educational and employment opportunities for young people/girls, unequal gender norms and values, and tolerance and acceptance of gender-based violence.[2]

Obesity has also become a major health challenge in Latin America. While it once was a problem for wealthy populations and concentrated in urban areas, data show that these trends are shifting.[4,5] In some Latin American countries, obesity rates are increasing among low-income settings and in rural communities, creating further health disparities among these populations.[5] For instance, in Argentina, Venezuela, and Mexico, obesity rates are concentrated among low wealth and education groups.[5] Furthermore, although obesity rates are still higher among urban populations, most Latin American countries present larger increases in obesity among rural settings.[5] The prevalence of overweight and obesity among children and adolescents in Latin America is also remarkably high (around 20–25%) and has statistically increased over time;[6] although, this increase has been identified mainly among females.[7]

Numerous studies associate adolescent fertility with obesity at different stages in a woman's life course.[8–10] Among youth, there is evidence that pregnant adolescents gain and retain

more weight during pregnancy than adults,[11] and that they develop more central adiposity that,[12] in turn, leads to deleterious effects on long-term weight, contributing to obesity in adulthood.[11] Among middle-aged women, researchers have reported associations between higher obesity prevalence among those who gave birth during adolescence,[8] and these findings have been replicated among cohorts of older women.[9] Overall, a notable body of research suggests that adolescent fertility contributes to a greater weight gain over the life course than adult fertility;[13] although, less is known about how and why these associations exist. Alternatively, there is also research, mostly from high-income countries, associating obesity in childhood to adolescent fertility due to early initiation of puberty.[14] Therefore, it is possible that adolescent fertility is a consequence, not a cause of obesity.

The pathways explaining the association between adolescent fertility and obesity are insufficiently theorized, especially with regard to the factors in Latin America that may underpin both. Here, we present several pathways to explain how giving birth in adolescence may be related to obesity. We frame these pathways using adaptations of two well-accepted theoretical frameworks in life course epidemiology: the critical period approach and the cumulative risk model.[15] The critical period approach posits that events occurring during salient periods of development may permanently alter health trajectories.[15,16] The cumulative risk model implies that exposures accumulate over the life course and this accumulation is increasingly important over time. These frameworks are not mutually exclusive and cumulative social, environmental, and/or behavioral exposures may alter the risk of disease *in combination* with any critical period events. Some early life exposures are known risk factors for obesity in adulthood and adolescent fertility may be in this pathway. A careful examination of these pathways provides theoretical clarity, identifies research gaps, and offers hypotheses-testing opportunities for future research.

Pathway #1: Critical Period Approach Factors

Adolescence is a critical period and second only to fetal and infant life with regard to the rapidity of growth and pervasiveness of change across body systems.[14,16] Puberty results in very rapid somatic growth, brain development, sexual maturation of multiple organ systems, major central nervous system changes, and dramatic psychological changes.[16] Because childbearing can act as a medical stress test for women through dramatic alterations in physiology and metabolism,[17] it may permanently alter biochemical pathways during the critical period of adolescence. These alterations may affect weight gain and growth trajectories, which may predispose adolescents to obesity as described below. Figure 1 presents a visual representation of the hypothesized causal model under this theoretical framework.

Adolescent pregnancy may affect weight gain trajectories and favor weight gain and retention—Longitudinal studies have established the significant potential for growth after menarche, both in terms of height and the accumulation of fat.[12,18–20] Thus, excessive weight gains during adolescence, such as those associated with pregnancy, could exacerbate normal maturation-related processes of fat deposition.[12]

It is hypothesized that, because they are still growing, weight-gain trajectories differ for pregnant adolescents compared to pregnant adult women.[21] While there is evidence from the United States (US) that the shape of the weight gain curve for adolescents is similar to adults, the median gain and rates of gain for adolescents are higher than adults.[12] Further, differently from what is observed among adults, adolescents seem to continue accumulating fat rather than mobilizing fat stores after 28 weeks of gestation; this is despite similar average daily energy intakes and percent contributions of protein, carbohydrates, and dietary fat to total intake.[12,22] Finally, in the postpartum period, adolescents retained significantly more weight than adult controls.[12] This may be a consequence of the earlier exposure of biologically immature organs to a high dose of estrogen from the pregnancy that induces subtle, deleterious changes in glucose metabolism,[23] contributing to persistent insulin resistance and weight gain. Thus, adolescent fertility may cause unique lifelong health risks and/or it may accentuate otherwise subtle health risks associated with pregnancy and childbirth (e.g. effect modification).

Adolescent pregnancy may limit maternal growth and impact fat distribution —Existing research is conflicting as to whether or not adolescents continue linear growth during pregnancy or the pregnancy limits linear growth.[24] Fetal competition for nutrients is a factor in growth retardation or interruption in adolescents.[25] This may prevent adolescent mothers from achieving their expected height and favor an increased body mass index (BMI).[26] Moreover, the adolescent's age may also determine whether or not her body invests in growth. The pubertal growth spurt among healthy non-pregnant female adolescents normally begins at 9–10 years old and lasts around 2.5 years, but there is a considerable variation among individuals and contexts[27]. This suggests that pregnancy during the first years of adolescence may interfere more with linear growth compared to late adolescence. Additionally, there are data to suggest that the physiology of younger adolescents invests in growth while for older adolescents, their bodies privilege reproductively valuable reproductive tissue.[24] However, evidence is still conflicting[28,29] and studies targeting Latin American teenagers are particularly absent. Further studies are needed to understand this mechanism.

The higher incidence of adolescent fertility among Latin American populations may be one of the many contributors to the shorter stature and greater BMI that they achieve during adolescence compared to populations from Europe or North America, even when presenting about the same height at age 5 years.[30] Similarly, it has been described that median body weight among adolescents is low in many low-to-middle income countries (LMIC), and that the observed high BMI may be a consequence, at least in part, of stunting and suboptimal linear growth.[26] However, studies are needed to investigate the impact of adolescent fertility on linear growth among the Latin American population.

Pathway #2: Cumulative Risk Factors from Adolescent Fertility leading to Obesity

Cumulative adversity, the occurrence of multiple, cascading adverse events across a lifetime, may be initiated by early childbirth. Different aspects associated with adolescent pregnancy and childbirth may act as cumulative factors and impact health over the life

course. Socioeconomic disadvantages and behaviors following adolescent pregnancy can accumulate over the years and lead to obesity as described below (Figure 2).

Adolescent childbirth may initiate a sequence of socioeconomic adversities that contribute to obesity—Adolescent childbirth can cause youths to drop out of school,[31] earn less over their lifetimes,[32] and experience stress.[33] These can be particularly important when adolescent fertility is associated with economic vulnerability and in cultural contexts that discourage girls from returning to school after giving birth.[31] A systematic review of educational attainment and obesity across the globe indicates that in high (e.g., the United States.) and upper-middle income countries (e.g. Brazil), education is inversely associated with obesity, and the association is stronger for women than men.[34] Greater educational attainment may be associated with lower levels of obesity, because of higher health literacy and healthier behaviors, as well as a greater sense of control and empowerment.[35,36] Higher levels of education are also associated with higher lifetime earnings and higher status jobs; in developed countries like the US, both higher incomes and higher status occupations are associated with lower obesity prevalence.[37]

The increased stress experienced by adolescent mothers because of social adversities may also contribute to obesity. Chronic stress has been associated with 6-month longitudinal weight gain in US adults.[38] Additionally, British adolescents reporting higher stress levels had higher overall adiposity than their peers reporting lower stress when followed over 5 years.[35] Interestingly, the study of adolescents did not observe differences in the rates of adiposity by stress levels during adolescence and hypothesized that early life stress may set adiposity trajectories before adolescence.[35] Studies investigating the long-term impact of adolescent fertility on social adversities and stress are needed to better understand these pathways in the Latin American context and provide insights for prevention efforts.

Multiparity may be in the pathway between adolescent fertility and obesity —It is possible that adolescent fertility leads to obesity because of its association with multiparity. Because they start childbearing at younger ages, adolescent mothers tend to have more children during their lifetimes.[8] As women have more children, their likelihood of post-pregnancy weight retention increases,[39] which puts women on a trajectory of obesity throughout the life course. Nevertheless, the association between multiparity and obesity seems to be dependent on the inter-pregnancy intervals. Multiparous women with short inter-pregnancy intervals have a higher risk of obesity after childbirth compared to multiparous women with longer inter-pregnancy intervals.[40]

A systematic review of studies conducted in LMIC, including Latin America,[41] reported significant associations between a short birth interval and a younger age of the mother. Moreover, current prevailing social norms in Latin America associate a female's status with fertility which indicates that a proportion of adolescent pregnancies are wanted.[42] This could potentially result in higher adolescent fertility and multiparity rates as prevailing social norms may influence adolescents' decisions in starting families earlier and emphasize motherhood as a desired status. Accordingly, a pilot study on adolescent pregnancy in Brazil determined that over 56% of adolescent pregnancies were planned.[43] Furthermore, socioeconomic factors such as education level, poverty status, and rural location may

result in adolescents wanting and planning their pregnancies due to lack of alternative opportunities to motherhood.[44] Finally, the socioeconomic adversities following early pregnancy described in the previous topic may be even more evident when adolescent fertility is followed by multiple childbirths over the life course.

Reduction in physical activity during and following pregnancy may contribute to weight gain and retention—Physical activity is a component of health and is known to be associated with fewer morbidities and improved quality of life.[45] On a population level, women are less physically active than men and it is well-established that women decrease physical activity even more during pregnancy.[46,47] Although we do not have data for pregnant adolescents, research from a study conducted in 26 Latin American and Caribbean countries found that, among 11–18 year-olds, only 15% meet the physical activity recommendations.[48] The same study also found that in 18 of the studied countries, girls are more inactive and accumulate more sedentary behavior compared to boys. [48] Also, there is evidence from other regions that minorities, such as Black girls, are less likely to be active, compared to whites,[49] and pregnancy is one of the factors leading to that outcome.

Despite many positive effects of physical activity during pregnancy,[50] many women give up regular exercise entirely when they become pregnant and do not resume it soon after birth, with evidence that some women only return to exercise up to four years after birth.[51] Similar to what is observed among adults, pregnancy during adolescence also leads to a reduction in physical activity and an increase in sedentary behaviors, such as TV viewing.[52]

Besides all the negative effects of physical inactivity, especially during pregnancy and puerperium, one that is easily observed is weight gain, as active women are more likely to present healthy gains throughout pregnancy compared to those who are inactive.[53] Weight gained during pregnancy is not only a major problem because it increases the chances of acute conditions such as gestational diabetes, eclampsia, and macrosomia,[54] but also excessive weight gains combined with physical inactivity make it harder for some women return to their pre-pregnancy weight. Weight retention following pregnancies is one of the main causes for adult obesity among women.[55]

Food insecurity and poor diet associated with adolescent fertility may contribute to obesity later in life—Pregnancy during adolescence might include a greater demand on nutrient requirements considering the growth of the mother-child dyad. In a life course approach, nutrition in adolescence and young adulthood is important for lifelong health with benefits to human capital, nutrition, and health in the next generation.[13] In Latin America countries, adolescent girls are poorly nourished, with a disbalance in terms of both macro- and micronutrients in their diet.[56] Diets rich in sugar and fat, insufficient vitamin and mineral density and poor bioavailability, and increased body requirements due to growth or infections are frequently observed.[56] Food insecurity is also highly prevalent in Latin America, with rates of moderate or severe food insecurity presenting an upward trend in the past few years (22.6% in 2014 to 31.7% in 2019).[57] Additionally, food insecurity has been associated with obesity among adults; women are more likely to be obese and to have food insecurity.[58] In a systematic review including 26 studies, food insecurity increased the risk of pre-pregnancy obesity and weight gain.[59] Inadequate

and excessive weight gain during pregnancy were also associated with food insecurity in the meta-analysis.[59] The authors found that social inequities, such as representing a racial minority, participation in social programs and low education level have increased food insecurity in pregnant women.[59] Additionally, in a single-center observational study,[60] although adolescent pregnancy was not considered, food insecurity was more likely among younger adult pregnant women and associated with additional social determinants related to adolescent pregnancy, such as being unmarried, unemployed, having less prenatal visits, and initiating prenatal care after the first trimester.[60]

The stress associated with adolescent fertility may also influence diet profile. Stress influences eating behaviors and food choices and may disrupt certain hormonal responses that regulate appetite and weight.[61,62] Chronic stress promotes the seeking and intake of high-fat and energy-dense foods.[61,63] It may also affect the secretion of cortisol and ghrelin, hormones associated with weight gain and food cravings, respectively.[38]

Pathway #3: Cumulative risk factors from childhood leading to adolescent fertility and obesity

Adolescent fertility may be a consequence of early life exposures, including disadvantageous economic conditions and obesity during childhood. These factors are also known risk factors for adulthood obesity. Adolescent fertility consequences may be cumulative with early life risk factors and lead to obesity (Figure 3).

Childhood obesity may contribute to early pregnancy through early menarche.—Research, mostly from high-income countries, demonstrates that obesity in childhood contributes to early puberty in girls.[14] Research from Latin America also found cross-sectional associations between earlier ages at menarche and higher BMI.[64] Obesity is one of the numerous factors that may influence the onset and timing of puberty among girls by affecting their hormonal profile. It has been reported that obesity during childhood may alter secretion and sensitivity of hormones and accumulated adipose tissue may therefore contribute to the orchestrated controls for pubertal development.[65] Early age at menarche, in turn, increases the time in which a girl can become pregnant, which explains the association between early menarche and adolescent fertility reported by previous research.[66,67] Thus, the association between adolescent fertility and obesity may have this opposite pathway, with obesity contributing to adolescent fertility. Nevertheless, studies investigating these pathways are all but absent in the literature.

Adolescent pregnancy may increase the effect of childhood nutrition and childhood obesity on adulthood obesity—It has been reported that obese children are at higher risk of being obese as adults and adolescent fertility may increase this effect.[68] Previous research has demonstrated that being overweight or obese before pregnancy is a strong predictor of excessive gestational weight gain and weight retention among adolescents.[69,70] Pre-pregnancy BMI is also related to BMI increase as these adolescents become adult women.[69] Thus, overweight or obese children, when entering adolescence with excessive weight and becoming adolescent mothers, are at higher risk of gaining and retaining more weight from pregnancy and becoming obese adults. Additionally, the

effect of pre-pregnancy BMI on long-term weight retention seems to be particularly harmful among younger mothers, ages 12–17 compared to those 18–19 years.[69]

Nutritional stunting, which is an indicator of chronic undernutrition and especially prevalent in LMIC,[71] may also promote later life obesity.[72] For example, in Brazil, one study from the 1990s in São Paulo provided mechanistic evidence that nutritionally stunted children ages 7–11 had impaired fat oxidation compared to non-stunted children.[73] The fetal competition for nutrients during pregnancy among the nutritionally stunted youngest adolescents may increase these effects. When fat is not oxidized, it must be stored, which is one mechanism by which an adverse childhood nutritional environment may contribute to lifetime obesity.

Children from impoverished backgrounds are more likely to be in poor nutritional status,[57,74] and they are also more likely to experience an early pregnancy.[75,76] Pregnancy then might accentuate health issues and health may deteriorate more rapidly over time than would be expected based on either risk alone. Although there are increased trends of pregnancy among younger adolescents,[2,77] as well as childhood obesity and malnutrition in Latin America,[6] the interaction between them has not been the focus of previous research.

Socioeconomic adversities from childhood may interact with adolescent fertility and lead to obesity—Pregnancy during adolescence may be one event in a cascade of cumulative socioeconomic adversities that women with disadvantageous childhoods face.[32] Cumulative socioeconomic adversities are a contributor to chronic stress, which, in turn, contributes to increased weight gain and visceral adiposity and obesity in adulthood.[78,79] Sexual and physical abuse are additional adverse childhood experiences that may contribute to obesity and adolescent fertility in Latin America. Research from nine Latin American countries show that approximately 58% of children experienced physical, sexual, or emotional abuse in the past twelve months.[80] Sexual violence produces serious health consequences for adolescents including increasing rates of adolescent pregnancy, encouraging unsafe abortion tactics, and generating high-risk pregnancies.[81] Studies have also shown that childhood sexual and physical abuse are positively associated with obesity in adulthood.[82] Therefore, there may also be an interaction effect between early childhood adversity and adolescent childbearing. Research specifically examining if adolescent childbirth is a mediator on the pathway between adversity in childhood and obesity is notably absent.

Pathway #4: Cumulative adversities and intergenerational consequences

There is evidence of the intergenerational transmission of adolescent fertility with adolescent mothers bearing future adolescent mothers.[83] Many of the factors described above, especially social influences on diet and physical activity, may contribute to the intergenerational transmission of obesity among adolescent mothers. It is also reported that parental obesity influences up to two generations of the offspring.[84] Disadvantageous early-life conditions are consistently related to poorer later-life health.[85,86] Factors that contribute to lifetime obesity begin at preconception and during fetal development. The well-known “developmental origins of health and disease” hypothesis posits that there are windows of opportunity to promote health or alternatively, increase disease risk.[86] Maternal

diet and nutritional status during pregnancy are well-documented to influence fetal growth.[87] For example, one study in Brazil reported that pre-pregnant individuals consuming a highly-processed diet, based on refined grains, high-fat foods, and low in fiber, were at increased risk of delivering a small-for-gestational age baby.[88] Similar findings have been reported elsewhere and in controlled trials testing nutritional supplements.[86] Critically, indicators of inadequate fetal growth, such as small-for-gestational age, low birth weight, etc. have been associated in numerous studies with lifetime obesity.[89] What is less clear is whether these indicators are associated with childhood obesity, especially during critical periods such as the first year of life and the prepubertal period, when there is rapid adipose tissue deposition.[90] It is plausible, indeed likely, that maternal nutrition and behaviors during pregnancy contribute to childhood obesity, which if associated with pubertal timing, may form a chain of causal factors that ultimately increase the risk of adolescent fertility. Figure 4 shows the depiction of this hypothesized causal model.

CONCLUSION

This paper presented hypotheses that may explain the interactions between adolescent fertility and obesity, with a special focus on Latin American populations. Given the complexity of this issue, it is critical to clearly articulate evidence, pathways, suppositions, and extrapolations from the evidence. Our goal is to better direct future research and interventions by improving the theoretical underpinnings of investigations and providing hypotheses-testing opportunities on this subject. It is unlikely that there is a single causal pathway and likely, multiple pathways contribute to obesity as it relates to adolescent fertility. For example, adverse childhood nutrition, economic, and social factors may contribute to earlier pubertal timing, which in a context where fertility, especially among poorer Latin American women, is highly socially valued, promotes adolescent fertility. Adolescent fertility, in turn, negatively affects nutritional, physical activity, and other health behaviors that protect against overweight and obesity status across the life course. The present state of the evidence suggests multiple causal pathways but is still too nascent to provide “evidence-based” recommendations to health professionals and/or policy-makers. Research focused on these issues must consider the complexity among the interactions between adolescent fertility and obesity, as well as context-specific aspects that may contribute to both conditions, so that effective health policies can be developed.

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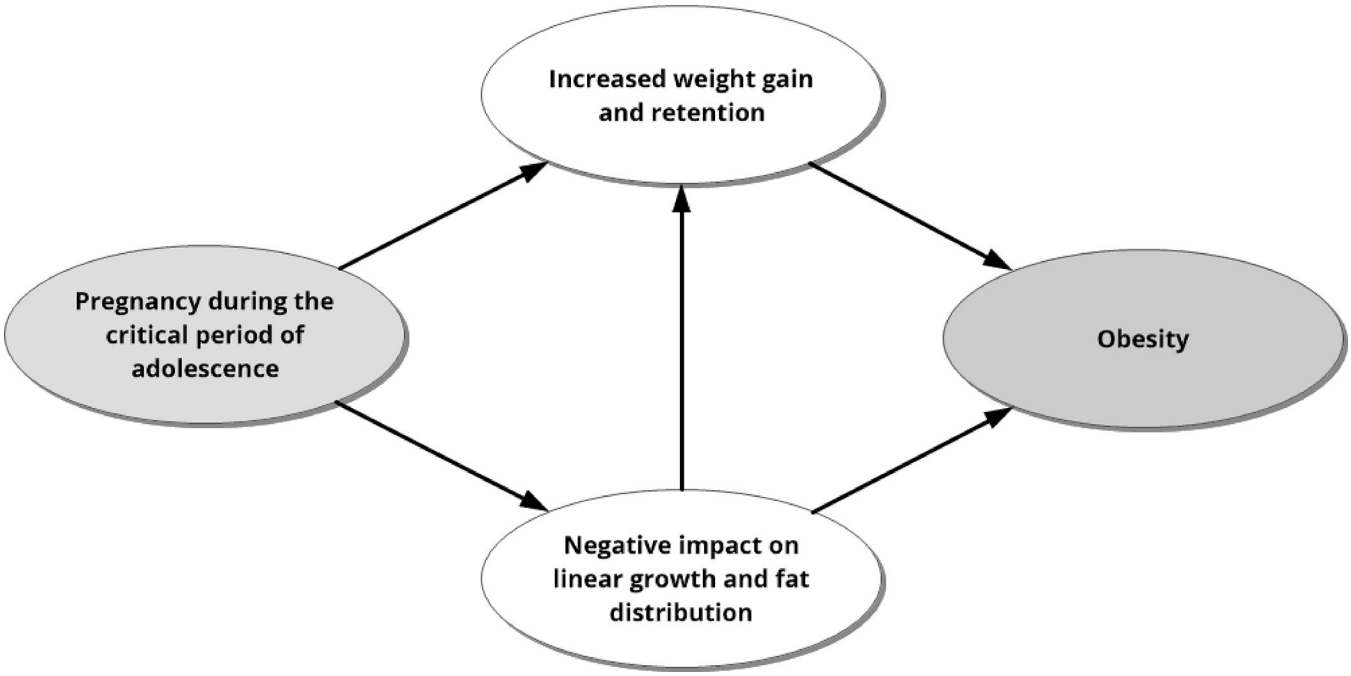

Figure 1.

Adolescence is a critical period in growth and development; pregnancy during this period may alter growth patterns and predispose a person to obesity. Critical Period Approach factors, such as alterations in physiology and metabolism during adolescent pregnancy, may explain the connection between pregnancy during adolescence and obesity.

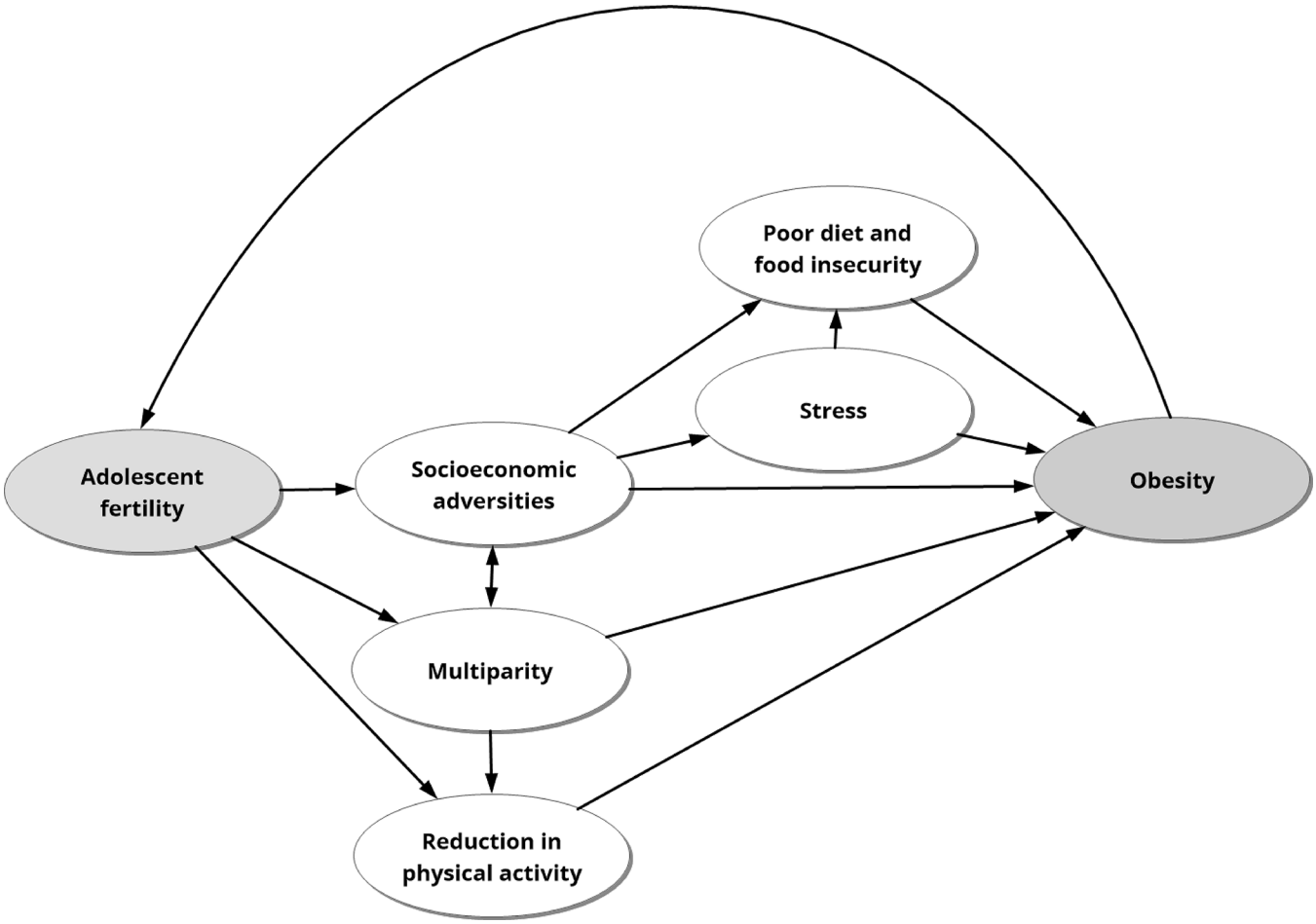

Figure 2.
Adolescent fertility may result in adverse life events, such as reduction in physical activity and food insecurity, that accumulate over time and increase the risk for obesity later in life.

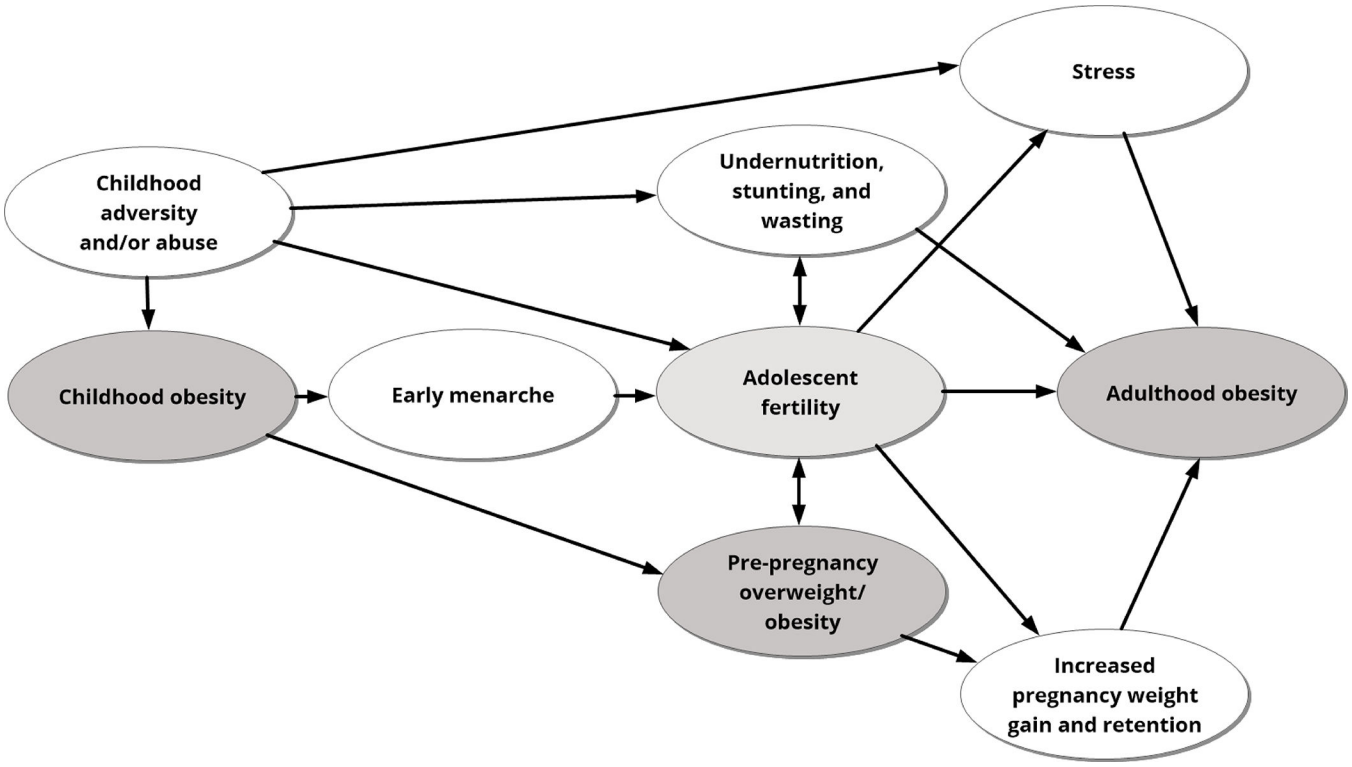

Figure 3.
Risk factors for obesity may start in childhood and accumulate over time, which can produce multiple pathways that set a person on a trajectory toward adolescent fertility and obesity in adulthood.

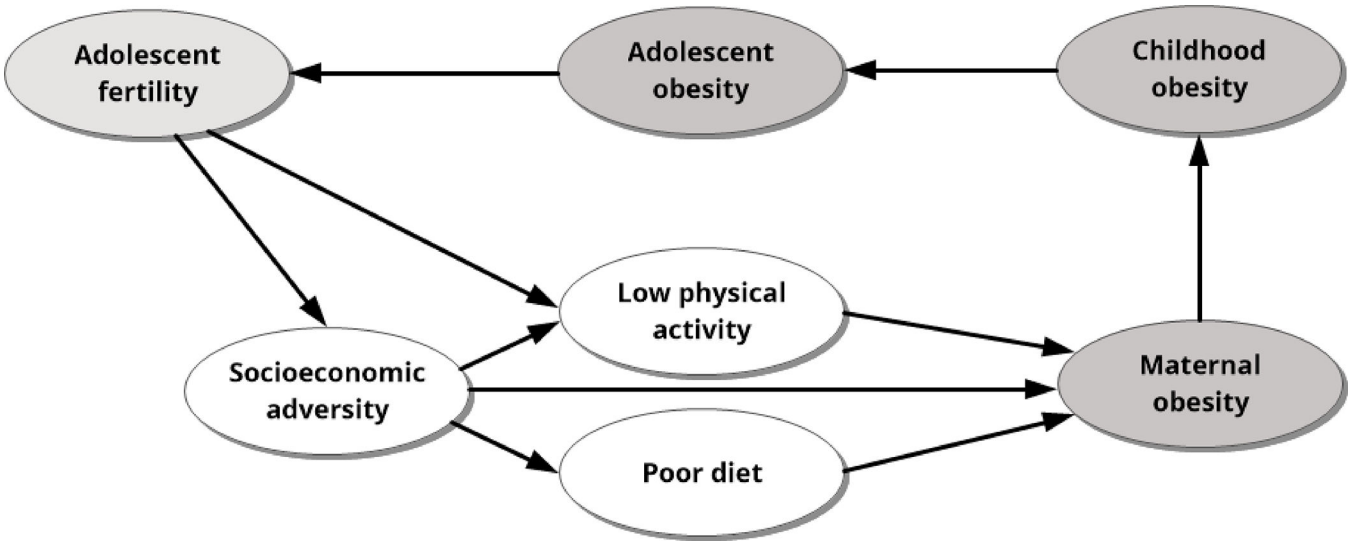

Figure 4.
Adolescent fertility may catalyze adversities which accumulate and can result in intergenerational cycles of obesity and adolescent fertility.