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RACE AND ETHNICITY DISPARITIES (M ALBERT, SECTION EDITOR)

Race/Ethnicity, Obesity, and Related Cardio-Metabolic Risk Factors: A Life-Course Perspective

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Abstract The adoption of health behaviors characterized by minimal energy expenditure and overconsumption of energy has led to cardiometabolic risk factors in pregnancy, childhood, and youth, all of which increase the prevalence of cardiovascular disease in adulthood. The propensity to develop abdominal obesity and cardiometabolic risk factors appears to disproportionally affect non-white ethnic groups. While the majority of observational research has been conducted in populations of European origin, studies in non-white ethnic groups across the life-course are underway and there is evidence that unique ethnic-specific differences exist. This review will focus on the life-course determinants of obesity and its related cardio-metabolic risk factors among diverse ethnic groups including people of Afro-Caribbean origin, South Asian, East Asian, and indigenous ancestry.

Keywords Obesity · Ethnicity · Cardiometabolic risk factors

Introduction

Globally, the most common causes of mortality are noncommunicable diseases (NCDs) of which the most prevalent is cardiovascular disease (CVD) [1]. Identifiable risk factors for CVD include dietary factors, sedentary behaviors, low physical activity, psychosocial stress, hypertension, tobacco use, abnormal lipids, glucose intolerance, and obesity [2]. Emerging as an epidemic in recent years, obesity has hastened the progress of CVD prevention efforts [3, 4]. The etiology of obesity across the lifespan is complex. Differences in the prevalence of obesity [5], its related cardio-metabolic risk factors, and CVD between ethnic groups [6] has uncovered unique ethno-cultural differences associated with the etiology, type, and consequences of obesity. With globalization, adoption of a westernized lifestyle, namely one characterized by minimal energy expenditure and overconsumption of energy has led to higher prepregnancy body weight, greater gestational weight gain, more overweight and obese children, and more young adults with cardio-metabolic risk factors. The distribution of this excess adiposity and adverse metabolic consequences appears to disproportionally affect non-white ethnic groups. This review will focus on the life-course determinants of obesity and related cardio-metabolic risk factors among diverse ethnic groups including Afro-Caribbean, South Asians, East Asians, and indigenous people of North America.

The *life course perspective* refers to the interaction of biological factors and environmental exposures during different stages of life (ie, prepregnancy, gestation, infancy, adolescence, and adulthood) and the impact on health outcomes [7]. A life course perspective of obesity is established by evidence that exposures as early as pregnancy/in utero and early childhood impacts the development of obesity later in life [8] (Fig. 1). While the majority of observational research has been conducted in populations of European origin, studies in non-white ethnic groups have been developed, and there is emerging evidence that unique ethnic-specific differences exist in maternal health behaviors [9], prevalence of gestational diabetes [10], birth weight [10], postnatal growth [9], and propensity to develop overweight/obesity and associated metabolic risk factors [11] (Table 1). This review will focus on the key sensitive periods over the lifespan, and within each important risk factors and health behaviors will be explored, including pregnancy/in

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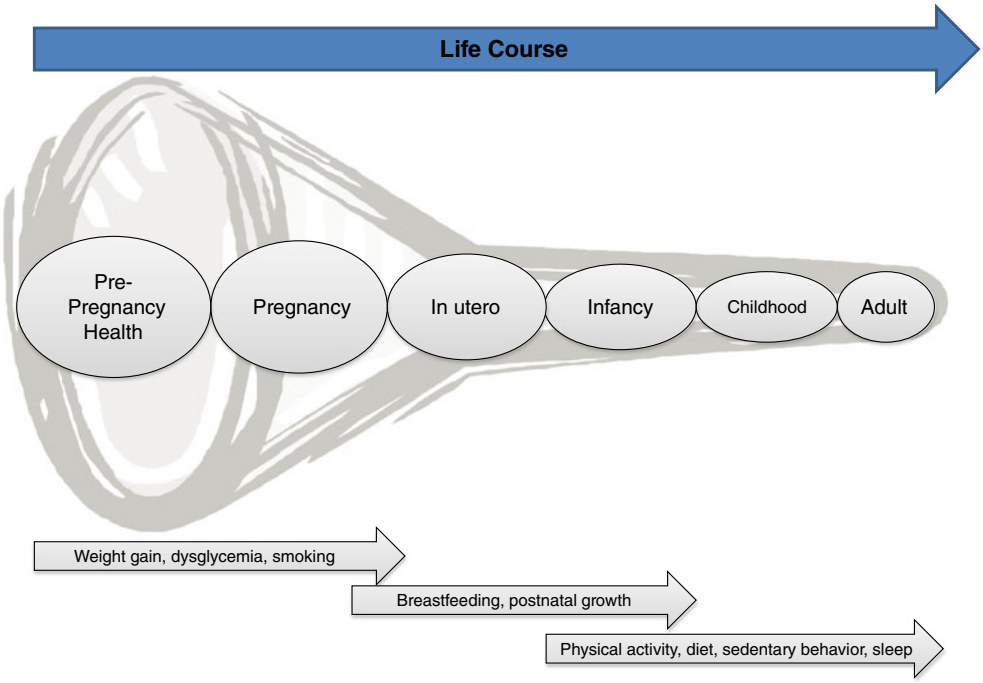

Fig. 1 A life-course approach to obesity: key health behaviors and risk factors

utero (maternal obesity and gestational weight gain [GWG]), infancy (rapid weight gain and breastfeeding), and childhood/adolescence (diet, physical activity, and sedentary behavior) into adulthood. In all ethnic comparisons when risk factors are compared with a reference population, most commonly white Caucasians, it is important to (1) consider the method of sampling with population-based studies of large size producing the most reliable results, and (2) take into account the socioeconomic differences between the groups which may suggest an apparent ethnic difference. Failure to consider the latter may lead to erroneous conclusions that ethnic differences exist when in fact they are due to a socioeconomic imbalance [9, 24].

The following sections will review the evidence of overweight, obesity, and associated cardio-metabolic risk factors and their associated health behaviors at different stages of the life-course for 4 ethnic groups—African-origin, South Asian, indigenous peoples, and East Asian.

Table 1 Overweight, obesity, and related early life determinants from diverse ethnic groups in North America

Factor	White Caucasian	South Asian	African Origin	East Asian	Indigenous
Maternal pre-pregnancy BMI >30 kg/m^2 (obese)	14.9 % [12]	U/A	26.1 % [12]	Asian/PI: 8.8 % [12]	27.7 % [13]
Gestational weight gain exceeded IOM recommendations	47.3 % [12]	U/A	52.1 % [12]	Asian/PI: 40 % [12]	52 % [14]
Prevalence of GDM	3.4 % [12]	Canada: 10 % [15]	3.4 % [12]	Asian/PI: 9 % [12]	8.1 % to 11.7 % [16–18]
Prevalence of maternal smoking	18.5 % [19]	U/A	10.1 % [19]	Asian/PI: 5.4 % [19]	Alaska Native: 36.3 % American Indians: 20.6 % [19]
Birth weight (@40 weeks)	Canada: 3030 g [10] US: 3326 g [12]	Canada: 2863 g [10]	Canada: 2900 g [10] US: 3120 g [12]	Canada: 2920 g [10] US, Asian/PI: 3252 g [12]	Male: 4030 g [10] Female: 3900 g [16]
Breastfeeding @ 6 months	US: 47 % [20] Canada: 25.8 % [21]	Canada, Asian: 30.2 % [21]	US: 30 % [20] Canada: 27 % [21]	Canada, Asian: 30.2 % [21]	Canada 16.6 % [21]
%Overweight/obese (>85th percentile) during childhood	27.9 % [4]	Asian: 12.5 % [22]	39.1 % [4]	Asian: 12.5 % [22]	US: 48.7 % [23]

U/A unavailable

African Origin

African origin includes people who originate from Africa and/or the Caribbean islands.

Pregnancy/In Utero

Most observational data of African-origin mothers/offspring originate from the United Kingdom (UK) and the United States (US). Population-based studies indicate that Black women are more obese than White Caucasian women [25] and have higher rates of excessive GWG [12]. However, they are also more likely to have inadequate GWG compared with White Caucasian women [26]. Further, the risk of gestational diabetes among Black women has been historically higher than White Caucasian women [27, 28]. Rates of smoking among pregnant African American women are traditionally lower than White Caucasian women in the US [29]. Disparities exist for neonatal outcomes with African Americans experiencing higher rates of infant mortality and low birth weight [30]. The etiology for these observations is complex, however, biological and socioeconomic factors likely play a role [31].

Infancy

Health behaviors in early infancy, including postnatal growth and infant diet, increase the risk of obesity in those of African ancestry. Rapid weight gain during the first year of life increased the risk of being obese (BMI≥30) by age 20 years in a cohort of 300 African American infants [32]. This trajectory of growth in early infancy has been linked to breastfeeding [33]. Positive gains in rates of breastfeeding in the US between 2000 and 2008, are lower than breastfeeding rates in W.C. [20] where 59 % of African American infants ever breastfed compared with 75 % of White Caucasian infants, and 30 % of African American women breastfed until 6 months compared with 47 % White Caucasian women [20]. Reasons for these differences include continued ethnocultural and socioeconomic factors.

Childhood/Adolescence

Table 1 illustrates the rates of childhood overweight/obesity among different ethnic groups. Black children in the US suffer a high prevalence of overweight and obesity compared with other groups, with the exception of Indigenous children. A constellation of health behaviors including physical inactivity, poor diet quality, and time spent in sedentary behaviors places children at risk of developing obesity. Data to date suggests that while Black children engage in more vigorous physical activity compared with White Caucasian children, they also are more likely to display sedentary behaviors. For example, data from the US National Health and Nutrition Examination Survey (NHANES) showed that Black children spend 8 min/day greater participating in moderate-vigorous physical activity compared with White Caucasian children, however, they also spent more time (25 min/day) in sedentary activities [34]. This observation is reinforced by the Child Heart Health Study in England (CHASE Study), which compared White Caucasian, South Asian, and African-Caribbean 9- to 10-year-olds and found African-Caribbean children spent more time (4 min/day greater) in moderate-vigorous physical activity than White Caucasian children [35]. However, in this same cohort, Black children also spent more sedentary time (26 min/day more) [35]. Interventions to promote activity (ie, culturally-tailored dance) and reduce screen time among African American girls, a particularly high-risk group, have had minimal impact on BMI [36]. Adverse dietary practices among Black children include excess high caloric and sugar sweetened beverage consumption compared with White Caucasian children [37] with a rising consumption in quantity between 1988 and 2004, in the US [38]. On balance consideration of activity, inactivity, and food consumption among Black children is complex, while there are some positive health behaviors displayed by African origin youth including vigorous activity, others are less desirable and suggest that population-based studies are required to track how these behaviors change and influence obesity rates over time.

South Asian

South Asian includes people who originate from the Indian subcontinent, including India, Pakistan, Bangladesh, and Sri Lanka.

Pregnancy/In Utero

South Asian adults have a higher burden of cardiovascular disease-related risk factors for the same BMI compared with White Caucasians [11]. This is explained by the higher percent body fat (ie, adiposity per unit BMI) and South Asians propensity to develop abdominal fat. A birth cohort from rural villages in India compared the anthropometric characteristics mothers and babies of European origin recruited from Southampton, UK [39]. This comparison revealed that South Asian mothers had a lower BMI (18 vs 23) compared with the European mothers, and South Asian babies were lighter (2.7 kg vs 3.5 kg) yet had comparable subscapular skin fold thickness, suggesting that for their weight they had relatively more adipose tissue. These South Asian babies were also found to have increased adiposity, glucose, and insulin at age 6–8 years [40]. Adding to this risk profile, is the observation that GDM is higher among South Asian women and is a predictor of infant weight gain and adiposity in the first year postpartum [41]. Predisposition for smaller weight at birth,

may lead to an inherent risk of misclassifying South Asian infants as small for gestational age (SGA) (Table 1). Prospective longitudinal studies are ongoing to understand the prepregnancy, pregnancy, and in utero determinants for the thin-fat phenotype among South Asians [42, 43].

Infancy

South Asian babies are observed as "short, underweight, but fat," which is referred to as the "thin-fat" phenotype [40]. The potential of assigning pathology to this observation may lead to unnecessary health visits and interventions when South Asian infants' weights are plotted on growth charts derived from data from White Caucasian infants. It has been shown, when plotted on ethnicity-based birth weight growth centiles, 29–46 per 1000 South Asian newborns from Ontario, Canada were misclassified as SGA [44]. In light of this observation the early postnatal life is important and is modified by the observation that South Asian infants have a propensity toward a higher rate of growth compared with White Caucasian infants [45]. This compounds the risk later in life of obesity related complications. A protective health behavior is demonstrated by South Asian mothers being more likely to breastfeed compared with Caucasian women [46, 47]. This may impact on the risk of developing of obesity and related complications later in life.

Childhood/Adolescence

Physical activity, diet, and sedentary behavior during childhood all impact the risk of developing obesity among those of South Asian ancestry. Data among South Asian children are limited from North America. In the UK, school-aged, South Asian children have lower levels of physical activity compared with White Caucasian children [35] and have demonstrated higher caloric and total fat intake [48] and increased adiposity, compared with White Caucasian children [49]. Reducing sedentary behaviors such as screen time has been associated with improving obesity [50]. South Asian children in the UK have been observed to participate in high, (ie, greater than 4 h per day) sedentary activities which is similar to White Caucasian children [51]. Similar trends of low levels of physical activity exist among South Asian adults in North America [52, 53]. Cultural attitudes promoting sedentary activities rather that sports-related leisure activities are thought to play a role in this health behavior pattern [53].

Indigenous Peoples of North America

Indigenous peoples of North America include those who originate from American Indian, Alaska Native, First Nations, Metis, or Inuit ancestry.

Pregnancy/In Utero

The presence of multiple risk factors including higher gestational weight gain, higher gestational dysglycemia, and smoking undoubtedly influence obesity and its related risk factors among Indigenous peoples. Evidence suggests the average pre-pregnancy BMI is significantly higher among Indigenous women compared with non-Indigenous women, in the US and Canada [13, 54]. Further, higher gestational weight gain among Indigenous mothers has been significantly associated with obesity during childhood years [14]. The effect of dysglycemia during pregnancy is particularly relevant to Indigenous women for whom the prevalence of gestational diabetes is high [55]. In Canada previous studies have estimated the rate of gestational diabetes in Indigenous people ranges from 8.1 % to 11.7 % [16–18] compared with 3.0 % to 4.8 % in non-Indigenous people [17, 18]. Furthermore, data from the Pima Indians in the US show that the effects of dysglycemia during pregnancy is associated with obesity later in life [56]. The prevalence of smoking during pregnancy among Indigenous women is substantially higher than among non-Indigenous women (Table 1) [19, 57]. While one may expect that higher smoking in pregnancy will result in more SGA newborns, this has not been observed in Indigenous populations. The presence of smoking in pregnancy with higher birth weight newborns increases the risk of childhood obesity and insulin resistance (OR=14.0, 95 % CI 3.8, 51.1) [58].

Infancy

Infants of Indigenous ancestry have a high risk for obesity based on their early life weight trajectory and nutritional exposures. Indigenous children are more likely to be born large for gestational age [16], have rapid postnatal growth [59], and those who have high BMI at age 1 year are 3 times more likely to be obese during childhood [14]. Each of these factors independently increases the risk of obesity later in life. Nutritional exposures during infancy also place infants at risk. Breastfeeding rates among Indigenous populations remain low in the United States (at 6 months 17 %) [59] and Canada (at 6 months 27 % [47, 60]) with cultural factors contributing to this disparity [61]. Further, among Indigenous children in the United States the absence breastfeeding imposes an increased risk of obesity by age 8 years [14].

Childhood/Adolescence

Although cultural heterogeneity exists among Indigenous peoples in North America there have been repeated observations from various tribes and reservations in North America documenting the high prevalence of risk factors for obesity. Among a cohort Pima Indian children in the US higher BMI

has been associated with 2 times higher rates of premature death [62]. Children of Indigenous ancestry in Canada living on-Reserves have lower rates of physical activity participation than Indigenous children living off-Reserves [63]. Similar findings have been observed among populations in the US [64]. Furthermore, the risk of obesity associated with a high consumption of sugary beverages [65] has also been seen among Indigenous children in rural US [66]. Indigenous youth in the US tend watch more television, a form of sedentary behavior, compared with White Caucasian youth [67]. This is concerning as sedentary behaviors among Indigenous youth has been significantly correlated with risk for overweight and obesity [68]. Specific multi-component interventions tailored to Indigenous youth in the US have not been successful in reducing body weight among children aged 8 to 10 years [69].

East Asian

East Asian refers to people who originate from China, Japan, Taiwan, Vietnam, and Korea.

Pregnancy/In Utero

East Asian children whose mothers were obese during pregnancy were more likely to be overweight by age 6–7 years [70]. Further, East Asian women with higher pre-pregnancy BMI demonstrate more insulin resistance than White Caucasian women [71], and dysglycemia during pregnancy has been shown to increase the risk of cardio-metabolic risk in offspring of East Asian women [72]. The Chinese famine of 1959–1961 illustrates an adapted response to undernutrition in utero, where fetal exposure to severe famine led to an increased risk of dysglycemia in adulthood (OR=3.92; 95 % CI: 1.64, 9.39) [73]. Finally, among a cohort of Taiwanese children, exposure to cigarette smoking in utero was associated with an increased risk of childhood obesity (OR 1.4 (95 % CI: 1.1–1 1.8) [74].

Infancy

The postnatal environment is important in shaping the trajectory of health. However, postnatal weight gain and breastfeeding, show diverging associations among East Asian children. Japanese adolescents who experienced greater weight gain in the first 18 months of life were more often overweight, compared with those who had a slower velocity of growth [75]. In contrast, there is evidence that breastfeeding may not be associated with obesity at age 6 to 7 years among a group of East Asian children in Hong Kong [70]. Although both are important observations the heterogeneity of the samples being compared makes a definitive conclusion difficult.

Childhood/Adolescence

The consequences of health behaviors among East Asian children are similar to other populations. Physical inactivity among a sample of Asian children in China showed an increased risk of obesity [76]. Further it has been shown that Asian children who spent more time participating in sedentary behaviors were more likely to be obese than those who spent less time engaging in similar activities [77]. Another study of East Asian children from China, showed a strong association between the consumption of sugar-sweetened beverages and obesity [78].

Adults, Obesity, and ethnicity

Overweight and obesity is associated with hypertension, type 2 diabetes, and abnormal lipids, which are strongly associated with the development of MI and stroke and may stall the decline in CVD in high-income countries [79]. Population-based studies from North America and the UK indicate that the prevalence of overweight and obesity differs substantially between ethnic populations. For example, using similar BMI cut-offs, overweight and obesity are highest among African origin and Hispanic populations, intermediate amongst White Caucasians, and lowest among South Asians and East Asians (Table 2). However, BMI as a measure of adiposity is limited and may over or underestimate adiposity differently in different ethnic groups. Whether BMI or another metric of adiposity is used to compare ethnic populations, or to study the association with cardio-metabolic risk factors, it is important to consider the following: (1) for some populations, there is greater adiposity per same given level of BMI, (2) adipose tissue distribution is important when considering metabolic consequences of overweight/obesity, and (3) adjustment for socio-economic and sex differences is crucial. The propensity to develop abdominal adiposity is a risk factor for cardio-metabolic risk factors, diabetes, coronary artery calcification, and CVD [102]. Even among individuals with abdominal obesity who show no signs of abnormal risk factors, ie, metabolically healthy, abdominal obese (MHAO), are at higher risk of mortality compared with their non-abdominally obese metabolically healthy counterparts (mortality risk for MHAO was around 40 % higher (hazard ratio 1.43; 95 % CI: 1.00–2.04) [103]. Certain non-White populations have a higher prevalence of abdominal adiposity including people of South Asian, African origin, and Indigenous people. Some studies also suggest that East Asians also have greater abdominal and visceral adiposity compared with White Caucasians [104]. However, indirect comparisons in which detailed measures of visceral fat area using CT or MRI suggest that South Asian men and women have particularly high amounts visceral fat compared with other ethnic groups,

although more data using direct comparisons and similar measurements techniques are required. Visceral fat is strongly associated with the presence of fatty liver, which is then in turn associated with cardio-metabolic risk factors and CVD (Table 2). Sex differences in visceral adipose tissue (VAT) consistently show that men have greater VAT than women. However, more research to identify the VAT (in cm^2) thresholds above which metabolic risk factors develop in each ethnic group are required as the cut points may differ [105].

Genetics, Epigenetics and Microbiome Life-Course Studies of Obesity

Recently, genome wide association studies (GWAS) have identified multiple genetic variants associated with increased CVD risk, lipid traits, diabetes, adiposity, body weight, and hypertension, demonstrating that CVD is a complex disease with both polygenic and environmental influences [106–108]. Most GWAS have been performed in adults, and we have only started to elucidate the possible impacts of these genetic variants on metabolic factors early in life. Recently, in the ALSPAC birth cohort, a gene score made up of 8 SNPs (in/near: FTO, MC4R, TMEM18, GNPDA2, KCTD15, NEGR1, BDNF, and etV5) showed little association with birth weight per se (regression coefficient: 0.01 standard deviation score per allele; 95 % CI 0.00–0.02), but did have a large positive effect on early infancy weight gain (within first 6 weeks of life) (0.119 standard deviation scores/allele/year) but not on subsequent childhood weight gain (0.004 standard deviation scores/allele/year) [109]. These data suggest that it is possible that different genes influence a trait at different developmental stages, or that the same genes can have a differential impact on a trait as it develops. In support of this concept SNPs associated with lipid levels harbour a 2-fold higher effect size in children than in adults [110]. The situation is different for glucose and hypertension-associated SNPs, where similar effect sizes are observed in children and adults for genetic variants associated with these traits [111, 112]. These recent data reveal a complex pattern of association during childhood for SNPs modulating cardio-metabolic risk factors that require further investigation, especially in diverse ethnic populations, ideally in prospective birth cohorts.

Although studies of gene–environment interactions require even larger sample sizes than genetic studies, some early investigations are underway. For example, in 2 Greek birth cohorts, while the FTO SNP was associated with increased body weight, this effect was reduced among children who

Table 2 Overweight, obesity and adiposity comparing adults from diverse ethnic groups

	African Americans	Chinese	Hispanic/Latino	South Asian	White Caucasian
Prevalence of overweight (%) (BMI ≥25 <30)	73.7 (71.2–76.2) [5]	15.7 (0.4) [80]	77.9 (74.5–81.4) [5]	Men: 40.9 [6] Women: 45.8 [6]	66.7 (64.1–69.3) [5]
Prevalence of obesity (%) (BMI ≥30)	44.1 (40.0–48.2) [5]	10.7 (0.3) [80]	38.7 (33.5–43.9) [5]	Men: 12.9[6] Women: 16.1 [6]	32.4 (28.9–35.9) [5]
Greater body fat per given BMI compared with White Caucasians (Yes/No)	No [81]	Yes [82]	Yes [83]	Yes [82, 84]	N/A
Abdominal obesity %	Men: 34.6 [85] Women: 75.8 [85] (>102 cm in men and >88 cm in women)	Men: 44 [6] Women: 22 [6] (>90 cm for men and >80 cm for women)	Men: 37.8 [85] Women: 73.6 [85] (>102 cm for men and >88 cm for women)	Men: 64.7 [6] Women: 72.7 [6] (>90 cm for men and >80 cm for women)	Men: 45.1 [85] Women: 58.0 [85] (>102 cm for men and >88 cm for women)
Mean W/H circumference (cm) in population based studies	Men: 0.94 (0.07) [86] Women: 0.89 (0.08) [86]	Men: 0.90 (0.1) [6] Women: 0.81 (0.1) [6]	Men: 0.99 (0.06) [86] Women: 0.90 (0.07) [86]	Men: 0.93 (0.06) [6] Women: 0.84 (0.08) [6]	0.85±0.01 [87]
Mean VAT measured by MRI or CT in population based sample (cm^2)	Men: 108±63 [88] Women: 94±58 [88]	100.0 (72.7, 126.6) [82]	Men: 127±62 [88] Women: 107±59 [88]	Men: 153.5±8.8 [87] Women: 97.3±7.3 [87]	Men: 134.5±12.1 [87] Women: 95.6±6.8 [87]
Fatty liver % in population based sample	Men: 23 [89] Women: 24 [89]	Men: 36.8 [90] Women: 22.7 [90]	Men: 45 [89] Women: 45 [89]	Men: 10.8 [87]–31.1 [91] Women: 9.4 [87]–37.4 [91]	Men: 42 [89] Women: 24 [89]
VAT and/or Fatty liver linked to T2D (Yes/No)	VAT: Yes [92] Fatty liver: Yes [89, 93]	VAT: Yes [94] Fatty Liver: Yes [95]	VAT: Yes [96] Fatty Liver: Yes [89]	VAT: Yes [97, 98] Fatty liver: Yes [98]	VAT: Yes [99] Fatty liver: Yes [99]
VAT and/or fatty liver linked to heart disease	VAT: Yes [100] Fatty liver: Yes [101]	VAT: Yes [82] Fatty liver: Yes [95]	VAT: Yes [99] Fatty liver: Yes [99]	VAT: Yes [97] Fatty liver: Yes [98]	VAT: Yes [99] Fatty liver: Yes [99]

CT computed tomography, *MRI* magnetic resonance imaging, *T2D* type 2 diabetes, *VAT* visceral adipose tissue, *W/H* waist/hip

were breastfed for at least 1 month. In contrast, this interaction of FTO and breastfeeding was not replicated in the UK-based ALSPAC cohort [113]. Furthermore emerging technologies have enabled life-course studies of epigenetic patterning and infant microbiome as related to obesity [114–117]. Offspring of mothers exposed to the Dutch Famine have low birth weight and adult onset central adiposity and insulin resistance. Famine exposure is associated with widespread but modest differential DNA methylation in genes involved in growth and metabolic diseases including insulin like growth factor 2 and the insulin promoter in famine exposed offspring compared with their non-exposed siblings [118]. The recent development of chip-based technology enables the study of genome-wide epigenetic marks that is reliable and accurate, and together with detailed environmental exposures, opens up unprecedented possibilities for epigenetic research in human populations [119, 120].

Infant gut microflora composition is emerging as a host factor which may play an important role in the development of responses to food intake. Several factors shape gut microflora during infancy, including mode of nourishment delivery, type of infant feeding, hospitalization, antibiotic use, type of delivery (vaginal vs Caesarean section), and prematurity [117]. The gut microbiota is profoundly altered in humans during pregnancy [121]. This suggests that the microbiome of the pregnant mother may be associated with metabolic changes in pregnancy, and its transfer to the newborn may influence the metabolic response of the developing infant. Furthermore, recent studies suggest that nutritive and non-nutritive components of breast milk, formula, and solid foods, and feeding practices may also shape the infant microbiome, programming growth rates and body composition and shape behavioral responses to foods and eating [122–124]. "Western" diets may also play a role in promoting "obesogenic" gut microbiota, but this has not been comprehensively studied early in life [122, 125–127]. Thus genetic, epigenetic and microbiome studies across the life-course are underway and are expected to shed new light on the etiology of overweight and obesity in human populations. Such investigations in diverse ethnic groups are also needed, as in addition to adding new etiologic knowledge to high risk populations, are an efficient research strategy which capitalizes on their wide diversity in health behaviors and cultural practices.

Conclusions

To mitigate the global epidemic of obesity, a sustained effort is needed toward better understanding the increasingly complex scope of risk factors and the role they play in promoting disease. Because the etiology and related complications of obesity span multiple stages of the life-course, a robust and coordinated approach to capturing information across these stages is needed. This can be accomplished by (1) repeated observations over sensitive periods in the life course such as through birth cohorts, and (2) by observing and comparing different ethnic groups, as this has the potential to advance knowledge about the pathophysiology of obesity.

There have been many attempts to reduce the burden of obesity in adults, with few having long-term success. With the burden of obesity-related diseases being shifted earlier in life [3], the need to develop and test of interventions that impact the trajectory of obesity early in life is critical. The impact of promoting healthy lifestyles to women during pregnancy may lead to intergenerational benefits. Further *a life course approach* to obesity prevention should be multi-generational, family-focused, and consider unique ethno-cultural influences.

Compliance with ethics Guidelines

Conflict of Interest Gita Wahi declares that she has no conflict of interest. Sonia S. Anand declares that she has no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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