Obesity in China: What are the Causes?

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Abstract: To address the causes for obesity in all of China is not feasible in a single article. There are hundreds of ethnic groups over a vast number of provinces. The diet and lifestyle of each is different based upon cultural/ethnic traditions and the environment. Several studies mentioned in this review have been done in particular areas and/or on specific population groups with regards to obesity and health risks associated with being overweight. Obesity is a multifactorial disease that is associated with genetic, physiological, environmental, and cultural/traditional perspectives in order to provide a broadened view on this epidemic in China. In this review, we will assess specific obesity gene and environment interactions, childhood obesity etiology, metabolic syndrome, and dietary and behavioral causes. We attempt to discuss obesity issues particularly in the Han Chinese population ranging from children, adolescents, adults to geriatrics.

Keywords: Obesity, overweight, China, insulin resistance, hypertension, type 2 diabetes mellitus, metabolic syndrome, dyslipidemia.

INTRODUCTION

Obesity in China is becoming a pandemic disease resulting from a shift in energy balance caused by altered genes, a sedentary lifestyle, and neurohormonal imbalances. It is spreading to low-income and middle-income countries, such as China, as a result of novel dietary habits, promoting chronic diseases and premature mortality [1]. Work-related activities declined recently in industrialized countries, whereas leisure time is dominated by television/computer programs and other physically inactive pursuits [2,3]. In China, overweight rates doubled between 1991 and 2006, and the number of obese individuals tripled [4]. The prevalence of obesity in men was 10.5% in mainland China [5], and 16.3% in the Hong Kong population [6]. The prevalence of diabetes in China parallels that in the United States, with more than 92 million cases [7]. More importantly, related health care costs are also substantial [8]. The vicious obesity cycle begins with excess adipose leading to chronic low grade inflammation that results in insulin resistance (IR) along with hypertension, atherosclerosis, dyslipidemia and type 2 diabetes mellitus (T2DM), which are consistent findings of metabolic syndrome (MetS) [9]. In the past, obesity has been defined by the body mass index (BMI) [10]. BMI is the result (kg/m^2) of dividing the weight (kg) by the height squared (meters). The accepted range for BMI <18.5 is underweight, 18.5-24.9 is normal, 25.0-29.9 is overweight, and >30 is obese. Obesity consists of three other categories including Class I: BMI 30.0 - 34.9, class II: BMI 35.0 - 39.9, and class III: BMI ≥40.0. Morbid obesity is considered to be >35 [11]. However, as seen by a few of our pilot studies, BMI is not the best method for measuring obesity in China as well as other populations such as body-builders and geriatrics [12]. Therefore, to obtain a better picture of the weight status of an individual, BMI should be measured along with waist circumference (WC) and waist-hip ratio (WHR). WC of >102 cm in men and >88 cm in women and a WHR >0.90 for men and >0.85 for women indicates obesity and can be used to predict T2DM, hypertension, cardiovascular disease, gall bladder disease and specific cancers [13,14]. Certain studies on Asian populations have shown that the Chinese have more body fat at a lower BMI and WC than Western populations [15,16]. Therefore, the waist-height ratio (WHtR) index appears to be an even more reliable measurement of obesity for lower statue ethnic groups such as the Chinese [17].

Studies have shown that obesity can be linked to lower ghrelin concentrations in obese individuals [18,19,20]. Salivary ghrelin levels were lower in obese and non-obese subjects with T2DM [21]. Ghrelin levels have been found to be negatively correlated with body fat and WC [22]. Serum and saliva ghrelin levels were correlated with BMI [23]. Saliva has been used as a simple, non-invasive diagnostic tool [24]. Parotid and submandibular glands were the primary sources of ghrelin [23]. Hence, this may be a useful application in Chinese obesity clinics. In the following sections, the potential causes for obesity in China will be discussed in detail.

GENE-ENVIRONMENT INTERACTION

Gene-environment interaction is defined as "the response or the adaptation to an environmental agent, a behavior, or a change in behavior is conditional on the genotype of the individual" [25]. Mechanisms for this phenomenon consist of decreased resting metabolism and lipid oxidation rate, and poor appetite control with increased fat mass [26]. McMillen *et al.* [27] suggested that specific periods during pregnancy predisposed individuals to obesity, therefore maternal nutrition and perinatal lifestyle played a major role in fetal programming. Over-nutrition during pregnancy led to larger offspring or gestational diabetes associated with obesity, while breastfeeding could counter the effects of obesity [28]. For example, in Fujian province, the rate of obesity has increased due to poor nutrition before and during pregnancy, economic development, urbanization and improved living standards [29].

Two recent studies [30,31] identified novel genetic variants associated with obesity and/or BMI. The genetic loci were: *NEGR1*, *SEC16B-RASAL2*, *TMEM18*, *SFRS10-ETV5-DGKG*, *GNPDA2*, *NCR3-AIF1-BAT2*, *LGR4-LIN7CBDNF*, *MTCH2*, *BCDIN3D-FAIM2*, *SH2B1-ATP2A1*, *KCTD15*, *FTO* (fat mass and obesity associated) [32,33,34] and *MC4R* (melanocortin 4 receptor) [35,36]. The *FTO* gene is present in all tissues and encodes a non-heme (FeII)-dioxygenase that adapts to hypoxia, lipolysis, or DNA methylation [37,38]. This key protein may serve as a link between the central nervous system and energy homeostasis.

Several studies associated *FTO* variants (rs8050136 and rs9939609) with obesity and BMI in Hong Kong, Taiwan, and Singapore populations [32,33,34]. On the other hand, Li *et al.* [39] found no association of the *FTO* variants with obesity and BMI in Shanghai and Beijing individuals. Obesity-associated genetic vari-

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ants studied in Caucasians impacted the development of obesity in the Chinese. For all obesity-associated single-nucleotide polymorphisms (SNPs), researchers observed higher odds ratios than previously reported [30,31]. This may be caused by differences in genetic compositions between Chinese and Caucasian populations, even though these SNPs are less common in the Chinese. There was a 17-23% increased risk of obesity for every unit increase in the combined genetic risk scores. Further research needs to be done on obesity susceptibility genes for clinical applications. The estimated heritability of BMI in Chinese ranged from 0.42 to 0.54 [40,41,42]; however, 4.1% of this could be correlated with seven obesity-associated SNPs [43]. Associations with BMI and WC were observed for rs10938397, rs17782313, and rs8050136 [43]. Identification of *FTO* and variants within this gene were extensively researched in various populations [44,45,46,47]. rs8050136 and rs9939609 are located within a linkage disequilibrium block in the first intron of *FTO*. rs10938397 is located about 450 kb upstream of *GNPDA2* (glucosamine-6-phosphate deaminase 2), which is involved in carbohydrate metabolism [48]. rs17782313 is located about 190kb downstream of *MC4R;* this SNP is associated with obesity, fat mass, weight, and height [35]. rs12970134 is associated with WC and IR [36]. The relationship between obesity-associated SNPs and glycemia needs further research. rs29941 is located about 4.4kb downstream of *KCTD15* (potassium channel tetramerization domain containing 15). The exact function of *KCTD15* is currently unknown, although it has been suggested that it acts as a transcription factor and may be involved in T2DM [49,50]. One study found a relationship between *FTO* SNP rs8050136 and BMI. It showed that the combined genetic risk of SNPs may be useful in predicting obesity [43], perhaps in the Chinese population.

Furthermore, rs9939609 was associated with BMI in Taiwan [51]. rs9939609 and other SNPs coupled with obesity were found in those living in Singapore [52]. The rs9939609 A allele frequency was lower in Chinese and Japanese subjects compared to that in Europeans [53]. rs9939609 SNP was strongly associated with obesity and T2DM in Chinese adults [54]. AA/AT genotype carriers had higher obesity and BMI prevalences than the TT genotype [53]. The rs9939609 A allele frequency was lower as compared with the Europeans [53]. Only 1.2% of the Chinese were homozygous for the A allele [53]. The A allele was indeed linked to obesity in Chinese adults [54]. On the other hand, there has been conflicting information with regards to rs9939609 or rs8050136 polymorphisms of the *FTO* gene and obesity in the Chinese population [55]. One study concluded that *FTO* polymorphisms were associated with obesity but not with T2DM in China [56]. Another study demonstrated a strong association between *FTO* and *MC4R* with BMI in Chinese individuals [57]. BMI of subjects with three or four risk alleles outweighed those without the *FTO* or *MC4R* risk allele [57]. The *FTO* rs9939609 A allele was associated with obesity and BMI mostly in women [57]. *FTO* may also regulate gender-specific development of obesity and insulin resistance in all people [57]. The Chinese are thus at risk for T2DM at a lower level of obesity due to their increased predisposition to abdominal fat deposition and decreased pancreatic β-cell function [58]. The Chinese have different genetic architecture, hence these SNPs act differently in them [57]. On the other hand, variants in the *FTO* gene were not associated with obesity in Chinese subjects [39]. The reason for this inconsistency is unknown. Possible explanations include different environmental exposures or lifestyle factors between the populations [52]. Associations of the A allele with T2DM remained significant with adjustment for age, sex and BMI [54].

Finally, the adiponectin (ADIPOQ) gene in T2DM and obesity has been reported. The synonymous and intronic polymorphisms in the ADIPOQ gene were found to be associated with BMI in Japanese, Korean, Chinese and Caucasian individuals [59,60,61,62]. AC3 genetic polymorphisms decreased the risk of obesity in Chinese adults [63]. The AC3 genetic effect on BMI may interact with factors relating to ageing and the environment [63].

ETIOLOGY OF CHILDHOOD OBESITY

In China alone, the prevalence of obese children aged 2-6 years increased from 1.5% to 12.6% in urban areas from 1989 to 1997 [64]. Parents and extended family members play a crucial role in shaping their children's eating and exercise habits [65]. Approximately 22% of Chinese parents regarded their children as underweight even if their children weren't. Meanwhile, 23% of overweight children were perceived by their parents as being normal [66]. Parental assessment of the weights of their children was associated with the physical appearance of the parents themselves [67]. Overweight daughters were more likely to be criticized by their mothers [68,69]. Chinese parents tended to misperceive their sons' weights more than their daughters.' Mothers had a better ability to discriminate their children's size. This gender difference could be related to social values and status [69], hence exacerbating the obesity problem. For example, girls with slim and graceful bodies were deemed acceptable by Chinese society, while overweight boys were regarded as "strong and healthy" [68,69]. Mothers' perception of their children could be influenced by emotional factors rather than known facts about obesity and body image, which could be related to their educational level [70]. Studies showed that parents' and other family members' 'pressure to eat' strategy was correlated with children's caloric consumption [71] and overall fat mass [72].

Another important factor leading to childhood obesity is that a high portion of Hong Kong school children spends too many hours watching television (TV) and playing computer or video games [73]. Overweight or obese adolescents had a tendency to view TV programs and become less physically active. Childhood obesity in Hong Kong children is associated with other factors, such as obstructive sleep apnea [73]. A cross-sectional cohort study conducted in 2003 demonstrated that study participants had high systolic blood pressure, increased IR, elevated inflammatory marker (high sensitivity C-reactive protein level), low high-density lipoprotein-cholesterol levels and high alanine aminotransferase levels; these were all independently associated with obesity [74].

As mentioned before, TV is a potential cause of increased obesity in children and adolescents [75]. In China, access to Westernized TV programming and food advertising has increased [76]. Over a decade ago, urban Chinese children spent 17.2 hours a week (1.7 hours on weekdays, and more than 8 hours on weekends) watching TV [77]. Advertisements for food products, such as soft drinks and salty snacks, constituted more than 80% of commercials in China [78]. According to mothers surveyed in urban areas in China, many children have their own spending money, and they often use this money to buy snacks and beverages [79]. Chinese children reported using TV as a main source of information about junk food [79], and Chinese parents stated that their children influenced most of their purchases, especially of snacks [80]. This can be witnessed in most Chinese cities with large supermarkets today. Children especially are snacking more than adults [81]. In Xi'an, 19.9% adolescents were overweight or obese, and some reported drinking sugar-sweetened beverages (SSB) one to four times per week [82]. In 2004, 11.4% of young Chinese children were overweight or obese. The rate of child obesity had more than doubled since 1991 [83]. In theory, attention gained from TV advertisements regarding snacks, SSB, and other food products would increase the chances of children wanting to buy and/or consume those products. TV exposure alone can be linked to snacking behaviors [84]. For example, people who are affected by food cues in the environment will be more likely to eat when exposed to those food cues even though they may not be hungry [85]. Food products and restaurant chains seen in TV programs and commercials provide food cues to children, thus enhancing the need to snack while watching TV [86]. TV is present in almost every Chinese household, and TV advertising in China increasingly promotes high-calorie foods [87]. Chinese viewers who paid attention to TV commercials were more persuaded to engage in advertized snacking [84]. Chinese teenagers were more likely to report buying advertized food, but less likely to request snacks from their parents. This group in general makes more independent purchases [80]. Low-income families spent more hours watching TV than their counterparts [88,89]. However, snacks seen on TV tended to be purchased more by those with higher incomes [81]. One study in 1997 did not find significant differences in TV (and video) use and snacking behaviors among overweight and normal weight Chinese children [90]. All in all, this evidence portrays that non-physical entertainment does play a major role in weight management in young people.

METABOLIC SYNDROME

This is defined as "a combination of clinical disorders that increase the risk for diabetes and cardiovascular disease, including atherosclerosis, stroke and hypertension" [9]. The components of MetS include abdominal fat, atherogenic dyslipidemia, hypertension, pro-inflammatory state, pro-thrombic state and IR with or without glucose intolerance [91]. Other criteria include impaired glucose tolerance, IR, blood pressure ≥140/90 mmHg, triglyceride levels ≥1.695 mmol/L, high density lipoprotein cholesterol (HDL-C) levels ≤0.9 mmol/L (men) or ≤1.0 mmol/L (women), WHR >0.90 (men) or >0.85 (women), BMI >30 kg/m^2, microalbuminuria or urinary albumin excretion ratio ≥20 mg/min, and albumin:creatinine ratio ≥30 mg/g [91]. These figures are incorporated into the low grade inflammation hypothesis which states that macrophages and T-lymphocytes in accumulating fat are a source of various cytokines/chemokines that modulate the innate and adaptive immune responses [92,93]. Fat cells along with macrophages have been shown to respond to lipopolysaccharide (LPS)-induced inflammation by increasing pro-inflammatory nuclear factor kappa beta (NFkβ) production which triggers inflammatory cytokine synthesis such as serum amyloid A3 (SAA3) and interleukin-6 (IL-6). The adipocytes then express LPS-activated Toll-like receptors-4 (TLR-4) [93,94]. Another condition is adipocyte hyperplasia/hypertrophy which may trigger the local inflammatory responses and cause fat deposition in skeletal muscle, liver, and pancreas resulting in non-alcoholic steato-hepatitis (NASH) [95]. Hypertension accompanying obesity is based on leptin resistance and a putative perivascular adipose tissue factor that sensitizes vascular tissues to endogenous contractile agonists [96,97]. Based on the leptin resistance theory, leptin exerts a sympatho-excitatory effect and at high levels, it causes increased sympathetic nervous system activity leading to hypertension [98]. Another major player in diagnosing MetS is resistin, which is an adipocyte hormone that initially caused IR in mice [99]. Resistin causes the production of inflammatory cytokines such as tumor necrosis factor alpha (TNF-α), IL-1, IL-6, and IL-12 via NFkβ [100,101]. Therefore, obesity, MetS and IR development have been associated with increasing resistin levels causing inflammation specifically in T2DM [102]. Hyperglycemia is also a major contributing factor to MetS in that it reduces glutathione peroxidase (GPx), glutathione (GSH) and catalase while increasing thiobarbituric acid reactive substance (TBARS) levels (markers for oxidative stress) [103,104]. High blood sugar levels promote activation of the stress-activated protein kinase (SAPK/JNK) pathway described as glucose toxicity which inhibits pancreatic beta cell function leading to IR [105].

Studies have found that MetS has been linked to the development of cancer [106,107,108]. Obesity, dyslipidemia and hyperglycemia are all risk factors for colorectal cancer [109-112]. Interestingly, only a large WC was linked with the development of colon adenoma [113]. Furthermore, BMI and high triglyceride levels were also related to the development of cancer [114,115]. Another study found a relationship between colorectal adenoma and low serum HDL-C levels [116]. Possible causes for this cancer include MetS symptoms of inflammation, IR and oxidative stress [117]. The actual makeup of adipose promotes procarcinogenic inflammatory cytokine production [118-120]. Another study found that the high-sensitivity C-reactive protein level was higher in MetS patients with adenoma [116].

One study showed that MetS was very common in southern China [63]. Researchers found that 42.1% of hypertensive patients and 73.1% of diabetics had MetS [63]. Studies in Japan [121], Korea [122] and China [123,124] indicated that decreased insulin secretion contributed more to T2DM. Other research [125] demonstrated that both IR and impaired β-cell function occurred prior to abnormal glucose tolerance. Latest surveys indicated that the prevalence of total diabetes in China was about 9.7% [126]. Two thirds of Chinese diabetics had a BMI less than 25 kg/m^2 [127]. The Chinese theoretically have less IR compared to obese Westerners [128]. Non-obese, normal-glucose tolerant, first-degree relatives of diabetics have a higher IR [128]. IR was found to be independent of obesity and blood glucose level [128]. Increased plasma free fatty acids (FFA) in obese Chinese people was an important link between obesity and IR, and plasma FFA levels were negatively correlated with insulin sensitivity [129,130,131]. The Chinese exhibited impairment of peripheral insulin sensitivity and elevated circulating FFA levels [128]. For example, in Han Chinese adults living in Shanghai, risk factors for obesity included lower education levels, family history of T2DM, cigarette smoking, high systolic blood pressure, glycemia and dyslipidemia. Over 70% of T2DM cases were between 45 and 65 years old [132]. The Chinese were five times more likely to have a family history of T2DM than normal subjects [133].

Abdominal fat distribution may also be associated with metabolic risks than BMI [134,135]. In 2005, it was recommended that BMI and WC be used to classify obesity-related cardiovascular disease (CVD) risk in adults [136,137]. Another study confirmed that both BMI and WC were ideal screening tools used to assess CVD risk in China [138]. Hong Kong Chinese asymptomatic patients with central obesity had a high rate of CVD risk factors [139]. For example, 19% of these patients had carotid atherosclerotic plaques and 10% of them had abnormal carotid intima-media thickness. As a result, this could serve as an appropriate screening tool in diagnosing atherosclerosis in the centrally obese middle-aged Chinese population [139].

In summary, those in the north or urban areas of China have more problems with obesity, T2DM, dyslipidemia, hypertension, and MetS [140]. In northeast China, the local cuisines are high in sodium and fat, with wheat as the staple food due to the dry arctic winter. Atherosclerosis cases are increasing as influenced by a Westernized lifestyle [141-143]. Finally, in middle-aged and elderly Chinese living in northeast China, there was a higher incidence of MetS and cardiovascular disease, especially atherosclerosis [144].

DIETARY ALTERATIONS

China can be portrayed as a "double burden of malnutrition" where under-nutrition coexists with obesity [145]. A fifth of overweight and obese individuals are located in China [146]. Mortality rate related to over-nutrition is estimated to reach 3 million by 2030 in China [147]. Since 1989, every 1,000 kcal increase in energy intake was associated with a 0.28 increase in BMI [148]. Another study showed a smaller 0.18 BMI increase for every 1,000 kcal increase in energy intake since 2006 [149].

The world-wide shift in food selection and consumption has resulted in a diet that is more energy-dense and laden with saturated animal fat and processed sugars, and is low in complex carbohydrates, fiber, and fresh fruits and vegetables [150-152]. Socioeconomic factors play a major role in influencing diet and the general health of the population [153], such as in China. Underprivileged individuals tend to stock up on non-nutritious, high-calorie foods as low-budget staples, whereas nutrient-rich foods and high-quality diets are consumed by more affluent customers [154]. Continuous exposure to foreign food commercials [155] is also considered as another possible cause of obesity in China.

Increasing the consumption of SSB on a global scale leads to obesity [156,157]. In Hong Kong, women who consumed higher amounts of SSB had an 8% higher rate of central obesity and a 1.5 cm larger WC. On the other hand, the men were more likely to drink SSB frequently (20% vs. 10%), to eat more meat (2.30 vs. 2.14 portions), and were less likely to exercise (31% vs. 39%). Younger individuals were also more likely to consume SSB [158]. To date, most of the data associated with SSB and obesity came from cross-sectional studies in children and teenagers [159,160]. In China, SSB is a major food source with a high glycemic index [161,162]. Another study found associations between frequent SSB intake and obesity predominantly in Chinese women, while lack of exercise, smoking, and high meat consumption increased the risk for greater weight gain [158].

Diets in Asian countries have undergone a nutritional transition, shifting from more traditional plant-based diets to Westernized, highly-processed foods with added animal products [163]. Recent malnourished Asian populations are more susceptible to obesity [164]. One study found that overweight children and adolescents consumed more energy, protein, and fat and ate fewer carbohydrates than did the controls [163]. They consumed less grain, fewer vegetables, more fruits, meats and cooking oil, eggs, fish, milk, and legumes. Those who ate at least 25g of cooking oil, 200g of meat, and 100g of dairy products had a higher chance of being overweight [165].

From a recent cross-sectional survey done in Jiangsu Province, researchers found that a higher socio-economic status and urban residency were associated with energy-dense foods such as animal and dairy products, soft drinks, Western food, and increased snacking/breakfast skipping behaviors [166]. Rural and lower income students normally consumed rice porridge, a traditional, thin breakfast gruel. However, they also preferred hamburgers, ice cream, milk, fruits, chocolate, and SSB [166]. The traditional Chinese high-glycemic diet consists of a variety of high-glycemic staple rice products such as boiled rice, rice congee, and glutinous rice which pose adverse cardiovascular and MetS risks [167]. When the Chinese population was lean and active, this diet did not pose as much risk. However, China today has an obesity epidemic and a dietary transition shifting toward more processed foods such as SSB [167]. Chinese overweight and obese children's and adolescents' metabolic risk factors were due to their higher high-glycemic diets [163]. Dependence on processed foods and foods consumed outside the home has been observed among Chinese youth [166]. The reason is that these foods are "appetizing, convenient and ready to eat, portable, affordable in single portions," and widely marketed for the younger generation. These include soft drinks, biscuits, snacks, and fast-food sandwiches [163]. Higher incomes in China allow families to purchase SSB, snacks, and fast food. Supermarkets are packed with highly-processed, energy-dense, nutrient-poor, and lower-priced foods. Preferences include polished grains/white rice products, because Chinese consumers are unaware of the benefits from whole grains [163]. Global trading and the food industries' aggressive marketing have also increased the availability of more affordable, palatable over-processed foods [163].

Another major dietary component is glutamate, which is a major taste ingredient of dietary protein described as 'Umami' [168]. Free glutamate functions as a signal to regulate protein intake and energy homeostasis [169-173]. Major public concern has been raised in modern countries with regards to the use of monosodium glutamate (MSG) as a flavor enhancer [174]. Findings concluded that MSG had no long-term, serious health consequences [175]. Studies in primates, mice and rats provided some evidence that weight gain may be associated with MSG intake [176-178]. Human studies linking MSG and obesity were limited, but increasing obesity in Westernized nations with the addition of MSG to commercially prepared foods is evident [179]. In one study, MSG intake in Jiangsu province was not associated with obesity or weight gain after 5 years [179]. A study of underweight, protein and energy malnourished infants found that they preferred higher concentrations of MSG due to a heightened sensory response to amino acids to relieve this protein deficiency [170]. Another study found that enhancing the taste of the meal increased protein intake in geriatric patients, but it did not lead to a higher overall energy intake or weight gain [180]. There was a positive association between MSG consumption and the socio-economic status in rural China [179]. For instance, highly seasoned restaurant food contained as much as 5g or more of MSG [174]. Both rice and wheat are staple foods in southern China, whereas wheat starch is a staple food in northern China [179], which can be easily made more palatable with MSG.

Kazaks, Uyghurs and Mongolians are the major minorities in Xinjiang. The Kazaks have been reported to have hypertension [181], while obesity is common in the Uyghurs and Mongolians [182]. Significant differences in mean blood pressure between Han, Kazaks, Uyghurs and Tibetan ethnic groups were deemed to be caused by different diet-related habits. It is well-known that alcohol, high-sodium foods and meat are traditionally popular among these groups, which are associated with surviving the cold weather in Xinjiang. Traditionally among Kazaks, Uyghurs and Mongolians in Xinjiang, alcohol consumption is paired with eating large amounts of animal fat or salty dishes, which could lead to an increase in fibrinogen levels. Males in particular traditionally drink spirits to deal with the cold. Additionally, salted milk tea is consumed in large amounts; vegetables are also rare in this region, hence they are not commonly consumed [183].

BEHAVIORAL CAUSES

Eating disorders ranged from 1.3% to 5.21% among young Chinese females [184-188]. However, these data do not represent the entire population. Currently, there is little knowledge about weight control concerns and behaviors in China. BMI, dieting, and eating disorder symptoms are not clearly defined [189]. BMI was associated with eating disorders as well as obesity among teenagers [190-194]. One study demonstrated that those that were overweight or obese exhibited greater weight control concerns or behaviors compared to controls because they were more likely to be actively trying to lose weight and thus place themselves at risk [189]. Greater shape and weight concerns were observed among overweight females [195,196]. Another important study of adolescents in China found a strong association between smoking and the belief that smoking was important in weight control [197].

CONCLUSIONS

There exists a vast amount of knowledge on the topic of obesity, yet more research needs to be conducted in the Chinese population. Obesity in China is a multifactorial disease where intervention is not always clear-cut or applicable. For instance, specific gene therapy may be available in the future to prevent childhood and adulthood weight gain and endocrine disorders. Lifestyle and behavioral changes need to be addressed and applied to prevent unhealthy physiques. Alternative medicine intervention, such as acupuncture and Traditional Chinese Medicine remedies, may be most appropriate for this part of the world. Overall, obesity is preventable and now is the ideal time in implementing current scientific methods and techniques to battle this epidemic.

CONFLICT OF INTEREST

The authors report no conflict of interest.

REFERENCES

[1] Cecchini M, Sassi F, Lauer JA, Lee YY, Guajardo-Barron V, Chisholm D. Tackling of unhealthy diets, physical inactivity, and obesity: health effects and cost-effectiveness. Lancet 2010; 376: 1775-84.

[2] Popkin BM. The nutrition transition and obesity in the developing world. J Nutr 2001; 131: 871S-3S.
[3] Poskitt EM. Countries in transition: underweight to obesity non-stop? Ann Trop Paediat 2009; 29: 1-11.
[4] Lu Y, Goldman D. The effects of relative food prices on obesity—evidence from China: 1991-2006. NBER Working Paper Series [w15720, 2010]. 2010 Dec 7; [cited 2010 February 1]. Available from: http://ssrn.com/abstract=1548778.
[5] Lee SA, Wen W, Xu WH, *et al.* Prevalence of obesity and correlations with lifestyle and dietary factors in Chinese men. Obesity (Silver Spring) 2008; 16: 1440-7.
[6] Janus ED. Epidemiology of cardiovascular risk factors in Hong Kong. Clin Exp Pharmacol Physiol 1997; 24: 987-8.
[7] Yang W, Lu J, Weng J, *et al.* Prevalence of diabetes among men and women in China. N Engl J Med 2010; 362: 1090-101.
[8] Brown WV, Fujioka K, Wilson PW, Woodworth KA. Obesity: why be concerned? Am J Med 2009; 122: S4-11.
[9] Achike FI, To NH, Wang H, Kwan CY. Obesity, metabolic syndrome, adipocytes and vascular function: a holistic viewpoint. Clin Exp Pharmacol Physiol 2011; 38(1): 1-10.
[10] Rossner S. Adolphe Quetelet (1796-1874). Obes Rev 2007; 8(2):183.
[11] WHO Consultation on Obesity. Obesity: preventing and managing the global epidemic: report of a WHO consultation; 1999; Geneva, Switzerland: WHO technical report series 894.
[12] Lee CD, Blair SN, Jackson AS. Cardiorespiratory fitness, body composition, and all cause and cardiovascular disease mortality in men. Am J Clin Nutr 1999; 69: 373-80.
[13] Bray GA. In: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, Hauser SL, Longo DL, Eds. Harrison's Principles of Internal Medicine. New York: McGraw-Hill. 1998; pp. 454-62.
[14] Lean ME. Pathophysiology of obesity: Proc Nutr Soc 2000; 59(3): 331-6.
[15] Hyun YJ, Kim OY, Jang Y, *et al*. Evaluation of metabolic syndrome risk in Korean premenopausal women: not waist circumference but visceral fat. Circ J 2008; 72: 1308-15.
[16] Li R, Lu W, Jia J, Zhang S, Shi L, Li Y: Relationships between indices of obesity and its cardiovascular comorbidities in a Chinese population. Circ J 2008; 72: 973-8.
[17] Shao J, Yu L, Shen X, Li D, Wang K. Waist-to-height ratio, an optimal predictor for obesity and metabolic syndrome in Chinese adults. J Nutr Health Aging 2010; 14(9): 782-5.
[18] Groschl M, Topf HG, Bohlender J, *et al.* Identification of ghrelin in human saliva: production by the salivary glands and potential role in proliferation of oral keratinocytes. ClinChem 2005; 51: 997-1006.
[19] English PJ, Ghatei MA, Malik IA, Bloom SR, Wilding JP. Food fails to suppress ghrelin levels in obese humans. J Clin Endocrinol Metab 2002; 87: 2984.
[20] Shiiya T, Nakazato M, Mizuta M, *et al*. Plasma ghrelin levels in lean and obese humans and the effect of glucose on ghrelin secretion. J Chin Endocrinol Metab 2002; 87:240-4.
[21] Aydin S. A comparison of ghrelin, glucose, alpha-amylase and protein levels in saliva from diabetics. J Biochem Mol Biol 2007; 40: 29-35.
[22] Fagerberg B, Hulten LM, Hulthe J. Plasma ghrelin, body fat, insulin resistance, and smoking in clinically healthy men: the atherosclerosis and insulin resistance study. Metabolism 2003; 52: 1460-3.
[23] Li BB, Chen ZB, Li BC, *et al.* Expression of ghrelin in human salivary glands and its levels in saliva and serum in Chinese obese children and adolescents. Arch Oral Biol 2010; [Epub ahead of print].
[24] Srivastava S, Krueger KE. In: Wong, Ed. Salivary diagnostics. Iowa: Wiley-Blackwell Press 2008; .pp.94-103.
[25] Bouchard C. Childhood obesity: are genetic differences involved? Amer J Clin Nutr 2009; 89: 1494S-1501S.
[26] WHO Expert Consultation. WHO Technical Report Series 894: Obesity: preventing and managing the global epidemic; Geneva, Switzerland. World Health Organization 2000.
[27] McMillen IC, Rattanatray L, Duffield JA, Morrison, *et al.* The early origins of later obesity: pathways and mechanisms. Advan Exp Med Biol 2009; 646: 71-81.
[28] Martorell R, Stein AD, Schroeder DG. Early nutrition and later adiposity. J Nutr 2001; 131: 874S-80S.
[29] Mcauley KA, Williams SM, Mann JI, *et al*. Diagnosing insulin resistance in the general population. Diabetes Care 2001; 24: 460-4.
[30] Thorleifsson G, Walters GB, Gudbjartsson DF, *et al*. Genome-wide association yields new sequence variants at seven loci that associate with measures of obesity. Nat Genet 2009; 41: 18-24.
[31] Willer CJ, Speliotes EK, Loos RJ, *et al.* Six new loci associated with body mass index highlight a neuronal influence on body weight regulation. Nat Genet 2009; 41: 25-34.
[32] Scuteri A, Sanna S, Chen WM, *et al*. Genome-wide association scan shows genetic variants in the FTO gene are associated with obesity-related traits. PLoS Genet 2007; 3:e115.
[33] Frayling TM, Timpson NJ, Weedon MN, *et al.* A common variant in the FTO gene is associated with body mass index and predisposes to childhood and adult obesity. Science 2007; 316: 889-94.
[34] Dina C, Meyre D, Gallina S, *et al*. Variation in FTO contributes to childhood obesity and severe adult obesity. Nat Genet 2007; 39: 724-6.
[35] Loos RJ, Lindgren CM, Li S, *et al*. Common variants near MC4R are associated with fat mass, weight and risk of obesity. Nat Genet 2008; 40: 768-75.
[36] Chambers JC, Elliott P, Zabaneh D, *et al*. Common genetic variation near MC4R is associated with waist circumference and insulin resistance. Nat Genet 2008; 40: 716-8.
[37] Gerken T, Girard CA, Tung YC, *et al*. The obesity-associated FTO gene encodes a 2-oxoglutarate-dependent nucleic acid demethylase. Science 2007; 318(5855): 1469-72.
[38] Sanchez-Pulido L, Andrade-Navarro MA. The FTO (fat mass and obesity associated) gene codes for a novel member of the non-heme dioxygenase superfamily. BMC Biochem 2007; 8: 23.
[39] Li H, Wu Y, Loos RJ, *et al*. Variants in the fat mass- and obesity-associated (FTO) gene are not associated with obesity in a Chinese Han population. Diabetes 2008; 57: 264-8.
[40] Deng FY, Lei SF, Li MX, Jiang C, Dvornyk V, Deng HW. Genetic determination and correlation of body mass index and bone mineral density at the spine and hip in Chinese Han ethnicity. Osteoporos Int 2006; 17: 119-24.
[41] Wu KD, Hsiao CF, Ho LT, *et al.* Clustering and heritability of insulin resistance in Chinese and Japanese hypertensive families: a Stanford-Asian pacific program in hypertension and insulin resistance sibling study. Hypertens Res 2002; 25: 529-36.
[42] Liu PY, Li YM, Li MX, *et al.* Lack of evidence for a major gene in the Mendelian transmission of BMI in Chinese. Obes Res 2004; 12: 1967-73.
[43] Cheung CY, Tso AW, Cheung BM, *et al.* Obesity susceptibility genetic variants identified from recent genome-wide association studies: implications in a Chinese population. J Clin Endocrinol Metab 2010; 95(3): 1395-403.
[44] Al-Attar SA, Pollex RL, Ban MR, *et al*. Association between the FTO rs9939609 polymorphism and the metabolic syndrome in a non-Caucasian multi-ethnic sample. Cardiovasc Diabetol 2008; 7:5.
[45] Hubacek JA, Bohuslavova R, Kuthanova L, *et al*. The FTO gene and obesity in a large eastern European population sample: the HAPIEE study. Obesity (Silver Spring) 2008; 16: 2764-6.
[46] Hotta K, Nakata Y, Matsuo T, *et al.* Variations in the FTO gene are associated with severe obesity in the Japanese. J Hum Genet 2008; 53: 546-53.
[47] Grant SF, Li M, Bradfield JP, *et al*. Association analysis of the FTO gene with obesity in children of Caucasian and African ancestry reveals a common tagging SNP. PLoS One 2008; 3:e1746.
[48] Kanehisa M, Goto S. KEGG: Kyoto encyclopedia of genes and genomes. Nucleic Acids Res 2000; 28: 27-30.
[49] Aravind L, Koonin EV. Fold prediction and evolutionary analysis of the POZ domain: structural and evolutionary relationship with the potassium channel tetramerization domain. J Mol Biol 1999; 285: 1353-61.
[50] Ding XF, Luo C, Ren KQ, *et al.* Characterization and expression of a human KCTD1 gene containing the BTB domain, which mediates transcriptional repression and homomeric interactions. DNA Cell Biol 2008; 27: 257-25.
[51] Chang YC, Liu PH, Lee WJ, *et al.* Common variation in the fat mass- and obesity-associated (FTO) gene confers risk of obesity and modulates BMI in the Chinese population. Diabetes 2008; 57(8): 2245-52.

[52] Tan JT, Dorajoo R, Seielstad M, *et al.* FTO variants are associated with obesity in the Chinese and Malay populations in Singapore. Diabetes 2008; 57(10): 2851-7.

[53] Fang H, Li Y, Du S, *et al.* Variant rs9939609 in the FTO gene is associated with body mass index among Chinese children. BMC Medical Genetics 2010; 11: 136.

[54] Li X, Song F, Jiang H, *et al.* A genetic variation in the fat mass-and obesity-associated gene is associated with obesity and newly diagnosed type 2 diabetes in a Chinese population. Diabetes Metab Res Rev 2010; 26(2): 128-32.

[55] Xi B, Shen Y, Zhang M, *et al.* The common rs9939609 variant of the fat mass and obesity-associated gene is associated with obesity risk in children and adolescents of Beijing, China. BMC Med Genet 2010; 11: 107.

[56] Jellema A, Zeegers MP, Feskens EJ, *et al.* Gly972Arg variant in the insulin receptor substrate-1 gene and association with type 2 diabetes: a meta-analysis of 27 studies. Diabetologia 2003; 46(7): 990-5.

[57] Huang W, Sun Y, Sun J. Combined effects of FTO rs9939609 and MC4R rs17782313 on obesity and BMI in Chinese Han populations. Endocrine 2010; [Epub ahead of print].

[58] Ng MC, Park KS, Oh B, *et al.* Implication of genetic variants near TCF7L2,SLC30A8, HHEX, CDKAL1, CDKN2A/B, IDF2BP2 and FTO in type 2 diabetes and obesity in 6,719 Asians. Diabetes 2008; 57(8): 2226-33.

[59] Hara K, Boutin P, Mori Y, *et al.* Genetic variation in the gene encoding adiponectin is associated with an increased risk of type 2 diabetes in the Japanese population. Diabetes 2002; 51: 536-40.

[60] Vasseur F, Helbecque N, Dina C, *et al.* Single nucleotide polymorphism haplotypes in the both proximal promoter and exon 3 of the ADIPOQ gene modulate adipocyte-secreted adiponectin hormone levels and contribute to the genetic risk for type 2 diabetes in French Caucasians. Hum Mol Genet 2009; 11: 2607-14.

[61] Gu HF, Abulaiti A, Ostenson CG, *et al.* Single nucleotide SNPs in the proximal promoter region of the adiponectin (APM1) gene are associated with T2D in Swedish Caucasians. Diabetes 2004; 53(Suppl 1): 31-5.

[62] Miyake K, Yang W, Hara K, *et al*. Construction of a prediction model for type 2 diabetes mellitus in the Japanese population based on 11 genes with strong evidence of the association. J Hum Genet 2009; 54: 236-41.

[63] Wang H, Wu M, Zhu W, *et al.* Evaluation of the association between the AC3 genetic polymorphisms and obesity in a Chinese Han population. PLoS One 2010; 5(11): e13851.

[64] Luo J, Hu FB. Time trends of obesity in pre-school children in China from 1989 to 1997. Int J Obesity 2002; 26: 553-8.

[65] Rhee K. Childhood overweight and the relationship between parent behaviors, parenting style, and family functioning. Annals Amer Acad Pol Soc Sci 2008; 615: 11-37.

[66] Shi Z, Liena N, Nirmal Kumara B, Holmboe-Ottesen G. Perceptions of weight and associated factors of adolescents in Jiangsu Province, China. Public Health Nutr 2007; 10: 298-305.

[67] Huang JS, Becerra K, Oda T. Parental ability to discriminate the weight status of children: results of a survey. Pediatrics 2007; 120: e112-9.

[68] Maynard LM, Galuska DA, Blanck HM, Serdula MK. Maternal perceptions of weight status of children. Pediatrics 2003; 111: 1226-31.

[69] Campbell MW, Williams J, Hampton A., Wake M. Maternal concern and perceptions of overweight in Australian preschool-aged children. Med J Aust 2006; 184: 274-7.

[70] Wen X, Hui SS. Chinese parents' perceptions of their children's weights and their relationship to parenting behaviors. Child Care Health Dev 2010; [Epub ahead of print].

[71] Drucker RR, Hammer LD, Agras WS, Bryson S. Can mothers influence their child's eating behavior. J Dev Behav Pediatr 1999; 20: 88-92.

[72] Spruijt-Metz D, Lindquist CH, Birch LL, Fisher JO, Goran MI. Relation between mothers' child-feeding practices and children's adiposity. Amer J Clin Nutr 2002; 75: 581-6.

[73] Kong AP, Chow CC. Medical consequences of childhood obesity: a Hong Kong perspective. Res Sports Med 2010; 18: 1,16-25.

[74] Kong AP, Choi KC, Ko GT, *et al*. Associations of overweight with insulin resistance, beta-cell function and inflammatory markers in Chinese adolescents. Pediatr Diabetes 9: 488-95.

[75] Hancox R, Milne B, Poulton R. Association between child and adolescent television viewing and adult health: a longitudinal birth cohort study. Lancet 2004; 364: 257-62.

[76] Hong J. The internationalization of television in China: an evolution of ideology, society, and media since the reform. Westport, CT: Praeger Publishers 1998.

[77] McNeal JU, Ji MF. Chinese children as consumers: an analysis of their new product information sources. J Consum Market 1999; 16: 345.

[78] Ji MF, McNeal JU. How Chinese children's commercials differ from those of the United States: a content analysis. J Advert 2001; 30: 79.

[79] Zhang YB, Harwood J. Modernization and tradition in an age of globalization: cultural values in Chinese television commercials. J Commun 2004; 54: 156-72.

[80] McNeal JU, Yeh C. Development of consumer behavior patterns among Chinese children. J Consum Market 1997; 14: 45-59.

[81] Wang Z, Zhai F, Du S, *et al*. Dynamic shifts in Chinese eating behaviors. Asia Pac J Clin Nutr 2008; 17: 123-30.

[82] Li M, Dibley MJ, Sibbritt D, *et al*. Factors associated with adolescents' overweight and obesity at community, school and household levels in Xi'an City, China: results of hierarchical analysis. Eur J Clin Nutr 2008; 62: 635-43.

[83] Popkin BM, Conde W, Hou N, *et al*. Is there a lag globally in overweight trends for children compared with adults? Obesity 2006; 14: 1846-53.

[84] McGuire WJ. Input and output variables currently promising for constructing persuasive communications. In: Rice RE, Atkin CK, Eds. Public Communication Campaigns. Thousand Oaks, CA: Sage Publications 2001; pp. 22-48.

[85] Schachter S. Obesity and eating. Science 1968; 161: 751-6.

[86] Coon KA, Goldberg J, Rogers BL, *et al*. Relationships between use of television during meals and children's food consumption patterns. Pediatrics 2001; 107: E7.

[87] Parvanta SA, Brown JD, Du S, Zimmer CR, Zhao X, Zhai F. Television use and snacking behaviors among children and adolescents in China. J Adolesc Health 2010; 46(4): 339-45.

[88] Livingstone S. Young People and New Media. Thousand Oaks, CA: Sage Publications 2002.

[89] Roberts DF, Foehr UG. Kids and Media in America. New York: Cambridge University Press 2004.

[90] Waller CE, Du S, Popkin BM. Patterns of overweight, inactivity, and snacking in Chinese children. Obes Res 2003; 11: 957-61.

[91] Grundy SM, Brewer B, Cleeman JI, Smith SC, Lenfant C. Definition of metabolic syndrome: Report of the national heart, lung, and blood institute/American heart association conference on scientific issues related to definition. Circulation 2004; 109: 433-8.

[92] Halberg N, Wernstedt I, Scherer PE. The adipocyte as an endocrine cell. Endocrinol Metab Clin North Am 2008; 37(3): 753-67.

[93] Lin Y, Rajala MW, Berger JP, Moller DE, Barzilai N, Scherer PE. Hyperglycemia induced production of acute phase reactants in adipose tissue. J Biol Chem 2001; 276(45): 42077-83.

[94] Berg AH, Lin Y, Lisanti MP, Scherer PE. Adipocyte differentiation induces dynamic changes in NF-kappaB expression and activity. Am J Physiol Endocrinol Metab 2004; 287(6): E1178-88.

[95] Unger RH. Lipotoxic diseases. Annu Rev Med 2002; 53: 319-36.

[96] Rahmouni K, Correia ML, Haynes WG, Mark AL. Obesity-associated hypertension: new insights into mechanisms. Hypertension 2005; 45(1): 9-14.

[97] Knudson JD, Dincer UD, Dick GM, et al. Leptin resistance extends to the coronary vasculature in prediabetic dogs and provides a protective adaptation against endothelial dysfunction. Am J Physiol Heart Circ Physiol 2005; 289(3): H1038-46.

[98] Hall JE, Brands MW, Hildebrandt DA, Kuo J, Fitzgerald S. Role of sympathetic nervous system and neuropeptides in obesity hypertension. Braz J Med Biol Res 2000; 33(6): 605-18.

[99] Steppan CM, Bailey ST, Bhat S, et al. The hormone resistin links obesity to diabetes. Nature 2001; 409(6818): 307-12.

[100] Singer G, Granger DN. Inflammatory responses underlying the microvascular dysfunction associated with obesity and insulin resistance. Microcirculation 2007; 14(4-5): 375-87.

[101] Silswal N, Singh AK, Aruna B, Mukhopadhyay S, Ghosh S, Ehtesham NZ. Human resistin stimulates the pro-inflammatory cytokines TNF-alpha and IL-12 in macrophages by NF-kappaB-dependent pathway. Biochem Biophys Res Commun 2005; 334(4): 1092-101.

[102] McTernan CL, McTernan PG, Harte AL, Levick PL, Barnett AH, Kumar S. Resistin, central obesity, and type 2 diabetes. Lancet 2002; 359(9300): 46-7.
[103] Dave GS, Kalia K. Hyperglycemia induced oxidative stress in type-1 and type-2 diabetic patients with and without nephropathy. Cell Mol Biol 2007; 53: 68-78.
[104] Lastra G, Manrique C. The expanding role of oxidative stress, renin angiotensin system, and beta-cell dysfunction in the cardiometabolic syndrome and type 2 diabetes mellitus. Antioxid Redox Signal 2007; 9: 943-54.
[105] Kaneto H, Nakatani Y, Kawamori D, Miyatsuka T, Matsuoka TA. Involvement of oxidative stress and the JNK pathway in glucose toxicity. Rev Diabet Stud 2004; 1: 165-74.
[106] Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. N Engl J Med 2003; 348(17): 1625-38.
[107] Jee SH, Ohrr H, Sull JW, Yun JE, Ji M, Samet JM. Fasting serum glucose level and cancer risk in Korean men and women. JAMA 2005; 293(2): 194-202.
[108] Lukanova A, Bjor O, Kaaks R, *et al*. Body mass index and cancer: results from the northern Sweden health and disease cohort. Int J Cancer 2006; 118(2): 458-66.
[109] Giovannucci E. Modifiable risk factors for colon cancer. Gastroenterol Clin North Am 2002; 31: 925-43.
[110] Limburg PJ, Anderson KE, Johnson TW, *et al*. Diabetes mellitus and subsite-specific colorectal cancer risks in the Iowa women's health study. Cancer Epidemiol Biomarkers Prev 2005; 14(1): 133-7.
[111] Ahmed RL, Schmitz KH, Anderson KE, Rosamond WD, Folsom AR. The metabolic syndrome and risk of incident colorectal cancer. Cancer 2006; 107(1): 28-36.
[112] Colangelo LA, Gapstur SM, Gann PH, Dyer AR, Liu K. Colorectal cancer mortality and factors related to the insulin resistance syndrome. Cancer Epidemiol Biomarkers Prev 2002; 11(4): 385-91.
[113] Kim JH, Lim YJ, Kim YH, *et al*. Is metabolic syndrome a risk factor for colorectal adenoma? Cancer Epidemiol Biomarkers Prev 2007; 16(8): 1543-6.
[114] Lee GE, Park HS, Yun KE, *et al*. Association between BMI and metabolic syndrome and adenomatous colonic polyps in Korean men. Obesity (Silver Spring) 2008; 16(6): 1434-9.
[115] Wang YY, Lin SY, Lai WA, Liu PH, Sheu WH. Association between adenomas of rectosigmoid colon and metabolic syndrome features in a Chinese population. J Gastroenterol Hepatol 2005; 20(9): 1410-5.
[116] Liu CS, Hsu HS, Li CI, *et al*. Central obesity and atherogenic dyslipidemia in metabolic syndrome are associated with increased risk for colorectal adenoma in a Chinese population. BMC Gastroenterol 2010; 10:51.
[117] Cowey S, Hardy RW. The metabolic syndrome: a high-risk state for cancer? Am J Pathol 2006; 169(5): 1505-22.
[118] Gunter MJ, Canzian F, Landi S, Chanock SJ, Sinha R, Rothman N. Inflammation-related gene polymorphisms and colorectal adenoma. Cancer Epidemiol Biomarkers Prev 2006; 15(6): 1126-31.
[119] Kim S, Keku TO, Martin C, *et al*. Circulating levels of inflammatory cytokines and risk of colorectal adenomas. Cancer Res 2008; 68(1): 323-8.
[120] Trevisan M, Liu J, Muti P, Misciagna G, Menotti A, Fucci F. Markers of insulin resistance and colorectal cancer mortality. Cancer Epidemiol Biomarkers Prev 2001; 10(9): 937-41.
[121] Fukushima M, Usami M, Ikeda M, *et al.* Insulin secretion and insulin sensitivity at different stages of glucose tolerance: a cross-sectional study of Japanese type 2 diabetes. Metabolism 2004; 53: 831-5.
[122] Rhee SY, Kwon MK, Park BJ, *et al*. Differences in insulin sensitivity and secretory capacity based on OGTT in subjects with impaired glucose regulation. Korean J Intern Med 2007; 22: 270-4.
[123] Liu J, Li YB, Shao H, *et al.* Evaluation of islet beta cell function in subjects with normal glucose tolerance, impaired glucose regulation, and type 2 diabetes mellitus. Zhonghua Yi Xue Za Zhi 2007; 15: 1252-5.
[124] Qian L, Xu L, Wang X, Fu, *et al.* Early insulin secretion failure leads to diabetes in Chinese subjects with impaired glucose regulation. Diabetes Metab Res Rev 2009; 25: 144-9.
[125] Han XY, Ji LN, Zhou XH. Cross-sectional study of the pathophysiologic and clinical features in the first-degree relatives of type 2 diabetic patients. Beijing Da Xue Xue Bao 2005; 37: 159-62.
[126] Yang W, Lu J, Weng J, *et al*. China national diabetes and metabolic disorders study group: prevalence of diabetes among men and women in China. N Engl J Med 2010; 362: 1090-101.
[127] Jia WP, Xiang KS, Chen L, Lu JX, Wu YM. Epidemiological study on obesity and its comorbidities in urban Chinese older than 20 years of age in Shanghai, China. Obes Rev 2002; 3: 157-65.
[128] Wang C, Tan H, Yu H, *et al*. Impairment of insulin action in non-obese, normal-glucose tolerant, first-degree relatives of Chinese type 2 diabetic patients. Diabetes Res Clin Pract 2011; 91(1): 67-71.
[129] Li HL, Yu YR, Yu HL, Wang C, Zhang XX. Relationship between peripheral insulin resistance and beta-cell function in obese subjects. Sichuan Da Xue Xue Bao Yi Xue Ban 2005; 36: 378-81.
[130] Perseghin G, Ghosh S, Gerow K, Shulman GI. Metabolic defects in lean nondiabetic offspring of NIDDM parents. Diabetes 1997; 146: 1001-9.
[131] Van Haeften TW, Dubbeldam S, Zonderland ML, Erkelens DW. Insulin secretion in normal glucose-tolerant relatives of type 2 diabetic subjects. Diabetes care 1998; 21: 278-82.
[132] Hu FB, Manson JE, Stampfer MJ, *et al*. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. N Engl J Med 2001; 345(11): 790-7.
[133] Xu H, Song Y, You NC, *et al*. Prevalence and clustering of metabolic risk factors for type 2 diabetes among Chinese adults in Shanghai, China. BMC Public Health 2010; 10: 683.
[134] Janssen I, Katzmarzyk PT, Ross R. Body mass index, waist circumference, and health risk: evidence in support of current national institutes of health guidelines. Arch Intern Med 2002; 162: 2074-9.
[135] Ardern CI, Katzmarzyk PT, Janssen I, *et al*. Discrimination of health risk by combined body mass index and waist circumference. Obes Res 2003; 11: 135-42.
[136] Department of Disease Control Ministry of Health, P.R. China. The guidelines for prevention and control of overweight and obesity in Chinese adults. Biomed Environ Scienc 2004; Suppl 17: 1-36.
[137] Corporative Meta-analysis Group of China Obesity Task Force. Predictive values of body mass index and waist circumference to risk factors of related diseases in Chinese adult population. Chin J Epidemiol 2003; 23: 5-10.
[138] Du SM, Ma GS, Li YP, *et al*. Relationship of body mass index, waist circumference and cardiovascular risk factors in Chinese adult. Biomed Environ Sci 2010; 23(2): 92-101.
[139] Ko GT, Chow CC, Leung G, *et al*. High rate of increased carotid intima-media thickness and atherosclerotic plaques in Chinese asymptomatic subjects with central obesity. Int J Cardiovasc Imaging 2010; [Epub ahead of print].
[140] Yu Z, Lin X, Haas JD, *et al*. Obesity related metabolic abnormalities: distribution and geographic differences among middle-aged and older Chinese populations. Prev Med 2009; 48: 272-8.
[141] Mi J, Cheng H, Zhao XY, Hou DQ, Chen FF, Zhang KL. Developmental origin of metabolic syndrome: interaction of thinness at birth and overweight during adult life in Chinese population. Obes Rev 2008; 9(Suppl 1): 91-4.
[142] Kokubo Y, Okamura T, Yoshimasa Y, *et al*. Impact of metabolic syndrome components on the incidence of cardiovascular disease in a general urban Japanese population: the suita study. Hypertens Res 2008; 31: 2027-35.
[143] Wang J, Li H, Franco OH, Yu Z, Liu Y, Lin X. Adiponectin and metabolic syndrome in middle-aged and elderly Chinese. Obesity (Silver Spring) 2008; 16: 172-8.
[144] Liu H, Zhang X, Feng X, Li J, Hu M, Yambe T. Effects of metabolic syndrome on cardio-ankle vascular index in middle-aged and elderly Chinese. Metab Syndr Relat Disord 2010; [Epub ahead of print].
[145] Popkin BM, Paeratakul S, Zhai F, Ge K. A review of dietary and environmental correlates of obesity with emphasis on developing countries. Obes Res 1995; 3(Suppl 2): 145s-53s.
[146] Wu Y. Overweight and obesity in China. BMJ 2006; 333: 362-3.
[147] Chen CM. Eating patterns—a prognosis for China. Asia Pac J Clin Nutr 1995; 4(Supp 1): 24-8.
[148] Paeratakul S, Popkin BM, Keyou G, Adair LS, Stevens J. Changes in diet and physical activity affect the body mass index of Chinese adults. Int J Obes Relat Metab Disord 1998; 22: 424-31.
[149] Shankar B. Obesity in China: the differential impacts of covariates along the BMI distribution. Obesity 2010; 18(8):1660-6.

[150] Joint FAO/WHO Expert Consultation. WHO Technical Report Series 916: Diet, Nutrition and the prevention of chronic diseases; Geneva, Switzerland. World Health Organization 2003.
[151] Zhai F, Wang H, Du S, *et al*. Prospective study on nutrition transition in China. Nutr Rev 2009; 67: S56-61.
[152] Madanat HN, Troutman KP, Al-Madi B. The nutrition transition in Jordan: the political, economic and food consumption contexts. Promotion et Educ 2008; 15: 6-10.
[153] Drewnowski A. Obesity, diets, and social inequalities. Nutr Rev 2009; 67: S36-9.
[154] Jones N, Furlanetto DL, Jackson JA, Kinn S. An investigation of obese adults' views of the outcomes of dietary treatment. J Human Nutr Diet 2007; 20: 486-94.
[155] James WP. The fundamental drivers of the obesity epidemic. Obes Rev 2008; 9: 6-13.
[156] Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. J Clin Endocrinol Metab 2008; 93: S9-30.
[157] Olsen NJ, Heitmann BL. Intake of calorically sweetened beverages and obesity. Obes Rev 2009; 10: 68-75.
[158] Ko GT, So WY, Chow CC, *et al*. Risk associations of obesity with sugar-sweetened beverages and lifestyle factors in Chinese: the ‘Better Health for Better Hong Kong’ health promotion campaign. Eur J Clin Nutrition 2010; 64: 1386-92.
[159] Ariza AJ, Chen EH, Binns HJ, Christoffel KK. Risk factors for overweight in five-to six-year-old Hispanic-American children: a pilot study. J Urban Health 2004; 81: 150-61.
[160] Berkey CS, Rockett HR, Field AE, Gillman MW, Colditz GA. Sugar-added beverages and adolescent weight change. Obes Res 2004; 12: 778-88.
[161] Murakami K, Sasaki S, Takahashi Y, *et al*. Dietary glycemic index and load in relation to metabolic risk factors in Japanese female farmers with traditional dietary habits. Am J Clin Nutr 2006; 83: 1161-9.
[162] Villegas R, Liu S, Gao YT, *et al.* Prospective study of dietary carbohydrates, glycemic index, glycemic load, and incidence of type 2 diabetes mellitus in middle-aged Chinese women. Arch Intern Med 2007; 167: 2310-6.
[163] Guldan, GS. Asian children's obesogenic diets—time to change this part of the energy balance equation? Res Sports Med 2010; 18: 1,5-15.
[164] Corvalan C, Dangour AD, Uauy R. Need to address all forms of childhood malnutrition with a common agenda. Arch Dis Child 2008; 93(5): 361-2.
[165] Li Y, Zhai F, Yang X, *et al*. Determinants of childhood overweight and obesity in China. British J Nutr 2007; 97: 210-5.
[166] Shi Z, Lien N, Kumar BN, Holmboe-Ottesen G. Socio-demographic differences in food habits and preferences of school adolescents in Jiangsu Province, China. Eur J Clin Nutr 2005; 59: 1439-48.
[167] Ding EL, Malik VS. Convergence of obesity and high glycemic diet on compounding diabetes and cardiovascular risks in modernizing China: an emerging public health dilemma. Globalization and Health 2008; 4: 4.
[168] Kurihara K, Kashiwayanagi M. Physiological studies on umami taste. J Nutr 2000; 130(Suppl 4): 931S-34S.
[169] Smriga M, Torii K. Release of hypothalamic norepinephrine during MSG intake in rats fed normal and nonprotein diet. Physiol Behav 2000; 70: 413-5.
[170] Vazquez M, Pearson PB, Beauchamp GK. Flavour preferences in malnourished Mexican infants. Physiol Behav 1981; 28: 513-9.
[171] Murphy C. Aging and chemosensory perception of and preference for nutritionally significant stimuli. Ann N Y Acad Sci 1989; 561: 251-66.
[172] Mori M, Kawada T, Ono T, *et al*. Taste preference and protein nutrition and L-amino acid homeostasis in male Sprague-Dawley rats. Physiol Behav 1991; 49: 987-95.
[173] Laska M, Hernandez Salazar LT. Gustatory responsiveness to monosodium glutamate and sodium chloride in four species of nonhuman primates. J Exp Zool A Comp Exp Biol 2004; 301: 898-905.
[174] Food standards Australia New Zealand (2003) monosodium glutamate-a safety report. technical report (series no. 23); June 2003 [updated 2011 February 3; cited 2011 Feb 1]. Available from: http://www.foodstandards.gov.au
[175] Walker R, Lupien JR. The safety evaluation of monosodium glutamate. J Nutr 2000; 130(Suppl 4S): 1049S-52S.
[176] Olney JW, Sharpe LG. Brain lesions in an infant rhesus monkey treated with monosodium glutamate. Science 1969; 166: 386-8.
[177] Olney JW. Brain lesions, obesity, and other disturbances in mice treated with monosodium glutamate. Science 1969; 164: 719-21.
[178] Hermanussen M, Garcia AP, Sunder M, *et al*. Obesity, voracity, and short stature: the impact of glutamate on the regulation of appetite. Eur J Clin Nutr 2006; 60: 25-31.
[179] Shi Z, Luscombe-Marsh ND, Wittert GA, *et al*. Monosodium glutamate is not associated with obesity or a greater prevalence of weight gain over 5 years: findings from the Jiangsu Nutrition Study of Chinese adults. Br J Nutr 2010; 104: 457-63.
[180] Essed NH, van Staveren WA, Kok FJ, *et al.* No effect of 16 weeks flavour enhancement on dietary intake and nutritional status of nursing home elderly. Appetite 2007; 48: 29-36.
[181] Jumabay M, Kawamura H, Mitsubayashi H, *et al.* Urinary electrolytes and hypertension in elderly Kazakhs. Clin Exp Nephrol 2001; 5: 217-21.
[182] Wang K, Ao YT, Zhao L, *et al.* Analysis on obesity and its risk factors among inhabitants of Bortala prefecture of Xinjiang autonomous region. Chinese J Public Health 2006; 22(9): 1128-30.
[183] Xi B, Mi J. FTO polymorphisms are associated with obesity but not with diabetes in East Asian populations: a meta-analysis. Biomed Environ Sci 2009; 22(6): 449-57.
[184] Fu D, Wang J, Chen W, Bi Y: Disordered eating attitudes and behaviours and related mood states among female university students in Beijing. Chin Ment Health J 2005; 19(8): 525-8.
[185] Liang X, Guo L, Liu K. A cross-sectional investigation on eating disorders in 1486 female students from universities, senior high schools and junior high schools in Chengdu. Chin J Epidemiol 2008; 29(4): 321-4.
[186] Lu X, Zhang N. Research on adolescents' eating behaviors and eating disorders. Master degree paper of Nanjing Normal University 2006; 2: 35-8.
[187] Qian M, Liu X. Dieting and eating disorder of female students in colleges in Beijing. Chin Ment Health J 2002; 16(11): 753-7.
[188] Xiao G, Qian M, Huon G, Wang Y. Rate of eating disorder in Beijing girls. Chin Ment Health J 2001; 15(5): 362-4.
[189] Fan Y, Li Y, Liu A, Hu X, Ma G, Xu G. Associations between body mass index, weight control concerns and behaviors, and eating disorder symptoms among non-clinical Chinese adolescents. BMC Public Health 2010; 10: 314.
[190] Burrows A, Cooper M. Possible risk factors in the development of eating disorders in overweight pre-adolescent girls. Inter J Obes 2002; 26(9): 1268-73.
[191] Fairburn C, Welch S, Doll H, Davies B, O'Connor M. Risk factors for bulimia nervosa: a community-based, case-control study. Arch Gen Psychiatry 1997; 54(6): 509-17.
[192] Fairburn C, Doll H, Welch S, Hay P, Davies B, O'Connor M. Risk factors for binge eating disorder: a community-based, case-control study. Arch Gen Psychiatry 1998; 55(5): 425-32.
[193] Striegel-Moore R, Fairburn C, Wilfley D, Pike K, Dohm F, Kraemer H. Toward an understanding of risk factors for binge-eating disorder in black and white women: a community-based case-control study. Psychol Med 2005; 35(6): 907-17.
[194] Bas M, Bozan N, Cigerim N. Dieting, dietary restraint, and binge eating disorder among overweight adolescents in Turkey. Adolescence 2008; 43(171): 635-48.
[195] Neumark-Sztainer D, Story M, Hannan P, Perry C, Irving L. Weight-related concerns and behaviors among overweight and nonoverweight adolescents implications for preventing weight-related disorders. Arch Pediatr Adolesc Med 2002; 156(2): 171-8.
[196] Neumark-Sztainer D, Wall M, Eisenberg M, Story M, Hannan P. Overweight status and weight control behaviors in adolescents: longitudinal and secular trends from 1999 to 2004. Prev Med 2006; 43(1): 52-9.
[197] Ge K, Weisell R, Guo X, *et al*. The body mass index of Chinese adults in the 1980s. Eur J Clin Nutr 1994; 48(Suppl 3): S148-54.

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