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Environmental risk factors contributing to childhood overweight and obesity

Środowiskowe czynniki ryzyka mające związek z nadwagą i otyłością dziecięcą

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ABSTRACT

The rising prevalence of overweight and obesity in children and teenagers is a major challenge for public health. Obesity is a complex and heterogeneous disorder, affected by many interacting genetic and non-genetic factors.

The aim of this article was to focus on the environmental risk factors for childhood obesity. Among different factors contributing to an increase in BMI, we highlighted the role of exposure to cigarette smoke, DDT, bisphenol A, pesticides, and noise. The correlation between exposure to environmental toxins during prenatal period and obesity development in later life was underlined. According to obesogenic environment hypothesis, some features of distal and proximal neighbourhood also have a pivotal impact on children's behaviour and may contribute to increasing the risk for overweight. The area of residence (urban or rural) may affect access to sports facilities or other opportunities for physical activity.

Therefore, for designing adequate prophylaxis, it is essential to take into account modifiable risk factors present in residential neighbourhood. Prevention of childhood obesity should integrate activities for both micro- and macro-environment surrounding the child.

Key words: environmental exposure, overweight, children, pediatric obesity

STRESZCZENIE

Wzrastająca częstość występowania nadwagi lub otyłości u dzieci i nastolatków stanowi poważne wyzwanie dla zdrowia publicznego. Otyłość to złożone i heterogenne zaburzenie wynikające z wielu interakcji pomiędzy czynnikami genetycznymi i pozagenetycznymi.

Celem artykułu było skupienie się na środowiskowych czynnikach ryzyka dziecięcej otyłości. Spośród różnorodnych czynników przyczyniających się do wzrostu BMI naświetliliśmy rolę ekspozycji na dym tytoniowy, DDT, bisphenol A, pestycydy oraz hałas. Podkreślony został związek pomiędzy narażeniem na środowiskowe toksyny w okresie prenatalnym a rozwojem otyłości w późniejszym życiu. Zgodnie z hipotezą obesogennego środowiska niektóre cechy dalszego i bliższego sąsiedztwa mają bardzo ważny wpływ na zachowania dzieci i mogą przyczynić się do wzrostu ryzyka nadwagi. Miejsce zamieszkania (wieś lub miasto) może warunkować dostępność do obiektów sportowych czy innych możliwości wykonywania aktywności fizycznej.

Dlatego też, niezwykle ważnym jest uwzględnienie przy projektowaniu odpowiedniej profilaktyki modyfikowalnych czynników ryzyka obecnych w lokalnym środowisku. Prewencja dziecięcej otyłości powinna integrować działania na rzecz mikro- i makrośrodowiska otaczającego dziecko

Słowa kluczowe: narażenie środowiskowe, nadwaga, dzieci, otyłość dziecięca

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INTRODUCTION

Childhood obesity has become a global epidemic and a pivotal public health problem. The prevalence of infant, childhood and adolescent overweight and obesity has risen dramatically not only in high-income but also in low- and middle-income countries [1, 2]. According to World Health Organization (WHO), in absolute numbers, more children are overweight and obese in low- and middle-income countries than in high-income countries [1].

Obesity is a chronic disease defined as abnormal or excessive fat accumulation which occurs as a result of imbalance between energy intake and output. Diagnosis is based on the child's weight-for-length/height or BMI, depending on the child's age and where it falls on the appropriate growth charts [1]. In children, BMI varies not only with weight but also with age. To find out whether the child is overweight or obese, it is essential to plot the child's BMI value on the BMI centile chart, which allows an assessment of BMI percentile according to the child's age and gender [1, 3].

In view of the rising prevalence of childhood obesity and related health consequences, the aim of this paper is to concentrate on environmental factors which contribute to the development of overweight and obesity in the child population. The focus is on environmental factors because of their importance as potentially modifiable determinants which should be taken into consideration in designing adequate prevention for both individuals and the whole population.

HEALTH CONSEQUENCES

Raised BMI is a major risk factor for hypertension, dyslipidaemia, hyperinsulinaemia, cardiovascular diseases, type-2 diabetes, and many cancers (e.g., colorectal cancer, kidney cancer, and oesophageal cancer) [1, 4–6]. The prevalence of pulmonary diseases, such as asthma or sleep-disordered breathing (sleep apnea), non-alcoholic fatty liver disease, and intracranial hypertension, is significantly higher in overweight and obese children [5]. Proteinuria and focal segmental glomerulosclerosis diagnosed in extremely obese adolescents may result in renal dysfunction [7]. Complications of childhood obesity include acceleration in the timing of thelarche and menarche in girls and pubertal advancement in boys, as well as adverse effects of early maturation on developing bones in both sexes [2]. The orthopedic disorders which seem to be more common in obese children are fractures, musculoskeletal discomfort, impaired mobility and lower-limb complaints (ankle and foot problems), tibia vara, and slipped capital femoral epiphyses [2, 8]. Apart from physical consequences, childhood obesity can contribute to behavioural and emotional difficulties, such as depression, stigmatisation, poor socialisation, social isolation, and a greater risk of teasing, bullying, and significant reductions in the quality of life [1, 4].

Childhood obesity is a strong predictor of obesity in adulthood [1], especially when it starts before the age of 5 years or after the age of 15 [9]. Prospective studies have demonstrated that obesity in adolescents (15–17 years of age) is connected with over seventeen times higher risk of adult obesity [5].

RISK FACTORS

Obesity is a complex and heterogeneous disorder that is affected by many interacting genetic and non-genetic factors [10]. It arises from a combination of exposure of the child to an unhealthy environment and inadequate behavioural and biological responses [1]. In the literature, risk factors of obesity are often divided into biological, environmental, socioeconomic, and behavioural (Table 1) [1, 11].

Table I. Risk factors associated with childhood obesity

Biological	Environmental	Socio-economic	Behavioural
genetics	environmental chemicals	socio-economic status	excessive calorie intake
brain-gut axis	noise	culture	eating patterns
prenatal determinants	built environment		sedentary lifestyle
gut microbiome			
viruses			
neuroendocrine conditions			
physical disability			

In most cases of obesity, biological susceptibility requires interaction with environmental factors. The aim of this paper is to concentrate on environmental risk factors connected with childhood obesity, primarily environmental pollutions and obesogenic environment.

Environmental toxins

Recent scientific evidence has shown that the imbalance between energy intake and energy expenditure does not fully explain the obesity epidemic [12]. The study conducted by Brown et al. over a period of nearly four decades drew a conclusion that "factors other than diet and physical activity may be contributing to the increase in BMI over time" [13]. These findings support the obesogen hypothesis that environmental pollutants may contribute to the obesity epidemic by altering the differentiation of adipocytes or the development of neural paths regulating feeding behavior [12, 14]. Many studies have demonstrated that maternal exposure during pregnancy to environmental pollutants is associated with childhood overweight [15, 16]. Considering the specific vulnerability of the early development period, the effects induced at that time are likely to be more profound or irreversible than those caused by the exposure occurring later in life [12].

A group of potentially obesogenic substances are endocrine disrupting chemicals (EDCs), which increase BMI probably by interrupting the homeostatic control of adipogenesis [16]. Many studies focus on environmental exposure to one of the EDCs - a nonsteroidal xenoestrogen bisphenol A (BPA). It is a substance mainly used in the production of polycarbonate plastics or as an additive in other plastics from which food containers or bottles are made, including baby polycarbonate bottles [16, 17]. Human exposure occurs mainly through consumption of food or liquids (increasing temperature or acidic pH of liquids may result in BPA leaching into food or beverage) [17]. The fetus can have contact with BPA due to maternal exposure, while in newborns the potential sources of BPA are breast milk and tinned food [16]. In animal models, exposure to BPA is related to high lipid levels, increased levels of insulin and serum markers of oxidative stress and inflammation, which are mechanisms connected with the development of obesity [18]. Mackay et al. reported a mechanism in which exposure to BPA could lead to increased food intake due to changes in the brain resulting in stimulating appetite [19]. Shankar et al. observed a positive association in adults between increasing levels of urinary BPA and obesity, independent of potential confounding factors, including smoking, alcohol consumption and serum cholesterol levels [18]. By contrast, Braun et al.'s results did not confirm that prenatal and early-childhood BPA exposures were associated with increased BMI; however, they did demonstrate that higher early-childhood BPA exposures were associated with accelerated growth during this period [20].

There is a positive association between childhood overweight and prenatal exposure to several other environmental chemicals, including dichlorodiphenyldichloroethylen (DDE), dichlorodiphenyltrichloroethane (DDT), hexachlorobenzene, phthalates, perfluorooctanoic acid (PFOA), dioxins, tributyltin and polycyclic aromatic hydrocarbon [15]. Research conducted by La Merrill et al. showed that exposure to DDT during pregnancy could reduce the basal metabolism in the offspring and thus explain why these offspring gained extra weight for a given energy intake [21]. Another widely discussed problem is developmental exposure to PFOA (used for manufacturing plastic, waterproof materials, food containers – e.g., containers for popcorn or non-stick pan coatings) [17]. Although there are insufficient data to prove their toxicity in humans, the correlation between PFOA exposure in mice and obesity has been noted. The association between low-doses of PFOA exposure during prenatal period in humans and the development of obesity in female offspring at the age of 20 has also been observed [17].

The possible role of pesticides in elevating the risk for obesity remains unclear. Although some active ingredients of pesticides, especially insecticides, may affect glucose homeostasis by altering neurotransmitter and ion channel systems which are involved in regulating pancreatic function, there is much less evidence that pesticides might affect adiposity or other components for metabolic syndrome [14].

Worth pointing out is the role of nicotine and other components in cigarette smoke (e.g., polycyclic aromatic hydrocarbons) which may contribute to overweight or obesity [14, 22]. Maternal prenatal tobacco smoking and exposure to secondhand smoke (SHS) in children may increase the risk for obesity [14, 15, and 22]. The agents in cigarette smoke are suspected endocrine disruptors, which can negatively affect the utilization of insulin and promote metabolic imbalance. Furthermore, exposure to SHS is associated with inflammation and systemic oxidative stress, which could play a role in the development of obesity [22]. Wen et al. observed a positive relationship between prevalence of obesity

among children at 7 years of age and maternal smoking during pregnancy (more than 20 cigarettes a day) [23]. Similarly, a study conducted by Apfelbacher et al. among children between the ages of 5–7 years revealed that parental self-report of household smoking was associated with childhood obesity [24]. Moore et al.'s findings suggest that the prevalence of obesity among children with both high exposure to SHS and low levels of dietary fiber, eicosapentaenoic acid or docosahexaenoic acid is greater than among children exposed to one of those factors alone [22].

Environmental noise

Noise, which can be classified as an environmental pollutant, is another obesogen that merits consideration. Exposure to residential noise leads to extra-aural health effects, such as sleeping disturbances and cardiovascular, hormonal, and metabolic disorders [25, 26]. Although its potential role as a risk factor for obesity has been investigated, there is still need for more research. The ways in which noise can affect body weight include hormonal changes connected with stress reaction and sleep disturbances [25, 27]. In adults, exposure to noise has been associated with higher BMI, waist circumference and increased prevalence of diabetes mellitus type 2 or prediabetic states [25, 26]. However, there are only limited studies describing the correlation of noise and obesity in children, who may be an especially vulnerable group. According to Christensen et al., exposure to environmental noise during pregnancy and childhood was positively associated with childhood overweight [27]. Both prenatal and postnatal exposure to road traffic noise resulted in 6% higher risk of childhood overweight per 10 dB [27]. In the same study, there was no significant correlation between road traffic noise and BMI z-scores among children, nor between railway noise and adiposity [27].

Obesogenic environment

WHO defines obesogenic environment as an environment that promotes high energy intake and sedentary behaviour [1]. In 1999, Swinburn was the first to suggest the complex and multi-dimensional idea of obesogenic environment as "excessive weight not as the result of a single cause but as the significant effect of environment on nutrition and physical activity." Therefore, prevention should focus on reduction of obesogenic factors [9]. Swinburn divided obesogenic environment into micro-environment (school, workplace, home, neighborhood) and macro-environment (education and health systems, government policies, societal involvement, cultural belief structure) [28]. Another concept was suggested by Nesbit et al., who indicated groups of proximal (home) and distal (neighborhood) environmental correlates of obesity, characterised by specific physical and social properties. The proposed physical features of the proximal environment include the space, materials and resources at home, while community structures, such as sidewalks, parks, playgrounds and recreation centers, are physical features of the distal environment [29,30].

Urbanisation is one of the most important aspects of an obesogenic environment. Most studies have shown that especially the environment around a child's home is related to childhood obesity [9]. The area of living (urban vs. rural) may affect access to sports facilities and opportunities for physical activity. Lack of play areas or areas suitable for walking or cycling results in children preferring to stay indoors, playing computer games or watching television, rather than play outside. Obviously, this inactive lifestyle increases the risk for overweight [9]. Gurzkowska et al. reported that in Poland, overweight and obesity were significantly more frequent among school pupils in urban than in rural areas [31].

Although there are many elements contributing to one's physical activity, some of them seem to be crucial. The characteristic environmental features influencing the decision to walk are well presented in the Hierarchy of Walking Needs model, which indicates five levels of needs; according to it, basic needs have to be fulfilled to take higher-order needs into consideration in a decision making process [32]. For the purpose of this paper, we have adapted this model to propose a hierarchy of being active needs, as a theoretical explanation for the possible impact of environmental factors on physical activity behaviours in general (illustrated in Fig. 2). In this article, we concentrate on basic needs..

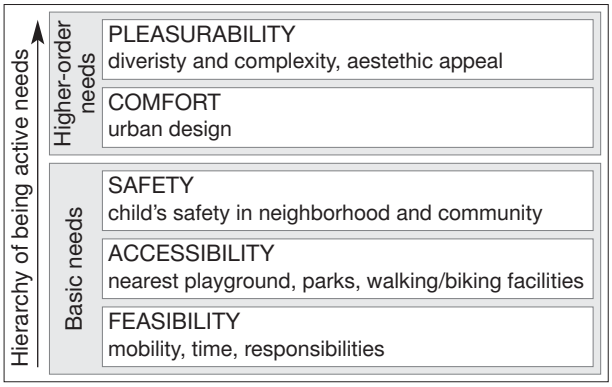

Fig. 1. Illustration of being active needs model (own work based on Alfonzo's hierarchy of walking needs model [32])

Although there are limited data connected with feasibility, both accessibility and safety have been analysed in many studies. According to Taylor et al., there is a significant negative correlation between accessibility and the prevalence of obesity in children [33]. While Nesbit et al. also demonstrated a significant relation between access to physical activity and decreasing risk of obesity in early adolescents (11-14 years), this connection was insignificant for middle adolescents (15-17 years) [30]. The same study revealed differences in the associations between access to physical activity and a reduction in the risk for obesity according to gender; namely, a significant association was observed in boy adolescents but not in girl adolescents [30]. In contrast, Liu et al. found no differences in children BMI status and the average distance to play areas [34]. In a survey conducted among children aged 3 and 4 years, Burdette and Whitaker found no association between proximity to playgrounds, proximity to fast-food restaurants, or neighborhood crime and the risk of overweight [34].

Safety is another pivotal factor affecting the parental decision to allow their child to play outside rather than keep them at home. A cross-sectional survey of children aged 10 to 12 years conducted by Timperio et al. demonstrated that parental worries about road safety (stranger danger or crossing several roads to reach playgrounds) reduced walking or cycling among their children [35]; therefore, those children were more likely to be obese [29]. Also Lumeng et al.'s findings confirmed the role of parental perception of the neighborhood as less safe in increasing the risk of overweight in their children (in a group of 7-year-olds). The association was not affected by the region of residence or the socioeconomic status [36].

CONCLUSIONS

The pathogenesis of obesity is complex and comprises many heterogeneous elements. The rising prevalence of obesity and overweight in children is an urgent challenge because health consequences of childhood obesity affect not only the period of their growth and development but also their adulthood. Among different factors contributing to an increase in BMI, we highlighted the role of pollutants and characteristic features of the local neighbourhood. Although the correlations between environment and obesity have been the subject of several studies, it is necessary to expand the knowledge of the mechanisms in which environmental risk factors increase individual risk for obesity. The identification of modifiable elements in human communities is essential for designing adequate prophylaxis. In the process of planning and designing proper solutions, specific residential features and conditions should be taken into consideration. Prevention focused on reducing the exposure to environmental obesogen chemicals or noise and minimising the effect of the physical environment properties which increase the risk for obesity should integrate activities for both micro- and macro-environment surrounding the child.

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