

<https://doi.org/10.17816/PED11233-42>

THE ROLE OF HUMAN CONSTITUTIONAL TYPE IN FORMING OF BODYMASS EXCESS AND DEFICITE IN CHILDREN OF VARIOUS AGES

© V.O. Erkudov¹, A.P. Pugovkin¹, A.J. Volkov², O.I. Musaeva², T.N. Slyaptsova³, M.V. Chistyakova³, S.S. Rogozin², M.A. Pakhomova¹, A.A. Kravtsova¹

¹ St. Petersburg State Pediatric Medical University, Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia;

² Saint Petersburg District Polyclinic No. 109, Children's Polyclinic department No. 3, Saint Petersburg, Russia;

³ School No. 225 of Admiralteysky District of Saint Petersburg, Saint Petersburg, Russia

For citation: Erkudov VO, Pugovkin AP, Volkov AJ, et al. The role of human constitutional type in forming of bodymass excess and deficit in children of various ages. *Pediatrician (St. Petersburg)*. 2020;11(2):33-42. <https://doi.org/10.17816/PED11233-42>

Received: 10.02.2020

Revised: 23.03.2020

Accepted: 24.04.2020

Nowadays there are no decisive descriptions of constitutional features and body mass deviations in a narrow group taking into consideration their sex, age, habitation region, and a concrete type of body mass deficit or excess. The present study focuses at comparative assessment of number and frequency of pronounced deficit, deficit, nominally normal body mass, preobesity and obesity in junior schoolchildren and adolescents with lepto-, meso- and hypersomal somatotype. All in all 274 persons took part in the study, 130 of junior schoolchildren aged 7 to 9 years (64 boys and 66 girls) as well as 142 adolescents 14–17 years old (65 boys and 77 girls). Somatotype was determined in all children with the help of I.I. Salivon and V.A. Melnik's method, as well as body mass index was calculated. Comparing the number of children with various deviations of body mass in case of lepto- meso- and hypersomal somatotype was accomplished with the help of precise Fisher's criterion for conjugated features tables 3 × 5. In boys and girls of junior school age with leptosomal (boys – 62%, girls – 81%) somatotype body mass deficit was predominant, while in adolescents with leptosomal somatotype body mass deficit was found in 37% boys and 15% of girls. Hypersomal boys form obesity and excessive body mass when they enter adolescence. On the other hand 40% girls of junior school age with hypersomal somatotype are already obese and 100% of them preserve obesity till puberty. The results of the study may be useful for specifying individual recommendations for children with metabolic pathology at pre-hospital stage for correction of both excess and deficit of body mass should be made prior to entering puberty.

Keywords: constitution type; somatotype; body mass deficit; body mass excess; children.

РОЛЬ КОНСТИТУЦИИ ЧЕЛОВЕКА В ФОРМИРОВАНИИ ДЕФИЦИТА И ИЗБЫТКА МАССЫ ТЕЛА У ДЕТЕЙ РАЗЛИЧНОГО ВОЗРАСТА

© В.О. Еркудов¹, А.П. Пуговкин¹, А.Я. Волков², О.И. Мусаева², Т.Н. Сляпцова³, М.В. Чистякова³, С.С. Рогозин², М.А. Пахомова¹, А.А. Кравцова¹

¹ Федеральное государственное бюджетное образовательное учреждение высшего образования «Санкт-Петербургский государственный педиатрический медицинский университет» Министерства здравоохранения Российской Федерации, Санкт-Петербург;

² Санкт-Петербургское государственное бюджетное учреждение здравоохранения «Городская поликлиника № 109», детское поликлиническое отделение № 3, Санкт-Петербург;

³ Государственное бюджетное образовательное учреждение средняя общеобразовательная школа № 225 Адмиралтейского района Санкт-Петербурга

Для цитирования: Еркудов В.О., Пуговкин А.П., Волков А.Я., и др. Роль конституции человека в формировании дефицита и избытка массы тела у детей различного возраста // Педиатр. – 2020. – Т. 11. – № 2. – С. 33–42. <https://doi.org/10.17816/PED11233-42>

Поступила: 10.02.2020

Одобрена: 23.03.2020

Принята к печати: 24.04.2020

В настоящее время характеристики взаимоотношений конституции человека и отклонений массы тела в узкой группе испытуемых с учетом их пола, возраста, региона проживания и конкретного типа дефицита или избытка веса тела не описаны достаточно полно. Цель данной работы – сравнительный анализ количества и определение частоты встречаемости выраженного дефицита, дефицита условно нормальной массы тела, предожирения и ожирения у детей младшего школьного возраста и подростков с лепто-, мезо- и гиперсомным соматотипом.

Методы. Всего в работе приняли участие 274 человека. Из них 130 детей младшего школьного возраста от 7 до 9 лет (64 мальчика и 66 девочек), а также 142 подростка в возрасте от 14 до 17 лет (65 мальчиков и 77 девочек). Всем детям определяли соматотип по методике И.И. Саливон и В.А. Мельник и рассчитывали индекс массы тела. Сравнение количества детей с различными отклонениями массы тела с лепто-, мезо- и гиперсомным телосложением проводилось с применением точного критерия Фишера для таблиц сопряженности признаков 3×5 . **Результат.** У мальчиков и девочек младшего школьного возраста с лептосомным (мальчики – 90%, девочки – 76%) и мезосомным (мальчики – 62%, девочки – 81%) типом телосложения выявлен преимущественно недостаток массы тела, а в подростковом возрасте это отклонение имеет место у 37% юношей и 15% девушек с лептосомным соматотипом. Испытуемые мужского пола с гиперсомным типом конституции формируют ожирение и избыток массы тела только к подростковому возрасту. При этом 40% девочек школьного возраста с данным соматотипом имеют ожирение уже в младшем школьном возрасте, которое в 100% случаев сохраняется до пубертатного периода. **Выводы.** Полученные результаты могут быть полезны для составления индивидуальных рекомендаций по ведению детей с патологией обмена веществ на догоспитальном этапе, поскольку коррекцию избытка и недостатка массы тела необходимо осуществить до вступления ребенка в процесс полового созревания.

Ключевые слова: конституция человека; соматотип; дефицит массы тела; избыток массы тела; дети.

INTRODUCTION

There are numerous factors that can influence body weight. Obesity and low body weight are general medical problems that require detailed study [2, 15, 22, 32]. At that, taking into account the polyetiological nature of these problems, the idea of finding out the relation of somatotype and deviation of body weight remains incomplete. The human constitution is an individual typological marker of the functional and morphological properties of the human body. It not only determines the shape and physical appearance of an individual, but it is also associated with body's functional reactivity [28], the anatomical structure of internal organs [8], and the blood system [9]. The somatotype is undoubtedly associated with obesity; overweight is a characteristic of hypersthenic type of the human constitution [13]. This fact was confirmed by us in modern works [10]. However, the relationship between the human constitution and deviation in body weight remains poorly understood. The results of such studies can be useful in relation to the creation and development of the principles of dynamic monitoring of the physical condition of contemporary schoolchildren of different ages in normal and in metabolic pathologies [13, 14].

This study was performed to do a comparative analysis of the level and determination of the incidence of severe deficit, deficit of conditional normal body weight, preobesity, and obesity in primary school children and adolescents with leptosomal, mesosomal, and hypersomal somatotypes.

MATERIALS AND METHODS

The study was performed during a routine preventive examination of children, which was con-

ducted in accordance with order No. 514n dated 08/10/2017 "On the procedure for conducting preventive medical examinations of minors"¹ in the children's outpatient department No. 3 of St. Petersburg City Polyclinic No. 109. All study participants signed the Voluntary Informed Consent for Routine Examinations and the Processing of Personal Data. A total of 274 patients took part in the study, 130 of them were children of primary school age from 7 years to 9 years 11 months and 29 days (64 boys and 66 girls) and 142 adolescents aged 14 years to 17 years 11 months and 29 days (65 boys and 77 girls). Somatotype of all children was calculated using the method proposed by I.I. Salivon and V.A. Melnik [20]. The application of this method involves the determination of the leptosomal, mesosomal, and hypersomal body types based on 12 anthropometric parameters. Apical body length was evaluated by using a floor-mounted medical stadiometer RM2- "Diacoms" (Diacoms, Russia) with an accuracy of up to 5 mm. Body weight was measured by VEM150- "Massa-K" electronic medical scales (Massa-K, Russia) with an accuracy of 50–150 g depending on the load. The overall dimensions of the chest (transverse and sagittal) were measured by using a spreading caliper (Argentum, Russia) with the accuracy of up to 1 mm. The thickness of the skin fat folds was measured by using calipers (Slim Guide Caliper, China) with a pistol grip and a graduating spring to create equal pressure on both sides of the fat fold (10 g/mm^2) with an accuracy of up to 0.5 mm. Circumference in the narrowest places of the forearm,

¹ Order of the Ministry of Health of the Russian Federation dated August 10, 2017 No. 514n "On the Procedure for Conducting Preventive Medical Examinations of Minors" with amendments and additions of July 3, 2018, June 13, 2019.

above the wrist, and lower leg, above the ankles, was measured with a sliding caliper (Argentum, Russia) with an accuracy of up to 1 mm. The width of the epiphyses of the shoulder and thigh was measured using a measuring tape.

Body mass index (BMI) was calculated in all patients, which is a generally accepted method for determining the type of deviation in body weight [7, 30, 32]. A BMI of 15.99 or less was regarded as a pronounced body mass deficit; the value of 16–18.49 indicated body mass deficit (BMD); 18.5–24.99 was conditional norm; 25–29.99 indicated preobesity; 30 or more indicated obesity [30, 32].

Deviations in body weight were determined by BMI. Comparison of the frequency of occurrence of various somatotypes in primary school children with adolescents was done by assessing the uniform distribution of various somatotypes in children of different age groups, as well as the distribution of severe deficit, deficit of normal body weight and overweight, and obesity in children with lepto-, meso-, and hypersomal physique. For this, the Fisher exact test was used for tables of contingency of signs 3×5 and 3×2 with the calculation of the proportion of children with one or another deviation of body weight. The results were considered statistically significant for $p < 0.05$. The calculations were performed using the integrated Excel functions from the Microsoft Office 2010 application package and the StatXact-8 statistical data processing algorithm with the Cytel Studio version 8.0.0 software shell [28, 31]. All data are presented

as the mean values of the fraction of body mass deviation, and the upper and lower bounds for the 96% confidence interval for body mass deviation (μ [L.L.; U.L. 95% CI]) are indicated.

RESULTS AND DISCUSSION

An analysis of the data showed that the distribution of various somatotypes in primary school children and adolescents is uniform and does not differ in boys and girls (Table 1), as indicated by the obtained p values. This means that quantitative differences in children with lepto-, meso-, and hypersomal somatotypes were not included in this study.

The distribution of body mass deviations determined by BMI is not uniform, and differs in boys (Table 2) and girls (Table 3) of both primary school age and adolescents with different somatotypes. Consequently, the presence of a pronounced deficit or deficit of body mass, normal weight, or overweight, as well as obesity depends on the type of physique and is significantly different in children with different constitution types at a chosen level of significance.

This work presents the constitutional features of the distribution of various deviations of body weight in children of different ages. Low body weight was found in boys and girls of primary school age with leptosomal (boys 90%, girls 76%) and mesosomal (boys 62%, girls 81%) body types. In adolescents, with a leptosomal somatotype, this deviation occurs in 37% of boys and 15% of girls. In boys with hypersomal body types, obesity and

Table 1 / Таблица 1
Prevalence of somatotypes in primary school boys and girls and male and female teenagers (μ (L.L.; U.L. 95% CI))
Распространенность различных типов телосложения у мальчиков и девочек младшего школьного и подросткового возраста (μ (L.L.; U.L. 95% CI))

Somatotype / Соматотип	Leptosomal / Лептосомный	Mesosomal / Мезосомный	Hypersomic / Гиперсомный
Prevalence of somatotypes in boys / Распространенность различных типов телосложения у мальчиков			
Primary school boys / Младший школьный возраст	0.47 (0.32; 0.62)	0.45 (0.31; 0.60)	0.08 (0.02; 0.19)
Male teenagers / Подростки	0.57 (0.42; 0.72)	0.34 (0.20; 0.49)	0.09 (0.03; 0.20)
Prevalence of somatotypes in girls / Распространенность различных типов телосложения у девочек			
Primary school girls / Младший школьный возраст	0.52 (0.36; 0.66)	0.41 (0.27; 0.56)	0.08 (0.02; 0.19)
Female teenagers / Подростки	0.70 (0.57; 0.82)	0.26 (0.15; 0.39)	0.04 (0.007; 0.12)

Note. The distribution of different somatotypes: in boys — $p = 0.3969$; in girls — $p = 0.0705$. *Примечание.* Распределение различных соматотипов: у мальчиков — $p = 0.3969$; у девочек — $p = 0.0705$.

Table 2 / Таблица 2

Body mass deviations, determined by BMI in boys of primary school and teenagers with different somatotypes (μ (L.L.; U.L. 95% CI))

Отклонения массы тела, определяемые по индексу массы тела, у мальчиков младшего школьного и подросткового возраста с различными соматотипами (μ (L.L.; U.L. 95% CI))

Body weight deviation / Отклонение массы тела	Severe underweight / ВДМТ	Body weight deficiency / ДМТ	“Normal” body weight / «Нормальная» масса тела	Overweight / Избыточная масса тела	Obesity / Ожирение
Distribution of body mass deviation in boys of primary school with different somatotypes / Распределение отклонения массы тела у мальчиков младшего школьного возраста с различными соматотипами					
Leptosomic / Лептосомный					
Leptosomic / Лептосомный	0.68 (0.44; 0.85)	0.23 (0.08; 0.46)	0.097 (0.01; 0.31)	0 (0; 0.15)	0 (0; 0.15)
Mesosomic / Мезосомный	0.25 (0.08; 0.46)	0.37 (0.16; 0.61)	0.40 (0.20; 0.65)	0 (0; 0.16)	0 (0; 0.16)
Hypersomic / Гиперсомный	0 (0; 0.60)	0 (0; 0.60)	1.00 (0.40; 1.00)	0 (0; 0.60)	0 (0; 0.60)
Distribution of body mass deviation in male teenagers with different somatotypes / Распределение отклонения массы тела у юношей подросткового возраста с различными соматотипами					
Leptosomic / Лептосомный					
Leptosomic / Лептосомный	0.02 (0.0002; 0.15)	0.35 (0.18; 0.55)	0.63 (0.43; 0.79)	0 (0; 0.10)	0 (0; 0.10)
Mesosomic / Мезосомный	0 (0; 0.19)	0.05 (0.0005; 0.27)	0.81 (0.55; 0.96)	0.14 (0.02; 0.40)	0 (0; 0.19)
Hypersomic / Гиперсомный	0 (0; 0.54)	0 (0; 0.54)	0 (0; 0.54)	0.83 (0.29; 0.998)	0.17 (0.002; 0.71)

Note. Distribution of body mass deviation in boys of primary school with different somatotypes: $p = 0.0001532$. Distribution of body mass deviation in teenagers with different somatotypes $p = 2.811 \cdot 10^{-6}$. Примечание. Распределение отклонения массы тела у мальчиков младшего школьного возраста с различными соматотипами: $p = 0,0001532$. Распределение отклонения массы тела у юношей подросткового возраста с различными соматотипами: $p = 2,811 \cdot 10^{-6}$. ВДМТ — выраженный дефицит массы тела, ДМТ — дефицит массы тела.

excess body weight are found only by adolescence. At the same time, 40% of school age girls with hypersomal somatotype are obese even in primary school age, and remain so until the puberty in 100% of cases.

In a child younger than 6–7 years of age, the type of physique cannot be defined with sufficient reliability [16]. The results obtained in this work indicate the relative stability of the physique, as the distribution of lepto-, meso-, and hypersomal somatotypes is the same both in primary school children and adolescents. Since the formation of the mechanism of regulation of the mass condition and its relation to the human physique occurs by puberty, the correction of deviations in body weight in a child must be performed by 11–12 years of age [13].

Special studies identify a significant deficit of motor activity in modern schoolchildren, since adolescents spend about 18–20 hours a day either sitting or lying down [4, 13, 32]. Moreover, they tend to overestimate their motor activity [32]. Such a lifestyle will certainly lead to obesity. Evidence

has shown that greater rates of television viewing are directly associated with a higher risk of being overweight or obese [13]. Overweight children can perform fewer movements than their non-obese peers [13]. Thus, the most important aspect of raising children of primary school age and preventing them from becoming obese, especially girls with a hypersomal somatotype, is the correct organization of physical activity when planning the day regimen.

The following factors are responsible for rapid weight gain in an individual: a high content of fats and carbohydrates in the diet [1, 32], predominance of night and evening meals [5, 32], an incorrect estimate of food volumes [32], a hyperphagic reaction to psycho emotional stress (up to 8000 kcal/day) with an increase in carbohydrate intake [5]. It should be noted that overfeeding children with protein foods during the formation of adipose tissue functions leads to an increase in the level of amino acids in the blood, hyperinsulinemia, and, as a result, increased proliferation of adipocytes [6, 30, 23, 24]. The results of this work can be an indirect

Table 3 / Таблица 3

Body mass deviations. determined by BMI in girls of primary school and teenagers with different somatotypes (μ (L.L.; U.L. 95% CI))

Отклонения массы тела. определяемые по индексу массы тела. у девочек младшего школьного и подросткового возраста с различными соматотипами (μ (L.L.; U.L. 95% CI))

Body weight deviation / Отклонение массы тела	Severe underweight / ВДМТ	Body weight deficiency / ДМТ	“Normal” body weight / «Нормаль- ная» масса тела	Overweight / Предожирение	Obesity / Ожирение
Distribution of body mass deviation in girls of primary school with different somatotypes / Распределение отклонения массы тела у девочек младшего школьного возраста с различными соматотипами					
Leptosomal / Лептосомный	0.47 (0.26; 0.68)	0.29 (0.13; 0.52)	0.24 (0.09; 0.45)	0 (0; 0.14)	0 (0; 0.14)
Mesosomal / Мезосомный	0.37 (0.17; 0.62)	0.44 (0.22; 0.70)	0.19 (0.05; 0.42)	0 (0; 0.17)	0 (0; 0.17)
Hypersomic / Гиперсомный	0.20 (0.002; 0.78)	0 (0; 0.60)	0.40 (0.03; 0.89)	0.40 (0.03; 0.89)	0 (0; 0.60)
Distribution of body mass deviation in female teenagers with different somatotypes / Распределение отклонения массы тела у девушек подросткового возраста с различными соматотипами					
Leptosomal / Лептосомный	0.04 (0.003; 0.15)	0.11 (0.03; 0.26)	0.82 (0.65; 0.92)	0.04 (0.003; 0.15)	0 (0; 0.09)
Mesosomal / Мезосомный	0 (0; 0.22)	0.05 (0.0005; 0.30)	0.85 (0.56; 0.98)	0.10 (0.009; 0.37)	0 (0; 0.22)
Hypersomic / Гиперсомный	0 (0; 0.78)	0 (0; 0.78)	0 (0; 0.78)	0.33 (0.003; 0.94)	0.67 (0.06; 0.997)

Note: Distribution of body mass deviation in girls of primary school with different somatotypes: $p = 0.0137$. Distribution of body mass deviation in female teenagers with different somatotypes: $p = 0.011$. Примечание. Распределение отклонения массы тела у девочек младшего школьного возраста с различными соматотипами: $p = 0.0137$. Распределение отклонения массы тела у девушек подросткового возраста с различными соматотипами: $p = 0.011$. ВДМТ — выраженный дефицит массы тела. ДМТ — дефицит массы тела.

evidence of the role of rational nutrition in primary school children to prevent the development of body mass deficit in adolescents with leptosomal physique and obesity in children with hypersomal somatotype.

The development of physique-dependent obesity undoubtedly has a genetic component [1, 5, 13, 22, 24–26]. It has long been known [13] and confirmed by contemporary studies [24] that when both parents are obese, their children have an 80 percent chance of obesity [24]. Now, more than 1000 genes associated with obesity are identified that create a threshold effect, as the disease occurs when the minimum critical level of their interaction is reached [24]. It is generally accepted that gene polymorphism of the leptin receptor (*LEPR*) [2, 11, 18, 21, 29, 33] and, to a lesser extent, proopiomelanocortin (*MC4R*) [18] represent a specific marker of this pathology. According to modern concepts, the role of genetic factors in the formation of somatotype is undeniable [36]. In addition, a relationship between *LEPR* and *MC4R* polymorphism and the human somatotype was revealed [35]. Therefore, it

is possible to assume that in children with obesity and a hypersomal body type, the *LEPR* receptor becomes insensitive to leptin, which reduces the excitability of appetite centers in the hypothalamus, activates a sympathetic effect on adipose tissue, promotes the fat cleavage in adipocytes, and causes their apoptosis [22, 29]. Accordingly, they have hyperleptinemia, which correlates with BMI [21, 35] and has gender differences; in girls it progressively increases during puberty, and in boys it is compensated during puberty [33].

As described previously, the somatotype is determined based on the measurement of 12 anthropometric parameters, one of which is the skin fat fold thickness. Accordingly, the volume of adipose tissue is a constitutionally dependent sign [13], including the uniformity of adipose and connective tissue, which, according to A.A. Bogomoltz, defines the type of physique [3]. The increase of adipose tissue during primary obesity in the somatotype associated with it can be caused by the combined effect of enhanced expression of the PPAR- γ adipocyte differentiation gene [30] and

impaired regulation and functions of insulin [13]. An increase in the effect of PPAR- γ , as a rule, develops according to the compensation mechanism due to fetoplacental insufficiency in the perinatal period of development [30].

The sensitivity of fat cells to insulin is inversely proportional to their size, which links the somatotype and insulin resistance and, as a consequence, hyperinsulinism results [13]. An increase in the concentration of blood insulin leads to hypertrophy of adipocytes without any increase in their number [13]. At the endothelium of the capillaries of the adipose tissue microvasculature, it is excessively active in individuals with primary obesity and the corresponding tissue lipase somatotype [13, 22]. Its activity does not reduce after weight loss [22]. It is shown that there is an association of tissue lipase gene polymorphism with excess body weight [22]. This enzyme cleaves lipoproteins and chylomicrons with the formation of unesterified fatty acids which compete with glucose for transport systems in skeletal muscles [13, 22]. Hyperglycemia leads to an increase in insulin concentration, which regulates the formation of new adipocytes, and hyperplastic obesity develops [13, 22].

With overweight and, probably, a hypersomal somatotype, the activity of adipose tissue lipase reduces [22], its reactivity to adrenaline and sympathetic effects also reduces, probably due to polymorphism of genes that control β_3 -adrenoreceptors of adipose tissue, leading to changes in their function [5].

Weight deficit in children is significantly associated with a number of chronic diseases [12, 17, 19, 27], and it is a kind of "marker" for their development in the adults [34]. For example, gastrointestinal tract pathology occurs in case of underweight in 80% of cases [12].

This study revealed that children of different ages with leptosomal physique have low body weight, which is compliant with the literature [17, 19]. This fact can be explained by the notion of asthenia (leptosomy) as a marker of slow growth and development of a person [14], in contrast to hypersthenia (hypersomy), which indicates increased anabolic processes [28]. These data may be relevant for predicting the susceptibility of a child to diseases in puberty. In addition, these results can be useful for making individual recommendations for the management of such children at the prehospital stage, since the correction of excess body weight must be performed before the child enters the process of puberty. In addition, weight

deficit is significant for monitoring the health status of young men by doctors of the preconscription commission [12].

CONCLUSIONS

- Based on the results of this work, it can be assumed that somatotyping in children of primary school age provides the possibility to predict with a high degree of probability the development of primary obesity or deficit of body weight in the pubertal period.
- The relationship of a certain deviation of body weight in the examined adolescents and children of primary school age with the type of physique has been proved.
- The hypersomal somatotype in a child in the prepubertal period enables to predict the development of excess body weight in his adolescence and determine with a high degree of probability his lifestyle, metabolic characteristics, and his genetic and hormonal status.
- A tendency to underweight is the characteristic of children with leptosomal body type, especially young men.

REFERENCES

- Бардымова Т.П., Михалева О.Г., Березина М.В. Современный взгляд на проблему ожирения // Acta Biomedica Scientifica. – 2011. – № 5. – С. 203–206. [Bardymova TP, Mikhaleva OG, Berezina MV. Modern View On The Problem Of Obesity. Bull Vost Sib Nauch Sent. 2011;(5):203-206. (In Russ.)]
- Беляева И.А., Намазова-Баранова Л.С., Турти Т.В., и др. Значение грудного вскармливания в профилактике отдаленных нарушений метаболизма: обзор литературы // Педиатрическая фармакология. – 2015. – Т. 12. – № 1. – С. 52–58. [Belyaeva IA, Namazova-Baranova LS, Turti TV, et al. Role of Breastfeeding in Preventing Long-Term Metabolic Disorders: Review. Pediatric pharmacology. 2015;12(1):52-58. (In Russ.)]
- Богомолец А.А. Введение в учение о конституциях и диатезах. – М.: Издательство Наркомздрава РСФСР, 1928. – 230 с. [Bogomolets AA. Vvedenie v uchenie o konstitutsiyakh i diatezakh. Moscow: Izdatel'stvo Narkomzdrava RSFSR; 1928. 230 p. (In Russ.)]
- Бокарева Н.А., Милушкина О.Ю., Овчинникова З.А., и др. Гигиеническая оценка влияния организаций образовательного процесса на физическое развитие школьников г. Москвы // Вестник Российского государственного медицинского университета. – 2016. – № 3. – С. 63–69. [Bokareva NA, Milushkina OYu, Ovchinnikova ZA, et al. Impact of learning environments on the physical development of Mos-

- cow schoolchildren: hygiene aspects. *Bulletin of RSMU*. 2016;(3):63-69. (In Russ.)]
5. Григорьев К.И., Князев Ю.А. Ожирение – теория и практика. Техника медицинского работника // Медицинская сестра. – 2006. – № 8. – С. 2–7. [Grigor'ev KI, Knyazev YuA. Ozhirenje – teoriya i praktika. Taktika meditsinskogo rabotnika. *Med Sestra*. 2006;(8):2-7. (In Russ.)]
 6. Денисов М.Ю., Коваренко М.А., Петрусенко О.И., Шведкина Е.Ю. Оценка взаимосвязи некоторых факторов риска развития ожирения у детей раннего возраста // Вестник Новосибирского государственного университета. Серия: Биология, клиническая медицина. – 2012. – Т. 10. – № 1. – С. 115–121. [Denisov MYu, Kovarenko MA, Petrusenko OI, Shvedkina EYu. Estimation of interrelation of some risk factors of development of obesity at children of early age. *Vestnik NGU. Serija biologija, klinicheskaja meditsina*. 2012;10(1):115-121. (In Russ.)]
 7. Еркудов В.О., Пуговкин А.П., Волков А.Я., и др. Конституциональное разнообразие размеров внутренних органов у подростков // Российский вестник перинатологии и педиатрии. – 2019. – Т. 64. – № 2. – С. 94–99. [Erkudov VO, Pugovkin AP, Volkov AYa, et al. Constitutional diversity in the dimensions of internal organs of teenagers. *Russian Bulletin of Perinatology and Pediatrics*. 2019;64(2):94-99. (In Russ.)]. <https://doi.org/10.21508/1027-4065-2019-64-2-94-99>
 8. Еркудов В.О., Скрипченко Н.В., Заславский Д.В., и др. Значение конституциональных факторов в развитии дефицита и избытка массы тела у подростков // Вопросы практической педиатрии. – 2019. – Т. 14. – № 4. – С. 21–29. [Erkudov VO, Skripchenko NV, Zaslavskiy DV, et al. Role of constitutional factors in the development of underweight and overweight in adolescents. *Problems of practical pediatrics*. 2019;14(4):21-29. (In Russ.)]
 9. Еркудов В.О., Волков А.Я., Пуговкин А.П., Мусаева О.И. Конституциональные особенности клеточного состава крови у подростков и юношей // Морфология. – 2018. – Т. 154. – № 5. – С. 50–56. [Erkudov VO, Volkov AYa, Pugovkin AP, Musaeva OI. Constitutional Characteristics Of The Blood Cell Composition In Male Teenagers. *Morfologija*. 2018;154(5):50-56. (In Russ.)]
 10. Еркудов В.О., Пуговкин А.П., Волков А.Я., и др. Гендерные различия размеров внутренних органов у 17-летних подростков с различными соматотипами // Педиатр. – 2017. – Т. 8. – № 5. – С. 67–73. [Erkudov VO, Pugovkin AP, Volkov AY, et al. Gender differences in the normative dimensions of internal organs of 17-years teenagers with different somatotypic characteristics. *Pediatrician (St. Petersburg)*. 2017;8(5):67-73. (In Russ.)]. <https://doi.org/10.17816/PED8567-73>.
 11. Иевлева К.Д., Рычкова Л.В., Шенеман Е.А., Баирова Т.А. Полиморфный локус Q223R гена LEPR и ожирение // Бюллетень восточно-сибирского научного центра сибирского отделения российской академии медицинских наук. – 2016. – Т. 1. – № 5. – С. 170–174. [Ievleva KD, Rychkova LV, Sheneman EA, Bairova TA. Q223R polymorphism of the LEPR and obesity. *Bull Vost Sib Nauch Sent*. 2016;1(5):170-174. (In Russ.)]
 12. Катаева И.В., Шульга И.М., Безроднова С.М. Дефицит массы тела и патология органов пищеварения у юношей-подростков, подлежащих постановке на первичный воинский учет // Экология человека. – 2008. – № 3. – С. 14–17. [Kataeva IV, Shul'ga IM, Bezrodnova SM. Body mass deficiency and digestive organs' pathology in boys-adolescents subject to be registered in military recruitment offices. *Ecology, human*. 2008;(3):14-17. (In Russ.)]
 13. Клиорин А.И. Ожирение в детском возрасте. 2-е изд., исправленное и дополненное. – Л.: Медицина, Ленинградское отделение; 1989. – 164 с. [Kliorin AI. Ozhirenje v detskom vozraste. 2-e izd., ispravlennoe i dopolnennoe. Leningrad: Meditsina, Leningradskoe otdelenie; 1989. 164 p. (In Russ.)]
 14. Клиорин А.И., Чтецов В.П. Биологические проблемы учения о конституциях человека. – Л.: Наука; 1979. – 254 с. [Kliorin AI, Chtetsov VP. Biologicheskie problemy ucheniya o konstitutsiyakh cheloveka. Leningrad: Nauka; 1979. 254 p. (In Russ.)]
 15. Красноперова О.И., Смирнова Е.Н., Чистоусова Г.В., и др. Факторы, способствующие формированию ожирения у детей и подростков // Ожирение и метаболизм. – 2013. – Т. 10. – № 1. – С. 18–21. [Krasnoperova OI, Smirnova EN, Chistousova GV, et al. Determinants of obesity in children and adolescents. *Obesity and metabolism*. 2013;10(1):18-21. (In Russ.)]. <https://doi.org/10.14341/2071-8713-5066>.
 16. Мельник В.А. Лонгитудинальное исследование изменений телосложения школьников г. Гомеля в период полового созревания // Вестник Московского университета. Серия 23: Антропология. – 2016. – № 1. – С. 86–92. [Mel'nik VA. Longitudinal study of body types in schoolchildren of Gomel at puberty. *Vestnik Moskovskogo universiteta. Seriya 23: Antropologiya*. 2016;(1):86-92. (In Russ.)]
 17. Ненартович И.А., Жерносек В.Ф. Индекс массы тела у детей с бронхиальной астмой при наличии структурных изменений лёгких // Медицинский журнал. – 2014. – № 4. – С. 92–97. [Nenartovich IA, Zhernosek VF. Body mass index in children with asthma and lung structure pathology. *Med Zhurnal*. 2014;(4):92-97. (In Russ.)]
 18. Панков Ю.А. Мутации в ключевых генах, контролирующих развитие ожирения и сахарного диабета //

- Молекулярная биология. – 2013. – Т. 47. – № 1. – С. 38. [Pankov YuA. Major gene mutations associated with obesity and diabetes mellitus. *Mol Biol (Mosk).* 2013;47(1):38. (In Russ.)]
19. Рябиченко Т.И., Скосырева Г.А., Карцева Т.В. Состояние репродуктивного здоровья девочек-подростков с дефицитом массы тела // Вестник Новосибирского государственного университета. Серия: Биология, клиническая медицина. – 2011. – Т. 9. – № 2. – С. 44–47. [Ryabichenko TI, Skosyrev GA, Kartseva TV. Condition Of Reproductive Health Of Girls Of Teenagers With Deficiency Of Hypotrophy. *Biologija, klinicheskaja meditsina.* 2011;9(2):44-47. (In Russ.)]
20. Саливон И.И., Мельник В.А. Способ определения типов телосложения человека по комплексу антропометрических показателей. Курский научно-практический вестник «Человек и его здоровье». – 2015. – № 1. – С. 93–98. [Salivon II, Mel'nik VA. Method of defining human constitution type by the complex of anthropometric parameters. *Kurskiy nauchno-prakticheskiy vestnik "Chelovek i ego zdrav'ye".* 2015;(1):93-98. (In Russ.)]
21. Солнцева А.В., Аксенова Е.А., Сукало А.В., и др. Полиморфизм гена рецептора лептина и изменения показателей лептинемии у детей с экзогенно-конституциональным ожирением // Весці Нацыянальнай акадэмії навук Беларусі. Серыя медыцынскіх наукаў. – 2011. – № 1. – С. 69–76. [Solntseva AV, Aksenova EA, Sukalo AV, et al. Leptin receptor gene polymorphism and leptinemia changes in children with adiposity. *Proceedings of the National Academy of Sciences of Belarus. Medical sciences series.* 2011;(1):69-76. (In Russ.)]
22. Строев Ю.И., Чурилов Л.П., Бельгов А.Ю., Чернова Л.А. Ожирение у подростков. – СПб.: Медкнига ЭЛБИ; 2006. – 216 с. [Stroev Yul, Churilov LP, Bel'gov AYu, Chernova LA. Ozhirenie u podrostkov. Saint Petersburg: Medkniga ELBI; 2006. 216 p. (In Russ.)]
23. Трашков А.П., Панченко А.В., Каюкова Е.С., и др. Лейкемия Р-388 у мышей линии CDF1 как тест-система опухоль-ассоциированного неоангиогенеза и гиперкоагуляции // Бюллетень экспериментальной биологии и медицины. – 2014. – Т. 158. – № 10. – С. 500–502. [Trashkov AP, Panchenko AV, Kayukova ES, et al. Leykemiya R-388 u myshey linii CDF1 kak test-sistema opukhol'-assotsirovannogo neoangiogeneza i giperkoagulyatsii. *Biull Eksp Biol Med.* 2014;158(10):500-502. (In Russ.)]
24. Трашков А.П., Васильев А.Г., Коваленко А.Л., Тагиров Н.С. Метаболическая терапия мочекаменной болезни на различных моделях поражения почек у крыс // Экспериментальная и клиническая фармакология. – 2015. – Т. 78. – № 3. – С. 17–21. [Trashkov AP, Vasil'ev AG, Kovalenko AL, Tagirov NS. Metabolic therapy of nephrolithiasis in two different rat models of kidney disease. *Eksp Klin Farmakol.* 2015;78(3):17-21. (In Russ.)]
25. Туркина Т.И., Щербо С.Н., Талицкий В.В. Некоторые вопросы генетики ожирения и метаболизма у детей и подростков // Медицинский алфавит. – 2017. – Т. 1. – № 6. – С. 5–8. [Turkina TI, Shcherbo SN, Talitskiy VV. On some aspects of obesity and metabolism genetics in children and adolescents. *Meditinskii alfavit.* 2017;1(6):5-8. (In Russ.)]
26. Струков Д.В., Александрович Ю.С., Васильев А.Г. Актуальные проблемы сепсиса и септического шока // Педиатр. – 2014. – Т. 5. – № 2. – С. 81–87. [Strukov DV, Aleksandrovich YuS, Vasil'ev AG. Actual aspects of sepsis and septic shock. *Pediatrician (St. Petersburg).* 2014;5(2):81-87. (In Russ.)]. <https://doi.org/10.17816/PED5281-87>.
27. Файзуллина Р.А., Киясова Л.М. Состояние вегетативной нервной системы у подростков с хроническим гастродуоденитом и дефицитом массы тела // Практическая медицина. – 2011. – № 1. – С. 128–131. [Fayzullina RA, Kiyasova LM. State of the vegetative nervous system in adolescents with chronic gastro-duodenitis and underweight. *Prakticheskaya meditsina.* 2011;(1):128-131. (In Russ.)]
28. Фефелова В.В., Фефелова Ю.А., Казакова Т.В., и др. Изменение активности ферментов основных метаболических путей лимфоцитов крови при пищевой нагрузке у девушек с разным компонентным составом тела (жировым, мышечным, костным) // Бюллетень экспериментальной биологии и медицины. – 2015. – Т. 159. – № 3. – С. 285–289. [Fefelova VV, Fefelova YuA, Kazakova TV, et al. Effect of Food Load on Activities of Enzymes of the Main Metabolic Pathways in Blood Lymphocytes in Girls with Different Anthropometric Parameters. *Biull Eksp Biol Med.* 2015;159(3):285-289. (In Russ.)]
29. Фильченков А.А., Залесский В.Н. Лептин, адипоциты и ожирение организма // Российский биотерапевтический журнал. – 2007. – Т. 6. – № 3. – С. 30–37. [Fil'chenkov AA, Zalesskiy VN. Leptin, adipocytes, and obesity. *Rossiiskii bioterapevticheskii zhurnal.* 2007;6(3):30-37. (In Russ.)]
30. Ходжиева М.В., Скворцова В.А., Боровик Т.Э., и др. Современные взгляды на развитие избыточной массы тела и ожирения у детей. Часть I // Педиатрическая фармакология. – 2015. – Т. 12. – № 5. – С. 573–578. [Khodzhieva MV, Skvortsova VA, Borovik TE, et al. Contemporary Views on Development of Excess Body Weight and Obesity in Children. Part I. *Pediatric pharmacology.* 2015;12(5):573-578. (In Russ.)]
31. Хромов-Борисов Н.Н. Биостатистические программы свободного доступа // Травматология и ортопедия России. – 2015. – № 4. – С. 154–159. [Khromov-Borisov NN. Free biostatistical software. *Traumatology and Orthopedics of Russia.* 2015;(4):154-159. (In Russ.)]

- [https://doi.org/10.21823/2311-2905-2015-0-4-154-159.](https://doi.org/10.21823/2311-2905-2015-0-4-154-159)
32. Щербакова М.Ю., Порядина Г.И., Ковалева Е.А. Проблема ожирения в детском возрасте // Экспериментальная и клиническая гастроэнтерология. – 2010. – № 7. – С. 74–82. [Shcherbakova MYu, Poryadina GI, Kovaleva EA. Problema ozhireniya v detskom vozraste. *Eksp Klin Gastroenterol.* 2010;(7):74-82. (In Russ.)]
33. Яковенко В.В. Особенности уровня лептина у детей с избыточной массой тела и ожирением // Академический журнал Западной Сибири. – 2013. – Т. 9. – № 1. – С. 10–11. [Yakovenko VV. Osobennosti urovnya leptina u detey s izbytochnoy massoy tela i ozhireniem. *Akademicheskiy zhurnal Zapadnoy Sibiri.* 2013;9(1):10-11. (In Russ.)]
34. Gunnell DJ, Frankel SJ, Nanchahal K, et al. Childhood obesity and adult cardiovascular mortality: a 57-y follow-up study based on the Boyd Orr cohort. *Am J Clin Nutr.* 1998;67(6):1111-1118. <https://doi.org/10.1093/ajcn/67.6.1111>.
35. Lahlou N, Landais P, De Boissieu D, Bougnères PF. Circulating leptin in normal children and during the dynamic phase of juvenile obesity: relation to body fatness, energy metabolism, caloric intake, and sexual dimorphism. *Diabetes.* 1997;46(6):989-993. <https://doi.org/10.2337/diab.46.6.989>.
36. Peeters MW, Thomis MA, Loos RJ, et al. Heritability of somatotype components: a multivariate analysis. *Int J Obes (Lond).* 2007;31(8):1295-1301. <https://doi.org/10.1038/sj.ijo.0803575>.

◆ Information about the authors

Valeriy O. Erkudov – MD, PhD, Senior lecturer of Normal Physiology Dept. St. Petersburg State Pediatric Medical University of the Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia. E-mail: verkudov@gmail.ru.

Andrey P. Pugovkin – PhD, Dr. Biol. Sci., Full Professor of Normal Physiology Dept. St. Petersburg State Pediatric Medical University of the Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia. E-mail: apugovkin@mail.ru.

Aleksej J. Volkov – Head of Children's Polyclinic Department No. 3. Saint Petersburg Regional Polyclinic No. 109, Saint Petersburg, Russia. E-mail: pd3@zdrav.spb.ru.

Oksana I. Musaeva – Head of the School-Preschool Department of Children's Polyclinic Department No. 3. Saint Petersburg Regional Polyclinic No. 109, Saint Petersburg, Russia. E-mail: oksana-musaeva@yandex.ru.

Tatiana N. Slyaptsova – Teacher of Biology, Vice-director. School No. 225 of Admiralteysky Region of Saint Petersburg, Russia. E-mail: tanjasl@mail.ru.

Mar'jana V. Chistyakova – 10-year Student. School No. 225 of Admiralteysky Region, Saint Petersburg, Russia. E-mail: m.chistyakova714@yandex.ru.

Sergei S. Rogozin – Senior Lab. Attendant of Normal Physiology. St. Petersburg State Pediatric Medical University of the Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia. E-mail: box.rogzin@yandex.ru.

Maria A. Pahomova – MD, Senior Researcher. Research Center. St. Petersburg State Pediatric Medical University of the Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia. E-mail: mariya.pahomova@mail.ru.

◆ Информация об авторах

Валерий Олегович Еркудов – канд. мед. наук, старший преподаватель кафедры нормальной физиологии. ФГБОУ ВО СПбГПМУ Минздрава России, Санкт-Петербург. E-mail: verkudov@gmail.ru.

Андрей Петрович Пуговкин – д-р биол. наук, ст. научн. сотрудник, профессор кафедры нормальной физиологии. ФГБОУ ВО СПбГПМУ Минздрава России, Санкт-Петербург. E-mail: apugovkin@mail.ru.

Алексей Яковлевич Волков – врач, заведующий, детское поликлиническое отделение № 3. СПбГУЗ «Городская поликлиника № 109», Санкт-Петербург. E-mail: pd3@zdrav.spb.ru.

Оксана Иосифовна Мусаева – врач, заведующая, школьно-дошкольное отделение детского поликлинического отделения № 3. СПбГУЗ «Городская поликлиника № 109», Санкт-Петербург. E-mail: oksana-musaeva@yandex.ru.

Татьяна Николаевна Сляпцова – учитель биологии, заместитель директора по развитию естественно-научного направления обучения. ГБОУ СОШ № 225 Адмиралтейского района Санкт-Петербурга. E-mail: tanjasl@mail.ru.

Марьяна Владимировна Чистякова – ученица 10 класса. ГБОУ СОШ № 225 Адмиралтейского района Санкт-Петербурга (ЛНМО «Биотоп»), Санкт-Петербург. E-mail: m.chistyakova714@yandex.ru.

Сергей Степанович Рогозин – старший лаборант кафедры нормальной физиологии. ФГБОУ ВО СПбГПМУ Минздрава России, Санкт-Петербург. E-mail: box.rogzin@yandex.ru.

Мария Александровна Пахомова – ст. научн. сотрудник Национального исследовательского центра. ФГБОУ ВО СПбГПМУ Минздрава России, Санкт-Петербург. E-mail: mariya.pahomova@mail.ru.

◆ Information about the authors

Aleftina A. Kravtsova – PhD, Assoc. Professor. St. Petersburg State Pediatric Medical University of the Ministry of Health-care of the Russian Federation, Saint Petersburg, Russia.
E-mail: aleftinakravcova@mail.ru.

◆ Информация об авторах

Алефтина Алексеевна Кравцова – канд. биол. наук, доцент кафедры патологической физиологии с курсом иммунопатологии. ФГБОУ ВО СПбГПМУ Минздрава России, Санкт-Петербург. E-mail: aleftinakravcova@mail.ru.