development of independence at physical culture classes are understudied. Consequently, the aim of the paper is to develop a method for optimizing the students' individual work when studying the subject "Physical Education". In order to achieve the aim of the research, the following research methods were used: method of formal self-record for estimating motion activity (Framingham method), A. Pirogova's method of estimating physical state index. The study involved examination of 92 students of 1-4 years of study at the Institute of Pedagogy and Practical Psychology on the basis of Sumy State Pedagogical University named after A. S. Makarenko. During the study period, the students had to estimate the index of physical state and the level of motion activity. The developed method of optimizing students' individual work when studying the subject "Physical Education" was based on individual approach to interest in the students' own physical condition. The effectiveness of the proposed method has been proven (28.7%). The efficiency of performing individual work on the subject "Physical Education" has been evaluated: prior to the implementation of the proposed method, the efficiency of individual work performance amounted to 34.6 ± 1.32%, while afterwards it increased by 28.7% (63.3 ± 1.46%).

Keywords: physical education, students, individual work, motion activity.

Подано до редакції 15.08.2016

UDC: 37.015:575+575.16

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INFLUENCE OF SEXUAL GENOMIC IMPRINTING ON CHILD ONTOGENESIS

The article presents the adapted form of the conceptual issues of the sexual imprinting phenomenology for students and pedagogues of psycho-pedagogical field. In the critical ontogeny periods individual's emotional reactions may have bright and impetuous manifestations so during these age periods imprinting effects may be different – from positive to catastrophic ones. The role of paternal and maternal genes during ontogenesis and their effect on initiation of child's psychophysical development deviations are determined.

Keywords: *genomic sexual imprinting, ontogenesis, deviations.*

Problem statement. Genomic sexual imprinting is a rather common phenomenon and it has a leading role in the human ontogenesis. Its phenomenology is that the chromosomes of germ cells acquire individual "mark" of gender and descendants get one chromosome set with father's marking of certain genes, and another set – with mother's one. Later, when the germ cells are being created, the obtained "mark" is erased and these genes will be marked according to the gender of the person.

The studying of sexual genomic imprinting (SGI) mechanisms is important for understanding the human biological evolution, the embryonic and postnatal individual development and identifying initiating factors of various kinds of developmental and neuropsychiatric disorders.

The importance and at the same time, insufficient knowledge of imprinting phenomenology explains the interest in it of not only theorists but also experts in the field of such applied specialties as psychophysiology of sport and professional achievements, age and pedagogical psychology, preventive pedagogy.

The article is aimed at highlighting the importance of genetic mechanisms of sexual imprinting in the determination of the individual peculiarities of child development and the initiation of developmental disorders.

The main tasks: 1) to describe the phenomenon of imprinting in the context of individual adaptive response to exo- and endogenous factors; 2) to outline the significance of genomic sexual imprinting in child psychophysical development determination.

Major content. Imprinting is a part of epigenetic inheritance: due to this phenomenon, properties and individual features that are not associated with changes in the basic molecules nuclear DNA structure are inherited. The essence of imprinting is that some genes in germ parent cells are marked in a certain way (for example, by cytosine bases methylation). The "marked" gene shows no activity in descendants: some genes inactivate in the spermatozoon, the others – in the ovules. As a result, the descendant inherits some features only from the mother (if these genes inactivate in the sperm) and part of the features – only from the father (if the gene is disabled in the egg). In the new generation germ cells' old labels are removed and replaced with new ones, as a result, the features of grandfathers or grandmothers may appear in grandchildren.

The importance of special labeling methods (methylation of DNA nucleotides) is overestimated. For example, the tumor cases studying have shown that the cancer pathology development is caused by genomic imprinting,

which is manifested in the spontaneous desamination of DNA 5-methylcytosine bases (the mechanism of human renal carcinoma). The role of methylation in carcinogenesis have been found, particularly retinoblastoma development (retina tumor) in children. Genomic imprinting plays a leading role in the development of different types of pathology such as transient neonatal diabetes, low blood pressure, obesity, mental retardation, acromicria, hypogonadism, ataxia, hyperkinesis, paroxysmal laughter, language development disorders, obsessive-compulsive syndrome and other neural diseases [1, 5].

A good example of the role of genomic imprinting is Turner's syndrome, when the human gene undergoes imprinting that determines social contacts. The dysfunction of this gene by receiving X-chromosome from the mother leads to abnormalities in behavior and a significant reduction in IQ. This syndrome is caused by a chromosomal abnormality X0 when the female receives just one X-chromosome from the father or mother (the similar anomalies occur in case of violation of the chromosomes separation during the gametes formation). It has been revealed that female patients with X-chromosome obtained from the father have significantly less abnormalities in social behavior and are less prone to autistic disorders. These female patients with insufficiently developed secondary sexual features look like women and have close to normal intelligence. The comparison of psychological tests results with maternal or paternal X-chromosome origin in patients with Turner's syndrome leads to the conclusion that the X-chromosome contains a gene that undergoes sexual imprinting and determines the behavior and intelligence of the person.

When discussing the importance of genomic imprinting for gender determination and gender differences, it is appropriate to present historical overview of the studies in this area.

In the middle of the XIX century Gregor Mendel in his experiments concerning crossbreeding of peas found that after mating homozygous plants with smooth and wrinkled seeds all plants will be identical and would only be smooth. This result did not depend on which plants have been selected for crossbreeding – male or female: the seeds were smooth. So Mendel has discovered the equivalence reciprocal crosses principle (ERCP): the gene comes out at the descendants equally no matter from which parent it has he been inherited. It has been found that this pattern (now known as a Mendel's First Law) is performed in case of inheritance of most alternative characters in all organisms and in people. But later genetics and embryologists have described a number of features for which this rule does not work; as a result of a number of studies the mechanisms of these deviations have been gradually discovered, including the effects of genomic imprinting manifestations.

Exceptions to the rule of identity of first hybrids generation is case of the reciprocal crossings are well known, but earlier they were attributed to one of two classes. The first one involves the features, which are determined by genes located in the sex chromosomes (sex linkage inheritance). The second class of non-equivalent reciprocal crosses includes attributes defined by non-nuclear genes contained in ovum mitochondria and cytoplasm, so these features are inherited only maternally (eg, mitochondrial encephalomyopathy).

Complementary to the aforementioned exceptions to ERCP the third class – the sexual genomic imprinting has been singles out by scientists [3]. SGI characteristics are not necessarily determined by sex chromosomes genes and are not associated with cell organelles. The principal is that SGI can affect any gene. The exceptions, related to the sex linkage inheritance or maternal organelle inheritance, are caused by the differences in the genetic parents' contribution in the descendant's genotype. In the case of SGI, on the contrary, both parents can pass absolutely identical genes to descendants, but the effect of these genes is varying depending on the maternal or paternal imprinting.

Manifestations of SGI in some way are related to some human diseases. R. Nicholls with his colleagues at Harvard Medical School of Harvard University have found that many patients with Prader-Willi syndrome (this disease is characterized by mental retardation, extreme obesity, short stature and disproportionately small arms and legs) inherited both chromosomes *15* from the mother [6]. R. Nicholls, J. Knoll and Ch. Williams have found the relation between Engelmann syndrome and genomic imprinting (this syndrome is characterized by inadequate risibility, sudden convulsive movements and mental retardation). These patients have as a rule partial deletion of *15*-chromosome inherited from the mother, so it is only paternal *15*-chromosome which is functionally active. These two syndromes, in spite of the difference in clinical picture, are associated with the differences in imprinting of the same genes and the same chromosome *15*.

Genomic imprinting can play a significant role in metastatic cancer pathology genesis in children, namely embryonic rhabdomyosarcoma (muscle cancer), Wilms tumor (kidney cancer) and osteosarcoma (bone cancer). There is data about carcinogenesis relations at an early age with recessive inactive oncogenes of paternal origin. For example, if father's gene *Rd* (antioncogene) is imprinted and inactivated, the sickness rate of embryonal rhabdomyosarcoma can be very high, because any genetic change in the inherited from the mother *Rd*-gene leads to the occurrence of cancer cells in the body. But because this disease is rare (it occurs about one child per 20 000), it is unlikely that every person inherits inactive recessive oncogene from his/her father. This fact can be explained by the fact that not all men (and probably not all women) have the same genes imprinted and inactivated. It is assumed that there are one or several genes responsible for imprinting (not those genes that undergo the imprinting). Thus, the imprinting is considered as a process determined by non-strictly fixed group of genes, which work in female and male organisms in a different way. Therefore, individuals of the same sex with different genes controlling imprinting should have different genes sets that are modified in this process. For example, the *Rd*-gene is not

inactivated for most men, but in rare cases, inactivation of the *Rd*-gene will happen in individuals who have aberrant gene that controls imprinting.

The statement that gene expression or its absence may be determined by other genes, in fact, describes a phenomenon long known in genetics as "dominance modification": many features depend on the activity of genes that influence the genes expressions that directly determine these characteristics [7, 8]. Genomic imprinting can be regarded as a special case of dominance modification. For genes-modifiers, which control genomic imprinting, only one unusual property postulates, namely these genes work differently in the persons of different sexes.

An interesting example of a feature that is modified by gender genomic imprinting is Huntington's disease. This disease is inherited as a dominant feature that appears in every person who inherited the mutated gene from at least one parent. Approximately in 10% of cases the disease develops under maturity age (sometimes it starts when a child is 2,5 years old). It has been ascertained that 90% of sick children with early manifestations of Huntington disease inherit the gene determining this disease from their fathers. It is quite clear that in these cases, both children and their parents have the same gene responsible for the disease; but its modification as a result of paternal imprinting leads to the manifestation of this disease at an early age.

Various hypotheses were made with the aim to explain the Huntington's disease and the best one was made by Ch. Leyrd from Washington University. He suggested that if the differences at the age of Huntington disease starting reflect mosaicism in the gene expression that causes the disease, this mosaicism probably depends on the genes-modifiers. If the gene-modifier acts that the mosaicism balance shifts toward mutant cells and appears almost entirely mutant tissue – the disease develops early; in the case of the opposite action of this gene it appears in later age. The fact that the gene causing the disease is inherited from the father and in most cases the disease begins at an early age is explained by Leyrd by the location of a gene-modifier in the X-chromosome. As men have one X-chromosome, any abnormality of the gene-modifier will be manifested, but not compensated as in women who have two X-chromosomes. Therefore, it is more likely for a man that the mosaicism balance will be shifted towards larger proportion of mutant cells in his descendants' tissues and the disease will develop at an earlier age. In 10% of early cases of Huntington's disease the child receives the corresponding gene from the mother (not from the father); this is to be expected, because so rare, but sometimes women can have the abnormal gene-modifier in both X-chromosomes.

As to genotype of sex, a human being belongs to the type *XX-XY*; there is a typical Mendel splitting of sexual chromosomes during the gametes formation. The sex with *XX* genotype is called homogametic (female sex) because it has the same gametes containing only X-chromosomes, while sex with *XY* genotype is heterogametic because half of its gametes contain X- and half – Y-chromosome. In the cell nuclei of the female organism one X-chromosome is always active and has a normal look but the other X-chromosome usually looks like a tight dark-stained stuff called Barr-body (facultative heterochromatin). There are no Barr-bodies in the cells nuclei of the male organism, and Y-chromosome is genetically relatively inert as it has significantly less genes. However, the influence of Y-chromosome on the sex determination is very strong and holandric: the genes cause psychophysical development of the male organism. If the normal male karyotype is 44+*XY* and female – 44+*XX*, the person with a 44+*X* karyotype is a woman while this one with a 44+*XXY* karyotype is a man [9]. In both cases people with distorted karyotype have developmental disorders but their look is determined by the presence or absence of Y-chromosome. A human being with 44+*XXX* genotype is a barren woman; while with 44+*XXXY* genotype is a sterile mentally retarded man (these genotypes appear as a result of sex-chromosomes nondisjunction and lead to the developmental disorders, such as for example Klinefelter syndrome with 44+*XXY* karyotype).

Thus, there are two basic genetic mechanisms for human sex determination and differentiation: chromosomal aberrations and, in fact, the genes expression. Despite the fact women have two X-chromosomes while men have only one, X-chromosome genes expression takes place at the same level in both sexes, because one of the woman's X-chromosome in each cell is completely inactivated (Barr-body). As noted above, the X-chromosome is inactivated at the early stages of embryonic development, and paternal or maternal X-chromosome becomes inactivated by chance in the female organism. The X-chromosome inactivation state is inherited during the subsequent cell divisions.

Sexual imprinting has a leading role in the individual development trajectories determination in ontogenesis. The phenomenon of imprinting is peculiar not only for behavioral reactions but also for morphogenesis at the early stages of embryonic development when the presumptive germs of future tissues at a certain time perceive chemical stimuli from the molecules-inductors (neuropeptides, hormones, signaling molecules) and thereby the transformation to specialized tissues and organs (morphogenesis) is ensured. If not, then the morphogenesis is chronologically implemented in advance or with delay and the embryonic development is violated [2].

It is appropriate to describe the genetic aspects of gender differences caused by the male and female genes participation at the determination of psychophysiological and psychological personal characteristics.

The scientists have managed to identify the genetic determination of cell division specifics in the embryo. The research results have shown that the cerebral cortex, hippocampus and basal ganglia consist of cells controlled by maternal chromosomes, while these cells are almost absent in the hypothalamus. It is well-known that various signals from the environment are processing and the be-

havioral reactions programs are being formed in the cerebral cortex. Paternal chromosomes are poorly represented in the cerebral cortex but there are much more of them in muscle tissue; as to the brain parts, they influence the hypothalamus and pituitary [3]. These brain areas are the morphofunctional basis of the limbic system, responsible for initiating and managing emotions. It is suggested that the cerebral cortex is responsible for the the communication with relatives and other people, while the hypothalamus is a completely "egoistic" organ the functioning of which is aimed at satisfying the vital needs of the person.

The Japanese scientist Yoh Ivasiv proposed an interesting theory explaining the above mentioned phenomenon. He suggested that due to the fact the father's chromosome (X- or Y-chromosome) determines sex of the embryo, it is the father's X-chromosome that works in the female organism, i.e. the peculiarities of female behavior should be determined by male chromosomes genes. If the female X-chromosome works as well, the feminization effect will manifest itself in sons and with the effect force in daughters. It is reasonable to conclude that behavioral sexual dimorphism can be controlled by male genes.

Thus, the X-chromosome has a gene or genes with sexual imprinting, making these genes work only in the paternal chromosome and are almost always inactive in the maternal one. These genes provide some influence on the social development of a child, in particular, his/her ability to assess the feelings of others correctly.

Now it becomes clear why autism, dyslexia and other problems with speech development more often occur in boys than in girls. As boys have only one X-chromosome (inherited from the mother) necessary for the optimal cognitive development, genes in this chromosome may not only be damaged, but inactivated as a result of sexual imprinting. [1]

GSI plays a significant role in the manifestation of the individual physiological characteristics and determines the restrictions in direct extrapolation of inherited psychological traits from parents to descendants. By now, not only sexual imprinting genes have been detected, but also sexual benefits genes determining sex-role behavior and communication features of men and women in the society [4].

According to V. Efroyimson, imprinting phenomenology should be considered as the basis for individual approach to life, as this phenomenon is observed in case of clear, distinctive and irreversible ontogeny stages during the formation of individual aspirations, preferences and inclinations. It means that the value criteria and subconscious decisions are formed and fixed (usually for whole life) for each person in the childhood and adolescence so that he/she will be guided by them throughout his/her life. This is the opinion of V. Efroyimson, the author of "Pedagogical Genetics", who believed a musical piece or a story heard at a certain moment (which somehow affects the emotional component of personal consciousness) may act like imprinting. The good example of this is the musical talent of P. Tchaikovsky. When he was five years old when he heard Mozart's "Don Giovanni" performed by mechanical orkestrino and later he admitted that he felt the music in his head and it bothered him greatly. Years later, after he graduated from Law School he joined the military service but having worked for less than a year, left it to be enrolled in conservatory, and only then found his true purpose in life. Therefore, the music heard in the early childhood caused the development of musical creativity manifested in the future. Another example is a famous mathematician S. Kovalevska, who saw huge figures on a wall when she was 3 years old. These figures have sunk into the child's mind so that they have determined her fate and mathematical talent in the future. There are enough similar examples in the society to conclude that imprinting can be the leading determinant of human approach to life.

Conclusions and prospects of further studies. 1. From psycho-physiological point of view, imprinting is closely associated with the emotional component of the individual's psyche, and different emotional reactions lead to intensification or suppression of various forms of mental activity. It should be noted that in the critical ontogeny periods individual's emotional reactions may have bright and impetuous manifestations, so during these age periods imprinting effects may be different – from positive to catastrophic.

2. Genomic sexual imprinting influence the determination of various features, including physiological personal characteristics. If the majority of psychological traits were dependent on the sex of one of the parents (from who the corresponding genes is inherited) psychogenetics surely would have noticed it. However, not all human features are studied thoroughly; the issue of genomic imprinting effect on the genes expression determined by certain individual psychological characteristics still remains understudied. The degree of gene expression may be not important for some traits, but for others it can be crucial for possible initiating of some developmental disorders.

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ВПЛИВ ГЕНОМНОГО СТАТЕВОГО ІМПРИНТИНГУ НА ОНТОГЕНЕЗ ДИТИНИ

Автори представили в адаптованому вигляді для студентів та фахівців психолого-педагогічного профілю основні положення феноменології статевого імпринтингу. Геномний статевий імпринтинг – досить поширене явище і воно має провідне значення в онтогенезі людини. Феноменологія його полягає в тому, що хромосоми статевих клітин індивіда набувають «відбиток» його статі і внаслідок цього нащадки отримують один набір хромосом з батьківським маркуванням деяких генів, а інший набір – з материнським. Надалі при утворенні в новому організмі статевих клітин отриманий «відбиток» стирається, ці гени маркуватимуться згідно зі статтю особи. Проаналізовано роль батьківських і материнських генів в онтогенезі й їхній вплив на ініціацію відхилень у психофізичному розвиткові дитини. Слід звернути увагу на те, що в критичні періоди онтогенезу емоційні реакції за рахунок епігеномних механізмів спадковості мають яскравий і бурхливий характер індивідуальних проявів, тому в ці вікові періоди наслідки статевого імпринтингу можуть бути різними – від позитивних до катастрофічних. Відомо, що у ґенезі метастатичної онкопатології у дітей суттєву роль відіграє геномний імпринтинг, зокрема при таких видах злоякісних новоутворень: ембріональна рабдоміосаркома (рак м'язів), пухлина Вільмса (рак нирок) і остеосаркома (рак кісток). Геномному імпринтингу також відводиться провідна роль у розвитку таких видів патології, як хвороба Гентінгтона, синдроми Прадера-Віллі та Енгельмана, транзиторний неонатальний цукровий діабет, синдром Шерешевського-Тернера, гіпотонія, ожиріння, розумова відсталість, акромікрія, гіпогонадизм, атаксія, гіперкінези, пароксизмальний сміх, пору-

шення мовного розвитку, обесивно-конвульсивний синдром та інші нейрогенні захворювання та психопатологічні розлади. Геномний статевий імпринтинг впливає на детермінацію різних ознак у людини, зокрема психофізіологічних характеристик індивіда. Якби для більшості психологічних ознак існувала чітка залежність їхнього прояву від статі батька, від якого успадковано відповідний ген, психогенетики, безсумнівно, помітили б це. Але механізми генетичної детермінації психологічних ознак людини вивчені недостатньо; ще належить з'ясувати вплив геномного статевого імпринтингу на експресію генів, які відповідають за певні психофізіологічні характеристики індивіда. Для одних ознак ступінь експресії генів може бути і неважливим, але для інших він виявляється вирішальним для можливої ініціації відхилень від нормативних траєкторій індивідуального розвитку.

Ключові слова: геномний статевий імпринтинг, онтогенез, відхилення.

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ВЛИЯНИЕ ГЕНОМНОГО ПОЛОВОГО ИМПРИНТИНГА НА ОНТОГЕНЕЗ РЕБЕНКА

Авторы в адаптированном виде представили для студентов и специалистов психолого-педагогического профиля основные положения феноменологии полового импринтинга. Геномный половой импринтинг – достаточно распространенное явление, которое имеет ведущее значение в онтогенезе человека. Его феноменология заключается в том, что хромосомы половых клеток индивида приобретают «отпечаток» его пола, и вследствие этого потомки получают один набор хромосом с отцовской маркировкой некоторых генов, а другой набор – с материнской. Далее, при образовании в новом организме половых клеток полученный «отпечаток» стирается, эти гены будут маркироваться в соответствие с полом индивида. Проанализирована роль отцовских и материнских генов в онтогенезе и их влияние на инициацию отклонений в психофизическом развитии ребенка. Следует обратить внимание на то, что в критические периоды онтогенеза эмоциональные реакции из-за включения эпигеномных механизмов наследственности имеют яркий и бурный характер индивидуальных проявлений, поэтому в эти возрастные периоды последствия полового импринтинга могут быть разными – от положительных до катастрофических. Известно, что в генезе метастатической патологии у детей существенную роль играет геномный импринтинг, в частности при таких видах злокачественных новообразований: эмбриональная рабдомиосаркома, опухоль Вильмса (рак почек), остеосаркома. Геномному импринтингу также отводится ведущая роль в развитии таких видов патологии, как болезнь Гентингтона, синдромы Прадера-Вилли и Энгельмана, транзиторный неонатальный сахарный диабет, синдром Шерешевского-Тернера, гипотония, ожирение, умственная отсталость, акромикрия, гипогонадизм, атаксия, гиперкинезы, пароксизмальный, нарушения речевого развития, обессивно-конвульсивный синдром, а также другие нейрогенные заболевания и психопатические расстройства. Геномный половой импринтинг влияет на детерминацию различных признаков у человека, в частности и психофизиологических характеристик индивида. Если бы для большинства психологических признаков существовала четкая зависимость их проявления от пола родителя, от которого унаследован соответствующий ген, психогенетики, несомненно, заметили бы это. Однако механизмы генетической детерминации психологических признаков человека изучены недостаточно; еще предстоит выяснить влияние геномного полового импринтинга на экспрессию генов, которые отвечают за формирование определенных психофизиологических характеристик индивида. Для одних признаков степень экспрессии генов может быть не такой важной, но для других она оказывается решающей для возможной инициации отклонений от нормативных траекторий индивидуального развития.

Ключевые слова: геномный половой импринтинг, онтогенез, отклонения.

Подано до редакції 15.08.2016