DOI: https://doi.org/10.17816/ecogen34777

About some genetic terms, their content and education

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Ambiguity of genetic terms usage leads to their misinterpretation and learning difficulties. The content of some concepts is analyzed as well as haziness and mistakes in usage of corresponding terms such as gene, allele, genotype, phenotype, polymorphism, heritability, variability and few others. Limitations of the model dividing separately impact of the environment and genotype in a feature formation are explored. The interaction of the environment and hereditary material is suggested as basic factor of genetic and phonetic variability.

Keywords: education; scientific terminology; gene; allele; genotype; phenotype; polymorphism; heritability; variability; "environment–genotype" interaction.

To cite this article:
Daev EV. About some genetic terms, their content and education. *Ecological genetics*. 2021;19(2):181–192. DOI: https://doi.org/10.17816/ecogen34777

Received: 18.06.2020 **Accepted:** 13.04.2021 **Published:** 21.06.2021

DOI: https://doi.org/10.17816/ecogen34777

О некоторых генетических терминах, их содержании и образовательном процессе

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Неточность употребления генетических терминов создает трудности в их понимании и препятствует качественному обучению. В статье проанализировано содержание некоторых понятий, а также неточности и ошибки в употреблении их описывающих терминов, таких как ген, аллель, генотип, фенотип, полиморфизм, наследуемость, изменчивость и некоторых других. Рассмотрены недостатки модели, разделяющей вклад генов и среды в формирование признака. Предлагается рассматривать взаимодействие среды с наследственным материалом как основной фактор генотипической и фенотипической изменчивости.

Ключевые слова: образование; научная терминология; ген; аллель; генотип; фенотип; полиморфизм; наследуемость; изменчивость; взаимодействие «среда–генотип».

Как цитировать:
Даев Е.В. О некоторых генетических терминах, их содержании и образовательном процессе // Экологическая генетика. 2021. Т. 19. № 2. С. 181–192. DOI: https://doi.org/10.17816/ecogen34777

Рукопись получена: 18.06.2020 **Рукопись одобрена:** 13.04.2021 **Опубликована:** 21.06.2021

ЭКО•ВЕКТОР

INTRODUCTION

Accurate usage of concepts

In any branch of science, terminology should be clearly defined and appropriately used. When adding new information to existing concepts, one should not create semantic contradictions, as many authors of scientific biological publications do. For example, "polymorphic variant" [1, 2] means "many forms of one specific form." The authors of several textbooks, books and monographs, particularly those of educational literature, do not always separate the terms "gene" and "allele"; they are considered different in some cases, but inaccurately referred to as equivalent in others [3, 4]. Furthermore, words or phrases such as "polymorphisms" and "single nucleotide polymorphism (SNP)" are frequently used incorrectly in the scientific and educational literature. More precisely, these would be "variants," "a variant of a polymorphic locus (gene)," or "a variant of replacement of a single nucleotide." Often, this is simply a substitute for the term "allele" (see, for example, [5]). Furthermore, in our opinion, the phrase "polymorphic variability" [6] should not be used as the second word simply repeats the first and is therefore redundant. In Russian educational sources, incorrect expressions such as "allelic genes," or "non-allelic genes," and representations such as "gene **A**" at the same time with "gene **a**" are sometimes used [7]. Such inaccuracies in textbooks considerably complicate the learning process and even distort the results/inferences thereby confusing the untrained reader.

CONCEPTS IN "TWIN" METHOD

The role of hereditary factors and environment in the formation of traits in humans was first studied by F. Galton in twins [8]. After a nearly 50-year hiatus, the foundations of the method were further developed by H. Siemens, K. Holzinger, and others [9–12] and are still used in studies on twins [13–16]. Variants of the twin method are widely used to study the **heritability** of various traits, especially when studying the human psyche [17].

The main postulates of the twin method are briefed in "Genetics with the basics of selection" [18, 19] and "Human genetics" [20, 21] and can be simplistically reduced to three points:

1. Identical twins or "**M**onozygotic **T**wins" (MT) have the same genotype, and non-identical or "**D**izygotic **T**wins" (DT) have different genotypes.
2. For a studied pair, the environment in which MT develop may be the same or unequal.
3. All properties of the body are determined by the interaction of only two factors – genotype and environment [18, 19].

Continuous developments in science have shown that mathematical models do not yet accurately describe biological phenomena. This requires the improvement of previously formulated scientific concepts. When reanalyzing data obtained by the twin method, for the beginning, necessary terms must be defined and filled by correct content.

The first and most significant concept is that of the "gene." Defining this fundamental term in genetics in the light of all available data is difficult. Unfortunately, some authors do not define "gene" at all. For example, in Vogel and Motulsky's "Human Genetics," 4th edition, the "gene" is understood as "a unit of heredity," based on information fragments spread throughout the book, primarily based on the definition that it is a "sequence of DNA bases containing information for protein synthesis in all living forms" [4, p. 59 and 75]. This understanding of the "gene" is far from complete, as not all genes encode proteins. In addition, speaking of "all living forms", it is difficult to understand what the living are limited to.

If a hypothetical diploid organism that needs only five genes is heterozygous in all five genes, would it have five or 10 genes? Can one person have two times the genes observed in another? An inaccurate definition of the "gene" could result in an inference that different people could have different number of genes, when comparing individuals who are homozygous with heterozygous in all genes.

Statements such as "... dizygotic twins, however, share on average 50% of their genes..." [22] or "... non-identical twins, like sibs, have an average half of common genes..." [3], warrant the following question: how many genes are general and "not common?" Only a qualified specialist would understand that identical or different **alleles** of genes are being referred to here. Nevertheless, following statements have been retained for 25 years in Vogel and Motulsky's "Human Genetics," 4th edition [4]: "... monozygotic twins have twice as large genes than dizygotic ones..." [4, p. 310], and "It is believed that dizygotic twins... have only half of the general genes..." (p. 325).

Use of the vague term "common genes" when comparing MT and DT is unacceptable, in our opinion, especially in educational literature. Each person has a complete set of all genes, but alleles can be different. In a pair of DT and simply sibs, this implies that not half of the "common genes" [4] but 50% of the same (or identical) **alleles** were transferred from the parents.

Upon clearly defining "alleles" as a specific form (specific variant) of a gene, one can easily deduce how many genes exist in humans, and explain the phenomenon of multiple allelism or heterozygosity. Not distinguishing between "gene" and "allele" (Speicher et al. [4]) would lead one to conclude that higher the degree of heterozygosity, the higher would be the number of genes. The concept of "gene" should be a generalization that combines all various alleles of a gene, i.e., the combination of all similar

sequences of nucleotides underlying the formation of a certain function(s) in the population of organisms of one species.

All people have the same number of genes with some exceptions (for example, in some genes of sex chromosomes, aneuploidy, duplications, or deletions, etc.). In general, all genes are the same in humans; only alleles can be different. Unfortunately, in scientific and educational literature, there is still the concept of "allelic genes," which is identical to alleles. Using the term "allele," for example, for polymorphic genes of the histocompatibility locus (*HLA*), some of which are represented in human populations by more than 300–500 alleles, appears more accurate than the usage of 300–500 "allelic genes."

Genotype, a basic concept in genetics, can also have different interpretations. It could be correlated with the allelic composition of one gene (for example, AA, Aa, and aa are different genotypes) or associated with a combination of alleles of all genes of the studied organism. The genotype most accurately describes the totality of hereditary factors (alleles of all genes) of one cell or unicellular organism.

The **genotype of a multicellular organism (GeM) should include the totality (mosaic, conglomerate) of all the different genotypes of the cells that make up the multicellular organism.** Mechanisms of emergence of mosaicism, even during the early stages of embryonic development of a multicellular organism, can be different [23, 24] and are not discussed here. Mosaicism within one organism will be specific to that organism. Even MT after some time represent different mosaics (in terms of localization and percentage) of different cells, with spontaneously arising differences in genotypes, which contradicts the first postulate underlying the twin method. If, for example, at the four-blastomere stage, one of MT has a cell carrying a balanced translocation that disrupts the functioning of cells of the central nervous system, a significant percentage of such cells (up to 25% or more) could cause sharp differences in IQ or any other indicators between the MT. These differences may turn out to be much stronger than those between DT, in which such an event did not occur.

As the degree of mosaicism increases with age, the number of differences between twins increases as the changes occurring in them are independent of each other. Nevertheless, this is often not taken into account, as the degree of similarity of **GeMs** in MT is generally higher than that in other compared groups.

Development of scientific concepts further shows that the second postulate of the twin method is also an oversimplification. Since a person is mainly adapted to the simultaneous bearing of one fetus, twins (both MT and DT) are initially in dissimilar conditions. Different positions in space, limited resources from the mother, and other factors contribute to different environments even for MT. The severity of competition between MT during pregnancy correlates with the time of their occurrence; a later separation of embryos increases their dependence on each other (closer location of implantation sites, the presence of common fetal membranes, etc.). Therefore, twins can be born different in mass, which correlates with the degree of their development and, in particular, their nervous system. In DT development, the time of conception may vary; occurrences of common membranes are less common, but insufficient maternal resources could lead to competition (opposite-sex twins are not considered here because of the peculiarities of the twin method). It can be assumed that the conditions when carrying siblings one after the other would be more similar in some respect if the mother were healthy, her lifestyle had not changed significantly, the time gap between pregnancies is small, and there are no extraordinary circumstances. Environmental conditions after birth can be more similar, near-identical (if MT are in the same family), or less similar (separated twins), **but never identical.**

Since the twin method begins with an assessment of the phenotypic similarity of various groups of twins (according to a particular trait), the concept of phenotype must be understood. Johannsen introduced the term "phenotype" as a set of external traits of an organism and stated: "... the description of tens of thousands of phenotypes in shape, structure, size, color, and other characteristics of living organisms was the main goal of natural science, which, as always, is a science in essence of a morphological and descriptive nature..." [25]. He also defined the phenotype differently with respect to trait, pure lines, or populations. He described the phenotype as:

a) A specific external manifestation of a particular trait (for example, weight, height, or color);
b) A combination of several traits of one organism;
c) Generalized or even averaged characteristics of groups (lines, populations) of organisms phenotypically similar in any one trait [25].

Improved understanding of the molecular genetic foundations of life encourages the idea that even the synthesis of a protein molecule can be considered a primary (proximal) phenotypic manifestation of a genotype [26]. Thus, a living organism can be characterized by its transcriptome, metabolome, proteome, or cell type, which significantly complements and expands the definition of "phenotype." In addition, **the phenotype of a multicellular organism (PheM)** is the result of the interaction of both genotypes and phenotypes of the various cells that constitute it. Cells differ not only within the normal reaction range of their genotype, but also in terms of genotype (G, or G) due to a mutation process, polyploidy, aneuploidy, etc. When stating that "the degree of manifestation of the phenotype is different in different individuals" and with the same genotype [3], it should be clarified that this refers to

individuals with identical genotypes for alleles of the same gene, which may nevertheless have a different phenotype. The authors refer to the expressiveness and penetrance of the trait, which must be clarified in textbooks [3]. At the same time, it is clear that this is the phenotype of a population (groups, lines).

However, **there can be no two identical GeMs, and no perfect coincidence of environmental conditions.** The results of twin analysis should not be interpreted based on the mathematical meaning of the operations used, when the influence of the environment (E) is estimated by subtracting the heredity coefficient (H) from the unit and the numbers obtained using these parameters [27].

A biologist is unlikely to argue against the fact that initially, the cell genotype arises as a function of unique combinations of environmental conditions continuously interacting with a specific pool of bioorganic molecules that make up the hereditary material ($G = f_{(H \times E)} = G_{(H \times E)}$, where ***G***, ***H***, ***E*** and "×" indicate genotype, hereditary material, environment and sign of interaction, respectively).

This implies an extension of the third postulate previously cited of the twin method [18], which in our opinion, should be considered as the first and main one:

1. **PheM** should be considered the result of continuous interaction of changing environmental conditions with the hereditary material of all its cells (**GeM**). As **GeM** is a function of a similar interaction ($GeM = f_{(H \times E)}$), **PheM** can be represented as a complex interaction function dependent on the direct ($\mathbf{f}_{\underline{E}}$) and genotype-mediated ($fGeM_{(H \times E)}$) influence of the environment on the phenotype ($PheM = f_E \times f_{GeM(H \times E)}$, where $0 < E \leq 1$).

The interaction index is often neglected due to its seeming small value, apparently, mechanically transferring to it a purely mathematical operation of multiplication. But if there were no continuous, constantly changing (and unique in each case) interactions of the hereditary material with the surrounding environment (*H*×*E*), there would be no **GeM**, no organism or trait studied by the researcher. Thus, a model describing the phenotype (**Ph**) of an individual as a simple sum of the average values of the contributions of the genotype (**G**) and the environment (**E**) ("The phenotypic value of an individual is the sum of the genotypic plus the environmental value: **Ph** = **G** + **E**" [4]) should be considered too simplified and apparently obsolete.

A **GeM** is the product of the interaction of the genotype of the initial zygote and all cells derived from it with environmental conditions. No matter how metrically the gradations of the studied phenotypic trait are expressed, each specific value reflects the result of the inseparable interaction of a unique (at each moment of ontogenesis) combination of environmental conditions with an equally unique **GeM**. Any **PheM** from a biological point of view is an even more complex function, not reducible to the formula **Ph** = **G** + **E.**

The second postulate of the twin method (formerly the first one) should be formulated more precisely as follows:

2. MT have more similar (but not identical) **GeM**s, and fraternal twins are less similar (different).

The third postulate of the method can also be modified as follows:

3. For the studied pair of MT, the environment in which they develop may be more similar (but not identical) than that for DT, and sometimes even less the same.

Additional difficulties can be created by:

a) the polygenic nature of inheritance of the trait under study, b) its incomplete penetrance, c) the different nature of interallelic (intragenic) and intergenic interactions, d) the epigenetic nature, and e) other differences arising in the process of individual ontogenesis.

CONTRIBUTION OF GENOTYPE AND ENVIRONMENT IN MANIFESTATION OF A TRAIT

The contribution of genotype (small or large additive genetic contribution) and environment to the manifestation of a trait [20, 21] or "the influence of the genotype and environment on the development of a trait" [18] is continually evaluated; the formula **Ph = G + M** is used [4]. It is generally accepted that the first term, which takes values from 0 to 1 (or 100%), estimates the genetic contribution to the studied trait in the population [20] or the share of heredity in the development of the feature [18]. "A low value of heritability implies small," and "high value indicates a large" contribution of genes, and additionally acting genes [20].

However, two biologically relevant conditions must be considered: 1) The **genotype** contribution cannot be "1," as this denotes the complete absence of the influence of the environment; 2) The contribution of the genotype also cannot be "0," as **Ph** will not exist in the absence of **G**.

One can submit conditions of the environment in which the studied form of a trait can either not manifest or will always manifest (100% dependence on the environment ***E***). The presence of any mutation that changes the traits analyzed proves that the genotype (***G***) contributes entirely (100%) to the formation of a specific form of this feature in this case. But is the effect of the environment (***E***) at the same time zero (?), if interaction with environmental factors ($E \times G$) allowed or did not allow the manifestation of the mutant phenotype (***Phe***). Thus, the simplified model using ($G + E$) incorrectly describes the phenomena, dividing the deposits of the genotype and the environment in the development of a trait and then adding them.

Analysis of the studied trait by the twin method begins with comparison of the pair concordance ($\mathbf{C}_\mathbf{p}$) of MT and DT of the same sex. The degree of genetic determination

of the phenotypic similarity in the sample of twin pairs is estimated based on the trait, separating it from the contribution of the environment [**H = (1 – E)**, where **1** is the ideal (100%) similarity of **PheM**s of MT, **H** and **E** are the contributions of the genotype and environment, respectively, in this similarity]. The study indicates that complete similarity of any particular shape of a studied attribute does not always occur. The difference ($\mathbf{1 - C_{pMT}}$) shows that in reality is not always and not all similarities are determined by the **genotype**; some part of this is contributed by the interaction of unaccounted differences in environmental factors, which also leads to differences in **GeM**. Absence of differences on the monogenic-controlled feature, i.e., $\mathbf{C_{pMT} = 1}$, may indicate that in a particular sample of the analyzed MT pairs of the **GeM**s changes, the environment and interactions with it do not affect the attribute being studied. However, this does not mean that heredity contributes 100% to the formation of a trait, as some authors note [3]. It is just that the **question about the degree of influence of genotype and environment on the development of a trait is formulated incorrectly. It is impossible to separate the influence of genotype from the effect of the environment** [3, 28, 29]. In this case, the effect of the factors under consideration (genotype and environment) "is not accumulative or optional" [30]. They are interdependent, and the nature of this interdependence is dynamic, i.e., constantly changing.

The problem of the ratio of genetic and environmental contributions (*nature vs. nurture*) to the development of traits is often viewed by comparing the variability of a trait in groups with different degrees of kinship. At the same time, it is not the concordance that is compared, but the variability of a trait in samples from genetically different populations, for which new concepts are introduced–heritability and coefficient of heritability [20]. Certain semantic inconsistencies are associated with these terms. Unlike inheritance, the process of passing on a trait by inheritance, heritability in a broad sense is the property (ability) of traits, or rather of any particular form of trait, to be inherited (see Table 1).

Heritability (both in a broad and narrow sense) should be a characteristic (estimate) of constancy, and not variability of the feature (or a specific form of a trait) which is transmitted. It should depend only on the properties of hereditary material, such as inheritance, method of breeding, and environmental influences. When it is limited to the analysis of concordance (similarity on the studied basis), the contribution of similarity on the studied attribute estimated as $\boldsymbol{H_c = (C_{MT} - C_{DT})/(1 - C_{DT})}$ can indeed be considered dependent on the genotype (inherited, i.e., the heritability coefficient). However, we compare several model examples.

When samples of separated MT and DT raised in countries with different languages are compared, the pair concordance on the basis of the native language will strive for zero, as the $\boldsymbol{C_{MT}}$ will be equal to the corresponding $\boldsymbol{C_{DT}}$. **Thus,** the inheritance indicator $\boldsymbol{H_c}$ will also be zero. It is clear that the feature entirely depends on the differences in the environment and is not genetically inherited.

Consider another example where the trait is rigidly controlled by alleles of one gene. The paired concordance of MT will be **1** and for the dizygotic ones – no: $\boldsymbol{C_{DT}}$ will be less than $\boldsymbol{C_{MT}}$ and $\boldsymbol{H_c}$ will not be **0**.

However, if the sample randomly comprises DT, identical on the corresponding allele of the gene as in the second example, $\boldsymbol{C_{DZ}}$ will also be **1**. Therefore, $\boldsymbol{H_c}$ would be reduced to "0/0," representing uncertainty, a result difficult to interpret in biology, although the "inheritance" of this feature (contrary to the first example) is clearly **1**. If the concordance of both MT and DT is the same, but less than 1, $\boldsymbol{H_c}$ will be 0.

Thus, $\boldsymbol{H_c}$ reflects not the heritability of a feature, and the degree of differences in the genetic structure of the samples of monozygotic (genetically homogeneous pairs) and dizygotic (genetically heterogeneous pairs) twins and, at the same time, differences in the interaction of different genotypes with environmental conditions.

As J. Lush (a quote from: [3]) restricted the definition of heritability by frame of genetically determined phenotypic characteristic variability, at least a Russian-language reader had to be issued. In contrast to inheritance

Table 1. Trait properties and their assessment

Trait properties	Trait's manifestation	Assessment criterion used
Variability: the ability of a trait to exist in the form of several specific forms ($n > 1$)	Existence of changes (i.e., availability of the trait's different variants)	1. Assessment of the total **variance** of the trait's forms (V_t) and environmental variance (V_e). 2. Assessment of genetic variance ($V_g = V_t — V_e$). 3. Calculation of the "heritability coefficient" ($H = V_g/V_t$) or, more precisely, the variances ratio: of genotypically induced **changes** to total spectrum of changes
Heritability: the ability to transmit a specific form of trait to subsequent generations	Constancy of the trait's specific variant	Comparison of the degree of samples concordance according to the studied form of the trait

DOI: https://doi.org/10.17816/ecogen34777

(transference of something permanent, inherited) of some particular form of a trait that ensures similarity, variability is the ability of a trait to change (i.e., to take several different forms). The variability of each trait should also be considered as partly inherited by the characteristic of this feature. Here, it would be more accurate to define the "heritability coefficient" of J. Lush as **"the share of changes in the trait defined by the genotype"** ("**S**hare of **P**henotypic **C**hanges", further abbreviated as $\mathbf{SPC_G}$ or $\mathbf{SPC_E}$, if defined correspondingly by the genotype or the environment). We do not refer to heritability of a trait in general, but to the latitude of the spectrum of its changes in the studied sample (population). It is also only rarely possible to clearly separate $\mathbf{SPC_G}$ from $\mathbf{SPC_E}$ (the share of changes in a trait defined by the environment), as the latter is also determined by the reaction rate of all individual **GeM**s of the analyzed samples. It should be noted that although "trait" in this context is also a generalized concept that combines all various specific forms of this feature, it is often also used as the designation for a single particular form of a trait.

"Inheritance coefficients" are purely conventional indices that have no specific genetic interpretation [11, 31]. David Boueno's review on the influence of improper understanding of scientific concepts on the educational process [32] states: "...some common misunderstandings concerning the biological meaning and significance of the concepts of gene function and heritability have led to some educational proposals allegedly having a scientific basis when in fact they had none at all...".

Inconsistencies in the definitions of calculated indices such as the "heritability coefficient" cause erroneous data interpretations, which could probably be avoided by renaming "heritability coefficient" to "variability coefficient." However, as it only describes the share of observed changes in a trait dependent on the genotypic diversity ($\mathbf{SPC_G}$) in an analyzed sample, and not all observed changes, the generally accepted designation of h^2 should be replaced by v_g^2.

The need to separate "variability" from actually analyzed "changes" [33] when calculating "inheritance coefficients" must be explored. Variability is a general property (ability) to change, but it refers here only to the share of genotypically determined changes in a trait (Table 1).

Despite "heritability" and "variability" having opposing meanings, many researchers use the definition of Lush. Heritability (or hereditability) was considered a fraction of the phenotypic variability of any feature (for example, growth), which can be attributed to hereditary due, as opposed to the environment **(**"...proportion of observed variation in a particular trait, such as height, that can be attributed to inherited genetic factors in contrast to environmental ones..." [34]**)**. Thus, heritability in a broad sense, is currently defined as the ratio of variability due to individual genotypic differences, to the total phenotypic variability in the population ("...heritability, in a general sense, is the ratio of variation due to differences between genotypes to the total phenotypic variation for a character or trait in a population..." [35]).

The phenotypic variability of a feature ($\mathbf{V_{phe}}$, which is equal to 1) can be simplistically described using the expression $\mathbf{V_{phe} = V_G + V_E}$, where $\mathbf{V_G}$ and $\mathbf{V_E}$ are the proportions of variability determined by heredity (genotype) and environment, respectively. The environmentally controlled variability is calculated as a difference ($\mathbf{1 - V_G = V_E}$).

In some studies, $\mathbf{V_E}$ is defined as a dispersion of a specific form in the first generation obtained after crossing pure (homozygous) lines, assuming uniformity in offspring F1 [3]. Here, the component $\mathbf{V_{(E \times G)}}$ (interaction of the environment and genotype) is often neglected, making the assumption of independence of the corresponding dispersions [20, 21].

As noted above, the influence on a phenotype is incorrectly divided into the contributions of the genotype and the environment [3, 28, 29]. A more accurate definition would consider a spectrum of all observed phenotypic symptoms as a function of the interaction of changes induced by direct action on a phenotype ($\mathbf{V_{ph(E)}}$), with changes in the phenotype caused by an indirect action of the genotype ($\mathbf{V_{ph[G\,(E)]} : V_{ph} = V_{ph\,(E)} \times V_{ph[G\,(E)]}}$), where $0 < \mathbf{e} < 1$, and "×" is a sign of interaction. Here both the genotype and environment can change under the action of the phenotype (Fig. 1).

The second factor is a complex function, as the genotype itself is a product of the interaction of hereditary material with the environment, i.e., a variable dependent on the environment. Thus, the environment of a multicellular organism would be a multi-level system of embedded media (environment of cells, tissue, organ, organ systems, and all organism systems) continuously interacting with each other with an environment outside the body and with hereditary material in the process of ontogenesis (Fig. 2).

Life is the result of the interaction of polygenic systems and multifactor environmental impacts. Therefore, defining reliable quantitative characteristics with understanding of these associations is a substantial task ("... if real life gene × environmental interactions are both polygenic and poly environmental, the task of reliably quantifying meaningful explanatory associations is currently difficult...") [36].

However, many publications use the traditional calculation of "heritability coefficients" as $h^2 = V_G / V_P$, referring to **heritability of a trait** but scipping "variability" (or "changes" in our opinion) of a trait. Heritability of a trait is not the same as the "inheritance" of its genetically determined changes ($\mathbf{SPC_G}$).

Some genetic textbooks describe examples where the ratio of genetically determined dispersion to the general is called, for example, "heritability of the flower length" [3],

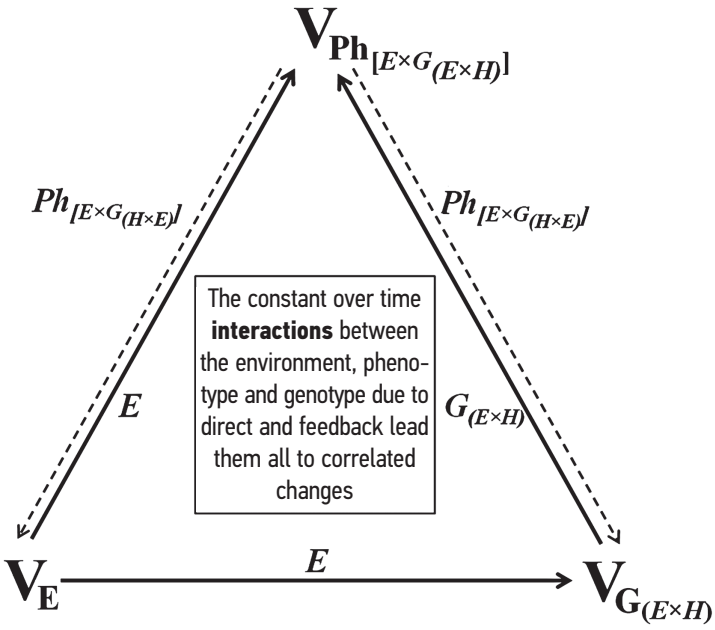

Fig. 1. Changes in environmental factors ($\mathbf{V_E}$) can directly affect both the genotype and phenotype (***E***), determining their changes ($\mathbf{V_G}$ and $\mathbf{V_{Ph}}$). The environment can also affect the genotype indirectly, inducing phenotypic changes leading to changes in the genotype. Conversely, by directly acting on hereditary material (***H***) and inducing genotypic changes ($\mathbf{V_{G(E\times H)}}$), факторы environmental factors cause genotype-mediated [$\boldsymbol{G_{(E\times H)}}$] phenotypic changes. In turn, phenotype changes, representing a complex function of the interaction of the genotype and the environment ($\boldsymbol{Ph_{[E\times G_{(H\times E)}]}}$), can have an opposite effect on them. The arrows indicate the direct effects, and the dotted arrows and italics indicate the reverse effects

although it is only a characteristic of the variability of this feature in a particular population, a fact noted by the authors themselves. Some authors write: "... calculate the heritability factor of a feature, which shows what the estimated proportion of genotypic variability is in the observed phenotypic variability" [19]. Here, $\mathbf{V_G}$ is occasionally defined as "total genotypic variability in a population" [37], when in fact it is part of the phenotypic variability of the population determined by its genetic structure. Use of the phrase "heritability coefficient of a trait" with subsequent explanations (if any) leads to false impressions of the degree of genetic determination of a trait (or its formation) in inexperienced readers (especially in students), which can lead in future to socially significant negative consequences [32, 38].

Comparison results of dispersions of a studied feature in genetically different populations with a firm standard obtained for genetically homogeneous groups are often considered as a contribution of heredity in the development of a trait. This interpretation, in our opinion, does not correspond to the true meaning of the calculated "heritability coefficients".

Foreseeing these problems, several authors dissuaded the use of similar indices of "heritability" [31]. Such calculations hold little sense if a trait with a high level of heritability is drastically affected by environment factors ("... a trait with high heritability might be greatly altered by the environment..." [32]). In different studies, the heritability of traits, such as the mass of chicken eggs or the cow's milk yield varies from 0.1 to 0.8 and from 0 to 0.7, respectively [16]. Nevertheless, "heritability coefficients" are continuously used and discussed while interpreting "heritability" as a certain part of variability and dispersion [4]. In fact, it appears that the average indicators of "heritability" reflect the concordance degree of phenotype of a population (sample) individuals to mean conditions of the surrounding environment.

If the phenotypic manifestations of a trait do not interfere with the survival of an organism, then they would all be presented in the population. Indirectly, selection for (or against) some phenotypes will affect the genetic structure of a population. Comparison of samples with a contrasting degree of homozygosity (heterozygosity) for such a trait would reveal differences in phenotypic concordance and high "heritability coefficients." If some forms of manifestation of a feature reduce viability, then both phenotypic diversity and genetic heterogeneity of one of the samples will decrease thereby reducing the "heritability coefficient." Thus, indicators of "heritability" as calculated by Lush actually evaluate the dispersion of the trait and reflect the degree of differences in the genetic heterogeneity of comparable groups. Heterogeneity, in turn, is determined by the variability of environmental conditions of comparable samples. It, thus, depends on the degree of concordance of each of the trait specific forms to environmental conditions and natural selection intensity.

If such arguments hold true, then a high or low value of **H** can, in no case, be interpreted to contribute to the formation of a trait studied, especially in educational materials (see, for example, http://buzani.ru/zadachi/genetika/792-bliznetsovyj-metod-antropogenetiki-zadachi-1-8 and other educational sites). This contribution of heredity would, thus, be only into the general variability of the trait in the particular group of individuals studied, and based on an oversimplified mathematical model.

The incorrect interpretation of high "heritability coefficients" of a trait would lead to false inferences such as in physicians and patients that could lead to ineffective treatment plans in patients with a genetically determined disease that has a high coefficient. In addition, the same term (heritability) should not be used in the analysis of both qualitative and quantitative traits, as the calculated values most often have different content; in the first case, it is a genetically determined concordance, and in the second, it is a genetically determined variability of the feature.

Researchers emphasize that "heritability coefficient" can vary significantly between experiments [39], as in reality it reflects the difference in the genetic structure of the compared samples and in environmental conditions. It would be more accurate to assume that high or low values of $\mathbf{SPC_G}$ relate to the degree of importance of a particular form of a trait for the body's fitness in changing environmental conditions (*i.e.*, in accordance with environmental conditions). They can also, to some extent, relate to features of biochemical mechanisms of molecules involved in various vital functions (branching paths of biosynthesis, features of the structure of a protein, etc.).

Incorrect jargon in biology and, in particular, genetics arises from the under-development and fuzziness in the definitions of the terms used [33, 40]. Terms may be incorrectly used as synonyms, or their definitions are varied with context, or they are also used in parallel immediately in several values [41]. As a result, there are differences in their subjective perception, leading to an erroneous worldview, especially among the general public. Inattention to the use of terms is a substrate of pseudoscience [40]. It would appear that a minor inaccuracy, especially in a widely used textbook; however, just as an inconspicuous error in the first few nautical miles of a voyage can lead to a tragedy (for example, the ship may not dock at the right port or miss an island, even lose a mainland as in the famous novel by Jules Verne).

The vague use of the term "stress" by researchers may lead a non-biologist to think that plants and bacteria have a hypothalamus, pituitary gland, and adrenal gland. The term "stress" applied to temperature, ultraviolet, humidity, or hunger (as well as polymorphism, SNP applied to a single nucleotide replacement) could be regarded as unprofessional usage or indifference to scientific clarity.

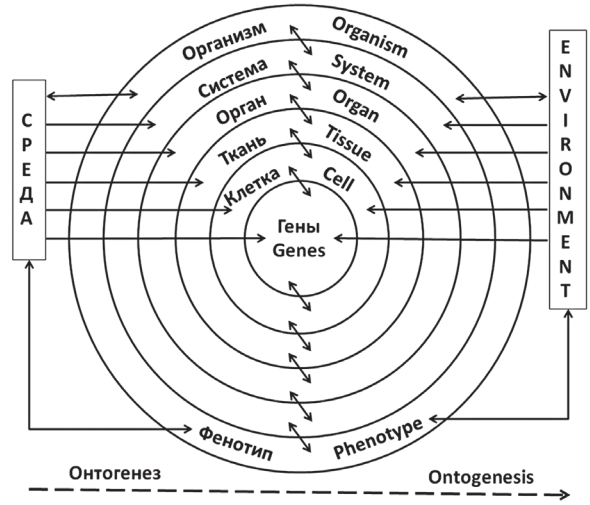

Fig. 2. Scheme of direct and indirect (through genes, cells, organs, systems and organismic levels) environmental influence on phenotype of multicellular organism (modified from [29])

From such fuzzy usages, those acquainted with the principle of mathematical induction (or at least with the "deductive" method of Mr. S. Holmes) can easily compile a chain of false scientific reasoning.

The timely improvement of a specific terminology and its correct use is necessary to prevent pseudoscience development. That is why of very important to choose the right educational material that competently highlights the modern problems of genetics, and each term is filled by correct content to prevent misinterpretation of scientific concepts by readers.

CONCLUSIONS

1. Currently, the definition of many terms traditionally used in genetics is expanded too wide that the creation of whole families of terms related to the original ones has become necessary. The content of terms "genotype" and "phenotype," for example, should be expanded to include multicellular organisms. Refined terms such as **GeM** and **PheM** may be used, where necessary. Furthermore, the conceptual apparatus should be continuously and **correctly developed** as rapid progress is made in biological sciences.

2. Interpretation of the results obtained, particularly those by the twin method, as well as determination of the proportions of the influence of heredity and environment on the processes of a trait formation (as it is often interpreted) appears incorrect. Such an interpretation develops in a large readership (particularly students) erroneous ideas about the separation of environmental and genetic influences on biological processes, and leads to an underestimation of the role of genotype-environmental interactions. It is ecologically more accurate to emphasize the constant dynamic relationships in the "environment↔genotype↔phenotype↔environment" interdependent system.

3. The erroneous assignment of the term "coefficient of heritability" to data comparing the variability of changes in a trait in genetically heterogeneous samples creates the impression of the existence of a degree of "heritability" of a trait among certain researchers and students. Additional explanations are required for clarity, which can be avoided by simply renaming the characteristic to be calculated, for example, "genotype-determined rate of change for a trait" (**SPC_G**) as opposed to the share of environment-driven non-inherited change (**SPC_E**).

4. When using mathematical symbols like "+," "–," "×," and ":" in biology, it should not automatically transfer the mathematical properties of an operation to the biological phenomena described. Although 2 + 2 always equals four in mathematics, this is not the case in biology.

5. With the rapid development of information technology and the weakening control over information quality, educational sources (printed materials, Internet, etc.) must be carefully chosen to deliberately prevent the formation of an erroneous scientific worldview in the reader.

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