

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/315378645>

Practical 3. Genes interaction. Dominance, incomplete dominance, co-dominance and lethal alleles

Chapter · February 2017

CITATIONS

0

READS

5,611

5 authors, including:



Boris M. Sharga

Uzhhorod National University

60 PUBLICATIONS 85 CITATIONS

[SEE PROFILE](#)



Volodymyr Feketa

Uzhhorod National University

57 PUBLICATIONS 4 CITATIONS

[SEE PROFILE](#)

Some of the authors of this publication are also working on these related projects:



radiation protection [View project](#)



Bacillus as antagonists and biocontrol agents of plant pathogenic bacteria and fungi [View project](#)

Uzhhorod National University
Department of Fundamental Medical Disciplines

MEDICAL BIOLOGY PRACTICALS. GENETICS.

Practical 3. Genes interaction. Dominance, incomplete dominance, co-dominance and lethal alleles

Compiled by B.M. Sharga, M.Y. Hliudzyk, Y.P. Sanislo, D.B. Pilipiv, V.P. Feketa

There is nothing difficult for understanding of **dominance** of particular gene. Here one (dominant) gene product totally suppressing the effect of other (recessive) gene. For example, the dominance of the genes for yellow seed color and round pea shape over the genes of green seed color and wrinkled pea shape were showed in Mendel's experiments [1].

The **incomplete dominance** could be regarded as "dilution" of the effect of particular gene by the effect of other gene product in heterozygote, resulting in new phenotype (as it could be seen in pink flower color in snapdragons or level of lip protrusion, shape of hair, voice pitch, Tay-Sachs disease severity and hands size in human).

The **co-dominance** allows manifestation of both genes effects, like it is observed, for example, in human blood groups [1].

Although an organism can carry only two alleles of one gene, many alleles of a single gene can be present in population. This situation is called **multiple allelism**, and the set of alleles itself is called **allelic series**. An example is a self-incompatibility alleles in plants. When same alleles met in pollen grains and in egg cells no pollination take place. This encourages the exchange of genes between different plants in population. Multiply alleles determine the chevron pattern on the leaves of white clover. The coat color in rabbits is also controlled by multiply alleles with dominance row $C > c^{ch} > c^h > c$. (Table 1) [1].

Table 1. C gene in Rabbits

| Coat-color Phenotype | Genotype |
|----------------------|--|
| Full color | CC or Cc^{ch} or Cc^h or Cc |
| Chinchilla | $c^{ch}c^{ch}$ or $c^{ch}c^h$ or $c^{ch}c$ |
| Himalayan | c^hc^h or c^hc |
| Albino | cc |

Other example is the human ABO blood-group alleles. There are 4 blood types (or phenotypes) in the ABO system (Table 2). There are 3 major alleles — i , I^A , and I^B — but, of course, any person has only 2 of the 3 alleles (or 2 copies of 1 of

them). The alleles I^A and I^B each determine a unique antigen; allele i confers inability to produce an antigen. In the genotypes I^Ai and I^Bi , the alleles I^A and I^B are fully **dominant**, but they are **codominant** in the genotype I^AI^B .

Table 2. ABO blood groups in human

| Red blood cell phenotype (group) | Genotype | Antigens in red blood cells | Antibodies in plasma |
|----------------------------------|----------------------|-----------------------------|----------------------|
| O (I) | ii | None | A and B |
| A (II) | $I^A I^A$ or $I^A i$ | A | B |
| B (III) | $I^B I^B$ or $I^B i$ | B | A |
| AB (IV) | $I^A I^B$ | AB | None |

Two other blood polymorphisms human have in addition to the ABO system. Two alleles L^M and L^N determine the M , N , and MN blood groups. The dominant allele R of a different gene causes a person to have the Rh^+ (rhesus positive) phenotype whereas the homozygote for r is Rh^- (rhesus negative).

Very interesting and some time confusing effects may be observed in action of **lethal alleles**.

Lethal alleles (or in other words, **lethals** or **lethal genes**) are alleles causing the death of the organism, in which they are present. Lethal alleles are usually result of mutations in genes that are essential to growth or development [2]. They may be recessive, dominant, or conditional depending on the gene or genes involved. Lethals can cause death of an organism prenatally or in any time after birth, though they usually manifest early in development.

Dominant lethal examples. Lethal alleles were first discovered by Lucien Cuénot in 1905 while studying the inheritance of yellow coat colour in mice [3]. W.E.Castle and C.C.Little confirmed the findings of L.Cuénot in 1910. The lethal A^Y allele discovered by Lucien Cuénot is dominant. It controls 2 characters: color and survival. Such genes that have more than one distinct phenotypic effect are said to be **pleiotropic**. It is entirely possible, however, that both effects of A^Y pleiotropic allele result from the same basic cause, each promotes yellowness of coat in a single dose and they are lethal in a double dose [1].

Huntington disease is a progressive brain damage that causes uncontrolled movements, emotional problems, and loss of thinking ability. Adult-onset of the disease, the most common form of this disorder, usually appears in a person's thirties or forties. It is caused by mutations in the *HTT* gene. The *HTT* gene codes production of a protein *huntingtin*. The gene has trinucleotide repeat variable in length between individuals. This repeat may change in length between generations. When the length of this repeated section reaches a certain threshold, it results in production of the mutant Huntingtin protein (*mHTT*). The presence of this gene in homozygotes and heterozygotes (*HH* and *Hh*) leads to Huntington's disease.

Early symptoms can include depression, irritability, small involuntary movements, poor coordination, and learning and making decisions problems. Ill individuals may have problems in walking, speaking, and swallowing. They usually live about 15 to 20 years after first symptoms appear.

The "creeper" allele in chicken confers the legs to be short and stunted. Heterozygous chickens display the *creeper* phenotype. If these they are crossed, the deviation from Mendelian ratio is observed: the offspring obtained is 2/3 creeper and 1/3 normal. This is because of homozygous *creeper* chickens death.

Recessive lethals examples. *IHH* gene encodes protein for bone growth and differentiation. A single mutated copy of this allele results in few deformations of skeletal bones (*brachydactyly* only) as one dose of functional *IHH* allele is almost enough to produce a required amount of a protein. When two *IHH* alleles inherited, no protein essential for skeletal bones formation is produced and embryo stops to develop and dies.

Cystic fibrosis is fatal to every homozygous recessive person by age 30. Sticky mucus accumulates in the lungs giving rise to frequent and chronic respiratory infections. It is caused by chloride ion channels malfunctioning in ducts.

Congenital ichthyosis. Children are born with crusted leathery skin with deep splits. These splits lead to bleeding, infection and death. The skin's natural shedding process is slowed or inhibited and in some types, skin cells are produced too rapidly. Most types of ichthyosis are treated by suppression of scale build up and humidification of the underlying skin.

Recessive or dominant, the lethal genes are commonly fatal only in the homozygous condition. Heterozygotes may display a form of disease phenotype, as in the case of *acatalasia* [4] or *achondroplasia* [1, 5]. Some lethal alleles produce a recognizable phenotype in the heterozygote, as in the yellow mouse and *Manx cat* (see Problems 15 and 16 here). However, some lethal alleles are fully dominant, killing in one dose in the heterozygote and some confer no detectable effect in the heterozygote at all, and the lethality is recessive [1].

We see, the lethals differ in the developmental stage at which they manifested. Some are expressed as deaths in uterus, where they usually unnoticed, or regarded as spontaneous abortions. Other as those responsible for *Duchenne muscular dystrophy*, phenylketonuria, and cystic fibrosis, exert their effects in childhood. The Huntington's disease leads to death in adulthood. The sum of all the deleterious and lethal genes that are present in individuals is called **genetic load**. It has been estimated that we each carry a small number of recessive lethals in our genomes [1].

Usually, lethal alleles kill, because of deficiency in essential chemical reaction or structural defect (e.g., Sickle-cell anemia, phenylketonuria, abnormal cartilage, etc.).

Whether an allele is lethal often depends on the environment in which the organism develops. However, certain alleles would be lethal in virtually any environment [1].

The favism is an example of a **sex-linked conditional lethal**, conferring to carrier glucose-6-phosphate dehydrogenase deficiency that causes the organism to develop hemolytic anemia after eating of fava beans (*Vicia faba*). An estimated 400 million people on the globe have this lethal [6].

Sometime, the lethal allele is said to be **subvital**, or **semilethal**, because the lethality is expressed in only some individuals. Thus, lethality may range from 0 to 100%, depending on the gene itself, the rest of the genome, and the environment [1].

Dominance

Problem 1. Normal pigmentation (C) in human is dominant over albino (c). A diabetic man marries a normal woman whose mother was an albino and whose father was diabetic. What are the genotypes of the man and the woman? What proportion of their children would be expected to be both non-diabetic and have normal pigmentation?

Solution: We are looking at 2 traits in this question. The man is *ccdd*. The woman was normal (for both traits) so she must have at least 1 dominant allele for each character: *C_D_*.

♀ *CcDd* × ♂ *ccdd*

| ♀G: \ ♂G: | <i>cd</i> | Answer: ¼ of their children will be expected to be non-diabetic and have normal pigmentation. |
|---------------|-------------|---|
| <i>CD</i> , ¼ | <i>CcDd</i> | |
| <i>Cd</i> , ¼ | <i>Ccdd</i> | |
| <i>cD</i> , ¼ | <i>ccDd</i> | |
| <i>cd</i> , ¼ | <i>Ccdd</i> | |

She has a mother that is albino, supplied her with the recessive allele, *c*. Her father is diabetic, so he provided her recessive allele, *d*. Therefore her genotype, with respect to these two traits is *CcDd*.

Problem 2. A person with Rh+ blood has a specific protein in his/her blood. Persons with Rh- blood do not have this particular protein in their blood. Rh+ is dominant in respect to Rh-. Also, normal insulin production dominates over abnormal insulin production. If two individuals are heterozygous for Rh+ and insulin production, what probable phenotypes might their children be?

Solution: The individuals in question are heterozygous for both traits, so here we can regard the mating:

♀ *+ - Ii* × ♂ *+ - Ii*

| ♀G: \ ♂G: | <i>+I</i> , ¼ | <i>+i</i> , ¼ | <i>-I</i> , ¼ | <i>-i</i> , ¼ |
|---------------|--------------------|--------------------|--------------------|--------------------|
| <i>+I</i> , ¼ | <i>++II</i> , 1/16 | <i>++Ii</i> , 1/16 | <i>+-II</i> , 1/16 | <i>+-Ii</i> , 1/16 |
| <i>+I</i> , ¼ | <i>++Ii</i> , 1/16 | <i>++ii</i> , 1/16 | <i>+-Ii</i> , 1/16 | <i>+-ii</i> , 1/16 |
| <i>-I</i> , ¼ | <i>+-II</i> , 1/16 | <i>+-Ii</i> , 1/16 | <i>--II</i> , 1/16 | <i>--Ii</i> , 1/16 |
| <i>-I</i> , ¼ | <i>+-Ii</i> , 1/16 | <i>+-ii</i> , 1/16 | <i>--Ii</i> , 1/16 | <i>--ii</i> , 1/16 |

Answer: 9 Rh+, normal insulin production; 3 Rh+, diabetic; 3 Rh-, normal; 1 Rh-diabetic

Incomplete dominance and codominance

Problem 3. What progeny is expected from the couple in which father is heterozygote on Sickle cell anemia and mother is normal on this trait?

Solution: P: ♀ *Hb^AHb^A* × ♂ *Hb^SHb^A*
 G: *Hb^A* *Hb^S, Hb^A*

F1 : *Hb^AHb^S* - survive; *Hb^AHb^A* - no anemia, 1 : 1.

Answer $Hb^A Hb^S$ – no anemia, red blood cells sickle only under low oxygen concentration. Sickle cell disease is the result of **incomplete dominance** of Hb^A . However, in regard to hemoglobin we see **codominance**. Those individuals who have the disease carry both: 50% normal (fast in electrophoresis) and 50% changed (slow in electrophoresis) hemoglobin.

Problem 4. Mather has A group of the blood, the father has B. They have two children. The boy has A and his sister O group of blood. What are the genotypes of this couple? Can they have child with another group of blood?

Solution: The problem's information suggests the mating: female II(A) \times male III (B)

P: ♀ $I^A I^O \times \text{♂ } I^B I^O$ or P: ♀ $I^A I^A \times \text{♂ } I^B I^B$
 G: I^A, I^O I^B, I^O G: I^A I^B
 F: $I^O I^O$ (I); $I^A I^O$ (II); $I^B I^O$ (III); $I^A I^B$ (IV). F: $I^A I^B$ (IV).

Answer: They can produce the child of any of the groups.

Problem 5. A man with blood group A marries a woman with blood group B. Their child has group O blood. What are the genotypes of these individuals? What other genotypes and in what frequencies would you expect in offspring from this marriage?

Solution: Based on the information provided above, we can summarize.

| Genotype | Phenotype | Type of genes interaction |
|------------------------|-----------|---------------------------|
| $I^A I^A$ or $I^A I^O$ | A | A dominance over O |
| $I^B I^B$, $I^B I^O$ | B | B dominance over O |
| $I^A I^B$ | AB | A and B codominance |
| $I^O I^O$ | O | |

In order for someone to have type O blood, they must be $I^O I^O$ (or in other symbols - ii) by genotype. Therefore, they must have received one O allele from one parent and the other O allele from the other parent. In this marriage the father must be $I^A I^O$ and the mother must be $I^B I^O$. To determine the genotypes in the offspring of this couple, you can draw up the Punnett square:

♀ $I^B I^O \times \text{♂ } I^A I^O$.

| | | | |
|--------------------|------------------------|------------------------|--|
| ♂ G: | $I^A, \frac{1}{2}$ | $I^O, \frac{1}{2}$ | <u>Answer.</u> We would expect $\frac{1}{4} I^A I^B$; $\frac{1}{4} I^B I^O$; $\frac{1}{4} I^A I^O$; $\frac{1}{4} I^O I^O$. |
| ♀ G: | | | |
| $I^B, \frac{1}{2}$ | $I^A I^B, \frac{1}{4}$ | $I^B I^O, \frac{1}{4}$ | |
| $I^O, \frac{1}{2}$ | $I^A I^O, \frac{1}{4}$ | $I^O I^O, \frac{1}{4}$ | |

Problem 6. In a maternity ward, 4 babies become accidentally mixed up. The ABO types of 4 babies are known to be O, A, B, and AB. The ABO types of the 4 sets of parents are determined. Which baby belongs to each set of parents: a) $AB \times O$, b) $A \times O$, c) $A \times AB$, d) $O \times O$ [1]?

Solution: If we write the crosses for each set of parents, we can estimate the family for each child (table 3).

Table 3. Parents and their children

| Babies | Parents |
|--------|---------------|
| O | $O \times O$ |
| A | $A \times O$ |
| B | $AB \times O$ |
| AB | $A \times AB$ |

Problem 7. Two men took a paternity dispute to court, each claiming three children to be his own. The blood groups of the men, the children, and their mother were as indicated in table 3:

Table 4. Blood types of children and parents

| Person | Blood group | | |
|--------------|-------------|-----------|------------|
| Husband | O | <i>M</i> | <i>Rh+</i> |
| Wife's lover | AB | <i>MN</i> | <i>Rh-</i> |
| Wife | A | <i>N</i> | <i>Rh+</i> |
| Child 1 | O | <i>MN</i> | <i>Rh+</i> |
| Child 2 | A | <i>N</i> | <i>Rh+</i> |
| Child 3 | A | <i>MN</i> | <i>Rh-</i> |

From this evidence, can the paternity of the children be established [1]?

Solution: To estimate the paternity, we must take into account that *Rh+* dominates over *Rh-*; alleles *MN* are codominant; alleles I^A and I^B are fully dominant in genotypes $I^A i$ and $I^B i$, but codominant in genotype $I^A I^B$ (AB blood group) and *i* genotype determines O group of the blood, the inability to produce antigen; $I^A i$ or $I^A I^A$ and $I^B i$ or $I^B I^B$ are for the A and B groups, respectively.

On the basis of this information we can conclude the paternity of the children (table 5):

Table 5. Paternity of the disputed children

| Children | Blood group | | | Father | Answer. Only 1 st child belongs to husband and other two of children are from the lower. |
|----------|-------------|-----------|------------|--------------|--|
| Child 1 | O | <i>MN</i> | <i>Rh+</i> | Husband | |
| Child 2 | A | <i>N</i> | <i>Rh+</i> | Wife's lover | |
| Child 3 | A | <i>MN</i> | <i>Rh-</i> | Wife's lover | |

Problem 8. Sickle cell anemia is inherited as autosomal trait with incomplete dominance. Homozygous individuals generally die before puberty; heterozygous are viable, the anemia in them is often subclinical to mild. The malarial plasmodium cannot feed on hemoglobin of heterozygotes. The people with this form of hemoglobin can resist malaria. What is the likelihood of having children resistant to malaria in families where both parents are heterozygous on the gene of sickle cell anemia [4]?

Solution: Hb^A - fast hemoglobin, normal gene condition; Hb^S - slow, abnormal hemoglobin. Thus, $Hb^A Hb^A$ is healthy individual, $Hb^A Hb^S$ - mild anemia, resistance to malarial *Plasmodium* and $Hb^S Hb^S$ is lethal condition.

Parents, P: ♀ $Hb^A Hb^S$ × ♂ $Hb^A Hb^S$
 Gametes, G: Hb^A, Hb^S Hb^A, Hb^S
 Children, F: 1 $Hb^A Hb^A$: 2 $Hb^A Hb^S$: 1 $Hb^S Hb^S$ **Answer:** 50%.

Problem 9. Familial **hypercholesterinemia** is an abnormally high level of cholesterol in the blood. This disease is inherited as dominant autosomal character. In heterozygotes the disease appears as high cholesterol level in blood and homozygotes develop atherosclerosis and xanthomas (benign tumors) in skin and tendons. Determine the severity of possible hypercholesterinemia development in children if both parents have just high level of cholesterol in the blood [2].

| | |
|---|--|
| Solution: <i>C</i> - dominant allele of familial hypercholesterinemia; <i>c</i> -recessive allele; <i>CC</i> - atherosclerosis and xanthomas; <i>Cc</i> - high cholesterol level in blood, Parents, P: ♀ <i>Cc</i> × ♂ <i>Cc</i> Gametes, G: <i>C, c</i> <i>C, c</i> Children, F: 1 <i>CC</i> (25%) : 2 <i>Cc</i> (50%) : 1 <i>cc</i> (25%) | Answer: The probability of completely healthy children is 25%, the probability of high cholesterol level in blood is 50% and the chance to have severe familial hypercholesterinemia , the atherosclerosis and xanthomas is 25%. |
|---|--|

Problem 10. Both grandfathers have just wavy hair and both grandmothers have curly hairs. The mother of the child has wavy hair, and the both: father and child have curling hair. Determine

the genotypes of all individuals. What is the probability of the birth of the next child with wavy hair and with straight hair (curly hair gene dominates incompletely over the gene of straight hair) [2]?

| | |
|---|---|
| <p><u>Solution:</u> C - curly hair gene; c – recessive allele. CC - curly hair; Cc - wavy hair; cc - straight hair. Grandparents, GP: ♀CC × ♂Cc Parents, P: ♀Cc × ♂CC Gametes, G: C, c C 1st Child CC Children, F: 1 CC (50%) : 1 Cc (50%).</p> | <p><u>Answer:</u> The probability of child birth with curled hair is 50% and it is not possible to have child with strait hair.</p> |
|---|---|

Problem 11. A is autosomal gene with incomplete dominance. When in homozygous state, it causes severe deformation of limbs, but in heterozygotes only brachydactyly develops, the shortening of the fingers and toes due to unusually short bones. Identify possible phenotypes of children in the family if one parent is healthy, and another has brachydactyly [2].

| | |
|--|--|
| <p><u>Solution:</u> A - the gene that determines the development of skeleton anomalies; a - gene that determines their absence; AA - deformation of extremities; Aa - shortening of the fingers; aa - normal skeleton. Parents, P: ♀Aa × ♂aa Gametes, G: A, a a Children, F: 1 Aa (50%) : 1 aa (50%).</p> | <p><u>Answer:</u> Probability of children with normal skeleton in the family is 50% and of children with brachydactyly also 50%.</p> |
|--|--|

Problem 12. Acatalasia caused by a rare autosomal recessive gene. In heterozyhote catalase activity decreased slightly. A woman with a normal catalase activity have husband with low catalase activity. What is the probability of children birth without anomaly in the family, if grandparents from both sides have reduced activity of catalase [2]?

| | |
|--|---------------------------|
| <p><u>Solution:</u> A - the gene that determines the normal activity of catalase; and - the gene that determines Acatalasia; AA- normal catalase activity Aa- reduced catalase activity aa acatalasia. Grandparents, GP: Aa × Aa Aa × Aa Parents, P AA × Aa Gametes: A A, a Children, F AA : Aa or 1:1</p> | <p><u>Answer:</u> 50%</p> |
|--|---------------------------|

Lethal alleles

Problem 13. Pelger-Huët anomaly involves an arrest of the segmentation of the nuclei of certain white cells. It was first reported in 1928 by Pelger, a Dutch hematologist, who described neutrophils with dumbbell-shaped bilobed nuclei, a reduced number of nuclear segments, and coarse clumping of the nuclear chromatin. The nuclei of several types of white blood cells (neutrophils and eosinophils) have unusual shape (bilobed, peanut or dumbbell-shaped instead of the normal trilobed shape) and unusual structure (coarse and lumpy).

In 1931 Huët, a Dutch pediatrician, identified it as an inherited disorder. The disease was previously thought to be inherited in an autosomal dominant manner; however, **co-dominant inheritance** has been suggested also [7, 8]. In homozygotes on this basis segmentation of nuclei completely absent and in heterozygotes it is unusual.

Determine the character of segmentation of nuclei in leucocytes of grandchildren, knowing that mother of children and her parents have unusual segmentation of leukocytes' nuclei and father of children and his parents are normal in segmentation of nuclei of leukocytes.

| | |
|---|---|
| <p><u>Solution:</u></p> <p>P- Pelger anomaly gene, problems with segmentation of leukocyte nuclei; N- gene normal segmentation of nuclei of leukocytes; PP- complete lack of segmentation in leukocyte nuclei; PN- unusual segmentation of nuclei of leukocytes; NN - normal segmentation of nuclei of leukocytes.</p> <p>GP: ♀ $PN \times PN$ ♂ ♀ $NN \times \text{♂} NN$ P: ♀ PN × ♂ NN G: P, N N</p> <p>F1: PN - 50% unusual segmentation of nuclei of leukocytes NN - 50% normal segmentation of nuclei of leukocytes</p> | <p><u>Answer:</u></p> <p>The chances to have grandchildren with normal segmentation leukocyte nuclei is 50%, and with unusual segmentation - 50%.</p> |
|---|---|

Problem 14. Hans Nachtsheim investigated an inherited anomaly of the white blood cells of rabbits. This anomaly, termed the Pelger anomaly, involves an arrest of the segmentation of the nuclei of certain white cells. The disease does not appear to seriously inconvenience the rabbits.

a. When rabbits showing the typical Pelger anomaly were mated with rabbits from a truebreeding normal stock, Nachtsheim counted 217 offspring showing the Pelger anomaly and 237 normal progeny. What appears to be the genetic basis of the Pelger anomaly?

b. When rabbits with the Pelger anomaly were mated to each other, Nachtsheim found 223 normal progeny, 439 showing the Pelger anomaly, and 39 extremely abnormal progeny. These very abnormal progeny not only had defective white blood cells but also showed severe deformities of the skeletal system; almost all of them died soon after birth. In genetic terms, what do you suppose these extremely defective rabbits represented? Why do you suppose there were only 39 of them?

c. What additional experimental evidence might you collect to support or disprove your answers to part b?

d. In Berlin, about one human in 1000 shows a Pelger anomaly of white blood cells very similar to that described in rabbits. The anomaly is inherited as a simple dominant (or codominant [4], but the homozygous type has not been observed in humans. Can you suggest why, if you are permitted an analogy with the condition in rabbits?

e. Again by analogy with rabbits, what phenotypes and genotypes might be expected among the children of a man and woman who both show the Pelger anomaly [9]?

Solution:

a. 217 offspring showing the Pelger anomaly and 237 of young rabbits are normal progeny. Here is relation close to 1:1 ratio. As pure line was mated with Pelger anomaly animals, we conclude, the cross was $PN \times NN$, where P is Pelger anomaly and N is for no disorder. Progeny: $PN : NN$ as 1:1.

b. This is result of heterozygotes cross, $PN \times PN$; 439 are PN , the Pelger anomaly; 39 are PP - extremely abnormal progeny; 223 are normal progeny – NN .

c. We can make analyzing cross of animals participating in mating (b) with pure healthy line (NN). If we observe 1:1 ratio (healthy: Pelger anomaly) in result, the animals in cross (b) are PN , the heterozygotes.

d. The zygotes with two copies of the P gene will be reabsorbed, or fetus stillborn or die shortly after birth.

e. In this case we can presume the ratio $PP: PN: NN$ as 1:2:1. However, PP are not alive, only PN and NN progeny viable.

Problem 15. In 1904, Lucien Cuenot studied mice having yellow coat color. The mating of yellow mouse to a gray mouse from a pure line resulted in 1:1 ratio (yellow : normal mice) in the progeny, suggesting that a single gene determines these phenotypes, that the yellow mouse was heterozygous for this gene, and that the allele for yellow is dominant to an allele for normal color. However, when Cuenot crossed yellow mice with one another the result was always the same:

Yellow × yellow → 1/3 normal color 2/3 yellow

When he studied of uteri from pregnant females of the *yellow × yellow* cross; one-fourth of the embryos were found to be dead. In 1910, W.E.Castle and C.C.Little confirmed Cuénot's work, further demonstrating that one quarter of the offspring were dying during embryonic development. How the L. Cuenot's results can be explained?

Solution: Note first, the 2:1 ratio is a departure from Mendelian expectations. Second, because no cross of *yellow × yellow* ever produced all yellow progeny, as there would be if either parent were a homozygote, it appeared that there were no homozygous yellow mice.

The heterozygotes cross would be expected to yield a genotypic ratio of 1:2:1. If all of the mice in one of the homozygous classes died in uterus, the live births would then show a 2:1 ratio of heterozygotes to homozygotes. The allele A^Y for yellow might be dominant to the normal allele A in its effect on color, but it might act as a recessive **lethal** allele with respect to viability. Thus, $A^Y A^Y$ genotype mice die before birth and are not observed in progeny. All surviving yellow mice were heterozygous $A^Y A$, so a cross between yellow mice will always yield the following results:

$$A^Y A \times A^Y A \rightarrow \begin{array}{l} \frac{1}{4} AA \text{ normal color} \\ \frac{1}{2} A^Y A \text{ yellow} \\ A^Y A^Y \text{ die before birth} \end{array}$$

The Mendelian ratio of 1:2:1 would be observed among the zygotes, but it is altered to a 2:1 ratio, because the $A^Y A^Y$ genotype progeny do not survive.

Problem 16. The tailless Manx phenotype (absence of tail in cats) is produced by an allele that is lethal in the homozygote state. A single dose of the Manx allele, M^L , severely interferes with normal spinal development, resulting in absence of a tail in the $M^L M$ heterozygote. Two tailless Manx cats were crossed? What kind of progeny is expected?

Solution:

P: ♀ $M^L M$ × ♂ $M^L M$

G: M^L, M M^L, M

F1: $1M^L M^L$ (died in uterus) $2M^L M$ (tailless) $1MM$ (normal)

In $M^L M^L$ homozygotes, the double dose of the gene produces such extreme developmental abnormality that the embryos not survive [1].

Problems for individual work of students:

Problem 17. Mother has III group of blood and father has IV. What blood groups can be in their children?

Problem 18. Father has AB NN blood group and mother has O MM group their children have A MN and B MN. Which child is adopted?

Problem 19. At which genotypes of parents children can't inherit the same blood group?

Problem 20. The court hears the case of paternity. Mother has 1st group of blood, her baby has a 2nd group of blood. Can a man be the father of a child, if he has 3rd blood group. What blood group may be in natural father of the child?

Problem 21. In one family both parents have hazel eyes. This character is inherited as autosomal and dominant in respect to blue eyes. Family has 4 children 2 of them are blueeyed and have 1st and 2nd group of blood and other 2 have hazel eyes and 2nd and 3rd blood groups. Determine the probability of hazel eyes and 1st blood group in next child.

Problem 22. The recessive gene of anencephaly causes phetus death in homozygotes, while in heterozygotes is manifested as *spina bifida* of different severity. *Spina bifida* is a birth defect where there is incomplete closing of the backbone and membranes around the spinal cord. The condition often leads to death. Young phamily need counsulting, because two of their children were stillborn with anencephaly and one is alive, however has severe case of *spina bifida*. What is the probability of healthy child birth?

Literature

1. Griffiths Anthony J.F. An Introduction to genetic analysis.- 5th edition Freeman and Company. – 1993.- 840 p.
2. Gluecksohn-Waelsch, Salome. Lethal Genes and Analysis of Differentiation// Science.- 1963.- Vol. **142** (3597).- p. 1269–76. doi:10.1126/science.142.3597.1269. PMID 14074837
3. Cuénot L. L'heredite de la pigmentation chez les souris//Arch Zoo Exp Get.- 1904.- Vol. 2.- P. 45–56.
4. Барна І.В. Загальна біологія: збірник задач.- Тернопіль: підручники і посібники, 2013.- 736 с.
5. Lobo I. Mendelian Ratios and Lethal Genes. Nature Education. 2008- Vol. **1**.- N1.- P.138.
6. Glucose-6-phosphate dehydrogenase deficiency. Genetics Home Reference.- <https://ghr.nlm.nih.gov/condition/glucose-6-phosphate-dehydrogenase-deficiency>
7. Nachtsheim H. The Pelger-Huët anomaly in man and rabbit. A Mendelian character of the nuclei of leukocytes// J. Hered.- 1950.- Vol.41.- P.131-137.
8. Borovik L., Modaff P., Waterham H.R., Krentz A.D., Pauli R.M. Pelger-Huët anomaly and a mild skeletal phenotype secondary to mutations in LBR// *American Journal of Medical Genetics*. - 2013.- Vol. 161A.- N8.- P. 2066-2073. <http://www.ncbi.nlm.nih.gov/pubmed/23824842>.
9. Srb A.M., Owen R.D., Edgar R.S. General Genetics, 2d ed. W. H. Freeman and Company, 1965.