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MORPHOLOGICAL CHARACTERISTICS OF THE MYOCARDIAL CONTRACTILE APPARATUS IN RATS UNDER THE INFLU- ENCE OF HYPOXIA (LITERATURE RE- VIEW AND RESEARCH PERSPECTIVES)

Kobeza P.A.  , Cherkas O.A. , Marchenko D.G. , Khripkov I.S.  Morphological characteristics of the myocardial contractile apparatus in rats under the influence of hypoxia (literature review and research perspectives). Dnipro State Medical University, Dnipro, Ukraine.

ABSTRACT. Background. Conducting model experiments on different age groups of laboratory rats under chronic hypoxic conditions is relevant and aids in studying structural changes in cardiac tissues from a scientific perspective. Understanding the mechanisms of heart adaptation to hypoxia. Research into cardiac adaptation to hypoxia helps uncover the mechanisms that enable the heart to function under low oxygen conditions. Studies on different age groups of rats can help determine how age affects adaptation and the ultrastructure of the heart during hypoxia. **The objective** of this study is to understand the impact of hypoxia on the hearts of rats at different stages of their lives, including neonates, young, and adult animals. This helps expand our knowledge of the adaptive mechanisms of the heart and may have significant implications for the normal anatomy of laboratory animals, their biological significance, and a comprehensive understanding of the ultrastructural remodeling of the components of the myocardial contractile apparatus. **Methods.** Systematic Literature Review, Meta-Analysis, Content Analysis, Assessment of Source Quality, Information Retrieval. **Results.** Chronic hypoxia affects the structure of the myocardial contractile apparatus in rats during different stages of development (from birth to 6 months) as follows: Newborn rats: Hypoxia can lead to myocardial hypertrophy and an increase in the number of capillaries to improve blood supply. 1 month after birth: Under hypoxia, there is myocardial hypertrophy, an increase in the amount of fibrous tissue, and possible vessel remodeling. 3 months after birth: Hypoxia can result in further enlargement of the myocardium, changes in structure and functional parameters, and the development of new vessels. 6 months after birth: At this stage, significant changes are possible, including hypertrophy, vessel remodeling, alterations in functional parameters, and structural peculiarities of the myocardium. **Conclusion.** Chronic hypoxia affects the morphology of the myocardium in rats at different stages of their development, leading to changes in size, structure, and cardiac functionality.

Key words: tissue hypoxia, cardiomyocyte, contractile apparatus, ultrastructure, myofibrils.

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Introduction

Hypoxia, characterized by insufficient oxygen supply to the body's tissues, is one of the key issues in modern normal anatomy, cell biology and histology. It can arise from various physiological and

pathological processes, and its consequences for tissues and organs can be severe [1]. One of the most vulnerable organs to hypoxia is the heart. The cardiac muscle, or myocardium, requires a constant supply of oxygen for its normal function as it is respon-

sible for the continuous mechanical work of the heart. Hypoxia can lead to a range of changes in the myocardium, including structural and functional adaptations that can affect its performance [2]. This scientific publication is dedicated to the study of the morphological characteristics of the contractile apparatus of the myocardium in rats subjected to hypoxia. Our work aims to uncover the changes that occur in the structure and organization of the myocardium under the influence of hypoxia at different stages of rat development, from neonates to adulthood.

Background

Conducting model experiments on different age groups of laboratory rats under chronic hypoxic conditions is relevant and aids in studying structural changes in cardiac tissues from a scientific perspective. Understanding the mechanisms of heart adaptation to hypoxia. Research into cardiac adaptation to hypoxia helps uncover the mechanisms that enable the heart to function under low oxygen conditions. Hypoxia can lead to various pathological changes in cardiac muscle. Investigating these changes can lead to an understanding of the causes and mechanisms of cardiovascular diseases associated with hypoxia. Age can influence the heart's response to hypoxia. Studies on different age groups of rats can help determine how age affects adaptation and the ultrastructure of the heart during hypoxia. The overall relevance lies in advancing our understanding of the heart's response to hypoxia.

Research of this kind is of great importance for our understanding of the adaptive mechanisms of the myocardium and its response to stressful conditions such as hypoxia. The obtained results will contribute to improving our understanding of the impact of hypoxia on the structure and function of the myocardium and the components of the heart's contractile apparatus at various levels of its organization, from tissue to ultrastructure.

Objective

The aim of this study is to understand the impact of hypoxia on the hearts of rats at different stages of their lives, including neonates, young, and adult animals. This helps expand our knowledge of the adaptive mechanisms of the heart and may have significant implications for the normal anatomy of laboratory animals, their biological significance, and a comprehensive understanding of the ultrastructural remodeling of the components of the myocardial contractile apparatus.

Research on the influence of hypoxia on the hearts of laboratory rats of different ages may encompass the following aspects [3]: Assessment of Morphological Changes: The study aims to determine specific morphological changes occurring in the structure of the hearts of rats of different ages under the influence of hypoxia [4]. This may include changes in the size and shape of the heart, myocardial structure, vessels, and cardiomyocytes. Func-

tional Changes: Evaluation of the impact of hypoxia on the functional characteristics of the heart, such as cardiac output, rhythm, and overall cardiac performance [5]. Analysis of Molecular Mechanisms: Investigation of the molecular mechanisms underlying the changes in the heart due to hypoxia [6]. This may involve studying gene expression, biochemical alterations, and other molecular processes. Age-Comparative Analysis: Comparing the effects of hypoxia on the hearts of rats at different ages to determine potential differences in responses between adult and young animals [7]. Development of Research Strategies: Developing potential research strategies for modeling experiments to determine aspects of ultrastructural remodeling of myocardial components related to hypoxia based on the study's findings. This research contributes to our understanding of how hypoxia affects the hearts of rats at various developmental stages, shedding light on both morphological and functional adaptations and providing insights into the molecular mechanisms involved.

Materials and methods

When writing an analytical scientific article based on the analysis of scientific papers, it is important to use appropriate research methods to ensure that the article is well-grounded and reliable. The following methods are crucial in conducting such research:

1. Systematic Literature Review: Conducting a structured search and analysis of relevant sources to gather all available information on a specific topic.
2. Meta-Analysis: Using statistical methods to combine and analyze data from multiple studies to obtain reliable conclusions.
3. Content Analysis: Studying the content of scientific articles to identify themes, patterns, and relationships among variables.
4. Assessment of Source Quality: Considering the relevance and quality of literature sources, including publications in peer-reviewed scientific journals.
5. Information Retrieval: Utilizing databases and websites to search for relevant literature, including PubMed, ScienceDirect, and Google Scholar.

It is also essential to consider the publication date and examine the bibliography to find additional sources of information. These research methods help ensure the rigor and validity of the analytical scientific article based on the analysis of existing literature.

Results and discussion

The morphological characteristics of the myocardial contractile apparatus in rats under hypoxia can exhibit several changes and features, as hypoxia (oxygen deficiency) can significantly impact the functioning of the cardiac muscle [8]. Here are some possible alterations in the structure and function of the myocardial contractile apparatus during hypoxia:

1. **Changes in Cellular Composition [9]:** Under the influence of hypoxia, apoptosis (programmed cell death) of cardiomyocytes, the heart muscle cells, may occur, resulting in the loss of viable cells and a reduction in the number of active muscle fibers.

2. **Changes in Cell Morphology [10]:** Hypoxia can lead to alterations in the appearance and structure of cardiomyocytes, including an increase in cell size and an accumulation of vacuoles in their cytoplasm.

3. **Altered Intercellular Connections:** Hypoxia can affect intercellular junctions, such as desmosomes and their adhesive proteins [11]. This can lead to the disruption of connections between cells, potentially affecting signal transmission and contraction coordination.

4. **Functional Changes in the Contractile Apparatus:** Hypoxia can result in impaired contractility of the myocardium, reducing its ability for rhythmic contractions and lowering cardiac output [12].

5. **Vascular Changes:** Hypoxia may trigger angiogenesis, the formation of new blood vessels, as an attempt by the body to restore oxygen supply to the heart [13].

6. **Inflammatory Processes:** Hypoxia can activate inflammatory processes in cardiac tissues, which can influence the structure and function of the contractile apparatus [14].

In summary, hypoxia can induce various changes in the structure and function of the myocardial contractile apparatus, which may lead to disruptions in heart function and its ability to pump blood effectively [15]. These changes warrant further investigation in histology and morphology to understand the mechanisms occurring in the heart during hypoxia.

The Impact of Hypoxia on the Myocardial Contractile Apparatus should be considered comprehensively [16]. The myocardial contractile apparatus is a complex system of structures responsible for the rhythmicity and coordination of heart muscle (myocardium) contractions. This apparatus includes the following components:

1. **Sinoatrial Node (SA Node or Sinus Node):** The SA node is located in the upper part of the right atrium, near the open upper chamber of the heart. It generates electrical impulses that determine the rhythm of heart contractions and is considered the "primary pacemaker" of the heart [17].

2. **Atrioventricular Node (AV Node):** Situated in the lower part of the right atrium, the AV node delays the electrical impulse, transmitting it downward through the conduction pathway to ensure a brief pause between atrial and ventricular contractions. This pause allows for efficient blood transfer from the atria to the ventricles [18].

3. **Bundle of His (AV Bundle):** The AV bundle is located in the wall between the atria and ventricles. It consists of fibers that transmit electrical impulses from the AV node to the myocardium of

the ventricles [19].

4. **Purkinje Fiber System:** The Purkinje fiber system is a network of conducting fibers that divides into numerous branches, spreading along the walls of the ventricles [20]. These branches distribute electrical impulses to coordinate and facilitate contractions of the ventricular myocardium.

5. **Myocardium:** The myocardium is the cardiac muscle that contracts upon receiving electrical impulses from various parts of the contractile apparatus [21]. The myocardium performs the primary function of the heart, which is pumping blood.

6. **Intercellular Junctions:** The contractile apparatus also includes intercellular junctions, such as desmosomes, which provide strong connections between cardiomyocytes (myocardial cells) and facilitate the transmission of contractile signals from one cell to another [22].

Together, these components allow for the perception and transmission of electrical signals that coordinate the rhythm and force of heart contractions [23], efficiently pumping blood throughout the body. Structural components of cardiomyocytes that participate in the process of contraction are highly susceptible to the influence of hypoxia, resulting in their active remodeling at the ultrastructural level of cell organization [24]. Cardiomyocytes are the cells of the heart muscle that participate in the process of heart contraction. The structural components of cardiomyocytes that play a key role in contraction include the following:

1. **Myofibrils:** Myofibrils are long fibers composed of myosin and actin proteins. They form the primary structural component of cardiomyocytes and are responsible for the cell's ability to contract. In response to electrical signals, myofibrils undergo displacement relative to each other, leading to muscle cell contraction [25].

2. **Sarcomeres:** Sarcomeres are the fundamental functional units of myofibrils. They consist of striated bands of myosin and actin filaments that slide relative to each other during muscle contraction. Sarcomeres determine the length of the myofibril and are responsible for its contraction [26].

3. **Sarcoplasmic Reticulum (SR):** The SR is a network of membrane layers within the intracellular reticulum that surrounds sarcomeres. It stores calcium, which is necessary for the functioning of the contractile apparatus [27]. Under the influence of electrical signals, calcium is released from the SR, triggering myofibril contraction.

4. **T-Tubules (Transverse Tubules):** T-tubules are membranes that penetrate deep into cardiomyocytes and facilitate the even distribution of electrical signals throughout the cell [28]. They play a crucial role in coordinating the contraction of myofibrils and the sarcoplasmic reticulum.

5. **Mitochondria:** Mitochondria generate the energy required for heart muscle contraction. Since the heart is a constantly working organ, it requires a

significant amount of energy to sustain continuous contraction, and mitochondria serve this function [29].

6. **Desmosomes and Adherens Junctions:** These structures provide strong adhesion between cardiomyocytes and allow the transmission of contraction signals from one cell to another [30]. They ensure the coordination of many cells' work during heart contraction.

These structural components collectively enable cardiomyocytes to perform their primary function—generating rhythmic and coordinated contractions that facilitate blood flow throughout the body [31]. The morphology of cardiomyocyte myofibrils in laboratory rats under the influence of chronic acute hypoxia can undergo changes during the first 6 months of life. Here's how these changes may appear:

Newborn rats (1 week old): Under the influence of hypoxia, myofibrils may be less developed and less organized [32]. However, these changes at this stage may be moderate, as the heart is just beginning to develop. **Young rats (1 month old):** Hypoxia can lead to further destruction and disorganization of cardiomyocyte myofibrils. Changes in the length and thickness of myofibrils may be possible. **Young rats (3 months old):** Due to prolonged hypoxia, myofibrils may become elongated but still disorganized. This may be an attempt at myocardial adaptation to insufficient oxygen supply [33]. **Adult rats (6 months old):** At this stage, the morphology of cardiomyocyte myofibrils may reflect adaptation to hypoxia, with increased length and improved organization of myofibrils.

In general, chronic acute hypoxia can affect the morphology of cardiomyocyte myofibrils, leading to their disorganization and changes in length and thickness. Myocardial adaptation may result in certain changes in myofibrils aimed at supporting heart function under inadequate oxygen load. For a more precise examination of these changes, it is recommended to conduct microscopic analysis of heart tissues during hypoxia experiments and compare them with normal conditions [34].

Chronic hypoxia affects the structures of the myocardial contractile apparatus in the left and right ventricles of rats during the first 6 months of life as follows: **Newborn rats (1 week old):** Hypoxia can lead to myocardial hypertrophy in both ventricles. Changes in myocardial structure, including increased fibrous tissues and possible vascular remodeling to improve blood supply [35]. **Young rats (1 month old):** Under the influence of hypoxia, myocardial hypertrophy in both ventricles is observed. Changes in myocardial structure, including increased fibrous tissues and potential vessel remodeling. **Young rats (3 months old):** Hypoxia may lead to further myocardial hypertrophy in both ventricles. Changes in myocardial structure, development of new vessels, and alterations in functional parameters

may occur [36]. **Adult rats (6 months old):** Myocardial hypertrophy in both ventricles may be possible due to hypoxia. Vessel remodeling, changes in myocardial morphology, and potential alterations in functional characteristics [37].

Thus, chronic hypoxia affects the structures of the myocardial contractile apparatus in the left and right ventricles, leading to hypertrophy, vascular remodeling, morphological changes, and potential alterations in functional characteristics during the first 6 months of their life [38]. The process of chronic hypoxia affects the structures of the myocardial contractile apparatus in the hearts of rats during the first 6 months of life as follows in the heart wall: **Newborn rats (1 week old):** Hypoxia may lead to myocardial hypertrophy and changes in the structure of cardiomyocytes and blood vessels. **Young rats (1 month old):** Under the influence of hypoxia, further myocardial hypertrophy occurs in both atria. Changes in myocardial structure, including fibrous tissues and vascular remodeling. **Young rats (3 months old):** Hypoxia may cause further changes such as myocardial hypertrophy in both atria. Development of new blood vessels, alterations in myocardial morphology, and functional characteristics. **Adult rats (6 months old):** Possible myocardial hypertrophy in both atria due to hypoxia. Vessel remodeling, changes in myocardial morphology, and potential alterations in functional characteristics of the atria [39].

In summary, chronic hypoxia affects the structures of the myocardial contractile apparatus in the hearts of rats in the atrial walls during the first 6 months of life, leading to hypertrophy, vascular remodeling, and changes in morphology that occur at different stages of heart development [40].

The observed clear interdependence between age groups and specific structural adaptations in the contractile apparatus of rat hearts under the influence of chronic acute hypoxia results in various changes during different periods: **Newborns:** Morphology of the contractile apparatus in the hearts of laboratory rats on the first day after birth under the influence of chronic acute hypoxia may differ from how it appears under normal developmental conditions [41]. Hypoxia, a condition where tissues receive insufficient oxygen, can impact the development and structure of the heart's contractile apparatus. Here are possible changes that can occur:

Changes in myocardial size and structure: Chronic acute hypoxia may lead to an increase in the size of the myocardium [42]. This could be an attempt by the heart to adapt to the insufficient oxygen by increasing the amount of muscle tissue to improve blood supply.

Changes in myocardial structure: Hypoxia can result in structural changes in the myocardium, such as an increase in the amount of fibrous (connective) tissue, which can affect the effectiveness of myocardial contraction [43].

Changes in intercellular junctions: Under the influence of hypoxia, the integrity of intercellular junctions, such as desmosomes and adherens junctions, may suffer [44]. This can lead to the disruption of connections between cardiomyocytes and a decrease in their coordination. Changes in mitochondria: Hypoxia can affect the function of mitochondria, potentially leading to disturbances in energy metabolism and reducing the ability of muscle cells to contract effectively [45]. Changes in the number and size of cardiomyocytes: Under the influence of hypoxia, the number and size of cardiomyocytes may change [46]. This can affect the overall structure and function of the heart. Changes in blood vessels: Hypoxia can induce adaptive changes in blood vessels, such as an increase in the number of capillaries to enhance better blood supply to the heart [47].

These changes can be either pathological or adaptive responses to hypoxia, depending on the duration and intensity of acute hypoxia [48]. To thoroughly study the morphology of the contractile apparatus in the hearts of laboratory rats one day after birth under the influence of hypoxia, it is recommended to conduct microscopic tissue analysis and study structural changes resulting from this condition.

One week after birth: The morphology of the myocardium in newborn rats and rats at one week of age under chronic acute hypoxia may significantly differ due to the influence of hypoxia on heart development and structure [49].

Here's a comparative characterization of myocardial morphology in these two groups: Myocardium of newborn rats (without hypoxia): Size and volume of the myocardium: In newborn rats, the myocardium typically has a small size and volume corresponding to their age [50]. Morphology of cardiomyocytes: Cardiomyocytes in newborns have small, immature nuclei, which is characteristic of early cardiac development [51]. Number of vessels and capillaries: Young myocardium has fewer blood vessels and capillaries compared to adults since vascular network development is still ongoing [52]. Myocardium of rats at 1 week of age under the influence of chronic acute hypoxia: Size and volume of myocardium: Under the influence of hypoxia, the myocardium may increase in size and volume [53]. This hypertrophy can be an adaptive response to insufficient oxygen load.

Changes in myocardial structure: Hypoxia can induce changes in the structure of the myocardium, including an increase in the amount of fibrous tissue or remodeling of blood vessels [54]. This can affect the morphology and functionality of the myocardium.

Changes in blood vessels: Hypoxia can lead to changes in blood vessels, such as an increase in the number of capillaries to improve myocardial blood supply [55].

Changes in mitochondria: Hypoxia can affect the function of mitochondria, which can result in reduced efficiency of energy exchange in myocardial cells.

Changes in the location and size of cardiomyocyte nuclei: Hypoxia can influence the location and size of cardiomyocyte nuclei. These changes in myocardial morphology after the birth of newborn rats under the influence of hypoxia can be an attempt by the organism to adapt to insufficient oxygen load [56]. To accurately study morphological changes, it is recommended to conduct microscopic analysis of heart tissues and compare their structural features in different groups of rats.

1 month after birth. The morphology of the myocardium of laboratory rats at 1 month of age under the influence of chronic acute hypoxia may differ from the myocardium of rats at 1 week of age under the same hypoxia. Prolonged hypoxia can lead to more significant changes in the structure and function of the heart [57].

Here is a comparative description of the myocardial morphology in these two groups. Myocardium of rats at 1 week of age under the influence of chronic acute hypoxia: Size and volume of myocardium: At this stage of heart development, the myocardium may be smaller in size compared to adult animals [58]. Morphology of cardiomyocytes: Cardiomyocytes may have immature nuclei and be less specialized compared to older individuals [59]. Number of vessels and capillaries: Young myocardium may have fewer blood vessels and capillaries compared to adult animals.

Myocardium of rats at 1 month of age under the influence of chronic acute hypoxia: Size and volume of myocardium: Under the influence of prolonged hypoxia, the myocardium may increase in size and volume due to hypertrophy. This adaptation is aimed at ensuring better oxygen supply [60]. Changes in myocardial structure: Hypoxia can lead to changes in myocardial structure, including an increase in the amount of fibrous tissue or vessel remodeling. Changes in blood vessels: Hypoxia can stimulate the development of new vessels or an increase in the number of capillaries to improve myocardial blood supply [61]. Changes in mitochondria: Hypoxia can affect the function of mitochondria, resulting in reduced efficiency of energy exchange in myocardial cells [62]. Changes in the location and size of cardiomyocyte nuclei: Under the influence of hypoxia, changes in the location and size of cardiomyocyte nuclei may be observed.

Overall, the morphology of the myocardium of laboratory rats at 1 month of age under chronic acute hypoxia differs from the myocardium of rats at 1 week of age under the same hypoxia due to the longer duration and intensity of hypoxia, which can lead to more significant changes in the structure and function of the heart for adaptive purposes [63].

3 months after birth. The morphology of the

myocardium of laboratory rats at 3 months of age under the influence of chronic acute hypoxia may differ from the myocardium of rats at 1 month of age under the same hypoxia due to the duration of exposure to hypoxia and the longer period of heart development [64]. Here is a comparative description of myocardial morphology in these two groups:

Myocardium of rats at 1 month of age under the influence of chronic acute hypoxia. Size and volume of myocardium: At this stage of heart development, the myocardium may be smaller in size compared to adult animals [65]. Morphology of cardiomyocytes: Cardiomyocytes may have immature nuclei and be less specialized compared to older individuals. Number of vessels and capillaries: Young myocardium may have fewer blood vessels and capillaries compared to adult animals.

Myocardium of rats at 3 months of age under the influence of chronic acute hypoxia: Size and volume of myocardium: Under the influence of prolonged hypoxia, the myocardium may increase in size and volume due to hypertrophy [66]. At this age, the heart is more developed compared to younger individuals. Changes in myocardial structure: Hypoxia can lead to changes in myocardial structure, including an increase in the amount of fibrous tissue or vessel remodeling [67]. Changes in blood vessels: Hypoxia can stimulate the development of new vessels or an increase in the number of capillaries to improve myocardial blood supply. Changes in mitochondria: Hypoxia can affect the function of mitochondria, resulting in reduced efficiency of energy exchange in myocardial cells.

Changes in the location and size of cardiomyocyte nuclei: Under the influence of hypoxia, changes in the location and size of cardiomyocyte nuclei may be observed.

These changes in myocardial morphology at 3 months of age under chronic acute hypoxia may indicate adaptation of the heart to insufficient oxygen load over an extended period [68]. To accurately study morphological changes, it is recommended to conduct microscopic analysis of heart tissues and compare their structural features in different groups of rats [69-73].

The morphology of the myocardium of laboratory rats at 6 months of age under the influence of chronic acute hypoxia may exhibit certain changes compared to normal myocardial development [74]. Chronic acute hypoxia, which persists for an extended period, can impact the structure and function of the heart. Here are possible changes in myocardial morphology in this group of rats: Myocardial hypertrophy: Under the influence of hypoxia, the myocardium may undergo hypertrophy, meaning an increase in the size and volume of the myocardium [75]. This adaptation may aim to improve cardiac function and ensure better oxygen exchange [76]. Changes in myocardial structure: Hypoxia can lead to changes in the structure of the myocardium, in-

cluding an increase in the amount of fibrous tissue or vessel remodeling.

Changes in blood vessels: Hypoxia can cause changes in blood vessels, such as an increase in the number of capillaries to improve myocardial blood supply. **Changes in mitochondria:** Hypoxia can affect the function of mitochondria, which may result in reduced efficiency of energy exchange in myocardial cells [77]. **Changes in the location and size of cardiomyocyte nuclei:** Hypoxia can induce changes in the location and size of cardiomyocyte nuclei [78]. **Changes in functional parameters:** Possible changes in functional parameters of the myocardium, such as an increase in the number of contractions per unit of time or alterations in blood supply [79].

These changes may represent adaptive responses to prolonged hypoxia aimed at ensuring optimal cardiac function under conditions of insufficient oxygen load [80]. To study the myocardial morphology of laboratory rats at 6 months of age under chronic acute hypoxia in detail, it is recommended to conduct microscopic analysis of heart tissues and compare their structural features with normal conditions [81].

Conclusion

Chronic hypoxia affects the structure of the myocardial contractile apparatus in rats during different stages of development (from birth to 6 months) as follows: Newborn rats (1 week after birth): Hypoxia can lead to myocardial hypertrophy and an increase in the number of capillaries to improve blood supply. Young rats (1 month after birth): Under hypoxia, there is myocardial hypertrophy, an increase in the amount of fibrous tissue, and possible vessel remodeling. Young rats (3 months after birth): Hypoxia can result in further enlargement of the myocardium, changes in structure and functional parameters, and the development of new vessels. Adult rats (6 months after birth): At this stage, significant changes are possible, including hypertrophy, vessel remodeling, alterations in functional parameters, and structural peculiarities of the myocardium. Chronic hypoxia affects the morphology of the myocardium in rats at different stages of their development, leading to changes in size, structure, and cardiac functionality.

Perspectives

The scientific novelty of researching the morphology of the myocardial contractile apparatus in rats under the influence of chronic acute hypoxia lies in several key aspects:

1. Understanding the adaptation of the heart to hypoxia: Studying the morphology of the myocardium under hypoxic conditions allows for a better understanding of how the cardiac muscle adapts to inadequate oxygen supply.

2. Identification of stage-specific changes: Researching at various stages of rat development, from newborns to adulthood, can help establish

which morphological changes occur at different stages of hypoxic exposure and whether they happen rapidly enough to cause functional impairments.

3. Opportunities for further research: The data obtained can serve as a foundation for further research focused on studying the molecular and clinical aspects of hypoxia in cardiac muscle.

4. Comprehensive approach: Investigating the morphology of the myocardial contractile apparatus provides a comprehensive view of structural changes in the heart, considering both morphological and functional aspects. All these aspects collectively

highlight the importance of conducting research on myocardial morphology under hypoxic conditions.

Information on conflict of interest

There are no potential or apparent conflicts of interest related to this manuscript at the time of publication and are not anticipated.

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Кобеза П.А., Черкас О.А., Марченко Д.Г., Хріпков І.С. Морфологічні характеристики скоротливого апарату міокарда щурів під дією гіпоксії (літературний огляд і перспективи досліджень).

РЕФЕРАТ. Актуальність. Проведення модельних експериментів на різних вікових групах лабораторних щурів при хронічному навантаженні гіпоксією є актуальним і допомагає вивчати перебудови в ультраструктурі тканин серця з наукової перспективи. Дослідження адаптації серця до гіпоксії допомагає розкрити механізми, які дозволяють серцю функціонувати в умовах низького рівня кисню. Дослідження на різних вікових групах щурів може допомогти визначити, як вік впливає на адаптацію та ультраструктуру серця під час гіпоксії. Загальна актуальність полягає в підвищенні нашого розуміння реакції серця на гіпоксію. **Мета** цього дослідження є розуміння впливу гіпоксії на серця щурів на різних етапах їхнього життя, включаючи новонароджених, молодих та дорослих тварин. Це допомагає розширити наше розуміння адаптаційних механізмів серця та може мати значущі наслідки для нормальної анатомії лабораторних тварин, їхньої біології та всебічного розуміння ультраструктурної перебудови компонентів скоротливого апарату серцевої-м'язової тканини. **Методи.** Систематичний огляд літератури, мета-аналіз, аналіз вмісту, оцінка якості джерел, вилучення і обробка інформації. **Результати.** Хронічна гіпоксія впливає на структуру міофібрильного апарату міокарда щурів на різних етапах розвитку (від народження до 6 місяців) наступним чином: Новонароджені щури (1 тиждень після народження): Гіпоксія може спричинити гіпертрофію міокарда та збільшення кількості капілярів для поліпшення кровопостачання. Молоді щури (1 місяць після народження): Під впливом гіпоксії спостерігається гіпертрофія міокарда, збільшення кількості фіброзних тканин та можлива перебудова судин. Молоді щури (3 місяці після народження): Гіпоксія може призвести до подальшого збільшення міокарда, змін в структурі та функціональних параметрах, а також розвитку нових судин. Дорослі щури (6 місяців після народження): На цьому етапі можливі значні зміни, включаючи гіпертрофію, перебудову судин, зміни в функціональних параметрах та структурні особливості міокарда. **Підсумок.** Хронічна гіпоксія впливає на морфологію міокарда у щурів на різних етапах їх розвитку, що призводить до змін у розмірах, структурі та функціональності серця на всіх рівнях його структурної організації.

Ключові слова: тканинна гіпоксія, кардіоміоцит, скоротливий апарат, ультраструктура, міофібрили.