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Physiological Activity Of Platelets In Vivo During Aging Against The Background Of Physical Exertion.

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ABSTRACT

The aging process inevitably affects all the systems of the body. This is a complex extinction of the body's functions, leading to its death. The aging changes also affect blood parameters in the mammalian organism, including influencing platelet hemostasis, sometimes causing thrombophilic changes. These changes are very significant in the pathogenesis of virtually all cardiovascular diseases, which often occur in a person with age. Due to the fact that membranes of activated platelets provide hemostasis processes, it becomes clear the importance of weakening the disaggregation mechanisms, which can easily be disrupted. In this regard, it is very important to carry out experimental work on the study of their various aspects. Very dosage physical exercises are very accessible and have no side effects. As it is explained, they can often attenuate platelet activity especially in cardiovascular pathology. This helps reduce the risk of developing thrombosis, which severely limit life expectancy. The aim of the work is to elucidate in the model of aging rats the possibility of inhibition of platelet activity in vivo with the help of regular physical exertion. The study included 26 healthy male rats 12 months. Age, experienced during the year, daily physical activity. The control is represented by 91 healthy male colors: 30 animals of 12 months of age, 32 rats of 18 months and 29 animals of 24 months of age. Biochemical, hematological and statistical methods of investigation has been applied. In control rats, as the age increases, there is a gradual increase in the activity of thrombocytes. Regular daily exercise in rats between 12 and 24 months of life provided stabilization of intravascular activity of platelets, inhibiting its age-related enhancement.

Keywords: platelets, intravascular activity, aging, rats, physical activity.

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INTRODUCTION

The process of aging - inevitably affects all the systems of the body [1]. This is a complex extinction of the body's functions, leading to its death [2]. Old age changes also affect blood parameters in the mammalian organism, including influencing platelet hemostasis, sometimes causing thrombophilic changes [3, 4]. These changes are very significant in the pathogenesis of virtually all cardiovascular diseases, which often occur in a person with age [5, 6]. Due to the fact that membranes of activated platelets provide hemostasis processes, it becomes clear the importance of weakening disaggregation mechanisms [7, 8], which can easily be disrupted [9, 10]. In this regard, it is very important to carry out experimental work on the study of their various aspects. Very dosage physical exercises are very accessible and have no side effects. As it was found out, they can often attenuate platelet activity especially in cardiovascular pathology [11, 12]. This helps reduce the risk of developing thrombosis, which severely limit life expectancy [13, 14].

The aim of the work is to elucidate in the model of aging rats the possibility of inhibition of platelet activity in vivo with the help of regular physical exertion.

MATERIALS AND METHODS

The research was conducted in strict accordance with ethical principles established by the European Convent on protection of the vertebrate used for experimental and other scientific purposes (adopted in Strasbourg in March, 18th, 1986, and confirmed in Strasbourg in June, 15th, 2006) and approved by the local Ethics Committee of Russian State Social University (Record №16, dated December, 7th, 2015).

The study included 26 healthy male rats 12 months. Age, which during the year spent daily physical activities on the horizontal treadmill TORNEO of KETLER, moving at a speed of 5m / min. The animals were placed in one of the sections of a wooden frame of a rectangular shape mounted on a treadmill, divided by wooden partitions into 3 parts for the individual placement of the animal. On the first day, the duration of the load was 1 minute, followed by its lengthening by 1 minute per day, bringing it up to 25 minutes per day and following its unchanged duration for a day until the end of the observation [15]. The control consisted of 91 healthy male colors, including 30 animals of 12 months of age, 32 rats of 18 months and 29 animals of 24 months of age.

The activity of lipid peroxidation (LPO) of plasma was evaluated by the amount of Thiobarbituric acid (TBA) -active products in it by the Agat-Med (Russia) and acyl hydroperoxides (AGP) kit taking into account the antioxidant plasma activity (AOA) [16]. The number of platelets in the capillary blood was determined in Goryaev's chamber. Intravascular activity of platelets (IAP) was recorded by phase contrast. The registration of all indicators in the experimental group was conducted three times - at the age of 12 months, 18 months and 24 months. Three age groups of rats (12 months, 18 months and 24 months) who made the control were examined once. The statistical processing of the results is carried out by Student's t-test.

RESULTS AND DISCUSSION

Experimental and control animals 12 months of age before the start of the study there were no differences in all the indicators considered. As the age increased, the control showed an increase in the amount of AGP and TBA products in the plasma, which was accompanied by a decrease in its AOA. For experimental rats, the stability of the LPO plasma and its antioxidant protection proved to be characteristic. So, at the age of 24 months. AHP in them was 1.59 ± 0.019 D233 / 1 ml, TBA-active products were 3.66±0.021 µmol/l and at AOA value 32.2±0.37%. In the control 24 months. Rats, these indices were 1.95±0.033 D_{233}/1 ml, 4.22±0.042 µmol /l and 26.2 ± 0.27%, respectively.

Initially comparable levels of platelet-dislocates in the blood of rats of both groups began to differ with age; in the control they decreased by 12.8% with an increase in the sum of active forms of platelets compared to experimental animals to 29.6±0.17%. Small and large aggregates in the blood of control rats during the observation period increased by 38.6% and 65.8%. At the same time, the number of platelets included in the aggregates in control animals increased by 18.6% in the second year of life.

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Regular physical loads were accompanied in the rats of the experimental group by stably normal IAP (Table). The number of discs in the bloodstream of these animals at the age of 24 months. Was 77.9±0.19% with a low level of the total value of active forms of blood platelets (22.1±0.16%)? This provided an invariably low level of free circulating aggregates of various sizes in the blood of animals with little involvement of platelets in them.

Table: Biochemical and hematological parameters in rats of the second year of life against the background of regular physical activity

Indicators	Experienced group, M±m, n=26			Control group, M±m, n=91		
	12 months, n=26	18 months, n=26	24 months, n=26	12 months, n=30	18 months, n=32	24 months, n=29
Acylhydroperoxides of plasma, D_{233}/l ml	1.53±0.015	1.56±0.014	1.59±0.019	1.52±0.018	1.60±0.024*	1.95±0.033**
Thiobarbituric acid-products of plasma, umol/l	3.59±0.012	3.62±0.016	3.66±0.021	3.61±0.022	3.80±0.016*	4.22±0.042**
Antioxidant activity of plasma, %	32.8±0.33	32.4±0.29	32.2±0.37	32.6±0.24	30.7±0.32*	26.2±0.27**
Platelets-dissociates, %	78.9±0.22	78.2±0.14	77.9±0.19	79.4±0.18	77.2±0.15*	70.4±0.19**
Sum of platelets' active forms, %	21.1±0.18	21.8±0.15	22.1±0.16	20.6±0.14	22.8±0.19*	29.6±0.17**
number of platelets in the aggregates, %	4.9±0.08	4.9±0.07	5.1±0.09	4.8±0.12	4.9±0.05*	5.9±0.09**
Number of little aggregates (in 100 free platelets)	3.6±0.10	3.7±0.09	3.8±0.12	3.5±0.07	3.6±0.09*	5.7±0.10**
Number of medium and large aggregates (in 100 free platelets)	0.14±0.006	0.15±0.005	0.14±0.006	0.13±0.008	0.17±0.004*	0.38±0.003**

Note: there were no significant differences between 12 months of experimental and control rats and age-related dynamics in experimental rats.
Legend: the reliability of age-related dynamics in the control rats relative to 12 months of age:
* - $p < 0.05$; ** - $p < 0.01$.

It is recognized that regular physical exertion can have an optimizing effect on the body in conditions of pathology [17, 18]. Moreover, their effect on age-related changes in IAP remained poorly investigated [19]. To close the gap in scientific knowledge, an assessment was made of the dynamics of IAP in rats during the 2nd year of life - the stage of their ontogenesis, during which the manifestation of signs of aging increases [2]. In the rats of the control group, activation of plasma LPO and IAP was noted. This was based on the activation in them of the exchange of arachidonic acid with increased synthesis of thromboxane [20, 21]. In addition, it is possible that on the platelets of control rats of the second year of life, the number of different receptors increased [22, 23].

In the course of aging in rats, there was an increase in the synthesis of thromboxane A_2 with a depression of the production of its ant platelet agents, prostacyclin and NO. This was the basis for reducing the limiting effect of the vascular wall on IAP. At the same time, plasma thromboplastin, which is excessively formed in aging rats on platelet membranes, accelerates thrombin formation, leads to further growth of aggregates of blood plates and accelerates the formation of fibrin fibers [25], gradually increasing hemostasis.

In aging animals, a gradual increase in the density of platelet receptors to collagen Ia-IIa and VI can be noted, as indicated by an increase in IAP. This is accompanied by an increase in platelet activity of

phospholipase C, stimulation of the synthesis of diacylglycerol and protein kinase C followed by pronounced phosphorylation of proteins of the contractile system [26,27]. Under these conditions, instill triphosphate begins to actively stimulate the entry of Ca^{2+} from the platelet depot, contributing to the increasingly pronounced reduction of act myosin [28]. In addition, excessive adhesion of blood plates in aging animals was also associated with the increase in the factor of von Will brand synthesis in their vessels and the enhancement of its interaction with its receptors (Ic) on the platelet membrane [29]. In addition, aging apparently causes platelet amplification of fibrinogen receptor expression, gradually increasing the activation of phospholipase A_2, which cleaves arachidonic acid from phospholipids [30], to synthesize an increased amount of thromboxane A_2. All these changes are obviously well inhibited by regular physical loads.

At the same time, in the rats, during the second year of life, the survival at the level of the outcome (12 months of life), the activity of LPO in the plasma, ensured the stimulating effect on the surface structures of the platelets. Low IAP in physically loaded rats is a consequence of low LPO and the optimal state of receptor and post receptor mechanisms of platelet function. The presence of a low IAP also indicated that they had an optimal level of aggregation inducers in their blood [26]. The low level of aggregates of platelets in the blood of the observed physically loaded rats also indicated the preservation of the activity of the platelet ant oxidation system, which further inhibited their aggregation abilities with age.

CONCLUSION

For healthy rats the second year of life is characterized by a gradual increase in platelet activity in vivo. The feasible daily exercise in rats in the second year of life was able to maintain intravascular activity of platelets at a normal level, inhibiting its age-related enhancement.

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