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Theory-Physiological and biomechanical features of the interconnected functioning of the systems of accommodation, and aqueous production and outflow. Hypotheses and actuating mechanisms of growth of the eye's optical axis in the metabolic theory of adaptive myopia and in the theory of retinal defocus.

1. Physiological features of the interconnected functioning of the systems of accommodation, and aqueous humor production and outflow

The regulation systems for accommodation, production and outflow of aqueous humor have one common actuating unit – the ciliary muscle (CM), both in animals and humans. At the same time, the control signals from these systems to the CM can be directly opposite [4,6,8]. For example, to open the trabecular pathway of outflow (TPO), it is necessary to reduce the CM. But at the same time, it is necessary to relax the CM in order to see the approaching danger in the distance. So which command will be executed first? In the eye, there is an overriding priority for executing commands from the accommodation system, since the survival of the species as a whole depends on this. The signals of the control system of aqueous production are in the second place by priority, and the signals from the outflow control system are performed last. It is because that the task of maintaining metabolic processes in the eye is more important than the task of removing the spent aqueous humor [4,6,17]. Such physiological representations are key to understanding the features of the interrelated functioning of these three physiological systems of the eye. Most of the animals have only one aqueous outflow pathway – uveoscleral pathway of outflow (USPO), which then passes into the outflow through the sclera. Only in humans and in four species of highly evolved monkeys, during the course of evolution, an additional aqueous outflow pathway was formed through the trabeculae (TPO, trabeculae outflow pathway). This happened because of changes in the habitat, which required to develop the ability of a long visual work at a short distance, and at that moment the USPO is blocked (in the ciliary muscle, the interfiber spaces with the matrix are compressed at that moment). In Table 1 it is shown that TPO is open only when looking near, and USPO is closed at that time. In visual work at medium and long distances, on the contrary, only USPO is open, and TPO is closed. It should be noted that USPO is the main way by which the necessary ingredients are delivered to maintain normal metabolism and reproduction of collagen in the middle and back parts of the sclera. Also, along this basic pathway, prostaglandins are delivered to the sclera, which are normally produced by the intraocular epithelium. The sclera can regulate its permeability with the help of a large number of prostaglandin receptors located in it [1]. That is why the pharmacotherapy of glaucoma with prostaglandins is so effective. The eye does not control the level of IOP directly, since morphologists have not yet detected baroreceptors in it. The level of IOP in the eye is directly determined primarily by the level of rigidity of the sclera [2-4]. A large number of mechanoreceptors have been found in the sclera [1], which allow to control the reciprocal displacement of scleral plates during micro fluctuations of the eye volume. Conclusion: the eye does not control the level of IOP, but constantly monitors its volume with the help of mechanoreceptors, as well as the receptors of prostaglandins [4].

Table 1 [2,4,6].

Functioning of the outflow pathways of aqueous humor depending on the phase of accommodation			
Name of outflow pathways of intraocular fluid	The phases of accommodation and the ciliary muscle tonus		
	The «completely near» phase of accommodation. Ciliary muscle tonus is maximal.	The «partially near» and «partially afar» phases of accommodation. Ciliary muscle tonus is average.	The «completely far» phase of accommodation. Ciliary muscle tonus is minimal.
Trabecular outflow pathway through trabeculae (TPO)	Opened	Closed	
Uveoscleral outflow pathway through the matrix of the ciliary muscle and then through the sclera (USPO)	Closed	Maximally opened	Partially opened
In fact, the ciliary muscle regulates the intensity of the outflow pathways of intraocular fluid			

The outflow (slow filtration) of the aqueous humor occurs through the three main eye filters: juxtacanicular tissue, inter-fiber ciliary matrix and scleral matrix. The outflow efficiency is determined by the main functional characteristic of the sclera – its fluctuability (this is a new concept in ophthalmology). Fluctuation is the functional ability of the sclera to "push out" the waste intraocular fluid from the eye with the help of elastic fibers and fibroblasts located in the sclera. Concurrently the volume of the eye decreases. We have learned to reliably measure *in vivo* the level of fluctuation and rigidity of sclera, as well as the level of IOP in youth and even in elderly patients, using an ORA air analyzer by our own method [2-4].

2. The key links of adaptive myopia (AM)

The development of the most pandemic myopia in the history of mankind shows that until recently there was no workable theory of acquired myopia in the world. The forecast of an increase in the number of myopes to 5 billion people by 2050 (half of the world's population, figure 1) [5] shows that if acquired myopia is a disease, it is transmitted "visually". But the clinical facts show another pattern. AM develops rapidly both in animals and in a healthy person under the age of 45 years. And this apparently is a normal adaptive reaction, which provides the possibility of prolonged strenuous visual work at a short distance. Adaptive myopia is a clear example of the implementation of the energy saving law in anatomical development of biological systems. The length of the optical axis of the eye should be such as to ensure the necessary long-term near visual work with minimal tonus of the ciliary muscle [4,6]. Since children of all animals and humans at an early age are weak hyperops, even the transition to emmetropia or initial myopia can significantly reduce the energy consumption of the eye if necessary to perform long-term near work. Highly developed monkeys, such as gorillas, orangutans and others, are even forced to post a sentry along the perimeter of the troop, because their way of life implies gathering food and looking at it closely.

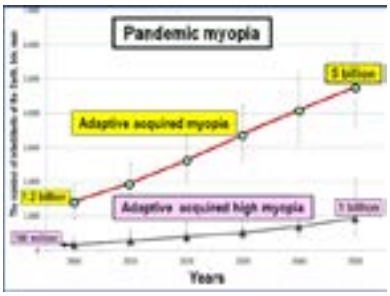

Fig. 1. Prognosis of the adaptive myopia development by 2050 [5].

This led them to adaptive myopia: they lost the ability to see well into the distance. On the contrary, you will not find AM in wolves or, for example, reindeer herders over the age of 50, because near visual work is almost not necessary for them. But now young reindeer herders since the age of 15 are almost all myops, as they actively and constantly use modern gadgets. These observations

lead to important conclusions. Both animals and humans clearly have a normal adaptive mechanism for adjusting the optic axis of the eye to the required visual load. So the implementation of the energy saving law is directly ensured. Therefore, the possibility of adaptive lengthening of the eye`s anteroposterior axis (APA) is directly related to the accommodation system, the commands of which the ciliary muscle performs first and foremost, necessarily and always. In addition, this adaptation mechanism should be common in animals and in humans, and it must be present at the physiological (fast) level, but not at the genetic (slow) level. The actuating mechanisms of lengthening of the eye`s APA, according to our metabolic theory of myopia, are as follows. During active near visual work USPO is blocked. This leads to a temporary functional insufficiency of the collagen production level in the posterior part of the sclera. The strength of the scleral collagen skeleton temporarily decreases, and the intraocular pressure easily "blows" the posterior pole of the sclera. As the result APA increases.

3. The actuating mechanisms of adaptive myopia (in our metabolic theory of AM)

Classical morphological studies of sclera in acquired myopia show that only in the posterior pole of the sclera collagen fibers are located equatorially [4,6,7] (see our figure 2). The equatorial, not meridional, arrangement of collagen fibers in the posterior pole of the sclera (in the form of a spring) facilitates the possibility of its adaptive extension. But now, for example, the average ciliary muscle tonus is required to perform the same visual work, not the maximal tonus. Therefore, the level of USPO and the reproduction of collagen come back to normal. If concurrently the visual load is comfortable, then the conditions for further progression of myopia are absent. If not, the adaptation will continue. This actuating mechanism of adaptive elongation of eye's APA is based on the creation of temporary functional insufficiency of the USPO.

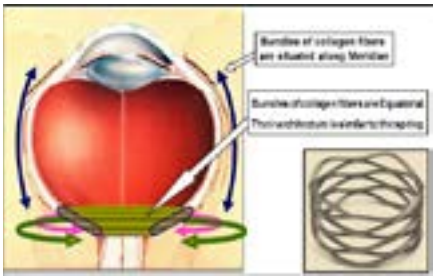

Fig. 2. The posterior pole of the sclera [4,6].

The arrangement of collagen fibers is equatorial. This facilitates the adaptive elongation of the optical axis of the eye.

The mechanism is simple and reliable, and it is the same in animals, as in humans. Therefore, adaptive myopia should be considered as a classic case of the predominance of accommodation over the aqueous humor outflow. Our research has shown that the extreme phases of accommodation (completely near sight and completely far sight) are unfavorable for USPO. In completely near-sight accommodation USPO is blocked, and in completely far-sight accommodation USPO is minimal. In the first case AM develops according to the loading type, and in the second case it develops according to the unloading type (Fig. 3).

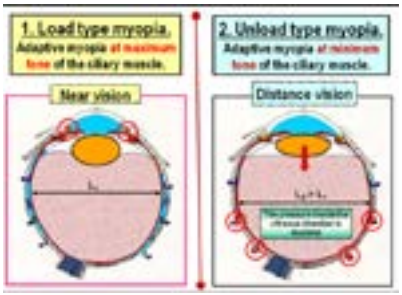

Fig. 3. Load (1) and unload (2) type adaptive myopia [6]. 1 - Functional overlap USPO during intense near work temporarily worsens nourishment of the back part of sclera.

2 - Blood consumption of ciliary muscle and production of intraocular fluid are reduced thrice. Compression of interfiber spaces of CM with filtration matrix worsens the level of USPO and nourishment of the sclera. Compression of the suprachoroidal gap complicates the delivery of aqueous humor to the sclera below the equator.

4. Key linchpins of early refractogenesis

Fig. 4 shows the generalized age curves of the mutually related growth of human organs [9]. It is clearly seen that by the age of 14 the "genetic" growth of the human eye stops (red dot). Fig. 4 shows, that the genetically "slowed" youthful growth of the eye at the age of 10-14 years (unlike the early childhood from 2 to 5 years) can not ensure quick adjustment of the eye`s APA directly, without attracting any other actuators – physiological, for example. Indeed, adaptation requires that the axial growth of the eye is accelerated many times compared to the relatively slow growth of the body and brain in early adolescence.

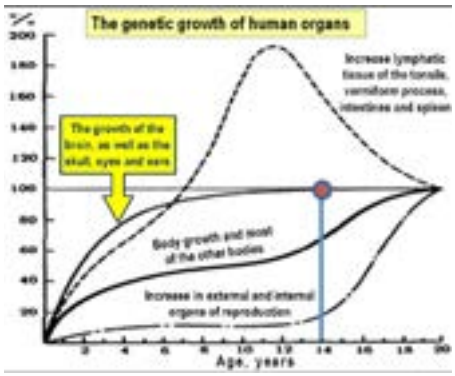

Fig. 4. Curves of growth of separate human organs [9].

So we know that every child has a genetic program for the gradual growth of the eye in all directions, according to the corresponding parallel growth of the skull and orbit. And this program will be executed, even if we`ll cut the optic nerve. And normally the eyes will certainly reach the state of weak hyperopia. In a norm, early refract genesis in childhood ends with this condition: the eyes grow synchronously with the body and internal organs. However, in those children who have a hereditary predisposition, the genetic weakness of the collagen formation process in the fibrous membrane of the eye can lead to expanding beyond the hyperopic "end-point" even at the stage of early refract genesis. And during the early "genetic" refract genesis the optical axis of such eye will grow longer due to poor heredity. In this case, the rapidly growing children's eye will mainly expand in length, as there are no other directions to grow in the orbit. That is, already in the period of early refract genesis this child will starts not from the level of weak hyperopia, but from the level of weak myopia, for example. Probably, this is the congenital myopia, which may not progress under low visual loads.

5. Key linchpins of late refractogenesis

This period refers to the age of 14–23 years and has been relatively little studied. There are few clinical data that confirm that a significantly faster growth of eye`s APA is possible during this period, compared with the period of early refractogenesis. And this requires the possibility of additional rapid adaptation of the visual mechanism. And this is a key issue in the philosophy of the adaptive myopia development! If the mechanism of accelerated ocular "axial" genetics does not work in adolescence and adulthood, then what executive mechanisms lead to such a massive and rapid emergence and development of progressive myopia (PM)? In other words, the adaptive PM rapidly develops through non-genetic, but physiological actuators. And it is possible even in the period of early adolescence with normal genetic growth of the eye in the background. The emergence and adaptive progression of emmetropy and initial myopia presumably are linked to the manifestation of a regular physiological mechanism, which is the same for humans and animals. The length of the eye, corresponding to visual loads, is formed in such a way to ensure the lowest possible level of energy consumption during intense and prolonged near visual work. The adaptive anatomical elongation of APA in initially hyperopic eye is a result of strenuous near work; and perhaps this is a manifestation of the energy

saving law in the development of biological systems at any age. But if that's true, then it must be concluded that the acquired myopia is not a disease, but a normal adaptation to the visual habitat. From this perspective, only any degree of myopia with complications should be considered as a disease. So in the prevention and control of weak and moderate degrees of PM the "main attack" should focus on this task: to switch off the physiological functional ability of the human eye – the adaptive elongation of optical axis. Our clinical studies (with co-authors) in 2772 patients have convincingly demonstrated the real effectiveness of our metabolic theory of adaptive myopia. We used all modern means of optical correction, and observation periods was 3, 5 and 7 years [11,12]. The new theory allows to explain all available clinical facts, and also it allows to justify the choice of the most effective and adequate means of individual optical correction in practice. Our theory of rational optical correction (our special generalized term) allows not only to take into account the individual visual acuity of the patient, but also to apply optical correction in cases of glaucoma and other pathologies. The effective way to control adaptive myopia is linked to the field of eye physiology. It is necessary to exclude the work of the ciliary muscle in the extreme phases of accommodation with the help of rational preventive optical correction. I. e. it is necessary to provide an average tonus of the ciliary muscle to maintain USPO. This problem is most successfully solved with the help of orthokeratology, when the eye has a weak hyperopia during the working day. The two main objectives of rational optical correction are: 1) using of the entire functional working range of the ciliary muscle for a given visual load; 2) selection of such CM tonus, which does not worsen the USPO. In cases of irrationally chosen optical correction and with intense long-term visual work, the process of natural adaptation of APA length can continue even after successful keratorefractive surgery. Therefore, such patients will need preventative glasses for intense long-term work with displays.

6. Key points of the incremental retinal-defocus theory

Now that we have an idea of the possible physiological mechanisms of adaptive myopia development, some clearly incorrect positions in the incremental retinal-defocus theory (IRDT) will also become more clear [10].

According to the IRDT, the retina is the "brain center" of the eye growth even if the optic nerve is cut. This is a strong, but dangerous hypothesis. It turns out that the eyes of animals can grow rapidly even without connection with the brain, if the focus shifts in a certain direction. Thus, the processes of early and late refractogenesis in IRDT are "unified and inseparable": the genetic mechanisms of morphosis in childhood are simply extrapolated to adolescence and adulthood. It is assumed that the speed of the work of genetic mechanisms can increase several-fold. And then we can understand, why the accommodation management system should not participate at all in the adaptive lengthening of the eye axis (according to the IRDT). Let's quote from [16]: "...since the accommodation mechanism can not compensate for a large defocus area of the retinal image, induced by convex or concave lenses of considerable optical power (used in animal experiments), the accommodation system in these experiments, in fact, does not have any influence on the observed effect." But these submissions already drive the IRDT back in the 19th century. This vulnerability of the retinal-defocus theory has already been noted by a number of researchers. It should be noted that humankind has accumulated so much knowledge about connection between accommodation and current length of the eye, that under this spate of facts IRDT will not survive in the historical perspective. However, the authors of the IRDT made a separate "curtsey" to indirect influence of accommodation on APA growth. To explain the results of various clinical studies about effect of correction degree on myopia progression in children, the authors of the IRDT even invoked the method of accommodative response, which allows to evaluate the work of the accommodative system at a different level of the visual stimulus.

7. Criticism of the IRDT

Let's sum up. "In the opinion of the authors of the IRDT, their theory provides a simple and physiologically real mechanism explaining how a lenses of great positive force, complete correction or incomplete correction in progressive myopia can contribute to a change in the axial length of the eye according to the hyperopic, emmetropic or myopic type, respectively" (quoted after [16]). In fact, at the present time this is only a hypothesis without clinical confirmation of the proposed actuating mechanisms. But let's try to preserve objectivity. To the undoubted advantages of the IRDT we can include the fact that finally in the West the direct connection been understood between lengthening of eye`s axis and possible insufficiency of collagen formation in scleral structures. This connection previously was revealed by Russian scientists [4,6-8,12]. Let's quote from [16]: "The authors also believe that the flow of chemicals [from the producer, i. e. retina – our insert] can cross the choroid to get in the sclera. In the sclera, these

substances seem to be able to control the rate of synthesis of proteoglycans and, consequently, the rate of growth of the sclera."

7.1. Advantages of IRDT:

7.1.1. This is another historical attempt to explain the refractogenesis of progressive myopia by disorganization in the work of mechanisms of collagen formation in the sclera. Truly speaking, this was already said in 1974, in the classical doctoral dissertation by T.E. Nikolayeva [7]. And the direct connection between accommodation and aqueous outflow was noted by us back in 1997 [17].

7.1.2. G.K. Hung and K.J.A. Ciuffreda tried to explain the mechanism of eye focusing through the work of neuromodulators, which are sensitive to changes in the contrast of the retinal image [13-15]. This attempt should certainly be welcomed. This is a promising path and an important attempt, although not the first one. After all, until recently we did not fully understand, how the mechanism of feedback from the brain to the ciliary muscle is organized. This mechanism changes ciliary muscle tonus and allows us to get the eye in sharp focus. It is clear to us and other researchers [4,6,17], that it is not related to the "blurring of the image", but to something else – perhaps, to the comparative sensitivity of exciting fields on the retina. And, most likely, the contrast of the incoming optical signal can be one of the additional factors that ensure greater efficiency of such a mechanism.

7.1.3. Also worthy of respect is the attempt of G.K. Hung and K.J.A. Ciuffreda to find direct actuating mechanisms of the influence of visual load on the processes of collagen formation in the posterior part of the sclera. Truly speaking, this attempt was clearly not successful, but its great importance lies in the fact that it forms the public opinion of the professional community of ophthalmologists and other eye researchers. Certainly in different clinics there will be a lot of parallel research in this scientific direction at the most modern level, and it will accelerate the understanding of the essence of adaptive myopia.

7.2. Disadvantages of IRDT:

7.2.1. The assumption that the brain does not participate in the process of late refractogenesis contradicts a huge number of clinical facts and in the long term will do more harm than good. A significant part of researchers in the world will be distracted by testing this physiologically incorrect hypothesis.

7.2.2. The hypothesis that there is a growth center of the eye in the retina, separated from the brain, is the most incorrect one. Here we immerse ourselves in the field of mythology, remaining in the two-dimensional (retina), and not in the three-dimensional world of a higher control system called the brain, with the periphery in the form of retina.

7.2.3. The very idea that the retina develops, and then delivers along its own ways to the sclera some chemical substances, that ensure the regulation of the rate of refractogenesis at a genetic level, is certainly taken from the science fiction. For today the biochemical processes taking place in the retina are described in sufficient detail, although not completely. But nowhere will we find the message that the retina is a biochemical "machine" that directly controls the growth of the sclera.

7.2.4. A serious disadvantage of IRDT is the low response rate to the change in the surrounding visual environment (years). Also they have no clear idea about the functioning of one of the supposed links in the general genetic mechanism of eye growth, primarily along optical axis. We do not even say that the authors of the IRDT lack any consideration of the energy saving law and ergonomics in the development of biological systems.

7.2.5. The authors of the IRDT propose challenging hypotheses, but in general we should note their obvious one-sidedness and limitation.

Again, we will not blame the authors. Like many other ophthalmologists, they are still trapped in myths about secondary role of the uveoscleral outflow pathway – the only one in eyes of a huge number of animal species. The authors do not even suggest that the sclera is an important filter for the drainage of spent aqueous humor out from the eye cavity. For this, the sclera has prostaglandin receptors that regulate its permeability, and mechanoreceptors that regulate the volume of the eye. It should also be understood that aqueous humor is the main carrier of metabolites necessary to maintain normal collagen formation in the sclera, as well as for maintenance of normal metabolism in the retinal structures.

8. Conclusions

The equatorial arrangement of collagen fibers in the posterior pole of the sclera provides a process of normal adaptive elongation of

the optic axis of the eye. The length of the optical axis adapts to the intense visual load in accordance with the energy saving law – one of the laws governing the development of biological systems. The actuating regulatory mechanism of rapid physiological (not genetic!) adaptation of the anteroposterior axis length to the visual load is the organization of temporary functional insufficiency of the level of USPO. This is a common mechanism in animals and humans. Therefore, adaptive emmetropia and myopia are not a disease, but a normal adaptation of the visual system to the environment. To create a comfortable visual environment, it is necessary to work out the rules and regulations of visual work in each country. The regulations should contain specific requirements for the video safety of all modern displays and artificial light sources. The absence of such rules does not allow highly effectively deal with adaptation myopia even using sound optical correction. However, today a total preventive correction when working with any displays can significantly slow down the natural process of development of adaptive myopia. Thus, the main task of preventive or rational optical correction - delete work ciliary muscle at maximum or minimum tone.

References:

1. Vit V.V. Structure of the human visual system.- Odessa, Astroprint.- 2003.- 655 P. [in Russian]
2. Svetlova O., Koshits I., Guseva M., Makarov F. Two key managers in the pathogenesis of OAG and innovative methods of early diagnosis.- 7-th World Glaucoma Congress: Abstract book, 2017.- Helsinki.- P. 532.
3. Gorodnjanskaja E., Makarovskaia O., Pankratov R., Koshits I., Svetlova O., Makarov F. Low confidence of the IOP criterion and clinical efficacy of OAG diagnostic by sclera rigidity and fluctuations levels and by IOP value in the youth.- 7-th World Glaucoma Congress: Abstract book, 2017.- Helsinki.- P. 505.
4. Svetlova Olga. Functional peculiarities of interaction of sclera, accommodative and eye drainage systems in glaucoma and myopic pathology. Thesis of doctor of medical sciences.- Moscow,2010.- 55 p. [in Russian]
5. Brien A. Holden at al., 2016, American Academy of Ophthalmology; www.aaojournal.org. Brien A. Holden at al., 2016, American Academy of Ophthalmology; www.aaojournal.org.
6. Koshits I.N., Svetlova O.V. A mechanism of formation of the eye adequate length in the norm and metabolic theory of pathogenesis of acquired myopia.- Ophthalmic journal, 2011.- 5 : 4-23.
7. Nikolaeva T.E. Histological, histochemical and electron microscopic study of sclera for myopia: Thesis of medical sciences.- Moscow, 1974.- 216 p. [in Russian]
8. Svetlova O.V., Koshits I.N. Biomechanical aspects of possible future areas of causes of hereditary and acquired myopia.- «Myopia, violation accommodation, refraction and the oculomotor system»: proceedings of the International Symposium.- Moscow: Helmholtz Institute of eye illnesses, 2001: 77–78. [in Russian]
9. http://botan0.ru/?cat=2&id=126.
10. Koshits I.N., Svetlova O.V., Guseva M.G., Balashevich L.I., Makarov F.N., Egemberdiev M.B. Adaptive myopia. Part 1. Actuating mechanisms of optic axis growth in the incremental retinal defocus theory.- Ophthalmic journal,2016.- 6: 45-54.
11. Guseva M.G., Svetlova O.V., Koshits I.N. Stabilization of the acquired myopia in children with the aid of contact lenses from the standpoint of the metabolic theory of myopia.- Ophthalmic journal, 2011.- 5: 29-36.
12. Koshitc Ivan, Svetlova Olga, Guseva Marina. Physiological principles of rational optical correction. Practical recommendations. Normal physiology of the eye. Tutorial.- St. Petersburg: Northwestern State Medical University by name I.I. Mechnikov, 2016. - 152 p. [in Russian].
13. Hung G.K., Ciuffreda K.J. An incremental retinal defocus theory of the development of myopia. Comments on Theoretical Biology,2003.- V. 8: 511-538.

14. Hung G.K., Ciuffreda K.J. Differential retinal-defocus magnitude during eye growth provides the appropriate direction signal.- Med Sci Monit, 2000.- 6(4): 791-795.

15. Hung G.K., Ciuffreda K.J. Incremental retinal defocus theory predicts experimental effect of under-correction on myopic progression. JBO 2004.-3: 59-63.

16. Lagase J.P. Theory of retinal defocus changes and the progression of myopia.- Optometry bulletin, 2011, Moscow.- 1: 48-57. [in Russian]

17. Volkov V.V., Kotliar K.E., Koshits I.N., Svetlova O.V., Smolnikov B.A. Biomechanical features of interaction adjustment of regulatory systems and drainage of the eye and when contusion lens.- Vestn. ophthtalmol.,1997.- 3: 5-7. [in Russian]

Biography

Ivan N. Koshits, mechanical engineer, a member of the St. Petersburg Sechenov Society of physiologists, biochemists, pharmacologists. Author of 59 scientific works in biomechanics, normal and pathological physiology of the eye and its elements, 2 monographs (2016). Organizer and co-head of the first scientific conferences in Russia on the Biomechanics of the eye in 1998-2011. Co-author and developer of the theory of functions of fibrous sheath eyes, the theory of open angle glaucoma and metabolic adaptation theory of acquired myopia. As well as the co-author of dynamic diagnostic methods to determine the *in vivo* new physiological and biomechanical characteristics of the eye. General Director of Petercom-Network / Management Systems Consulting Grope Cl. Corp.

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