

**INFLUENCE OF ANTIHYPXANTS AND SEROTONIN ON THE VIABILITY AND BASIC METABOLISM OF ANIMALS IN NORMAL CONDITIONS, IN HYPOXIA AND UNDER THE EFFECT OF SNAKE VENOM**

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**ABSTRACT**

Influence of antihypoxants and serotonin on the basal metabolism

Influence of antihypoxants on the basal metabolism of animals

Antihypoxants – benzonal, katatsin, cavergal and gutimin are interesting because they increase the body's resistance to extreme effects

[Aleksandrova A.E., Govorova L.V., 1977; Valieva L.B., Litvinov V.S., Rodin A.P., 1980; Nazrullaev S.S., 1994; Zakharovsky A.S., Smelyanskaya G.N., Saksanov P.P. et al., 1996; Yuldashev N.M., Khakimov Z.Z., Ziyaeva Z.Z., 1996; Ziyaeva A.V., 1997; Asanova K.A., 2002; Abdullaev S.F., 2005] and chronic emotional and pain stress [Karimova G.M., Abdullaev G.R., Khalikov Zh.R., Almatov K.T., 2010; Almatov K.T., Karimova G.M., Abdullaev G.R., Khalikov Zh.R., 2011; Karimova G.M., Abdullaev G.R., Almatov K.T., 2011; Abdullaev G.R. Karimova G.M., Mirzakulov S.O., Almatov K.T., 2012; [Karimova G.M., Abdullaev G.R., Almatov K.T., 2014; Abdullaev G.R., Karimova G.M., Almatov K.T., 2015; Karim T. Almatov, Gafurdzon R. Abdullaev., 2016].

Analysis of the impact of antihypoxants on the body's oxygen consumption rate and respiratory rate revealed that these medications inhibit (Table 3.1). The rats' respiratory rate and oxygen absorption gradually dropped with increasing antihypoxant dosage. Thus, if the rate of oxygen consumption in the animals drops by only 16 percent after 20 mg kg of katacin is introduced, then the rate of oxygen consumption drops by 26 point 2, 38 point 1, and 48-point 5 percent, respectively, after 30 mg kg, 40 mg kg, and 50 mg kg. When compared to oxygen consumption, antihypoxants reduce rats' respiratory rates more slowly. The animals' respiratory rates drop by -12, 23, 3, 28, and 34.6 percent, respectively, from the control level after katatsin is introduced into their bodies at doses of 20, 30, 40, and 50 mg kg. After benzonal, cavergal, and gutimin are introduced into animals, a similar suppression of gas-oxygen exchange and respiratory rate is also seen.

**Table 3.1 The effect of antihypoxants on gas-oxygen exchange and respiratory rate (M±m; n=10-14)**

Antihypoxants, mg/kg body weight	O <sub>2</sub> consumption rate, ml O <sub>2</sub> /kg h		Respiratory rate		SPO <sub>2</sub> /CHD
Katatsin		%		%	
0	4690±410	100	159,0±13,6	100	1,0
20	4014±346	85,6	138,8±12,7*	87,3	0,98
30	3463±302**	73,8	121,9±11,9**	76,7	0,96
40	2902±286***	61,9	114,5±10,8****	72,0	0,86
50	2414±205****	51,5	104,0±11,2****	65,4	0,79
Benzonal					

0	4782±286	100	160,2±14,4	100	1,0
20	3993±370	83,5	136,5±13,8*	85,2	0,98
30	3653±314**	76,4	127,7±12,4**	79,7	0,96
40	3146±280***	65,8	115,2±12,0****	71,9	0,91
50	2821±244****	60,0	110,2±11,6****	68,8	0,87
Kavergal					
0	4698±370	100	155,9±13,8	100	1,0
20	4355±378	92,7	145,7±13,5*	93,5	0,99
30	4090±355	87,0	139,4±14,2**	89,4	0,97
40	3805±384**	81,0	135,5±14,0****	86,9	0,93
50	3654±344***	77,8	132,3±13,3****	84,9	0,91
Gutimin		%			
0	4795±386	100	162,0±15,5	100	1,0
40	3951±355	82,4	135,7±15,6*	83,8	0,98
60	3529±312**	73,6	125,5±12,7**	77,5	0,95
80	3006±284***	62,7	115,0±12,0****	71,0	0,88
100	2628±224****	54,8	106,7±12,6****	65,9	0,83

It should be noted that the animals received intraperitoneal antihypoxants. The basal metabolic rate and respiratory rate were measured an hour after antihypoxants were administered. The respiratory rate (RR) is expressed as a percentage, and the rate of oxygen consumption (BMR2) is expressed as ml g kg of body weight. In this and other tables, the asterisks \* $p < 0.05$ , \*\* $p < 0.02$ , \*\*\* $p < 0.01$ , and \*\*\*\* $p < 0.001$  denote the reliability coefficient. This was accomplished by using gutimin at a concentration twice as high as other antihypoxants. Both of these outcomes align with the S.S data. Nazrullaev [Nazrullaev SdotS. in 1994]. He demonstrated that gutimin was 1 point 65 times less effective than catacin and benzonal. Thus, a reduction in oxygen consumption rate surely contributes to the way hypometabolic agents work.

American scientists testified before the royal commission. They included R.H. MacArthur and E.O. Wilson [1967]. The scholars introduced coefficients  $r$  and  $k$  into his population dynamics analysis; in the dissertation, these were not treated mathematically but used as recommended by the authors to identify two strategies that organisms employ in their evolutionary process: K-type with a low reproductive rate and long-life span, and R-type with a high reproductive rate and short life span.

In addition to promoting the emergence of new traits and the capture of new areas, the  $r$  strategy can be helpful for populations at turning points and their evolution with a changing environment. Under comparatively stable environmental conditions and in an area that has already been captured, the K strategy is indicative of a population's well-being. A higher rate of reproduction and a greater generational change—that is, a shorter life expectancy for individuals—will undoubtedly increase the likelihood of creating something new. Including in the strategy an increase in both the rate of reproduction and life activity in general (let's call it the power of the organism) over a brief period of time allotted by nature for an individual's life would be desirable in order to solve the problem of transitional forms as formulated above.

Generally speaking, this assumption makes sense: increasing activity and fertility come at a cost, and that cost is money.

The respiratory rate and basal metabolism are thus decreased by antihypoxants. Notably, antihypoxants lower the respiratory rate after first lowering the basal metabolism. Here, the reduction in basal metabolism caused by cavergal is more pronounced than that caused by katazin and benzonal. There is a greater disparity between respiratory rate and basal metabolism as antihypoxant concentration rises. When the rate of oxygen consumption declines, the body's energy expenditures also decrease.

### **The influence of serotonin on the basal metabolism of animals**

Because it suppresses mitochondrial respiration, affects numerous bodily functions, and builds up in hibernating animals, serotonin is an intriguing neurotransmitter [Aripova A. A. . Bogdanov, E. G. . Zaitsev, A. A. . Ignatov Yu. A. , 1996] and makes the body more resilient to strong stimuli [Medvedev A. A. 1990; N. Brustovetsky. No. M.V Egorova. and others. 1991; G. Bronnikov. A. Vinogradova, M. D. Mezentseva, V. S. . (1991); Voronova I. P. . K. Svechnokova. V. . Popova, N. K. . 1991; Lebkova N. P. . Shortanova, T. K. Samoilik, N. Me. Zetlenok, L. No. 1991; D.A Ignatiev. Vorobiev, V. V. . Yarkov, A. V. . V. Sviryaeve. Me. R.J Berger, 1992. Phillips, N. H. . 1995; S.M Gibarova., 1997].

The question of how an endogenous hypometabolic substance like serotonin, which typically builds up in the body and tissues of hibernating animals, affects oxygen metabolism was of interest to us [Voronova I. P. K. Svechnokova, V. Popova, N. K. 1991]. The primary indicator of an organism's hibernation or torpidity is the inhibition of metabolic functions, or the shift to a hypobiotic state, which guarantees a rise in the body's resistance and the conservation of metabolic resources.

The primary physiological function of serotonin is linked to neuronal activity, particularly in the hypothalamus. In the pathways linked to sleep and sensory perception, serotonin plays a role [Savina MdotV]. Sapronova, A. (1992). Well, yes. Proshlyakova, MdotV. S.V. Panaev. And so on. (1996); Whitter J. Ms. R.T Mason. Hemeric A. (1996). Belpaire F., and De Vriendt S. D. in 2000]. Serotonin levels rise fifty times while you're hibernating [Brustovetsky N. N. Egorova, M. V. And so on. (Bostovetsky N. 1991). N. Egorova, M. V. And so on. in 1993]. Apart from its role in digestion, the gastrointestinal tract also serves as an organ involved in hormone regulation. Most of the serotonin produced by the stomach and intestines enterochromatid cells is about 90% [Brustovetsky N. N., Egorova, M. V. and so on. R.J Berger, 1993. N. Phillips. A. 1995; Aripova, A. E. Bogdanov, G. A. Zaitsev., U. D. Ignatov in 1996].

The remarkable function of serotonin in the hypothalamic regulation of body temperature has been demonstrated, involving two opposing effects: serotonin on the one hand, and norepinephrine and adrenaline on the other [Kubarko A. M. Pereverzev, V. A. Balakleyevsky, A. Me. Gomolko, N. M. 1991]. Considerable evidence has been gathered that serotonin has a hypothermic effect [Berger R. J. Phillips, N. H. 1995; S. M. Gibarova, 1997].

The way that serotonin causes hypothermia is not well understood. Serotonin's central effect on hypothalamic thermoregulation is supported by data, but the hypothalamic temperature curve remains unchanged when serotonin is injected into the brain's third ventricle, anterior hypothalamus, or preoptic zone. Thus, serotonin suppresses peripheral thermogenesis

mechanisms [Berger R. J. Phillips, 1995; A. N. Aripova, A. E. Bogdanov, G. A. Zaitsev, U. D. Ignatov 1996].

According to our research findings (Table 3.2), this biogenic amine reduces the force of breathing. To more precisely ascertain the hypometabolic efficacy of serotonin, we employed higher dosages than those found in the literature [I. P. Voronova, K. V. Svechnokova, V. N. Popova, 1991]. The amount of serotonin given to the rats and mice caused a progressive decrease in oxygen absorption.

**Table 3.2** The effect of serotonin on gas-oxygen metabolism, respiratory rate and general condition of animals ( $M \pm m$ ;  $n = 7-10$ ).

Animals	Mg/kg massy tela 16 / 5 000 Mg/kg body weight	O <sub>2</sub> consumption rate, ml O <sub>2</sub> /kg h	%	Respiratory rate	%	SPO <sub>2</sub> CHD
Mouse	0	4335,5±274,5	100	835,0±53,4	100	1,00
	20	2896,4±164,4****	66,8	656,4±58,8***	78,6	0,85
	40	2610,0±151,6****	60,2	620,4±51,6****	74,3	0,81
	60	2271,8±140,8****	52,4	581,1±43,2****	69,6	0,75
	80	1978,8±131,2****	45,6	546,7±36,8****	65,5	0,70
Rat	0	2451,2±126,5	100	158,2±16,8	100	1,00
	20	1641,4±100,2****	66,9	125,4±14,6*	79,2	0,84
	40	1495,2±96,6****	61,0	119,3±13,8**	75,4	0,81
	60	1272,2±80,4****	51,9	110,7±13,0****	70,0	0,74
	80	1124,4±61,7****	45,9	101,4±12,6****	64,1	0,71

Note: Serotonin was administered intraperitoneally to the animals. One hour after the administration of serotonin, the basal metabolic rate and respiratory rate were determined.

After administering 20, 40, 60, and 80 mg/kg body weight to rats, the oxygen consumption rates decreased by 33.2%, 39.8%, 47.6%, and 54.4% compared to the control group, while in mice, the reductions were 33.1%, 39.0%, 48.1%, and 54.1%. The decrease in the respiratory rate of the animals following serotonin injection was less pronounced than the reduction in the intensity of respiration. Specifically, after administering serotonin at doses of 20, 40, 60, and 80 mg/kg to rats, the respiratory rates dropped by 21.4%, 25.7%, 30.4%, and 34.5%, respectively, compared to the control, while in mice, the decreases were 20.8%, 24.6%, 30.0%, and 35.9%. This indicates that with serotonin present, the respiratory rate declines 15%, 19%, 25%, and 30% more slowly in mice, and 16%, 19%, 26%, and 29% more slowly in rats compared to the basal metabolic rate. As the concentration of serotonin increases, the gap between the basal metabolism and the respiratory rate widens. Additionally, the animals exhibit reduced mobility but do not experience narcotic suppression of the basal metabolism, which decreases by 47-58%.

As a result, serotonin lowers respiratory rate and basal metabolism. It should be mentioned that serotonin lowers respiratory rate after first lowering baseline metabolism. The discrepancy between the respiratory rate and basal metabolism grows as the serotonin concentration does.

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