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CHANGES OF THE GLUCIDIC METABOLISM DETERMINED BY THE PHYSICAL EFFORT OF THE TREATMENT WITH THE ASLAVITAL AND APILARNIL

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ABSTRACT. We studied changes of glycaemia, liver and muscle glycogen during physical effort after the treatment with Aslavital and Apilarnil. The experiments have monitored the effects of Aslavital and Apilarnil treatment on glucidic metabolism used in physical effort. The experiments were done on Wistar white rats aged 6-10 months, weighing 140-200 g. The animals had to pass a tiredness test consisting of free swimming: 90 minutes and 5 hours acute, five hours swimming in 7 days and five hours swimming in 14 days. The swimming took place in the absence or in the presence of Aslavital (1 ml in a 10 mg/kg of body) and Apilamil (3x1 p.o.). We investigated glycaemia following the God Perid method *(Werner et al,* 1970) and the glycogen following the Montgomery method. The results are presented in tables 1-6 and graphics 1-6. They provide us with useful information in order to better understand the action mechanisms of Aslavital and Apilarnil, as metabolic answer to effort. The interference of Aslavital under physical effort consists of a positive action of equalizing the biologic parameters often treated animals to the control values. The Apilamil increases the muscle glycogen depletion to control, exerting a catabolic influence on the glucidic metabolism. The results allow the Apilarnil to be classified as a strong energizer with an intense catabolic influence.

Keywords: glycaemia, muscle glycogen, physical effort, Aslavital, Apilarnil, glucidic metabolism, liver and muscle glycogen

INTRODUCTION

Case Study. Procaine administration - as a basic substance of Aslavital - increases brain resistance to various aggressors, including rat's resistance to swimming, protecting against hypoexy *(Stroienescu et al,* 1981). Starting fram indirect analyses (studies) and from empirical practice, we believe that Apilarnil - a substance based on bee drones - plays a positive rale in preventing and diminishing tiredness, in improving vital capacities of the disabled organism.

Although we do not possess all necessary information, it is supposed that the two Romanian pharmaceutical products mentioned above have an inf1uence on physical effort, prabably increasing animals'resistence to effort.

Our purpose of this study to determine if the treatment with the Apilarnil and Aslavital used in swimming effort, influiences for changes in lever and muscle glucogenolysis.

MATERIAL AND METHOD

The experiments were done on white Wistar rats aged 6-10 months and weighing 140-200g. Animals were submitted to a tiring test, consisting of free swimming: 90 minutes and 5 hours acute swimming, a five-hour-swimming within 7 days, a five-hour-swimming within 14 days. Swimming took place in the absence or after previous treatment of the ani mals with Aslavital (1 ml, in a 10 *mg/kg/body)* and Apilarnil (3x1 p.o.). Glicemy was investigated following the God-Perid method *(Werner et al,* 1970) and lever

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glycogen and muscle glycogen following the *Montegomery* method.

RESULTS AND DISCUSSIONS

The received data concerning the effect of the acute and chronicle swimming in the presence or in the absence of Aslavital over glicemy can be seen in fig. 1 and in table 1. The effects on lever glycogen in table 2 and fig. 2, and those over muscle glycogen and muscle glycogen following the *Montgomery* method.

I. Changes of certain parameters of the glucidic metabolism in effort after the treatment with Aslavital

1. Glicemy variations. As it can be seen from table 1 and fig. **1,** except for the 90 minutes acute swimming, which shows a significantly high value of the glicemy in all animal batches submitted to effort by swimming, the glicemy values obtained were visibly inferior to those ofthe witness animals. The decrease in glicemy was possible only in the case of the 5 hours swimming both in the acute experiment and in the chronicle swimming, 14 days after beginning swimming. The decrease is not significant from the statistic point of view, 7 days after the training.

Our results show the inf1uence of the Aslavital treatment on the glicemy variation inducted under effort by swimming. Although differences are not statistically significant.

When there is an important hypo-glicemy increase, the treatment with Aslavital seems to constantly induce the glicemy decrease. This effect can be closely related to the recent dataprovided by *Madar et al,* (1975) that signalled stimulation under Aslavital inf1uence both in glucose penetration and in glucose spreading marked by glycogen in the rat's diaphragm under "in vitro" conditions.

Table 1

Glycaemia variations under swimming effort and the Aslavital treatment

Experimental	Parameters	Witness	Control		Treated
type	(Dif. %; p; n; mg/gm)		90'	5h	body
Acute swimming	mg/gm	70±1.27	85±2.26	59±0.68	50±1.29
	Dif. % (x)	-	+21.43	-15.71	-28.58
	p	< 0.001	< 0.001	< 0.001	< 0.001
	n	9	9	9	8
	Dif. % (0)	-	-	-	-15.71
	p	0.001	0.001	0.001	0.001
7 days swimming	mg/gm	86±3.14	-	81±4.94	79±4.09
	Dif. %(6x)	-	-	-5.81	-8.14
	p	-	-	< 0.05	< 0.05
	n	13	-	13	13
14 days swimming	mg/gm	74±2.32	-	56±1.32	52±2.31
	p	0.001	0.001	0.001	0.001
	n	11	-	12	11

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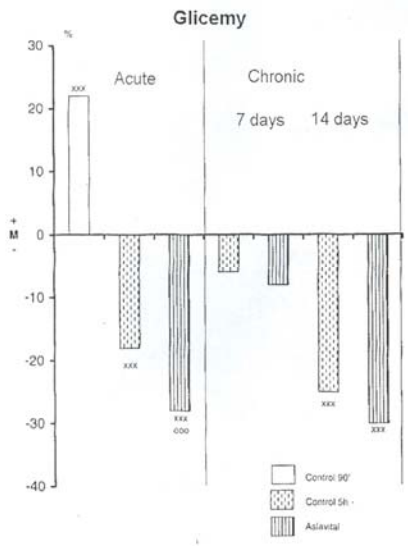

Fig. 1 Glycemy varialions under effort and treatment with Aslavital (xxx = p < 0,001 compared to the vllitness; 000 = p < 0,001 compared to the control).

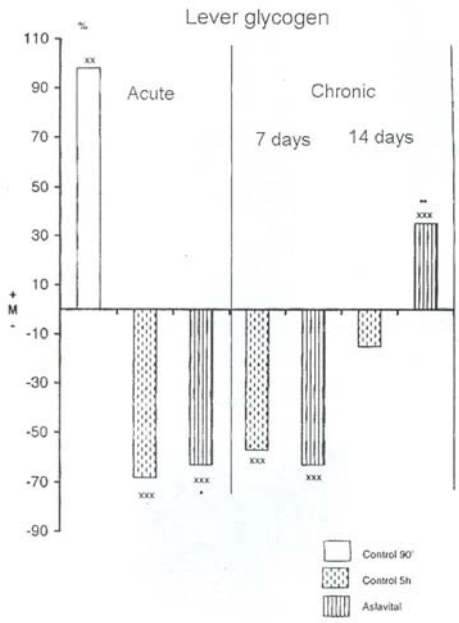

Fig. 2 Changes of the lever glycogen on rats submitted to effort and treated with Aslavital (x = p<0,05; xxx = p<0,001 compared to the witness; 0 = p < 0,001; 00 = p < 0,001 compared to the witness)

2. The variations of the lever glycogen concentration. Our results presented in table 2 and Fig. 2 focus on the dynamics of lever glycogen concentration at different moments of the effort and provide new data, useful for the understanding of the metabolic mechanisms involved in the adapting and effort resistance processes, as well as in the nature of the interference degree of the Aslavital with these processes.

As it can be seen from the presented data, under acute effort we can notice a significant increase in lever glycogen to 90 minutes and it will, on the contrary, significantly decrease after five hours of swimming. The results focus on the great variability of this parameter. These increases are not mentioned in the literature, which, in exchange, offers very controversial data concerning the lever glycogen variations during the first 30 minutes of swimming, even under thermo-neutral conditions *(Dawson and Horvath, 1970).*

The increase in the glycogen concentration is caused by a possible glycogenesis activity under the lactate concentration increase. Such a hypothesis is supported by histo-enzymatic results that we have followed.

Most interesting is the fact that the five-hour-swimming is followed by important decreases of the lever glycogen 01' sometimes unimportant decreases according to the training duration (period). As it can be seen from our data, compared to a - 69,62 % de crease of the lever glycogen content (percentage) following the non-trained swimming, after 7 days the decrease is of - 56,67 % (which is + 48,96 % more compared to the non-trained batch). 14 days after the training, the lever glycogen content is unchanged compared to that of the witnesses. Therefore, our data show that training has a huge impact on the body's capacity to adapt to over-effort conditions by a more economical use of the energetic reserves.

A special attention has been given to the results concerning the influence that Aslavital exerts over the changes inducted by effort in as far as the lever glycogen concentration is concerned. Our results show that there is a stimulation of the synthesis processes of the glycogen in treated rats, compared with the non-treated ones. The conclusion is that Aslavital action/activity influences the lever, as well as the skeletal muscle, through its "insulin-like" effects, mentioned above.

Table 2

Variations of the lever glycogen concentration in rats submitted to swimming effort and the Aslavital treatment (mg/100g of fresh tissue)

Experiment type	Parameters (Dif. %, n, p, mg%)	Witness	Control		Treated body
			90'	5h	
Acute swimming	mg%	316±18.10	616±106^{xxx}	96±9.61^{xxx}	117±1.29^{xxx}
	Dif. % (x)	-	+94.94	-69.62	-63.00
	p	< 0.01	< 0.01	< 0.001	< 0.001
	Dif. % (0)	-	-	-	+21.88
	p	-	-	-	0.05
	n	9	9	9	9
7 days swimming	mg%	330±33.07		143±17.51^{xxx}	118±14.77^{xxx}
	Dif. % (x)	-	-	-56.67	-64.24
	p	-	-	(p < 0.001)	(p < 0.001)
	Dif. % (0)	-	-	-	-17.48
	p	-	-	-	(p 0.1)
	n	11	-	13	13
14 days swimming	mg%	71±2.67	-	61±4.61	99±13.94^{00}
	Dif. % (x)	-	-	-14.18	+39.44
	p	-	-	< 0.1	< 0.1
	Dif. % (0)	-	-	-	+62.30
	p	-	-	-	0.02
	n	10	-	12	12

Note: The statistically significant variations were marked xxx = p 0,001 for the witness and 0 = p < 0,05; 00 = p < 0,02 for the control batch.

3. Muscular glycogen variations. The results of the present experiment are in perfect accordance with the data reported by other authors *(Dawson and Horvath,* 1970). Table 3 and figure 3 show these results. 90 minutes and especially 5 hours swimming even under thermo-neutrality conditions reduces considerably the muscle glycogen concentration.

The training seems to significantly improve muscle capacity to keep and to use the energetic reserves. Compared to the values obtained after the five hours swimming in the acute and the chronicle experiment, slight differences can be noticed. The – 84.60% decrease obtained in the first case goes back to – 54.76% under the 14 days training conditions.

In as far as the Aslavital inf1uence is concerned, although the value variation does not attain the statistical significance limit, under effort conditions, the glycogen concentration records differences of + 41,56 % (acute swimming) and + 15,79 %

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(chronicle swimming). The increasing tendency of the muscle glycogen concentration together with the more significant decreasing tendency of glycaemia suggests the possible effect called "insulin-like".

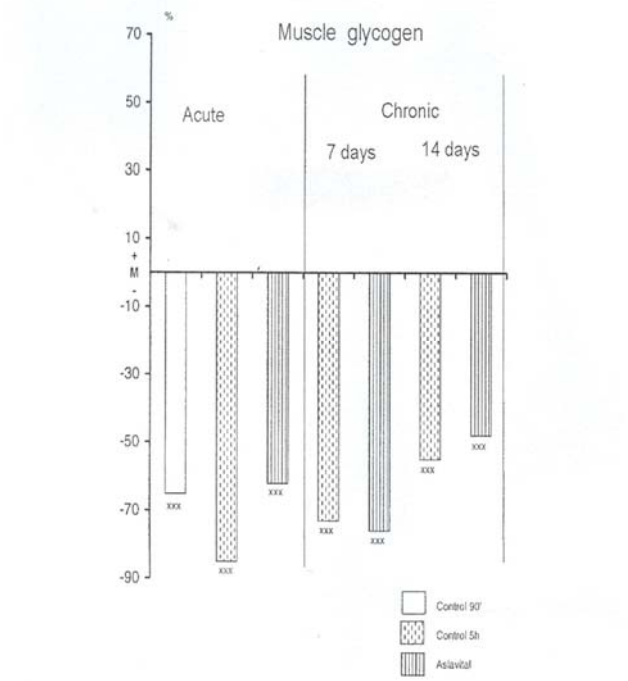

Fig. 3 The effects of the effort and treatment with Aslavital on themuscle glycogen concentration (xxx = $p < 0.001$ compared to the witness).

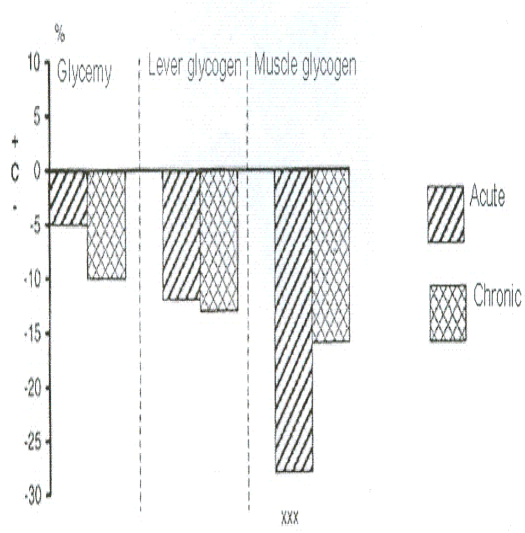

Fig. 4 Changes of the glycemy, of the lever and muscle glycogen under effort and treatment with Apilarnil

II. Changes of some parameters of glucidic metabolism in effort after the Apilarnil treatment

1. Glycaemia variations. By analyzing the results from table 4 and the Fig. 4, 5, 6, we underlined a strong decrease of the glycaemia after the acute swimming. The treatment slightly increases hypo-glycaemia induced by effort. The training does not inf1uence the glycaemia values and the hypo-glycemic estate is maintained after 14 days of chronicle swimming. Yet, a slight decrease of the variation amplitude can be noticed. Although with no statistical significance, the glycaemia decrease in the treated batch, also noticed within the acute swimming, shows up to be more stressed compared to the control batch. Therefore, we can say that this may be one of the causes of mortality within the treated batch.

2. The variations of the lever glycogen concentrations. The glycogen concentration of the lever significantly changes, with important decreases that could be seen in both batches submitted to swimming, as can be seen from the table 5 and Fig. **4,** 5 and 6. Compared to the witness batch, there is an important decrease for the control batch and the treated batch under acute swimming, while 14 days after chronic swimming although significant compared to the witness' values, the decrease is less important, and these are the favorable effects of the training on a more economic al use of the liver glycogen.

The Apilarnil treatment has the tendency to increase lever glycogen depletion and its values are low in eight experiment types, compared to the control, although the changes are not statistically significant.

Table 3

Changes of the muscle glycogen concentration in rats submitted to swimming effort and the Aslavital treatment (mg/100g of fresh tissue)

Experiment type	Parameters (Dif. %,p,n,mg)	Witness	Control		Treated body
			90'	5h	
Acute swimming	mg	512±53.50	170±28.42^{xxx}	77±7.23 ^{xxx}	109± 15.86^{xxx}
	Dif. % (x)	-	-66.80	-84.60	-78.30
	p	-	< 0.001	< 0.001	< 0.001
	n	9	9	9	9
	Dif. % (0)	-	-	-	+41.56
	-	-	-	-	(p < 0.1)
7 days swimming	mg	436±19.98	-	111±14.23^{xxx}	109±13.47^{xxxx}
	Dif. % (x)	-	-	-74.54	-75.00
	p	-	-	< 0.001	< 0.001
	Dif. % (0)	-	-	-	-1.80
	n	12	-	14	13
14 days swimming	mg	462±15.44	-	209± 18.16^{xxx}	242±24.25^{xxx}
	Dif. % (x)	-	-	-54.76	-47.60
	p	-	-	< 0.001	< 0.002
	Dif. % (0)	-	-	-	+15.79
	p	-	-	-	> 0.1
	n	9	-	12	9

Note: The statistically significant variations were marked xxx = p 0.001.

Table 4

The Apilarnil effect on white rat`s glycemy (mg%)

Witness	Acute effort		Chronicle effort	
	Control	Treated body	Control	Treated body
81±1.46	46±1.95	44±1.52	52±2.55	47±1.86
Dif. %1	-43.21	-45.68	-35.80	-41.98
p	< 0.001	< 0.001	< 0.001	< 0.001
Dif. %2	-	-4.35	-	-9.62
p	-	> 0.25	-	> 0.25
n8	9	8	9	9

Table 5

The variations of the lever glycogen in the white rat submitted to effort and treated with Apilarnil (mg/100g of fresh tissue)

Witness	Acute effort		Chronic effort	
	Control	Treated body	Control	Treated body
X±ES 1217±73.23	83±4.20	74±5.89	581±23.34	513±61.74
Dif. %1	-93.16	-93.90	-52.14	-57.74
p	< 0.001	< 0.001	< 0.001	< 0.001
Dif. %2	-	-10.84	-	-11.70
p	-	> 0.25	-	> 0.25
n9	10	8	9	9

3. The variations of the muscle glycogen concentration. According to the data from table 6 and graphics **4-6,** the treatment with Apilarnil increases musc1e glycogen raising, and this is obvious under acute effort, when the decrease variation touches highly significant values (p<0.001) and less present as a consequence of chronic effort. Therefore, as it can be seen from figure 6, Apilarnil seems to exert a catabolic influence on the glucidic metabolism, even if the constant decreasing tendency does not always touch (reach) the statistic signification level.

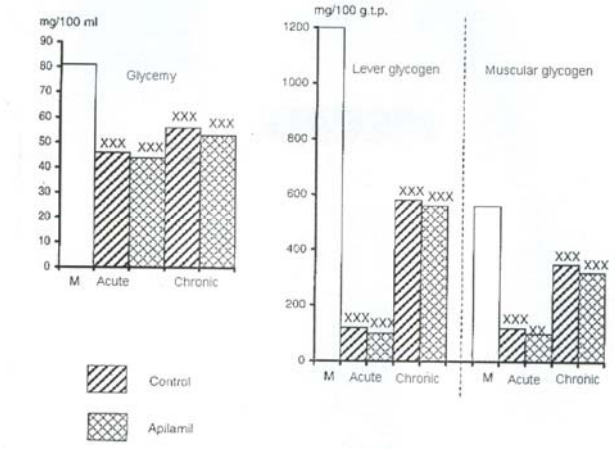

Fig. 5 Percentage changes of the glycemy of lever and muscle glycogen under effort and treatment with Apilarnil, compared to the witness

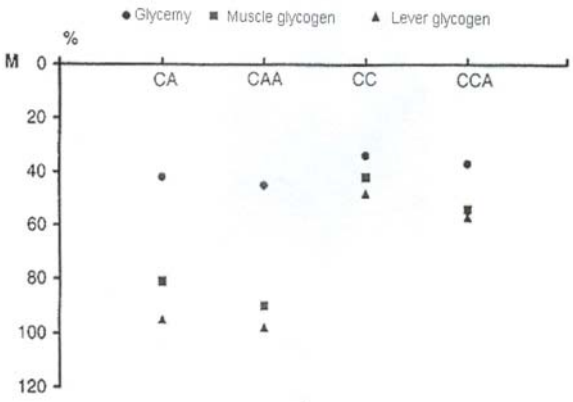

Fig. 6 Indicators for glucidic metabolism on rats submitted to effort and treatment with Apilarnil.

General Debates concerning

I. The Aslavital interference with the glucidic metabolism during effort. The results supply (provide) useful information in order to better understand the action mechanisms of Aslavital - metabolic adaptation and neuro endocrino10gic answer under the effort.

Aslavital produces changes in the glucidic metabolism in effort - a decrease in glycemy simultaneously with a synthesys stimulation of the muscle glycogen but especially the lever one, as well as of the mitocondrial enzymes (CyOx, SDH, ATP) and of the glicogenesys (G-6-P and G-6-PDI-I). We should also like to underline the improvement ofthe oxygen in the lever tissue, certified by the Aslavital's action in maintaining LDH activity in the batches submitted to effort. The result is a protective action on mitocondrial membranes.

The effect of the Aslavital treatment can be seen in the complex neuro-endocrine answer, and an important component ofthis are the hormones.

Table 6

The variations of muscle glycogen quantity in the white rat submitted to effort and treated with Apilarnil (mg/100g of fresh tissue)

Witness	Acute effort		Chronic effort	
	Control	Treated body	Control	Treated body
X±ES 515±20.84	84±4.58	59±4.44	285±14.03	246±13.52
Dif. %1	-83.69	-88.54	-44.16	-52.23
p	< 0.001	< 0.001	< 0.001	< 0.001
Dif. %2	-	-29.76	-	-13.68
p	-	< 0.001	-	>0.25
n	10	8	9	9

II. The Apilarnil interference with effort glucidic metabolism. Animals treated with Apilarnil were agitated (nervous) and tired during the swimming and this shows obvious effects of the product both on the glucidic metabolism and on the endocrine feedback.

The significant decrease of glycaemia in treated batches, compared to the control batches probably is the cause of mortality (16-19 %) of the animals submitted to effort.

The Apilarnil increases glycogen depletion, exerting a catabolic influence on the glucidic metabolism.

The Apilarnil constantly induces a decrease of the enzymes' activity in as far as the lever is concerned. The Apilarnil intensifies the rats' kidney gland, the glycogen depletion and it decreases acids and substances. It makes us believe that there takes place intensification of the energetic processes and of bio-synthesis of glucocorticoids hormones.

The product is characterized by an increase in the changes induced by effort and their maintenance in time. Therefore, our results allow us to classify the Apilarnil among the strong energizers, with an intense catabolic influence.

CONCLUSIONS

The Aslavital administration on rats leads to a hormone balance, an insulin-like effect ensuring an economical use of the energetic reserves, simultaneously with an amplification of the anabolic processes in trained animals. This fact makes that Apilarnil is a complex product for the increase of effort resistance and preventing tiredness.

Due to its strong catabolic influence on the body, Apilarnil is a strong energizer, stimulating oxidative processes giving birth to energy. In order to maintain it to performance levels, it must be administered together with energetic supplies necessary to the muscles.

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