EFFECT OF VITAMIN P ON ASCORBIC ACID METABOLISM IN ANIMALS TEMPORARILY EXPOSED TO A HIGH ENVIRONMENTAL TEMPERATURE

A. Ya. Tseitina

Laboratory of Vitamins C and P (Head, Prof. N. S. Yarusova), Research Institute of Vitaminology (Director, Candidate Biol. Sci. M. I. Smirnov), Ministry of Health of the USSR, Moscow
(Presented by Active Member AMN SSSR B. A. Lavrov)
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 56, No. 12, pp. 52-55, December, 1963
Original article submitted February 16, 1963

There are reports in the literature that a short period of overheating causes a marked fall in the ascorbic acid level in the organs of experimental animals [2,5,7,9,10]. A functional relationship has been established between ascorbic acid and vitamin P. In particular, although vitamin P preparations have no significant effect on the accumulation of ascorbic acid in the healthy organism [1,8], they influence this process favorably in pathological conditions [6].

The object of the present investigation was to study the effect of vitamin P preparations (bioflavonoids) on ascorbic acid metabolism of animals exposed to overheating, employing for this purpose both animals which synthesize vitamin C (albino rats) and animals incapable of synthesizing vitamin C (guinea pigs).

EXPERIMENTAL METHOD

Experiments were conducted on 78 male rats weighing about 250 g, kept on a casein diet [8], and on 73 guinea pigs (also males) weighing about 500 g, receiving a modified Lecoq's diet [1] and 25 mg vitamin C daily.

The rats and guinea pigs were kept on these diets for 3-4 weeks, after which they were exposed to one period of overheating for 1 h to a temperature of 40-42° and relative humidity of 26-31%. The overheating took place in an incubator fitted with a device for ensuring circulation of air.

The animals were divided into four groups (each containing 10-20 animals). The animals of the first (control) group were kept at 20-22°. The rats and guinea pigs of the second, third, and fourth groups were overheated in the conditions described above, and in addition the animals of the third group received rutin, and those of the fourth group received tea catechins (rats 5 mg each, guinea pigs 10 mg each, daily).

Before the animals were placed in the incubator, their rectal temperature was taken with a mercury thermometer. Immediately after exposure of the animals to heat, their rectal temperature was again taken, after which they were decapitated and the content of ascorbic acid (AA), dehydroascorbic acid (DAA), and diketogulonic acid (DGA) in the adrenals, liver, and blood serum was determined [12]. Organs taken simultaneously from 2 rats were combined for one analysis because of the small weight of their adrenals.

EXPERIMENTAL RESULTS

Keeping the animals at a high atmospheric temperature for 1 h led to elevation of their rectal temperature by 1.0-1.5°. The presence of vitamin P preparations in the diet had no significant effect on the changes in the body temperature.

It is clear from Table 1 that a single exposure to a high temperature caused a marked, statistically significant decrease in the AA content in the adrenals and liver of the experimental animals not receiving vitamin P by comparison with the control animals kept at room temperature. In the animals receiving tea catechins, practically no decrease in the AA content of the test organs as a result of overheating could be observed. Rutin was less effective and did not always prevent a decrease (by 50-100%) was observed in the DAA level in these organs in the rats and guinea pigs compared with the control values (Table 2).

TABLE 1. Effect of Vitamin P (Rutin or Tea Catechins) on Content of AA (in mg-%) in Adrenals and Liver of Experimental Animals Exposed to Overheating

Test animals	Organ	Group of animals			
		first	second	third	fourth
Rats	Adrenals	497,3±12,9 $P<0,001$	403,0±19,5	383,0±14,0 $P>0,5$	470,0±13,4 $P<0,01$
	Liver	34,9±1,18 $P<0,01$	30,3±0,87	33,6±0,87 $P<0,02$	34,5±1,18 $P<0,01$
Guinea pigs	Adrenals	125,5±4,88 $P<0,001$	94,2±3,78	96,6±4,19 $P>0,5$	123,0±3,15 $P<0,001$
	Liver	11,4±0,54 $P<0,001$	7,8±0,51	9,1±0,60 $P<0,05$	9,7±0,44 $P<0,01$

Note. 1) Mean values deduced from determinations in not less than 10 animals. 2) Values of significance of differences given by comparison with results obtained with animals of second group.

In the animals preliminarily receiving tea catechins, hardly any changes were observed in the DAA content of the liver and adrenals as a result of exposure to a high temperature. The action of rutin was less marked, and it almost failed to prevent an increase in the DAA content of the organs of animals exposed to overheating. In contrast to AA and DAA, the DGA content of these organs underwent very little change after exposure to a high temperature, irrespective of the presence of bioflavonoids in the diet.

In addition to the changes in the AA and DAA content of the liver and adrenals during overheating of the rats and guinea pigs, a marked increase in the AA level in the blood serum was observed (Table 3). The preliminary administration of tea catechins to the animals inhibited the increase in AA concentration during overheating only in the case of the guinea pigs. Rutin, on the other hand, prevented the increase in the serum AA concentration in the rats.

Concurrently with the rise of temperature, an increase in the combined DAA and DGA concentrations in the serum was observed (see Table 3). Catechins prevented this increase only in guinea pigs. Rutin, on the other hand, had no such action and the DAA and DGA levels in the serum were close to those observed in the overheated animals not receiving vitamin P.

Analysis of the results shows that the decrease in the AA concentration in the adrenals during exposure to a high temperature is the result of a general, nonspecific reaction of the organism to the thermal stimulus. During the action of an excessively strong agent, exceeding the normal limits (cold, heat, trauma, muscular effort, and so on), a state of stress develops in the body, giving rise to a general adaptation syndrome [15]. The stimulation of the secretory function

TABLE 2. Effect of Vitamin P (Rutin and Tea Catechins) on DAA Content (in mg-%) of Adrenals and Liver of Experimental Animals Exposed to Overheating

Test animals	Organ	Group of animals			
		first	second	third	fourth
Rats	Adrenals	10,7±0,54 $P<0,001$	16,4±1,22	13,9±1,20 $P>0,2$	10,0±0,69 $P<0,001$
	Liver	0,45±0,040 $P<0,01$	0,81±0,092	0,50±0,036 $P<0,01$	0,39±0,029 $P<0,001$
Guinea pigs	Adrenals	3,9±0,35 $P<0,001$	8,1±0,68	7,2±0,71 $P>0,5$	3,8±0,38 $P<0,001$
	Liver	0,22±0,02 $P<0,001$	0,39±0,03	0,33±0,005 $P>0,2$	0,21±0,021 $P<0,001$

TABLE 3. Effect of Vitamin P (Rutin and Tea Catechins) on AA, DAA, and DGA Contents (in mg-%) in Serum of Experimental Animals Exposed to Overheating

Test animals	Components	Group of animals			
		first	second	third	fourth
Rats	AA	1,08±0,051	1,28±0,040 $P<0,01$	1,10±0,066 $P<0,05$	1,16±0,103 $P>0,2$
	DAA+ DGA	0,21±0,021	0,27±0,032 $P>0,2$	0,26±0,034 —	0,22±0,27 $P>0,2$
Guinea pigs	AA	0,26±0,02	0,39±0,03 $P<0,01$	0,39±0,03 —	0,28±0,03 $P<0,02$
	DAA+ DGA	0,13±0,009	0,19±0,018 $P<0,01$	0,21±0,027 $P>0,5$	0,14±0,015 $P<0,05$

of the cortex arising in these circumstances is regularly accompanied by a decrease in the AA content of the adrenals [14]. It may also be postulated that the decrease observed in the AA level, accompanied by a decrease in the DAA level in the test organs, takes place as a result of a weakening of the reduction phase in the reaction:

$$AK \underset{\text{reduction}}{\overset{\text{reversible oxidation}}{\rightleftharpoons}} DAK,$$

observed during exposure to a thermal stimulus [3]. The addition of tea catechols to the animals' diet practically prevented these changes in the AA and DAA contents of the organs, presumably on account of the ability of catechins to stimulate reduction of DAA to AA with the aid of glutathione, and thereby to diminish the oxidative losses of AA in the body [4,11].

The increase in the concentration of AA in the blood following exposure to overheating could take place as a result of an increased output of AA from the adrenals into the blood stream [13].

LITERATURE CITED

1. N. N. Berzovskaya, A. Ya. Tseitina, and S. A. Lapina, Vopr. pitaniya, 5, 26 (1962).
2. S. I. Vinokurov, Ukr. biokhim. zh., 2, 89 (1938).
3. S. I. Vinokurov and M. L. Butom, Ukr. biokhim. zh., 12, 551.
4. I. I. Matusis and M. I. Pechenina, Abstracts of Proceedings of the 3rd Scientific Session of the Institute of Vitaminology, Moscow (1960), p. 23.
5. É. L. Perel'man and F. F. Sultanov, Izv. Akad. Nauk Turkmensk. SSR, seriya biol., 1, 48 (1962).
6. E. F. Shamrai, N. S. Verkhratskii, U. A. Kuz'minskaya, et al., in the book: Vitamin Resources and their Utilization [in Russian], collection 4, p. 30, Moscow (1959).
7. F. M. Shleifman, Gig. i san., 5, 14 (1961).
8. N. S. Yarusova, V. M. Selivanova, and S. A. Lapina, Vopr. pitaniya, 5, 66 (1957).
9. T. Juszkiewicz, Am. J. vet. Res., 22, 537 (1961).
10. M. Kurioka, Vitamins, 16, 415 (1959).
11. J. L. Parrot and J. M. Gazave, C. R. Soc. Biol., 145, 821 (1951).
12. J. Roe, M. Mills, M. Oesterling, et al., J. biol. Chem., 174, 201 (1948).
13. S. L. Salomon, Tex. Rep. Biol. Med., 16, 137 (1958).
14. M. A. Sayers, G. Sayers, and L. A. Woodbury, Endocrinology, 42, 379 (1948).
15. H. Selye, The Story of the Adaptation Syndrome, Montreal (1952).