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Effects of Ethanol on Body Temperature of Rats at High Ambient Pressure

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BERGE, O.-G. AND I. GARCIA-CABRERA. *Effects of ethanol on body temperature of rats at high ambient pressure.* PHARMACOL BIOCHEM BEHAV **39**(1) 37–41, 1991.—Separately, ethanol and high ambient pressure cause hypothermia in laboratory animals. However, ethanol and high pressure have mutually antagonistic effects on several biological functions and the present experiments investigate their combined action on body temperature. Rats given saline, 1.5 g/kg ethanol or 3.5 g/kg ethanol were exposed to 1 bar air at 25–26°C, 1 bar helium-oxygen at 30–31°C, or 48 bar helium-oxygen at 33.5–34.5°C. Ambient, colonic and tail-skin temperatures were monitored for 60 min. There were no significant differences in mean ambient or tail-skin temperatures between groups belonging to the same ambient condition. Colonic temperatures under the 1 bar conditions were 1.5–2°C lower in the 3.5 g/kg ethanol group than in the saline and 1.5 g/kg ethanol groups, while no significant differences were observed between the groups at 48 bar. Comparisons of the colonic temperatures at the end of the observation period, i.e., 60 min after administration of ethanol, demonstrated that their values at 48 bar were significantly lower than at 1 bar after saline, significantly higher after 3.5 g/kg ethanol and identical across conditions in the 1.5 g/kg groups. The results suggest that high ambient pressure may counteract rather than potentiate the hypothermic effect of ethanol.

Ethanol Hyperbaric conditions Hypothermia Rat Colonic temperature Thermoregulation

REVERSAL of some effects of ethanol by high ambient pressure has been demonstrated in several species (1, 13, 17, 19, 20, 34). Conversely, recent studies show that ethanol protects animals against some of the aversive effects of high pressure (14,15). Although alteration in body temperature is a possible confounding factor in in vivo experiments dealing with high pressure and ethanol, the literature contains little information on the combined effects of these factors on body temperature and thermoregulation.

The biological effects of ethanol depend on tissue temperature. Hypothermia antagonizes behavioral intoxication in rats and mice (10, 25, 28). The ability of ethanol to reduce the amplitude of squid giant axon action potentials (32) and to reduce the duration of action potentials in cultured rat sensory neurons (8) varies directly with temperature. The net effect of low temperature is thus to reduce the potency of ethanol, although the rate of ethanol elimination may also be decreased (30).

Ethanol does, on the other hand, cause hypothermia in both rats and mice under conditions that favor thermal balance in animals which are not intoxicated (23, 26, 28). The effect may partially be due to a change in thermoregulatory set-point (5, 16, 23). Increased ambient pressure may also lower the set-point for body temperature regulation (7,24). Thus the combined effect of pressure and ethanol is difficult to predict. Mice given a narcotic dose of ethanol and then compressed to 6 bar in heliox at 34.5°C did not exhibit significant changes in rectal temperature (25). At higher pressures, aggravated hypothermia due to combined pressure- and ethanol-induced interference with temperature regulation may be expected. On the other hand, it is possible that mutually antagonistic effects of pressure and ethanol may prevent the hypothermic effects observed when either factor is applied separately. The present study addresses this problem and examines whether ethanol administration under standard hyperbaric conditions may affect body temperature to such an extent as to contribute to the reversal of ethanol intoxication observed in rats in previous behavioral studies.

METHOD

Subjects

Male Sprague-Dawley rats (Mol:SPRD, Møllegård, Denmark) were housed three to a cage. Food was limited to 15 g of pellets per animal per day. Water was freely available. Retrospective analysis of data from several stress-sensitive behavioral experiments indicates that this feeding procedure reduces differences in drug effects caused by inhomogeneity of body weight without interfering with the behavioral tests. The weight at the time of testing was 286 ± 22 g (mean $\pm$ SD) and there were no statistical differences between groups. The light phase lasted from 0800 to 2000 hours and ambient temperature was 22–23°C. All experiments took place between 0830 and 1500 and the various treatment groups were tested in balanced order across

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days and with regard to time of the day. Food but not water was removed one hour before testing.

The Hyperbaric Chamber

Experiments were carried out in a 24.5 l steel hyperbaric chamber equipped with video monitoring, gas supply and temperature control systems as described in detail previously (13).

Colonic and Tail-Skin Temperature Recordings

Each rat was restrained in an acrylic cylinder with an internal diameter of 6.2 cm and of variable length (15–22 cm). The restrainer was perforated to allow free exchange of gas with the rest of the chamber. Colonic temperature probes (Pt-100, length: 6.0 mm, diameter at the tip: 1.3 mm) were inserted 5 cm. Skin temperature probes (Pt-100, $10.2 \times 3.2 \times 0.6$ mm) were taped to the dorsal aspect of the tail, 3 cm from the base. Heat-conducting paste was applied between the skin and the probe which was supplied with a layer of insulating material towards the surrounding gas. Animals were tested two at a time.

Procedure

The animals were exposed to one of three ambient conditions (1 bar air at 25–26°C, 1 bar heliox at 30–31°C, or 48 bar heliox at 33.5–34.5°C, referring to the period 20–60 min after the start of the experiment when all groups were at stable pressure). The temperature and pressure parameters were chosen to allow comparison with previous behavioral studies (13). The actual chamber temperatures recorded during the experiments were analyzed as described below and did not differ significantly between groups. For each condition, the animals were divided into three groups which received intraperitoneal injections of either isotonic saline or a solution of 1.5 g/kg or 3.5 g/kg ethanol in 21 ml/kg saline (corresponding to 0.07 and 0.17 g/ml).

Immediately after injection, the animals were placed in the pressure chamber. When rats belonging to the 1 bar air groups were tested, the chamber was flushed with air from 2 to 20 min after injection. The 1 bar heliox groups received similar treatment except that a mixture of 80% helium and 20% oxygen was used for flushing. The 48 bar heliox groups were treated as the 1 bar heliox groups until 4 min 20 s after injection when compression was started.

The compression was performed with helium at a rate of 3 bar/min so that stable pressure was reached 20 min after injection. Throughout the experiments, the partial pressure of oxygen in the breathing gas was kept between 0.2 and 0.4 bar.

Statistics and Data Presentation

Temperature data for each subject was recorded as the median of 6 sampled temperatures per minute.

For statistical analysis, data for the following four periods were averaged: 1) Zero to 4 min 20 s (the precompression period of the 48 bar groups). 2) Four min 20 s to 20 min (the compression period). 3) Twenty to 40 min. 4) Forty to 60 min. Analysis of variance (ANOVA; 3 groups × 4 periods unless otherwise specified) was employed throughout.

In addition, colonic temperatures recorded during the first and last minute of the experiment were evaluated by ANOVA (3 conditions × 3 doses × 2 periods) and when appropriate, Scheffé's test was used after one-way ANOVA for post hoc comparison of mean values.

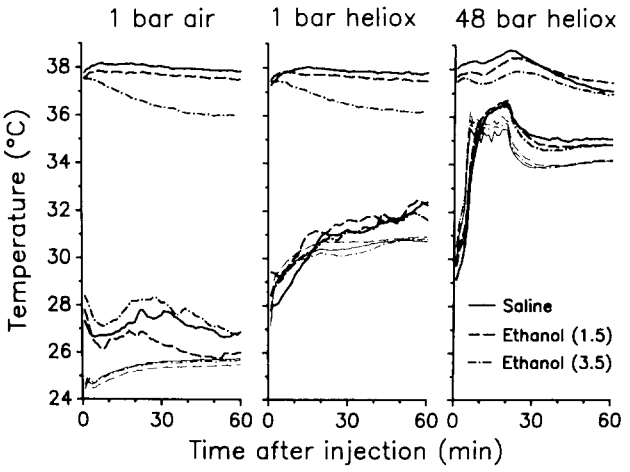

FIG. 1. Colonic (upper thick lines), tail (lower thick lines) and chamber temperatures (thin lines) obtained from experiments in air at 1 bar and heliox at 1 bar or 48 bar. Saline or ethanol were given by IP injection at time 0. Compression to 48 bar took place between 4 min 20 s and 20 min at a rate of 3 bar/min. Each line represents mean values of 7–8 experiments, calculated for each min of recording.

RESULTS

Tail-Skin Temperature

At 1 bar air, the group that had received 1.5 g/kg ethanol had slightly lower tail-skin temperatures than the other two groups (Fig. 1), but the difference did not reach significance, $F(2,21) = 2.96$, $0.05 < p < 0.10$. At 1 bar heliox, the progressive rise in temperatures throughout the recording period yielded a significant periods effect, $F(3,60) = 26.63$, $p < 0.00001$, but there was no tendency to group difference or interaction. Similarly, analysis of the data from the groups that were compressed to 48 bar showed a significant periods effect, $F(3,63) = 235.56$, $p < 0.00001$, but no difference between groups and no interaction. Thus there were no significant differences in mean tail-skin temperature between groups belonging to the same ambient condition.

Colonic Temperature

At 1 bar air, the mean colonic temperatures in the saline and 1.5 g/kg ethanol groups increased slightly during the first 10 min after injection and subsequently dropped steadily (Fig. 1) so that the average temperature at the conclusion of the observation period was the same as at the start for both groups. In contrast, the colonic temperatures in the 3.5 g/kg ethanol group gradually dropped between 1.1 and 2.3°C (mean: 1.5°C). Statistical analysis of the average temperatures recorded during the observation periods demonstrated highly significant differences between groups, $F(2,21) = 35.42$, 2.96, $p < 0.000001$, and between periods, $F(3,63) = 23.62$, $p < 0.000001$, and a reliable interaction, $F(3,63) = 15.63$, $p < 0.000001$. Further comparison between the saline-treated animals and the rats that received 1.5 g/kg ethanol (2 groups × 4 periods ANOVA) demonstrated only a nonsignificant tendency to group differences, $F(1,14) = 4.13$, $0.05 < p < 0.10$, and no interaction between groups and periods, whereas a similar comparison between the saline and 3.5 g/kg group revealed highly significant effects of group, $F(1,14) = 55.09$, $p < 0.000001$, period, $F(3,42) = 24.82$, $p < 0.000001$, and interaction, $F(3,42) = 19.74$, $p < 0.000001$.

TABLE 1

COLONIC TEMPERATURES (MEAN ± S.E.M., n = 7–8 IN EACH GROUP) BASED ON THE MEDIAN OF VALUES RECORDED DURING THE FIRST (INITIAL) AND THE SIXTIETH MIN (FINAL) AFTER ADMISSION TO THE CHAMBER

Pressure	Dose (g/kg)	Temperature (°C) Initial	Final
1 Bar air	0.0	37.8 ± 0.2	37.8 ± 0.1
	1.5	37.6 ± 0.1	37.5 ± 0.1
	3.5	37.5 ± 0.2	36.0 ± 0.1‡
1 Bar heliox	0.0	37.3 ± 0.2	37.8 ± 0.2
	1.5	37.5 ± 0.2	37.5 ± 0.2
	3.5	37.4 ± 0.2	36.2 ± 0.2‡
48 Bar heliox	0.0	38.0 ± 0.2	37.1 ± 0.2*
	1.5	37.7 ± 0.2	37.5 ± 0.1
	3.5	37.5 ± 0.1	37.0 ± 0.1†

*Significantly different from the final temperatures of the saline groups at 1 bar air and 1 bar heliox, $p<0.02$; †significantly different from the final temperatures of the 3.5 g/kg groups at 1 bar air, $p<0.0005$ and 1 bar heliox, $p<0.002$; ‡significantly different from the saline group and the 1.5 g/kg ethanol group within the same condition, $p<0.001$, Scheffé's test subsequent to ANOVA.

The data obtained at 1 bar heliox were similar to the results of the air groups. ANOVA demonstrated highly significant effects of group, $F(2,20)=12.51$, $p<0.0005$, period, $F(3,60)=9.10$, $p<0.0001$, and interaction, $F(3,60)=11.41$, $p<0.00005$, while further comparison between the saline-treated animals and the rats that had received 1.5 g/kg ethanol (2 groups × 4 periods ANOVA) demonstrated no significant group difference or interaction. As at 1 bar air, significant effects of group, $F(1,13)=18.75$, $p<0.002$, period, $F(3,39)=9.82$, $p<0.0002$, and interaction, $F(3,39)=22.74$, $p<0.000001$, were evident between the saline and the 3.5 g/kg group.

The compressed groups showed a different pattern with temperatures rising by 0.5–1.0°C during the second half of the compression period and dropping by between 1.7°C (saline group) and 0.9°C (3.5 g/kg ethanol group) during the 40 min at stable pressure. The two groups that had received ethanol followed a parallel course, with the values of the 1.5 g/kg group being 0.3–0.6°C higher. Statistical analysis demonstrated significant differences between periods, $F(3,63)=26.91$, $p<0.000001$, and significant groups × periods interaction, $F(3,63)=3.45$, $p<0.01$. Further analysis (3 groups × 2 periods ANOVA) restricted to the last two observation periods did not, however, yield significant group difference or interaction. Thus no difference in mean colonic temperatures could be demonstrated between the groups during the periods at 48 bar.

The colonic temperatures recorded during the first and the last minute of observation are shown in Table 1. There was a highly significant overall interaction between ambient conditions, doses and sampling periods [$F(4,62)=9.89$, $p>0.00001$; $3\times3\times2$ ANOVA]. The initial temperatures were similar in all groups and separate analysis of these data revealed no main effects of conditions or doses and no interaction effect (3×3 ANOVA). Analysis of the data obtained at 60 min showed a significant interaction between pressure and dose [$F(4,62)=11.48$, $p<0.00001$, 3×3 ANOVA] and one-way ANOVA demonstrated significant differences between groups at 1 bar air, $F(2,21)=63.10$, $p<0.00001$, and at 1 bar heliox, $F(2,20)=29.23$, $p<0.00002$, but not at 48 bar, $F(2,21)=2.95$, $0.05<p<0.10$. Post hoc evaluation of the final colonic temperatures confirmed that at 1 bar, the 3.5 g/kg groups had significantly lower temperatures than the other groups (Table 1). Furthermore, the values at 48 bar were significantly lower than at 1 bar after saline, significantly higher after 3.5 g/kg ethanol and identical across conditions in the 1.5 g/kg groups.

DISCUSSION

Several factors may have contributed to alleviate ethanol-induced hypothermia at 48 bar in this study. It seems likely that pressure reversal of ethanol-induced hypothermia took place in the 3.5 g/kg ethanol group. We have previously reported complete reversal of the depressant effect on spontaneous behavior of 1.5 g/kg ethanol in rats exposed to 48 bar (13). The effect of 3.5 g/kg was not significantly reversed, even at a pressure of 72 bar (15), but the study employed a method less suitable to detect partial reversal of intoxication and do not preclude pressure reversal in the present experiments. Others have found reversal of the depressant effect of high doses of ethanol on the righting reflex of mice at pressures of 4–12 bar (25).

The magnitude and direction of body temperature changes in ethanol-intoxicated animals are modified by ambient temperature (12, 26, 28). It is unlikely, however, that the lack of ethanol-induced hypothermia at 48 bar was due to the ambient temperature. Under the conditions of this experiment, the heat loss to the environment is augmented both by the thermal properties of helium which is substituted for nitrogen to prevent narcosis, and by the higher density of the hyperbaric gas. At 1 bar of 80% helium and 20% oxygen (heliox), an ambient temperature of approximately 30°C is required to prevent increased oxygen consumption in rats (22). At pressures greater than 35 bar, temperatures between 33°C and 35°C are needed to maintain thermal balance in several species, including the rat (31, 33, 35). Previous experiments have shown identical levels of oxygen consumption in groups of rats exposed to ambient conditions similar to the three conditions employed in the present experiments (33). Thus the heat transfer to the environment should be approximately equal across conditions in the present studies. Also, the pronounced drop in body temperature exhibited by the saline group at pressure demonstrates that heat-loss was possible in the 48-bar condition. The situation may have been different during parts of the compression period when the ambient temperature was approximately 1.5°C higher than at pressure. The parallel rise in body temperature shown by all groups during the last part of this period may have been caused by a combination of factors, including environmental temperature and compression effects.

Under pressure, the saline-treated group showed a more pronounced drop in temperature than the other groups, and its mean temperature at the end of the observation period was significantly lower than at 1 bar air or heliox, a finding which is compatible with a pressure-induced lowering of the thermoregulatory set-point (7,24). It is possible that this effect was reduced in the other groups since anesthetics, including ethanol, may protect against some actions of pressure (14,17).

It is unlikely that any of the observed effects were due to differences in blood and brain ethanol concentrations between groups given the same dose of ethanol in the present study. No differences in blood or brain ethanol concentrations were previously detected between groups of rats given the same doses and exposed to the same ambient temperatures and pressures as in these experiments (13,15). In other studies, no difference in the ethanol clearance rate was found between hyper- and normothermic rats (9). Similarly, blood and brain concentrations were

not affected in mice after hyperbaric exposure (25).

In order to determine the relevance of the present experiments with regard to behavioral studies on pressure reversal of ethanol intoxication one must consider whether colonic temperature is representative of brain temperature. A high correlation between rectal and thalamic temperature was found in guinea pigs at 50 bar in a heliox atmosphere (35). The thalamic temperature was slightly but consistently higher, and the difference increased during cold challenge. Under normobaric conditions, evidence that brain and deep body temperature may change with some degree of independence has been obtained from several species, including the rat (2, 6, 18). Recent experiments in mice have indicated that brain temperature may be lowered as a compensatory response to ethanol intoxication, but remains less affected than rectal temperature by ambient temperatures (4).

Thus some difference may occur between brain and rectal temperature and the latter appears to be more susceptible to thermal challenge, particularly after ethanol administration. Under the conditions of the present experiments, however, it seems unlikely that the overall temperature pattern would differ substantially between the brain and the colon.

Several stressors affect the core temperature of experimental animals. Introduction into a novel observation chamber or either the intermittent or continuous presence of a rectal temperature probe causes persistent hyperthermia in rats (29). Restraint reduces the ability for behavioral thermoregulation at both low and high temperatures in this species (3,11). At normal laboratory temperatures, restraint has been reported as lowering colonic temperature in mice and guinea pigs (21,35), but as elevating it in rats (21,36). Mild stressors, e.g., combined handling and rectal temperature recording, which induce hyperthermia in the absence of ethanol, may potentiate ethanol-induced hypothermia (27,37). In the present study, the animals that received saline showed an initial increase in colonic temperature compatible with the results cited above concerning the hyperthermic effects of handling, immobilization and rectal probing. There was no evidence for hypothermia after 1.5 g/kg ethanol, but it is possible that the moderate but long-lasting hypothermia observed after 3.5 g/kg ethanol at 1 bar was enhanced by the stress caused by restraint and temperature measurement.

On this basis, it seems possible that the difference in colonic temperature between saline- and ethanol-treated rats at 1 bar would have been less pronounced in unrestrained animals and in animals that had been habituated to the observation chamber. It is also possible that the fluctuations in colonic temperature associated with the increase in ambient temperature during compression would have been smaller in unrestrained rats. Since both restrained and freely moving animals are used in hyperbaric studies, further investigations comparing restrained and unrestrained animals are warranted.

In conclusion, high ambient pressure may counteract rather than potentiate the hypothermic effect of ethanol. The chamber temperatures employed supported normal colonic temperature in saline-treated rats and in animals which had received a moderate dose of ethanol and supported normal body temperatures even after a higher dose at high pressures. On this basis it is unlikely that alterations in body temperature contributed significantly to the pressure reversal of ethanol intoxication previously reported.

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REFERENCES

1. Alkana, R. L.; Malcolm, R. D. Antagonism of ethanol narcosis in mice by hyperbaric pressures of 4–8 atmospheres. Alcohol.: Clin. Exp. Res. 4:350–353; 1980.
2. Baker, M. A. Brain cooling in endotherms in heat and exercise. Annu. Rev. Physiol. 44:85–96; 1982.
3. Bartlett, R. G.; Bohr, V. C.; Helmendach, R. H.; Foster, G. L.; Miller, M. A. Evidence of an emotional factor in hypothermia produced by restraint. Am. J. Physiol. 179:343–346; 1954.
4. Bejanian, M.; Finn, D. A.; Syapin, P. J.; Alkana, R. L. Rectal and brain temperatures in ethanol intoxicated mice. Psychopharmacology (Berlin) 92:301–307; 1987.
5. Berge, O.-G.; Garcia-Cabrera, I. Changes in tail-skin temperature during ethanol-induced hypothermia in the rat; submitted.
6. Blumberg, M. S.; Mennella, J. A.; Moltz, H. Hypothalamic temperature and deep body temperature during copulation in the male rat. Physiol. Behav. 39:367–370; 1987.
7. Brauer, R. W.; Beaver, R. W.; Lahser, S.; Mansfield, W. M.; Sheehan, M. E. Time, rate, and temperature factors in the onset of high-pressure convulsions. J. Appl. Physiol. 43:173–182; 1977.
8. Eskuri, S. A.; Pozos, R. S. The effect of ethanol and temperature on calcium-dependent sensory neuron action potentials. Alcohol Drug Res. 7:153–162; 1987.
9. Ferko, A. P.; Bobyock, E. Physical dependence on ethanol: rate of ethanol clearance from the blood and effect of ethanol on body temperature in rats. Toxicol. Appl. Pharmacol. 46:235–248; 1978.
10. Finn, D. A.; Boone, D. C.; Alkana, R. L. Temperature dependence of ethanol depression in rats. Psychopharmacology (Berlin) 90:185–189; 1986.
11. Frankel, H. M. Effects of restraint on rats exposed to high temperature. J. Appl. Physiol. 14:997–999; 1959.
12. Freund, G. Hypothermia after acute ethanol and benzyl alcohol administration. Life Sci. 13:345–349; 1973.
13. Garcia-Cabrera, I.; Berge, O.-G. Pressure reversal of the depressant effect of ethanol on spontaneous behavior in rats. Pharmacol. Biochem. Behav. 29:133–141; 1988.
14. Garcia-Cabrera, I.; Berge, O.-G. Interaction of ethanol and the high pressure nervous syndrome in rats. Undersea Biomed. Res. 17:375–382; 1990.
15. Garcia-Cabrera, I.; Berge, O.-G. Interaction of high pressure and a narcotic dose of ethanol on spontaneous behavior in rats. Pharmacol. Biochem. Behav. 37:577–581; 1990.
16. Gordon, C. J.; Stead, A. G. Effect of alcohol on behavioral and autonomic thermoregulation in mice. Alcohol 3:339–343; 1986.
17. Halsey, M. J.; Wardley-Smith, B. Pressure reversal of narcosis produced by anaesthetics, narcotics and tranquillisers. Nature 257:811–813; 1975.
18. Hayward, J. N.; Baker, M. A. A comparative study of the role of the cerebral arterial blood in the regulation of brain temperature in five mammals. Brain Res. 16:417–440; 1969.
19. Johnson, F. H.; Brown, D. E. S.; Marsland, D. A. Pressure reversal of the action of certain narcotics. J. Cell. Comp. Physiol. 20: 269–276; 1942.
20. Johnson, F. H.; Flagler, E. A. Hydrostatic pressure reversal of narcosis in tadpoles. Science 112:91–92; 1950.
21. Jørgensen, H. A.; Fasmer, O. B.; Berge, O.-G.; Tveiten, L.; Hole, K. Immobilization-induced analgesia: Possible involvement of a non-opioid circulating substance. Pharmacol. Biochem. Behav. 20: 289–292; 1984.
22. Leon, H. A.; Cook, S. F. A mechanism by which helium increases metabolism in small mammals. Am. J. Physiol. 199:243–245; 1960.
23. Lomax, P.; Bajorek, J. G.; Chesarek, W. A.; Chaffee, R. R. J. Ethanol-induced hypothermia in the rat. Pharmacology 21:288–294; 1980.
24. Macdonald, A. G.; Marshall, N. R.; Pertwee, R. G. Behavioral thermoregulation in mice subjected to high pressure. J. Appl. Physiol. 66:238–244; 1989.

25. Malcolm, R. D.; Alkana, R. L. Hyperbaric ethanol antagonism: Role of temperature, blood and brain ethanol concentrations. Pharmacol. Biochem. Behav. 16:341–346; 1982.
26. Myers, R. D. Alcohol's effect on body temperature: Hypothermia, hyperthermia or poikilothermia? Brain Res. Bull. 7:209–220; 1981.
27. Peris, J.; Cunningham, C. L. Handling-induced enhancement of alcohol's acute physiological effects. Life Sci. 38:273–279; 1986.
28. Pohorecky, L. A.; Rizek, A. E. Biochemical and behavioral effects of acute ethanol in rats at different environmental temperatures. Psychopharmacology (Berlin) 72:205–209; 1981.
29. Poole, S.; Stephenson, J. D. Core temperature: Some shortcomings of rectal temperature measurements. Physiol. Behav. 18:203–205; 1977.
30. Romm, E.; Collins, A. C. Body temperature influences on ethanol elimination rate. Alcohol 4:189–198; 1987.
31. Sheehan, M. E.; Brauer, R. W. Temperature regulation and energy balance in mice under high pressures. In: Lomax, J.; Schönbaum, E.; Jacob, J., eds. Temperature regulation and drug action. Basel: Karger; 1975:367–382.
32. Spyropoulos, C. S. The effects of hydrostatic pressure upon the normal and narcotized nerve fiber. J. Gen. Physiol. 40:849–857; 1957.
33. Stetzner, L. C.; De Boer, B. Thermal balance in the rat during exposure to helium-oxygen from 1 to 41 atmospheres. Aerospace Med. 43:306–309; 1972.
34. Syapin, P. J.; Chen, J.; Finn, D. A.; Alkana, R. L. Antagonism of ethanol-induced depression of mouse locomotor activity by hyperbaric exposure. Life Sci. 43:2221–2229; 1988.
35. Unger, H.; Hempel, F. G.; Kaufmann, P. G. Correspondence of brain and rectal temperatures of guinea pigs in helium environments. Undersea Biomed. Res. 7:27–34; 1980.
36. Vidal, C.; Suaudeau, C.; Jacob, J. Regulation of body temperature and nociception induced by non-noxious stress in rat. Brain Res. 297:1–10; 1984.
37. York, J. L.; Regan, S. G. Conditioned and unconditioned influences on body temperature and ethanol hypothermia in laboratory rats. Pharmacol. Biochem. Behav. 17:119–124; 1982.