substrata. Although these ants are specifically attracted, under experimental conditions, to sterile urea solutions, the possibility that urea serves as a chemical signal for the ingestion of an organic base, bound to be rich in microorganisms and, hence, a potential source of nutrients, cannot be ruled out. Some species of *Camponotus* are known to be fungus-feeding and fungus-growing ants[10]. This particular species is not involved in fungus cultivation[11], so the possibility that urea is ingested as a source of nitrogen for the cultivation of fungus does not arise.

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Chronic exercise does not alter the chronotropic response of isolated rat atria to catecholamines[1]

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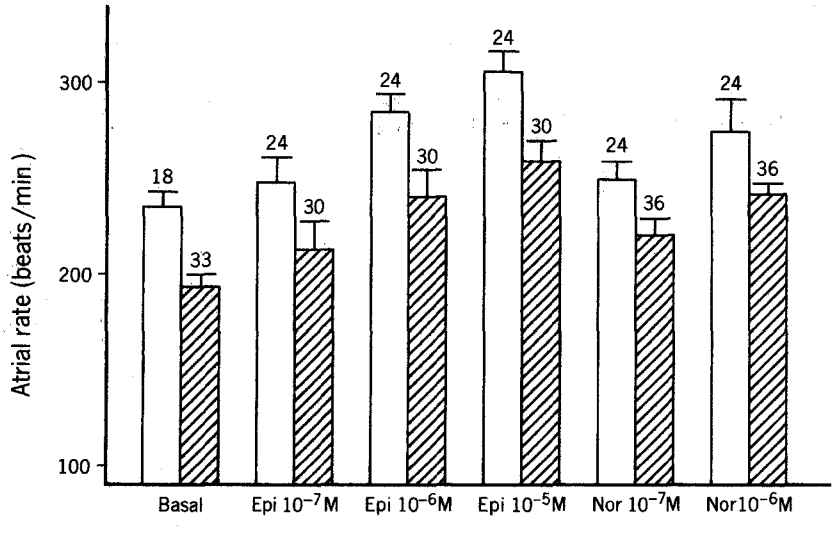
Summary. Atria isolated from rats after 7 weeks of exercise training beat at a slower rate than did atria from sedentary controls. There is no significant difference between the chronotropic responses of the 2 groups to epinephrine or norepinephrine.

The mechanism for exercise bradycardia has been suggested to be an increase in parasympathetic[2] or a decrease in sympathetic tone[3]. It has been shown that chronic exercise increases cardiac acetylcholine content[4] and increases the chronotropic response of isolated atria to atropine[5]. With regard to sympathetic effects, acute exercise has been reported to decrease cardiac catecholamine content[6] or to increase catecholamine synthesis without increasing content[7]. Leon[8] reported no change in heart norepinephrine levels after 3 months of chronic exercise. Raab et al.[9] consider that the non-exercised heart is under preponderant adrenergic control, while exercise exerts an 'anti-adrenergic' effect of the heart. Since this anti-adrenergic effect might mean a reduced chronotropic response to catecholamines, we have undertaken an evaluation of the sensitivity of isolated rat atria to catecholamines following chronic exercise.

Methods. Male Sprague-Dawley rats were assigned to control (sedentary) or experimental (exercised) groups. Experimental rats were run in a 6 compartment motor driven activity wheel twice daily for 1 h, 6 days per week for a total of 7 weeks. The rate of running was 12 m/min. Control rats were quartered with experimentals and were regularly placed in the activity cage, but were not exercised. The animals were sacrificed at the end of the training period and the hearts rapidly removed to a dish of Krebs-Hensleit solution (pH 7.2 ± 0.1). The atria were dissected free and placed in a bath of Krebs-Hensleit solution at 34 °C which was continuously oxygenated with 95% O_2, 5% CO_2. Atrial beats were recorded by means of a strain gauge with



Average rate of beating of isolated atria, basal, and in the presence of the indicated concentrations of epinephrine (Epi) or norepinephrine (Nor). White bars are sedentary controls; shaded bars are exercised rats. Extension bars represent SEM. Numbers over each bar show the number of rats in that group.

tension adjusted such that the atria were beating at the peak of the length-tension curve. The preparation was allowed to stabilize for a period of 0.5–1 h at which time the atrial rate was counted. The chronotropic response to catecholamines was tested by adding epinephrine (E) or norepinephrine (NE) to the bath to bring the bath content of E to the following concentrations: 10^{-7} M, 10^{-6} M, 10^{-5} M or of NE to these concentrations: 10^{-7} M, 10^{-6} M. The atrial rate was then counted after stabilization. Statistical analyses were done according to methods described by Schefler[10].

Results. The spontaneous rate of atria isolated from sedentary controls was 235 ± 7.8 beats/min (mean $\pm$ SEM). The rate from exercised animals was 194 ± 5.7 beats/min. The difference between sedentary and exercised was significant ($p < 0.001$).

The figure shows these basal rates as well as the responses of atria to the various concentrations of E and NE. Because atria from exercised animals beat at a significantly lower rate than atria from controls, we analyzed the data by means of a factorial analysis of variance. By this test, the response to E (with the exercise effect factored out) was significant ($p < 0.005$) at all concentrations except 10^{-7} M. The response to NE was significant at all concentrations. However, interaction effects between exercise and catecholamines were not significant at any concentration indicating that chronic exercise does not alter the chronotropic response to catecholamines. Nevertheless, trained atria always beat at a significantly ($p < 0.01$) lower rate than did untrained atria at all concentrations of E and NE. The rate of trained atria only exceeded the basal rate of control atria by a significant amount ($p < 0.05$) when in the presence of E in a concentration of 10^{-5} M.

Discussion. Our results corroborate those of Bolter et al.[11] which show that isolated atria from exercise-trained rats beat at a slower rate than atria from sedentary controls. The mechanism for exercise bradycardia therefore exists at least in part at the level of the atrium itself. This bradycardia could certainly be related to the increased amount of acetylcholine found in the exercised heart tissue. At least a part of the bradycardia effect might be explained in terms of altered sensitivity of the pacemaker to the autonomic neurotransmitters. However, our results indicate that atria from exercised animals are equally as sensitive to catecholamines as atria from sedentary rats. Whatever anti-adrenergic mechanism exists in these exercised hearts, it is not exerted by a reduction in chronotropic responsiveness to catecholamines. It is noteworthy, however, that the rate of trained atria was always less than untrained atria and that only with E in a concentration of 10^{-5} M did exercised atria beat at a faster rate than the basal rate of non-exercised controls. These results are consistant with an anti-adrenergic role of exercise as expressed by Raab et al.[9].

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Complete cold substitution of noradrenaline-induced thermogenesis in the Djungarian hamster, *Phodopus sungorus*[1]

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Summary. The thermogenic response to injections of noradrenaline at thermoneutrality was substituted by thermogenesis at low ambient temperatures. This demonstrates that noradrenaline-induced heat production is equivalent to physiologically induced nonshivering thermogenesis during cold exposure.

In small mammals, nonshivering thermogenesis (NST) is the dominating pathway for thermoregulatory heat production, and shivering thermogenesis is only used when heat production by NST is insufficient[2,3]. The most important site of NST appears to be brown adipose tissue (BAT)[4], where heat is liberated by oxidation of fatty acids following a stimulation of BAT cells by noradrenaline (NA) released from the sympathetic nerve endings of BAT[5,6]. This mechanism of NST induction is deduced from the fact that NST may also be induced artificially by injections of NA or it may be inhibited by injections of β-adrenergic inhibitors like propranolol[7,8]. The artificial stimulation of NST is most commonly used for quantitative determination of NST. Provided that an optimum dosage of NA is used, NST may be stimulated to its maximum in a curarized or deeply anesthetized mammal even in a warm environment. However this artificial stimulation always leaves the question whether the observed calorigenic response to NA is equivalent to the physiologically available NST in an unrestrained mammal during cold exposure.

If NA-induced NST corresponds to the cold-induced NST, than the calorigenic effects of exogenous NA and endogenous NA should be able to substitute for each other, i.e. if NA is injected during cold exposure the same NST maxima should be obtained as at thermoneutrality (for theoretical considerations see also Mejsnar and Jansky[9]). Differing from previous authors we want to use the term 'cold substitution' instead of 'temperature substitution' for a more precise description of the phenomenon. A complete cold substitution of the calorigenic response to exogenous