zen preservation of embryos also permits stockpiling of embryos for biochemical analysis. The present method of rapid freezing and thawing of rabbit embryos is less tedious than those reported hitherto, requires no specialized equipment, and permits higher survival rates. Thus it should be more useful in practice.

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Endurance training and antioxidants of lung[1]

A. Salminen, H. Kainulainen and V. Vihko

Division of Muscle Research, Department of Cell Biology, University of Jyväskylä, SF-40100 Jyväskylä 10 (Finland),
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Summary. Mice and rats were adjusted to daily treadmill training programs, which were heavy enough to increase the oxidative capacity of skeletal musles. Endurance training did not affect the activities of catalase and glutathione peroxidase and the concentration of vitamin E in the lungs of mice and rats. Thus increased ventilation and oxygen utilization induced by exercise training do not modify lung antioxidants, in contrast to hyperoxia and hypoxia.

Lung injuries appear in animals exposed to excessive concentrations of oxygen and other oxidants[2]. Hyperoxia increases H_2O_2 release by lung mitochondria and microsomes[3] and thus may cause peroxidative injuries. Lung tissue adapts to oxidant stress by increasing the level of antioxidants such as vitamin E, or the activities of catalase, glutathione peroxidase and superoxide dismutase[4,5]. On the other hand, exposure to hypoxia affects the antioxidant capacity of lung and causes structural changes and a tolerance to hyperoxia[6].

Some studies using expired pentane as the measure of lipid peroxidation have shown that physical exercise increases pentane expiration[7,8]. The real origin of this lipid peroxidation product has not been demonstrated. Physical exercise increases pulmonary ventilation and O_2 utilisation. Some studies also suggest that increased physical activity may induce structural changes in the lung tissue[9]. Hence, we studied whether endurance training modulates the level of some lung antioxidants.

Materials and methods. Male NMRI-mice, aged 4 months, were trained on a motor-driven treadmill (6° uphill tracks) once a day for 1 h at a speed of 25 m/min. A group of mice were killed the next day after 3, 10, and 20 training sessions. Male Sprague-Dawley rats, aged 8–9 weeks at the beginning of the study, were trained on a motor-driven treadmill with horizontal tracks. The daily running time and speed were gradually increased so that after 4 weeks' training the rats run for 1 h/day during 5 days a week. The running speed of each session was increased from 20 to 36 m/min for the last 30 min of the session. The total duration of the protocol was 6 weeks. The effect of the termination of regular endurance training (detraining) was also studied in this experiment. The first group of rats was killed the day after the last training session, the second 2 weeks after the cessation of training and the third group 5 weeks after the withdrawal of training.

The lungs were removed, washed in homogenization buffer (0.1 M potassium phosphate, pH 7.4), and trimmed free of connective tissues and major vessels. The activities of catalase and glutathione peroxidase and the concentration of vitamin E were assayed as described earlier[10] after modifying the reaction conditions optimal for lung tissue. Hydrogen peroxide was used as the substrate for both enzymes. The significance of the difference in the means between groups were tested by Student's t-test.

Results and discussion. Endurance training (commencement, prolonging, or termination) did not affect the activities of catalase and glutathione peroxidase and the concentration of vitamin E in the lungs of mice (table 1) and rats (table 2). The training programs were heavy enough to induce statistically

Table 1. Endurance training and the level of some antioxidants in mouse lung

Variable	Control	Endurance training (days)		
		3	10	20
	(n = 9)	(n = 9)	(n = 8)	(n = 8)
Catalase	13.9 ± 0.5	14.3 ± 0.6	15.0 ± 0.9	14.2 ± 0.6
Glutathione peroxidase	14.6 ± 0.5	15.1 ± 0.2	14.8 ± 0.7	14.7 ± 0.6
Vitamin E	19.9 ± 1.4	18.6 ± 1.1	21.5 ± 0.9	20.0 ± 2.0

Values are means ± SE. Catalase activity is expressed as arbitrary units x g^{-1} wet weight. Glutathione peroxidase activity is given as μmol x min^{-1} x g^{-1} wet weight. Vitamin E concentration is given as μg/g wet weight.

significant changes in the energy metabolic capacity of skeletal muscles (data not shown). The increase of citrate synthase activity, for instance, varied between 20–30% in different muscles.

Several studies suggest that in highly oxygenated tissues, such as heart and lung, vitamin E is the major cellular antioxidant[11]. In the microsomes of lung and heart the ratio of vitamin E to peroxidizable polyunsaturated fatty acids is several-fold higher than in microsomes of other tissues[11] and therefore the microsomes of these tissues are more resistant to lipid peroxidation. During inhalation of oxidants (nitrogen dioxide) vitamin E is assimilated by the lungs[5]. We observed no increase in lung vitamin E content in association with exercise-induced hyperventilation. The concentration of vitamin E was 47.8% higher in rat than in mouse lungs suggesting that the lungs of rat are more resistant to oxidant stress.

The activity of glutathione peroxidase was assayed using hydrogen peroxide as the substrate. This activity corresponds to the selenium dependent enzyme activity[12]. In lungs there exists also non-selenium dependent glutathione peroxidase activity. This activity consists 14.3% of the total activity in rat lung and 59.3% in mouse lung[12].

Table 2. Endurance training, detraining and the level of some antioxidants in rat lung

Variable	Control (n = 8)	Trained (n = 6)	Detraining 14 days (n = 5)	35 days (n = 4)
Catalase	8.31 ± 0.22	8.27 ± 0.13	7.68 ± 0.20	7.46 ± 0.27
Glutathione peroxidase	22.4 ± 1.0	20.3 ± 0.8	18.9 ± 1.2	23.0 ± 0.5
Vitamin E	29.4 ± 1.0	28.5 ± 1.3	28.7 ± 1.1	32.1 ± 1.3

Legends are as in table 1.

In oxidant stresses the activity of Se-dependent glutathione peroxidase has been increased, as has also that of catalase[4,13]. The changes are age-related, being greater in neonatal than in adult animals[4]. The activities of catalase and superoxide dismutase (not assayed) are low in the lung tissue compared to many other tissues of rats[13], and hence their role in the total protection may be slight. The lack of adaptive changes in lung antioxidants of exercised animals suggests that the pentane production during exercise[7,8] does not originate in the lungs.

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Somatostatin and 3-oxy-methyl-D-glucose (3-OMG) uptake in isolated chicken intestinal epithelial cells

M. Abalde, C. Taboada and P. Fernández

Departamento de Fisiologia Animal, Facultad de Farmacia, Universidad de Santiago, Santiago de Compostela (Spain), 7 July 1983

Summary. The direct effect of somatostatin on the absorption of 3-oxymethylglucose in epithelial cells isolated from the small intestine of chicken was studied. The presence of somatostatin in the incubation medium at concentrations of 3.5×10^{-8} M and 7×10^{-8} M produced significant dose-dependent increases in the accumulation of sugar in the enterocytes. This effect might be due to an increase in the cell membrane permeability caused by hormone action.

Unger et al.[1] have proposed the hypothesis that somatostatin plays an important physiological role in the homeostasis of nutrients by controlling the entry of ingested substances into the blood stream. Also, Wahren and Feling[2] observed that the administration of somatostatin to diabetics after the oral administration of glucose reduces the rise in the blood glucose level without affecting i.v. glucose tolerance, which suggests the action of somatostatin in the small intestine.

However, whether SRIF (somatotropin release inhibiting factor or somatostatin) acts directly on the absorption of nutrients by modifying the transport process in the wall of the digestive tract[3] or whether it acts indirectly by altering the splanchnic blood flow[2,4], intestinal motility or gastric emptying[5], is still open to question. For this reason the aim of the present work is to study the direct effect of somatostatin on the uptake of 3-oxy-methyl-D-glucose by isolated chicken intestinal epithelial cells.

Methods. Intestinal epithelial cells were isolated from 3–6-week-old male broiler chickens by the method reported by Kimmich[6]. The cells were separated from the medium by centrifugation. Somatostatin-14 was purchased from Sigma Chemical Co. and was added directly to 3 ml of the incubation medium containing 1 ml of the cell suspension, in concentrations of 3.5×10^{-8} M and 7×10^{-8} M. The standard incubation medium contained 80 mM NaCl, 100 mM mannitol, 20 mM Tris-Cl (pH 7.4), 3 mM K_2HPO_4, 1 mM $MgCl_2$, 0.1 mM EGTA, 2.5 mM $CaCl_2$, and 1 mg/ml of BSA.

For the determination of sugar accumulation, 3-O-methyl-D-glucose 2 mM was added to the cellular suspension together with 0.5 μCi of 2-O-methyl-D-(U-^{14}C)glucose (sp. act.: 295 mCi/mmol)[7] purchased from Radiochemical Centre, Amersham. Samples were extraced at intervals of 1, 2, 4, 6, 8 and 10 min. Incubation was not extended beyond 10 min to avoid the possible degradation of the hormone and the loss of this effect.