740 SPLANCHNIC BLOOD FLOW RESPONSES TO NICOTINE INFUSION AT REST AND DURING TREADMILL RUNNING IN MINISWINE.

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Nicotine administration or dynamic exercise increases resistance and decreases blood flow to the splanchnic circulation. In both cases, sympathetic vasconstriction is the primary mechanism. We hypothesized that nicotine administration during treadmill running exaggerates exercise-induced increases in resistance and decreases in blood flow to the splanchnic circulation. Ten miniswine (32±2 kg) were aseptically instrumented with catheters in the descending aorta and left atrium. Recording electrodes were sewn to the epicardium. Two weeks later, three protocols were performed. *Protocol 1.* Mean arterial pressure (MAP), heart rate (HR), and blood flow (Q̇) in the stomach (STO), proximal colon (COL), small intestine (INT), and pancreas (PAN) were determined at rest, and after 2 min of nicotine infusion (INF; 20 µg/kg/min) into the left atrium. Q̇ was measured using radioactive microspheres. *Protocol 2.* The same variables were determined before (PRE-X), and during 20-25 min of treadmill exercise (EX) at workloads sufficient to elicit 80% of each animal's maximal HR reserve. *Protocol 3.* After 48 hours, miniswine repeated identical workloads while nicotine was infused during the last 2 min of exercise (EX + NIC).

	MAP (mmHg)	HR (b/min)	STO (ml/g/min)	COL (ml/g/min)	INT (ml/g/min)	PAN (ml/g/min)
1. REST	90±3	112±6	41±6	77±10	160±14	173±10
INF	103±3*	106±6	29±7*	54±7*	97±14*	129±15*
2. PRE-X	85±2	112±3	44±5	70±8	148±10	148±12
EX	95±1*	226±3*	14±2*	33±2*	57±6*	47±6*
3. PRE-X	89±1	108±3	45±5	72±9	146±9	150±12
4. EX+NIC	100±2*†	220±3*	13±2*	34±3*	48±5*	42±5*

Means±SEM. *p<0.05 REST or PRE-X *vs* INF, EX, or EX + NIC; †EX *vs* EX+NIC.

These results suggest that nicotine does not enhance exercise-induced sympathetic vasoconstriction in the splanchnic vasculature.

741 EFFECTS OF A SINGLE BOUT OF EXHAUSTIVE EXERCISE ON INTEGRATED BAROREFLEX FUNCTION FOLLOWING BEDREST.

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Altered baroreflex function is a frequently observed consequence of bedrest. Baroreflex function is enhanced in the 24 h following performance of exhaustive, dynamic exercise. Therefore, we tested the hypothesis that one bout of maximal exercise performed 24 h prior to re-ambulation from 16 days of 6° head-down bedrest (HDT) could increase the gain of integrated baroreflex function. To do this, heart rate (HR) and blood pressure (MAP) were continually measured during a 15-sec Valsalva maneuver (VM) performed by seven subjects at a controlled expiratory pressure of 30 mmHg before and after two periods of HDT separated by 11 months. On the last day of one HDT period, subjects performed a single bout of maximal cycle ergometry (exercise). Subjects did not exercise following the other HDT period (control). There were no differences in baseline HR ($r = 0.841$, $P = 0.012$) or MAP ($r = 0.864$, $P = 0.018$) between bedrest exposures. The ratio of the change in HR to change in MAP (ΔHR/ΔMAP) during phases II and IV of the VM was used as an index of cardiac baroreflex sensitivity. Sixteen days of HDT decreased ΔHR/ΔMAP during phase II by 79% (9.6 to 2.0 beats/mmHg; $P = 0.0001$). After completion of exhaustive leg exercise, ΔHR/ΔMAP in phase II returned toward pre-bedrest levels (6.4 beats/mmHg), while it remained substantially attenuated in control (1.7 beats/mmHg). There were no differences ($P > 0.05$) in ΔHR/ΔSBP during phase IV at any time. These observations suggest that global baroreflex function, which is diminished by bedrest, is enhanced in the 24 h period following completion of a single bout of maximal exercise.

742 INSTANTANEOUS HEART RATE RESPONSES TO ACTIVE AND PASSIVE CYCLING

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Shortening of the R-R interval (RRI) in response to volitional and passively induced unloaded cycling was investigated in 15 healthy volunteers (27.3 ±1.2 yrs; 178±2 cm; 81.8 ±4.1 Kg). It was hypothesized that the volitional or central command component occurring with the onset of active cycling (AC) would cause a greater shortening of the RRI than would the onset of passive cycling (PC) when central command was presumed to be absent. Subjects performed 20-30 bouts of AC in which they initiated their own movement (rest to ~60 rpm) following a breath hold at functional residual capacity, while (20-30) bouts of PC were accomplished using a tandem bicycle with a second rider. Exercise onset was recorded using an electrical triggering device to detect pedal movement; ECG and leg EMG activity were sampled continuously (1000 Hz). RRI shortening in response to pedal movement was taken as the ratio of the RRI in which movement occurred as compared to the average resting RRI (6 to 8 beats prior to pedal movement). Onset of AC produced a significantly greater reduction for the RRI in which exercise began than did PC (-4.2 ±0.3% vs -0.4 ±0.2%; $P<0.01$). Likewise, AC caused a -10.2 ±0.3% reduction in the subsequent RRI as opposed to a -2.5 ±0.2% decrease elicited by PC. Onset RRI responses during control tests in which the pedal was triggered but no movement occurred were similar to PC (-1.0 ±0.3%, $P>0.05$). These findings suggest that central command (i.e. volitional initiation) has a predominant effect on the initial heart rate response to dynamic exercise.

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743 ANTIOXIDANT ENZYME ACTIVITIES IN RAT SKELETAL MUSCLES: EFFECTS OF ATROPHY, VITAMIN E, & EXERCISE

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The purpose of this study was to determine whether tail-cast atrophy (7 d), vitamin E (vit E) supplementation (20 mg/kg/d), and three bouts of acute exercise (2 km/h, 15% grade, 30 min for 3 d after atrophy) alters the antioxidant enzymes (U/g total protein), superoxide dismutase (SOD), glutathione reductase (GRd), and glutathione peroxidase (GPx) of soleus (SO), plantaris (PL), red gastrocnemius (RG), and white gastrocnemius (WG). Male Sprague-Dawley rats (80; 175-225g) were assigned as follows: group 1) atrophy-vit E-exercise, 2) atrophy-vit E, 3) atrophy-exercise, 4) vit E-exercise, 5) atrophy, 6) vit E, 7) exercise, and 8) control (each n=10). Independent of atrophy and exercise, in all 4 muscles Vit E had no effect on GRd and GPx, but showed significantly higher ($p < 0.01$) SOD activity than without vit E. Independent of atrophy, exercise with vit E group had higher ($p<0.01$) SOD activity than exercise without vit E group. There was no significant difference between vit E non-exercise group and without vit E and non-exercise group in SOD activity. GPx activity was higher ($p < 0.01$) in exercise group compared with non-exercise group in SO and PL muscle. GRd activity was also significantly higher ($p < 0.05$) in exercise group vs. that of non-exercise group in PL, RG, and WG muscle. No muscle showed a significant response to exercise in SOD activity . Atrophy elicited significant increases ($p < 0.01$) only in GRd activity in RG. None of the antioxidant enzymes and muscles were effected by atrophy. It is concluded that both vit E and exercise together promote increases in all skeletal muscle antioxidant enzyme activities to protect tissue against oxidative stress. Atrophy may not cause much oxidative stress as demonstrated by unchanged antioxidant enzymes in most skeletal muscles.

744 N-ACETYLCYSTEINE ATTENUATES EXERCISE ASSOCIATED BLOOD GLUTATHIONE OXIDATION

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Oxidation of blood glutathione (GSH) does not only diminish GSH dependent direct antioxidant protection but also may impair the replenishment of oxidized antioxidants, ascorbyl and tocopheroxyl radicals. High concentration of oxidized glutathione (GSSG) is also cytotoxic. We studied the effect of oral N-acetylcysteine (NAC) on exercise-associated rapid blood GSH oxidation in nine healthy young men who performed two identical maximal bicycle ergometer exercises (mean duration 14 [SEM 0.84] minutes) three weeks apart. Before the second exercise test, the men took NAC tablets (200 mg x 4 /day) for two days, and an additional 800 mg in the test morning. Blood samples were drawn before, immediately and 24 hours after tests. Total glutathione (TGSH) and GSSG were determined from acidified blood extracts. Thiobarbituric acid reactive substances (TBARS) and net peroxyl-radical scavenging ability (PSA; phycoerythrin-fluorescence based assay) were determined from the plasma. NAC supplementation resulted in an increase in pre-exercise PSA (mean 816 [SEM 106] µmol/l vs. 1108 [70]; $p<.05$). The control maximal test resulted in a significant increase (91 [6] µM to 181 [33]; $p<.001$) in blood GSSG. The rapid decline in blood thiol redox status was markedly attenuated (104 [13] µM to 113 [13]) by NAC supplementation. Plasma TBARS did not change (1.26 [0.19] µM to 1.72 [0.19] vs. 1.03 [0.09] to 1.42 [0.23]) following either of the tests. These results reveal that oral NAC elevates circulatory antioxidative capacity. NAC supplementation also spares exercise-associated blood GSH oxidation and the thiol redox status perturbation.

745 EFFECT OF EXERCISE TRAINING ON ANTIOXIDANT SYSTEM IN BRAIN REGIONS OF RAT.

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The brain is more susceptible to lipid peroxidation damage because it contains an appreciable amount of unsaturated fat and it uses 20 percent of oxygen consumed. Antioxidant enzymes and glutathione protect the cell against the oxidative stress and lipid peroxidation. Because of the potential hazards of free radical generation during exercise, this investigation was undertaken in order to determine the status of GSH and GSSG and the magnitude of change in antioxidant enzymes in brain regions in response to exercise training in various brain regions. Sprague-Dawley rats were exercise trained on a treadmill for 7.5 weeks, and sacrificed by decapitation 18 hr. after the last exercise. Different brain regions corpus striatum (CS), cerebral cortex (CC), brainstem (BS) and hippocampus (H) were removed and GSH and disulphide of glutathione (GSSG) were determined by HPLC; superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GSH-Px) activity was measured by spectrophotometry. The exercise training increased SOD activity significantly (130% of sedentary control) in brain stem as well as in corpus striatum. SOD activity slightly increased in hippocampus, however, this region contained the lowest activity as compared to other brain regions. There was not change in SOD activity in cerebral cortex due to exercise training. Different brain regions showed GSH-Px activity in decreasing order for CS < BS < CC < H. GSH levels were 43% less in brain stem than cortex and striatum. GSH/GSSG ratio significantly increased from 6.8 to 8.3 in cortex; from 9.4 to 13.5 in brain stem due to exercise training. Different brain regions contained different activities of antioxidant enzymes, as well as GSH and GSSG levels, which were preferentially altered due to exercise training to cope with oxidative stress.