654 ALTERATION OF LYMPHOCYTE SUBSETS AND ENDOCRINE RESPONSE DURING 42 DAYS OF COMPETITIVE SWIM TRAINING.

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Seventeen male subjects ($\bar{x}$ age=19.2$\pm$ 1.2 yrs) were tested before (Pre) and within 5 minutes of completion (Post) of a typical swim workout at the beginning of the season and after 42 days of training. Training consisted of both swimming ($\bar{x}$=49,000 yds in 9 workouts/wk) and progressive weights ($\bar{x}$=2 sets/wk). Cortisol (Cort) and Growth Hormone (GH) were assayed by RIA. Thyroid Stimulating Hormone (TSH) and Prolactin (Pro) were assayed by EIA. Lymphocyte (LY) subsets were evaluated with two-color monoclonal antibodies for T4-T8, T3-I3, T11-B1, NKH1-T8, 2H4-T4 (Helper Inducer) and 4B4-T4 (Suppressor Inducer). All samples were assayed by the same technologists on the same instruments. A significant decrease in Pro and in T4, NK, 2H4-T4, 4B4-T4 and Helper/Suppressor Ratio (H/S) ($p<.002$) when expressed in absolute terms occurred between the two Pre sample times. Significant decreases in H/S, Cort, and Pro with an increase in GH ($p<.0004$) occurred in both Pre to Post samples. Responses of LY, T8, NK, and TSH differed significantly between the two Pre to Post samples ($p<.0001$). These data indicate that both chronic training and acute exercise can induce significant alterations in LY subsets as well as some pituitary hormones.

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655 EFFECT OF ACUTE EXERCISE ON LYMPHOCYTE MITOGENIC RESPONSES IN CONDITIONED AND NON-CONDITIONED MALE SUBJECTS

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Ten conditioned and ten non-conditioned male subjects were evaluated for immune reactivity before and after an acute exercise regime. Subjects were divided into conditioned catagories based on exercise schedule, body composition and resting heart rate. The procedure was explained to each volunteer and informed consent was obtained from each participant. Subjects were instructed to refrain from exercising 24 hrs before testing. At the beginning of each session resting heart rate and body composition were determined. Blood was then drawn immediately before initiating the exercise routine (T1). Each individual was instructed to exercise on a bicycle ergomter at a rate or tension to bring his heart rate to 75% of maximal. Once achieved, the clock was started and he was instructed to continue at this pace for the 15 min by continuous heart rate monitoring. Blood samples were drawn immediately after the 15 min session (T2) and both samples were evaluated. The results from this study show that lymphocyte function as determined by response to mitogenic stimulation was lower at T1 in the conditioned group vs the non-conditioned group. In addition, a significant depression in lymphocyte reponsiveness to the mitogens PHA, Con A and PWM was observed at T2 vs T1 in the conditioned group. No significant effects of acute exercise were observed in the non-conditioned group. The results from this study indicate that lymphocytes from conditioned subjects are less responsive to stimulation and are suppressed after an acute bout of exercise compared to non-conditioned controls. These results suggest a role of exercise in the susceptibility of athletes to infection.

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656 INFLUENCE OF HEAVY TRAINING ON IMMUNE RESPONSE TO ACUTE EXERCISE IN ELITE RUNNERS

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Ten elite runners (age= 29.8$\pm$1.7yrs; VO2max= 65.3$\pm$4.9ml/kg/min.; personal best 10km. time= 31:43$\pm$1:46min.) were evaluated before and after 3 weeks of heavy training. On average the heavy training constituted a 47% increase over baseline training load. Before and after the 3 weeks of heavy training each runner ran for 30 min. on a treadmill at 80% of their VO2max. Venous blood was sampled before and 5min. following the treadmill run. Mitogen induced proliferation of peripheral blood mononuclear cells was used as an assessment of immune function. The heavy training load did not alter VO2max. (pre $\bar{x}$=65.3, post $\bar{x}$=65.1). There was no difference in mitogen induced cell proliferation in resting bloods sampled before and after heavy training. Acute exercise (REST proliferation - 5 POST proliferation) did not suppress immune function before heavy training but the acute exercise challenge significantly ($p<.05$) suppressed immune function following the 3 weeks of heavy training.

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SECTION H-5, FREE COMMUNICATIONS

657 BONE MASS IN POSTMENOPAUSAL ATHLETES AND NON-ATHLETES

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Weight bearing activity is suggested to reduce bone loss in postmenopausal women, but swimming, a non-weight bearing activity is not. In this study Masters swimmers (Ss) and runners (Rs) were compared to non-athletes (NA) to determine if differences existed between groups in bone mass, measured by dual photometry at the proximal femur, sum of 5 skinfolds (SOS) and VO2max, measured by direct oxygen uptake during an incremental treadmill test (values are reported as means $\pm$SD, $p<.05$). The 23 women were healthy, 50-68 yr old, at least 6 months postmenopausal (verified by serum FSH and estradiol (E2) levels) and none had taken estrogen replacement therapy. All groups were similar in height, calcium intake, years postmenopausal, age at menarche, and socio-economic background. However, the Ss and NA had greater body mass (Ss=68.5$\pm$8.6 Rs=59.9$\pm$5.9 NA=68.0$\pm$12.7 kg) and SOS (Ss=97.2$\pm$14.4 Rs=67.4$\pm$26.8 NA=105.0$\pm$19.1 mm) and had lower VO2 max values (Ss=27.5$\pm$2.4 Rs=38.6$\pm$6.3 NA=23.8$\pm$4.0 $(ml \cdot kg^{-1} \cdot min^{-1})$ than the runners. However, both Ss and Rs had higher age-adjusted bone mass at the femoral neck than NA (Ss=0.94$\pm$.05 Rs=0.87$\pm$.04 NA=0.72$\pm$.05 g/cm^2). The Ss did not have lower bone mass values than the Rs which may be attributed to higher estradiol levels in the Ss compared to Rs (Ss=49.3$\pm$19.2 Rs=23.8$\pm$21.5 $pmol \cdot ml^{-1}$). Therefore, although swimming is not weight bearing, it seems that elevations in body mass or estradiol may preserve bone mass in Masters swimmers.

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658 COMPARISON OF ESTROGEN OR EXERCISE ON LUMBAR BONE MASS IN EARLY POSTMENOPAUSAL WOMEN

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Early postmenopause is associated with an accelerated rate of bone loss. We compared the effectiveness of hormone replacement therapy (HRT) or exercise training in maintaining lumbar bone mass in sedentary early postmenopausal women. The non-exercise group (n=8) was on HRT initiated at menopause, 2.4 $\pm$ 0.5 yr (mean $\pm$ SE) earlier. The exercise group (n=8) initiated a weight bearing exercise training program 3.0 $\pm$ 0.7 yr after menopause. Although menopausal age was similar (3.0 vs 2.4 yr), the exercise group was older than the HRT group (52.5 $\pm$ 1.3 vs 46.4 $\pm$ 1.2 yr, $p=0.005$). Both groups were maintained on 1500 mg calcium daily. $\dot{V}O_2max$ was similar initially (25.3 $\pm$ 1.3 vs 27.7 $\pm$ 1.4 ml/kg·min). After training the exercise group had a 20.5% increase in $\dot{V}O_2max$ ($p<0.001$). Initial lumbar bone mass was significantly lower in the exercise group than in the HRT group (1.061 $\pm$ 0.038 vs 1.228 $\pm$ 0.031 $g \cdot cm^{-2}$, $p=0.004$). After 15 months of exercise training or 19 months of continued HRT, there was no change in bone mass from baseline in the exercise or HRT groups (+0.8% vs 1.6%). An increase in bone mass was noted at 5 months with exercise (+3.4%, $p<0.05$), but was not maintained with continued training. These results support the benefit of HRT for women in early menopause. Exercise training was able to maintain, but not increase, bone mass when initiated in early postmenopause. Further research is needed to learn whether bone mass can be maintained if exercise is initiated at menopause.

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659 BONE DENSITY OF ELITE FEMALE ATHLETES WITH STRESS FRACTURES

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To investigate whether stress fractures occurring in elite female athletes are related to reductions in bone mass associated with exercised induced amenorrhea, we measured bone mass in 9 athletes with such fractures and compared them with 9 athletes without fractures, matched for age, weight, height and sport. Bone density was measured in three regions: upper limb (distal radius), axial skeleton (lumbar spine) and lower limb, weight-bearing (femoral neck) by photon absorptiometry. The age of menarche was significantly delayed in the fracture group (mean $\pm$ SD: 16.1 $\pm$ 0.4 vs 14.4 $\pm$ 1.5 years, $p<0.02$). Two girls were premenarchal and there was a trend to fewer menses per year in the stress fracture group. Lumbar spine bone mineral density (BMD) was significantly reduced in the fracture group (mean $\pm$ SD: 1.20 $\pm$ 0.10 vs 1.25 $\pm$ 0.1 g/cm^2, $p<0.01$). Femoral neck BMD was also reduced (1.07 $\pm$ 0.1 vs 1.15 $\pm$ 0.12) but this difference was not significant. There was no significant difference in BMD between groups in the radius. All athletes had lumbar spine and femoral neck BMD values within the 95% confidence limits for normal non-athletic women. Although extreme exercise was associated with menstrual disturbance and reductions in lumbar spine bone mass, these changes may not necessarily account for stress fractures in such patients since the fractures occurred predominantly in lower limb bones, at sites characterized by predominantly cortical bone.

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