Anabolic steroids increase exercise tolerance

Anabolic steroids are of course well-known in the sports and bodybuilding worlds as huge performance enhancers; specifically, they add muscle and strength, and cut fat mass. So how do they work? One way is by increasing exercise tolerance. The athlete who uses steroids can return to the gym or the field much more quickly than one who does not use them.

The influence of an anabolic androgenic steroid (AAS) on thymidine and amino acid uptake in rat hindlimb skeletal muscles during 14 days after a single exhaustive bout of weight lifting was determined. Adult male rats were divided randomly into Control or Steroid groups. Nandrolone decanoate was administered to the Steroid group 1 wk before the exercise bout. [3H]thymidine and [14C]leucine labeling were used to determine the serial changes in cellular mitotic activity, amino acid uptake, and myosin synthesis. Serum creatine kinase (CK) activity, used as a measure of muscle damage, increased 30 and 60 min after exercise in both groups. The total amount of weight lifted was higher, whereas CK levels were lower in Steroid than in Control rats. [3H]thymidine uptake peaked 2 days after exercise in both groups and was 90% higher in Control than in Steroid rats, reflecting a higher level of muscle damage. [14C]leucine uptake was approximately 80% higher at rest and recovered 33% faster postexercise in Steroid than in Control rats. In a separate group of rats, the in situ isometric mechanical properties of the plantaris muscle were determined. The only significant difference was a higher fatigue resistance in the Steroid compared with the Control group. Combined, these results indicate that AAS treatment 1) ameliorates CK efflux and the uptake of [3H]thymidine and enhances the rate of protein synthesis during recovery after a bout of weight lifting, all being consistent with there being less muscle damage, and 2) enhances in vivo work capacity and the in situ fatigue resistance of a primary plantarflexor muscle.

Bioavailable Testosterone and
A cross-sectional population-based study examined the association between endogenous sex hormones and depressed mood in community-dwelling older men. Participants included 856 men, ages 50–89 yr, who attended a clinic visit between 1984–87. Total and bioavailable testosterone, total and bioavailable estradiol, and dihydrotestosterone levels were measured by radioimmunoassay in an endocrinology research laboratory. Depressed mood was assessed with the Beck Depression Inventory (BDI). Levels of bioavailable testosterone and bioavailable estradiol decreased with age, but total testosterone, dihydrotestosterone, and total estradiol did not. BDI scores increased with age. Low bioavailable testosterone levels and high BDI scores were associated with weight loss and lack of physical activity, but not with cigarette smoking or alcohol intake. By linear regression or quartile analysis the BDI score was significantly and inversely associated with bioavailable testosterone (both P’s = 0.007), independent of age, weight change, and physical activity; similar associations were seen for dihydrotestosterone (P = 0.048 and P = 0.09, respectively). Bioavailable testosterone levels were 17% lower for the 25 men with categorically defined depression than levels observed in all other men (P = 0.01). Neither total nor bioavailable estradiol was associated with depressed mood. These results suggest that testosterone treatment might improve depressed mood in older men who have low levels of bioavailable testosterone. A clinical trial is necessary to test this hypothesis.

If you’re older and don’t want to be depressed, have high testosterone.

Androgenic-anabolic steroids (AAS) are synthetic derivatives of the male hormone testosterone. They can exert strong effects on the human body that may be beneficial for athletic performance. A
review of the literature revealed that most laboratory studies did not investigate the actual doses of AAS currently abused in the field. Therefore, those studies may not reflect the actual (adverse) effects of steroids. The available scientific literature describes that short-term administration of these drugs by athletes can increase strength and bodyweight. **Strength gains of about 5–20% of the initial strength and increments of 2–5kg bodyweight, that may be attributed to an increase of the lean body mass, have been observed. A reduction of fat mass does not seem to occur.** Although AAS administration may affect erythropoiesis and blood haemoglobin concentrations, no effect on endurance performance was observed. Little data about the effects of AAS on metabolic responses during exercise training and recovery are available and, therefore, do not allow firm conclusions.

The main untoward effects of short- and long-term AAS abuse that male athletes most often self-report are an increase in sexual drive, the occurrence of acne vulgaris, increased body hair and increment of aggressive behaviour. AAS administration will disturb the regular endogenous production of testosterone and gonadotrophins that may persist for months after drug withdrawal. Cardiovascular risk factors may undergo deleterious alterations, including elevation of blood pressure and depression of serum high-density lipoprotein (HDL)-, HDL2- and HDL3-cholesterol levels. In echocardiographic studies in male athletes, AAS did not seem to affect cardiac structure and function, although in animal studies these drugs have been observed to exert hazardous effects on heart structure and function. **In studies of athletes, AAS were not found to damage the liver.** Psyche and behaviour seem to be strongly affected by AAS. Generally, AAS seem to induce increments of aggression and hostility. Mood disturbances (e.g. depression, [hypo-]mania, psychotic features) are likely to be dose and drug dependent. AAS dependence or withdrawal effects (such as depression) seem to occur only in a small number of AAS users. Dissatisfaction with the body and low self-esteem may lead to the so-called ‘reverse anorexia syndrome’ that predisposes to the start of AAS use. Many other adverse effects have been associated with AAS misuse, including disturbance of endocrine and immune function, alterations of sebaceous system and skin, changes of haemostatic system and urogenital tract. One has to keep in mind that the scientific data may underestimate the actual untoward effects because of the relatively low doses administered in those studies, since they do not approximate doses used by illicit steroid users. The mechanism of action of AAS may differ between compounds because of variations in the steroid molecule and affinity to androgen receptors. Several pathways of action have been recognised. The enzyme 5-α-reductase seems to play an important role by converting AAS into dihydrotestosterone (androstanolone) that acts in the cell nucleus of target organs, such as male accessory glands, skin and prostate. Other mechanisms comprises mediation by the enzyme aromatase that converts AAS in female sex hormones (estradiol and estrone), antagonistic action to estrogens and a competitive
antagonism to the glucocorticoid receptors. Furthermore, AAS stimulate erythropoietin synthesis and red cell production as well as bone formation but counteract bone breakdown. The effects on the cardiovascular system are proposed to be mediated by the occurrence of AAS-induced atherosclerosis (due to unfavourable influence on serum lipids and lipoproteins), thrombosis, vasospasm or direct injury to vessel walls, or may be ascribed to a combination of the different mechanisms. AAS-induced increment of muscle tissue can be attributed to hypertrophy and the formation of new muscle fibres, in which key roles are played by satellite cell number and ultrastructure, androgen receptors and myonuclei.

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**Metabolic adaptation to high-intensity exercise**

This thesis investigated the acute and chronic responses of human skeletal muscle to high-intensity exercise, with a particular focus on markers of mitochondrial content, and the potential for nutrition to manipulate the adaptive response in recreationally active individuals. The acute response was primarily assessed via measurement of signalling proteins and mRNA species linked to exercise-induced mitochondrial biogenesis. The chronic response was determined via changes in the protein content or maximal activities of mitochondrial enzymes after training. Study 1 examined whether the manner in which a given amount of high-intensity cycling work was performed (i.e., in an intermittent or continuous fashion) altered the acute metabolic response to exercise, and whether the acute response was indicative of longer-term adaptations. **Despite the similar acute activation of signalling proteins after the intermittent and continuous matched-work exercise protocols, 6 wk of training with the continuous protocol did not increase mitochondrial content, contrary to what we have previously shown after 6 wk training with the intermittent protocol. This suggests that the intermittent application of a low-volume, high-intensity stimulus is important to elicit training-induced increases in mitochondrial content.** Furthermore, Study 1 showed that acute changes in specific signalling proteins did not necessarily predict chronic adaptations. Studies 2 and 3 examined whether specific nutritional interventions, previously shown to modulate acute exercise capacity or metabolic response, altered the mitochondrial...
adaptive response to several weeks of HIT. Neither manipulating carbohydrate availability between twice daily training sessions, or chronic ingestion of β-alanine, augmented skeletal muscle adaptations in response to 2-6 wk of HIT. It is possible that small influences of nutrition were overwhelmed by the potency of HIT, which stimulated marked increases in mitochondrial content in this population. Overall this thesis advances our basic understanding of the skeletal muscle adaptive response to HIT and the influence of nutrition.

So, endurance training caused NO increase in mitochondrial biogenesis, while a work-matched high intensity interval protocol did. The lesson seems to be that to obtain all the benefits of exercise, workouts must be of sufficient intensity. If they don’t leave you gasping for breath, they are not.

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**Resistance to type 2 diabetes mellitus: a matter of hormesis?**

Type 2 diabetes mellitus is characterized by subclinical systemic inflammation and impaired regulation of blood glucose levels. Interestingly, impairment of glycemic control occurs despite substantial insulin secretion early in the course of this disease. Dysfunction of several organs (including pancreatic islets, liver, skeletal muscle, adipose tissue, gut, hypothalamus and the immune system) has been implicated in the pathogenesis of type 2 diabetes mellitus. However, diabetes-promoting lifestyle factors do not inevitably cause disease in all persons exposed. Hence, defense mechanisms must exist that can keep the detrimental influence of these risk factors at bay. Hormesis describes the phenomenon that exposure to a mild stressor confers resistance to subsequent, otherwise harmful, conditions of increased stress. This Review discusses the emerging concept that the effectiveness of an adaptive (hormetic) response to detrimental lifestyle factors determines the extent of protection from progression to type 2 diabetes mellitus. Further analysis of these protective hormetic responses at the molecular level should help to identify novel targets for preventive or therapeutic intervention in patients at risk of developing type 2 diabetes mellitus or those with overt disease.
The Free Radical Theory of Aging Revisited: The Cell Signaling Disruption Theory of Aging

Significance: The free radical theory of aging has provided a theoretical framework for an enormous amount of work leading to significant advances in our understanding of aging. Up to the turn of the century, the theory received abundant support from observations coming from fields as far apart as comparative physiology or molecular biology. Recent Advances: Work from many laboratories supports the theory, for instance showing that overexpression of antioxidant enzymes results in increases in life-span. But other labs have shown that in some cases, there is an increased oxidative stress and increased longevity. The discovery that free radicals can not only cause molecular damage to cells, but also serve as signals; led to the proposal that they act as modulators of physiological processes. For instance, reactive oxygen species (ROS) stimulate physiological adaptations to physical exercise. Critical Issues: A critical blow to the free radical theory of aging came from epidemiological studies showing that antioxidant supplementation did not lower the incidence of many age-associated diseases but, in some cases, increased the risk of death. Moreover, recent molecular evidence has shown that increasing generation of ROS, in some cases, increases longevity. Future Directions: Gerontologists interested in free radical biology are at a crossroads and clearly new insights are required to clarify the role of ROS in the process of aging. The hurdles are, no doubt, very high, but the intellectual and practical promise of these studies is of such magnitude that we feel that all efforts will be generously rewarding.

Influence of exercise intensity on
**systemic oxidative stress and antioxidant capacity**

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The aim of the current study was to examine the influence of exercise intensity on systemic oxidative stress (OS) and endogenous antioxidant capacity. Non-smoking, sedentary healthy adult males (n = 14) participated in two exercise sessions using an electronically braked cycle ergometer. The first session consisted of a graded exercise test to determine maximal power output and oxygen consumption (VO2max). One week later, participants undertook 5-min cycling bouts at 40%, 55%, 70%, 85% and 100% of VO2max, with passive 12-min rest between stages. Measures of systemic OS reactive oxygen metabolites (dROM), biological antioxidant potential (BAP), heart rate (HR), VO2, blood lactate and rating of perceived exertion were assessed at rest and immediately following each exercise stage. Significant (P<0·05) differences between exercise bouts were examined via repeated measures ANOVA and post hoc pairwise comparisons with Bonferroni correction. Increasing exercise intensity significantly augmented HR (P<0·001), VO2 (P<0·001), blood lactate (P<0·001) and perceived exertion (P<0·001) with no significant effect on dROM levels compared with resting values. In contrast, increasing exercise intensity resulted in significantly (P<0·01) greater BAP at 70% (2427 ± 106), 85% (2625 ± 121) and 100% (2651 ± 92) of VO2max compared with resting levels (2105 ± 57 μmol Fe2+ /L). The current results indicate that brief, moderate-to-high-intensity exercise significantly elevates endogenous antioxidant defences, possibly to counteract increased levels of exercise-induced reactive oxygen species. Regular moderate-to-high-intensity exercise may protect against chronic OS associated diseases via activation, and subsequent upregulation of the endogenous antioxidant defence system.

One thing I think this study shows is that exercise is a kind of hormesis, upregulating stress defense mechanisms.

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**Exercise is an antioxidant**

**Moderate exercise is an antioxidant: Upregulation of antioxidant genes by training**
Exercise causes oxidative stress only when exhaustive. Strenuous exercise causes oxidation of glutathione, release of cytosolic enzymes, and other signs of cell damage. However, there is increasing evidence that reactive oxygen species (ROS) not only are toxic but also play an important role in cell signaling and in the regulation of gene expression. Xanthine oxidase is involved in the generation of superoxide associated with exhaustive exercise. Allopurinol (an inhibitor of this enzyme) prevents muscle damage after exhaustive exercise, but also modifies cell signaling pathways associated with both moderate and exhaustive exercise in rats and humans. In gastrocnemius muscle from rats, exercise caused an activation of MAP kinases. This in turn activated the NF-κB pathway and consequently the expression of important enzymes associated with defense against ROS (superoxide dismutase) and adaptation to exercise (eNOS and iNOS). All these changes were abolished when ROS production was prevented by allopurinol. Thus ROS act as signals in exercise because decreasing their formation prevents activation of important signaling pathways that cause useful adaptations in cells. Because these signals result in an upregulation of powerful antioxidant enzymes, exercise itself can be considered an antioxidant. We have found that interfering with free radical metabolism with antioxidants may hamper useful adaptations to training.

Effect of supplementation with a cysteine donor on muscular performance

Oxidative stress contributes to muscular fatigue. GSH is the major intracellular antioxidant, the biosynthesis of which is dependent on cysteine availability. We hypothesized that supplementation with a whey-based cysteine donor [Immunocal (HMS90)] designed to augment intracellular GSH would enhance performance. Twenty healthy young adults (10 men, 10 women) were studied presupplementation and 3 mo postsupplementation with either Immunocal (20 g/day) or casein placebo. Muscular performance was assessed by whole leg isokinetic cycle testing, measuring peak power and 30-s work capacity. Lymphocyte GSH was used as a marker of tissue GSH. There were no baseline differences (age, ht, wt, %ideal wt, peak power, 30-s work capacity). Follow-up data on 18 subjects (9 Immunocal, 9 placebo) were analyzed. Both peak power [13 ± 3.5 (SE) %, P < 0.02] and 30-s work capacity (13 ± 3.7%, P < 0.03) increased significantly in the
Immunocal group, with no change (2 ± 9.0 and 1 ± 9.3%) in the placebo group. Lymphocyte GSH also increased significantly in the Immunocal group (35.5 ± 11.04%, \( P < 0.02 \)), with no change in the placebo group (−0.9 ± 9.6%). This is the first study to demonstrate that prolonged supplementation with a product designed to augment antioxidant defenses resulted in improved volitional performance.

Note that this was done with whey protein; also note that no exercise training was involved – the subjects developed more power from whey protein alone. N-acetylcysteine is also a cysteine donor and one may speculate that the results from that might be as good.

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**Supplemental Vitamin C Appears to Slow Racing Greyhounds**

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During strenuous exercise, markers of oxidation increase and antioxidant capacity decreases. Antioxidants such as vitamin C may combat this oxidation stress. The benefits of vitamin C to greyhounds undertaking intense sprint exercise has not been investigated. The objective of this experiment was to determine whether a large dose (1 g or 57 mmol) of ascorbic acid influences performance and oxidative stress in greyhounds. Five adult female, trained racing greyhounds were assigned to receive each of three treatments for 4 wk per treatment: 1) no supplemental ascorbate; 2) 1 g oral ascorbate daily, administered after racing; 3) 1 g oral ascorbate daily, administered 1 h before racing. Dogs raced 500 m twice weekly. At the end of each treatment period, blood was collected before and 5 min, 60 min and 24 h after racing. Plasma ascorbate, \( \alpha \)-tocopherol, thiobarbituric acid-reducing substances (TBARS) and Trolox equivalent antioxidant capacity (TEAC) concentrations were measured and adjusted to compensate for hemoconcentration after racing. TBARS, TEAC and \( \alpha \)-tocopherol concentrations were unaffected by supplemental vitamin C. Plasma ascorbic acid concentrations 60 min after racing were higher in dogs that received vitamin C before racing than in dogs that either received no vitamin C or received vitamin C after racing. The dogs ran, on average, 0.2 s slower when supplemented with 1 g of vitamin C, equivalent to a lead of 3 m at the finish of a 500-m race. Supplementation with vitamin C, therefore, appeared to slow racing greyhounds.
**Vitamin C may hamper exercise recovery**

Ascorbic acid supplementation does not attenuate post-exercise muscle soreness following muscle-damaging exercise but may delay the recovery process.

Exercise involving lengthening muscle actions, such as downhill running, results in delayed onset muscle soreness (DOMS), which may be attributable to reactive oxygen species (ROS). Although exercise causes oxidative stress, any link between ROS and DOMS remains speculative. There is emerging evidence to suggest that ROS play an important physiological role, assisting in the recovery process and protecting the cell from future damage; however, this has not been fully established. Despite this uncertainty as to the precise role of ROS, attempts to prevent post-exercise ROS production through antioxidant intervention are still common. The study investigated the effects of ascorbic acid supplementation on ROS production and DOMS following downhill running. Subjects were assigned to two groups. The ascorbic acid group (group AA) received 1 g ascorbic acid 2 h pre- and for 14 d post-downhill running, whilst the placebo group (Pl group) received a placebo. Blood samples were drawn pre-supplement, pre- and post-exercise, and then 1, 2, 3, 4, 7 and 14 d post-exercise for analysis of ascorbate, malonaldehyde and total glutathione. DOMS was assessed using a visual analogue scale and pressure algometry. Muscle function was assessed using isokinetic dynamometry. Plasma ascorbate was elevated throughout in group AA compared with the Pl group. Downhill running resulted in DOMS in both groups. Muscle function was impaired post-exercise in both groups, although a delayed recovery was noted in group AA. Malonaldehyde increased 4 d post-exercise in the Pl group only. Ascorbic acid supplementation attenuates ROS production following downhill running, without affecting DOMS. Furthermore, ascorbic acid supplementation may inhibit the recovery of muscle function.

**Effect of resistance training and whey on body composition**

The effect of whey isolate and resistance training on strength, body composition, and plasma glutamine.
Abstract
Different dietary proteins affect whole body protein anabolism and accretion and therefore, have the potential to influence results obtained from resistance training. This study examined the effects of supplementation with two proteins, hydrolyzed whey isolate (WI) and casein (C), on strength, body composition, and plasma glutamine levels during a 10 wk, supervised resistance training program. In a double-blind protocol, 13 male, recreational bodybuilders supplemented their normal diet with either WI or C (1.5 gm/kg body wt/d) for the duration of the program. Strength was assessed by 1-RM in three exercises (barbell bench press, squat, and cable pull-down). Body composition was assessed by dual energy X-ray absorptiometry. Plasma glutamine levels were determined by the enzymatic method with spectrophotometric detection. All assessments occurred in the week before and the week following 10 wk of training. Plasma glutamine levels did not change in either supplement group following the intervention. The WI group achieved a significantly greater gain (P < 0.01) in lean mass than the C group (5.0 +/- 0.3 vs. 0.8 +/- 0.4 kg for WI and C, respectively) and a significant (P < 0.05) change in fat mass (-1.5 +/- 0.5 kg) compared to the C group (+0.2 +/- 0.3 kg). The WI group also achieved significantly greater (P < 0.05) improvements in strength compared to the C group in each assessment of strength. When the strength changes were expressed relative to body weight, the WI group still achieved significantly greater (P < 0.05) improvements in strength compared to the C group.

Full paper here, well worth reading. The whey group gained 5 kg lean mass (!) and lost 1.5 kg fat. These were not novice bodybuilders either, which is usually the case when you see huge effects like this.

The atherogenic potential of dietary carbohydrate

The atherogenic potential of dietary carbohydrate.

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Source
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Abstract
OBJECTIVE:
To investigate the role of dietary carbohydrate in atherogenesis.
METHOD:
Search of the literature for relevant papers concerning the relationship of insulin/hyperinsulinemia and carbohydrate on the one hand, and the renin-angiotensin system, the sympathetic nervous system, growth factors, i.e. platelet-derived growth factor and insulin-like growth factor-I, C-reactive protein, and dyslipemia, on the other hand, factors well known to be involved in the atherogenic process, as well as for epidemiologic studies investigating the relationship between high-carbohydrate diets and the development of cardiovascular disease.

RESULTS:
High-carbohydrate nutrition is shown to have the ability to induce vascular inflammation and plaque formation through an insulin-mediated activation of the RAS, growth factors, cytokines, the SNS, and C-reactive protein and to cause an atherogenic lipid profile in normal humans. Epidemiologic studies as well as studies in experimental animals corroborate an important role of dietary carbohydrate in atherogenesis.

CONCLUSION:
High-carbohydrate diets, particularly in the form of high-glycemic index carbohydrate, have the ability to directly induce atherosclerosis. Based on anthropologic facts, the reason for these dietary-induced, insulin-mediated, atherogenic metabolic perturbations are suggested to be an insufficient adaptation to starch and sugars during human evolution. Restriction of insulinogenic food (starch and sugars) may help to prevent the development of atherosclerosis, one of the most common and costliest human diseases.

Testosterone, body composition and aging

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In addition to growth hormone (GH), sex hormones are important determinants of body composition. Aging is accompanied by a decrease in free testosterone levels and, as BMI as well as fat mass increase with age (with a redistribution of body fat), whereas muscle mass decreases, it is tempting to attribute a causal role to the decrease in androgen levels. In our study involving 372 males aged >20-85, age was found to be positively correlated with BMI and fat mass as measured by impedance, and negatively correlated with
levels of free testosterone and free insulin-like growth factor-I. Multiple regression analysis revealed that BMI and age were independent determinants of testosterone levels. The latter decreased from 598+/−188 (SD) ng/dl in the young controls to 453+/−161 ng/dl in the elderly group, free testosterone decreasing from 15.35+/−4.10 to 8.38+/−2.51 ng/dl. Fat-free mass decreased by 18.9%. In a subgroup of 57 men aged 70-80 years, testosterone levels correlated negatively with percentage body fat (r=−0.57), abdominal fat (r=−0.56) and plasma insulin levels (r=−0.40). As GH levels and pulsatility also decrease with age and as, moreover, androgens amplify endogenous secretion of GH, it is not easy to determine the relative role of androgen deficiency in the age-associated changes in body composition. Moreover, increase in fat mass (obesity), as occurs in aging males, is in itself associated with low levels of free testosterone and GH which both normalize after weight reduction. The role of testosterone in the age-associated changes in body composition is, however, further suggested by the increase in lean body mass and in mid-arm circumference and the decrease in waist-to-hip ratio observed after testosterone treatment of elderly men with decreased testosterone levels. Also in healthy eugonadal men, testosterone treatment, at least in supraphysiological doses, causes an important increase in fat-free mass (+/−10%) and in muscle size. The changes in muscle volume are associated with an increase in muscle fibre diameter, suggesting that testosterone induces muscle cell hypertrophy. In conclusion, aging in males is accompanied by an important increase in fat mass and a decrease in lean body mass. Several indices of body composition are significantly correlated with plasma testosterone levels before and after correction for BMI and age. It is evident, however, that in addition to testosterone levels, the age-associated somatopause is also a determinant of the changes in body composition.

**Effect of Testosterone Treatment on Body Composition and Muscle Strength in Men Over 65 Years of Age**

As men age, serum testosterone concentrations decrease, the percentage of body mass that is fat increases, the percentage of lean body mass decreases, and muscle strength decreases. Because these changes are similar to those that occur in hypogonadal men, we hypothesized that increasing the serum testosterone concentration of men over 65 yr of age to that in young men would decrease their fat mass, increase their lean mass, and increase their muscle strength.

We randomized 108 men over 65 yr of age to wear either a testosterone patch or a placebo patch in a double blind study for 36 months. We measured body composition by dual energy x-ray absorptiometry and muscle strength by dynamometer before and during treatment. Ninety-six men completed the entire 36-month protocol.
Fat mass decreased ($-3.0 \pm 0.5 \text{ kg}$) in the testosterone-treated men during the 36 months of treatment, which was significantly different ($P = 0.001$) from the decrease ($-0.7 \pm 0.5 \text{ kg}$) in the placebo-treated men. Lean mass increased ($1.9 \pm 0.3 \text{ kg}$) in the testosterone-treated men, which was significantly different ($P < 0.001$) from that ($0.2 \pm 0.2 \text{ kg}$) in the placebo-treated men. The decrease in fat mass in the testosterone-treated men was principally in the arms ($-0.7 \pm 0.1 \text{ kg}$; $P < 0.001$ compared to the placebo group) and legs ($-1.1 \pm 0.2 \text{ kg}$; $P < 0.001$), and the increase in lean mass was principally in the trunk ($1.9 \pm 0.3 \text{ kg}$; $P < 0.001$). The change in strength of knee extension and flexion at 60° and 180° angular velocity during treatment, however, was not significantly different between the two groups. **We conclude that increasing the serum testosterone concentrations of normal men over 65 yr of age to the midnormal range for young men decreased fat mass, principally in the arms and legs, and increased lean mass, principally in the trunk, but did not increase the strength of knee extension and flexion, as measured by dynamometer.**

One thing to keep in mind here is that T supplementation increased lean mass and decreased fat mass without exercise. Just imagine what it would do with some. One study I read stated that the increase in lean mass in T supplementation (again, without exercise) was on the order of 10%, although that wasn’t seen in the above study.
Exercise vs inactivity

These are MRIs, which speak for themselves. Link. Personally, I’m not planning on ever being sedentary, especially after seeing these.

Extremely short duration high intensity interval training
substantially improves insulin action in young healthy males

Extremely short duration high intensity interval training substantially improves insulin action in young healthy males

Background
Traditional high volume aerobic exercise training reduces cardiovascular and metabolic disease risk but involves a substantial time commitment. Extremely low volume high-intensity interval training (HIT) has recently been demonstrated to produce improvements to aerobic function, but it is unknown whether HIT has the capacity to improve insulin action and hence glycemic control.

Methods
Sixteen young men (age: 21 ± 2 y; BMI: 23.7 ± 3.1 kg·m-2; VO2peak: 48 ± 9 ml·kg-1·min-1) performed 2 weeks of supervised HIT comprising of a total of 15 min of exercise (6 sessions; 4–6 × 30-s cycle sprints per session). Aerobic performance (250-kJ self-paced cycling time trial), and glucose, insulin and NEFA responses to a 75-g oral glucose load (oral glucose tolerance test; OGTT) were determined before and after training.

Following 2 weeks of HIT, the area under the plasma glucose, insulin and NEFA concentration-time curves were all reduced (12%, 37%, 26% respectively, all P < 0.001). Fasting plasma insulin and glucose concentrations remained unchanged, but there was a tendency for reduced fasting plasma NEFA concentrations post-training (pre: 350 ± 36 v post: 290 ± 39 μmol·l-1, P = 0.058). Insulin sensitivity, as measured by the Cederholm index, was improved by 23% (P < 0.01), while aerobic cycling performance improved by ~6% (P < 0.01).

The efficacy of a high intensity exercise protocol, involving only ~250 kcal of work each week, to substantially improve insulin action in young sedentary subjects is remarkable. This novel time-efficient training paradigm can be used as a strategy to reduce metabolic risk factors in young and middle aged sedentary populations who otherwise would not adhere to time consuming traditional aerobic exercise regimes.

Check out the amount of exercise: those 15 minutes were not daily, but was the total amount over two weeks. The secret is that those were 15 high intensity minutes – with of course rest periods in between the 30 s all-out cycle bouts.