SEÇÃO ESPECIAL

"WHY EXERCISE ?"

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RESUMO

ASTRAND,P.-O. Por que fazer exercícios? Revista Brasileira de Ciência e Movimento, vol. 06, nº 03, pp 25-37, 1992.

Há pronunciada plasticidade e adaptabilidade nas propriedades funcionais e estruturais das células, tecidos e sistemas orgânicos do corpo humano quando expostos a estímulos diversos. Enquanto há concordância unânime de que é essencial haver exercícios regulares para funcionamento perfeito do corpo humano, há também, fatores extrínsecos evidentes, como dieta e hábitos de exercícios físicos, que se refletem nas estatísticas de doenças e mortalidade, em especial entre os idosos. O envelhecimento está associado obrigatoriamente à redução da energia máxima aeróbica e à redução da força muscular, ou seja, à redução da forma física. Como decorrência da diminuição da tolerância de exercícios físicos, um número crescente de idosos está vivendo abaixo, no, ou pouco acima dos patamares da habilidade física, bastando apenas uma ligeira doença para os tornar completamente dependentes. O treinamento físico pode produzir melhoras profundas e imediatas das funções essenciais para a forma física na velhice. A adaptação à atividade física regular ajuda a diminuir o rompimento do "milieu interieur" da célula e reduz muito o cansaço, aumentando, desse modo, o desempenho e a economia de produção energética durante o exercício.

UNITERMOS - Treinamento, Envelhecimento, Otimização de Funções, Fisiologia do Exercício.

ABSTRACT

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There is a pronounced plasticity and adaptability in the structural and/or functional properties of cells, tissues, and organ systems in the human body when exposed to various stimuli. While there is unanimous agreement that regular exercise is essential for optimal function of the human body, it is evident that extrinsic factors, such as diet and exercise habits, are reflected in the morbidity and mortality statistics, especially in the aged. Aging is obligatorily associated with reduced maximal aerobic power and reduced muscle strength, i.e., with reduced physical fitness. As a consequence of diminished exercise tolerance, a large and increasing number of elderly persons will be living below, at, or just above "thresholds" of physical ability, needing only a minor intercurrent illness to render them completely dependent. Physical training can readily produce a profound improvement of functions essential for physical fitness in old age. Adaptability to regular physical activity serves to cause less disruption of the cell's "milieu interieur" and minimizes fatigue, thereby enhancing performance and the economy of energy output during exercise.

UNITERMS - Exercise Training, Aging, Optimal Function Exercise Physiology.

An important question to ask is "Why has a subdiscipline titled 'exercise physiology' developed?" As a physiologist,

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O Comitê Científico gostaria de informa-los que devido a problemas técnicos a tradução em português do "Por que fazer exercícios"? será publicada no no próximo número 7(1), 1993.

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it is a pleasure to quote Starling: "The physiology of today is the medicine of tomorrow." Therefore, physiologists should be a basic science group that uses the most advanced technology to understand both the normal function of living systems and also the mechanisms of diseases. The study of the normal human individual provides an important baseline for the study of disease. There is a definite trend that new scientific frontiers like molecular biology, genetics, immunology, developmental biology, and neuroscience dominate both in research and in teaching medical students. It is, however, the physiologist's responsibility to convert the lifeless pieces of molecular and cellular biology into whole living systems. Morgan (34), former president of the American Physiological Society, emphasizes that:

> The research approach that is taken should not impact on the ability of the physiologist to teach a broad-based course in human physiology to medical students. Those physiologists who work at the systems level must be fully aware of advances at the cellular and molecular level, whereas those who work on molecular mechanisms must be able to integrate their data into a model of systemic function.

In my opinion, "exercise physiology" is from these viewpoints particularly important because an exercise situation in various environments provides a unique opportunity to study how different functions are regulated and integrated. In fact, most functions and structures are in one way or another affected by acute and chronic (i.e., in a training program) exercises. Therefore, exercise physiology is to a high degree an integrated science that has as its goal the identification of the mechanisms of overall bodily function and its regulation. It is regrettable that so few pages in standard textbooks of physiology for medical students are devoted to discussions of the effects of exercise on different functions and structures. Thay may explain why a majority of physicians do not recommend regular physical activity!

A review of some factors of key importance in exercise situations follows:

LIMITING FACTORS FOR MAXIMAL AEROBIC POWER

Symmorphosis. For an individual's aerobic performance the maximal oxygen uptake is decisive. However, in prolonged exercise the availability of substrates for the oxygen-consuming metabolism is another factor to consider, as well as the movements. During this century much research has been devoted to analyzing whether a particular step in the oxygen cascade from environmental air to the mitochondria in the skeletal muscle is the limiting factor, these include: pulmonary ventilation, pulmonary diffusion capacity, pulmonary ventilation, pulmonary diffusion capacity, cardiac output, oxygen diffusion from blood to the mitochondria, and the quantity of oxidizing enzymes in the mitochondria.

Weibel and Taylor (61) have presented a "common sense" hypothesis that, if animals are built reasonably, they should build and maintain just enough, but no more, structure than they need to meet functional requirements ("symmorphosis"). They noted that maximal oxygen uptake varied with the 0.8 power of body mass. Thus, a 30 g mouse consumed per unit body mass 6 times as much O_2 as a 300 kg cow. This difference in metabolic rate was matched by a similar difference im metabolic rate was matched by a similar difference in mitochondrial volume in skeletal muscles. However, larger animals have structural capacities of the pulmonary system that exceeds the demand for O_2 transport. The link, or symmorphosis, between lungs and muscles was not included in this early study (61). In 1987, Taylor and coworker (56) published reports in which "athletic" animals (dogs and ponies) were compared with sedentary animals of the same size (goats and calves). The ratio of maximal oxygen uptake to body mass for the more "athletic" animals, and their total mitochondrial volume, was about 2.5-fold higher than the corresponding values for goats and calves. The calculated maximal oxygen uptake per ml of mitochondria was therefore independent of the aerobic potential of the animal. They concluded that all four species were operating close to their upper limit for convective transport of oxygen in the circulatory system. In addition, they noted a 2.5-fold higher rate of O_2 transport with contribution from both cardiac output (stroke volume) and arteriovenous O_2 difference (the more aerobic species had a higher hemoglobin concentration) in the more "athletic" animals. These studies provide some support to the concept of symmorphosis in O_2 transport, owing to both structural and functional adaptions (30).

What is the situation in humans? At present there is general agreement that the central circulation of the human limits the maximal oxygen uptake during exercise in which large muscle groups are involved.

There is a high correlation between this maximum and maximal cardiac output times oxygen concentrations of the arterial blood ($C_{a_{O_2}}$). An acute increase in $CC_{a_{O_2}}$, e.g., by elevation of the hemoglobin concentration or by oxygen enrichment of the inspired air, will increase the maximal cardiac output and O_2 uptake (see refs.4, pp. 183-185; 9,30). Evidently the mitochondrial potential to consume oxygen normally exceeds the capacity of the circulation to provide oxygen. Saltin (45) has calculated that the potential of human skeletal muscle in terms of blood flow is 2.0-2.5 $1.kg^{-1}.min^{-1}$ under normoxic conditions. During maximal exercise the oxygen uptake is approximately 0.35 $1.kg^{-1}.min^{-1}$. A person with a total skeletal muscle mass of 30 kg (body mass 75 kg) would, with all muscles activated at maximal aerobic rate, require a blood flow of 60-75 $1.min^{-1}$ to the muscles alone. However, a realistic maximal cardiac output in this person would be 20-25 $1.min^{-1}$ including a blood flow to nonmuscular tissues of, say 4 $1.min^{-1}$. A similar calculation of the potential to take up oxygen by 30 kg of muscle mass would give some 10.5 $1.min^{-1}$ as compared with a "normal" maximal aerobic power of 3-4 $1.min^{-1}$ in an untrained to moderately trained young individual when running. How large a muscular mass with maximal blood flow can be activated to create a maximal demand on the cardiac output? Again, using Saltin's figures (45) and making allowance for a blood flow of 4 $1.min^{-1}$ to other tissues, the exercising muscle mass would be approximately 8 kg. With a larger muscle mass engaged vasoconstriction must occur in those 8 kg of muscle to allow for an

increased blood flow through the additional muscle mass activated. In fact the peak oxygen uptake in leg exercise (cycling) and combined arm and leg exercise (cranking plus cycling) is approximately the same. Actually with the arm exercise superimposed on the leg exercise, vasoconstriction occurs and blood flow in the leg muscles is reduced. Similarly, when during maximal one-legged exercise the other leg also starts exercising, there is a reduced blood flow in the "first" leg (the muscle mass involved in the one-legged exercise amounts to 7-8 kg in a man of body mass 75 kg (45). It should be emphasized that the heart weight in humans is only 0.3-0.4% of the body mass, 0.7-0.8% in the dog, and 0.8 - 1.0% in the horse (45).

In conclusion, the oxygen supply to one large muscle group of activated muscles is limited by vasoconstriction when another large group of muscles is exercising simultaneously. By this adaptation the arterial blood pressure can be maintained at an adequate level. With a large muscle mass activated in vigorous exercise the central circulation limits the whole body oxygen uptake. On the other hand, beyond a certain power output involving large muscle groups, the local peak oxygen uptake is limited by peripheral factors, i. e., by vasoconstriction. The muscle groups "steal" blood flow and oxygen supply from each other. So, when vascular conductance exceeds cardiac pumping capacity in the severe exercise, both muscle chemoreflexes and arterial baroreflexes must maintain blood pressure by vasoconstricting active muscle (44).

Another conclusion is that the sym - morphosis concept is not applicable to humans. It was mentioned that the maximal cardiac output did not match the 2.5-fold higher maximal oxygen uptake in "athletic" animals. Thanks to their higher Hb-concen tration, a widening of the a-$\bar{v}$ O_2 difference could support the cardiac output for an efficient O_2 transport. However, "athletic" endurance trained humans characterized by high maximal aerobic power have, if anything, a subnormal Hb-concentration (4,p. 452). The results from animal experiments cannot always be applied on humans.

Can maximal aerobic power be trained? It is not necessary to activate the total skeletal muscle mass in order to train central circulation. On the other hand, by engaging large muscle groups the perceived exertion at a given oxygen uptake and heart rate is modest as compared with an activity involving the requirement of small muscle groups. A training program including interval or continuous exercises at 50% (beginners) and up to some 80% of maximal aerobic power for 30-40 min three times per week can effectively increase stroke volume and, therefore, maximal cardiac output by about 15% or more, albeit with large individual variations (39,62). Actually, Bouchard et al. (8) report that in their studies 77% of the variance in the training response in maximal aerobic power seemed to be genotype - dependent. Their subjects were monozygotic twins. Those who with a given training program improved less than 5% were about 5% of a sedentary population. About 5% of the popu lation fell into the category that improved their maximal aerobic power by 60% or more. In extreme situations one can find large variations in maximal oxygen uptake: After 3 wk bed rest this maximum averaged 1.74 $1.min^{-1}$ in three subjects; then, after 50 d with intensive training, it had almost doubled to 3.41 $1.min^{-1}$ (4,p.442).

Aging. From cross-sectional studies it is concluded that there is a decline in maximal oxygen uptake amounting to 0.5 - 1.0% per year. However, as has been reported the degree of habitual physical activity and heredity can markedly affect this maximum (32).

LIMITING FACTORS FOR MAXIMAL ENDURANCE

Untrained persons with maximal oxygen uptake of 2.5 $1.min^{-1}$ may be able to exercise at 90% of their maximum for some 20 min. If after a period of training they have raised their maximal aerobic power to 3 $1.min^{-1}$, the submaximal rate of oxygen uptake of 2.25 $1.min^{-1}$ will only demand 75% of this new maximum. The trained person can tolerate this metabolic rate for some 90 min, i.e., there is a 4.5-fold improve ment of endurance that most likely is not limited by the oxygen transport system. It is more likely that emptied glycogen stores are the limiting factor. With a glycogen-

saving, training-induced, increased utilization of free fatty acids (FFA) as a substrate for activated skeletal muscles, the performance time at 75% of maximal aerobic power will be longer than when a similar relative rate of exercise was maintained before training.

Numerous studies have demonstrated a significant increase in mitochondrial density as a consequence of aerobic training with a proportionate increase in mitochondrial enzymes (7,20). If one expresses skeletal muscle's oxidative capacity for a sedentary individual in 1 "unit," the endurance trained elite athletes have a 3 "unit" capacity. In a rabbit muscle, 3-5 wk of chronic electric stimulation with a frequency of 10 Hz can bring the capacity up to 6 "units." In a leg subjected to encasement in plaster, the activity may drop to 0.7 "units" (20). From these data we can conclude that with aerobic training there is a shift in the trained skeletal muscle to greater reliance on oxidative metabolism to provide energy for ATP resynthesis.

An increase in capillary density redues the distance between blood and cell interior, which enhances the exchange rate of gases, substrates, and metabolites. The surface area available for this exchange also increases. With more capillaries in a given tissue volume, more blood can flow through the vascular bed per unit of time. The mean transit time (MTT) is increased, which allows a more complete exchange of material. The primary advantage of high capillary density is highly endurance trained muscle is probably that it allows for an adequate MTT at high flow rates, thereby promoting a more complete exchange of materials (45).

The physiological site of the action of the enzyme lipoprotein lipase (LPL) is in the luminar surfaces of the capillary epithelium. With an increased capillary bed, more binding sites for LPL will be available. This may favor the provision of FFA to the muscle, because the degradation of triglyceride-rich particles depends largely on the activity of LPL. In addition, the activity of LPL also transfers more surface material into high density lipoproteins (HDL). This mechanism may explain the elevated plasma HDL concentration noticed in endurance-trained individuals (15,26), and may be one factor of many behind the observation that habitual physical activity seems to reduce the risk for coronary heart disease.

LIMITATIONS IN MAXIMAL STRENGTH

With age there is a decrease in muscle strength that seems to parallel the reduction in muscle mass. This age-associated loss of muscle fibers is related to a loss of α-motoneurons (2). The force-generating capacity of residual contractile material is, however, unaltered. It should be noted that there are normally no signs of degenerated fibers in the aging muscle and the fiber composition is relatively constant, independent of age. However, the muscle fibers per motor unit have been reported to increase, which is interpreted as a reduction in motoneurons, partly compensated for by peripheral sprouting from "healthy" nerve terminals and thereby a reinnervation of muscle fibers that lost their original motoneurons. To what extent activation of the muscles can promote nerve fiber sprouting is unknown. In this context it is of interest to not that 24-month-old rats who had been submitted to strength training for 5 months had 14% more muscle fibers than nontraining controls (28).

When starting a strength-training program, a 20-40% increase in strength may occur during the first weeks, without a noticeable increase in the cross-sectional area of the muscles involved. This suggests a more efficient activation of the muscles. With continued strength training there is an adaptive hypertrophy exclusively achieved by an increase in fiber size without increase in the number of muscle fibers (31). It is remarkable that as little as a 6-s isometric contraction repeated five times, three times per week can prevent loss of muscle mass and muscle function during periods of recovery from injury with joint immobilization (5).

Fiatarone et al. (14) studied nine frail, institutionalized volunteers 90 yr old (range 87-96) who undertook 8 wk of high-intensity resistance training. They employed an adaptation of standard rehabilitation principles of progressive resistance training with concentric (lifting) and

eccentric (lowering) activation of knee extensor muscles. Three times per week the subjects performed three sets of eight repetitions with each leg in 6- to 9-s per repetition, with a 1-to 2-min rest period between sets. Except for the first week the load was 80% of the one repetition maximum. Strength gain averaged 174% ± 31% (mean ± SEM). Midthingh muscle area increased 9.0% ± 4.5%. Mean tandem gait speed improved 48% after training. These findings would suggest that age does not appear to affect the trainability of skeletal muscle.

SPECIFICITY OF TRAINING

Training adaptations, whether for strength or endurance, in the skeletal muscles are limited to the muscles actually engaged in the training performance. Specificity in training is also obvious with the circulatory response to exercise. Saltin (45) reports that the heart rate was approximately 25 beats.min^{-1} higher at a given oxygen uptake when one leg, which had been inactivated by a cast for 4-6 wk, was exercising on a cycle ergometer than when the other leg exercised at the same submaximal aerobic power.

To maintain or improve both flexibility in joints and dexterity and endurance in technically demanding skills, the specific activities must be practiced. It is generally thought that elderly person cannot execute coordinated, quick, precise finger movements. However, it is impressive that many musicians can peform perfectly at an advanced age. Arthur Rubinstein played very demanding Chopin compositions at the age of 88 yr. Andrés Segovia, at 92 yr of age, gave concerts on the classical guitar. Hours of daily "training" are behind these achievements. Most likely their fantastic performances were not only a question of talent but of continuous practice.

Since there is such a specificity in training effects, it is advisable that one undertakes variation in the types of habitual physical activity, i.e., a "smörgasbord" of activities should be practiced throughout life.

IS PHYSICAL INACTIVITY A RISK FACTOR FOR CARDIOVASCULAR DISEASES?

During 1987, coronary heart disease (CHD) accouted for 27.5% of the 2.1 million deaths in the United States (1). Well-documented risk factors for CHD, independent and in some cases interrelated, are sedentary lifestyle, increases in triglyceride and low density lipoprotein levels in the blood, decrease in the high density lipoprotein leval, cigarette smoking, hypertension, diabetes, obesity, post-prandial hyperinsulinemia, and carbohydrate intolerance. During the last decade, there have been several reports showing a significant reduction in morbidity and mortality in CHD in physically active persons compared with sedentary control groups (3,21,35,38, 40,41). Paffenbarger et al. (38) conclude:

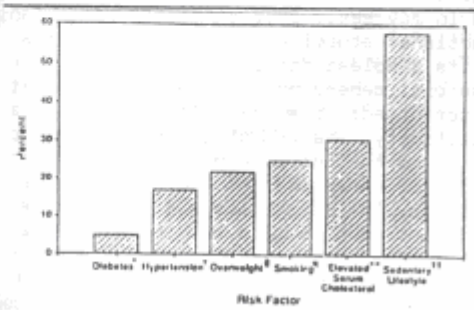
> Discounting the influence of blood pressure status, cigarette habit, net weight gain since college, and parental history of early death, the more active alumni students are estimated to have lived on average 1.25 yr longer than less active men.

The data were obtained in a longitudinal study of approximately 17,000 Harvard alumni. Men expending 8.4 + MJ (2000 + kcal) weekly in walking, stair - climbing, and sport play were at 39% lower risk of developing coronary heart desease than less active classmates.

Figure 1 is based on data from the 1988 Behavioral Risk Factor Surveillance System (BRFSS) and the 1976 - 1988 Second National Health and Nutrition Examination Survey (NHANESII) (1). In the BRFSS survey with 37 state health departments participating, sedentary lifestyle was the most prevalent (58%) modifiable risk factor for CHD reported and was followed by cigarette smoking (25%), obesity (22%), hypertension (17%), and diabetes (5%). The estimate for serum cholesterol levels (2g.1^{-1} or 5.2 mmol.1^{-1}) was 31%.

These studies indicate a substantial independent risk for CHD for persons with sedentary lifestyles. A high level of habitual physical activity will reduce premature mortality. Physical activity also confers other important health benefits. Habitual physical activity reduces the incidence of, or is otherwise beneficial to, hypertension, hyperlipidemia, obesity,

diabetes type II, impaired glucose tolerance, osteoporosis, psychologic impairment, colon cancer, stroke, and back injury (1). The effect on some of these factors may be small and statistically nonsignificant; however, the sum of these effects may have an important positive impact, influencing health and disease, life and death.



* Respondent self-reported having been told by physician that respondent has diabetes.

† Respondent self-reported having been told by a physician that blood pressure is high on multiple checks, or respondent is on medication for high blood pressure.

§ Based on bodly mass index 27.8 for men and 27.3 for women from self-reported height and weight of respondent.

¶ Respondent self-reported currently smoking.

** Measured 200mg/dl by NHANES II.

†† Respondent self-reported no physical activity or irregular physical activity (i.e., fewer than three times per week and/or 20 min. per session).

FIGURE 1 - Prevalence of modifiable risk factors for CHD - behavioral risk factor surveillance system, 1988, and second national health and nutritional evaluation survey, 1976-1980 (1).

The majority of studies of levels of physical activity and life expectancy available in 1988 appear to documenta a 1- to 1-yr increased life expectancy for men who are physically active in their lifestyle compared with inactive men (21).

In the United States life expectancy has increased from 47 yr in 1900 to approximately 75 yr in 1988. What about upper limits to human longevity? It has been calculated that eliminating ischemic heart disease would only increase life expectancy at birth by 3.0 yr for females and 3.5 yr for males. Eliminating all forms of cancer (22.5% of all deaths in 1985) would increase life expectancy by 7.0 yr for females and 8.1 yr for males (37). In other words, the effects of eliminating major diseases on life expectancy are not dramatic. Advances in medical treatment more than improvements in risk factors may be allowing elderly persons who are frail and who suffer from fatal degenerative diseases to survive longer after the onset of the disease than was the case in the past. Current research efforts, by the medical community are focused on prolonged life rather than preserving the quality of life. An obvious conclusion, therefore, is that the time has come for a shift toward ameliorating the function for people with nonfatal disease of aging (37).

PHYSICAL ACTIVITY AND CARBOHYDRATE METABOLISM

With reference to carbohydrate metabolism, there are recent hypotheses that glucose can permanently alter some proteins. In doing so, it may contribute to age-associated declines in the functioning of cells and tissues (10). An excess blood glucose concentration in people with uncontrolled diabetes could potentially contribute to diabetic complications. The authors point out that the effects of diabetes on many organs and tissues can be described as accelerated aging, including senile cataracts, joints stiffness, and atherosclerosis. These disorders are typical in the aging person, but in people with hyperglycemia they develop earlier. Without excess glucose, there may be a slower onset of age-associated dysfunction.

Sedentary people often have a reduced response to insulin in their untrained muscles. Elderly individuals may develop a reduced glucose tolerance and blood glucose concentrations is, at times, elevated and may trigger the mentioned glucose-protein reaction, and by a feedback also cause a hyperinsulinemia, in itself a risk factor for CHD.

A training program, as described above, can normalize the glucose tolerance in elderly people and enhance the muscle's

sensitivity to insulin (7,24,58). It should be emphasized that muscle contractions **per se** will promote the muscle's uptake even with no insulin present (58,60).

LOCOMOTOR ORGANS - OSTEOPOROSIS

Structural changes in bones, ligaments, and tendons are more easily demonstrated in connection with decreased rather than with increased physical activity (52,57). Immobilization will markedly diminish the number and size of collagen fiber bundles and negatively change the collagen cross-links. In contrast, trained animals have an increased concentration of collagen in ligaments and tendons (57). Inactivity will not only affect the strength of muscle, bone, and junctions, but also diminish the forces being transmitted by ligaments and tendons. Therefore, clinicians must constantly reevaluate the necessity and the duration of a prescribed immobilization.

Studies of astronauts and immobilized persons have consistently recorded osteoporosis. Bone involution also poses a serious health risk, especially for aging women. A progressive decline in bone density starts after maturity, and osteoporosis is a serious disease in the elderly. In the United States one-third of women, 65 yr old, will have vertebral fractures, and by age 81 one-third of women and one-sixth of men will have suffered a hip fracture, often a catastrophic, if not terminal, event (42).

Bone mass is subject to both local (mechanical) and systemic (hormonal) homeostatic control mechanisms. The local forces acting on bone tissue are due to gravity and muscular contraction (52). Standing upright is a better stimulus for bone hypertrophy than exercise in the bed, and walking is better than swimming in this respect.

Athletes have a greater bone mass than the sedentary population, with the greatest hypertrophy noted in the areas most stressed (e.g., the preferred arm of a tennis player). Exercise intervention also promotes bone hypertrophy and prevents osteoporosis. Both middle-aged and elderly women increase bone mass or reduce the rate of bone tissue loss in response to exercise intervention programs (11,12,46, 47,57). Rowe and Kahn (42) suggest that the marked reductions in bone density with "usual" aging may in large part be preventable or modifiable by regular exercise, reduced smoking, and an adequate intake of calcium.

In summary, prescribed exercises that increase the forces being transmitted to ligaments, tendon, and bones will maintain- and in may cases increase-the strength an functional sturdiness of these structures. In its simplest form, the effect of exercise will depend on the manner in which it is performed: the result displays a specificity. The only structures to respond are those that have been exposed to the forces of exercise. To counteract osteoporosis, the strategies are for adolescents and young adults to increase peak bone mass, and then adults and the elderly should focus on maintaining and/or slowing down the rate of bone loss (46).

NUTRITIONAL ASPECTS OF HABITUAL PHYSICAL ACTIVITY

The more physically active a person is, the higher the energy intake can be without risking obesity. From a nutritional viewpoint the advantage with such activity is that higher energy intake will better secure an adequate intake of essential nutrients (4,pp. 574-576; 13). From an evolutionary point of view, our energy expenditure and intake of essential nutrient were quite high. It may become critical for the low energy consumer-the sedentary person-to be nutritionally well-balanced, particularly if exposed to so-called "junk" food. At increase age there is gradual reduction in the basal metabolic rate, but no proportional reduction of the demand for essential nutrients. For this reason it is recommended that old people try to stay physically active. In treatment of obese patients it is essential to combine a recommendation of restrictions of energy intake with an increase in energy output by daily physical activity (33). After all, physical activity is the most variable component of energy expenditure.

PHYSICAL ACTIVITY AND THE TOXIC POTENTIALITIES OF OXYGEN

Aerobic metabolism involves production

of molecules that are intermediates in electrical charge between O_2 and H_2O. These intermediates, also named free oxygen radicals, include superoxide ion, hydroxyl radical, and derivates of these radicals like H_2O_2 and singlet oxygen. They can elicit lipid peroxidation, enzyme inactivation, denaturation of proteins, and structural changes in nucleic acids. A particular problem is apparently that, mediated by some sort of chain reactions, the oxidizing free radicals can damage membranes by lipid peroxidation. During evolution, living organisms had to develop adequate anti - oxidant defenses to combat the destructive properties of these oxygen radicals. Thus, aerobic cells, such as muscle fibers, have been equipped with enzymes and nonenzymatic scavenger systems as protection against these radicals. There are speculations that physically very active individuals would also be more exposed to the destructive tendency of oxygen. However, cytochrome oxidase, which is responsible for most of the biological oxygen consuption, produces water without the release of intermediates. In other words, the production of free oxygen radicals does not increase in proportion to the increase in oxygen uptake by the body. There are also reports concerning an adaptive increase in the biosynthesis of the scavenger superoxide dismutase with increasing metabolic rate (16, p. 263). There are reports that endurance training increased the resistance of skeletal muscles to injuries caused by lipid peroxidation. It may also be that damaged molecules are more efficiently replaced by resythesis (51).

From a nutritional viewpoint it should be pointed out that selenium is a cofactor for glutathione peroxidase, an effective scavenger of hydrogenperoxid (H_2O_2), and that vitamin E, C, and A can act as antioxidants. Thus although "oxygen possesses a good Dr. Jekyll - evil Mr. Hyde pesonality" (16, p. 386), the overall picture is the Dr. Jekyll aspect, that increased whole body metabolism and thereby oxygen consumption as elicited by regular physical activity are essential for optimal functioning of the body.

CARDIOVASCULAR RISKS OF EXERCISING

Vuori's (59) estimation of the incidence of sudden cardiovascular death in all recreational exercise and sports in the total Finnish male population is as follows: one death in 11.5 million exercise sessions in 20-to39-yr-old men, one death in 2.6 million sessions in 40-to 49- yr - old men, and one death in 2.2 million sessions in 50-to 69-yr-old men.

Other studies also suggest that sudden death during exercise is a rare event. On the other hand there is transient increased risk of sudden death during vigorous exercise compared with that during more sedentary activities. However, that risk should be compared with benefits of habitual exercise. Siscovick (50) reports that among habitually vigorous men, the overall risk of primary cardiac arrest calculated on a 24-h basis was only 40% that of sedentary men.

IMMUNOLOGIC EFFECTS OF EXERCISE

Simon (48) conclude that a variety of **in vitro** assays can be used to demonstrate that exercise affects most defense mechanisms including granulocyte counts, lymphocyte counts and functions, cytotoxicity, cytokine production, and secretory immunoglobulin levels. In general however, these changes are small in magnitude and brief in duration. The biological significance of these changes is uncertain.

SECRET TO LONGEVITY?

"If you slow down the matabolic rate, you reduce wear and tear on the system," and that means "the process of aging may be slowed" (quotation of Joseph Kemnitz, ref. 230. One can find popular statements pointing out that "you have a given number of heart beats allotted, don't waste them by exercising." However, after a few months of aerobic training you may save some 20,000 heart beats per day! In the long run a sedentary lifestyle may induce more "wear and tear on the system" than a physically active lifestyle.

SUMMARY OF EFFECTS OF HABITUAL PHYSICAL ACTIVITY AND POSSIBILITIES TO COUNTERACT CONSEQUENCES OF "NORMAL" AGING

Between 1985 and the end of this

century, we can expect an approximately 50% increase in the proportion of the population aged 65 and over in the "developed" countries. With aging there is an impairment in bodily functions and structures, diseases develop, and the endpoint is death. Rowe and Kahn (42) point out:

> Research in aging has emphasized average age-related losses and neglected the substantial heterogeneity of older persons. The effect of the aging process itself has been exaggerated, and the modifying effects of diet, exercise, personal habits, and psychosocial factors underestimated. Within the category of normal aging, a distinction can be made between usual aging, in which extrinsic factors heighten the effects of aging alone, and successful aging, in which extrinsic factors play a neutral or positive role. Research on the risks associated with usual aging and strategies to modify them should help elucidate how a transition from usual to successful aging can be facilitated.

So, the key question is to what extent impairment, morbidity, and mortality are inevitable consequences of the individual's innate genetic composition, i.e., intrinsic factors, and to what extent environment and the individual's lifestyle, i.e., extrinsic factors, can modify these processes?

It has been pointed out that aging is obligatorily associated with reduced maximal aerobic power and reduced muscle strength, i.e., with reduced physical fitness. Being overweight in addition to these handicaps is additionally unfortunate because these factors taken together make walking, climbing stairs, getting up from bed or chair, entering a bus or train more difficult and fatiguing, and eventually impossible. The ability to lift and carry weights becomes reduced. The aging persons will lose their independence and autonomy. As a consequence of diminished exercise tolerance, a large and increasing number of elderly persons will be living below, at, or just above "thresholds" of physical ability, needing only a minor intercurrent illness to render them completely dependent. Now we are back to the question of the respective places of intrinsic and extrinsic factors in the deteriorations typical for "usual" aging. Physical training can readily produce a profound improvement of functions essential for physical fitness in old age and thus effectively postpone physical deterioration for some 10-20 yr. It was mentioned that three are remarkably many musicians who can perform extremely well in their 80th-90th yr. Now consider how many old individuals continue or take up physically demanding activities ranging from walking and playing golf up to participation in endurance events like long-distance running (marathon!), orienteering, and cross-country skiing. We know very little about the relative contributions from intrinsic and extrinsic factors behind these achievements. Rowe and Kahn (42) argue that: "a direct-assistance treatment is typical of approaches to older people that do for them what they could do to learn to do for themselves."

TABLE 1 - Effects of habitual physical activities.

Increase in maximal oxygen uptake and cardiac output (4, chp. 10;6,45)
Reduced heart rate at given oxygen uptake (4, chap. 10;6,45,53).
Reduced blood pressure (18,49)
Reduced heart rate x blood presure product (18)
Improved efficiency of heart muscle (4, chap. 10,27)
Improved myocardial vascularization? (4, chap. 10)
Favorable trend in incidences of cardial morbidity and mortality (1,3,21,35,38,40,41)
Increased capillary density in skeletal muscle (7, 17,20)
Increased mitochondrial density in skeletal muscle (7,17,20)
Reduced lactate production at given percentage of maximal oxygen uptake (4, chap. 10,25)
Reduced perceived exertion at given oxygen uptake (2, chap. 10)
Enhanced ability to utilize free fatty acids as substrate during exercise - is glycogen saving (4, chap. 10 + 14; 17,20)
Improved endurance during exercise (4, chap. 10+14)
Increases metabolism - advantageous from a nutritional viewpoint (4, chap. 14;13,49)
Counteracts obesity (13,33,49)
Increases HDL concentrations in blood (15,19,26)
Improved structure and function of ligaments, tendons, and joints (52,57)
Increased muscular strength (4, chap. 10;5,14,29,36)
Increased production of endorphines (55)
Enhances nerve fiber sprouting to reinnervate muscle fibers? (54)
Enhances tolerance to hot environment - increased

sweat production (43)
Reduced platelet aggregation? (41)
Counteracts osteoporoses (11,12,46,47,52,57)
Can normalize glucose tolerance (24,55,58)

The body quickly will adapt to reduced demands on muscle activity, with an accelerated rate of functional impairment and physical fitness as consequences. Table 1 summarizes some effects of habitual physical activity.

EVOLUTIONARY ASPECT

Evolution of all living materials is based on heterogeneity in the individual's genetic pool within a species. Survival of the species is a measure of the success of evolution in adapting an organism to particular environmental conditions. Close to 100% of the existence of the **hominid** 3 homo species has been dominated by outdoor activities. Hunting and foraging for food and other necessities in the wilds have been a condition of human life for millions of years. Evolutionary changes in the genetic code that made survival of the hominids possible were promoted. We became adapted to a lifestyle as hunter - gatherers; this also applies to our emotional and social lives and to our intellectual skills. Major adaptations for this survival were consonant with habitual physical activity, including endurance and peak effort alternated with rest and socialization. Now, after a very brief spell in an agrarian culture, we have in some "privileged" societies ended up in an urbanized, highly technologic society dominated by a sedentary lifestyle (Fig. 2). One may conclude that the relationship between aging and various diseases - and physical fitness - has become very abnormal in our modern society. Over millions of years the old persons were very important and respected in society because they were credited with experience essential for the well-being and survival of the people. Therefore, the aging individual was still an important member of the group. More recently - but before our time - when the old man in an North American Indian tribe did not fully qualify as a scout, a hunter, or a warrior, he became the teacher of the boys in a "lecture hall" outdoors, with the lessons full of excitement.

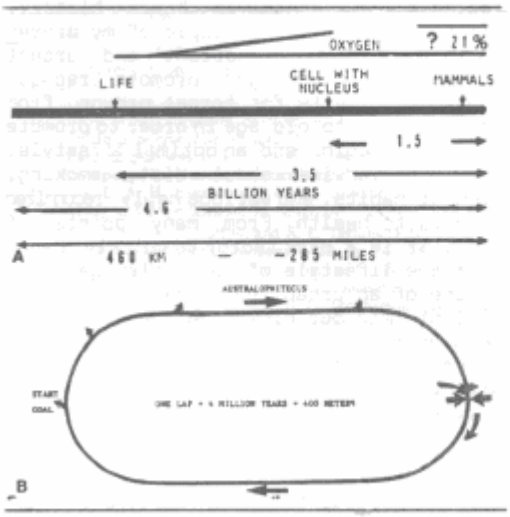


FIGURE 2 - A) Our planet is probably some 4.6 billion yr old. Let us translate those years into a 460 km journey. Biological evolution began some 1 billion yr later. Cells with nucleus functioning thanks to the ATP=ADP cycling, glycolysis, Krebs citric cycle, electron transport chain can be traced 1.5 billion yr ago-150 km from "now," mammals appeared on the scene after another almost 130 km.B). The hominid Australopithecus can be traced approximately four million yr back. Let us symbolize those 4 million yr by a 400 m track. He "gave up" after about 200 m. At this point, 2 million yr ago, true Homo individuals appeared on the scene (Homo habilis, later on Homo erectus). After 399 m - 10,000 yr ago-farming and agriculture started, probably in the Middle East. This country = the last 10 mm! Homo sapiens neanderthalianeis became extinct approximately 35,000 yr ago (3.5 m from "now"). Conclusion : during more than 99% of our existence, we were hunters and food gatherers. Now we are exposed to an enormous experiment-without control groups.

The old man entered a new niche in the society, still intellectually and physically demanding and of vital importance for the future of the tribe, and eventually:

"this is a good day to die!" (22). Without experience and scientific backups, we are facing a new phase in human history. Limiting myself to the topic of my presentation: it is an important and urgent challenge to teach and promote regular physical activity for target groups from childhood up to old age in order to promote successful aging and an optimal lifestyle, including advice about diet, smoking, alcohol habits, and various newly recognized threats to health. From many points of view, it is a risk factor to quickly change from the lifestyle of a hunter-gatherer to one of an urbanized high-technologist. Insight into our biological heritage may help us to modify our current lifestyle in a positive way. At any rate, adverse effects of noncompetitive exercises are very small in comparison with the health benefits. Cardiologists who do not believe that regular physical activity is a useful "medicine" in the primary and secondary prevention of cardiovascular disease are wise if they prescribe exercise for many of the other reasons. Health education is complicated; there are case reports of somenone with a lifestyle considered to be perfect who may die young from a heart attack. In contrast, a man with a "catastrophic" lifestyle may have a life span extending to one century. The genetic background is not democratic...

PRESCRIPTION FOR EXERCISE FOR HEALTH LIVING

With "support" from Table 1, the following recomendations represent an exercise program for good function and health.

Daily. At least 60 min of physical activity, not necessarily vigorous, not necessarily continuously. During the daily routine of moving, walking, climbing stair, etc., whether for 1 min 60 times a day, 12 min five times a day, or any combination totaling 60 min may demand 1.2 MJ (300 kcal).

Weekly. Exercises, say 30-45 min, three times per week (e.g., brisk walking, jogging, cycling, swimming, "aerobic dancing / gymnastics," rowing, skiing) are efficient efforts to achieve and maintain good cardiovascular fitness and may consume an additional 3 MJ (750 kcal) per week. The extra weekly energy output by these exercises will exceed 8 MJ (2000 kcal), an energy output recommended by Paffenbarger et al. (38) for better health.

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