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Review Paper

MECHANICAL FORCE AND BONES DENSITY

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Abstract. *The fact that physical activity has a positive influence on the skeleton has already become an axiom. Referring to this there is substantial evidence including the prolific documentation of the often devastating consequences of the imobilisation and the comparative data which point to the connection between increased physical activity and increased mass of the bones. Having already said that regular physical activity can have a beneficial effect on the bones one can pose a question as to what the specific parts of the programme which is going to be the most favourable for the bones are. Guidelines for the exercise can be defined according to the type of exercise, intensity, duration and frequency although it is impossible to give absolute recommendations. This is so because there are no grounds that indicate the optimum duration and frequency of the exercise for the skeleton. The subject of this paper is to define the mechanism of the influence of different physical activities on the bones status. The aim is to enhance the understanding of the forms of different physical activities and the way they influence the skeleton status.*

Key words: *mechanical force, bones density, strength training, adaptation*

INTRODUCTION

A specific build of bones represents a joint of hard inorganic and flexible organic components and is equally resistant to the forces of compression and extension as well. On the exertion of the force of compression a bone has a high limit of safety to endure the load and the impact. Muscle contraction during active movement exerts the biggest pressure on the bones and wrists. Compression forces that can cause the fracture are 2450 and 3000 kg/cm respectfully.

The thickness of the compact bone tends to be the biggest in the body i.e. in the middle part of the long bones where the torsion and bending forces are the biggest. In the

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areas of wrist end parties which are mainly exposed to the often big forces of compression a layout of the bone tissue differs from a layout in the body of the bone. A wrist part of the bone is completely built up of the sponge bone tissue covering the thin external layer of the compressed and compact bone. Most bones have this kind of bone tissue layout.

Long bones (ossa longa) have this layout as well while their middle part is adapted to the local mechanical needs. This layout is seen in the short bones (ossa brevia), too, for example in the hand, legs and spinal discs bones.

Bone mass is a quantity of bone tissue that skeleton contains. It is expressed as a mineral content of the bones (MCB), as a total quantity of bone minerals in grams within a certain surface of the bone or as a bone mineral density (BMD) or the quantity of minerals in bones in cm^2 of the measured surface (i.e. g/cm^2).

Maximum bone mass (MBM) is defined as the highest reached level of the bone mass due to the normal growth. It is usually reached in youth before the involutive (age related) loss of bones.

MBM of a person is conditioned by a host of factors including heritage (considered the most important one), muscle power, physical activity, plasma level of sex hormones and nutrition. It is estimated that MBM is reached at the earliest in 17–18 and in 35 at the latest. Quantity of the skeleton bone minerals in adults is a difference between the maximum quantity of bone minerals obtained during the growth of the skeleton and maturity (i.e. from the childhood till the middle 30 ties) and the quantity lost later on. Hence bigger MBM provides bigger depots of the bone mass during life time. Therefore primary protection against osteoporosis is achieved through the biggest possible MBM.

A low bone mineral density can be caused by a low MBM or by very sudden bone mass loss after the completion of the maximum mass. Factors influencing the bone density can be roughly classified into hereditary and environmental factors. Although heritage obviously influences the maximum bone mass its role in the later bone loss is not defined.

Habits as smoking and alcohol consumption are connected to the degree of the bone loss.

Those interwoven relations make difficult attempts to separate the influence of heritage from the influence of the environment on the bone loss.

Review of the previous research

Having in mind that the central problem of this research is the influence of mechanical force (different physical activities) on the bone status (bone mass and density) we are going to review the findings of the previous research directly or indirectly connected to this topic.

One of the key factors of the sound bone maintenance are probably muscle forces exerted on the bones. A view that exercising has beneficial effects on the skeleton is supported in the immobilization consequences, increased bone mineral density in sportsmen and the results of exercise. Recent research studies show that "active sportsmen" or recreational ones have significantly bigger bone mineral density in comparison to those who do not do any physical activities and that difference ranges from 8-30% no matter what activity is in question.

Physical activity can play a vital role in the development of bone mass during childhood and adolescence as well as in skeleton mass maintenance in young persons. This conclusion is based partly on research in which young sportsmen have bigger bone density compared to their non-active peers, then on research in which sportsmen have different bone density in relation to the sport they play and finally on the fact that the bone mass increase in students is connected to the higher level of physical activity.

Beside this hypothetic function during youth physical activity plays a vital role in normal structure and functional bone hardness maintenance during the whole life. Long staying in bed or immobility can cause sudden and significant bone density decrease.

Bone is a dynamic tissue which constantly renews (remodels) its structure through the resorption and formation processes. Physical activity by effects of loading of the skeleton is an important factor affecting bone density and its architecture. Bone cells react on mechanical loads by making balance between formation and resorption of bones which results in bone mass increase. (Lanyon, 1987). The greater the load the bigger a bone mass. Inversely when the skeleton is not under load (as in non-active state) bone mass is decreased. Having in mind that muscle exerts the biggest force on the bone during physical activity the role of muscle mass and force on the skeleton integrity should be researched in more detail.

Strenuous physical activity of the active sportsmen causes bone mass increase. One research shows that exercising in the period of growth can increase the thickness of he cortical bone by 25-30%. Research on the influence of physical activity has given controversial results ranging from the loss of bones up to the increased mineral volume.

Whether mild physical activity can influence the bone mass in adults is to be seen. However, all agree that insufficient physical activity can cause the increase in bone mass loss. It was suggested that there should be "minimum efficient effort" which is the lowest effort needed to maintain the balance in bone changes so that the relative values of the bones could be saved (Frost, 1986). Yet, size is just one of the factors contributing to the functional load as a stimulus for the changes in bones. Three factors modifying the bone are: load quantity, frequency of load and the distribution of load (Lanyon, 1987).

Immobilization and a diminished load (on the skeleton) stimulate bone resorption and cause a sudden bone loss. Healthy persons oriented towards frequent staying in bed and bed-ridden patients in intervals of from two to nine weeks show a significant bone mass loss. During space flights bone loss in astronauts is 3% on average.

Courteix et al. (1998) claim that physical activity has an anabolic effect on the bone tissue. The authors have concluded that physical activity increases bone mineral density in pre-puberty children as well as in teenagers. But evidence on the influence of intensive physical activity in childhood and in pre-puberty period is still obscure. In order to investigate the influence of the intensive training in that period on bone mineral density selected group of girls (in pre-puberty period) has been tested in the initial phase of their maximum bone mass. Subjects were playing either high impact loading sports on the skeleton or non impact loading sports. There was no significant statistical difference between the groups in relation to age, body mass and height and body composition. In all BMD measurements there was no significant statistical difference between the swimmers and the control group, whereas the middle BMD in female gymnasts was statistically bigger than in the control group.

Up to now controversial findings were reported on the favorable influence of swimming on bone mass (BM) because this activity does not have load impact on the skeleton.

Increase in physical activity contributes to the mineralization of bones in adults. However, in teenagers sport that can slow down growth can do the same with the mineralization of bones while sports with normal calories intake favor mineralization.

In order to evaluate the effects of the physical activity intensity and the kind of sport on mineralization of bones pre and after the puberty (Burrows et al., 1996) have investigated the sample of 144 pupils of both sexes, aged 7 to 14 attending different kinds of physical activities. Better values of BMD of the whole body and the spine were found in pupils with increased physical activity. Female gymnasts in pre-puberty have had smaller BMF of the whole body. Puberty pupils with insignificant physical activities have had smaller BMD. The authors have concluded that physical activity benefits mineralization of bones in vertebra and hips especially in puberty.

To follow the adaptation of bones to the load effort intensity might be more important than number of repetition. In order to validate this hypothesis (Bennell et al, 1977) have conducted 12month longitudinal research comparing bone mass and metabolism in top and average athletes and the control non-sport group. The group consisted of 50 power athletes (sprinters, jumpers, hurdle runners, 23 female athletes, 27 male athletes) 61 endurance athletes (middle and long distance runners; 30 female and 31 male athletes) and 55 controls (28 female and 27 male subjects) aged 17–26. The results show that power athletes have bigger BMD of lower limbs, lumbal part of the vertebrae and upper limbs in comparison to the controls. Endurance athletes have bigger BMD than the controls only in lower limbs. Maximum difference in BMD between the athletes and the control group was noticed on the exercise loaded parts. Both male and female power athletes had bigger bone density in lumbal part of the vertebrae than the endurance athletes. During 12 months both athletes and the control group showed a slight but significant increase of BMD of the whole body and the femur. Changes in bone density did not depend on exercise except for the lumbal part of the vertebrae. On that particular place power athletes had significantly bigger bone density than the other groups. The results confirm general view that bone reaction to mechanical loads depends on the place (on the bone and the exercise type).

Frost (1986) assumed that hormones in interaction with cone adaptation by change of mechanical load intensity threshold determine (condition) bone cells sensitivity to mechanical loads. This theory called mechanistic theory (mehanostat) underlies that hormones either increase or hinder the effects of mechanical load on some basic levels within connected bone cells of transduction mechanism. Although there is a written anecdote which corroborates Foster's theory cell mechanism to prove this has not yet been discovered. Further research on mechanic-transduction of bone cells can give answers to this one and the basic questions in the field of bone biology.

THEORETICAL CONSIDERATIONS OF THE PROBLEM

Wolf's law and mechanostat hypothesis

More than 100 years ago German scientist Julius Wolff set a theory which is now called Wolff's law and it reads a bone adapts to the forces applied to it by the change in

quantity and layout of its mass. This theory is almost translated into the general theory of bone mass regulation or mechanostat model. Mechanisms by which mechanical oad changes bone mass are not yet defined while Carter et al. (1989) and Obradović (2000) have suggested that trabecular bone density is determined by its usual history of load. This history represents the sum of all loads where each load is defined according to the quantity of load and the number of repetition. Besides Whalen et al. (1989) have given corroborative evidence for the conclusion that the size of load is a much more useful determiner of the bone mass than the number of repetitions. For example, weightlifting activity of big load and small number of repetitions would have greater effect on the bone than the activities of more frequent repetitions and smaller loads (Obradović, 2000).

One of more plausible and logical explanations of the muscle force influence on the bone integrity is offered in "mechanostat hypothesis". Combination of factors making a sound bone and having a satisfactory effect on amphibians, birds, mammals and reptiles of all sizes, age and sexes is called "mechanostat".Mechanostat is a combination of bone modeling and remodeling mechanisms, their thresholds, bone marrow mediator mechanisms, signal mechanism that connect them and perhaps more mechanisms.

Negative feedback would determine if, where and when bones need more firmness or if the bone does not need it from some mechanical reasons. Different non-mechanical factors including hormones and other humeral factors can change (assist or repel) mechanostat effects on the bone firmness. Mechanostat can be compared to the combination of steering wheel, brake and accelerator in cars. Osteoblasts and osteoclasts would be wheels while mechanical force is represented in a driver.

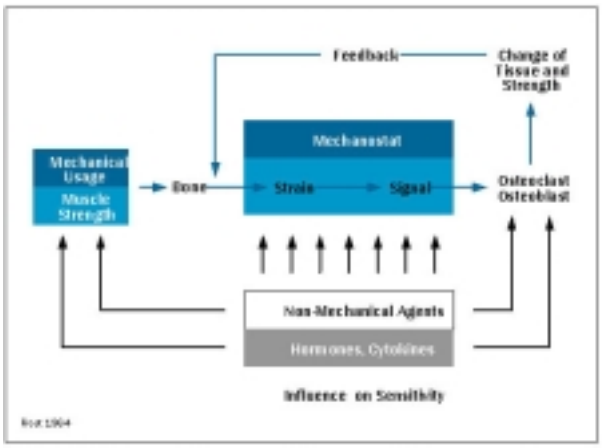

Fig. 1. Figure of "Mechanostat" theory (Schoenau & Frost, 2002)

Functional model of bone development on the basis of "mechanostat" theory. A central part of bone regulation is a feedback between bone deformation (strain of the tissue) and a firmness of the bone. During growth this homeostatic system is always forced to adapt to the surroundings influences. Factors shown below create different forms of the central system of regulation (Schoenau & Frost, 2002).

In the previous research it was claimed that postnatal bone firmness (and mass) in children and adolescents is controlled by hormones, calcium, vitamin D and other

humeral and non-mechanical factors. However later evidence which brought to light a case of Juta in the skeleton physiology show that monitoring of postnatal bone firmness depends to great extent on the biggest mechanical load on the bone. Muscles cause the biggest load and strain on bones and those strains help control biological mechanisms which determine the firmness of the whole bone. Therefore bone firmness in children depends greatly on the muscle power development and the way a bone reacts to it. Many hormones and other non-mechanical factors which influence bone firmness can add or retract from the relationship "bone firmness – muscle power" but they can not change it.

Besides some factors long considered to influence the bones acting directly on the bone cells influence muscle power as well. Thus they can indirectly influence the bone firmness. Those factors are growing hormone, adrenal-corticosteroid compounds, androgens, calcium, vitamin D and its metabolites, etc. Namely bone and muscle form indeed a kind of functional unit. This is a part of the "Jute case" which connects previous views with previous evidence and claims. The above mentioned case explains the functioning of relation "bone firmness – muscle power" (Schoenau & Frost, 2002).

Models of different influences of physical activites on the bone status

Model 1: Fitness level and bone status

Research has shown that there is a positive relation between the level of fitness and the bone mass and density. (Chow et al., 1986) and (Pocock et al., 1986) have proved that lumber spine discs and femoral density is in correlation with fitness level including maximum muscle power and aerobic power. Some studies suggest that physical activity in childhood can significantly influence bone density in adulthood (McCulloch et al., 1990). Yet not all programs of exercise give out the same results. It can be expected that dominant limbs of the sportsmen are exposed to greater stress and total functioning than non-dominant limbs and this can refer to the difference in bone mineral incorporation and bone mass and density. Humeral of the dominant hand in tennis player has shown bigger mass (Jones et al., 1977) and greater bone width and mineral content. (Montoye et al., 1980).

Similar results were obtained for baseball players. In many different sportsmen femoral of the dominant leg also shows greater density than the non-dominant leg. (Nilsson & Westlin, 1971), (Komi, 1996).

Model 2: Aerobic training and the bone status

Transversal research in highly trained sportsmen (during aerobic exercise) has given different results on the effects of aerobic activities on the bone density, especially jogging. Male long distance runners (over 64 km per week) had a similar density of tibial and radial bones but significantly lower density of vertebral bones than short distance runners below 64 km a week. (Bilanin et al., 1989). Young male and female long distance runners aged 13.1 compared to non-trained controls of the same age, height, weight have shown during control checks (Rodgers et al., 1990) significantly smaller ulna length and bone mineral density. This difference in density was more emphatic in males than in females (Komi, 1996).

Chow et al. (1986), has divided 58 women in three groups: control one (n = 19) aerobic dance one (n = 19) and aerobic dance combined with low intensity weightlifting with small weights group (n = 20). After a year the results showed some density increase in combined exercise type group whereas the aerobic group showed slight difference and the control group showed small bone density decrease. This indicates that with postmenopausal women a combined aerobic and power building exercise can remold bones efficiently.

Different effects of the aerobic training on bone mineral density can be achieved by the change in the load, quantity and intensity of exercise.

Small effort (low intensity exercise) will not add to the efficient change on bones no matter how it is distributed (Komi, 1996).

Model 3: Power training and bone status

It is known that sportsmen playing sports demanding movements repetition and greater force such as weightlifting and throws have bigger bone density compared to long distance runners and footballer. Swimmers (load-free exercise) have the smallest bone density (Nilsson & Westlin, 1971).

Colletti et al. (1989) has researched twelve males with regular muscle building training for at least a year and compared them with the control group of fifty man aged 19-50. Load impact training was connected with the increase in bone mineral density in lumbal vertebrae bones, trohanter and femoral neck but not in radius which shows that load impact training causes increase in bone density in weightlifting but not in the non load places (Komi,1996).

Granhed et al. (1987) has demonstrated that in eight weightlifters force exertion on the third lumbal disc (L3) was calculated and the total weight lifted during training in the previous year was referred to the bone mineral content in vertebrae bones. Compression forces on L3 ranged from 18–36,4 kN. Bone mineral content was in high correlation to training difficulty (r = 0,82) (Komi, 1996).

Lane et al. (1988) compared aerobic training (jogging) with power training over five months. Load impact training has increased significantly bone density in lumbal part of vertebrae than the aerobic training.

Some power exercise types can adequately satisfy the criteria for the bone status change. These are exercises including different swift movements and the whole body exercises where one should provide adequate size, frequency and distribution of strain. These are meant to increase to a maximum bone mass and density or prevent their decrease. Load impact training especially with weight carrying component can change essentially bone mineral density (Komi, 1996).

Three rules of bone adaptation to mechanical stimulus

Basic mechanical function of the bones is to provide firm levers as a support for muscles to enable efficient moving. In order to achieve this bones must change form and structure so as to use material in adequate way. By adapting during growth and development of the skeleton bones constantly adjust skeleton mass and structure to the change of mechanical influences.

There are three fundamental rules for the adaptation of bones:

- adaptation of bones occurs more often under the influence of dynamic and rare to a static load,
- certain period of application of mechanical load is needed to bring about a change as a response,
- bone cells adapt to usual influences of mechanical load and this makes them less susceptible to usual load signals.

From these rules one can deduce several mathematical equivalents which constitute parameters for bone adaptation (Turner, 1998).

Bone structures are elegant and structurally fit as if created on the basis of engineering design. This skeleton design is partly contained in the genetic blueprint of the bone cells but there is also epigenetic element of the skeleton creation which is constantly renewed with respect to mechanical forces influencing the bones. Bone cells begin with genetic design and create it until it entirely corresponds to the load demands.

This process called cone adaptation requires bone cells to detect mechanical signals and to convert them to certain bone structure changes. More than a hundred years ago Roux and Wollf claimed that bone structure is in harmony with mathematical laws: thickness and number of trabeculs (or the distribution of bone mass) must correspond to the quantitative distribution of mechanical pressures and the pressure on trabeculars must be exerted axially. Pauwels (Figure 2) has complited this paper by describing the effects of mechanical pressure on the square and bone healing. These laws make a basis of the existing concepts of bone adaptation out of which all other concepts are derived. A great number of experimental findings was collected in the last thirty years and the ideas occurred that make it possible to give a mathematical description of the bone adaptation. (Turner, 1989).

Above mentioned rules are translated into mathematical formulae which will be described in detail.

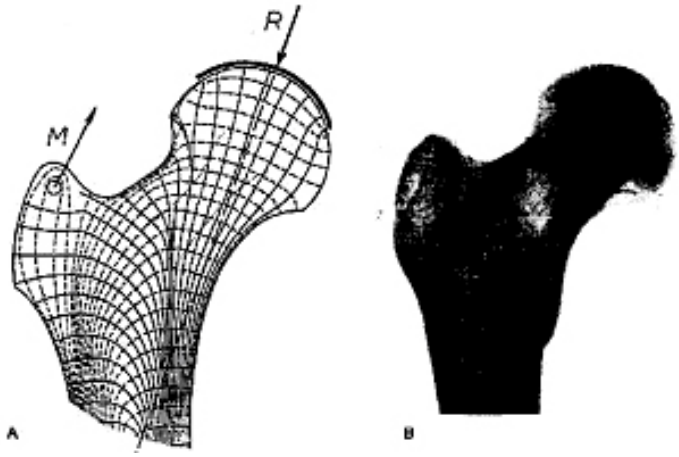

Fig. 2. Effects of the mechanical pressure on square cut of bone (Turner, 1998)

According to Wollf bone structure is based on mathematical laws: thickness and number of trabeculs (i.e. distribution of mass) must respond to the quantitative distribution of mechanical pressure and pressure on trabeculs must be exerted axially. This rule is shown in Pauwels where concordance of pressure direction (A) and trabeculs structure (B) in proximal femoral part is indicated.

Rule 1: Dynamic stimulus of strain

On nature of mechanical stimulus for bone adaptation people discuss for more than a hundred years. We give a short review on this topic.

Wollf gave an opinion in 1892 that pressure on bones determines their structure. Later Thompson claimed the following: "it is exceptionally important truth that state of tension, result of pressure is a direct stimulus for the self development." Frost confirms Thompson's view and adds that mechanical strain is not only a basic indicator of bone adaptation but that " minimal efficient strain", limit of intensity must be overcome before bone adaptation. The most important view gave Hert and associates showing that dynamic and not static strain increases bone formation. Dynamic strain represents basic stimulus for bone adaptation. Lanyon and Rubin confirmed Hert's results using " isolated avian ulna model". These researchers have shown that bone adaptation in this model is directly proportional to some maximum strain (Figure 3). Rubin and Mcleod showed that frequency (i.e. number of cycles in a minute) on the strain curve is extremely important for the bone adaptation. Recent laboratory experiments show that frequencies and repetition of strain are important indicators of bone adaptation (Turner, 1998).

These results show us the following:

- dynamic strain brings about bone adaptation,
- stimulus of strain increases if the size or frequency of dynamic signal is increased,
- frequent repetition of strain increases the stimulus of strain.

In order to incorporate these facts into mathematical formula we must know that frequency of maximum strain is proportional to frequency of strain curve and the size of strain. On careful consideration we find out that stimulus of strain is proportional to frequency of strain as experimentally verified:

$$E = k_1 ef \tag{1}$$

E stands for stimulus of strain
k_1 is a constant of proportion
e is a size of strain from the highest point of one to the highest point of the other strain
f is a frequency of strain in one second cycle

Equation 1 represents stimulus of strain for sinusoid load curve. Yet the result can be generalized by use of Fourier's method which expends the curve s of any periodical strain into series of sinus waves of different amplitudes and frequencies so that stimulus of strain is defined in the following way:

$$E = k_1 \sum_{i=1}^{n} \varepsilon_i f_i \tag{2}$$

Skeleton forming can be shown as a function of strain stimulus which is calculated by formula 2. On the basis of this Equation 2 one can predict the results of different

experiments and different conditions of strain. It can have limitations when predicting the results of applications of high frequency strain. It represents a linear model of relation between size and frequency of strain whereas biological systems are rarely completely linear. For example, on the basis of Equation 2 one can predict that very small strain can cause remarkable strain stimulus if high frequencies are applied, but still there can be limited number of strain frequencies that can influence the bone. Also statical strain does not affect bone adaptation because $f = 0$. It is assumed that the bone adaptation will be proportional to the corresponding size of dynamic strain and that the higher frequency of strain will have bigger effect on bone adaptation stimulus (Turner, 1998).

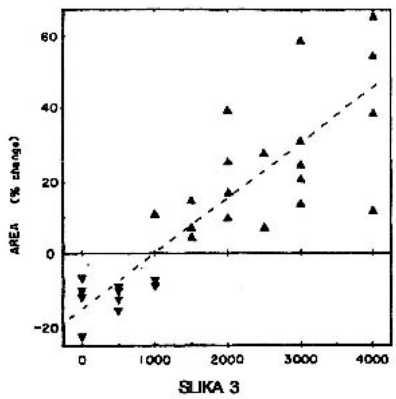

Fig. 3. Relations of bone adaptation and maximum strain (Turner, 1998)

Rubin and Lanyon have demonstrated by use of "isolated avian ulna model" that newly formed bone mass is proportional to corresponding size of strain. This experiment was often interpreted in different inadequate ways but it turned out that size of strain is a primary stimulus for bone adaptation whereas frequency of strain curve is ranked second. Yet frequency of strain, frequency and size of dynamic strain signal are mutually interconnected so that these strain curves shown by Rubin and Lanyon differ not only in size of strain but in their frequency as well (Turner,1998).

Rule 2: A case of reduced response

Prolonged load on the skeleton does not cause proportional increase of bone mass. After longer load as a result over forming of bones can occur. This phenomenon of result absence and reduced response is shown in studies made by Umemur, Rubin and Lanyon.

The following mathematical approach explains absence of results and it was shown by Carter et al. (1989). They claimed that daily strain stimulus (S) should be shown as:

$$S \propto \left[\sum_{j=1}^{k} N_j \sigma_j^m \right]^{\frac{1}{m}} \tag{3}$$

k is a number of different conditions of daily strains applied on bones
N is a number of strain cycles per day for each strain condition
O is an efficient pressure (or strain) for each condition of strain
m is a constant

Value m is a factor of difficulty for relative importance of pressure or strains applied on S. For example, if $m = 1$ effects of strain size and load cycles would be equal. This is faulty effect of strain frequency was not considered but idea of difficulty factor m is a considerable advance.

Experimental data show clearly that m should be bigger than 1. Carter et al. 1989 have concluded that value m should be four which is based on the mass of data given by

Rubin and Lanyon. On the basis of the curve of recent data by Umemur value m should be 3.5 (Figure 4) which is close to Carter's values (Turner, 1998).

Interaction between strain stimulus (ef) and load lasting (number of load cycles per day *N*) can be represented mathematically by logarithm or exponential relation used by Carter et al. (1989). We can combine rules 1 and 2 of bone adaptation into a new formula of daily load stimulus:

$$S \propto \sum_{j=1}^{k} \log(1+N_j)E_j \tag{4}$$

Where:

$$E_j = \sum_{i=1}^{n} \varepsilon_i f_i \, E_j$$

k stands for number of daily load conditions
N is a number of frequency components for each condition of load

Similarly daily load stimulus can be given by use of Carter's formula (Equition 3):

$$S \propto \left[\sum_{j=1}^{k} N_j E_j^m \right]^{\frac{1}{m}} \tag{5}$$

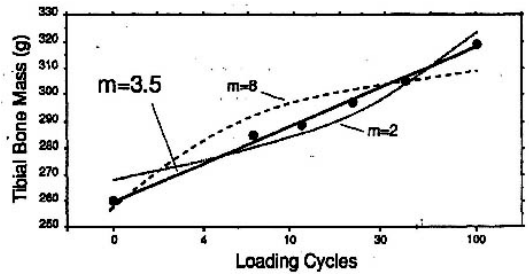

Fig. 4. Interaction of load stimulus and load lasting (Turner, 1998)

Carter's formula (Equation 3) corresponds to Umemura et al data if $m = 3.5$. This is to imply that daily load stimulus is proportional to EN, where E is a strain stimulus and N is a number of load cycles.

Rule 3: Bone cells adaptability to common loads

Application of too much load on skeleton causes its structural changes. As Lanyon claims: " a response to mechanical adaptation is not controlled by many cycles of "normal" strain changes caused by dominant activities but by rather small number of the cycles of relative "abnormal" strain changes caused by unusual conditions of load." This rule reflects adaptation on the cell level which causes bone cell adaptations to "normal" loads of wave forms of usual daily activities such as walking and running (Turner, 1998).

Adaptation means reconstruction of cytoskeleton mechanism cells and/or genetic expression so that the cells adapt to their external local strain. Biological signals that rule

cell adaptation are not completely tested although there is evidence about adaptation. For example, it was discovered that some osteoblasts exposed to mechanical stimuli reorganize their cytoskeletons so as to form actins stretched fibers.

During that process proteins essential for bone adaptation occur in great mass. Yet forming of these proteins is temporary and with continued exposition to mechanical stimuli cells transform into a new permanent biological state in which they adapt to bigger mechanical load. To cause new reaction through cytoskeleton signaling way one should control abnormal strain.

Bone cells adaptation to usual strain patterns in diatheses of many long bones should be expected having in mind data on typical load applied on these bones. Most long bones are bent along their axis and these bends increase rather than decrease mechanical strains. Bones could sustain more weight although their mass is small if they were loaded by axial pressure. Therefore bone bends is a paradox in mechanical design. Bertram and Biewener say that bone bends enable progress because they widen the scope of load pattern prediction on the square cut on bones under different dynamic strains varying both in direction and size. They have shown that small bends of bones increase load sustain possibility and strain patterns prediction. By this bone bends guaranties that part of bone along neutral axis of bending accepts very small strains so we can assume that bone cells have adapted to small loads, on the other hand bone along neutral axis of bend would absorb strains. Size of strain increases with the distancing from neutral axis of bending which means that cells should be adapted to different strains on each point along the parts of long bone.

The state of strain to which the bones are adapted is sometimes called equilibrium (balance) of strain. Great deviations from equilibrium (e.g. abnormal states of load) demand adaptation. This can explain why experimental control of load causing bending along neutral axis has such an enormous effect on bone forming although achieved sizes of strain are satisfactory within psychological limits. In the above mentioned difference between abnormal and normal stimulus of strain on different points of bone tissue can be pretty big (Turner, 1998).

Mathematical function explaining an error of bone adaptation has a general form:

$$\frac{\partial M}{\partial t} = B\{\Phi - F\} \tag{6}$$

M is a bone mass
t is time
F is local state of strain
B and F are constants

F stands for "normal' state of load to which cells are adapted. That means F $-F$ is a function of error causing changes in bone mass of that system. If it is shown by daily load stimulus (S) given in Equations 4 and 5, S–F is a force causing bone adaptation. By normal pattern of load remodeling of bones takes place on low levels but if error in functioning ($S-F$) exceeds lower threshold (e.i. abnormal low load) activity of osteoclasts connected with bone remodeling is increased. If an error in functioning exceeds upper threshold osteoblasts activity on the surface is activated causing long bones or trabeculas forming. Enormously high value S can cause increase in remodeling of bones because of micro damage. (Turner, 1998).

CONCLUSION

Bone adaptation depends on strain size, lasting time, frequency, history (origin), type (pressure, tension) and strain distribution. Three rules shown here make possible mathematical consideration which integrates influences of strain size, frequency, lasting and to some extent origin. Importance of strain types and distribution is yet to be considered. Generally, strains due to extension or under pressure are thought most important for bone adaptation and tension stain cause small effects. There is a vast body of evidence that the flow of fluids within canaliculae and lacunae of bone is mostly responsible for the transduction of mechano-chemical signal in bone cells.

If this is true than the level of hydrostatic strain in bone must be increased to cause bone adaptation. Hydrostatic strain has developed from dynamic dilatation strain and not from tension strain which means that bone adaptation is taking place in dynamic load, dilatation strain (i.e. volume change in tissue), and level of strain. It is not surprising that all studies concentrated on equation 1 use models of animals on which loads causing bending were applied (bending causes certain level of dilatation strain).

Since efficient bone adaptation causes skeleton change and decreases risk of fracture and often fatal pain in the old age process of development is probably continued and increases bone cells abilities to respond adequately to mechanical strains. Current mathematical concept of bone adaptation although not perfect enables understanding of mechano-sensory bone system. Bone adaptation can to certain extent be predicted by means of three basic rules: bone adaptation is more a consequence of dynamic than static strains; prolonged lasting of mechanical load or exercise has a lessening effect on further bone adaptation; bone cells and/or cell network tend to adapt to external mechanical loads so that their reaction to usual load signals is diminished.

Nature of cells adaptation is not yet completely comprehensible but there is evidence that it exists. In any case long bones significantly lose bone mass even if they are not under mechanical load while calviarium is usually under small mechanical strains (pressures) but it does not absorb them because the cells of these bones are adapted to different external loads. It is also possible that hormones and cytokines can cause bone cells adaptation.

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MEHANIČKA SILA I ČVRSTINA KOSTI

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Činjenica da fizička aktivnost ima pozitivan uticaj na skelet postala je već aksiom. S tim u vezi postoji nekoliko dokaza, uključujući obimnu dokumentaciju često poražavajućih posledica imobilizacije i komparativne podatke, koji pokazuju povezanost između povećane fizičke aktivnosti i povećane mase kosti. Pošto je već rečeno da redovna fizička aktivnost korisno deluje na skelet, može se postaviti pitanje koji su to specifični delovi programa koji bi bili najpovoljniji za kosti. Recept za vežbanje se može definisati prema vrsti vežbanja, intenzitetu, trajanju i frekvenciji, mada je nemoguće napraviti potpune preporuke. Naročito zbog toga što nemamo bazu na osnovu koje bi određivali optimalno trajanje ili frekvenciju vežbi za skelet. Predmet ovog rada je koštani status i mehanička opterećenja kosti. Problem rada je da se utvrdi mehanizam uticaja različitih fizičkih aktivnosti na koštani status. Cilj rada je da se doprinese shvatanju na koji način i u kom obliku različite vrste fizičke aktivnosti utiču na skeletni status.

Ključne reči: *mehanička sila, čvrstina kosti, trening snage, adaptacija*