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No.	Title	Page
01	The Prospective Effects of Exercise Sports on the Body's Immune Functions	01-09
02	Nutritional Status and Body Composition of Elderly consultants in outpatient medicine in Casablanca - Morocco	10-14

The Prospective Effects of Exercise Sports on the Body's Immune Functions

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ABSTRACT

Strenuous or intense exercise has severe and continual influences on a person's systemic immunity. When athletes and non-athletes are resting, their respective systems are more alike than dissimilar except the activities of the NK cell which have a tendency to be elevated in the athletes. Many constituents of the immune system display adverse changes after long-drawn-out heavy exertion. These changes occur in several sections of the immune system and the body (like the skin, lungs, blood, muscles and the mucosal tissues of the upper respiratory tract). Although it is still subject to interpretation, most exercise immunologists are of the opinion that it is during the said "open window" of impaired immunity (whereby the duration may vary from 3 to 72 hours, contingent on the immune measure) during which viruses and bacteria may possibly gain a foothold, thereby resulting in a higher risk of subclinical and clinical infection. The risk of infection can become greater than before when the other immune-related factors are present, such as being exposed to novel pathogens when travelling, not having enough sleep, undergoing severe mental stress, suffering from weight loss or malnourished.

Keywords: Exercise, training, leucocytosis, antibodies, immunoglobulins, infection

INTRODUCTION

The methods that can be used to assess the most important functional elements of the immune system are described here. The seriousness of the responses to exercise is dependent upon the intensity and period of the required activity as well as the fitness level of the individual. Moderate endurance exercises do not change or enhance indices such as granulocyte, leucocyte, lymphocyte, monocyte and total T-cell counts, serum immunoglobulin levels, helper-suppressor cell ratio, in vitro immunoglobulin production and cell proliferation in response to mitogens. But, very tiring exercises then to produce unfavorable changes in these

indices, especially if the physical activity occurs with environmental or competitive stress.^[1] Moderate and appropriately graded training decreases the reactions to any of the exercise with an absolute intensity. When undergoing a training regimen that is more demanding, it is vital for the person doing the exercise to optimize his immune responses. If the preparation for the athletic performance is worked at till it becomes stale and results in muscle injury, it may have extensive negative implications for several aspects of the immune functions and it includes resistance to acute infections, cancer, ageing, HIV infections as well as any other conditions that are influenced by the immune system.^[2]

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THE METHODS FOR ASSESSING THE RESPONSES OF THE IMMUNE FUNCTIONS TO EXERCISE

Exercise and its overall effect on the immune system can be observed by plotting a subject's responses to inoculations or general susceptibility to infections.

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The separate elements of the system can be assessed by carrying out differential blood counts, analyzing lytic activity, quantifying the degree of cell proliferation or immunoglobulin production in reaction to cytokines or external mitogens, and assessing cytokine or cytokine receptor densities.^[3]

Susceptibility to Infections

Susceptibility to infections can be assessed by inoculating subjects with a standard amount of a comparatively harmless virus like that of the common cold. But, rapid mutation of the virus can give rise to problems and result in a loss of immunity. Another possible method is to examine antibody production after the injection of anti-tetanus toxoid or Merieux Multitest.^[4]

Epidemiologists have established a link between incidences of specific infections with heavy training sessions or strenuous competition. But, do not forget that exercise can change the risk of infection through mechanisms apart from a change of immune function. Examples of the earlier assertion includes activities that may result in exposure to contaminated water or air, cold or polluted air when inspired may depress the function of the tracheal cilia, or a change of lifestyle by the subject may vary his chances of falling sick.^[5]

The Differential Blood Counts

The population of white blood cells includes polymorphs (basophils, eosinophils and neutrophils) and mononuclear cells (lymphocytes, monocytes and plasmocytes, a progeny of B lymphocytes). The various sub-populations in the blood can be ascertained through the use of monoclonal antibodies. The total and differential white cell counts provide some clues of the functional condition of the immune system, but many other factors may alter the reading during bouts of vigorous exercise.^[6]

An incomplete list of extraneous influences that can alter peripheral leucocyte counts during strenuous activity includes: (1) a reduction of blood volume; (2) higher cardiac output resulting in the demargination of hitherto sequestered cells; (3) the activation of adrenoreceptors that decreases the number of leucocytes that are attached to the endothelium depending on the level of circulating catecholamines; (4) autonomic nerve activity that results in the release of catecholamines and co-

transmitters; and (5) the secreting of cortisol that stimulates the release of granulocytes from the bone marrow.^[7] The analysis is complicated because a big portion of the total leucocyte count is ordinarily not circulated. The amount and whereabouts of the non-circulating leucocytes can be traced by injecting radiolabelled autologous cells into the body.^[8] The non-circulating neutrophils are generally located in the liver, lungs and spleen, but the non-circulating lymphocytes are predominantly located in the liver and spleen. During long exhaustive runs, the number of immature neutrophils in the blood circulation system increases by 17 times, and this indicates that the non-circulating cells have been carried into the general circulation system. In human beings, exercise has minimal influence on the dimensions of the spleen and splenectomy also has minimal influence on the exercise-induced leukocytosis. The changes in the total leucocyte or lymphocyte counts indicate the mobilization of cells from the liver and the 2A lymphocytes is mainly responsible for the cell-mediated immune function.^[9] The lymphocytes are divided into T cells (coded in the thymus in response to both specific allergens and nonspecific mitogens), B cells (it undergoes the maturing process in the bone marrow) and null cells. The T cells were initially distinguished due to their tendency to form “rosettes” with sheep.^[10]

Cell Proliferation

When lymphocytes are incubated with tritiated thymidine, the radioactive molecules are integrated with the DNA of the newly formed cells. From the radioactivity of the newly formed cells it is possible to study how the spontaneous proliferation rate is transformed by exercise, training and non-specific activators like hormones, antigens, cytokines and neuropeptides.^[11] Most of the time, the cell proliferation rate is assessed in vitro and whole blood or the mononuclear cells from washed peripheral blood are incubated with non-specific mitogens like plant lectins, concanavalin A (Con A) or phytohaemagglutinin (PHA). When the procedure is carried out using whole blood samples, the responses to PHA are known to be resistant to oral cortisone, but the responses to Con A are depressed by cortisone. The mitogens derived from plants act on receptor sites that are different from the receptors for specific antigens. The lectins stimulate a non-specific reproduction of all the subsets of T cells which can be employed to test

the effect of exercise and training on the particular kind of lymphocyte.^[12] The major actual problem with this assay is that the responses varies enormously depending on the concentration of the mitogen. Therefore, the tests have to be replicated by using different concentrations of mitogen so as to find the optimal dose. A 12 hour fast by the subject will enhance the chance of getting the same response from a given subject.^[13]

Immunoglobulin Synthesis

The changes in the concentration of immunoglobulins of the plasma or saliva do not mean that there are corresponding changes in the immunoglobulin synthesis.^[14] The body fluids concentration are affected by receptor binding, blood concentration, catabolism as well as the movement of protein involving the blood and other fluid compartments. The salivary concentrations are also affect by the amount of saliva that is secreted. The most common type of immunoglobulin is IgG, but it also includes anti-toxic, anti-bacterial and anti-viral antibodies as well as potent opsonins which can enhance phagocytosis. Macroglobulins (IgM) are found in cytoplasm as well as on the surface of B cells during the early stages of their maturation. The first group of antibodies is produced by the plasma cells that are developed from the activated B cells and it include cold agglutinins, haemagglutinins, IgA, IgD and IgE.^[15] The capability of the plasmocytes to manufacture immunoglobulin can be evaluated in vitro by using antihuman IgM and IgA after the peripheral blood mononuclear cells have been incubated with a non-specific mitogen like pokeweed, that can activate T and B cells.^[16]

THE REACTION OF THE IMMUNE FUNCTIONS TO ACUTE BOUTS OF EXERCISE

Even though exercise can induced noticeably severe responses in some components of the immune system, the said responses are normally transitory, and there have been queries about the extent such changes can affect the body's defense system in the presence of bacteria, viruses and neoplastic cells. If blood samples are taken more than 30 minutes after the exercise, the results of the analysis may be affected by the rebound phenomenon and the measurement of the various immune functions may even exceed that of the pre-exercise levels.^[17] Unfortunately, the responses are fairly

variable and changes from one day to another as well as one person to another thus making it difficult to generalize. The factors that can modify the reactions of the immune system consist of the amount of effort that is put in relative to the person's state of training, the amount of time spent doing the exercise as well as associate competitive and environmental stresses. With regards to the duration of the exercise, it has been noted that short-term activities will mobilize the sequestrated cells while longer duration of activities will result in them escaping into the tissues. The results that have been obtained show that it is dependent upon the methods that were used during the assessment of the immune function.^[18]

Leucocytosis of the Blood and Lymphocytosis

Previously, the studies merely report on the total white cell or lymphocyte counts. Acute exercise brings about a higher peripheral venous leucocyte count that is approximately proportional to the severity and period of activity. However, prolonged activity may reduce total leucocyte counts caused by the migration of monocytes and NK cells into the injured muscles. Strenuous exercise may result in a delayed leukocytosis about 30 minutes to 3 hours after the exercise and this is due to the release of white cells, stimulated by cortisol, from the bone marrow. After a marathon, the late leukocytosis may persevere for a few hours, but if the exercise is moderate, it is more often than not complete with 6 hours.^[19] However, the granulocytes and especially neutrophils causes most of the late increment in the white cell count. The response is most obvious in persons that have a high capacity for physical work whereby the eosinophil count drops whereas the basophil count remains relatively unchanged. How significant these responses has on body functions is still in doubt, but non-specific immunity may be improved.^[20]

The is a substantial increase in the monocyte count during or as soon as the exercise is completed, and at this stage the lymphocyte count also shows some increase. Some studies have put forward the idea that the response is dependent on the kind of exercise performed by the subject, for example, cycle ergometry gives rise to a larger lymphocytosis than treadmill exercise.^[21] Other inter-trial differences point to the time blood samples were taken relative to when the exercise was performed and this is due to the use of non-automated cell-counting methods in the early days which resulted in relatively smaller number of blood

samples and the immune function recovery was often already completed within 30 minutes after the exercise. Not less than two groups of hormones play a part to the changes in cell counts, At the beginning stages of the exercise, catecholamine secretion invigorates the discharge of lymphocytes from the endothelia of the venules, through the process of “demargination”. Further into the exercise, the cortisol that is secreted induces and overall leukocytosis which in turn stimulate the discharge of granulocytes from the bone marrow. However, it also impedes the entry and expedites the exit of lymphocytes from the circulation. Perhaps, some of the lymphocytes enter the muscle tissues together with the monocytes and NK, thereby expediting the repair processes. The remainder migrate to the lymphoid tissues, where they have a bigger probability of encountering macrophages as well as other antigen-loaded cells.^[22]

Lymphocyte Subsets

Outdated or archaic studies using non-specific markers indicated that exercise increases the proportion of B cells. But, the early investigators were not able to differentiate B cells from natural killer cells. It is an important source of blunder when stating B cell counts because exercise is known to have increased the NK counts significantly and therefore there is a need to cast doubts on the accuracy of such reports.^[23]

Nevertheless, current monoclonal antibody methods indicate that the absolute numbers of T and B cells rises after a 15 to 30 minute-round of submaximal exercise. However, the relative percentage change of the T and B cells varies from one study to another depending on the methodology, intensity of effort and the subject’s fitness. On the whole, immediately after a half-hour of energetic submaximal treadmill exercise the proportion of B cells shows a small decrease.^[24]

The helper-suppressor cell ratio has a significant influence upon susceptibility to infection. Berk *et al.*^[25] reported that immediately after the maximal treadmill exercise the overall percentage of T cells remained unchanged. However, the helper-suppressor ratio experienced a transitory drop from 1.94 to 1.36, an unsatisfactory level. Keast *et al.* reported findings that are essentially similar, and remarked that strenuous exercise heightened the sensitivity of T cell P-adrenoreceptors, *i.e.*, by 121% and 80% on the helper T cells and suppressor T cells respectively.^[26] Some researchers have, in general, found an increase

while others a reduction in the percentage of T cells when the subject is exercising, but most of them have corroborated the early reduction of the helper-suppressor cell ratio.^[27] Exercise apparently increases the number of cytotoxic T cells and at the later phases of recovery up to 24 hours after the exercise, the helper-suppressor cell ratio is higher largely due to a cortisol-induced reduction of suppressor cell numbers and this may possibly compensate for the snuffing out of natural killer cell activity.^[28]

The number of inter-cell differences of the P-adrenoreceptors is directly linked to the sensitivity to exercise-induced secretion of catecholamines and may lead to the lymphocyte subset changes for the duration of and after the exercise. B cells have about thrice the number of such receptors as T cells whereas the number of receptors T helper cells have is quadruple that of T suppressor cells. In addition to that, there is further a potential for up- or down-regulation of the system because the lymphocyte f3-receptors is controlled by normal levels of catecholamines and cortisol in the blood, and training.^[29]

The Natural killer Cell Numbers and Activity

Edwards *et al.* reported that 5 minutes of stair-running resulted in an instantaneous four- to five-fold rise in natural killer cell numbers as well as an upsurge of overall NK activity (based on the amount of chromium that was released from the labelled myeloid tumor cells). Other researchers have also document the early increase in the number and/or percentage of NK cells during moderate bouts of exercise.^[30] A catecholamine-mediated reduction of cell margination may also contribute the increase in NK cell count. However, the intensity and duration of exercise that can create such effects has yet to be defined. Exercise can also activate and bring about an instantaneous higher proportion of killer cells. Berk, L. S., *et al.* found that, immediately and within 24 hours after a 12.8 kilometer run, there was an enhancement of the cytotoxic activity of NK cells.^[31] Similarly, Brahmi, Zacharie, *et al.* noted that NK activity increased by 40% 1 hour after the exercise. Unfortunately, strenuous exercise appears to have a less favorable long-term impact upon the natural killer cells. Berk *et al.* noted that there was a 31% drop in NK cell activity 90 minutes after a 3-hour marathon as well as a 50% drop in the number cells carrying the NK specific CD16 antigen, but there were no changes in the number of cells carrying the CD56 antigen

that is common to cytotoxic T and NK cells. Shek & associates as well as Shinkai *et al.* have also reported a lengthened suppression of NK cell activity after a sustained round of exercise. Shek *et al.* discovered that there was a substantial drop of NK count and activity, and it persisted for not less than 7 days after one round of exercise that lasted between 90 to 120 minutes at 65% maximal oxygen intake.^[32] However, their important findings have not been replicated by other laboratories or researchers.^[33] The initial increase in of cell activity is restrained by naloxone, an endorphin inhibitor, implying that endogenous opioids could probably be mediating the preliminary stimulation of the natural killer cells. If that is so, the round of exercise probably ought to be fairly vigorous because moderate activity does not have much influence upon the secretion of endorphin. Exercise-induced fluctuations in the concentration of interleukins and interferons can also modify the surface properties of the NK cells, including their lytic activity. The NK activity is negatively correlated with the serum cortisol levels and a large gush of cortisol secretion will probably cause the late suppression of NK activity. Prostaglandins released by the monocytes may also cause the persistent late reduction of NK cell activity. If that is the case, the negative impact of sustained or repeated rounds of heavy exercise can possibly be neutralized by the administration of indomethacin.^[34]

Immunoglobulin Synthesis

Hanson and Flaherty noted that the serum immunoglobulin levels did not change 10 minutes following a 13 kilometer submaximal run, however the concentrations in serum, saliva and in the nasal, saliva and serum secretions generally dropped after extended exhausting activity and sometimes it take as long as 4 days to recover.^[35] Most researchers have deduced that there is a corresponding inhibition of immunoglobulin production. Zoher, *et al.* detected a half an hour after finishing a marathon run the *in vitro* production of tetanus antibodies was normal.^[36] However, Hedfors *et al.* stated that just 15 minutes of submaximal exercise was enough to slow down the pokeweed-stimulated production of IgG as well as IgM. Quite recently, it was observed that properly trained long distance runners who, after finishing a 30-minute round of submaximal treadmill exercise, showed higher pokeweed-stimulated *in vitro* production of IgG 5 minutes after the exercise. The discrepancies is due to the following differences: (1) the amount of

exercise performed; (2) the timing of the sampling; and (3) the subject's level of training. In addition to that, Hedfors *et al.* adopted the whole-blood culture method in their studies.^[37]

THE CASE OF THE IMMUNE FUNCTION AFTER ENDURANCE TRAINING

The use of cross-sectional comparisons between animals that have been trained and those that have not or endurance sports persons and sedentary subjects is advantageous because of the prolonged training as well as there being sufficient opportunity for the individuals to get use to the physical demands of the strenuous activities.^[38] During the process of making the said comparisons of immune responses between the endurance sports persons and sedentary subjects, it is crucial to distinguish the relative strength of any workout that is performed so as to be able to take into account the stresses of concurrent competition as well as the heavy travel plans, and to make sure that there is sufficient recovery from the recent training sessions.^[39]

If there is no "overtraining", the immune status of athletes at rest is generally normal, even though some researchers have noticed granulocytosis, lymphocytosis, higher antibody-dependent cytotoxic and NK cell activity as well as plasma IL-1 and IL-2 activity.^[40] At any given absolute work-rate, athletes will have less leukocytosis than sedentary persons, however if the two groups are stressed to the maximum or at a comparable fraction of maximum effort, the leukocytosis appears to be comparable in the two groups. There are essentially no dissimilarities in the overall T cell or trained and untrained persons' subset responses to exercise. In any case, the test results on mitogen responsiveness is conflicting.^[41]

Athletes that are undergoing active training may have poorer phagocytic activity. Simpson *et al.* remarked that compared to healthy men without training, athletes who are taking part in basic controlled intensity training have lower total lymphocyte, NK, T and T helper cell counts and the CD4-CD8 ratio was also lower in their resting blood samples.^[42] Oshida *et al.* have also observed that while exercise unvaryingly lowered the percentage of T and T helper lymphocytes cells, in trained athletes the percentage of T suppressor cells showed distinct increases. However, they noted that there was an increase the NK count and the preceding

finding was recently duplicated by Rhind et al. The higher NK count in athletes also appears to be linked to an upsurge in the number of cells bearing markers of the 70-75kDa P-receptor for IL-2 (but, it does not have the p55IL-2 receptor).^[43]

Pedersen et al. reported that the resting salivary IgA levels were lower in elite cross-country skiers when compared with the controls, although it was probably due to incomplete recovery from the previous exercise. The levels decreased further after 3 to 3 hours of exhaustive skiing, although the analysis of the data was complicated by the changes in the volume of saliva that was secreted.^[44] A few other researchers have also observed that elite performers have low serum immunoglobulin levels, but most researchers have discovered there were either no changes or even the immunoglobulin readings were higher in response to more moderate training, especially if due care was taken to allow for the expansion of plasma volume induced by training.^[45]

Tomasi et al. noted that complement levels, both during and after exercise were found to be lower in marathon runners than in age-matched controls.^[46] They conjectured that the demands of repetitive distance running may have overworked the liver's ability to synthesize, although the blood volume was altered, the catabolism of amino acids like glutamine in response to depressed levels of glycogen and urgent repair reactions in damaged muscles may also have contributed to this finding. The serum levels of C-reactive protein in athletes are also lower than those in the control group and this may be caused by the chain of events that were induced by muscle injury.^[47]

Longitudinal Training Studies

The response to planned training is dependent on the intensity, frequency and period of the applied regimen, as well as on the preliminary condition of the individual. Several researches involving training whereby the sports persons almost reached the point of overtraining, indicated that after training the T cells can account for a larger proportion of the total lymphocytes, but the helper-suppressor cell ratio is decreased. If the training was strenuous, the NK cell count may also drop and it is caused by the migration to injured tissues or they were converted into T cells. But, Woods et al. discovered that NK cell activity was increased in an elderly population upon the completion of a light training program.^[48]

Training tends to increase resting mitogen-induced lymphocyte proliferation, but the recovery period after the final round of exercise must be sufficient for the response to be present. Thus, Koch et al. noted that there was a decreased response to mitogens immediately after the cessation of a training program, but after 72 hours of recovery the response was higher.^[49] Training typically weakens the overall lymphocytosis of a sedentary person when he participate in an exhausting exercise. Rhind et al. observed that 12 weeks of moderate exercise weakened the exercise-induced lower in vitro IL-2 production and augmented the expression of IL-2 p-receptors. But, exhausting training has a calmative effect on animals and humans. In rats and mouse, the mass of the thymus is reduced and the lymphocytes of the spleen become less responsive to mitogen stimulation, and it may be because the proportion of T cells in relation to B cells have been altered and it may also be due to T suppressor cell actions or macrophage-secreted prostaglandin E2. Similarly, data from studies in humans indicate that after a round of submaximal exercise the lymphocyte response to mitogen usually decreases even though the proliferation may be increased in athletes who abuse the usage of anabolic steroids.^[50]

Moderate exercise seemingly increases resting plasma IgA levels whilst on the other hand, heavy exercise lowers the IgG and IgM resting levels, and mitogen-stimulated IgG synthesis. Immediately before and during major competitions, the levels of IgG, IgA and IgM are also low.^[51]

Interaction with Other Stressors

If an athlete's diet is insufficient to meet the demands of exercise, an absence or deficiency of amino acids like glutamine can have an adverse effect on the growth of immune cells. Athletic competition by itself can be perceived as stressful or it may be caused by environmental and psychological stress. The stress-induced secretion of cortisol may repress some aspects of the immune function. Finally, extended exercise by itself may stimulate the release of cortisol, even though it is a normal metabolic control mechanism there are parallel implications for the immune function.^[52]

Systemic infections can change the immune responses to training and can also directly cause a deterioration of physical performance and it can be a source of stress for an athlete. Even though an exercise may not be stressful to a healthy person, it can become physically and

psychologically stressful if the person has a developing infection. During the assessment of supposed training-induced responses, consideration must be given to the superimposed stresses including how the factors change as the subjects become familiar or get used to any given laboratory or competitive environment.^[53]

THE CLINICAL EMBODIMENTS

In conclusion, several clinical embodiments of the exercise-induced changes of the body's immune function will be briefly discussed.

Detection of Overtraining

There were hopes that the changes of the resting immune parameters or a disturbed immune response to training might give an early warning that an athlete's conditioning program was too intensive and was becoming over-trained. It is difficult to conduct such type of experiments because it is ethically incorrect to make athletes train until they reach the level of injury. Richardson *et al.* observed that when a group of long distance athletes undergoing intensive training intentionally, over a period of three weeks, increase their average training intensity by a whopping 38%, the resting mitogen-stimulated lymphocyte proliferation have a propensity to increase.^[54]

The helper-suppressor cell ratio decreased (perhaps, the athletes did not reach the overtraining threshold but the ratio was above the important level of 1.5) and the pokeweed mitogen-induced synthesis of immunoglobulins was less than normal. Furthermore, a 30 minute round of submaximal exercise (which formerly did not modify cell proliferation) has now induced an 18% suppression of lymphocyte proliferation and the immunoglobulin synthesis, stimulated by exercise, did not happen. Nonetheless, the changes that occurred were small and varied. As such, the researchers concluded that simple psychological tests may perhaps offer a simpler and more effective method to detect staleness in an athlete.^[55]

The Risk of Infection

Viral infections is a serious threat to international sports persons. Experiments and clinical studies of animals have linked too much physical activity to any increased risk of viral infections and viral myocarditis. Verde *et al.* opined that two out of ten long distance runners contracted acute upper respiratory

tract infection (URTI) in response to a 3-week training schedule that was deliberately intensified and they have linked this outcome to evidence of immunosuppression.^[56] Similarly, Peters discovered that the chances of developing URTI were doubled in runners who ran more than 97 kilometers per week during training as compared to those who ran less than 32 kilometers per week. When a runner takes part in a major marathon, the chances of infection is 6 times more than other runners who did not take part. On the other hand, moderate exercise evidently increase the human volunteers resistance to some diseases.^[57]

The Risk of Cancer

The role of the NK cells is to destroy tumor cells, but excessive exercise is believed to increase an athlete's risk of developing some form of cancer. A few early experiments carried out on animals indicated that moderate exercise make the animals more resistant to experimental tumors. Studies done with humans indicate that moderate, occupational and/or leisure activity offers protection against some types of cancer. In colon cancer, the mechanism probably involves an alteration of the colon's transit time instead of an exercise-induced modification of the immune function. Reproductive cancers in active women may also undergo small alterations, but the change appears to be linked to lowered body fat and lower estrogen levels, instead of enhanced immune function. Experiments to treat certain type of cancers with IL-2 were carried out recently. Excessive doses of interleukins gave rise to major complications and a training program can probably play an important role in the development of IL-2 receptors, thereby resulting in a lower dosage of cytokine to treat the patient.^[58]

Ageing

Ageing is linked to the progressive deterioration of the immune function as well as the development of various autoimmune disorders. Therefore, probably moderate exercise may offer some protection whereas excessive exercise may hasten the problems. Currently, there is not much evidence to show that habitual exercise delays the ageing process, however studies on animals indicate that animals with physical inactivity and a restricted diet has a slower inherent rate of ageing.^[59]

CONCLUSION

The evidence reviewed in this brief article indicates that a moderate dose of endurance exercise has

beneficial effects upon a person's immune responses, but more intense and stressful exercise may have a persistent adverse effect. Most of the changes that were acquired from one round of moderate exercise were fairly short-lived, but a prolonged round of exercise or training for an event like a marathon race can cause a prolonged suppression of NK activity, exposing the athlete to an immediate risk of viral infections as well as potential adverse changes in other more long-term manifestations of impaired immune function. The probability that any given round of exercise may have an adverse effect on immune function is dependent on the relative intensity of the effort that is demanded. Regular training can move the threshold for adverse reactions higher. Nevertheless, given the importance of the immune system in the many aspects of health, this writer is of the view that there is a need to have more information as to the dose of exercise that will optimize a person's responses and to avoid the long-term negative consequences.

REFERENCES

1. Pennebaker, James W., Janice K. Kiecolt-Glaser, and Ronald Glaser. "Disclosure of traumas and immune function: health implications for psychotherapy." *Journal of consulting and clinical psychology* 56.2 (1988): 239.
2. Adams, Jeremy, and Robert Kirkby. "Exercise dependence and overtraining: The physiological and psychological consequences of excessive exercise." *Sports Medicine, training and rehabilitation* 10.3 (2001): 199-222.
3. Nieman, DAVID C. "Exercise, upper respiratory tract infection, and the immune system." *Medicine and science in sports and exercise* 26.2 (1994): 128-139.
4. Woods, Jeffrey A., Victoria J. Vieira, and K. Todd Keylock. "Exercise, inflammation, and innate immunity." *Immunology and allergy clinics of North America* 29.2 (2009): 381-393.
5. Brenner, Ingrid KM, Pang N. Shek, and Roy J. Shephard. "Infection in athletes." *Sports Medicine (Auckland, NZ)* 17.2 (1994): 86-107.
6. Smith, Lucille Lakier. "Overtraining, excessive exercise, and altered immunity." *Sports Medicine* 33.5 (2003): 347-364.
7. Connolly, Peter H., et al. "Effects of exercise on gene expression in human peripheral blood mononuclear cells." *Journal of applied physiology* 97.4 (2004): 1461-1469.
8. Pedersen, Bente Klarlund, and Laurie Hoffman-Goetz. "Exercise and the immune system: regulation, integration, and adaptation." *Physiological reviews* 80.3 (2000): 1055-1081.
9. *Ibid*, 1058- 1077.
10. Engler, Harald, et al. "Effects of repeated social stress on leukocyte distribution in bone marrow, peripheral blood and spleen." *Journal of neuroimmunology* 148.1 (2004): 106-115.
11. Alonso-Fernández, Patricia, and Mónica De la Fuente. "Role of the immune system in aging and longevity." *Current aging science* 4.2 (2011): 78-100.
12. *Ibid*, 79- 90.
13. Shephard, Roy J., Shawn Rhind, and Pang N. Shek. "Exercise and the immune system." *Sports medicine* 18.5 (1994): 340-369.
14. Boyum, A., et al. "The effect of strenuous exercise, calorie deficiency and sleep deprivation on white blood cells, plasma immunoglobulins and cytokines." *Scandinavian journal of immunology* 43.2 (1996): 228-235.
15. *Ibid*, 230- 233.
16. Herbert, Tracy Bennett, and Sheldon Cohen. "Stress and immunity in humans: a meta-analytic review." *Psychosomatic medicine* 55.4 (1993): 364-379.
17. Walsh, Neil P., et al. "Position statement part one: immune function and exercise." (2011).
18. Shephard, Roy J., et al. "Physical activity and the immune system." *Canadian Journal of Sport Sciences* (1991): 85-163.
19. Pedersen, B. K., T. Rohde, and K. Ostrowski. "Recovery of the immune system after exercise." *Acta Physiologica Scandinavica* 162.3 (1998): 325-332.
20. *Ibid*, 325-330.
21. Shalaby, Mohamed. "The Effect of Physical Activity on the Support and Enhance the Natural Behavior of Stem Cells." PhD I medicine, Jilin Univ, China (2012): 12- 43.
22. *Ibid*, 22- 28.
23. Klarlund, and Hoffman-Goetz. "Exercise and the immune system." 1058-1079.
24. Shinkai, S., et al. "Acute exercise and immune function." *International journal of sports medicine* 13.06 (1992): 452-461.
25. Berk, L. S., et al. "Lymphocyte subset changes during acute maximal exercise." *Medicine & Science in Sports & Exercise* 18.6 (1986): 706.
26. Keast, D., K. Cameron, and A. R. Morton. "Exercise and the immune response." *Sports Medicine* 5.4 (1988): 248-267.
27. Shavit, Yehuda, Raz Yirmiya, and Benzion Beilin. "Stress neuropeptides, immunity and neoplasia." *The neuroendocrine-immune network* 163 (1990): 163.
28. Berczi, I. "The effects of growth hormone and related hormones on the immune system." *Pituitary function and immunity* (1986): 133-159.
29. David "Exercise, infection, and immunity." 131-141.
30. Edwards, A. J., et al. "Changes in the populations of lymphoid cells in human peripheral blood following physical exercise." *Clinical and experimental immunology* 58.2 (1984): 420.
31. Berk, L. S., et al. "The effect of long endurance running on natural killer cells in marathoners." *Med Sci Sports Exerc* 22.2 (1990): 207-212.
32. Brahmi, Zacharie, et al. "The effect of acute exercise on natural killer-cell activity of trained and sedentary human subjects." *Journal of clinical immunology* 5.5 (1985): 321-328.
33. Subotnick, Steven I. *Sports & exercise injuries: conventional, homeopathic & alternative treatments*. North Atlantic Books, 1991: 21- 155.
34. Hines, Melissa Trogdon, et al. "Exercise and immunity: a review with emphasis on the horse." *Journal of Veterinary Internal Medicine* 10.5 (1996): 280-289.
35. Hanson, P. G., and D. K. Flaherty. "Immunological responses to training in conditioned runners." *Clinical Science* 60.2 (1981): 225-228.
36. Kapasi, Zoher, et al. "Effect of duration of a moderate exercise program on primary and secondary immune responses in mice." *Physical therapy* 83.7 (2003): 638.
37. Hedfors, E., et al. "Physiological variation of blood lymphocyte reactivity: T-cell subsets, immunoglobulin production, and mixed-lymphocyte reactivity." *Clinical immunology and immunopathology* 27.1 (1983): 9-14.
38. Klarlund, and Hoffman-Goetz. "Exercise and the immune system." 1060-1072.
39. Shephard, Roy J., and Pang N. Shek. "Cancer, immune function, and physical activity." *Canadian Journal of Applied Physiology* 20.1 (1995): 1-25.
40. MacKinnon, Laurel T. "Overtraining effects on immunity and

- performance in athletes." *Immunology and cell biology* 78.5 (2000): 502-509.
41. Blannin, Andrew K. "Acute exercise function." *Immune Function in Sport and Exercise* (2006): 67.
 42. Simpson, Richard J., et al. "Exercise and the aging immune system." *Ageing research reviews* 11.3 (2012): 404-420.
 43. Oshida, Y., et al. "Effect of acute physical exercise on lymphocyte subpopulations in trained and untrained subjects." *International journal of sports medicine* 9.02 (1988): 137-140.
 44. Klarlund, and Hoffman-Goetz. "Exercise and the immune system." 1061-1078.
 45. Dimitriou, Lygeri, N. C. C. Sharp, and Michael Doherty. "Circadian effects on the acute responses of salivary cortisol and IgA in well trained swimmers." *British Journal of Sports Medicine* 36.4 (2002): 260-264.
 46. Tomasi, Thomas B., et al. "Immune parameters in athletes before and after strenuous exercise." *Journal of clinical immunology* 2.3 (1982): 173-178.
 47. Kasapis, Christos, and Paul D. Thompson. "The effects of physical activity on serum C-reactive protein and inflammatory markers: a systematic review." *Journal of the American College of Cardiology* 45.10 (2005): 1563-1569.
 48. Woods, Jeffrey A., et al. "Effects of 6 months of moderate aerobic exercise training on immune function in the elderly." *Mechanisms of ageing and development* 109.1 (1999): 1-19.
 49. Koch, Alexander J. "Immune response to resistance exercise." *American Journal of Lifestyle Medicine* 4.3 (2010): 244-252.
 50. Rhind, S. G., et al. "Differential expression of interleukin-2 receptor alpha and beta chains in relation to natural killer cell subsets and aerobic fitness." *International journal of sports medicine* 15.06 (1994): 311-318.
 51. Smith, Carine. *Exercise, stress and immune system functional responses*. Diss. Stellenbosch: University of Stellenbosch, 2004: 19-88.
 52. Rennie, Michael J., and Kevin D. Tipton. "Protein and amino acid metabolism during and after exercise and the effects of nutrition." *Annual review of nutrition* 20.1 (2000): 457-483.
 53. Venkatraman, Jaya T., and David R. Pendergast. "Effect of dietary intake on immune function in athletes." *Sports medicine* 32.5 (2002): 323-337.
 54. Richardson, Sean O., Mark B. Andersen, and Tony Morris. *Overtraining athletes: Personal journeys in sport*. Human Kinetics, 2008: 3- 56.
 55. Klarlund, and Hoffman-Goetz. "Exercise and the immune system." 1058-1076.
 56. Verde, T., S. Thomas, and R. J. Shephard. "Potential markers of heavy training in highly trained distance runners." *British Journal of Sports Medicine* 26.3 (1992): 167-175.
 57. Peters, E. M. "Exercise, immunology and upper respiratory tract infections." *International journal of sports medicine* 18.S 1 (1997): 69-77.
 58. Lakier. "Overtraining, excessive exercise, and altered immunity." 348-362.
 59. Licastro, Federico, et al. "Innate immunity and inflammation in ageing: a key for understanding age-related diseases." *Immunity & Ageing* 2.1 (2005): 8.

Nutritional Status and Body Composition of Elderly consultants in outpatient medicine in Casablanca – Morocco

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ABSTRACT

Objective: Senescence represents a public health disaster, which the nutritional profile is the main key to determine the status of seniors. The objective is the evaluation of the nutritional status and the body composition of the Moroccan elderly people of Casablanca. **Materials and Methods:** This is a cross-sectional survey of 62 people aged over 60 years, consults in ambulatory medicine in a public Moroccan hospital, from January to April 2015, in Casablanca we used questionnaire to determine sociodemographic characteristics and the anthropometric indicators. **Results:** The 62 subjects in the study reported a sex ratio of 0.5 and an average age of 71.08, Significant male and female varieties were recorded for lean and fat mass measurements. Negatively significant correlations were found between BMI and total cell water. The model of the multiple descending linear regression showed that fat mass is a factor associated with total body water (beta = -0.1, p = 0.05) in the elderly of our study. **Conclusion:** we found that the nutritional status and the body composition of the elderly consultants in outpatient medicine were associated.

Key words: Elderly, nutritional status, body composition.

INTRODUCTION

All over the world, the age of the elderly (old people OP) of 60 years and over is increasing, it is estimated that in 2050 the number will exceed 2 billion people across the planet. Morocco is no exception, according to the Moroccan Office of the Plan, the number of older persons increased from 8% in 2004 to 9.6% in 2014, as result the support of senescence need a new approach in terms of public health for the kingdom authorities (1,2,3).

It is proven that the nutrition occupies a major place in the cycle of life, especially among the elderly person. It is important to be aware that the situation of the health of the elderly person is in part affected by its nutritional profile because they suffer more of undernutrition, which is why there is a need to determine the nutritional status for either preventive or curative approach(4,5).

The evaluation of the nutritional status remains indispensable particularly among the OP with taking into account the main changes due to the age and repercussions of the general state of health and body composition in particular in the short and long-term(5,6,7).

In the light of these findings, there are only a few Moroccan studies in this subject; that is why we wanted to conduct a study to value the nutritional status of

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the elderly in Ambulatory medicine in a hospital in Casablanca, Morocco.

MATERIALS AND METHODS

This is an epidemiological inquiry cross on the nutritional status of the elderly consultant in Ambulatory medicine in a public hospital in the city of Casablanca conducted from January to April 2015.

Topics

A random 62 subjects, of men and women, aged 60 and over were selected from the database in the possession of the medical team of the service medicine at the hospital. This study has included elderly that not presenting any pathologies interfering with the body composition, such as, the presence of edema, the port of a pacemaker or the taking of diuretics. The subjects have formulated their informed consent for participation in the study informed of its goal and its conduct in accordance with the deceleration in Helsinki in 1964.

Data Collection

A questionnaire in face-to-face and a grid, previously tested and standardized, were used in this study to gather all of the data. The questionnaire focused on the sociodemographic characteristics of the patients, in particular, the age, sex, marital status and the education level, the grid allowed to identify the data relating to the measures of the anthropometric indicators namely the weight, size, the circumference of the calf, the brachial circumference and indicators of body composition including lean mass, fat mass, water, total cell, the intracellular water and extracellular water for each subject of the study.

Anthropometric Indicators

The measurement of anthropometric indicators have been carried out on two occasions by the same experienced investigator to minimize the variations intra-individual, with subjects heaved and wearing clothes light interiors. The measurement of the weight, size, of the brachial Circumference (BC) and the circumference of the Calf (CC) have been identified according to the standard techniques (WHO, 1995). The subject standing has been weighed on a scale Seca 761 to determine its weight in kg. Subsequently, the subject extends to determine its size which is calculated

from the distance heel-knee According to the formula of Chumlea(8) using a paediatric spacer Seca 207. The report of the weight on the size allowed to calculate the Body Mass Index(BMI) kg/m^2 . The values of the BMI were compared to the recommendations of the WHO(9).

$$\text{Height (Man)} = (2,02 \times \text{dHKcm}) - (0,04 \times \text{age}) + 64,19 \quad (1)$$

$$\text{Height (Women)} = (1,83 \times \text{dHKcm}) - (0,24 \times \text{age}) + 84,88 \quad (2)$$

dHK: distance heel-knee

Statistical Analysis

The quantitative variables were represented by the mean \pm standard deviation (ET) whereas the qualitative variables were represented by the absolute frequency (n) and relative (%). The univariate analysis has appealed to the test of Pearson for the calculation of the coefficient of correlation between the indicators of body composition and anthropometric indicators. Student's t-test was used to compare the difference of the averages of the variables of interest between the two sexes. The multiple linear regression descendant used to determine the factors associated with anthropometries indicators and body composition in our sample.

The results were statistically significant when the P value was less than 0.05 and the statistical treatment was carried out with the SPSS software 20.0.

RESULTS

Description of the Sample

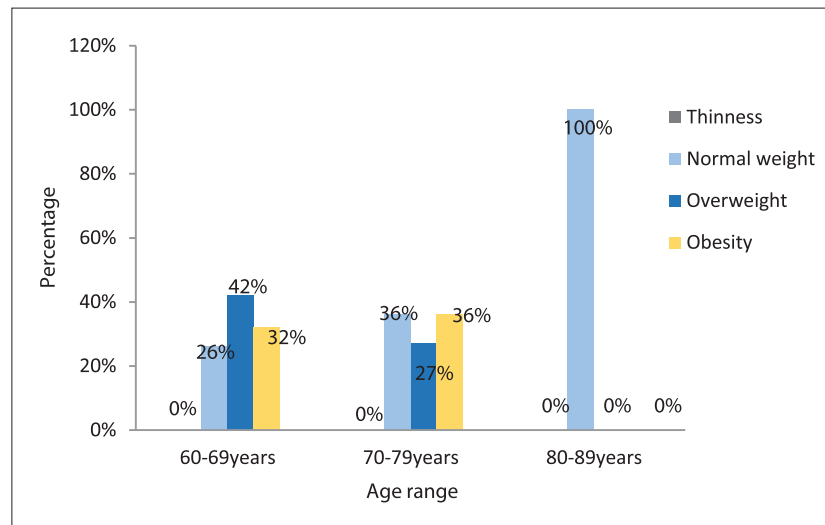
In this study, we had a total of 62 subjects, 31 men and 31 women, mean age, BMI and lean mass in respective 71.08 ± 6.5 ans, $26.2 \pm 4.8 \text{ kg/m}^2$ and $64.3 \pm 8.6 \text{ kg}$. The distribution of subjects in function of the age has been to 53.2 % (n=33) for the 60-69 years, 37.1% (n=23) for the 70-79 years, 9.7% (n=6) for 80-89 years.

Significant differences between the male and female subjects have been recorded in the measurement of indicators of body composition and anthropometric, including that of the lean mass ($p < 0.001$), fat mass ($p < 0.001$), total body water ($p < 0.001$) and of the BMI ($p < 0.001$). The whole of descriptive data of the sample is showed in the Table 1 (Table 1).

Table 1: General characteristics of elderly patients of the study

	Total (n=62)	Men (n=31)	Women (n=31)	P value
Marital status				
Married	54.8 (34)	77.4 (24)	32.3 (10)	<0.001
Not married	45.2 (28)	22.6 (7)	67.7 (21)	
Level of education				
Illiterate	77.4 (48)	64.5 (20)	90.3 (28)	0.49
Primary	17.7 (11)	29.0 (9)	6.5 (2)	
Secondary	4.8 (3)	6.5 (2)	3.2 (1)	
Middle of residence				
Urban	74.2 (46)	67.8 (21)	80.6 (25)	0.24
Rural	25.8 (16)	32.2 (10)	19.4 (6)	
BMI	26.2±4.8 (62)	24.6±4 (31)	27.4±5.0 (31)	0.02
Fat mass	34.9±8.7 (62)	28.3±5.9 (31)	41.5±5.6 (31)	<0.001
Lean mass	64.3±8.6 (62)	69.4±6.6 (31)	59.3±7.4 (31)	<0.001
Water total body	51.6±5.0 (62)	54.1±4.6 (31)	49.0±4.0 (31)	<0.001
Intracellular water	27.6±3.3 (62)	29.1±3.8 (31)	26.0±1.6 (31)	<0.001
Extracellular water	23.9±2.9 (62)	24.9±2.5 (31)	22.9±3.0 (31)	0.007
BC	27.2±3.8 (62)	26.7±4 (31)	27.2±3.2 (31)	0.3
CC	32.7±4.3 (62)	32.0±4.5 (31)	33.4±4.0 (31)	0.1

BMI : Body mass index ; BC : Brachial circumference; CC : Circumference of the calf

**Figure 1:** Distribution of BMI by age for women

BMI was used as an indicator of corpulence in the elderly, 1.6% (n = 1) in the sample represented a state of emaciation (BMI <16 kg/m²), 45.2% (n = 28) (25.0 kg/m² <BMI <29.9Kg/m²), and 24.2% (n = 15) were overweight (18.5kg/m² <body weight, body mass index (BMI) > = 30 kg/m²).

The distribution of BMI for both sexes showed that overweight and obesity affects more women 63%, especially the 70-79 age group, compared to men only 8% (Figures 1 and 2).

As well as, the repair of recommended values of indicators of body composition, including the fat mass, total body water, and of the extracellular water was different between the two sexes of the study (Table 2).

Correlation between Anthropometric Indicators and Body Composition Indicators

Negatively significant correlations were found between BMI and lean mass (r = -3.1, p = 0.01, between BMI and total cellular water (r = -0.3, r = 0.003 (R = -0.3,

$p = 0.002$) and positively significant, between BMI and fat mass ($r = 0.4, p = 0.001$), between BMI and extracellular water CB ($r = 0.6, p < 0.001$) and BMI and CM ($r = 0.7, p < 0.001$). ($R = 0.7, p < 0.001$), between ECT and lean mass ($r = 0.5, p < 0.001$), and negatively significant between ECT and BMI ($r = -0.3, p = 0.003$) and between ECT and fat mass ($r = -0.6, p < 0.001$).

Factors Associated with Body Composition

The quantitative and qualitative variables that demonstrated a significant correlation or difference in the univariate analysis were introduced in the model of the multiple descending linear regression to determine the factors associated with anthropometric indicators and body composition. Thus, 82% of body fat appears to be associated with total body water ($\beta = -0.1$) in elderly outpatient consultants ($p = 0.05$).

DISCUSSION

The anthropometric parameters of the subjects, including BMI, calf circumference and brachial circumference were greater than the values recommended by WHO. All subjects in the study were overweight with a BMI greater than 25 kg/m², which was higher in women with higher fat mass than the recommendations while MM and ECT were lower.

Table 2: Distribution of indicators of the body composition by sex

	Men (n=31)	Women (n=31)
Fat mass		
Normal	32.0 (10)	9.7 (3)
Superior to the standards	68.0 (21)	90.3 (28)
Lean mass		
Lower to the standards	90.3 (28)	90.3 (28)
Normal	9.7 (3)	6.5 (2)
Superior to the standards	0 (0)	3.2 (1)
Water total body		
Dehydration	93.5 (29)	58.1 (18)
Normal hydration	6.5 (2)	41.9 (13)
Intracellular water		
Dehydration	93.5 (29)	100 (31)
Normal hydration	6.5 (2)	0 (0)
Extracellular water		
Dehydration	71.0 (22)	29.0 (9)
Normal hydration	25.8 (8)	74.2 (23)

Our findings are consistent with those of a Moroccan study conducted in the same direction with PA, or 72.2% of women have a higher percentage of GP than normal, with MM and ECT lower than referential (7,10).

A high body mass profile with decreased lean mass in PAs is a predictive factor for muscle wasting, risk of sarcopenia and compromises the quality of life of subjects. This phenomenon highlights the profile of sarcopenic obesity affecting the elderly with significant weight status in our study (11,12,13).

The difference observed between the two sexes for body compartments and the anthropometric indicator goes hand in hand with the literature (14). A Swedish cohort found similar results in populations over 75 years of age (15). It appears that this difference in the anthropometric parameters between the two sexes can relate mainly to the anatomy and physiology of women and to the Moroccan culture where the elderly Moroccan woman are less active than men.

As far as the BC and CM measures seem to be similar between the sexes. But if certain authors have come to the same conclusion as ours, other authors have shown opposite results (16,17). We can hypothesize that a slight overweight tends not to influence the measures of the circumferences and does not affect them but studies need to be started in this direction.

Our results showed that the age group of 80 years and over registers the absence of an overweight or obesity BMI, we can assume that from this age onwards the main changes related to aging occur. This decrease in weight can be explained by the decrease in appetite and food intake with aging accompanied by an increase in metabolism without it being a corollary to an increase in dietary intakes as reported

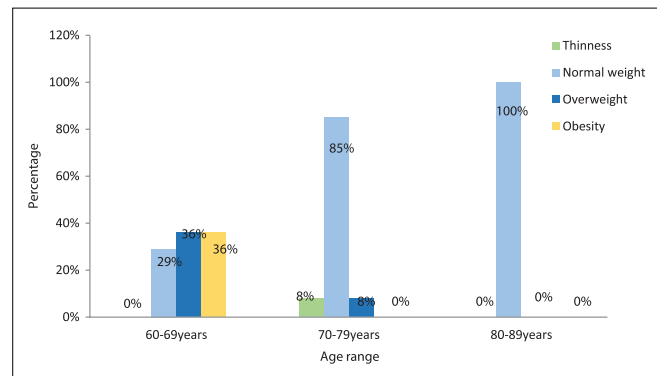


Figure 2: Distribution of BMI by age for men

by the Health ABC study (ABC = Aging and Body composition) (18,19).

Correlations were found between anthropometric indicators and body composition indicators, in this case, between BMI and MG, MM and ECT. Such relationships have been found in a study of 79 Moroccan elderly of both sexes (20,21).

Factors associated with body composition and anthropometric indicators indicated that MG is associated with ECT. It appears that the decrease in water induces an increase in fat main factor involved in comorbidity (20,21).

CONCLUSION

At the end of this study, the findings concerning the association between anthropometric indicators and body composition are an increased fat mass, a decreased lean mass and risk of dehydration. Thus, our study highlighted the importance of the evaluation of the nutritional status of the elderly consultant in ambulatory medicine. Longitudinal studies should be initiated to demonstrate a link between these two parameters, in order to limit all risks of late repercussions.

REFERENCES

- World Health Organization(WHO). World Report on Ageing and Health; 2015, p 3-233. <http://www.who.int/ageing/events/world-report-2015-launch/en>.
- Sajoux M, Nowik L. Vieillesse de la population au Maroc: Réalités d'une métamorphose démographique et sources de vulnérabilité des aîné(e)s. *Autrepart*.2005; 53(1):17-34. <http://www.cairn.info/revue-autrepart-2010-1-page-17.htm>.
- Centre d'Etude et de Recherches Démographiques (CRED). Recensement Général de la Population et de l'Habitat. 2014. http://www.hcp.ma/Presentation-des-premiers-resultats-du-RGPH-2014_a1605.html.
- Marion T. Statut nutritionnel de la personne âgée vivant à domicile: prévalence, facteurs associés et conséquences. *Santé publique et épidémiologie*. Université de Bordeaux; 2014. <https://tel.archives-ouvertes.fr/tel-01249581>.
- Fanello S, Faucoult S, Delbos V, Jousset N. Assessment of Nutritional State of Elderly in the hospital. *SantePublique*.31Janv2000;12(1):83-90. http://fulltext.bdsp.chesp.fr/Sfsp/SantePublique/2000/1/IMP_FANELLO_ps.pdf.
- Donini L.M, Savina C, Rosano A, Cannella C. Systematic Review Of Nutritional Status Evaluation And Screening Tools In The Elderly. *The Journal of Nutrition, Health & Aging*. 2007; 11(5):421-32. <https://search.proquest.com/openview/8e6f8fa633e24b4a79816de78315a012/1?pq-origsite=gscholar&cbl=28850>.
- Bour A, Hsaini HE, Kari KE, Janah K, Belghiti H, Cha-hid N, et al. P046 Mesure de la composition corporelle chez les personnes âgées marocaines par les méthodes des isotopes stables et d'impédancemétrie. *Nutr Clin Métabolisme*. 1 nov 2007;21:68-9. [https://doi.org/10.1016/S0985-0562\(07\)78848-9](https://doi.org/10.1016/S0985-0562(07)78848-9).
- Chumlea W.C, Roche A.F, Steinbaugh M.L. Estimating stature from knee height for persons 60 to 90 years of age. *J Am Geriatr Soc*.1985;33(2):116-20. <https://www.ncbi.nlm.nih.gov/pubmed/3968366>.
- World Health Organization (WHO). Physical status: The use and interpretation of anthropometry. Report No.: 854; 1995.p. 312-463. http://apps.who.int/iris/bitstream/10665/37003/1/WHO_TRS_854.pdf.
- Mathus-Vliegen M.H. Obesity and the elderly. *J Clin Gastroenterol*. août 2012;46(7):533-44. doi: 10.1097/MCG.0b013e31825692ce.
- Tengvall M, Ellegård L, Malmros V, Bosaeus N, Lissner L, Bosaeus I. Body composition in the elderly: reference values and bioelectrical impedance spectroscopy to predict total body skeletal muscle mass. *Clin Nutr*. févr 2009;28(1):52-8. doi: 10.1016/j.clnu.2008.10.005.
- Chang C.I, Huang K.C, Chan D.C, Wu C.H, Lin C.C, Hsiung C.A, and al. The impacts of sarcopenia and obesity on physical performance in the elderly. *Obesity Research & Clinical Practice*. 2015;9(3):256-65. doi: 10.1016/j.orcp.2014.08.003.
- Cetin D.C, Nasr G. Obesity in the elderly: more complicated than you think. *Cleveland Clinic Journal of Medicine*. 2014 Jan;81(1):51-61. doi: 10.3949/ccjm.81a.12165.
- Aloia J.F, Vaswani A, Flaster E, Ruimei Ma. Relationship of body water compartments to age, race, and fat-free mass. *J Lab Clin Med*. déc 1998;132(6):483-90. DOI: [http://dx.doi.org/10.1016/S0022-2143\(98\)90126-3](http://dx.doi.org/10.1016/S0022-2143(98)90126-3).
- Woo J, Kwok T, Lau E, Li M, Yu L.M. Body composition in Chinese subjects: relationship with age and disease. *Arch Gerontol Geriatr*. févr 1998;26(1):23-32. [https://doi.org/10.1016/S0167-4943\(97\)00026-5](https://doi.org/10.1016/S0167-4943(97)00026-5).
- Chaffal P. Composition corporelle et force musculaire des personnes de plus de 65ans en médecine générale. *Faculté de Médecine: Université Claud Bernard- Lyon1; 2014, N°45*. <http://portaildoc.univ-lyon1.fr>.
- Kilic M.K, Kizilarlanoglu M.C, Arik G, Bolayir B, Kara O, Dogan Varan H, and al. Association of Bioelectrical Impedance Analysis-Derived Phase Angle and Sarcopenia in Older Adults. *Nutr Clin Pract*. 2017 Feb;32(1):103-109. doi: 10.1177/0884533616664503
- Goodpaster B.H, Park S.W, Harris T.B, Kritchevsky S.B, Nevitt M, Schwartz A.V, et al. The loss of skeletal muscle strength, mass, and quality in older adults: the health, aging and body composition study. *J Gerontol A Biol Sci Med Sci*. oct 2006;61(10):1059-1064. DOI: <https://doi.org/10.1093/gerona/61.10.1059>.
- Sternfeld B, Ngo L, Satariano W.A, Tager I.B. Associations of body composition with physical performance and self-reported functional limitation in elderly men and women. *Am J Epidemiol*. 15 juill 2002;156(2):110-121. DOI: <https://doi.org/10.1093/aje/kwf023>.
- Montagnani M, Montomoli M, Mulinari M, Guzzo G, Scopetani N, Gennari C. Relevance of hydration state of the fat free mass in estimating fat mass by body impedance analysis. *Applied Radiation and Isotopes*.1998;49(5):499-500. [https://doi.org/10.1016/S0969-8043\(97\)00180-2](https://doi.org/10.1016/S0969-8043(97)00180-2).
- Visser M, Harris T.B, Langlois J, Hannan M.T, Roubenoff R, Felson D.T, et al. Body fat and skeletal muscle mass in relation to physical disability in very old men and women of the Framingham Heart Study. *J Gerontol A Biol Sci Med Sci*. mai 1998;53(3):M214-M221. DOI: <https://doi.org/10.1093/gerona/53A.3.M214>.