International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2015): 78.96 | Impact Factor (2015): 6.391

Exercise and Chronic Diseases

Oloo Micky Olutende(B.Sc.) [1], Issah W. Kweyu (M.SC.) [2], Edna Sabiri (B.Sc.)[3]

[1, 2, 3]Dept. of Health Promotion and Sport Science, School of Public Health, Biomedical Sciences and laboratory Technology. Masinde Muliro University of Science and Technology, Kakamega, Kenya

Abstract: ***Physical inactivity(PA) is fourth on the list of leading causes of deaths worldwide. An increased level of PA and exercise will not only reduce the risk for developing various chronic diseases, but also have a positive influence on morbidity and mortality resulting from these chronic diseases. While overwhelming success have been achieved in disease control through environmental interventions such as vaccinations and improved hygiene to increase life expectancy, many authorities in the field of preventative healthcare are of the opinion that too little has been done to target behavioral factors, particularly physical inactivity. PA promotion is one of the first treatment and management recommendations in a large number of ever-increasing evidence based clinical guidelines for many chronic conditions.***

Keywords: Exercise, chronic diseases prevention, health promotion, primary and secondary prevention, exercise is medicine

1. Introduction

The negative health effects of physical inactivity are well documented, the economic consequences are often neglected. As the population ages, chronic illnesses have become a common occurrence, putting pressure on the sustainability of healthcare systems as chronic diseases account for most of global healthcare expense (Huber et al., 2011). Physical inactivity was directly responsible for 3% of disability adjusted life years lost in the United Kingdom in 2002, with estimated direct costs to the National Health Service of £1.06 billion (Allender, Foster, Scarborough et al., 2007). In America, the annual cost directly attributable to inactivity is an estimated \$24 billion–\$76 billion (2.4%–5.0% of national healthcare expenditures) (Roux, Pratt, Tengs et al., 2008). In Canada, it is estimated that even modest reductions in inactivity levels could result in substantial cost savings. A 10% reduction in the prevalence of physical inactivity has the potential to reduce direct health care expenditures by \$150 million a year (Katzmarzyk et al., 2000). Over the past few decades, extensive knowledge has been accumulated relating to the significant contribution of PA in the prevention and treatment of a number of diseases (Chakravarthy et al., 2002; Leijon, Bendtsen, Nilsen et al., 2008), especially noncommunicable chronic diseases. A linear relationship exists between PA levels and overall health status (Sallis, 2009), evidenced by the strong links between increased levels of PA and aerobic fitness with a reduction in the risk for developing various chronic diseases, as well as the morbidity and mortality resulting from these chronic diseases (Oberg, 2007; Pedersen and Saltin, 2006). Increasing PA is now considered to be as important as tobacco control in lessening the burden of non-communicable diseases (Bauman et al., 2006; WHO, 2014). Conditions such as cardiovascular disease, type 2 diabetes, obesity, and cancer are drastically improved when PA and exercise are part of a medical management plan. Exercise provides many primary prevention health benefits, exercise also provides similar benefits in secondary disease prevention. When PA and exercise are initiated after a chronic disease is diagnosed, many of the harmful disease effects are ameliorated and in some cases (e.g., type 2 diabetes) the disease progression is slowed or halted (Sigal, 2006) exercise when used as part of the medical management plan for secondary disease prevention will almost always improve the quality of life and potentially extend the life of a disease individual (Durstine, 2000).In this regard, the benefits of PA and exercise depend on the type, severity, and comorbidities of the disease.

2. Methods

A comprehensive literature search was carried out for in the Pubmed, Crossref ,Genamics Journal Seek ,Global impact factor.com , Google Scholar , Academic keys, Open Academic Journals Index, Sherpa/RoMEO (University of Nottingham), Chemical Abstracts (CAS), Open-j-Gate ,Cochrane Library and MEDLINE databases (search descriptors: exercise therapy, training, physical fitness, physical activity, rehabilitation ,exercise is medicine; Chronic disease; Primary prevention; Secondary prevention). In addition, we sought literature by examining reference lists in original articles and reviews.

3. Exercise and Diabetes

The prevalence of Type 2 Diabetes Mellitus (T2DM) and pre-diabetic conditions such as impaired fasting glucose and impaired glucose tolerance are rapidly on the rise (Hordern, Dunstan, Prins et al., 2012). An estimated one million deaths which occurred during 2002 could be attributed to diabetes (LaMonte, Blair and Church, 2005). In 2009, it was estimated that the world prevalence of diabetes among adults (aged 20– 79 years) will be 6.4%, affecting 285 million adults in 2010 and will increase to 7.7% (439 million adults) by 2030 (Shaw, Sicree, and Zimmet, 2010). These figures were revised and a 69% increase in numbers of adults with diabetes in developing countries and a 20% increase in developed countries were postulated for the time period from 2011 to 2030 (Whiting, Guariguata, Weil et al., 2011).

A sedentary lifestyle is directly associated with the development of T2DM (Blair, 1989; Kokkinos and Myers, 2010), while moderate to vigorous PA can reduce onset rates of diabetes by between 6%-48% (Bassuk and Manson, 2005; Helmrich, Ragland, Leung et al., 1991; Sieverdes, Sui, Lee et al., 2010). Exercise plays a leading role in the prevention and control of insulin resistance, pre-diabetes, gestational diabetes mellitus, T2DM, and diabetes-related health complications (Colberg and Sigal, 2010; Hordern et al., 2012). The most important way by which PA decrease the threat for diabetes is by improvement in insulin sensitivity (Roberts and Barnard, 2005). As exercise is an insulin-independent stimulus for increased glucose uptake by the working muscle cells via the Glucose Transporter Type 4 transporter, both aerobic training and resistance training improve insulin action (Colberg and Sigal, 2010), and is considered one of the three cornerstones of treatment for diabetes mellitus (Woodard and Berry, 2001).

According to Colberg and Sigal (2010), an exercise prescription program for persons with diabetes should be individualised according to medication schedule, presence and severity of complications, and goals and expected benefits of the program (Colberg and Sigal, 2010). The majority of patients with T2DM can exercise without taking special precautions. Nonetheless, it is necessary that patients being treated with medication such as sulfonylurea, postprandial regulators or insulin are educated on precautions on how to avoid hypoglycaemia.

Precautions to avoid hypoglycaemia include blood glucose monitoring, adjustment of the insulin dose and dietary modification (Pedersen and Saltin, 2006). Patients with autonomic neuropathy should be carefully monitored since the absence of ischemic symptoms pose a risk for sudden cardiac death (Pedersen and Saltin, 2006) and silent myocardial ischemia (Boulton, Vinik, Arezzo et al., 2005). Similarly, patients with peripheral neuropathy should be educated to monitor for foot blisters or ulcers following exercise since neglect may result in complications leading to amputation (Mayfield, Reiber, Sanders et al., 1998). It is therefore recommended that, due to the high prevalence and incidence of comorbid conditions in diabetic patients, pre-exercise testing should precede training programs (Boulton et al., 2005), and preferably written and managed by individuals with appropriate qualifications and experience (Hordern et al., 2012).

Persons with no significant complications or limitations should follow exercise programs which accumulate to a minimum of 210 min per week of moderate-intensity exercise or 125 min per week of vigorous intensity exercise with no more than two consecutive days without training, as well as two or more resistance training sessions per week (Hordern et al., 2012).

4. Exercise and Hypertension

Results from the South African National Health and Nutrition Examination Survey (SANHANES-1) demonstrated that almost three quarters of South African adults over 50 years are hypertensive (Shisana, Labadarios, Rehle et al., 2013). Hypertension is defined as a systolic blood pressure (BP) above 140mmHg and/or a diastolic BP >90mmHg (Pedersen and Saltin, 2006). It is a risk factor for acute myocardial infarction, cardiac insufficiency and sudden death, while being considered as the single most important risk factor for strokes in SA (Connor, Rheeder, Bryer et al., 2005).

While cardiorespiratory and resistance training has both been shown to be effective in the prevention of hypertension, cardiovascular exercise training is the most effective type of exercise to prevent and treat hypertension (Wallace, 2003). During exercise, systolic BP may rise from baseline, but following 30 to 45 minutes of moderate exercise, a 10 to 20 mmHg decrease in systolic BP are noted which may last for up to ten hours (MacDonald, 2002). Although studies carried out in subjects with cardiovascular diseases or risk factors reported that decreases in cardiovascular risk associated with exercise training are connected to an up-regulation of endothelium mediated vasodilator function together with an increase in arterial compliance (Green, Spence, Halliwill et al., 2011), further research has shown that the prolonged effect of exercise on the lowering of systolic BP can be attributed to a transient decrease in stroke volume rather than peripheral vasodilatation (Fletcher, Ades, Kligfield et al., 2013).

An increase in exercise capacity is also linked to a lower mortality in hypertensive persons, with a greater risk reduction in younger individuals (18% reduction) compared to older persons (12% reduction) (Kokkinos and Myers, 2010).

5. Exercise and Cancer

Worldwide there is a progressive increase in the prevalence of cancer (Newton and Galvão, 2008). Nearly 40 000 cancer-related deaths are reported annually in SA (Mayosi et al., 2009). With the exception of non-melanoma skin cancers, an estimated 3.45 million new cases of cancer and 1.75 million deaths from cancer were reported in Europe in 2012 (Ferlay, Steliarova-Foucher, Lortet-Tieulent et al., 2013). The most common cancer sites were cancers of the female breast, followed by colorectal, prostate and lung cancer (Ferlay et al., 2013).

An increase in PA has been documented to be associated with reduced risk of developing several forms of cancer (Roberts and Barnard, 2005; Shann, 2000). The mechanism of protection is thought to be due to the favourable effect of PA on a person's antioxidant capacity (Franzoni, Ghiadoni, Galetta et al., 2005) and consequent ability to scavenge free radicals which have a carcinogenic affect (Dreher and Junod, 1996). This protective effect of PA has been observed over different populations with the potential to reduce the incidence of cancer by 40% (Newton and Galvão, 2008) while being most consistent for breast and colon cancer (Kruk, 2007; Shann, 2000).

PA increases bowel transit time and thus decreasing the duration of contact between faecal carcinogens and colonic mucosa (Shann, 2000) to prevent colon cancer. Furthermore, increased habitual PA modulate the production, metabolism and excretion of sex hormones implicated in the development of breast and endometrial cancer (Shann, 2000) as seen in the significant decrease of salivary estradiol levels with regular PA (Jasienska and Ziomkiewicz, 2006). PA therefore curbs the cancerous effects of certain hormones (Roberts and Barnard, 2005; Shann, 2000) such as estradiol which is a risk factor for developing breast cancer (Kruk, 2007) and testosterone which is a risk factor for developing prostate cancer (Shann, 2000). Even after cancer has been diagnosed, the survival rate of breast and colon cancer increase by 50–60% when engaging in regular PA (Shann, 2000), highlighting not only the preventative but also the therapeutic effect of PA on cancer.

Treatments for cancer include surgery as well as systemic and radiation therapy. However, these treatments compromise the physical function and quality of life of patients (Newton and Galvão, 2008). For many cancer patients' chemotherapy or hormone therapy results not only in muscle loss (general cachexia), but also bone mineral loss as a result of reduced PA (Newton and Galvão, 2008).Thus, there is a growing interest in the use of exercise in the treatment and rehabilitation of patients with cancer (Pedersen and Saltin, 2006; Shann, 2000). Aerobic and resistance exercise programs for cancer patients improve balance and bone remodelling while simultaneously reducing muscle weakness and wasting (Galvão, Taaffe, Spry et al., 2010). This result in reduced levels of fatigue, greater selfconfidence, maintenance of body weight, improved mood, less side effect severity, improved aerobic capacity, and a higher quality of life (Pedersen and Saltin, 2006).

6. Exercise and Cardiovascular Disease

Cardiovascular diseases (CVD) include high BP (hypertension), coronary heart disease, stroke, rheumatic heart disease and other forms of heart disease (Kruk, 2007). Physical inactivity, smoking, poor diet and neglect of chronic life-stress play key roles in the pathogenesis of CVD (Derman et al., 2008a). Atherosclerotic CVD is caused by the accumulation of lipids, macrophages, blood-clotting elements, calcium and fibrous connective tissue within the inner layer of arteries, resulting in endothelial dysfunction and vascular inflammation (Pearson, 2003a). These atherosclerotic changes later result in the formation of atheromatous plaques or lesions which may cause obstruction of normal blood flow (Pearson, 2003a). Dyslipidemia or high cholesterol level remains a major cardiovascular risk factor in SA, as familial hypercholesterolaemia occurs in one out of every 200 Afrikaners which may cause early CVD in affected individuals (Mayosi et al., 2009). From the SANHANES-1 project, it was found that one out of five males 15–65 years of age and older had abnormally high serum total- and low-density lipoprotein (LDL) cholesterol. In the South African females, the figures were even worse, where abnormal lipid concentrations were found in almost one out of three females 15–65 years of age (Shisana et al., 2013). Regular PA results in beneficial changes in persons with normal lipid and lipoprotein concentrations as well as in most persons with dyslipidemia (Kelley and Kelley, 2008; Kelley, Kelley, Roberts et al., 2011; Murphy, Blair and Murtagh, 2009).

Regular PA and exercise not only plays a substantial role in the primary prevention of CVD, but also in the secondary prevention and rehabilitation of patients with known CVD (Thompson, 2005; Thompson, Franklin, Balady et al., 2007). The purpose of PA in the form ofexercise training is to reduce physiological limitations and increase exercise capacity through specific exercise therapy (Derman, Whitesman, Dreyer et al., 2008b). Health benefits associated with regular exercise and increased exercise capacities include, among others, the following (Derman et al., 2008a; Giannuzzi, Saner, Björnstad et al., 2003; Leon, Franklin, Costa et al., 2005; Miles, 2007; Mora, Cook, Buring et al., 2007; Thompson et al., 2007): Reduced number of cardiovascular events ,Improvement in blood lipid concentrations ,Reduced systolic and diastolic BP ,Increased fibrinolysis, Reduced thrombocyte aggregation ,Reduced endothelial dysfunction of the coronary arteries ,Increased autonomic tone and heart rate variability ,Decreased cardiac arrhythmias, Improvement of insulin resistance and glucose intolerance, Improved psychosocial factors ,Improved lifestyle choices, Reduced obesity ,Reduced smoking habit ,Improved functional (exercise and work) capacity ,reduced hospitalization and decreased morbidity and total mortality.

Notwithstanding these benefits, exercise training is hardly ever prescribed for patients with CVD (Thompson, 2005). Some authors hypothesize that the complexity of prescribing exercise for patients with chronic disease may be a contributing factor to this tendency since the patient should not only be advised which exercise therapies to use, but the exercise goals should be defined, a suitable training intensity, duration and frequency chosen for the appropriate stage of the medical condition (Derman et al., 2008b).

Preceding regular exercise training all patients should be assessed by a cardiologist or medical practitioner skilled in exercise testing and prescription to have a baseline symptom-limited exercise test and exclude any contraindications before an exercise programme is initiated (Derman et al., 2008b). This will also detect important clinical signs such as a cardiac murmur, ischemia symptoms or arrhythmia that would alter the therapeutic approach, gallop sounds, pulmonary "wheezing," or pulmonary crepitation (Thompson, 2005). Patients with a history of worsening unstable angina or decompensated heart failure should not undergo exercise testing until their condition stabilizes (Fletcher, Balady, Amsterdam et al., 2001).

After an exercise stress test, the patient should be classified as either a high, moderate or low risk patients. For high and moderate risk patients (for example those with exercise-induced myocardial ischaemia with possible ST-segment depression and/or angina pectoris and those with left ventricular ejection fraction <30%, arrhythmia, clinical depression, low exercise tolerance or those patients unaccustomed to exercise) there should be medical supervision, as these patients should be monitored more closely and frequently than low- risk patients. Risk stratification should be repeated at 3 month intervals (Derman et al., 2008b; Thompson, 2005).

Family doctors have a key role as the central coordinating figures in promoting cardiovascular wellness through routine provision of and/or information on primary and secondary preventive services; identifying patients who would gain from a structured cardiac rehabilitation program and helping them to engage in these programmes, encouraging existing patients to complete the program and providing longitudinal follow-up for patients after program completion (Stephens, 2009). If a local cardiac rehabilitation program is not available, doctors should refer patients to physical therapy or fitness facilities or should advise the patient on how to start an exercise program. This necessitates doctors to design an exercise program for the patient (Thompson, 2005). When developing an individualized exercise prescription for aerobic and resistance training, take into consideration evaluation of exercise stress test findings, risk stratification, comorbidities (e.g., peripheral arterial disease and musculoskeletal conditions), in addition to patient and program goals (Balady, Williams, Ades et al., 2007). Besides PA, cardiac rehabilitation programs must also address diet, emotional, medication, and smoking cessation issues (Giannuzzi et al., 2003; Thompson, 2005).

7. Exercise and Stroke

Early definitions of a stroke stated it to be an event characterised by rapidly developing clinical symptoms and signs of focal, and at times global, loss of cerebral function lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin (Hatano, 1976). More recently, a statement by the American Stroke Association (Sacco, Kasner, Broderick et al., 2013) placed emphasis on the fact that a stroke is, in fact, a central nervous system infarction and defined it as an infarction of the brain, spinal cord, or retinal cell death attributable to ischemia, based on:

1) Pathological, imaging, or other objective evidence of cerebral, spinal cord, or retinal focal ischemic injury in a defined vascular distribution; or
2) Clinical evidence of cerebral, spinal cord, or retinal focal ischemic injury based on symptoms persisting ≥24 hours or until death, and other aetiologies excluded.

This include ischemic stroke, silent central nervous system infarction, stroke caused by intracerebral haemorrhage, silent cerebral haemorrhage, subarachnoid haemorrhage, stroke caused by cerebral venous thrombosis, and a stroke not otherwise specified (Sacco et al., 2013).

In addition to the high mortality associated with strokes (the second most common cause of death worldwide; Mathers and Loncar, 2005), strokes also have a high morbidity, leaving up to 50% of survivors chronically disabled (Lloyd-Jones, Adams , Brown et al., 2010). The cornerstone of the prevention of recurrent stroke and acute cardiac events in stroke survivors is the modification of multiple risk factors through a combination of comprehensive lifestyle interventions and appropriate pharmacological therapy (Gordon, Gulanick, Costa et al., 2004). Therefore, regular PA is recommended for both primary and secondary prevention of stroke (Lloyd-Jones et al., 2010) evidenced by the fact that moderately to highly active individuals have a lower risk of both ischemic and hemorrhagic strokes compared to individuals with inadequate PA levels (Lee, Folsom and Blair, 2003). The goals for exercise training in stroke survivors are (Gordon et al., 2004): Increased independence in ADLs, Increased walking speed/efficiency, Improved tolerance for prolonged PA ,Reduced risk of cardiovascular disease, Improved level of safety during ADLs, Increase range of motion (ROM) of involved extremities.

8. Exercise and Obesity

Thirty years ago the world were fixed on juvenile malnutrition, the 'protein gap' and how to feed the world's rapidly increasing population (James and Leach, 2001; Prentice, 2006). At present the WHO finds itself needing to deal with a different pandemic, namely obesity and its associated non-communicable diseases (Caballero, 2007; James and Leach, 2001). Meanwhile the challenge of juvenile malnutrition has not disappeared, thus creating a 'double burden' of disease that threatens to overwhelm the health services of many resource-poor countries (Prentice, 2006). According to the WHO, the criteria for obesity is defined as a body mass index (BMI) of >30 kg/m^2 and overweight a BMI of 25 – 29.9 (Alwan, 2011). Cardiovascular risks increase with higher degrees of obesity (Apovian, 2010). Physical inactivity is considered to be an important underlying reason for obesity (Kokkinos and Myers, 2010), while obesity in turn intensifies cardiovascular disease risk stratification through its indirect adverse effects on numerous recognised risk factors such as insulin resistance and hypertension (Khan, 2008; Lee, Sui and Blair, 2009). Furthermore, there is a strong correlation between high BMI and the development of several other non-communicable diseases, including site-specific cancers such as colon and prostate cancer in men, and breast, endometrial, cervical, and ovarian cancer in women (Kruk, 2007).

Historically, obesity has been regarded as a 'Western' problem associated with prosperity, but is of rising significance in low income countries (Hawkes, 2006; Zimmet, 2000). Unfortunately, the same holds true for the South African population. Globalisation is causing the increase in consumption of foods high in fats and sweeteners throughout the developing world. This "nutrition transition" as well as physical inactivity are considered to be important underlying reasons for obesity (Kokkinos and Myers, 2010) and is associated with the rapid rise of obesity and dietrelated chronic diseases worldwide (Hawkes, 2006). In the 1998 SA Demographic and Health Survey high levels of excess body mass were observed among South Africans, particularly women. It was found that more than 29% of men and 56% of women were classified as overweight (BMI >25) or obese (BMI >30) (Joubert et al., 2007). The South African National Health and Nutrition Examination Survey (Shisana et al., 2013) showed a deterioration of the nutritional status of adult males and predominantly females based on various anthropometric measures. It was also found that major changes across all BMI groups occurred; the percentage of persons regarded as being underweight or normal weight decreased, while individuals considered being overweight or obese increased (Shisana et al., 2013). Overall, Mean BMI increased across all age categories, provinces, and race groups, but specifically among females (Shisana et al., 2013). It is suspected that the low levels of PA among South African adults reported in the past (Joubert et al., 2007; Kruger et al., 2002) contributed to the reported excess body mass, although education status also has to be taken into consideration since research has shown a sharp rise in the incidence of obesity among people who seem to be better educated and financially more privileged than the general South African population (Senekal, Steyn and Nel, 2003).Research has shown that a high-volume-high-intensity exercise regimen had the greater beneficial effects on body weight, fat mass and central obesity than a low amount of exercise (Slentz, 2004). This approach will also aid in the preservation or increase of lean muscle mass which will alter body shape, even in the absence of dieting. Contrary to traditional beliefs, exercise on its own has a limited effect on weight loss (Franz, VanWormer, Crain et al., 2007; Macfarlane and Thomas, 2010). Instead, it has been shown that exercise in combination with a nutritional plan is the most effective approach for sustainable weight loss (Macfarlane and Thomas, 2010) due to the combined effect to create a positive caloric imbalance needed for weight loss. Although exercise is most effective for weight loss when used in conjunction with a nutritional plan, it is still more effective than diet on its own (Macfarlane and Thomas, 2010) and a key component for avoiding primary weight gain (Pedersen and Saltin, 2006). Probably the most noteworthy effect that an obese person engaging in regular PA will benefit from, is the decrease in risk factors for comorbidity problems such as diabetes (Pan, Li, Hu et al., 1997), together with the positive psychological effects associated with regular PA (King, Hopkins, Caudwell et al., 2009; Lee et al., 2009).

In Africa and SA the fight against obesity is complicated. An overweight body type very often has positive connotations within the black South African community, symbolising wealth and status (Mvo, Dick and Seyn, 1999). Obesity is therefore widely accepted and an avenue to a high level of body satisfaction among many middle-aged South African women (Prentice, 2006; Van der Merwe and Pepper, 2006). In contrast, being “thin” or of normal body weight according to WHO standards (Alwan, 2011) often has a negative associated with HIV/AIDS (Human Immunodeficiency Virus/Acquired Human Deficiency Syndrome) status which further accentuate the positive attitudes noted towards obesity among Africans (Clark, Niccolai, Kissinger et al., 1999; Prentice, 2006).

9. Exercise and Chronic Respiratory Disease

One of the major chronic respiratory diseases in SA is chronic obstructive pulmonary disease (COPD) (Mayosi et al., 2009). COPD is characterised by a poorly reversible airflow limitation that is usually progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases, particularly cigarette smoke (Fabbri and Rabe, 2007; Pauwels, Buist, Calverley, et al. 2001). While COPD affects the lungs, it also causes substantial systemic consequences such as weight loss, nutritional abnormalities and skeletal muscle dysfunction (Celli, 2008). Data released by Statistics SA showed that premature adult deaths caused by COPD increased by 24% from 1999-2003 (Mayosi et al., 2009). Unfortunately, research suggests that COPD is often under diagnosed (Garcia-Aymerich, Barreiro, Farrero et al., 2000; Rutschmann, Janssens, Vermeuen et al., 2004), raising concerns that the prevalence of COPD may be even higher than documented.

For persons with COPD, strong linear associations exist between their exercise capacity and their health-related quality of life (Puhan, Siebeling, Zoller et al., 2013). Furthermore, exercise capacity is one of the strongest predictors of mortality and showed reliably stronger associations than either lung function or dyspnoea (Puhan et al., 2013). As the disease progress, gas exchange becomes compromised and patients may develop respiratory failure (Puhan et al., 2013). As soon as dyspnoea develops, it occurs at even lower levels of exercise (Celli, 2008). In fact, the BODE index (body mass index, forced expiratory volume in one second (FEV1), dyspnoea and 6-minute walk distance) includes exercise capacity to predict mortality (Puhan, Mador, Held et al., 2008; Puhan et al., 2013). However, COPD is a multidimensional disease. Many patients with COPD may have decreased fat-free mass, impaired systemic muscle function, anaemia, osteoporosis, depression, pulmonary hypertension, and cor pulmonale, all of which are important elements of the effect COPD may have on patients (Celli, 2008). The objectives of management of COPD are prevention of further deterioration in lung function, improvement of symptoms (coughing, sputum production and dyspnoea) and quality of life, treatment of complications, and to prolong a meaningful life (Celli, 2008).

One of the most important advances in the therapy of COPD is the capability to influence the disease without having to automatically modify lung function. Pulmonary rehabilitation and oxygen therapy are established forms of treatment for COPD (Celli, 2008). Pulmonary rehabilitation comprises of a combination of holistic interventions on the respiratory system such as smoking cessation, psychological support to help with coping strategies, and physical activity in the form of exercise training (Spruit, Troosters, Trappenberg et al., 2004). Physical exercise improves exercise capacity and health related quality of life and is a cost effective intervention (Puhan, Schünemann, Frey et al., 2005; Reardon, Mckenna and Riddoch, 2005). Although exercise training does not improve lung function, it does ease other symptoms of COPD such as dyspnea, fatigue and anxiety (Casaburi and ZuWallack, 2009; Spruit et al., 2004). Because of increasing skeletal muscle dysfunction in advanced stages of COPD (Franssen, Broekhuizen, Janssen et al., 2004), exercise training has become the core component of pulmonary rehabilitation (Lacasse and Goldstein, 2006; Reardon et al., 2005; Spruit, Singh, Garvey et al., 2013).

The type of exercise training for pulmonary rehabilitation follows the FITT (frequency, intensity, type, time) principle and includes: High-intensity regimens which are generally the preferred type of exercise, although lower-intensity exercise is also beneficial (Gosselink, 2002; Puhan, sching, ch nemann et al., 2006), with exercise of the leg muscles as the focal point of endurance exercise (such as walking, stationary cycling, and treadmill exercise). Under the observation of rehabilitation staff the intensity of the exercise may be increased as tolerated by the patients. Resistance training that includes the upper arms aids the ability to carry out the activities of daily living and because some of the upper-arm muscles also function as auxiliary muscles of respiration, it is often advised (Casaburi & ZuWallack, 2009; Puhan et al., 2005; Spruit et al., 2013).

Commonly contraindications for pulmonary exercise rehabilitation includes the inability to walk, either due to orthopaedic or neurological disorders, unstable cardiac disease including unstable angina or recent myocardial infarction and psychiatric or cognitive problems that would prevent the patient from understanding what is required or cooperating with the exercise prescription plan (Casaburi and ZuWallack, 2009; Spruit et al., 2013). Mild to moderate cases of COPD can be managed by a GP (Chavannes, Vollenberg, van Schayck et al., 2002) with specific attention given to the PA habits of persons with COPD who visit their general practice (Chavannes et al., 2002).

10. Benefits of Exercise on other Conditions

Exercise and osteoporosis

Osteoporosis is a disorder characterised by a decrease in bone mineral density caused by inadequate bone development during growth, excessive bone loss, failure to replace bone loss and an imbalance between osteoblast and osteoclast functioning, all of which ultimately lead to microarchitectural deterioration of the skeletal structure (Raisz, 2005). Osteoporosis poses a major risk for sustaining skeletal fractures due to this decrease in bone mineral density and compromised bone architecture (Howe, Shea, Dawson et al., 2011; Kai, Anderson and Lau, 2003; Kruk, 2007). Hip fracture is the most serious complication of osteoporosis resulting in an overall negative impact on the life of patients due to the increased risk for mortality, long term disability and loss of independence (Korpelainen, Keinänen-Kiukaanniemi, Heikkinen, et al., 2006).

The incidence of osteoporosis in the US is estimated to increase to over 14 million people in 2020 (Burge, 2007). During 2005, the direct medical costs due to osteoporosis accumulated to approximately $17 billion in the US. Furthermore, it is estimated that by 2025, annual fractures and costs would have grown by 50% (Burge, 2007). Considering the substantial financial costs together with the fact that osteoporosis affects one out of every four postmenopausal Caucasian women (Siris, Brenneman, Miller et al., 2004), it is imperative that effective prevention and treatment regimes be put in place (Korpelainen et al., 2006).

Osteoporosis is typically treated by using pharmacological agents (Howe et al., 2011) despite the fact that mechanical loading associated with regular aerobic, weightbearing and resistance exercise are key to stimulate osteogenesis and increase bone mineral density (Howe et al., 2011; Langberg, Skovgaard, Asp, et al., 2000). Despite the fact that high-impact exercise has been suggested to be most effective for the prevention of osteoporosis in premenopausal women (Korpelainen et al., 2006), the evidence of the long-term effect of PA on postmenopausal bone loss is inadequate, mainly due to short follow-up times seen in the core body of literature. However, a meta-analysis concluded that after a year or longer, PA may be effective for slowing bone loss (Korpelainen et al., 2006), deeming exercise a safe and effective modality to avert bone loss in postmenopausal women (Howe et al., 2011). In addition to preventing osteoporosis through mechanical loading, exercise also improves muscle strength, mobility and balance, all of which will aid and protect the elderly against falls and fractures (Buchner, Cress, de Lateur et al., 1997), something dietary supplements and medication are unlikely to accomplish in isolation.

Exercise and Depression

According to the Global Burden of Disease study (Mathers and Loncar, 2005), mild to moderate Major Depressive Disorder (MDD) is one of the most pronounced causes for years of life lost due to premature death or disability, ranking second behind ischemic heart disease (Dunn, Trivedi, Kampert et al., 2005). It is estimated that approximately one in five adults experience MDD at some stage of their lives (Blumenthal et al., 2005). Depression is twice as likely to occur in women compared to men (Blumenthal et al., 2005) and often co-occurs with medical conditions such as obesity, diabetes, and cardiovascular disease (Blumenthal et al., 2005). In SA, the prevalence of MDD accounts to approximately 9.7% of adults with significantly higher numbers of MDD diagnoses seen among females compared to males, and those with a low level of education compared to those with a higher level of education (Tomlinson, Grimsrud, Stein et al., 2009).

There is scientific evidence to suggest that physical exercise is just as effective in the treatment of mild to moderate MDD as pharmacotherapy and psychotherapy (Dunn et al., 2005), and those who do participate in regular leisure-time exercise are less likely to experience depressive symptoms, irrespective of the intensity of the exercise they engage in (Harvey, Hotopf, Øverland et al., 2010). Positive changes in the psychological profile with exercise may include (Warburton, Katzmarzyk, Rhodes et al., 2007): Improved mood, Improved self-concept, Improved work behavior, Decreased depression and anxiety and Improved social networks.

The amount of exercise needed for persons suffering from depression is equivalent to the recommendations for healthy subjects since the effect of lower amounts of exercise is similar to that of placebo controls (Dunn et al., 2005). According to the American College of Sports Medicine (ACSM) guidelines (ACSM, 2010), the recommended exercise dose is at least 30 minutes a day, five days per week of moderate intensity, or 50 minutes three times per week of high intensity exercise, accumulating to 150 minutes of exercise per week (ACSM, 2010). Although most studies to date have focused on aerobic exercise, some studies found evidence that resistance training may also be effective (Blumenthal et al., 2005).

Exercise and Dementia

Alzheimer's disease (AD) is a progressive neurodegenerative disorder that is characterized by the presence of amyloid deposition and neurofibrillary tangles in the brain, coupled with a loss of cortical neurons and synapses (Nestor, Scheltens and Hodges, 2004; Terry, Masliah, Salmon et al., 1991). It is considered not only the most common cause of dementia (Nestor et al., 2004), but also the most widespread kind of dementia throughout the world with rates increasing exponentially with age (Kawas and Corrada, 2006). The prevalence rose from 3% among the 65-75 years age group to a staggering 50% among those 85 years and older (Zhu and Sano, 2006). In 2006, the worldwide incidence of Alzheimer's disease accounted to 26.6 million cases. It is furthermore estimated that by 2050, the incidence of Alzheimer's disease will quadruple to 1 in 85 persons worldwide who will be living with the disease (Brookmeyer, Johnson, Ziegler-Graham et al., 2007).

Alzheimer's disease, other dementias and alcohol-use disorders are projected to be among the top four causes of burden of disease in high-income countries in 2030 (Mathers and Loncar, 2005).Globally, around 13% (nearly 4.3 million) AD cases may be attributed to physical inactivity (Barnes and Yaffe, 2011).

Presently there is no cure for AD (Zhu and Sano, 2006). However, substantial evidence exists that PA have an important role in moderating dementia such as AD (Hillman, Erickson and Kramer, 2008; Larson and Wang, 2006; Scarmeas, Luchsinger, Schupf et al., 2009; Verdelho, Madureira, Ferro et al., 2012). By increasing PA by 25%, an estimated million cases of dementia can be prevented (Nagamatsu, Flicker, Kramer et al., 2014). Positive findings on the exercise response in AD include a slower decline in mental status (Arcoverde, Deslandes, Rangel et al., 2008), improved cognitive function (Guiney and Machado, 2013; Kramer, Colcombe and McAuley, 2005), improved health and an improvement in quality of life (Deslandes, Moraes, Ferreira et al., 2009; Lawlor and Hopker, 2001; Pedersen and Saltin, 2006).

11. Conclusion

As physical inactivity remains a pressing public health issue (Grandes, Sanchez, Sanchez-Pinilla et al., 2009), a great need exists to increase activity in the general population. Even though being physically active for health and well-being is accepted by much of the general population, the majority of people in developed countries fail to meet even minimal requirements (Lee et al., 2012). There's numerous preventative and therapeutic health benefits associated with a physically active lifestyle which include the effectiveness of exercise to treat various chronic diseases and alsothere's limited side effects associated with exercise in comparison to pharmaceutical therapies. Promotion of PA is a priority for health agencies (Heath, Parra, Sarmiento et al., 2012), evidenced by the shift in focus from monitoring, protecting and promoting general health, to injury prevention and control, chronic disease prevention and management, health-promoting public policies and environmental support for behavioral change to increase PA in a whole population (Alwan, 2011; Davis, Verhagen, Bryan et al., 2014)

References

[1] Sigal RJ, Kenny GP, Wasserman DH, Castaneda-Sceppa C, White RD (2006). Physical activity/exercise and type 2 diabetes: a consensus statement from the American Diabetes Association. Diabetes Care,29:1433-1438.

[2] Durstine JL, Painter P, Franklin BA, Morgan D, Pitetti KH, Roberts SO (2000). Physical activity for the chronically ill and disabled. *Sports Med*,30:207-219.

[3] Allender S, Foster C, Scarborough P, Rayner M(2007). The burden of physical activity-related ill health in the UK. *Journal of Epidemiology and Community Health*, *61*(4):344–8.

[4] Alwan A(2011). Global status report on noncommunicable diseases 2010., World Health Organization. World Health Organization.

[5] Apovian C(2010). The causes, prevalence, and treatment of obesity revisited in 2009: what have we learned so far? *American Journal of Clinical Nutrtion*, 91:277–279.

[6] Arcoverde C, Deslandes A, Rangel A, Rangel A, Pavão R, Nigri F, Engelhardt E, Laks J(2008). Role of physical activity on the maintenance of cognition and activities of daily living in elderly with Alzheimer's disease. *Arquivos de neuropsiquiatria*, 66(2B):323-327.

[7] Balady GJ, Williams M, Ades P, Bittner V, Comoss P, Foody J M, Franklin B, Sanderson B, Southard D(2007). Core components of cardiac rehabilitation/ secondary prevention programs: 2007 Update A scientific statement from the American heart association exercise, cardiac rehabilitation, and prevention committee, the council on clinical cardiology; the councils on cardiovascular nursing, epidemiology and prevention, and nutrition, physical activity, and metabolism; and the American association of cardiovascular and pulmonary rehabilitation. *Circulation*,115: 2675–2682.

[8] Barnes D, Yaffe K. (2011). The Projected impact of risk factor reduction on Alzheimer's disease prevalence. *Lancet Neurology*, 10: 819–828.

[9] Bassuk SS, Manson JE(2005). Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease. *Journal of Applied Physiology*, 99:1193–1204.

[10] Bauman A, Phongsavan P, Schoeppe S, Owen N (2006). Physical activity measurement--a primer for health promotion. *Promotion & Education*, **13**:92–103.

[11] Blair S(1989). Physical fitness and all-cause mortality. A prospective study of health men and woman. *JAMA*,262:2395–2401.

[12] Blumenthal J A(2005). Exercise and pharmacotherapy in the treatment of major depressive disorder. *Psychosomatic medicine*, 69(7):587-596.

[13] Boulton A J, Vinik A I, Arezzo J C, Bril V, Feldman E L, Freeman R, Malik R A, Maser R E, Sosenko J M, Ziegler D.(2005). Diabetic neuropathies. A statement by the American Diabetes Association. *Diabetes Care*, 28(4): 956–962.

[14] Brookmeyer R, Johnson E, Ziegler-Graham K, Arrighi M(2007). Forecasting the global burden of Alzheimer's disease. *Alzheimer's & dementia*, 3(3):186-191.

[15] Buchner DM, Cress ME, de Lateur BJ, Esselman PC, Margherita AJ, Price R, Wagner E H(1997). The effect of strength and endurance training on gait, balance, fall

risk, and health services use in community-living older adults. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 52(4):M218-M224.

[16] Burge R (2007). Incidence and Economic Burden of Osteoporosis-Related Fractures in the United States, 2005–2025. *Journal of bone and mineral research*, **22** (3):465–475.

[17] Caballero B (2007). The global epidemic of obesity: an overview. *Epidemiologic Review*, 29 (7):1–5.

[18] Casaburi R, ZuWallack R(2009). Pulmonary rehabilitation for management of chronic obstructive pulmonary disease. *The New England journal of medicine*,360 (13):1329–1335.

[19] Celli BR(2008). Update on the management of COPD. *Chest*,133 (6):1451–1462.

[20] Chakravarthy M, Joyner M, Booth F(2002). Sedentary Patients to Reduce the Risk of Chronic Health Conditions. *Mayo clinic proceedings*, **77** (2):165–173.

[21] Chavannes N, Vollenberg JJH, van Schayck CP, Wouters EFM(2002). Effects of physical activity in mild to moderate COPD: a systematic review. *The British journal of general practice*,52 (480):574–578.

[22] Clark R, Niccolai L, Kissinger P, Peterson Y, Bouvier V(1999). Ethnic differences in body image attitudes and perceptions among women infected with human immunodeficiency virus. *Journal of the American Dietetic Association*, 99**(**6):735-737.

[23] Colberg S, Sigal R (2010). Exercise and type 2 diabetes the American college of sports medicine and the American diabetes association: joint position statement executive summary. *Diabetes care*, 33(12):2692–2696.

[24] Connor M, Rheeder P, Bryer A, Meredith M, Beukes M, Dubb A, Fritz V (2005). The South African stroke risk in general practice study. *SouthAfrican Medical Journal*,95 (5): 334–339.

[25] Davis JC, Verhagen E, Bryan S, Liu-Ambrose T, Borland J, Buchner D, Hendriks MR, Weiler R, Morrow JR, van Mechelen W, Blair SN, Pratt M, Windt J, al-Tunaiji H, Macri E, Khan K M(2014). 2014 Consensus Statement from the first Economics of Physical Inactivity Consensus (EPIC) Conference (Vancouver). *British journal of sports medicine***48** (12):947–951.

[26] Derman E, Patel D N, Nossel C J, Schellnus M P(2008a). Healthy lifestyle interventions in general practice Part 1: An introduction to lifestyle and diseases of lifestyle. *South African Family Practice*50(4):19–22.

[27] Derman E W, Whitesman S, Dreyer M, Patel D N, Nossel C J, Schwellnus M P(2008b). Healthy lifestyle interventions in general practice Part 2: Lifestyle and cardiovascular disease. *South African Family Practice*,50(5): 6-10.

[28] Deslandes, A., Moraes, H., Ferreira, C., Veiga, H., Silveira, H., Mouta, R.,Pompeu, F. A., Coutinho, E. S. F., Laks, J. 2009. Exercise and mental health: many reasons to move. *Neuropsychobiology*,59 (4) pp 191–8.

[29] Dreher D, Junod F(1996). Role of oxygen free radicals in cancer development. *European journal of cancer*, *32*A(1):30–38.

[30] Dunn A L, Trivedi M H, Kampert J B, Clark C G, Chambliss H O(2005). Exercise treatment for depression: efficacy and dose response. *American journal of preventive medicine*28 (1):1–8.

[31] Fabbri L, Rabe K(2007). From COPD to chronic systemic inflammatory syndrome? *TheLancet* 370:797–799.

[32] Ferlay J, Steliarova-Foucher E, Lortet-Tieulent J, Rosso S, Coebergh J W W, Comber H, Forman D, Bray F(2013). Cancer incidence and mortality patterns in Europe: estimates for 40 countries in 2012. *European journal of cancer*49 (6):1374–403.

[33] Fletcher G, Balady GJ, Amsterdam E A, Chaitman B, Eckel R, Fleg J, Froelicher V F, Leon A S, Piña I L, Rodney R, Simons-Morton D A , Williams, M. A., Bazzarre, T(2001). Exercise standards for testing and training a statement for healthcare professionals from the American Heart Association. *Circulation*,104:1694–1740.

[34] Fletcher GF, Ades PA, Kligfield P, Arena R, Balady G J, Bittner V A, Coke L A, Fleg J L, Forman D E, Gerber T C, Gulati M, Madan K, Rhodes J, Thompson P D, Williams M A(2013). Exercise standards for testing and training: a scientific statement from the American Heart Association. *Circulation*, 128:873–934.

[35] Franssen F M, Broekhuizen R, Janssen P P, Wouters E F, Schols A M(2004). Effects of whole-body exercise training on body composition and functional capacity in normal-weight patients with COPD. *Chest*,125 (6): 2021–2028.

[36] Franz M J, Van Wormer J J, Crain A L, Boucher J L, Histon T, Caplan W, Bowman J D, Pronk N P(2007). Weight-loss outcomes: a systematic review and meta-analysis of weight-loss clinical trials with a minimum 1-year follow-up. *Journal of the American Dietetic Association***107**(10): 1755–1767.

[37] Franzoni F, Ghiadoni L, Galetta F, Plantinga Y, Lubrano V, Huang Y, Salvetti

[38] A(2005). Physical activity, plasma antioxidant capacity, and endotheliumdependent vasodilation in young and older men. *American journal of hypertension*, **18**(4):510-516.

[39] Galvão D A, Taaffe D R, Spry N, Joseph D, Newton R U(2010). Combined resistance and aerobic exercise program reverses muscle loss in men undergoing androgen suppression therapy for prostate cancer without bone metastases: a randomized controlled trial. *Journal of clinical oncology*, **28**(2):340-347.

[40] Garcia-Aymerich J, Barreiro E, Farrero E, Marrades R M, Morera J, Anto J M(2000). Patients hospitalized for COPD have a high prevalence of modifiable risk factors for exacerbation (EFRAM study). *The European respiratory journal*,**16** (6):1037–1042.

[41] Giannuzzi P, Saner H, Björnstad H, Fioretti P, Mendes M, Cohen-Solal A, Dugmore L, Hambrecht R, Hellemans I, McGee H, Perk J, Veress G(2003). Secondary prevention through cardiac rehabilitation position paper of the working group on cardiac rehabilitation and exercise physiology of the European Society of Cardiology. *European Heart Journal*,24(13): 1273-1278.

[42] Gordon NF, Gulanick M, Costa F, Fletcher G, Franklin B A, Roth E J, Shephard T(2004). Physical activity and exercise recommendations for stroke survivors: An American Heart Association scientific statement from the council on clinical cardiology subcommittee on exercise, cardiac rehabilitation, and prevention; the council on cardiovascular nursing; the council on nutrition, physical activity, and metabolism; and the stroke council. *Circulation*,**109**(16): 2031–2041.
[43] Gosselink R(2002). Respiratory rehabilitation: improvement of short- and long-term outcome. *European Respiratory Journal*,**20**(1):4–5.
[44] Grandes G, Sanchez A, Sanchez-Pinilla R O, Torcal J, Montoya I, Lizarraga K, Serra J(2009). Effectiveness of physical activity advice and prescription by physicians in routine primary care. *Archives of Internal Medicine*,**169**(7):694–701.
[45] Green D J, Spence A, Halliwill J R, Cable N T, Thijssen D H(2011). Exercise and vascular adaptation in asymptomatic humans. *Experimental physiology*. **96** (2):57–70.
[46] Guiney H, Machado L(2013). Benefits of regular aerobic exercise for executive functioning in healthy populations. *Psychonomic bulletin & review*,**20**(1):73–86.
[47] Harvey S B, Hotopf M, Øverland S, Mykletun A(2010). Physical activity and common mental disorders. *The British journal of psychiatry*,**197**(5):357–364.
[48] Hatano S(1976). Experience from a multicentre stroke register: a preliminary report. *Bulletin of the World Health Organization*, ***54***(5):541.
[49] Hawkes C (2006). Uneven dietary development: linking the policies and processes of globalization with the nutrition transition, obesity and diet-related chronic diseases *Globalization and health*, ***2***(1):4.
[50] Heath G W, Parra D C, Sarmiento O L, Andersen L B, Owen N , Goenka S, Monres F ,Brownson R C(2012). Evidence-based intervention in physical activity: Lessons from around the world. *TheLancet*,**380**(9838):272–281.
[51] Helmrich SP, Ragland DR, Leung RW, Paffenbarger RJ(1991). Physical activity and reduced occuring of non-insulin-dependent diabetes mellitus. *New England Journal of Medicine***325**(3):147–152.
[52] Hillman C H, Erickson K I, Kramer A F(2008). Be smart, exercise your heart: exercise effects on brain and cognition. *Nature Reviews Neuroscience*, ***9***(1):58-65.
[53] Hordern M D, Dunstan D W, Prins J B, Baker M K, Singh M A F, Coombes J S(2012). Exercise prescription for patients with type 2 diabetes and prediabetes: a position statement from Exercise and Sport Science Australia. *Journal of science and medicine in sport / Sports Medicine Australia*, **15**(1): 25–31.
[54] Howe T E, Shea B, Dawson L J, Downie F, Murray A, Ross C, Harbour R T, Caldwell LM, Creed G(2011). Exercise for preventing and treating osteoporosis in postmenopausal women. *The Cochrane Library*.
[55] Huber M, Knottnerus J A, Green L, Horst H V D, Jadad A R, Kromhout D,Leonard B, Lorig K, Loureiro M I, van der Meer JWM, Schnabel P, Smith R, van Weel C, Smid H(2011). How should we define health? *British medical journal*,**343**(2):d4163–d4163.
[56] James P, Leach R(2001). The worldwide obesity epidemic. Obes. Res. 9. James, P. T., Leach, R., Kalamara, E., & Shayeghi, M.,2001. The worldwide obesity epidemic. *Obesity research*, ***9***(S11):228S-233S.
[57] Jasienska G, Ziomkiewicz A(2006). Habitual physical activity and estradiol levels in women of reproductive age. *European journal of cancer prevention*, **15**(5):439–45.
[58] Joubert J, Norman R, Lambert E V, Groenewald P, Schneider M, Bull F, Bradshaw D, and Collaboration, S A C.R A(2007). Estimating the burden of disease attributable to physical inactivity in South Africa in 2000. *South African Medical Journal*.**97**(8):725–731.
[59] Kai M C, Anderson M, Lau E(2003). Exercise interventions: defusing the world's osteoporosis time bomb. *Bulletin of the World Health Organization*, **8**(11):827–830.
[60] Katzmarzyk PT, Gledhill N, Shephard RJ(2000). The economic burden of physical inactivity in Canada. *Canadian medical association journal*,**163**(11):1435–1440.
[61] Kawas C, Corrada M(2006). Alzheimer's and dementia in the oldest-old: a century of challenges. *Current Alzheimer Research*, **3**(5):411–419.
[62] Kelley G A, Kelley KS(2008). Efficacy of aerobic exercise on coronary heart disease risk factors. *Preventive cardiology*,**11**(2):71–75.
[63] Kelley G A, Kelley K S, Roberts S, Haskell W(2011). Efficacy of aerobic exercise and a prudent diet for improving selected lipids and lipoproteins in adults: a meta-analysis of randomized controlled trials. *BMC Medicine*,**9**(1):74.
[64] Khan K(2008). Wikinomics, Steven Blair and the Zurich International Conference on Concussion in Sport. Br. J. Sports Med. **42**(4):235.
[65] Kokkinos P, Myers J(2010). Exercise and physical activity: clinical outcomes and applications. *Circulation*,**122**(16):1637–48.
[66] Korpelainen R, Keinänen-Kiukaanniemi S, HeikkinenJ, Väänänen K, & Korpelainen J(2006). Effect of impact exercise on bone mineral density in elderly women with low BMD: a population-based randomized controlled 30-month intervention. *Osteoporosis International*,**17**(1):109–118.
[67] Kramer A, Colcombe S, McAuley E(2005). Fitness, aging and neurocognitive function. *Neurobiology of Aging*, **26**(1):124–127.
[68] Kruger H S, Venter C S, Vorster H H, Margetts B M(2002). Physical inactivity is the major determinant of obesity in black women in the North West province, South Africa: the THUSA study. *Nutrition*,**18**(5):422–427.
[69] Kruk J(2007). Physical activity in the prevention of the most frequent chronic diseases: an analysis of the recent evidence. *Asian Pacific journal of cancer prevention***8**(3):325–338.
[70] Lacasse Y, Goldstein R(2006). Pulmonary rehabilitation for chronic obstructive pulmonary disease. *Cochrane Database Syst. Rev* **4**(4).

[71] LaMonte M J, Blair S N, Church T S (2005). Physical activity and diabetes prevention. *Journal of Applied Physiology*, **99**(3):1205–1213.
[72] Langberg H, Skovgaard D, Asp S, Kjaer M(2000). Time pattern of exercise induced changes in type I collagen turnover after prolonged endurance exercise in humans. *Calcified tissue international*, **67**(1), pp.41–44.
[73] Larson, E., Wang, L., 2006. Exercise is associated with reduced risk for incident dementia among persons 65 years of age and older. *Annals of internal medicine***144**(2), pp.73–82.
[74] Lawlor, D.A., Hanratty, B., 2001. The effect of physical activity advice given in routine primary care consultations: a systematic review. *Journal of public health medicine***, 23**(3), pp.219–226.
[75] Lee, D. C., Sui, X., Blair, S. N.2009. Does physical activity ameliorate the health hazards of obesity? *British Journal of Sports Medicine*,**43**(1), pp.49–51.
[76] Do Lee, C., Folsom, A. R., Blair, S. N. 2003. Physical activity and stroke risk: a metaanalysis. *Stroke*,**34**(10), pp.2475–2481.
[77] Lee, I. M., Shiroma, E. J., Lobelo, F., Puska, P., Blair, S. N., Katzmarzyk, P. T. 2012.
[78] Effect of physical inactivity on the world's major non-communicable diseases. *TheLancet*, **380**(9838), pp.219–229.
[79] Leijon, M. E., Bendtsen, P., Nilsen, P., Ekberg, K., & Ståhle, A. 2008. Physical activity referrals in Swedish primary health care - prescriber and patient characteristics, reasons for prescriptions, and prescribed activities. *BMC health services research*,**8**(1), p.201.
[80] Leon, A. S., Franklin, B. A., Costa, F., Balady, G. J., Berra, K. A., Stewart, K. J., Thompson, P. D., Williams, M. A., Lauer, M. S. 2005. Cardiac rehabilitation and secondary prevention of coronary heart disease: an American heart association scientific statement from the council on clinical cardiology (Subcommittee on exercise, cardiac rehabilitation, and prevention) and the council on nutrition. *Circulation*,**111**(3), pp.369–376.
[81] Lloyd-Jones, D., Adams, R. J., Brown, T. M., Carnethon, M., Dai, S., De Simone, G., Ferguson, G. T., Ford, E., Furie, K., Gillespie C., Go, A., Greenlund, K., Haase N., Hailpern, S., Ho, P. M., Howard, V., Kissela, B., Kittner, S., Lackland, D., Lisabeth, L., Marelli, A., McDermott, M. M., Meigs, J., Mozaffarian, D., Mussolino,
[82] M., Nichol, G., Roger, V. L., Rosamond, W., Sacco, R., Sorlie, P., Stafford, R., Thom T., Wasserthiel-Smoller, S., Wong, N. D., Wylie-Rosett, J. 2010. Heart disease and stroke statistics—2010 update A report from the American heart association. *Circulation*,**121**(7), e46–e215.
[83] MacDonald, J.R., 2002. Potential causes, mechanisms, and implications of post exercise hypotension. *J. Hum. Hypertens.***16**(4), pp.225–236.
[84] Macfarlane, D.J., Thomas, G.N., 2010. Exercise and diet in weight management: updating what works. *British journal of sports medicine*, [Online] **44**, pp. 1197–1201. [Accessed 8 June 2010]. Available from: http://www.ncbi.nlm.nih.gov/pubmed/19846427
[85] Mathers, C., Loncar, D., 2005. Updated projections of global mortality and burden of disease, 2002-2030: data sources, methods and results. *Geneva World Health Organization.*
[86] Mayfield, J.A., Reiber, G. E., Sanders, L. J., Janisse, D., Pogach L.M. 1998. Preventive foot care in people with diabetes. *Diabetes Care***21**, pp. 2161–2177.
[87] Mayosi, B. M., Flisher, A. J., Lalloo, U. G., Sitas, F., Tollman, S. M., & Bradshaw, D. 2009. The burden of non-communicable diseases in South Africa. *The Lancet*, **374**, pp.934–947.
[88] Miles, L., 2007. Physical activity and health. *Nutrition Bulletin***, 32**, pp.314–363.
[89] Mora, S., Cook, N., Buring, J. E., Ridker, P. M., & Lee, I. M. 2007. Physical activity and reduced risk of cardiovascular events potential mediating mechanisms. *Circulation*,**116**, pp.2110–2118.
[90] Murphy, M. H., Blair, S. N., Murtagh, E. M. 2009. Accumulated versus continuous exercise for health benefit: a review of empirical studies. *Sports medicine***39**, pp.29–43.
[91] Mvo, Z., Dick, J., Steyn, K., 1999. Perceptions of overweight African women about acceptable body size of women and children. *Curationis*,**22**, pp.27–31.
[92] Nagamatsu, L. S., Flicker, L., Kramer, A. F., Voss, M. W., Erickson, K. I., Hsu, C. L., Liu-Ambrose, T. 2014. Exercise is medicine, for the body and the brain. [Online] *British journal of sports medicine***48**, pp.943–944. [Accessed 12 June 2014].
[93] Available from: http://bjsm.bmj.com/content/early/2014/03/21/bjsports-2013-
[94] 093224.short
[95] Nestor, P. J., Scheltens, P., Hodges, J. R. 2004. Advances in the early detection of Alzheimer's disease. *Nature reviews Neuroscience*, **5**, S34–41.
[96] Newton, R.U., Galvão, D. A, 2008. Exercise in prevention and management of cancer.
[97] *Current treatment options in oncology*, **9**, pp.135–46.
[98] Oberg, E., 2007. Physical activity prescription: our best medicine. *Integrative Medicine*, **6**, pp.18–22.
[99] Pan, X., Li, G.-W., Hu, Y., Wang, J., 1997. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. *Diabetis Care*,**20**, pp.537–544.
[100] Pauwels, R.A., Buist, A. S., Calverley, P. M. A., Jenkins, C. R., Hurd, S. A. 2001. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. NHLBI/WHO global initiative for chronic obstructive lung disease (GOLD) workshop summary. *American journal of respiratory and critical care medicine*, **163**, pp.1256–1276.
[101] Pearson, T.A., 2003a. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: a statement for healthcare professionals from the centers for disease control and prevention and the American heart association. *Circulation*,**107**, pp.499–511.
[102] Pedersen, B.K., Saltin, B., 2006. Evidence for prescribing exercise as therapy in chronic disease. *Scandinavian journal of medicine & science in sports*, **16** (S1), pp.3–63.

[103] Prentice, A., 2006. The emerging epidemic of obesity in developing countries. *International journal of epidemiology*, **35**, pp. 93–99.
[104] Puhan, M. A., Mador, M. J., Held, U., Goldstein, R., Guyatt, G. H., Schünemann, H. J. 2008. Interpretation of treatment changes in 6-minute walk distance in patients with COPD. *The European Respiratory Journal*,**32**, pp.637–643.
[105] Puhan, M. A., Schünemann, H. J., Frey, M., Scharplatz, M., Bachmann, L. M. 2005. How should COPD patients exercise during respiratory rehabilitation? Comparison of exercise modalities and intensities to treat skeletal muscle dysfunction. *Thorax*, **60**, pp.367–375.
[106] Puhan, M. A., Siebeling, L., Zoller, M., Muggensturm, P., ter Riet, G. 2013. Simple functional performance tests and mortality in COPD. *The European Respiratory Journal*, **42**(4) pp. 956-963.
[107] Raisz, L.G., 2005. Pathogenesis of osteoporosis: concepts, conflicts, and prospects. *The Journal of Clinical Investigation*,**115 (**12), pp. 3318–3325.
[108] Reardon, J., Casaburi, R., Morgan, M., Nici, L., Rochester, C. 2005. Pulmonary rehabilitation for COPD. *Respiratory medicine*,**99,** Suppl B, S19–27.
[109] Roberts, C.K., Barnard, R.J., 2005. Effects of exercise and diet on chronic disease. *Journal of Applied Physiology*, **98**, pp 3–30.
[110] Roux, L., Pratt, M., Tengs, T. O., Yore, M. M., Yanagawa, T. L., Van Den Bos, J., Rutt, C., Brownson, R. C., Powell, K. E., Heath, G., Kohl III, H. W., Teutsch, S., Cawley, J., Lee, I., West, L., Buchner, D. M. 2008. Cost effectiveness of community-based physical activity interventions. *American Journal of Preventive Medicine*,**35**, pp. 578–588.
[111] Rutschmann, O. T., Janssens, J. P., Vermeulen, B., Sarasin, F. P. 2004. Knowledge of guidelines for the management of COPD: a survey of primary care physicians. *Respiratory Medicine*, **98**, pp. 932–937.
[112] Sacco, R. L., Kasner, S. E., Broderick, J. P., Caplan, L. R., Culebras, A., Elkind, M. S., George, M. G., Hamdan, A. D., Higashida, R. T., Hoh, B. L., Janis, L. S., Kase, C.
[113] S., MD; Kleindorfer, D. O., Lee, J., Moseley, M. E., Peterson, E. D., Turan, T. N., Valderrama, A. L., Vinters, H. V. 2013. An updated definition of stroke for the 21st century: A statement for healthcare professionals from the American heart association/American stroke association. *Stroke*,**44**, pp. 2064–2089.
[114] Sallis, R.E., 2009. Exercise is medicine and physicians need to prescribe it! *British journal of sports medicine* [Online] **43**(1), pp. 3–4. [Accessed 2 December 2013]. Available from: http://www.ncbi.nlm.nih.gov/pubmed/18971243
[115] Scarmeas, N., Luchsinger, J. A., Schupf, N., Brickman, A. M., Cosentino, S., Tang, M. X., Stern. 2009. Physical activity, diet, and risk of alzheimer disease. *JAMA***302**, pp. 627–637.
[116] Senekal, M., Steyn, N. P., Nel, J. H.. 2003. Factors associated wiyh overweight / obesity in economically active South African populations. *Ethnicity & Disease*, 13, pp.109–116.
[117] Shann, F., 2000. Does physical activity prevent cancer? *British medical journal***321**, pp.1424–1425.
[118] Shaw, J. E., Sicree, R. A., & Zimmet, P. Z. 2010. Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes research and clinical practice*,**87**, pp. 4–14.
[119] Shisana, O., Labadarios, D., Rehle, T., Simbayi, L., Zuma, K., Dhansay, A., Reddy, P., Parker, W., Hoosain, E., Naidoo, P., Hongoro, C., Mchiza, Z., Steyn, N. P., Dwane, N., Makoae, M., Maluleke, T., Ramlagan, S., Zungu, N., Evans, M. G., Jacobs, L., Faber, M., SANHANES-1 Team. 2013. South African national health and nutrition examination survey 2012 (SANHANES-1). [Online]. [Accessed 1 September 2013]. Available from:*http://www.hsrc.ac.za/en/mediabriefs/population health/results-sanhanes*
[120] Siris, E. S., Brenneman, S. K., Miller, P. D., Barrett-Connor, E., Chen, Y. T., Sherwood, L. M., Abbott, T. A. 2004. Predictive value of low BMD for 1-year fracture outcomes is similar for postmenopausal women ages 50-64 and 65 and older: results from the National Osteoporosis Risk Assessment (NORA). *Journal of Bone and Mineral Research*, **19**(8), pp. 1215–1220.
[121] Slentz, C. 2004. Effects of the amount of exercise on body weight, body composition and measures of central obesity. *Archives of internal medicine*,**164**(1), pp. 31–39.
[122] Spruit, M. A., Troosters, T., Trappenburg, J. C., Decramer, M., & Gosselink, R. 2004. Exercise training during rehabilitation of patients with COPD: a current perspective. *Patient education and counseling*,**52**, pp. 243–248.
[123] Stephens, M.B., 2009. Cardiac Rehabilitation. *American Family Physician*, **80**, pp. 955– 959.
[124] Terry, R. D., Masliah, E., Salmon, D. P., Butters, N., DeTeresa, R., Hill, R., Hansen, L.A., Katzman, R. 1991. Physical basis of cognitive alterations in Alzheimer's disease: synapse loss is the major correlate of cognitive impairment. *Annals of Neurology*, **30**(4), pp.572–580.
[125] Thompson, P.D., 2005. Exercise prescription and proscription for patients with coronary artery disease. *Circulation*,**112**(15), pp.2354–2363.
[126] Thompson, P. D., Franklin, B. A., Balady, G. J., Blair, S. N., Corrado, D., Estes, N. M., Fulton, J. E., Gordon, N. F., Haskell, H. L., Link, M. S., Maron,B. J., Mittleman, M. A., Pelliccia, A., Wenger, N. K., Willich, S. N., Costa, F. 2007. Exercise and acute cardiovascular events Placing the risks into perspective: a scientific statement from the American heart association council on nutrition, physical activity, and metabolism and the council on clinical cardiology. *Circulation*,**115**(17), pp.2358– 2368.
[127] Tomlinson, M., Grimsrud, A. T., Stein, D. J., Williams, D. R., Myer, L. 2009. The epidemiology of major depression in South Africa: results from the South African stress and health study. *South African Medical Journal*,**99**(5), pp.367–373.
[128] Van der Merwe, M., Pepper, S., 2006. Obesity in SA. *Obesity Review*,**7**, pp.315–322.

[129] Verdelho, A., Madureira, S., Ferro, J. M., Baezner, H., Blahak, C., Poggesi, A., Hennerici, M., Pantoni, L., Fazekas, F., Scheltens, P., Waldemar, G., Wallin, A., Erkinjuntti, T., Inzitari, D. 2012. Physical activity prevents progression for cognitive impairment and vascular dementia: results from the LADIS (Leukoaraiosis and disability) study. *Stroke*, **43**(12), pp.3331–3335.
[130] Wallace, J., 2003. Exercise in hypertension: a clinical review. *Sports medicine*,**33**(8), pp.585–598.
[131] Warburton, D. E., Katzmarzyk, P. T., Rhodes, R. E., Shephard, R. J. 2007. Evidenceinformed physical activity guidelines for Canadian adults. *Applied Physiology, Nutrition, and Metabolism*,**32**(S2E), S16-S68.
[132] Whiting, D. R., Guariguata, L., Weil, C., Shaw, J. 2011. IDF diabetes atlas: global estimates of the prevalence of diabetes for 2011 and 2030. *Diabetes Research and Clinical Practice*,**94**(3), pp.311–321.
[133] WHO, 2014. World health organization [Online]. [accessed 6 July 2014]. Available from: *http://www.who.int/gho/ncd/en*
[134] Woodard, C.M., Berry, M.J., 2001. Enhancing adherence to prescribed exercise: structured behavioral interventions in clinical exercise programs. *Journal of Cardiopulmonary Rehabilitation and Prevention*, **21**(4), pp.201–209.
[135] Zhu, C.W., Sano, M., 2006. Economic considerations in the management of alzheimer's disease. *Clinical interventions in aging*, **1**(2), pp.143–154.
[136] Zimmet, P., 2000. Globalization, coca-colonization and the chronic disease epidemic: can the doomsday scenario be averted? *Journal of Internal MedIcine*, **247**(3), pp.301-310