## **Short Communication**

# Ageing, Obesity, Sedentary Lifestyle: Constituents for Poor Health and Performance

#### Archer T<sup>1,2\*</sup>

<sup>1</sup>University of Gothenburg, Department of Psychology, Sweden

<sup>2</sup>Network for Empowerment and Well-Being, Sweden

\*Corresponding author: Trevor Archer, University of Gothenburg, Department of Psychology, Sweden

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### Abstract

Physical exercise accompanied by dietary restriction-selection, as a lifespan procedure bequeaths beneficial health manifestations and well-being over a plethora of health and functional domains. The persistence of a lifestyle defined by unrestricted intake of unhealthy food and drink intakes and a chronically sedentary characteristic over the lifespan will inevitably prove disastrous for salubriousness and longevity.

Keywords: Ageing; Obesity; Sedentary; III health

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The accumulating bulk of plausible evidence supporting the notion that the incremental effects of restricted and/or 'tailor-made"/ selective diets in conjunction with physical exercise have enabled substantially improved outcomes over the lifespan of individuals presenting overweight and obesity under both clinical and laboratory conditions, whether or not accompanied by other disorder comorbidities [1-3]. In aging populations, an age-related condition, sarcopenia, metabolic disorders and the development of insulin resistance are linked to deteriorations in muscle mass subsequent to state of inactivity and inability or lack of interest in exercise that may accompany obesity/overweight. A lifestyle defined by the poverty and inadequacy of diet, estimated through the low diet quality score, and sedentary behavior, is linked to with greater prevalence of obesity, hypertension and a multitude of other long-term illnesses. In a study of the influence of exercise schedules combined with protein and vitamin D intake in older Koreans (N = 4452, aged > 60 years), [4] found a greater appendicular skeletal muscle Mass/ weight in the non-obese participants in comparison with the obese participants although the latter weighed more. The non-obese sarcopenic participants presented detrimental health related to increased insulin resistance and metabolic disorder which the nonsarcopenic group did not. Dietary deficiency in protein level and vitamin D was enhanced with the prevalence of obesity, sarcopenia and sarcopenic obesity. Resistance exercise combined with protein and vitamin D intake attenuated obesity and sarcopenia among these elderly. Ageing muscles present fat infiltration and muscle catabolism accompanied by expression of pro-inflammatory cytokines instead of exercise-induced anti-inflammatory cytokines and myokines [5-7]. Within this context, the associations between inactivity, body weight problems, inflammation markers and insulin resistance all contribute to a narrative that will probably not unfold into a 'happy' outcome [8,9].

Among obese patients presenting diabetes co-morbidity, the combined regime of diet and physical exercise administered concurrently with sibutramine, a monoamine reuptake inhibitor, caused a weight loss outcome which was found to have reduced the blood glucose levels and glycated hemoglobin when analysed with reference to similar weight loss among patients assigned to the placebo condition receiving the diet and exercise combination [10]. It ought to be noted that glycated hemoglobin acts as a biomarker for average blood glucose levels over the previous three months before the measurement as this is the lifespan of red blood cells; greater levels of glycated hemoglobin, implying poorer control of blood glucose levels, have been associated with several chronic conditions, including cardiovascular disorders [11], in a meta-analytic study of results from 28 different clinical trials, observed that physical exercise, by itself, independent of presence/absence of dietary alterations/restrictions/ selections was related to a remarkable decrease in intra-hepatic lipid content as well as reductions in alanine aminotransferase and aspartate aminotransferase. Applying a meta-regression technique, the authors showed that individuals hampered by an increased body mass index were destined to be more likely to benefit from the intervention. By all accounts, it seems that the physical activity regime reduced intrahepatic lipid content and markers of hepatocellular injury in patients presenting non-alcoholic liver disease with these reductions correlated with individuals' baseline body mass index. Furthermore, in spite of the unreliable impact of a continuous/repetitive high fat dietary intake, the regime of regular exercise and dietary alteration induced a positive effect on insulin resistance and the mammalian target of rapamycin signaling protein levels [12].

Among Australian adults presenting obesity co-morbid with hypertension (N = 4908, age =  $45.2 \pm 0.24$ ), the intervention of a higher quality of diet, assessed according to the Dietary Guideline Index, was related to a lower odds ratio of obesity among male and female participants. The odds ratio of hypertension was lesser in the male participants, but not in the female participants, with a high diet quality score compared with a low quality score, whereas obesitylinked hypertension was only related to diet quality score in male participants presenting obesity. In a group of sedentary, obese female patients [13], analysed inflammatory biomarkers, insulin resistance, muscle soreness and fasting C-reactive protein before and after a fiveday exercise intervention. The diet-exercise combinations, short or prolonged, decreased the insulin-resistance index and the c-reactive protein fasting concentrations in the case of the prolonged, 80-min, exercise schedule elevating IL-6 and lowering TNF- $\alpha$  concentrations, yet contrastingly, on those days wherein the exercise schedule was

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missing it was found that the cytokines were unaffected, thereby conferring upon these sedentary patients definite health benefits without the induction of detrimental effects upon inflammation and muscle soreness. Finally, excessive high-caloric diets combined with a sedentary lifestyle and subsequent development of obesity and diabetes mellitus lead to detrimental outcomes for structural integrity and functional outcomes for the brain and CNS [14-16], examined the early effects of a cafeteria-diet on gray and white brain matter volume by means of voxel-based morphometry and region-of-interest analysis in order to ascertain whether or not wheel-running exercise prevented the unfavorable effects of that cafeteria-diet in mice. They found that the mice recipient of the cafeteria diet showed only mild deficits in long-term memory performance assessed by the puzzle-box paradigm, whereas executive functioning and short term memory remained unaffected implying that the physical exercise regime seemed not to interact with these processes. This evidence implies that under laboratory conditions: (i) not all the effects of a poor diet are markedly detrimental to brain structure and function, and (ii) physical exercise may not reverse the effects of a poor diet. Applying a laboratory mouse model of obesity, without comorbidities [17], obtained evidence concerning the underlying mechanism exacerbating vascular dysfunction and the role of exercise in ameliorating this development. Obesity induced endothelium dysfunction in the mice accompanied by proinflammatory TNF- $\alpha$  and nitric oxide synthase isoform pathway up-regulation and a reduction of vascular contractility in the obese mice. The exercise schedule effectively reinstated control of nitric oxide synthase isoform-dependent nitric oxide production and preserved endothelial function in obese individuals. Taken together, the fundamental principle revolving around ageing, obesity and a sedentary lifestyle remains in existence, that longevity is restricted; contrastingly, an active, exercising lifestyle composed of a carefully selected diet and close regard for body and related parameters will hold assurances of a sufficient longevity and well-being.

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