Adipose tissue and resveratrol

Rationale The leading causes of mortality has transitioned from infectious diseases, which now have been successfully battled against by immunizations and antibiotics, to age-associated diseases such as cardiovascular diseases, which is caused by the deposition of adipose tissue on blood vessels. Several studies found that Resveratrol, a plant-produced polyphenolic flavonoid, delays age-related diseases later in the lives of young laboratory animals, and alleviates existing conditions of various health illnesses, such as obesity. This substance is abundant in grape skin, nuts, and pomegranates. The mechanism by which it acts was found to be similar to that of Dietary or Caloric Restriction (DR or CR), or reduction of food intake by 30-50% by every-other-day feeding. DR has been found to delay the onset of the deleterious effects of oxidative stress and subsequent diseases and functional decline. Despite its health-improving effects, it has its contraindications. It is not suitable for the weak and ill, because they need the calories for energy. Following DR also requires enormous amount of discipline, as it demands a change in eating habits and lifestyles. Thus, there is interest in developing substances that mimics the health-improving action of DR (Pearson, et al., 2008) Action on Adipose Tissue Resveratrol is found to activate sirtuins, a family of NAD+-dependent histone deacetylase SIRT1 that acts on PGC1a, which, in turn, unregulating mitochondrial gene expression and subsequently increasing metabolic efficiency (Pearson et al., 2008; Koi and Montminy, 2006). Several studies have already determined its action on various tissues and on various animals. This paper focuses on the polyphenol’s action on adipose tissues. In mice, it decreases adipose tissue by increasing the rates of emulsification through upregulation of the enzyme that converts cholesterol to bile acids, which occurs in the liver. As a result,

Resveratrol decreases plasma cholesterol and triglyceride levels that may deposit on the walls of blood vessels (Pearson et al., 2008; Lagouge et al., 2006). In addition, Shan et al. (2008) reported that Resveratrol decreases adipose tissue in pigs in time-dependent fashion by increasing the transcription rates of porcine adipose triglyceride lipase (pATGL), which is an adipocyte-specific, lipid-hydrolyzing enzyme. On the other hand, In white adipose tissue (WAT), Resveratrol causes a notable decrease in beta-defensins, a family of antimicrobial, inflammation-mediating substances involved in adaptive and inherent immunity (Pearson et al., 2008), and Interleukins-6 and -8, which causes chronic inflammation that underly atherosclerosis and type 2 diabetes. Other studies, on the other hand, have identified Resveratrol's ability to regulate other tissues such as adipose tissue, indirectly by regulating its neurotransmitter. Dasgupta and Milbrandt (2007) show that both in vivo and in vitro, Resveratrol increases the activity of neuronal Adenosine Monophosphate-activated Kinase (AMPK). The increase in AMPK, in turn, improves mitochondrial regeneration, which results to energy-consuming and metabolically inefficient adaptive thermogenesis in the brown adipose tissue (BAT). Aside from increasing lipolysis, Resveratrol decreases adipose tissue by its double-bladed effect on its differentiation. First, this flavonoid prevents preadipocytes from differentiating into mature adipocytes by decreasing the production of adipocyte-specific transcription factors and enzymes. This decrease in the decrease in protein synthesis, specifically during the S phase of mitosis, may be attributed to Resveratrol's antioxidant activity. ROS were found by Lee et al. (2009) to increase adipocyte differentiation. Finally, it decreases the viability of already existing adipocytes by causing their apoptosis (Baile et

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