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Editorial

Caffeine Consumption and Induction of Obesity in the Developed World

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Editorial

The susceptibility of humans to obesity favors the deposition of fat with fat deposition in man much higher than compared with other species. The global increase in Non Alcoholic Fatty Liver Disease (NAFLD) is linked to the obesity epidemic and appetite dysregulation [1-3] is possibly the major defect in man with over eating the major cause of adiposity and NAFLD. Environment and genes have been indicated to play a major role in the global obesity epidemic with alterations in the appetite centre early in life relevant to the defective adipose tissue-liver crosstalk [4]. Induction of NAFLD may favor adipogenesis with consumption of high calorie diets, lack of essential nutrients, palmitic acid rich diets, nuclear receptor inhibitors, magnesium deficiency and bacterial lipopolysaccharides all relevant to liver inflammation and steatosis (Figure 1).

After consumption of a fat meal fatty acids are transported to the adipose tissue for storage or metabolism. The liver is involved with rapid clearance of lipoproteins that increase in the blood plasma after a fat meal with fatty acids rapidly metabolized by mitochondria in hepatocytes [1]. The crosstalk between the adipose tissue and the liver for storage and metabolism of fat is important to prevent adiposity and adipogenesis [5-7] and this interplay between the adipose tissue and liver becomes defective in overweight individuals early in life. Overweight is defined with a Body Mass Index (BMI) between 25 and 30 with obesity defined as a BMI > 30 (weight in Kg/[height m]^2). Interests in caffeine and adipogenesis has accelerated with relevance to treatment of overweight/obese individuals and the prevention of adipocyte dysfunction early in life linked to caffeine ingestion important to the sympathetic nervous system, lipolysis and lipid oxidation [8-12].

Coffee contains caffeine and other components with the effects of other components also relevant to the reduced risk for obesity [10]. Coffee but not caffeine determines the expression of the Peroxisome Proliferator-Activated Receptor γ (PPAR γ) a transcription factor controlling the differentiation of adipocytes with PPAR γ [11] also closely linked to other nuclear receptors associated with fat metabolism in the adipose tissue and liver [13]. Caffeine in humans has become important to appetite regulation and is now considered to act as an appetite suppressant [14]. The dose of caffeine used in obesity [15] has become important with relevance to the NAFLD epidemic [16-22] and pharmacokinetics of caffeine [23,24] completely impaired in the liver (NAFLD) in overweight/obese individuals. Inducing factors for NAFLD (Figure 1) override the beneficial effects of caffeine on adipocyte/liver fat metabolism [25,26].

With excess transport of caffeine to the brain and induction of Type 3 diabetes [24,27]. The effects of brain and interactions between sympathetic/parasympathetic innervation (autonomic nervous system) are closely connected to fat metabolism in the adipocyte and liver with defective interactions (brain, liver and adipocytes) induced by excess brain caffeine in overweight/obese individuals with relevance to appetite dysregulation, circadian rhythm abnormalities, accelerated brain aging and Type 3 diabetes [2,3,28-30].

The effects of caffeine treatment to prevent adipocyte dysfunction is superseded in overweight/obese individuals by magnesium deficiency in overweight and obese individuals [31-35]. Magnesium deficiency may occur by inadequate intake in children and young adults and may induce obesity and brain disorders with relevance to defective brain and peripheral organ (adipose tissue, liver, and

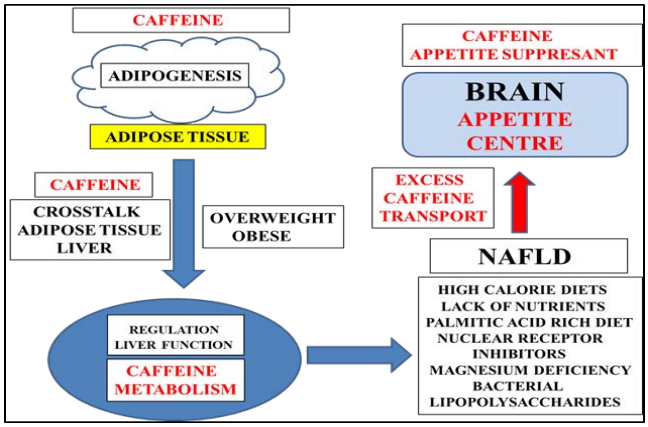

Figure 1: Caffeine consumption in overweight and obese individuals has become important to maintain the adipose tissue-liver crosstalk that is important to maintain lipid metabolism in adipocytes and hepatocytes. Obese individuals with NAFLD should be carefully monitored with relevance to diet, lifestyle and environment interactions that may prevent NAFLD induction and defective liver caffeine metabolism. Caffeine is an appetite suppressant but obesity and NAFLD may allow excessive caffeine transport to the brain with induction of brain disorders that are connected to the adipose-tissue crosstalk.

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pancreas) interactions. Caffeine may induce magnesium deficiency (Figure 1) [24] and the plasma concentrations of caffeine and magnesium should be carefully assessed in individuals with NAFLD [16-22] to prevent hyperglycemia induced mitochondrial apoptosis [36-39]. LPS as an inducing factor for obesity [40,41] has become of major concern with its effects overriding the beneficial effects of caffeine with LPS involved in magnesium deficiency [36,42,43]. Various caffeine sources for caffeine consumption include coffee, coca-cola, herbs and plants and excessive intake should be avoided to prevent caffeine toxicity to the liver and brain in childhood obesity.

Conclusion

Humans as a species are more susceptible to obesity and NAFLD with low magnesium intake connected to mitochondrial apoptosis in adipocytes/hepatocytes relevant to impaired mitochondrial oxidative capacity and systemic insulin sensitivity. Caffeine intake to improve lipid metabolism in obesity should be carefully controlled with relevance to defective caffeine metabolism in obese individuals with NAFLD. Defective caffeine metabolism in obese individuals can allow excessive caffeine transport to the central nervous system with effects on appetite regulation and induction of Type 3 diabetes. Connections between LPS and magnesium deficiency may override the beneficial effects of caffeine on the maintenance of the adipocyte-liver crosstalk important to the prevention of NAFLD in obesity.

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