Nutrigenomic Diets and Caffeine Determines the Intelligence Quotient and Thinking in Developing Countries
Ian James Martins*
Centre of Excellence in Alzheimer’s Disease Research and Care, School of Medical Sciences, Edith Cowan University, Joondalup Drive, Joondalup, Australia

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*For Correspondence
Ian James Martins, Centre of Excellence in Alzheimer’s Disease Research and Care, School of Medical Sciences, Edith Cowan University, 270 Joondalup Drive, Joondalup, Western Australia 6027, Australia. Tel: +61863042574.
E-mail: i.martins@ecu.edu.au

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ABSTRACT

Scientists have assessed various genes with relevance to human intelligence in the developing and developed world. Low intelligence quotients (IQs) have been linked to environmental factors and genes in the developing world. Developing world individuals lack the anti-aging gene Sirtuin 1 (Sirt 1) that determine brain circuitry circuits with relevance to information processing, thinking and synaptic transmission. Diets that contain caffeine, patulin, bacterial lipopolysaccharides (LPS) and xenobiotics interfere with IQs in diabetic individuals. Inactivation of genes such as Sirt 1 and the cholesterol interacting protein synatosomal associated protein 25 (SNAP 25) interfere with intelligence and insulin secretion with importance to adaptation and the survival of the species.

INTRODUCTION

Interests in intelligence and genes [1-4] has become of importance to international health organizations in the developing and developed world. The understanding between gene and intelligence quotients (IQ) has accelerated with relevance to learning and memory disorders in diabetes that has now reached an epidemic in the developing world [5]. Environmental factors in the developing world may be responsible for low IQs but genetic factors are fixed and cannot be altered in individuals from the developing world [1,2]. In diabetes the gene for SNAP 25 is now believed to be involved in learning and memory and strongly associated with human intelligence [3,4]. SNAP 25 is a synaptic protein that is associated with diabetes [6] and defective SNAP 25 membrane interactions associated with ingestion of bacterial lipopolysaccharides (LPS) and patulin [5,6]. Survival of the species now may involve nutritional interventions that regulate genes that improve intelligence associated with synaptic connections and insulin resistance. The gene Sirtuin 1 (Sirt 1) that is regulated by nutrition may be connected to intelligence with Sirt 1’s critical role in synaptic plasticity, learning and memory [6]. Sirt 1 is important to the regulation of circadian rhythms [7,8] with effects on sleep that determine IQ and thinking with effects on neuroplasticity [9-11]. Nitric oxide (NO) is important to memory and learning [12,13] with Sirt 1 regulation of circadian rhythm connected to NO homeostasis, short-term memory and mitochondrial apoptosis [8]. Sirt 1 posttranslational alterations of SNAP 25 involves Sirt 1 mediated NO regulation of SNAP 25 [3,4,6,14]. Dietary effects on SNAP 25 and Sirt 1 in neurons may be irreversible [6] with relevance to brain cholesterol toxicity that determines the survival of various species. IQ may be determined by cholesterol interacting proteins [3,4,6] that determine the brain circuits with relevance to information processing and synaptic transmission. Diabetes and the developing world indicate rapid early neurodegeneration induced by xenobiotics in the food, air and water with effects on mitochondrial apoptosis in neurons [15] that may be an important factor to learning and memory. Low calorie diets that maintain nutrigenomic Sirt 1 regulation of circadian clocks are relevant to synaptic plasticity with hepatic xenobiotic metabolism important to thinking and IQ in individuals in the developing world [5]. Caffeine has been used as a cognitive enhancer [16,17] but in diabetes and NAFLD [8] its delayed clearance may be relevant to neuron apoptosis and interruption of human circadian time keeping [7,8] with sleep disruption effects relevant to IQ scores [10,11]. In the developing world, caffeine, LPS and patulin in the diet should be carefully assessed to prevent Sirt 1...
gene repression \[18\] and to improve IQ and thinking in various species. Environmental factors such as diet and lifestyle factors that increase caffeine, LPS, xenobiotics and patulin in the brain may interfere with learning and memory that are strongly associated with human intelligence. Diabetes has indicated that rapid early neurodegeneration is associated with low IQs and that nutritional interventions early in life may allow genetic adaptation with improvement in intelligence relevant to man and the survival of various other species.

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REFERENCES