

Yesterday's Snack for Today's Thoughts

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Metabolic processes determine brain function including decision making, learning and memory. Metabolism depends on dietary patterns and therefore diet can affect the brain and modulate cognition and behaviour. In the past decade, scientific literature became significantly richer in data regarding the connection between cognitive function and diet. However, consistent conclusions to backup public health strategies that could promote cognitive resilience and performance based on dietetic interventions remain elusive. Part of the problem resides in the complex network of interactions between food and the brain. Available data presage a devastating effect of today's unhealthy Westernized eating habits on cognition and propensity to several neurodegenerative diseases. Yet, as dietary patterns seem to affect aging and longevity, today's knowledge also unveils the potential for longer, healthier and brainier lives [1-3]. As neuronal substrates for learning and memory, the hippocampus and frontal cortex seem to be extremely sensitive to any metabolic disruption caused by unhealthy eating and corrective dietetic interventions seem to afford protection and reverse damage [4-8,3].

Several animal models show diet-induced cognitive dysfunction and impaired synaptic plasticity in several brain regions [9,5]. Nonetheless, most are genetic or pharmacological models and use diets which are frequently too extreme in composition or delivery methods; thus, not being representative of the natural history of human disease or exposure. Most studies use long-term exposure resulting in multisystemic health issues and making a putative direct link between diet and cognition hard to prove. These animal models provide faster measurable effects and have yielded revolutionary discoveries on the interconnections between diet, metabolic (dys) function and cognition; albeit at the expense of unalienable limitations that foil safe extrapolation to humans in most, if not all cases. Significant human studies are even more challenging to design and have not been overall very successful in providing solid conclusions as intelligently reviewed by Vauzour et al. [3]. Despite this, evidence suggest that diet should substantially influence cognitive health based on significant associations only missing appropriate confirmation of causality. In a simplistic way, we know from common sense that diet affects the mind and most probably modulates behaviour and determines cognitive health over time; yet, the complex network of interactions between our brains and food have frustrated definite and practical conclusions. There is much controversy around this subject and a strong argument suggest that diet affects cognitive health secondarily to its multisystemic effects (i.e. cardiovascular and peripheral metabolic dysfunction) and not necessarily by a primary effect on brain cells. Furthermore, it could be argued that pursuing a diet for cognitive protection would be deemed unnecessary since we are already implementing dietary strategies applied to the well-known more traditional side-effects of unhealthy eating.

Fortunately, researchers are now aiming at more true-to-life models based on realistic dietary manipulations of healthy wild-type animals or healthy humans. Remarkably, a few of those studies now suggest that diet may significantly affect cognitive functions acutely and before any other significant consequences of chronic unhealthy eating. A few days of exposure to a high-fat diet might be sufficient to impair the performance of healthy adult human volunteers in tasks measuring attention and memory retrieval [10,11]. A similar high-fat ketogenic

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diet was previously shown to cause a mild and transient cognitive decline, but still being beneficial on the long-term, even among human subjects [12,6]. The overall benefits of ketogenic, hypocaloric or low-carbohydrate diets, especially on healthy organism, is still a matter of debate. Besides, it is highly relevant if the "high-fat" also means "high-energy" or "high-sugar" diet and the relative composition of fatty acids and other nutrients. High-fat normal or low-energy diets are expected to be ketogenic and neuroprotective, especially if low in sugars, adequate in protein composition and rich in unsaturated fatty acids like ω -3. Meanwhile, increased intake of sugar and saturated fats seem to be particularly associated with cognitive dysfunction in humans [1,9,13,14,3]. As a bottom line, we should not draw hasty conclusion as acutely changing diets may cause the need for biological and psychological adaptations, transiently affecting behaviour and cognition, and not being illustrative of a sustained exposure. Curiously, sugar has not been as well studied as fat, and recent animal studies suggest it may be even more deleterious for learning and memory [15-17] and able to disrupt behaviour and neuronal plasticity [5,8]. A few studies even suggest that saturated fats and/or refined carbohydrates, as in Western diets, may hinder cognitive development in humans [18-20]. Sugars are a major part of the Western diet resulting in its usually high glycaemic index. Nonetheless, a thorough systematic review by Philippou and Constantinou [21] could not conclude on the influence of the glycaemic index on cognition due to data inconsistencies. In this review, the authors perceptively describe how future research should consider comorbidities, age, type of cognitive domain tested and timing during the postprandial period, among other variables.

Tran and Westbrook [22] recently conducted a series of well-designed experiments on this subject. The authors focused on the acute cognitive and behavioural side effects of Western diets and designed the experiments to emulate human exposure. As described, Sprague-Dawley rats had ad libitum access to a 10% sucrose solution, lard and a range of "supermarket foods", in addition to standard chow and water. Interestingly the rats consumed the available westernized diet similarly to humans with increased energy intake in association with increased fat and sugar ingestion. The experiments assessed spatial memory and were designed to distinguish between geometrical and featural place-recognition. The westernized diet impaired geometrical orientation after only 5 days and persistently for 3 weekly

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tests. Curiously, featural place-recognition was unaffected and the rats could remember the location of objects if provided with non-geometrical visual landmarks. An ingenious experiment also verified that when faced with geometrical "blindness" the animals relied on visual features and, as a by-product, they could bypass geometrical confusion and perform better than controls if provide with visual landmarks. This happens because geometrical spatial memory seems to be dominant over featural in normal healthy rats.

In a flowing experiment, Tran and Westbrook [23] found that the impairment in the place-recognition tests caused by the Westernized-diet could be reversed by switching the animals to healthy chow. Interestingly, rats switched from chow to the unhealthy diet failed to express any significant impairment in place-recognition. The authors then designed an experiment combining diet and training history to assess if age or training could be responsible for this negative result, considering that the rats were 3 weeks older and had 3 weeks of previous testing. Generally, trained animals performed better than their naïve counterparts, even if performing poorly while on the unhealthy diet before being switched to chow. Remarkably, even untrained rats recovered gradually after switched to chow still suggesting at least partial reversibility. It seemed that training, even on the unhealthy diet, afforded a learning reserve later unlocked by the healthy diet, as these animals quickly achieved the performance of chow trained rats. Untrained rats were not protected against the Westernized diet regardless of age at exposure. The authors also concluded that training on chow afforded immunity to the Westernized diet as rats maintained their performance on the tests. At this point Tran and Westbrook [23] also perceptively postulated that training could be preserving the rats ability to use spatial geometry previously shown to be affected by the unhealthy diet. Critically, not only they found that training did not preserve the normal pattern of geometry over feature for spatial orientation, but concluded it only afforded cognitive protection against the Westernized diet for the specific place and task of the training. These are very interesting results as this duality of spatial orientation processing has been extensively described including in humans and can be disrupted in genetic disease with cognitive dysfunction such as in Williams syndrome [24,25].

A few other studies also suggest that unhealthy diets can acutely impair memory function immediately in the postprandial period. This could be somewhat expected in metabolically dysfunctional humans (i.e. diabetics), but was also found in non-diabetics, non-obese and even in young adults and children [26]. This makes us think about how meal patterns and composition, including breakfast and snacks, may affect our daily cognitive abilities and development. For an interesting review on children see Benton and a.i.s.b.l [27].

Overall, these are very interesting results and well-designed experiments such as Tran and Westbrook [23] are paramount to better understand the intricate connections between body and mind when it comes to food and eating behaviours. Duelling on the interconnections between food and related cues, eating behaviours, stress, emotions, reward, impulse control, addiction, personality and several other variables, is out of scope for this short commentary. However, these should always be kept in mind and more so when considering human subjects. In a worldwide crisis around diet-related health problems it seems that not even "our own selves" may be safe from what "we had last week". This should be regarded as a sensitive subject and careless statements can potentially further fuel the already saturated market of mostly unproved and potentially dangerous dietary and exercise

products. Therefore, scientifically based guidelines concerning diet, cognition and behavior are paramount to mitigate both the predicted cognitive risk of today's most popular diets and the emergent market of unproven interventions. Some may say that such research will not bring new insight on public health measures stating that all possible interventions are already justified based on traditional adverse effects of unhealthy diets. However, it seems that diet can quickly and significantly affect cognition and behaviour before other changes and this could have implications in several aspects of human life. For instance, we could design diets or at least prevent eating errors so that learning would be potentiated when most needed. Children and younger adults are particularly vulnerable to eating errors during the main stages of cognitive and behavioural development. For instance, it is common sense that studying causes stress and frequently induces unhealthy eating of sweet and salted snacks. Notwithstanding, older adult can also be susceptible, including in their cognitive performance, resilience and risk of neurodegeneration. Meal patterns may also affect our cognitive abilities, including executive functions, and in a world of diverse and changing working schedules, an adjusted meal pattern could be significantly beneficial. Certain tasks and hence jobs could also benefit from specifically designed diets to accomplish best health and performance benefits.

Idealisations aside, food is like a drug, it is an exogenous substance that our bodies assimilate and use every day to maintain overall homeostasis. As in drugs, food can be either protective or aggressive towards our internal milieu and countless food-derived substances have significant bioactive properties. Therefore, food is a daily necessity that can cause harm which drives the creation of an endless arsenal of medicinal drugs. Research on food should offer us a more efficient, natural and enjoyable way of specifically promoting cognition and preventing neurodegenerative diseases.

Competing Interests

The authors declare that no competing interests exist.

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