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A high-fat high-sugar diet-induced impairment in place-recognition memory is reversible and training-dependent

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ABSTRACT

A high-fat high-sugar (HFHS) diet is associated with cognitive deficits in people and produces spatial learning and memory deficits in rodents. Notable, such diets rapidly impair place-, but not object-recognition memory in rats within one week of exposure. Three experiments examined whether this impairment was reversed by removal of the diet, or prevented by pre-diet training. Experiment 1 showed that rats switched from HFHS to chow recovered from the place-recognition impairment that they displayed while on HFHS. Experiment 2 showed that control rats ("Untrained") who were exposed to an empty testing arena while on chow, were impaired in place-recognition when switched to HFHS and tested for the first time. However, rats tested ("Trained") on the place and object task while on chow, were protected from the diet-induce deficit and maintained good place-recognition when switched to HFHS. Experiment 3 examined the conditions of this protection effect by training rats in a square arena while on chow, and testing them in a rectangular arena while on HFHS. We have previously demonstrated that chow rats, but not HFHS rats, show geometry-based reorientation on a rectangular arena place-recognition task (Tran & Westbrook, 2015). Experiment 3 assessed whether rats switched to the HFHS diet after training on the place and object tasks in a square area, would show geometry-based reorientation in a rectangular arena. The protective benefit of training was replicated in the square arena, but both Untrained and Trained HFHS failed to show geometry-based reorientation in the rectangular arena. These findings are discussed in relation to the specificity of the training effect, the role of the hippocampus in diet-induced deficits, and their implications for dietary effects on cognition in people.

1. Introduction

The modern diet is rich in saturated fats and refined carbohydrates. It has been known for some time that excessive intake of this diet results in increased body weight, even obesity, and a range of adverse health effects, including cardiovascular disease, metabolic syndrome, and type 2 diabetes (e.g., Apovian & Gokce, 2012; Despres & Lemieux, 2006; Kahn, Hull, & Utzschneider, 2006; Weiss et al., 2004). More recent evidence has begun to link excessive intake of this diet with cognitive impairments. Prospective studies have shown that middle-aged and elderly adults whose diet is high in saturated fats and refined carbohydrates are at increased risk for neurological disorders, such as Alzheimer's disease, and a faster rate of normal age-related cognitive decline (e.g., Eskelinen et al., 2008; Kalmijn et al., 1997; Morris, Evans, Bienias, Tangney, & Wilson, 2004; Whitmer, Gunderson, Barrett-Connor, Quesenberry, & Yaffe, 2005). Such studies have also shown that intake of this diet in 14-year-old adolescents was negatively correlated with their performance at age 17 in tasks assessing visual-spatial learning and memory, even after controlling for a range of factors (Nyaradi et al., 2014). Intake of this diet has also been shown to be negatively correlated with school performance, especially with self-reported difficulties in mathematics (Overby, Ludemann, & Hoigaard, 2013). Similarly, body mass index in a large sample of children was negatively correlated with visual-spatial intelligence, again after controlling for socioeconomic, social, physical activity, and health factors (Li, Dai, Jackson, & Zhang, 2008). Cognitive deficits have also been reported after relatively short exposures to a high saturated fat diet; healthy adults who ate such a diet for 1 week performed worse on tasks measuring attention and speed of retrieval than they had prior to the diet (Edwards et al., 2011; Holloway et al., 2011).

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Rodent models have confirmed that diets rich in fat, sugar, or both fat and sugar produce cognitive impairments. These findings have been most notable in task that require the hippocampus and surrounding cortices, such as place-recognition memory and the use of spatial relations to navigate to the hidden platform in the water maze or to avoid the arms that have been visited and depleted of food in the radial arm maze (e.g., Molteni, Barnard, Ying, Roberts, & Gomez-Pinilla, 2002; Pistell et al., 2010; Ross, Bartness, Mielke, & Parent, 2009). Rodent models have even shown that impairments in such tasks can be detected within as little as one week of exposure to such diets, well in advance of diet-induced increases in bodyweight and associated adverse health effects, such as metabolic disorder (Beilharz, Maniam, & Morris, 2014; Kanoski & Davidson, 2010; Murray et al., 2009).

In a previous study, we (Tran & Westbrook, 2015) exposed rats to a diet that contained chow supplemented by a range of energy rich foods eaten by people and tested them every week for three weeks in place- and object-recognition memory tasks. These tasks consisted in familiarizing the rats with two identical objects and testing their memory for the location and identity of the objects several minutes later. In the place-recognition task, the two objects were the same as those in familiarization but one of them occupied a new location; in the object-recognition task, the two objects occupied the same locations as in familiarization but one of them was a new object. We found that rats fed the energy rich diet exhibited just as good memory for object identity but worse memory for object location than the control rats. Thus, the diet spared perirhinal-dependent object-recognition memory (Murray & Richmond, 2001) but impaired hippocampal-dependent place-recognition memory (Mumby, Gaskin, Glenn, Schramek, & Lehmann, 2002). This impairment was detected within one week of exposure to the energy rich diet, persisted across the tests conducted in each of the next two weeks, and was due to an inability to process geometrical information, including distance and direction. The aim of the present experiments was to determine conditions that reversed or prevented this diet-induced impairment in place-recognition memory.

2. Experiment 1

This experiment examined whether the impairment in place-recognition memory was reversed by removal of the high-fat high-sugar (HFHS) diet. As far as we are aware, two studies have examined recovery from a diet-induced impairment in cognition. Maesako et al. (2012) reported that amyloid precursor protein (APP)-overexpressing transgenic mice fed a high fat (HF) diet for 20 weeks exhibited worse performance in the water maze while on the diet than mice fed chow. They also fed another group of mice the HF diet for 10 weeks and then exposed them to chow for the remainder of the experiment. These mice showed better performance on the water maze than those fed the HF diet for 20 weeks, indicating either that the shift to chow had attenuated the deficit in the water maze or that the 10 weeks of exposure to the HF diet had produced a less severe deficit than had the 20 weeks. Sobesky et al. (2014) used a context fear conditioning protocol, which consisted in shocking rats shortly after exposure to a pre-exposed context. They found that rats fed chow for 20 weeks acquired fear of that context, indicating that they had learned about the context across pre-exposure, whereas rats fed a HF diet for 20 weeks acquired little or no fear of the immediately shocked context, indicating a failure to learn about the context across pre-exposure. Critically, rats fed the HF diet for 20 weeks and then chow for 4 weeks also acquired fear of the context, showing that 4 weeks of chow had been sufficient to rescue the deficit induced by the prior 20 weeks of exposure to the HF diet.

The results reported by Sobesky et al. (2014) show that shifting rats from a HF diet to chow rescues the impairment in context learning/context fear conditioning. The present experiment had two aims. The first was to show that shifting rats from the HFHS diet to chow also rescued the deficit in place recognition memory. The second aim was to assess how long it took for the shift to rescue the deficit induced by the HFHS diet. More specifically, we asked whether one week of exposure to chow was sufficient to rescue the deficit that had been observed in rats fed the HFHS diet for 3 weeks and tested each week.

2.1. Method

2.1.1. Design

This experiment was a continuation of a previous published study (Tran & Westbrook, 2015 Experiment 1) and used the same rats as subjects. These rats had been tested on both a place- and object-recognition memory task on their respective dietary conditions (chow control and HFHS). Across three weekly tests, HFHS rats performed comparably to chow rats on the object task, but were impaired on the place task. One day after the final test (the start of the current experiment), half of the rats in the chow and HFHS conditions continued their respective diets (groups Chow-Chow and HFHS-HFHS), and the remaining rats were switched to the other diet condition (groups Chow-HFHS and HFHS-Chow). All four diet groups then received three weekly test sessions on the place and object tasks (Table 1).

2.1.2. Subjects

Subjects were 32 male Sprague-Dawley rats used in Experiment 1 of Tran and Westbrook (2015). Rats were obtained from a commercial supplier (Animal Research Center, Perth, Western Australia, Australia) and weighed between 200 and 300 g on arrival in the laboratory. They were housed in plastic tubs (width = 65 cm, length = 40 cm, height = 32 cm) in a climate-controlled colony room (lights on from 7:00 a.m. to 7:00 p.m.). There were 4 rats in each tub and each rat had been handled for 2–3 min a day for 5 days prior to the start of the dietary manipulation from Experiment 1 of Tran and Westbrook (2015). All procedures were approved by the Animal Ethics Committee, University of New South Wales.

2.1.3. Diet

The diet for chow rats consisted of ad libitum access to water and standard laboratory chow. The nutrient breakdown of the chow diet was approximately 25% protein, 15% fat, 55% complex carbohydrates, and 5% sugar. In addition to the standard chow and water, HFHS rats had access to a 10% sucrose solution, lard, and a range of supermarket foods (various types of cakes and biscuits), which were replaced daily. Individual intake of the HFHS diet varied between home cages (4 rats) and across days, with the average nutrient breakdown consumed by a typical rat, across the duration of the experiment, being approximately 5% protein, 40% fat, 15% complex carbohydrates, and 40% sugar. Further details of each food contained in the HFHS diet can be found in Tran and Westbrook (2015).

2.1.4. Apparatus

The place- and object-recognition memory tasks were conducted in a square arena (width = 60 cm, length = 60 cm, height = 50 cm) constructed out of wood and coated with oil-based paint. The walls of this arena were uniformly black. The objects used were commercially available household items (maximum volume: width = 10 cm, length = 10 cm, height = 20 cm) that varied in material, texture, and shape (e.g., tin can, glass jar, plastic bottle; see Tran and Westbrook (2015) for a photograph of the