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Effects of high fat or high sucrose diet on behavioral-response to social defeat stress in mice

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ABSTRACT

Stress increases risk for psychopathology, and diet may moderate the impact of stress on mental health. A "Western" diet has been linked to psychopathology in humans; animal studies also show that diet can influence negative valence behavior in the presence or absence of stress, but findings are inconsistent. Contradictions in existing studies may result from differences in macronutrient content of diets and presence of metabolic syndrome. The present study exposed mice to 10 days of high fat or high sucrose diet concurrent with social defeat stress exposure and examined negative valence behavior at acute (< five days) and long-term (> 30 days) time points after stress/diet exposure. Predictably, stress increased negative valence behavior in the social interaction, open field, elevated zero maze, and tail suspension tests at the acute time point. While most stress-induced behaviors normalized after the 30-day recovery period, social avoidance was still highly significant for stress-exposed mice, supporting the hypothesis that avoidance of a trauma-related cue persists beyond non-specific anxiety-like behaviors. Supporting the hypothesis that an unhealthy diet contributes to psychopathology, non-stressed mice fed high fat or high sucrose diets spent less time exploring the center of the open field. This effect was no longer present after a 30-day recovery. Intriguingly, mice previously fed either high fat or high sucrose diets exhibited increased rearing behavior in the elevated zero maze 30 days post stress and diet exposure. This finding could be evidence that short-term diet administration can initiate a long-term increase in risk-assessment behavior.

1. Introduction

Exposure to chronic stress or a traumatic event increases the risk of developing psychiatric disorders, most notably major depressive disorder (MDD) and post-traumatic stress disorder (PTSD). Increasingly, researchers are also recognizing the importance of diet in mental health. A "Western" diet high in fat and sugar has been linked to psychopathology in humans (Jacka et al., 2010a, 2010b; Sinclair et al., 2016). A diet rich in processed meats, sugary foods, and high-fat dairy products is associated with an increased risk for depression (Akbaraly et al., 2009). Even after controlling for socioeconomic status and age, men who consumed a diet high in processed meats, fried food, and carbohydrates had a higher prevalence of symptoms of depression (Le Port et al., 2012). Longitudinal studies show that women who consume a diet high in sugar have a higher incidence of depression (Gangwisch et al., 2015). Experimentally, HSD administered to healthy weight and overweight humans resulted in higher depressive symptoms for both groups (Breymeyer et al., 2016).

The effects of diet on stress-sensitivity have also been explored in animal models, but results are inconsistent. Some studies support the hypothesis that an "unhealthy" but palatable diet decreases the impact of stress exposure. For example, when C57BL/6J mice were fed high fat diet (HFD, 45% kcal fat, Research Diets), the effects of a social defeat stress (SDS) x overcrowding procedure were minimized, measured by decreased depressive-like behavior in the forced swim test (FST) and decreased anxiety-like behavior in the light-dark box, compared to controls fed a low fat diet (LFD, 10% kcal fat, Research Diets) (Finger et al., 2011). Similarly, Wistar rats fed HFD (61% kcal fat) were protected from effects of SDS compared to rats fed a low fat, high carbohydrate diet (63% kcal carbohydrate), measured by normal locomotor activity and normalized body weight gain (Buwalda et al., 2001). Access to palatable diet with 45% kcal fat content also reduced stress-sensitivity in juvenile Sprague-Dawley rats exposed to seven days SDS (MacKay et al., 2017).

Other studies support the hypothesis that HFD exacerbates the impact of stress on behavioral and endocrine outcomes and have

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deleterious effects on behavior even without additional stressors. For example, in C57BL/6J mice, SDS plus 30 days on a HFD (42% kcal fat) led to greater social avoidance in the social interaction test compared non-stressed mice or SDS mice on control diet (4% kcal fat) (Chuang et al., 2010). C57BL/6J mice fed HFD (60.3% kcal fat, Harlan Laboratories) had increased anxiety-like behavior in the open field test (OF) and elevated zero maze (EZM), cognitive impairment in the Y maze, and increased depressive-like behavior in the FST compared to controls fed a standard diet (18% kcal fat, Harlan Laboratories) (Almeida-Suhett et al., 2017). In mice, six weeks of HFD (58% kcal fat) caused anhedonia in a sucrose-preference test and anxiety-like behavior in the elevated plus maze (EPM) compared to mice fed an ingredient-matched low fat control diet (11% kcal fat) (Sharma et al., 2013). Similarly, 12 weeks of HFD (58% kcal from fat, research diets) decreased time in the open arms of the EPM and time in the center of the OF, and increased immobility in the FST (Sharma and Fulton, 2013). Two months of HFD (45% kcal fat) in CD-1 mice increased negative valence behaviors in the OF and hole board test, but actually decreased immobility in the FST (Del Rosario et al., 2012).

While many diet x stress reports focus on HFD, high sucrose diets (HSD) may also increase risk for psychopathology in humans, and HSD has also been shown to modify the impact of stress in animal studies. Twelve weeks of HSD (74.2% carbohydrate, 5.8% fat, 20% protein, custom made of condensed milk, sugar, and Labina chow diet) in BALB/c mice, exacerbated the impact of 2-h restraint stress on anxiety-like behavior in the EPM, enhanced aversive memory in a fear conditioning task, and increased depression-like behavior in the tail suspension test (TST) (Santos et al., 2016). In absence of stress, Wistar Hannover rats fed HSD (25% sucrose) had increased anxiety-like behavior in the OF (Pinto et al., 2016). Other studies show that HSD decreases anxiety- and depressive-like behavior. Thirteen weeks on a custom-made "cafeteria" diet high in both fat and sugar decreased Swiss mice immobility in the FST and TST and increased time in the open arms of the EPM (Leffa et al., 2015).

The majority of animal studies employ long-term diet administration, which causes numerous downstream effects including metabolic syndrome, complicating the interpretation. Although less studied, short-term diet administration is also associated with neurological, physiological, and behavioral impacts. For example, impaired novel object recognition was observed after just one week of HFD (Gainey et al., 2016), and 10-days of HFD altered the cellular composition of the arcuate nucleus of the hypothalamus in mice (Balland and Cowley, 2017). Furthermore, previous research has suggested that the influence of HFD on anxiety-like behavior may vary by duration of diet exposure (Sweeney et al., 2017), providing additional rationale for evaluating the impact of a shorter term diet administration.

Very few studies have compared the influence of more than one diet on behavior, some animal models neglect to identify the source and macronutrient content of diets (e.g. (Baran et al., 2005)), others use a HFD that also has a high sucrose content (e.g. (Finger et al., 2011)), and still others employ a "control" LFD that is actually high in sucrose (e.g. (Balsevich et al., 2014)), highlighting the need for additional research on this topic. The present study employs a high fat/low sucrose diet (HFD), a high sucrose/low fat diet (HSD), and a control diet (chow). This study tests the hypothesis that unhealthy diets will influence sensitivity to social stress and allows us to determine if the effects differ for HSD and HFD. To test this hypothesis, mice are exposed to 10-days of SDS with concurrent exposure to one of the three diets. Tests of anxiety- and depressive-like behavior are conducted at acute and long-term time points following SDS and diet exposure.

2. Materials and methods

2.1. Animals and housing

All procedures and protocols were reviewed and approved by the Grand Valley State University Institutional Animal Care and Use Committee (IACUC). In accordance with IACUC, all efforts were made to minimize pain, suffering, and number of animals used.

Male C57BL/6N (C57, n = 78) mice were purchased from Charles River Laboratories (Portage, MI) at four weeks old and were seven weeks old (~20g) at the start of testing. Male CD-1 retired breeder mice (CD-1 n = 29) were purchased from Harlan (Lansing, MI). Mice were housed in polycarbonate cages with wire tops. Mice were housed four to a cage prior to group assignment. At the start of the study, non-stress (NS) mice were housed two per cage-one on each side of a Plexiglas divider. During the 10-day SDS procedure, SDS mice were housed in a separate room in the home cage of a CD-1 mouse but separated with a Plexiglas divider. After the 10-day SDS procedure, SDS mice were moved back to the original housing room and were housed two per cage-one on each side of a Plexiglas divider. At this time, CD-1 mice were also housed in the same room, but on a different cage rack than the experimental mice.

Water and food were available *ad libitum*. Both housing rooms used in this study were temperature and humidity-controlled with a 12 h light-dark cycle (lights on at 21:00).

In a 3 × 2 design, mice were assigned to SDS or NS and one of three diets: Chow, HSD, or HFD (n = 11–14/group). Group assignments are balanced for baseline body weight and baseline behavior in the OF (See Supplement). Content and nutritional details of control Chow, HSD, and HFD are shown in Table 1. Experimental timeline is shown in Fig. 1.

Table 1
Dietary content.

		Chow	HSD	HFD
Fat	%Kcal	11.41	10.52	58.02
	Source	Cholesterol, linoleic acid, arachidonic acid, omega-3 fatty acids, porcineanimal fat	Soybean oil and hydrogenated coconut oil	Soybean oil and hydrogenated coconut oil
Sucrose	%Kcal	3.25	60.16	12.66
	Source	Cane molasses	Maltodextrin 10 and unspecified source	Maltodextrin 10 and unspecified source
Protein	%Kcal	24.13	16.44	16.44
	Source	Unspecified	Casein and DL-methionine	Casein and DL-methionine
Minerals		Calcium, Phosphorus, Potassium, Magnesium, Sulfur, Sodium, Chloride, Fluorine, Iron, Zinc, Manganese, Copper, Cobalt, Iodine, Chromium, Selenium.	Calcium, Magnesium, Potassium Citrate, Potassium Sulfate, sodium chloride, Chromium Potassium Sulfate, Cupric Carbonate, Potassium Iodate, Iron, Manganous Carbonate, Sodium Selenite, and Zinc Carbonate.	
Vitamins		Carotene; Vitamin K, A, D, and E; Thiamin; Riboflavin; Niacin; Pantothenic acid; Choline Chloride; Folic acid; Pyridoxine; Biotin; and Ascorbic acid.	Vitamin A, D3, E, B12, B6, B2, and B1; Menadione, Biotin, Folic acid, Niacin, and Pantothenic acid.	
Product Information		50017 Labdiet, St. Louis, MO	D123298 Research Diets Inc., New Bruswick, NJ	D123318 Research Diets Inc., New Bruswick, NJ

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