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Sucrose ingestion induces glutamate AMPA receptor phosphorylation in dorsal hippocampal neurons: Increased sucrose experience prevents this effect

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Abstract

Evidence suggests that meal-related memory influences later eating behavior. Memory can serve as a powerful mechanism for controlling eating behavior because it provides a record of recent intake that likely outlasts most physiological signals generated by ingestion. Dorsal (dHC) and ventral hippocampal (vHC) neurons are critical for memory, and we demonstrated previously that they limit energy intake during the postprandial period. If dHC or vHC neurons control intake through a process that requires memory, then ingestion should increase events necessary for synaptic plasticity in dHC and vHC during the postprandial period. To test this, we determined whether ingesting a sucrose solution induced posttranslational events critical for hippocampal synaptic plasticity: phosphorylation of AMPAR GluA1 subunits at 1) serine 831 (pSer⁸³¹) and 2) serine 845 (pSer⁸⁴⁵). We also examined whether increasing the amount of previous experience with the sucrose solution, which would be expected to decrease the mnemonic demand involved in an ingestion bout, would also attenuate sucrose-induced phosphorylation. Quantitative immunoblotting of dHC and vHC membrane fractions demonstrated that sucrose ingestion increased postprandial pSer⁸³¹ in dHC but not vHC. Increased previous sucrose experience prevented sucrose-induced dHC pSer⁸³¹. Sucrose ingestion did not affect pSer⁸⁴⁵ in either dHC or vHC. Thus, the present findings show that ingestion activates a posttranslational event necessary for synaptic plasticity in an experience-dependent manner, which is consistent with the hypothesis that dHC neurons form a memory of a meal during the postprandial period.

Keywords

Memory; Plasticity; Serine 831; Serine 845

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Declarations of interest
None.

1. Introduction

Food-related memory can serve as a powerful mechanism for controlling eating behavior because it provides a record of recent intake that likely outlasts most physiological signals generated by ingestion. In support, evidence suggests that meal-related memory influences later eating behavior in humans. For instance, impairing the encoding of a recently eaten meal increases the amount that is subsequently ingested, and promoting meal-related memory decreases later ingestion [1,2]. Similarly, episodic memory deficits are associated with uncontrolled eating in healthy adults [3] and patients with episodic memory-type amnesia do not remember recent meals and will eat again if presented with food despite just having eaten to satiety [4–6].

The brain regions and neural mechanisms that mediate the inhibitory effects of ingestion-related memory on future intake are not known. We have proposed that hippocampal neurons, which are critical for memory, limit energy intake during the postprandial period [7,8]. In rodents, the hippocampus is divided into a dorsal (dHC; posterior in primates) and ventral region (vHC; anterior in primates) on the basis of significant differences in connectivity, patterns of gene expression, and function [9]. dHC neurons are critical for the cognitive processes associated with the hippocampus, namely episodic and spatial memory [10–17]. dHC neurons are anatomically poised to form a memory of a meal and control eating behavior because they receive ingestion-related neural signals from several brain regions (reviewed in [18]), express a multitude of receptors for pre- and postprandial signals (e.g., leptin [19,20], insulin [21], ghrelin [22], and melanocortins [23–25]), and project to many brain areas critical for energy regulation [26–29]. We discovered that muscimol-induced inactivation of dHC neurons after the completion of a sucrose meal when the memory of the meal would be undergoing consolidation accelerated the onset of the next meal and increased intake during that meal [30].

In contrast to dHC, vHC is important for motivation, affect and emotional memory [31–36]. vHC neurons also contain receptors for numerous food-related signals [18], vHC lesions increase food consumption and body mass [37], and activation of vHC receptors for gut hormones affects food intake and food-related memory [38–41]. Also, vHC neurons project to several brain regions critical for energy intake [42–44] and manipulation of specific vHC projections to BNST, LS and prefrontal cortex influences energy intake [40,45]. Given that vHC is involved in emotional and food-related memory and that memory is mediated by multiple brain areas that each represent different information contained within the same experience [46,47], vHC neurons may also contribute to the mnemonic representation of an eating episode and inhibit subsequent intake. In support, we determined that postmeal muscimol-induced vHC inactivation also promoted the initiation of the next meal, increased the amount consumed during that meal, and increased total intake [48].

Although our muscimol findings indicate that dHC and vHC neurons limit energy intake during the postprandial period, they do not directly implicate memory or reveal potential molecular mechanisms. Principal hippocampal glutamatergic neurons play a central role in memory [49–60], and synaptic plasticity at these excitatory synapses is a critical mechanism underlying memory formation [61,62]. We reasoned that if dHC or vHC neurons control

intake through a process that requires synaptic plasticity, then ingestion should increase events necessary for synaptic plasticity in dHC and vHC during the postprandial period. The most common form of synaptic plasticity in the hippocampus depends on glutamate NMDA receptor (NMDAR)-dependent increases in intracellular calcium that activate proteins and stimulate mRNA synthesis and protein translation that act collectively to increase Glu AMPA receptor (AMPA) function in the postsynaptic cell, thereby increasing Glu signaling and synaptic strength [51–58,63–65]. In the present study, we tested the effects of ingesting a sucrose solution on two protein activation events critical for augmenting hippocampal AMPAR function and increasing synaptic strength: phosphorylation of AMPAR GluA1 subunits at 1) serine 831 (pSer⁸³¹) and 2) serine 845 (pSer⁸⁴⁵) residues. We also determined whether increasing the amount of previous experience with the sucrose solution, which would be expected to decrease the mnemonic demands associated with the current sucrose meal and diminish hippocampal involvement in remembering that meal, would also attenuate sucrose-induced phosphorylation. Quantitative immunoblotting was used to assess the effects of sucrose ingestion on pSer⁸³¹ and pSer⁸⁴⁵ of AMPAR GluA1 subunits in dHC and vHC membrane fractions [66].

2. Methods

2.1. Subjects

Adult male Sprague-Dawley rats (N = 84; aged 53 days upon arrival from Charles River Laboratories, Wilmington, MA) were housed individually in OptiRat® cages (Animal Care Systems, Centennial, CO), kept on a 12:12 h light/dark cycle with lights on in the morning, and given *ad libitum* access to pelleted food (LabDiet 5001, Purina Mills, Gray Summit, MO) and tap water in their home cages. All procedures were performed in compliance with the NIH guidelines for care of laboratory animals and approved by the Georgia State University Institutional Animal Care and Use Committee.

2.2. Sucrose experience

One week after arrival to the laboratory, some of the rats were given a 32% (w/v) sucrose solution at the same time and place daily (SUC; n = 47). We used this sucrose solution as the meal because 1) it is very palatable/rewarding to rats [67,68], 2) its stimulus qualities are more specific than meals that include fats and proteins, 3) many of its peripheral and central processing sites and mechanisms have been identified [68,69], and 4) it cannot be hoarded. Cage-control rats were given comparable handling, transport, and access to food and water but were never offered sucrose (CTL; n = 33).

For each sucrose exposure day, the rats were brought to the testing room at the beginning of the light cycle and placed into polycarbonate testing cages (22 cm × 43 cm × 22 cm) with ALPHA-dri® bedding (Shepard Specialty Papers, Richland, MI). On the first day, food and water were removed for 8 h and then the SUC rats were offered sucrose for 10min. Sucrose consumption was measured by weighing the sucrose bottles before and after the 10-min sucrose exposure. One hour later, all of the rats were returned to their home cages where chow and tap water were available *ad libitum* until the following day. The same procedure was repeated the next day, with the exception that the rats were provided with *ad libitum*

access to tap water during the 8-h period. We removed the water on the first exposure day in order to increase the likelihood that the rats would approach and consume the sucrose solution. On the third exposure day and on any subsequent exposure days, the rats were offered sucrose in the same manner, with the exception that the sucrose was given after 3 rather than 8 h. We started with an 8-h chow deprivation period in order to increase the likelihood that the rats would approach the bottle, but then decreased it to 3 h to be within the range of an average postprandial intermeal interval (ppIMI) [70].

To manipulate sucrose experience, separate groups of SUC rats were offered sucrose for a different number of days prior to euthanasia and yoked CTL rats were euthanized at the same time. Specifically, one group of SUC rats was offered sucrose for 3 days, euthanized after consuming the sucrose solution on the third day (SUC-3; $n = 19$), and a subset of CTL rats was euthanized at the same time (CTL-3; $n = 16$). The other SUC rats were euthanized after either 5 or 10 days of sucrose experience (SUC-5; $n = 14$; CTL-5; $n = 9$; SUC-10; $n = 14$; CTL-10 $n = 8$).

2.3. Tissue collection and sample preparation

On the day of euthanasia, SUC rats were offered sucrose for 10 min and then injected with sodium pentobarbital (120 mg/kg, intraperitoneal; SomnaSol, Henry Schein, Dublin, OH) 25 min after they had started to lick from the sucrose bottle. Sodium pentobarbital was injected after the same interval in a yoked CTL rat. The rats were then moved to another room, euthanized by decapitation, and dHC and vHC were dissected and then dHC and vHC samples were placed in 2 mL of resuspension buffer (1 mM NaH_2PO_4 (pH 7.0), 10 mM NaCl, 1 mM $\text{Na}_4\text{P}_2\text{O}_7$, 0.5 mM EDTA, 0.5 mM EGTA, 0.1 μM okadaic acid, 0.1 μM microcystin, 1 U/mL aprotinin, 5 mM NaF, 0.1 mM Na_3VO_4), sonicated, and centrifuged (13,000 g) for 5 min. The pellets were resuspended in 1.5 mL of resuspension buffer, sonicated, and centrifuged again (13,000 g) for 5 min. These membrane fractions were resuspended in 200 μL of 1% SDS, boiled for 10 min, and then stored at -80°C .

2.4. SDS page and immunoblotting

A DC protein assay (BioRad, Hercules, CA) was used to quantify the protein concentration in the membrane fractions. Equal amounts of protein for each sample were loaded onto 7.5% acrylamide gels for SDS-PAGE. Each gel contained dHC and vHC membrane from one CTL and one SUC rat. After SDS-PAGE, the proteins were transferred to PVDF membranes, which were then blocked at room temperature (RT) for 1 h in one of three solutions depending on the primary antibody: 1% BSA solution (pSer⁸³¹ and pSer⁸⁴⁵), 5% nonfat dry milk solution (GluA1) or 5% nonfat dry milk + 1% BSA solution (β -actin). The membranes were then incubated overnight at 4°C in primary antibody (pSer⁸³¹, pSer⁸⁴⁵, or GluA1; 1:1000; PhosphoSolutions, Aurora, CO; β -actin; 1:50,000; Novus Biologicals, Littleton, CO) and then washed three times for 5 min each in Tris-buffered saline with tween (TBS-T; 0.20 mM Tris-base, 0.14 mM NaCl, 0.81 mM tween-20, pH 7.6). The blots were then incubated at RT for 1 h in either HRP-conjugated anti-rabbit secondary antibody for pSer⁸³¹, pSer⁸⁴⁵, and β -actin (1:20,000; Sigma Aldrich, St. Louis, MO) or anti-mouse secondary antibody for GluA1 (1:20,000; Santa Cruz Biotechnology, Santa Cruz, CA), washed three times for 5 min each in TBS-T, and then incubated at RT for 5 min in a chemi-luminescent

HRP substrate (Millipore, Billerica, MA). The membranes were then immediately exposed to Amersham Hyperfilm (GE Healthcare, Buckinghamshire, UK). Bands on the film were quantified with densitometry using Scion Imaging software (National Institutes of Health, Rockville, MD). The same membranes were used to probe for pSer⁸³¹, GluA1, pSer⁸⁴⁵ and β -actin (in that order), and the antibodies were stripped from the membranes between each probe by incubating in 1x phosphate buffered saline (3.8 mM NaH₂PO₄, 16.2 mM Na₂HPO₄, 150 mM NaCl, pH 7.4) for 15min, 0.1 M glycine (pH 2.5) for 15min, and then 1.0 M Tris (pH 8.0) for 2–5 min.

2.5. Statistical analysis

Raw densitometry data were used to calculate the following ratios for each rat: pSer⁸³¹/GluA1, pSer⁸⁴⁵/GluA1 and GluA1/ β -actin. A % of control value was calculated for each rat by dividing the ratio of an individual rat by the mean ratio of control rats sacrificed at the same time. Then, the mean % of control for each group was compared to the mean of their respective controls. Statistical analyses were conducted using the Statistical Package for the Social Sciences (IBM, Armonk, NY), which assessed the data for homogeneity of variance. The measures were analyzed for normality using a Shapiro-Wilk test. Total sucrose consumption during the exposure period and on the day of euthanasia were analyzed using a 1-way between subjects analysis of variance (ANOVA) and Tukey post hoc tests. Nonparametric statistical tests were used to analyze the % of control data due to non-normal distributions and heterogeneity of variance. Specifically, Mann-Whitney U tests were used to compare the % of control values of pSer⁸³¹, pSer⁸⁴⁵, and GluA1 in SUC vs. CNTL rats euthanized at the same time. In addition, Kruskal-Wallis and Mann-Whitney U post hoc tests with Bonferroni corrections were used to compare SUC 3, 5, and 10 rats to determine whether the amount of previous sucrose experience influenced any effects of sucrose ingestion on pSer⁸³¹, pSer⁸⁴⁵, and GluA1.

3. Results

3.1. Sucrose consumption on the day of euthanasia did not differ between rats given different amounts of previous sucrose experience

As expected, the number of days that the rats were exposed to sucrose significantly affected the total amount of sucrose they consumed during the experimental period [$F(2,47) = 121.41$, $p < 0.05$]. SUC-10 rats consumed more total sucrose during the sucrose exposure period than SUC-3 [$p < 0.05$] or SUC-5 rats [$p < 0.05$], and SUC-5 rats consumed more total sucrose than SUC-3 rats [$p < 0.05$; see Fig. 1A]. Importantly, there were no significant differences in the amount of sucrose that SUC-3, SUC-5, and SUC-10 rats consumed on the experimental day immediately prior to euthanasia and tissue collection [$F(2,47) = 0.69$, $p > 0.05$; see Fig. 1B].

3.2. Sucrose ingestion elevates dHC pSer⁸³¹ and increasing previous sucrose experience prevents this effect

The findings showed that the effects of sucrose ingestion on pSer⁸³¹ in dHC depended on the amount of previous sucrose experience. Sucrose ingestion significantly elevated pSer⁸³¹ in dHC of SUC-3 rats [$U = 80.00$, $p < 0.05$ vs. CTL-3], but not in SUC-5 [$U = 28.00$, $p >$

0.05 vs. CTL-5] or SUC-10 rats [$U = 27.00$, $p > 0.05$ vs. CTL-10], indicating that increased sucrose pre-exposure prevented sucrose-induced dHC pSer⁸³¹. In support, dHC pSer⁸³¹ was significantly different between the three sucrose experience groups [$X^2(2) = 15.85$, $p < 0.05$], such that pSer⁸³¹ was higher in SUC-3 rats compared to SUC-5 [$U = 39.00$, $p < 0.017$] and SUC-10 rats [$U = 20.00$, $p < 0.017$; see Fig. 2A].

3.3. Sucrose ingestion decreases vHC pSer⁸³¹ in rats with increased previous sucrose experience

The effects of sucrose ingestion on pSer⁸³¹ in vHC also depended on the amount of previous sucrose experience. In contrast to dHC, though, sucrose consumption decreased pSer⁸³¹ in vHC of rats, and this effect was only observed in rats that had the most amount of previous sucrose exposure. Specifically, sucrose ingestion significantly decreased pSer⁸³¹ in vHC of SUC-10 rats [$U = 13.00$, $p < 0.05$ vs. CTL-10] but did not significantly affect pSer⁸³¹ in SUC-3 [$U = 108.00$, $p > 0.05$ vs. CTL-3] or SUC-5 rats [$U = 38.00$, $p > 0.05$ vs. CTL-5]. However, vHC pSer⁸³¹ did not significantly differ between the three sucrose experience groups [$X^2(2) = 5.05$, $p > 0.05$; see Fig. 2B].

3.4. Sucrose ingestion does not affect pSer⁸⁴⁵ in dHC or vHC

Sucrose ingestion did not affect dHC pSer⁸⁴⁵ in SUC-3 [$U = 136.00$, $p > 0.05$ vs. CTL-3], SUC-5 [$U = 49.00$, $p > 0.05$ vs. CTL-5] or SUC-10 rats [$U = 45.00$, $p > 0.05$ vs. CTL-10] and dHC pSer⁸⁴⁵ did not differ between the three sucrose experience groups [$X^2(2) = 0.51$, $p > 0.05$; see Fig. 2C]. Similarly, sucrose consumption also did not significantly affect vHC pSer⁸⁴⁵ in SUC-3 [$U = 123.00$, $p > 0.05$ vs. CTL-3], SUC-5 [$U = 37.00$, $p > 0.05$ vs. CTL-5] or SUC-10 rats [$U = 49.00$, $p > 0.05$ vs. CTL-10], and there were no significant differences in pSer⁸⁴⁵ between the three sucrose experience groups [$X^2(2) = 0.92$, $p > 0.05$; see Fig. 2D].

3.5. Increasing previous sucrose experience increases vHC GluA1

Sucrose consumption did not significantly affect dHC GluA1 in SUC-3 [$U = 111.00$, $p > 0.05$ vs. CTL-3], SUC-5 [$U = 48.00$, $p > 0.05$ vs. CTL-5] or SUC-10 rats [$U = 44.00$, $p > 0.05$ vs. CTL-10], and there was no significant difference in GluA1 between the three sucrose experience groups [$X^2(2) = 2.79$, $p > 0.05$; see Fig. 2E]. Sucrose consumption increased vHC GluA1 in SUC-10 rats [$U = 24.00$, $p > 0.05$ vs. CTL-10] but did not significantly affect vHC GluA1 in SUC-3 [$U = 116.00$, $p > 0.05$ vs. CTL-3] or SUC-5 rats [$U = 37.00$, $p > 0.05$ vs. CTL-5]. In addition, vHC GluA1 significantly differed between the three sucrose experience groups [$X^2(2) = 10.98$, $p < 0.05$], such that GluA1 was higher in SUC-10 rats than in SUC-3 [$U = 41.00$, $p < 0.017$] or SUC-5 rats [$U = 30.00$, $p < 0.017$; see Fig. 2F].

4. Discussion

Collectively, the present results demonstrate that sucrose ingestion affects phosphorylation of the glutamate AMPA receptor GluA1 subunit in a brain region-, serine residue-, and sucrose experience-dependent manner. Specifically, sucrose ingestion increased GluA1 pSer⁸³¹ in dHC but not vHC during the postprandial period and did not affect dHC pSer⁸⁴⁵ or the total number of GluA1 subunits in dHC. Sucrose ingestion only increased dHC

pSer⁸³¹ in rats that had been given the least amount of sucrose exposure, indicating that increased sucrose experience prevented the effects of sucrose ingestion on dHC GluA1 pSer⁸³¹. Importantly, these differences between the different sucrose experience groups were not due to differences in the amount of the sucrose solution consumed prior to euthanasia.

The present finding that sucrose ingestion increased dHC GluA1 pSer⁸³¹ is consistent with our previous results showing that sucrose ingestion elevates dHC expression of the plasticity-related immediate early gene activity-regulated cytoskeleton-associated protein (*Arc*) [71]. Of note, both GluA1 pSer⁸³¹ and *Arc* expression are dependent on activation of the glutamate NMDA receptor [72,73] and are critical for synaptic plasticity [74,75]. The sucrose-induced increase in dHC GluA1 pSer⁸³¹ is noteworthy because GluA1 pSer⁸³¹ is a key posttranslational modification that increases AMPAR conductance, promotes insertion of AMPARs in postsynaptic spines, and increases synaptic strength [76–78]. The effects of sucrose ingestion on dHC GluA1 pSer⁸³¹ cannot be attributed to novelty or the context because the rats had consumed the sucrose solution in the same context and time prior to the experimental day. Importantly, the magnitude of the sucrose-induced increase in dHC pSer⁸³¹ and the specific effect on dHC pSer⁸³¹ and not dHC pSer⁸⁴⁵ is comparable to that produced by an inhibitory avoidance learning experience [66], suggesting that increased dHC pSer⁸³¹ is a component of the molecular events underlying the memory of a meal. Given that inhibitory avoidance is a single-trial aversive task that produces a long-lasting memory, it is possible that the reward from the first sucrose exposure may have been sufficiently arousing to increase dHC pSer⁸³¹ and produce a long-term memory. However, whether exposure to a palatable sucrose solution for the first time increases dHC pSer⁸³¹ was not assessed in the present experiment in order to avoid any possible contributions of novelty.

The finding that increased sucrose experience prevented the ability of sucrose ingestion to elevate dHC pSer⁸³¹ is consistent with previous findings showing that the ability of a stimulus to activate pSer⁸³¹ depends on the history of synaptic activation [74], and with our previous results showing that increased sucrose experience attenuates sucrose-induced increases in dHC *Arc* expression [71]. Together, the *Arc* and pSer⁸³¹ results suggest that repeated consumption of the same meal may decrease the mnemonic demands associated with that meal and/or that the role of the hippocampus in remembering the last meal may diminish when the energy source is not novel and consumed habitually. This interpretation is consistent with previous studies showing that repeated training diminishes dHC involvement in a memory task [79] and with evidence showing that extensive behavioral training in learning and memory tasks or repeated exposure to the same environment decreases hippocampal expression of several molecules critical for memory, including *Arc* [80–83], phosphorylated cAMP response element-binding protein (pCREB) [84] and protein kinase M- ζ [85].

It is possible that a portion of the changes in pSer⁸³¹ that were observed was due to variables other than orosensory stimulation and ingestion of calories, such as licking and ingesting a solution. A control group that ingested water was not included because the water deprivation needed to motivate rats to drink water would confound thirst and dehydration. Importantly, the difference in pSer⁸³¹ between the different sucrose experience groups cannot be due to

such variables because they ingested similar amounts of sucrose on the experimental day. It will be important to determine in the future whether nutritionally balanced liquid or solid diets that typically contain protein, fat, and carbohydrates have comparable effects.

In contrast to dHC, sucrose ingestion did not increase vHC pSer⁸³¹ and may have *decreased* it in rats with the *most* previous sucrose experience, although the decrease could be due to the fact that sucrose ingestion increased total GluA1 in this group. The increase in total GluA1 may be due to the effects of sucrose experience on trafficking or turnover. Although sucrose ingestion does not increase vHC pSer⁸³¹ it does elevate *Arc* mRNA in vHC [48], indicating that ingestion does activate synaptic plasticity processes in vHC. The finding that sucrose ingestion increases pSer⁸³¹ in dHC but not vHC suggests that ingestion activates synaptic plasticity mechanisms in a different manner in dHC vs. vHC.

5. Conclusions

In summary, the present findings indicate that ingestion activates a protein necessary for synaptic plasticity and memory in dHC during the postprandial period when the memory of the meal would be undergoing consolidation. The present findings raise the possibility that well-defined molecular events necessary for hippocampal synaptic plasticity play a role in controlling energy intake. Increased hippocampal synaptic strength augments functional connectivity between hippocampus and other brain regions [86], thereby providing a possible mechanism for hippocampal inhibition of intake. The present findings may have implications for the prevention and treatment of obesity. Excess intake of fats and/or sugars and obesity in rodents impair hippocampal synaptic plasticity [87–89] and hippocampal-dependent memory [90–95], and hippocampal dysfunction, in turn, increases meal frequency and food intake [30,48,96–98] and promotes weight gain [37,99,100].

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Fig. 1.

Total sucrose intake during the experiment and immediately prior to tissue collection. (A) Manipulating sucrose exposure significantly influenced the total amount of sucrose consumed during the experimental period. Rats given sucrose daily for 10 days consumed more total sucrose during the experiment than rats given sucrose for 3 or 5 days, and rats given sucrose for 5 days consumed more sucrose than rats exposed to sucrose for 3 days (all $ps < 0.05$). (B) Importantly, all sucrose experience groups consumed the same amount of sucrose immediately prior to tissue collection ($p > 0.05$).

Fig. 2.

The effects of sucrose ingestion and previous sucrose experience on GluA1 phosphorylation in dHC and vHC. (A) Sucrose consumption increased pSer⁸³¹ in dHC in rats exposed to sucrose for 3 days ($p < 0.05$), but not in rats given more sucrose experience (5 or 10 days) prior to euthanasia (all $ps > 0.05$). (B) Sucrose consumption decreased pSer⁸³¹ in vHC in rats given 10 days of sucrose experience ($p < 0.05$). Sucrose consumption did not affect pSer⁸⁴⁵ in (C) dHC ($p > 0.05$) or (D) vHC ($p > 0.05$). (E) Sucrose consumption did not affect total GluA1 in dHC ($p > 0.05$). (F) Sucrose consumption increased total GluA1 in

vHC in rats exposed to sucrose for 10 days ($p < 0.05$). (G) Representative immunoblots of p-Ser⁸³¹, pSer⁸⁴⁵, GluA1, and β -actin in the dorsal hippocampus (dHC) or ventral hippocampus (vHC) for control rats (C) and rats fed sucrose for 3, 5, or 10 days.

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