Acta Biomedica Scientia

e - ISSN - 2348 - 2168
Print ISSN - 2348 - 215X

www.mcmed.us/journal/abs

Research Article

EFFECT OF PALATABLE FOOD INGESTA ON FEEDING BEHAVIOR AND BLOOD GLUCOSE LEVEL IN RATS

Manjula Suri[1,2]*, Suneel Kumar[1], Suman Jain[1], Rashmi Mathur[1]

[1]Department of Physiology, All India Institute of Medical Sciences, New Delhi-110029, India.
[2]Department of Physiology and Promotive Health, Institute of Home Economics, University of Delhi, New Delhi-110016, India.

ABSTRACT

Diabesity has reached an epidemic proportion in India as well as worldwide. The use of non-nutritive sweeteners (NNSs) has been proposed to combat this menace. NNSs are calorie less healthy sugar substitutes while researchers suggest a contrary report of NNSs. Ingestive behavior plays an important role in diabesity development as it may provide excess calories. However, ingestive behavior is a complex process with a lack of experimental animal studies. Therefore, we studied ingestive patterns in the control, sucrose or saccharin (NNSs) fed rats along with a change in their body weight and blood sugar levels. The analysis allowed a comparison of ingestive behavior such as number of ingestive bouts, bouts licking rate, bouts duration as well as water and food intake patterns. Results show that control and sucrose-fed rats ingested 5 g food in the 2 h, and 4 h, respectively while saccharin fed rats have become “nibblers” during this period. Rats ingested sucrose in 6-bouts while saccharin in 4-bouts during the experiment. The study showed a temporal variation in sucrose versus saccharin induced ingestive behavior. The findings suggest that NNS's interferes with ingestive behavior which normally contributes to energy homeostasis and may induce metabolic derangements.

Keywords :-Diabesity, Non-nutritive sweeteners, Ingestive behavior, Palatability, Sucrose, Saccharin.

Access this article online	
Home page: http://www.mcmed.us/journal/abs DOI: http://dx.doi.org/10.21276/abs.2020.7.1.2	**Quick Response code**

Received:15.07.19	**Revised**:22.09.19	**Accepted**:15.10.19

INTRODUCTION

Various studies have authenticated the linkage between overeating, overweight, and diabetes. Environmental increases in palatability and variety in the food supply are potential contributors to the rise in adiposity seen worldwide [1], which is leading to a diabesity epidemic [2]. The main characteristic of the Western obesogenic food environment is highly palatable fast food products. Schroder et al. [3] reported that fast food products are often characterized by their high content of fat and sugars, high palatability, large portion size, and high energy density. Ingestive behavior responds to a multiplicity of factors including palatability [3-7] which in turn influences food intake [7-11], water intake [12, 13], and blood glucose [12, 14-18] amongst several other parameters [19-21]. Incidentally, each of these parameters per se may influence the nociceptive pain responses [19, 22-27]. There is a robust effect of food palatability and variety on short-term food intake. The additional calories provided by sucrose intake may inversely influence the food and water intake. Low-calorie sweeteners e.g. saccharin offer a palatable alternative to caloric sugars such as sucrose and are commonly found in soft drinks, sweetener packets, fast foods, dairy products, etc. Consumption of low-calorie sweeteners has increased significantly in recent years and controversy exists surrounding their effect on feeding

Corresponding Author **Manjula Suri** Email: - mnjlsuri@gmail.com

behavior. However, the underlying mechanism in sucrose or saccharin induced feeding behavior is still not clear. Therefore, we studied different parameters of palatability, feeding behavior, and blood glucose level in the present study utilizing adult male rats fed with sucrose or saccharin.

MATERIALS AND METHODS

Animals

All the animal experiments were conducted on male albino Wistar rats (200-250 g) bred in the Central Experimental Animal Facility under the Institutional Ethical Committee (All India Institute of Medical Sciences) approval. Rats were marked and housed in separate polypropylene cages after/during experimental procedure under standard laboratory conditions (25±2°C, light/dark cycles of 10/14 h)and rats were provided with *ad libitum* food (Ashirwad Industries, India) and water. In the present study, rats were provided with water (Control group, n=12), 20% sucrose (Sucrose group, n=12) or 0.1% saccharin (Saccharin group, n=12) along with food pellets. Food intake, water intake, body weight, sucrose intake, saccharin intake, licking behavior, and blood glucose level were studied in sucrose and saccharin fed rats in each group.

Food Intake Estimation

Rats were provided with pre-weighed food in spill-proof containers at the beginning of the experiment [28]. Care was taken to provide surplus food and the leftover food was repeatedly weighed at 0, 0.25, 1, 3, and 5 h.

Water Intake Estimation

Similarly, rats were also provided with a premeasured amount of water in a spill-proof container [28]. Care was taken to provide surplus water and left-over water was measured at 0, 0.25, 1, 3, and 5 h.

Sucrose and Saccharin Intake Estimation

Furthermore, sucrose (20%) and saccharin (0.1%) were freshly prepared and provided as premeasured to rats at the beginning of each experiment in a spill-proof container along with food and water. Care was taken to provide surplus sucrose and saccharin and then left-over saccharin and sucrose was measured (0, 0.25, 1, 3, and 5 h).

Licking Behavior in Sucrose and Saccharin Fed Rats

Licking behavior was studied in Skinner box which is provided with food dispenser and optical lickometer (Coulbourn Instruments, USA), light emitting diode emitter and receiver of the photocell sensor. The light beam is passed through glass rods across a gap at the end of the tube. With each lick, animal's tongue breaks the light beam which is registered every time in personal computer. The rat was kept in the Skinner box and the bottle was filled with either sucrose or saccharin. The number of licking responses at the tip of the bottle was computed every 5 min for 5 h.

Blood Glucose Estimation

Glucose meter (Accu-check sensor, Roche Group, India) was used for blood glucose concentration (mg/dL) measurement. The test strip was inserted into the test strip slot with silver colored bars facing up and glucometer turns on automatically. Lancet was used to obtain a drop of blood from the hind paws of the rat. A drop of blood is touched to the edge of the strip within the curve. The blood drop gets drawn into the strip automatically. The digital monitor displayed the blood glucose concentration.

Body Weight Measurement

Rats body weight was recorded weekly using a single pan torsion balance [28]. The accuracy of this balance was ± 100 mg and weight up to 1 kg could be accurately recorded on this.

Study Design

Rats were divided into Control, Sucrose fed, and Saccharin fed groups. Different parameters such as body weight, food intake, water intake, sucrose intake, saccharin intake, licking behavior, and blood glucose level were studied from a session I through V at 0.25, 1.0, 3.0, and 5.0 h (**Fig. 1**).

Statistical Analysis

Data are presented as mean±standard deviation (SD). The values were compared between Control and Sucrose fed group utilizing paired student t-test. One-way ANOVA was utilized to compare Sucrose versus Saccharin group.

RESULTS

Effect of Sucrose Ingestion

During the period of observation, the Control rats ingested food during session IV only, whereas sucrose-fed rats ingested almost the same amount distributed over session IV and V (**Table 1**). Total food intake during 5 h was comparable in Sucrose fed and Control group of rats, although there was a significant difference during sessions IV and V as compared to the Control group (**Fig. 2A**).

Water intake significantly varied during sessions II, III, and IV in Control rats whereas, in Sucrose fed rats it varied during sessions III and IV (**Table1**). Total water intake was reduced ($p<0.02$) in Sucrose-fed as compared to Control group of rats (**Fig. 2B**). Rats started drinking sucrose immediately within 5 min after the presentation

of bottles which continued until session V. Total sucrose intake during 5 h was 12.18±5.22 mL (**Fig. 2C**).

Body weight was comparable between Control and Sucrose-fed rats during the study period of 2 weeks (**Fig. 2D**). Blood glucose concentration was determined in the rats ingesting food and/ or sucrose ad libitum at various study hours. Blood glucose concentration varied from 83.75±5.22 mg/dL to 94.58±6.35 mg/dL during the 5 h period of observation in Control rats, whereas it varied from 87.0±2.66 mg/dL to 103±2.33 mg/dL in Sucrose-fed rats. The blood glucose concentration of sucrose-fed rats was comparable to Control rats throughout the period of observation (**Fig. 2E**).

Effect of Sucrose Ingestion on Licking Behavior

The number of licking responses at the tip of the sucrose bottle was recorded every 5 min for 5 h. The rats ingested sucrose in six bouts during the experiment. The duration and frequency of licks varied although the peak responses were about 500. The peak numbers 1-2 corresponded to sessions II-III, peak 3-4 appeared between sessions III and IV, and peak 5-6 during session V (**Fig. 3A**).

Effect of Saccharin Ingestion on Licking Behavior

Total saccharin intake was 15.72±6.73 mL during 5 h and comparatively more than sucrose intake (**Fig. 2C**) which did not attain statistical significance. Rats received saccharin solution at the start of the experiment and the number of licking responses were computed every 5 min for 5 h. The licking behavior varied from that of sucrose. The number of bouts was only four. The number of licks peaked during sessions I-II and then reduced to peak again at 90 min to a lower value. It then reduced to near nadir corresponding to session IV (**Fig. 3B**). It peaked again to a lower value than the sucrose response during session V (**Fig. 3A-C**).

Fig 1. Schematic presentation of the whole study design. Bl-blood

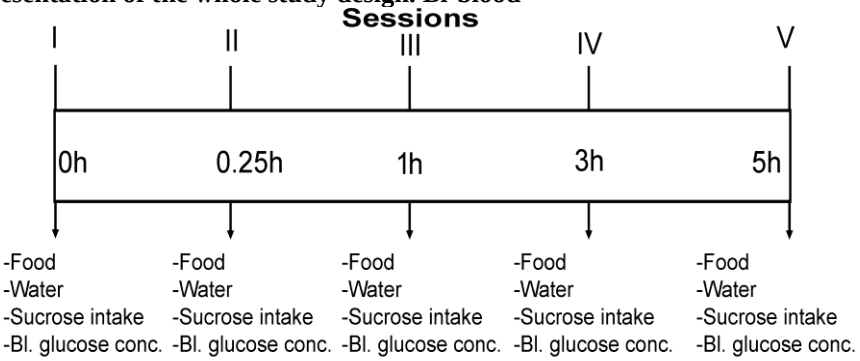
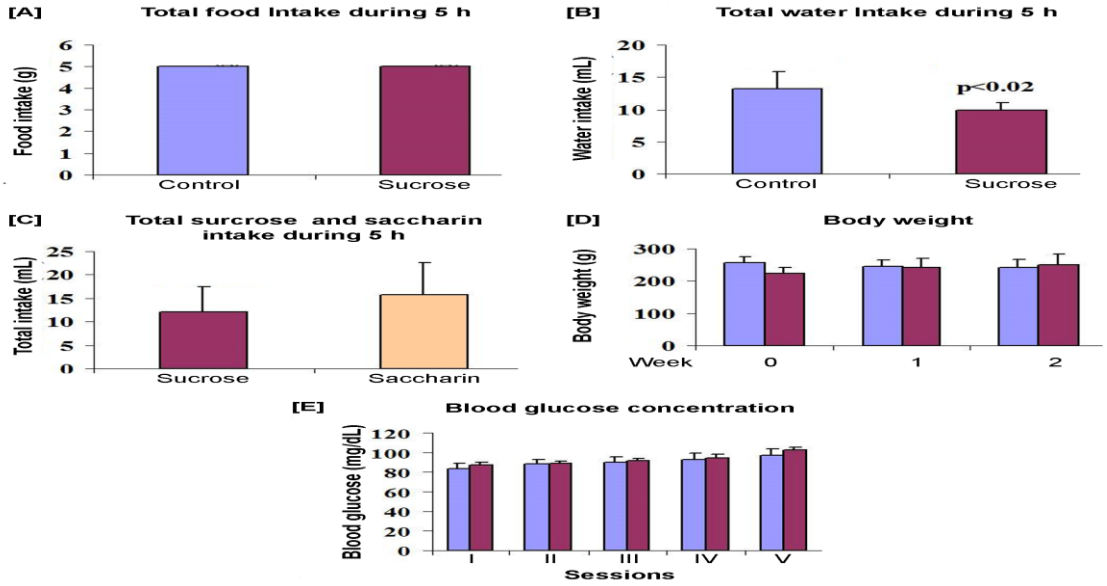
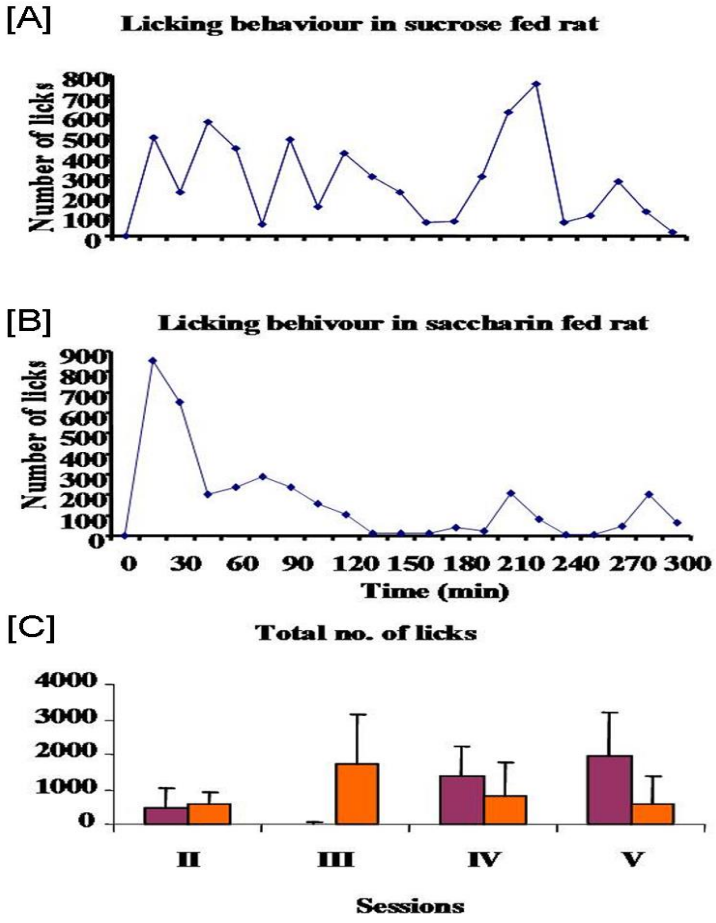


Fig 2. Physiological parameters



The figure depicts total food (A) and water intake (B) in Sucrose fed (Maroon) versus Control rats (Blue) during 5 h. Total sucrose and saccharin intake show no difference during 5 h (C). Body weight shows no significant variation in Sucrose fed versus Control rats over a period of observation (D). Blood glucose concentration varied insignificantly from a session I-V in both groups (E).

Fig 3. Effect of sucrose and saccharin ingestion on licking behavior of the rats.



The figure depicts the effect of sucrose and saccharin ingestion on licking behavior (A and B) responses (5 min for 5 h). However, the summated values of three blocks are being represented in the lower panel (C).

Table 1. Food, water, and sucrose intake in the Control and Sucrose fed rats

Session	Food intake (g)		Water intake (mL)		Sucrose intake (mL)
	Control	Sucrose fed	Control	Sucrose fed	
I	0.0±0.0	0.0±0.0	0.0±0.0	0.0±0.0	**0.0±0.0**
II	0.0±0.0	0.0±0.0	4.66±1.0^^	1.0±0.7**	**5.88±1.36**^^
III	0.0±0.0	0.0±0.0	3.33±1.3^^	2.77±0.6*^	**3.11±1.05**^
IV	5.0±0.0^^	2.26±2.6*^	3.66±2.1^^	4.88±1.1*^^	**3.77±0.87**^
V	0.0±0.0	2.26±2.6*^	1.77±1.7	1.11±0.3	**2.66±1.65**^
Total (5 h)	5.0	4.48	13.42	9.76**	**12.18±5.22**

The table depicts a significant variation (mean±SD) in food and water intake in Sucrose fed rats as compared to Control rats. * indicates a comparison between Control and Sucrose fed groups while ^ indicates comparison as compared to the session I within the group. */^P <0.05 and **/^^P <0.01.

DISCUSSION

Our study shows that *ad libitum*sucrose ingestion for 5 h did not make any significant difference in the blood glucose concentration and body weight in Sucrose fed as compared to Control rats. Whereas, in sucrose-fed rats, a significant variation in food intake was observed in session IV and V (3-5 h) and in water intake in sessions II and III as compared to the Control group. The total sucrose intake shows that the rats drank maximally immediately after sucrose presentation and continued to drink till 5 h although in lesser quantities. Licking behavior in sucrose-fed rats show 6-bouts of licking behavior spread over 5 h study period whereas in saccharin fed rat there were four bouts. The peak numbers 1-2 corresponded to sessions II-III while peak 3-4 appeared between sessions III and IV and peak 5-6

during session V. It is worth noting here that number of licks during a block corresponded to 500 licks except during the period 180-240 min, which roughly corresponded to 3 h and 45 min post-sucrose ingestion. Number of licks were approximately 750 (Session II) in saccharin fed rats. The number of licks gradually started increasing from 3 h onwards to 3.45 h and then suddenly decreased to <50. The number of licks suddenly increased to 200 but finally decreased to null at 5 h. It appears that the period from 150-195 min when number of licks was less than 50 corresponded to eualgesia recorded in Sucrose fed rats to most of the nociceptive responses [29] while the hyperalgesia recorded during session V [30] corresponded to extreme effort of 750 licks in 15 min followed by an exhaustive attempt around 285 min.

Bilateral electrolytic lesion of VMH has been reported to cause marked hyperphagia and obesity in rats, whereas bilateral lesion in adjacent LH resulted in aphagia [31-33]. Electrophysiological recordings from VMH have shown an increased activity following feeding or administering glucose whereas, LH activity tends to be lowered [11, 33-34]. These reactions are consistent with the role of VMH in limiting the intake of food and preference for palatable food [10]. Animals begin eating almost immediately after either procaine injections or electrolytic lesions into the VMH [35, 29]. Calorically rich sucrose sweet taste enhanced VMH activity and decreased LH activity. On the contrary, calorically inert saccharin sweet taste did not change the LH activity though the VMH activity was increased. It is correlated that VMH activity increased with sweet taste and it may be via VMH glucose receptors activation [11, 36] that dynamically respond to hypoglycemia [14, 37-40] to affect feeding behavior.

Food intake data of sucrose-fed rats is interesting because of the following observations, (1) The total amount of food ingested is equal in Control (5 g) and Sucrose (4.48 g) fed rat which has been ingested in session IV by control and session IV and V by sucrose fed rat. Duration of session III to V is four hours each session lasting for two hours. Therefore, Control rats ingested 5 g in 2 h while sucrose fed ingested 5 g in 4 h. It appears that sucrose-fed rats have become “nibblers” during this period. (2) Incidentally, first signs of hyperalgesia in some of the tests, in sucrose-fed rats have appeared in session IV. Strong hyperalgesia is seen in electrolytic VMH lesion [41]. (3) In chronic sucrose fed rat from weaning to adulthood pronounced hyperalgesia to noxious stimuli was noted [25]. Their food intake was higher which has been attributed to the functional physiological removal of VMH from the neural circuit. (4) Similarly, rats receiving continuous micro-infusion of 2DG in VMH [42] for 7 days show hyperalgesia and hyperphagia. (5) In light of these observations, VMH lesion produces hyperphagia and hyperalgesia. These hyperalgesic rats are nibblers and reportedly lose the diurnal rhythm to eat. Since nibbling and hyperalgesia are both characteristically seen in VMH lesion, therefore, it is tempting to hypothesize that in sucrose-fed rats VMH is having a “functional lesion” or it is out of the neural circuit. To summarize, it appears that sucrose-fed rats in the present study during session IV which extended from 1-3 h of sucrose ingestion have started showing the first sign of VMH being pushed out of circuit namely nibbling i.e. they ingested 5 g of food in 4 h compared to Control group who ingested the same amount in 2 h.

As compared to sucrose, saccharin has been reported to stimulate different hedonic properties and gustatory sweet receptors [43-45]. Bergmann et al. [42] also reported that saccharin consumption probably stimulates the release of endogenous opioid peptides via gustatory sweet receptors stimulation. Saccharin, a highly palatable sweetener that serves as an effective reinforcer of operant behavior and induces hyperphagia [4]. When electrical responses of VMH and LH to taste were recorded in a rat, ingestion of sucrose increases VMH activity and decreases LH activity while saccharin increases only VMH activity, though a reciprocal decrease in LH was not shown. Dynamics of LH/VMH neuronal activity is guided by the motivational state of the animal which in turn influences motivation to feed and associated behavior. Saccharin induced dietary hyperphagia is attributed to the interaction between innate and learned responses to the taste of foods. Rats innately respond to sweet tasting substances (saccharin) probably because they have some nutritive value [4].

In our study, blood-glucose concentration is increased little bit in Sucrose-fed rats compared to Control rats. As saccharin is non-calorigenic and unlikely to increase the blood glucose level, therefore, we did not measure the blood glucose level in the Saccharine fed rats. Anseloni et al. [46] reported that gustatory information from taste receptors is transmitted via facial, glossopharyngeal and vagal nerves to NTS, ascends to PBN and mid-and forebrain targets including taste thalamus, VMH, LHA, and insular cortex. Glucose-responsive cells in the VMH and LHA sense systemic and the ventricular chemical information. Further integration of information from other modalities, leads to feeding-related decision to control visceral organs and the motor system. The prefrontal cortex, amygdala, and reticular formation mediate signals from various sources and contribute, along with the hypothalamus, to the integration of hunger and satiation signals [47]. However, underlying mechanism of variation in palatable non-nutritive versus nutritive sweetener induced feeding behaviour is still not clear.

CONCLUSION

To summarize, there is temporal variation in sucrose versus saccharin induced ingestive behavior, but the underlying mechanism is still not clear. Further studies are needed to decipher the difference in the effect of palatable non-nutritive versus nutritive sweeteners on ingestive behavior.

ACKNOWLEDGMENTS

This project was funded by Institute Research Grant, All India Institute of Medical Sciences, New Delhi, India. We thank Mamta Sharma and Sanjeev Beniwal, Department of Physiology, for technical assistance and Department of Biostatistics for statistical analysis, All India Institute of Medical Sciences.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

REFERENCES

1. Johnson F, Wardle J. (2014). Variety, palatability, and obesity. *Adv Nutr*, 5, 851-59.
2. Zimmet PZ. (2017). Diabetes and its drivers, the largest epidemic in human history? *Clin Diabetes Endocrinol*, 3, 1-8.
3. Schroder H, Fito M, Covas MI. (2007). REGICOR investigators. Association of fast food consumption with energy intake, diet quality, body mass index, and the risk of obesity in a repetitive Mediterranean population. *Br J Nutr*, 98, 1274-80.
4. Ramirez I. (1990). What do we mean when we say "palatable food"? *Appetite*, 14, 159-61.
5. Yeomans MR, Wright P, Macleod HA, Critchley JA. (1990). Effects of nalmefene on feeding in humans, Dissociation of hunger and palatability. *Psychopharmacology*, 100, 426-32.
6. Yeomans MR, Wright P. (1991). Lower pleasantness of palatable foods in nalmefene treated human volunteers. *Appetite*, 16, 249-59.
7. Yeomans MR, Gray RW. (1996). Selective effects of naltrexone on food pleasantness and intake. *Physiol Behav*, 60, 439-46.
8. Ono T, Nishino H, Fukuda M, Sasaki K, Muramoto K, Oomura Y. (1982). Glucoresponsive neurons in rat ventromedial hypothalamic tissue slices *in vitro. Brain Res*, 232, 494-99.
9. Ono T, Oomura Y, Nishino H, Sakasi K, Muramoto K, Yano L. (1980). Morphine and enkephalin effects on hypothalamic glucoresponsive neurons. *Brain Res*, 185, 208-12.
10. Ono T, Sasaki K, Shibata R. (1987).Feeding and chemical related activity of ventromedial hypothalamic neurones in freely behaving rats. *J Physiol*, 394, 221-37.
11. Oomura Y, Oomura H, Yamamoto T, Naka F, Kobayashi N, Ono T. (1967). Neuronal mechanisms of feeding. *Prog Brain Res*, 27, 1-33.
12. Oomura Y, Ono T, Ooyama J, Wayner MJ. (1969). Glucose and osmosensitive neurons of the rat hypothalamus. *Nature*, 222, 282-84.
13. Oomura Y, Nishino H, Aou S, Lenard L. (1986). Opiate mechanism in reward related neuronal responses during operant feeding behaviour of the monkey. *Brain Res*, 365, 335-39.
14. Oomura Y. (1973). Central mechanism of feeding. *Adv Biophys*, 5, 65-142.
15. Borg MA, Sherwin RS, Borg WP, Tamborlane WV, Shulman GI. (1997). Local ventromedial hypothalamus glucose perfusion blocks counter regulation during systemic hypoglycemia in awake rats. *J Clin Invest*, 99, 361-65.
16. Challet EP, Pevet N, Lakhdar G, Malan A. (1997). Ventromedial nuclei of the hypothalamus are involved in the phase-advance of temperature and activity rhythms in food restricted rats fed during daytime. *Brain Res Bull*, 43, 209-18.
17. Moga MM, Moore RY. (1997).Organization of neural inputs to the suprachiasmatic nucleus in the rat. *J Comp Neurol*, 389, 508-34.
18. Levin BE. (2001). Glucosensing neurons do more than just sense glucose. *Int J Obes Relat Metab Disord*, 25(Suppl. 5), S68-S72.
19. Levin BE, Routh VH, Kang L, Sanders NM, Dunn-MeynellA. (2004). Neuronal glucosensing, what do we know after 50 years? *Diabetes*, 53, 2521-28.
20. Dum J, Gramsch C, Herz A. (1983). Activation of hypothalamic beta endorphin pools by reward induced by highly palatable food. *Pharmacol Biochem Behav*, 18, 443-47.
21. Bertiere MC, Mame Sy F, Baigts F, Mandenoff A, Apfelbaum M. (1984). Stress and sucrose hyperphagia, role of endogenous opiates. *Pharmacol Biochem Behav*, 20, 675-79.
22. Morley GE. (1980). The neuroendocrine control of appetite, the role of the endogenous opiates, cholecystokinin, TRH, gamma-amino-butyric-acid and the diazepam receptor. *Life Sci*, 27, 355-68.
23. Mukherjee K, Mathur R, Nayar U. (2001). Ventromedial hypothalamic mediation of sucrose feeding induced pain modulation. *Pharmacol Biochem Behav*, 6, 843-48.

24. Mukherjee K, Mathur R, Nayar U. (2001). Nociceptive response to chronic stress of restraint and noxious stimuli in sucrose fed rat. *Stress Health*, 17, 297-305.
25. Mukherjee K, Mathur R, Nayar U. (2002). Hyperalgesic response in rats fed sucrose from weaning to adulthood, role of VMH. *Pharmacol Biochem Behav*, 73, 3601-10.
26. Kanarek RB, Przypek J, D'Anci KE, Marks-Kaufman R. (1997). Dietary modulation of mu and kappa opioid receptor-mediated analgesia. *Pharmacol Biochem Behav*, 58, 43-9.
27. Segato FN, Castro-Souza C, Segato EN, Morato S, Coimbra NC. (1997). Sucrose ingestion causes opioid analgesia. Braz J Med Biol Res 30, 981-84.
28. Kumar S, Jain S, Behari J, Avelev VD, Mathur R. (2010). Effect of magnetic field on food and water intake and body weight of spinal cord injured rats. *Indian J Exp Biol*, 48, 982-86.
29. Suri M, Jain S, Mathur R. (2010). Pattern of biphasic response to various noxious stimuli in rats ingesting sucrose ad libitum. *Physiol Behav*, 101, 224-31.
30. Mercer E, Holder MD. (1997). Antinociceptive effects of palatable sweet ingesta on human responsivity to pressure pain. *Physiol Behavior*, 61, 311-18.
31. Brobeck JR, Tepperman J, Long CNH. (1943). Experimental hypothalamic hyperphagia in the albino rat. *Yale J Biol Med*, 15, 831-53.
32. Brobeck JR. (1946). Mechanism of the development of obesity in animals with hypothalamic lesions. *Physiol Rev*, 26, 541-559.
33. Anand BK. (1967). Handbook of Physiology: Alimentary Canal Washington DC, USA: American Physiological Society.
34. Anand BK. (1961).Nervous regulation of food intake. *Physiol Rev*, 41, 677-708.
35. BerthoudHR, Jeanrenaud B. (1979). Changes of insulinemia, glycemia and feeding behavior induced by VMH-procainization in the rat. *Brain Res*, 174, 184-87.
36. Rao BS, Prabhakara E. (1992). Effects of body weight loss and taste on VMH-LH electrical activity of rats. *Physiol Behav*, 52, 1187-92.
37. Anand BK, Chinna GS, Sharma KN, Dua S, Singh B. (1964). Activity of single neuron in the hypothalamic feeding centres, effect of glucose. *Am J Physiol*, 207, 1146-54.
38. Marrazzi MA. (1976). Hypothalamic Glucoreceptor Response: Biphasic Nature of Unit Potential. In: NovinD, Wywicka W, Bray GA, Ed. Hunger, Basic Mechanisms and Clinical Applications. New York: USA, Raven Press, 171-78.
39. Routh VH. (2003). Glucosensing neurons in ventromedial hypothalamic nucleus (VMN) and hypoglycemia-associated autonomic failure (HAAF). *Diabetes Metab Res Rev*, 19, 348-56.
40. Song Z, Levin BE, Mcardle JJ, Bakhos N, Routh VH. (2001). Convergence of pre- and postsynaptic influences on glucosensing neurons in the ventromedial hypothalamic nucleus. *Diabetes*, 50, 2673-81.
41. Mukherjee K, Mathur R, Nayar U. (2000). Effect of VMH lesion on sucrose-Fed nociceptive responses. *Jpn J Physiol*, 50, 395-404.
42. Bergmann E, Cohen I, Lieblich I. (1984).Biphasic effects of chronic saccharin intake on pain responses of healthy and diabetic rats of two genetically selected strains. *Psycho- pharmacology*, 82, 248-51.
43. Jakinovich W Jr. (1982). Stimulation of the gerbil's gustatory receptors by saccharin. *J Neurosci*, 2, 49-56.
44. Beczkowska IW, Koch JE, Bostock ME, Leibowitz SF, Bodnar RJ. (1993). Central opioid receptor subtype antagonists differentially reduce intake of saccharin and maltose dextrin solutions in rats. *Brain Res*, 618, 261-70.
45. Blass EM, Fitzgerald E, Kehoe P. (1987). Interactions between sucrose, pain and isolation distress. *Pharmacol Biochem Behav*, 26, 483-89.
46. Anseloni VC, Ren K, Dubner R, Ennis M. (2005). A brainstem substrate for analgesia elicited by intraoral sucrose. *Neurosciences*, 133, 231-43.
47. Sinha R, Nayar U, Mathur R. (2006). Pain modulation by VMH and LHA: A comparative study. In:Sharma KN, Bhattacharya N, Ed.Pain Updated: Mechanisms and Effects. New Delhi, India: Venus Printers and Publishers, 25-32.

Cite this article:
Manjula Suri, Suneel Kumar, Suman Jain, Rashmi Mathur. Effect Of Palatable Food Ingesta On Feeding Behavior And Blood Glucose Level In Rats. *Acta Biomedica Scientia.*, 2020; 7(1): 6-12. DOI: http://dx.doi.org/10.21276/abs.2020.7.1.2