

SOCIETY FOR THE STUDY

OF INGESTIVE BEHAVIOR

University of Pennsylvania
Philadelphia, Pennsylvania, USA
June 26-June 30, 2001

Program/Abstracts

2000-2001

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ACKNOWLEDGMENTS / SPONSORS

The Board of Directors of the Society for the Study of Ingestive Behavior (SSIB) and the Organizing Committee of the 2001 Annual Meeting thank, on behalf of the entire SSIB membership, the following corporations and groups for their generous support of the Society and its annual meeting.

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Distinguished Career Award:

Johnson and Johnson Career Award for Research in Ingestive Behavior Lecture:

A.J. STUNKARD: Eating disorders and the brain.

Early Career Award:

Early Career Award Lecture:

R. SEELEY: When worlds collide: GLP-1 and the response to visceral illness.

New Investigator Awards:

Listed alphabetically.

Dr. Deborah Clegg, University of Cincinnati, USA

Dr. Shachar Eylam, University of Florida, USA

Dr. Marci Gluck, Columbia University, USA

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Mr. Sean Stocker, University of Pittsburgh, USA

Mr. Robert Twining, Pennsylvania State University, USA

Dr. ChuanFeng Wang, University of Minnesota, VA Medical Center, USA

Mr. Robert Wheeler, Pennsylvania State University, USA

Tuesday, June 26

- 13:00-17:00 **Registration** – Sansom P West
- 13:00-17:30 **Penn Symposium**
Central Issues in the Foreground of Current Research on Caloric and
Hydromineral Homeostasis - Meyerson B1 (Organizers: S.J. Fluharty, H.J. Grill,
J.M. Kaplan, and G.P Smith)
Mickey Stunkard: Historical introduction.
Steve Fluharty and Randall Sakai: Fluid balance.
Mark Friedman and Randy Seeley: Central and peripheral control mechanism.
Harvey Grill and Rich Miselis: Central localization of function.
Gerry Smith and Joel Kaplan: Satiating and meal patterning.
Barbara Rolls and Harry Kissileff: Human intake control.
- 15:00 **Beverage Break** – Meyerson Lobby
- 17:30 **Open Bar** (provided by the University of Pennsylvania) - Bodek Lounge,
Houston Hall
- 19:00-21:00 **Welcome Reception** (Beer, Wine and Snacks) – Bodek Lounge, Houston Hall

Wednesday, June 27

- 07:30-08:30 **Breakfast** – Campus Dining
- 08:20-08:30 **Opening:** T.R. Scott – Logan 17
- 08:30-10:00 Symposium 1: Biological Mechanisms of Clinical Cachexias (Chair: G.J.
Schwartz) – Logan 17
- 08:30-08:32 **G.J. Schwartz:** Introduction
- 08:32-08:49 **R. Landmann,** K. Frei and M. Letiembre: Pattern recognition receptors in the
brain.
- 08:49-09:06 **W. Langhans:** Biological mechanisms of the anorexia during infection.
- 09:06-09:23 **M.J. Tisdale:** Mechanism of protein degradation in cancer cachexia.
- 09:23-09:40 **J.E. Morley:** Anorexia of aging.
- 09:40-10:00 General Discussion

- 08:45-10:00 **Oral Session 1: Mineral and Fluid Balance** – (Chair: M.G. Tordoff) – Terrace Room
- 08:45-09:00 **R.B. Puchalski**, A.A. Bachmanov, J. Barhanin and M.G. Tordoff: Regulation of NaCl intake is disrupted by null mutation of the gene encoding KCNE1, a potassium channel regulator.
- 09:00-09:15 **L.R. Lucas**, C.A. Grillo and B.S. McEwen: Interaction of brain dopaminergic and peptidergic systems in salt seeking behavior.
- 09:15-09:30 **S.N. Thornton**, C. Falconetti, M. Chapleur and B. Fernette. Actions of angiotensin and type-1 receptor specific non-peptide antagonists in different strains of rat, implications in sodium appetite.
- 09:30-09:45 **T.R. Houpt**: Hyperosmotic thirst in young pigs: the rapid drop in osmolality while drinking.
- 09:45-10:00 **S.A. Mc Caughey** and M.G. Tordoff: Magnesium appetite in the rat.
- 10:00-10:30 **Coffee Break**– Logan Foyer
- 10:30-12:00 **Oral Session 2: Metabolic Controls** (Chair: M.I. Friedman) – Terrace Room
- 10:30-10:45 **A. Doerflinger** and S.E. Swithers: Diet manipulation has persistent effects in the weaning rat.
- 10:45-11:00 **D.A. Levitsky**, E. Obarzanek and G. Mrdjenovic: Weight loss without hypophagia following overfeeding: Luxusconsumption lives.
- 11:00-11:15 **H. Ji** and M.I. Friedman: Fasting plasma triglyceride levels predict weight gain in rats fed a high-fat diet.
- 11:15-11:30 **L. Van Hanswijck De Jonge**, B.S. Birketvedt, N. Stettler and G. Waller: Environmental temperature during pregnancy and body mass index in late adolescence.
- 11:30-11:45 **M. Leonhardt** and W. Langhans: Effect of hydroxycitrate (HCA) on food intake and body weight regain in rats after hepatic branch vagotomy or sham vagotomy.
- 11:45-12:00 **C.C. Horn**, H. Ji and M.I. Friedman: Etomoxir, an inhibitor of fatty acid oxidation, affects hepatic energy status but does not increase feeding or reduce liver ATP.
- 10:30-12:00 **Oral Session 3: The Parabrachial Nucleus – Role in Ingestion and Beyond** (Chair: K.J. Simansky) – Logan 17
- 10:30-10:45 **J.P. Baird**, J.B. Travers and S.P. Travers: Taste and visceral representation in the PBN.
- 10:45-11:00 **T.A. Houpt**: c-Fos induction in the rat parabrachial nucleus after visceral, gustatory, and magnetic stimulation.

- 11:00-11:15 **R. Norgren**, B. Li and D. Wheeler: Medial and lateral parabrachial nucleus lesions affect learned aversions and sodium appetite differentially.
- 11:15-11:30 J.I.F. De Gobbi, L.A. De Luca Jr., A.K. Johnson and **J.V. Menani**: Serotonin and cholecystokinin in the lateral parabrachial nucleus in the control of sodium and water intake.
- 11:30-11:45 **K.J. Simansky** and D.M. Nicklous: Infusion of Neuropeptide FF (NPFF) into the parabrachial nucleus (PBN) inhibits feeding stimulated by the mu-opioid agonist DAMGO.
- 11:45-12:00 **H.J. Grill** and J.M. Kaplan: Effects of urocortin, leptin and melanocortin receptor ligands delivered to the parabrachial nucleus.
- 10:30-12:00 **Oral Session 4: Social and Physiological Aspects of Food Intake in Females** (Chair: T.A. Spiegel) – Meyerson B1
- 10:30-10:45 **J.O. Fisher**, D.C. Mitchell, H. Smiciklas-Wright, L.L. Birch: Maternal modeling and availability influences on young girls' beverage intakes.
- 10:45-11:00 **S.L. Johnson**: Cultural factors related to mothers' child feeding practices.
- 11:00-11:15 **I.R. Contento**, C.E. Basch and P. Zybert: Relationship of dietary restraint and dietary disinhibition to the quality of food choices of women and their young children.
- 11:15-11:30 **A. Drewnowski**: Dieting in adolescence: Biology, family, or the environment?
- 11:30-11:45 **B.J. Tepper**: Influence of mild gestational diabetes on appetite for sweet foods in pregnancy.
- 11:45-12:00 **A.R. Collins** and T. Spiegel: Psychosocial and life style determinants of weight gain and abdominal adiposity in women during transition to menopause.
- 12:00-13:30 **Free Time for Lunch** (lunch not provided)
- 13:30-15:30 **New Investigator Presidential Symposium** (Chair: T.R. Scott) – Meyerson B1
- 13:30-13:45 **D.J. Clegg**, E.L. Air, S.C. Woods and R.J. Seeley: Melanin-concentrating hormone (MCH)-induced hyperphagia is not blocked by naloxone, a non-specific opioid antagonist.
- 13:45-14:00 **M. Gluck**, A. Geliebter, A. Galvan, E. Yahav, D. Hui, J. Hung and S. Hashim: Obese binge eaters have more global stress and report more pain tolerance and hunger following a cold pressor test (CPT).
- 14:00-14:15 **C.J. Hukshorn**, M.S. Westerterp-Plantenga, L.A. Campfield and W.H.M. Saris: Pegylated human recombinant leptin causes additional weight loss and affects LH levels in energy-restricted overweight and mildly obese men.

- 14:15-14:30 **M.M.J.W. Kamphuis** and M.S. Westerterp-Plantenga: Taste perception of free fatty acids, i.e. CLA in humans.
- 14:30-14:45 **E.M.R. Kovacs** and M.S. Westerterp-Plantenga: Does (-)-hydroxycitrate reduce de novo lipogenesis?
- 14:45-15:00 **S.D. Stocker**, C. Kimbrough, E.M. Stricker and A.F. Sved: Elevated dietary salt suppresses renin release but not thirst evoked by hypotension.
- 15:00-15:15 **C. Wang** and C. Kotz: Effect of urocortin in the lateral septal area on food intake.
- 15:15-15:30 **R.A. Wheeler**, D.S. Wheeler and P.S. Grigson: Lewis and Fischer 344 rats exhibit different responses to natural rewards and drugs of abuse.
- 15:30-16:00 **Coffee Break**– Meyerson B1 and Logan Foyer
- 16:00-17:30 **NIDA-sponsored Symposium 2: Like Drugs for Chocolate: Separate Rewards Modulated by Common Mechanisms?** (Chair: P.S. Grigson, Co-Chairs: M. Lynch, S. Volman) – Logan 17
- 16:00-16:02 **P.S. Grigson**: Introduction
- 16:02-16:19 **M.L. Pelchat**: Of human bondage: Craving, obsession, compulsion, and addiction.
- 16:19-16:36 **A.E. Kelley**, M. Zhang, M. Will and T. Steininger: Opioid modulation of taste hedonics within the ventral striatum.
- 16:36-16:53 **K.D. Carr**: Augmentation of drug reward by chronic food restriction: Behavioral evidence and underlying mechanisms.
- 16:53-17:10 **R.M. Carelli**: Neurophysiological Analysis of Cocaine Self-Administration vs. "Natural" Reinforcement.
- 17:10-17:30 General Discussion
- 16:00-17:30 **Oral Session 5: Central Peptides** (Chair: T.H. Moran) – Terrace Room
- 16:00-16:15 **R.J. Bodnar**, R.M. Silva, M.M. Hadjimarkou, H.C. Grossman, P. Liu, G.C. Rossi and G.W. Pasternak. Beta-endorphin-induced feeding: Modulation by opioid receptor and G-protein antisense oligodeoxynucleotides.
- 16:15-16:30 **E. Air**, K. Blake and S. Woods: Acute reduction of food intake by intracerebroventricular insulin.
- 16:30-16:45 **S.F. Leibowitz**, P. Pamy, N. Levenkova and J. Dourmashkin: Hyperphagia on a high-fat diet is attributed, in part, to a rise in circulating triglycerides and hypothalamic galanin and a reduction in insulin and leptin.
- 16:45-17:00 **S. Aja**, K.J. Mills and T.H. Moran: Central neuropeptide Y (NPY) and peripheral cholecystokinin (CCK) have opposing, independent, and summative effects on food intake in rats.

- 17:00-17:15 **U. Smedh** and T.H. Moran: Suppression of gastric emptying, but not of sucrose ingestion, by hindbrain cocaine- and amphetamine regulated transcript (CART) peptide 55-102 is dependent on corticotropin-releasing factor receptors.
- 17:15-17:30 R. Ciccocioppo, M. Biondini, L. Antonelli, J. Wichmann, F. Jenck and **M. Massi**: The nonpeptidic ORL1 receptor agonist, Ro 64-6198, reverts restraint stress- and CRF-induced anorexia in rats.

19:00-22:00 **Poster Session I** – Bodek Lounge, Houston Hall

- 1 **S. Choi**, E. Jonak, L. Simpson, V. Patil and J.D. Fernstrom: Intermittent fenfluramine administration to rats suppresses food intake despite chronic brain serotonin suppression.
- 2 **I.G. Makarenko**, M.M. Meguid and M.V. Ugrumov: Distribution of Serotonin (5-HT) 1B Receptors in the rat diencephalon.
- 3 **J.-P. Voigt**, H. Hörtnagl, B. Bert and H. Fink: Effects of the 5-HT1A receptor agonist 8-OH-DPAT on food intake in obese Zucker rats of different ages.
- 4 **A. Hajnal**, G.P. Smith and R. Norgren: Sham-fed sucrose increases accumbens dopamine in a concentration-dependent manner: A microdialysis study in behaving rats.
- 5 **J.M. Barranco**, J. Lorch and T.A. Houpt: The effect of centrally administered lithium chloride on conditioned taste aversion and c-fos in rats.
- 6 M.H. Leszczuk, T. Allen, S. Judge and **C.F. Flaherty**: Effect of deprivation condition and inter-solution interval on anticipatory contrast obtained with sucrose-sucrose and saccharin-sucrose pairings.
- 7 **C.F. Flaherty**, M. McCool and M.H. Leszczuk: Sucrose intake in Sprague-Dawley and Lewis rats in two consummatory contrast procedures.
- 8 **N. Sakai** and S. Imada: The insular cortex and the prefrontal cortex are involved in the association between taste and odor in the rat.
- 9 **P.S. Grigson**, V. Sanchez and S.M. Ballard: Anticipatory contrast effects are disrupted by a local context change before and after the CS-US pairing.
- 10 **R.C. Twining** and P.S. Grigson: The suppressive effects of cocaine self-administration on saccharin vs. malic acid intake: Acquisition and reinstatement (New Investigator Awardee).
- 11 **P.L. Smith**, R. Norgren, G. Scalera, J.C. Smith and P.S. Grigson: Acquisition of conditioned trigeminal aversions in rats with parabrachial nucleus lesions.
- 12 **M. Mangiaracina** and N. Geary: Estradiol does not affect sham feeding of sucrose in ovariectomized rats.
- 13 **K.P. Myers** and A. Sclafani: Flavor preferences reinforced by the postingestive effects of glucose but not galactose in preweanling rats.
- 14 **K.P. Myers** and A. Sclafani: Conditioned changes in evaluation of a flavor paired with the postingestive effects of glucose: A taste reactivity analysis.
- 15 **A. Sclafani**: Conditioned flavor preferences as a function of sugar concentration.

- 16 **K. Ackroff** and A. Sclafani: Postingestive nutrient conditioning of flavor preferences does not require food deprivation.
- 17 **K. Ackroff** and A. Sclafani: Carbohydrate-conditioned preference for the flavor of ethanol.
- 18 **E. Na** and D.A. Fitts: Conditioned taste aversion and c-Fos expression in cholestatic rats.
- 19 **N.L. Turner**, J. Frieman, R. Mehiel, E.M. Douglass: Evidence for extinction of a conditioned flavor preference.
- 20 **L.J. Nolan**, B.A. Sousa and A.M. Siegel: Conditioned food aversion in the terrestrial hermit crab (*coenobita clypeatus*).
- 21 W. D. T. Kent, S. K. Cross-Mellor, M. Kavaliers and **K.-P. Ossenkopp**: Acute effects of corticosterone on LiCl-induced rapid gustatory conditioning in rats: Examining patterns of licking behavior.
- 22 **B. Raudenbush**, N. Corley, N. R. Flower, A. Kozlowski and B. Meyer: Nutritional intake differences in food neophobics and neophilics.
- 23 **B. Raudenbush**, N. Corley, N. R. Flower, A. Kozlowski and B. Meyer: Cephalic phase salivary response differences characterize level of food neophobia.
- 24 **C.H. Vaughan** and N.E. Rowland: Operant conditioning utilizing mice in a foraging paradigm.
- 25 **Y.E. Yiin** and L. Thibault: Training in acquisition of odor-cued fasting-anticipatory satiety in rats.
- 26 **A.W. Hartfield**, N.A. Moore and P.G. Clifton: The effects of clozapine, olanzapine and haloperidol on the microstructure of ingestive behaviour in the rat.
- 27 **C. Larue-Achagiotis**, M.L. Navis, A. Bensaid, M. Lacroix, G. Fromentin, N. Gausseres and D. Tome: Influence of nibblings and strains (Wistar vs Dark Agouti) on body weight gain and food intake of rats.
- 28 **Z. Chen**, S.P. Travers and J.B. Travers: Microinfusion of D-CPP into the brainstem reticular formation suppresses ingestion and rejection in the awake rat.
- 29 **M. Ciampolini**, M. Van Weeren, B. De Pont, W. De Haan and L. Borselli: Subjective recognition of a narrow glycemia range after training.
- 30 **M. Ciampolini**, V. Giannellini and N. Butte: Recognition of depletion manifestations (bearable hunger) in infants by trained caregivers and lower fecal energy loss.
- 31 **J.C. Gustavson**, J. Asarian-Anderson, N. Tayag, C. Young and A.R. Gustavson: Employing an evaluation framework to increase the effectiveness of a nutrition and physical activity intervention in a Latino community in Los Angeles.
- 32 **J.D. Latner** and A.J. Stunkard: The stigmatization of obese peers among 5th- and 6th- grade children: Comparison with a 1961 investigation (New Investigator Awardee).
- 33 **Y. Martins**, P. Pliner and J. Mills: Exploring the changeroom effect: Females' perceptions of bodily size and attractiveness.
- 34 **G. Mrdjenovic** and D.A. Levitsky: Do children regulate energy intake?

- 35 **J. Wilson**, H. Gwinn, S. Fernando and S. James: Caloric compensation in college students during lunchtime meals.
- 36 **C.L. Colbert** and N. E. Rowland: Hydrochlorothiazide induced sodium bicarbonate intake by rats in a simulated foraging paradigm.
- 37 **S. Eylam**, M. Garcea and A.C. Spector: Sodium appetite in DBA/2J mice (New Investigator Awardee).
- 38 **C.V. Pastuskovas**, A.K. Johnson and R.L. Thunhorst: Neuronal activity in rat insular cortex after sodium depletion: Manner of sodium delivery.
- 39 **M.F. Roitman**, G. Anderson, M.T. Koh, T.A. Jones and I.L. Bernstein: Behavioral sensitization to sodium (Na) depletion is correlated with increased dendritic length and spines in nucleus accumbens neurons.
- 40 **K.S. Curtis**, E.G. Krause and R.J. Contreras: Long-term food restriction and area postrema lesions: Effect on heart rate in rats.
- 41 **G.S. Fraley**, T.T. Dinh and S. Ritter: Immunotoxin lesion of norepinephrine (NE) and epinephrine (E) neurons innervating the medial hypothalamus elevates basal expression of and attenuates glucoprivation induced increases in agouti gene related-protein (AGRP) mRNA.
- 42 **M.G. De Vries**, L.M. Arseneau, B.L.M. Frenkel and J.L. Beverly: Hypoglycemia-associated autonomic failure does not involve diminishing of hypothalamic norepinephrine release.
- 43 **J.R. Komorowski**, J. De La Harpe, W.T. Cefalu, X.H. Zhang, Z.Q. Wang and D. Greenberg: JCR:LA-cp rats show improved lipid profiles in response to diets containing chromium picolinate and biotin.
- 44 **W. Langhans**, V.B. Hinderling, P. Schrauwen and M.S. Westerterp-Plantenga: Effect of repetitive etomoxir administration on 24h substrate oxidation and satiety in humans.
- 45 **K.J. Melanson**, M.R. Carpenter, S.J. Knipe, J. Dell'olio, D.P. Berlin and J.M. Rippe: Glycemic, insulinemic and appetitive responses to meals differing in protein content and glycemic index: a pilot study in obese adults.
- 46 **D. Salter** and A.G. Watts: Glucoprivic feeding, but not other glucoregulatory responses, is attenuated in dehydration anorexia (New Investigator Awardee).
- 47 **K. Torii**: Brain mechanism of the recognition for glucose utilization in rats.
- 48 **N.M. Sanders**, T.T. Dinh and S. Ritter: Selective immunotoxin lesions of hindbrain norepinephrine/epinephrine (NE/E) neurons impair feeding and glucocorticoid responses to insulin-induced hypoglycemia.
- 49 **Z.S. Warwick**: Diet-induced hyperphagia: role of fat content and caloric density.
- 50 **J.W. Jahng**, T.A. Houpt, G.T. Kim and D.G. Kim: Refeeding induced expression of neuronal nitric oxide synthase in the rat hypothalamic paraventricular nucleus.
- 51 **T.A. Lennie**: Hypothalamic c-fos expression in normal and weight-reduced rats during acute inflammation.
- 52 **E.P. Merrill**, F.M. Kramer, A. Cardello and H. Schutz: A comparison of satiety measures.

Thursday, June 28

- 07:30-08:30 **Breakfast** – Campus Dining
- 08:30-10:00 **Symposium 3:** Transgenic Models and Genetic Approaches to an Understanding of Food Intake Control and Obesity (Chair: S.P. Kalra) – Logan 17
- 08:30-08:32 **S.P. Kalra:** Introduction
- 08:32-08:49 **G. Barsh,** C. Kaelin, T. Gunn, L. He and K. Clement: Using forward and reverse genetics to study hypothalamic regulation of body weight.
- 08:49-09:06 **L.H. Tecott,** K. Nonogaki, E. Goulding, L. Abdallah and J. Wade: Chronic hyperphagia, hyperactivity and late-onset obesity in serotonin 5-HT_{2C} receptor mutant mice.
- 09:06-09:23 **E. Marathos-Flier:** Melanin concentrating hormone and energy homeostasis in transgenic and knockout mice.
- 09:23-09:40 **S.P. Kalra** and P.S. Kalra: Leptin and neurocytokine gene transfer to probe the etiology of hypothalamus based obesity and energy expenditure.
- 09:40-10:00 General Discussion
- 08:30-10:00 **Oral Session 6:** Human Ingestion (Chair: B.J. Rolls) – Terrace Room
- 08:30-08:45 **G.S. Birketvedt,** J.Sundsford and J. Florholmen: Hypothalamic-pituitary-adrenal axis in the night eating syndrome.
- 08:45-09:00 **C. Haynes,** M. Lee and M.R. Yeomans: The interactive effects of stress, dietary restraint, and disinhibition, on eating in a multi-item meal. (New Investigator Awardee)
- 09:00-09:15 **T.V.E. Kral,** L.S. Roe and B.J. Rolls: Effects of energy density with and without nutrition information on food intake in lean women.
- 09:15-09:30 **T.M. Robinson,** S.J. French, M.D. Lee, R.W. Gray and M.R. Yeomans: Effects of test meal palatability on responses to intragastric nutrient preloads. (New Investigator Awardee)
- 09:30-09:45 **A. Geliebter,** E. Yahav, D. Hui, M. Gluck, S. Haq and S.A. Hashim: Gastric capacity and test meal intake in obese binge and non-binge eaters.
- 09:45-10:00 **M.S. Westerterp-Plantenga,** E.M.R. Kovacs and K.J. Melanson: Energy intake regulation and habitual meal frequency in time-blinded men.
- 10:00-10:30 **Coffee Break** – Logan Foyer

- 10:30-12:00 **Symposium 4:** The Developing Story of Glucoreceptor Neurons (Chair: B.E. Levin, Co-Chair: H.J. Grill) – Logan 17
- 10:30-10:32 **B.E. Levin:** Introduction
- 10:32-10:49 **B.E. Levin:** Metabolic sensors: Viewing glucosensing neurons from a broader perspective.
- 10:49-11:06 **V.H. Routh:** Brain glucosensing: It's all in how you slice it.
- 11:06-11:23 **M.A. Cowley, J.L. Smart, S. Diano, M. Rubinstein, M.J. Low, T.L. Horvath and R.D. Cone:** POMC neurons integrate diverse metabolic signals including leptin, glucose and ghrelin.
- 11:23-11:40 **R.N. Bergman, A. Hevener and C. Donovan:** Relative roles of central and peripheral glucose sensors in the response to hypoglycemia.
- 11:40-12:00 General Discussion
-
- 10:30-12:00 **Oral Session 7:** Physiologic Mechanisms of Disordered Eating (Chair: B.T. Walsh) – Terrace Room
- 10:30-10:45 **N. Geary, L. Asarian, K. Korach, D. Pfaff and S. Ogawa:** Decreased influence of estradiol on CCK-induced satiation and brainstem c-Fos expression in estradiol receptor- α null mice.
- 10:45-11:00 **B. Gosnell:** Relationships between drug self-administration and sucrose intake.
- 11:00-11:15 **B.G. Hoebel, D. Chau, R.A. Kosloff, J.L. Taylor and P.Rada:** Possible role of accumbens dopamine and acetylcholine in sugar withdrawal and behavioral depression.
- 11:15-11:30 **J.E. Mitchell:** Substance abuse among patients with eating disorders.
- 11:30-11:45 **B.T. Walsh, E. Zimmerli, H.R. Kissileff, J.L. Guss and M.J. Devlin:** Disturbances of gastric function in bulimia nervosa.
- 11:45-12:00 **W. Kaye, G. Frank, C. Meltzer, J. Price, C. Mathis, W. Drevets, C. McConaha and K. Skovira:** Trait-related serotonin disturbances in anorexia and bulimia nervosa: PET imaging studies with radioligands.
-
- 12:00-13:30 **Free Time for Lunch** (lunch not provided)
-
- 13:30-15:00 **Keynote Lecture** (Chair: H.J. Grill) – Meyerson B1
J.S. Flier: Leptin and the regulation of body weight: Recent insights and unanswered questions
-
- 15:00-16:00 **Women's Forum** – Terrace Room
- 16:00-17:00 **New Investigators' Forum** – Terrace Room

- 15:30-18:00 **Long Range Planning Committee Meeting** - Bishop White Room, Houston Hall
- 18:00-20:00 **Reception at the Monell Chemical Senses Center**
- 20:00-22:00 **Board Meeting** – Bishop White Room, Houston Hall

Friday, June 29

- 07:30-08:30 **Breakfast** – Campus Dining
- 08:30-10:00 **Oral Session 8: Melanocortins** (Chair: J.M. Kaplan) – Logan 17
- 08:30-08:45 **L.M. Brown**, C.T. Hansen, R.L. Eskay and T.W. Castonguay: Chemiluminescent *in situ* hybridization of the melanocortin-4 receptor in adrenalectomized lean and obese Balb/c-Avy mice.
- 08:45-09:00 **J.D. Roth**, D.K. Yee, J. Hines and S.J. Fluharty: Melanocortin Regulation of Feeding: A cellular model.
- 09:00-09:15 **P.K. Olszewski**, M.M. Wirth, T.J. Shaw, M.K. Grace, C.J. Billington, S.Q. Giraud and A.S. Levine: Immunohistochemical evidence of the physiologic role for alpha-melanocyte-stimulating hormone (alpha-MSH) in the regulation of ingestive behavior.
- 09:15-09:30 C. Polidori and **N. Geary**: Failure of melanocortin-3/4 receptor agonism or antagonism to affect estradiol-induced inhibition of feeding in ovariectomized rats.
- 09:30-09:45 **T.H. Moran**, S. Knipp, S. Aja and E.E. Ladenheim: Meal pattern effects of central melanocortin receptor activation and blockade.
- 09:45-10:00 **D.L. Williams**, H.J. Grill and J.M. Kaplan: Food restriction in the hours after injection reduces long-term hyperphagic effects of SHU9119.
- 08:30-10:00 **Oral Session 9: Influences on Selections among Foods** (Chair: D.A. Booth) – Meyerson B1
- 08:30-08:45 **J. Sobal**, M.M. Connors, C.M. Devine and C.A. Bisognij: Marital careers and food choices.
- 08:45-09:00 **J. Pollard**, D. Greenwood and J. Cade: Motivations for fruit and vegetable consumption in the UK Women's Cohort Study.
- 09:00-09:15 **K. Brunsø** and J. Scholderer: Consumer health consciousness and the organic foods boom: Fact or fiction?

- 09:15-09:30 **M.K. Magnusson**, A. Arvola, U.-K. Koivisto Hursti, L. Åberg and P.-O. Sjödén: Perceived health and environmental consequences as predictors of consumer attitudes and behaviour towards organic foods.
- 09:30-09:45 **J. Scholderer**, T. Bech-Larsen and K.G. Grunert: Changing public perceptions of genetically modified foods: Effects of consumer information and direct product experience.
- 09:45-10:00 **K.G. Grunert** and J. Scholderer: More apples for the young: Can a means-end based framework explain intentions to increase apple consumption?
- 08:30-10:00 **Oral Session 10: Genetic and Molecular Approaches** (Chair: S.J. Fluharty) – Terrace Room
- 08:30-08:45 **D.K. Yee**, J. Hines and S.J. Fluharty: Use of a chimeric receptor strategy reveals commonalities and dissimilarities in the molecular mechanisms within the angiotensin II receptor family.
- 08:45-09:00 **J. Hines**, S.J. Fluharty and D.K. Yee: Activation of the different signal transduction pathways of the AT-1 receptor requires different structural determinants.
- 09:00-09:15 **J-A.M. McQuade**, S.C. Benoit, M. Xu and R.J. Seeley: Lack of a 2-deoxy glucose-induced feeding response in mice lacking the dopamine 3 receptor.
- 09:15-09:30 **M. Bagnasco**, M.G. Dube, H. Dhillon, P.S. Kalra, and S.P. Kalra: Microinjection of recombinant adeno-associated virus vector encoding rat leptin gene (rAAV-leptin) in discrete hypothalamic sites suppressed weight, adiposity and plasma insulin for extended period in adult rats.
- 09:30-09:45 **M.G. Dube**, E. Beretta, H. Dhillon, S.P. Kalra and P.S. Kalra: Central leptin gene therapy blocks high fat diet-induced weight gain, hyperinsulinemia, hyperleptinemia and leptin resistance in rats.
- 09:45-10:00 **M.G. Tordoff**, A.A. Bachmanov, X. Li, D.R. Reed, S. Li, Z. Chen, P.J. De Jong, C. Wu, D.R. West, A. Chatterjee, D.A. Ross, J.D. Ohmen and G.K. Beauchamp: Positional cloning of *Sac (Gpr98)*, a gene controlling sweetener ingestion.
- 10:00-10:30 **Coffee Break** – Logan Foyer
- 10:30-12:00 **Symposium 5: The Dorsomedial Hypothalamus Revisited** (Chair: M. Tang-Christensen) – Logan 17
- 10:30-10:32 **M. Tang-Christensen**: Introduction
- 10:32-10:49 **N. Vrang**, P.J. Larsen and M. Tang-Christensen: Neurocircuitry and neurochemistry of the dorsomedial hypothalamic nucleus.
- 10:49-11:06 **L.L. Bellinger** and L.L. Bernardis: The Dorsomedial Hypothalamic Nucleus (DMN), and its role in feeding behavior; lessons learned from lesioning studies.

- 11:06-11:23 **S. Bi**, E.E. Ladenheim and T.H. Moran: The DMH NPY system: lessons learned from the OLETF rats.
- 11:23-11:40 **K.L. Grove**, P. Chen and M.S. Smith: Neuropeptide Y (NPY) neurons in the dorsomedial hypothalamic nucleus (DMH): Implications in the development and regulation of hypothalamic feeding circuits.
- 11:40-12:00 General Discussion
- 10:30-12:00 **Oral Session 11: Scaling of Sensations and Intake Measurements** (Chair: H.R. Kissileff, Co-Chair: L. Bartoshuk) – Terrace Room
- 10:30-10:45 **L.M. Bartoshuk**, V.B. Duffy, K. Fast, B.G. Green and D.J. Snyder: The General Labeled Magnitude Scale provides valid measures of genetic variation in taste and may be a universal psychophysical ruler.
- 10:45-11:00 A. Oenema and **J. Brug**: Testing the effects of personalised feedback to influence dietary intake awareness.
- 11:00-11:15 **C.L. Pelkman**, L.S. Roe and B.J. Rolls: Strategies for increasing statistical power in studies on human energy intake.
- 11:15-11:30 **J.L. Guss**, H.R. Kissileff, B.T. Walsh and D.A. Booth: Words used by patients with bulimia nervosa and healthy controls to express sensations associated with different types of meals.
- 11:30-11:45 **H.R. Kissileff**, J.L. Guss, L. Bartoshuk and B.T. Walsh: Effect of rating scales for feelings in imagined eating situations by patients with Bulimia Nervosa and normal controls.
- 11:45-12:00 **D.M. Press**, J.L. Guss, C. Haynes, X. Pi-Sunyer and H.R. Kissileff: Appetitive ratings during a test meal measure effects of CCK on sickness and satiety.
- 12:00-13:30 **Free Time for Lunch** (lunch not provided)
- 13:30-14:30 **Award Lectures** (Chair: R. Norgren) – Meyerson B1
- 13:30-14:00 **Early Career Award Lecture, R. Seeley**: When worlds collide: GLP-1 and the response to visceral illness.
- 14:00-14:30 **Johnson and Johnson Career Award for Research in Ingestive Behavior Lecture, A.J. Stunkard**: Eating disorders and the brain.

14:30-17:30 **Poster Session II** – Bodek Lounge, Houston Hall

- 1 **J. Bogue:** The integration of market and sensory information to explore consumers' preferences for full-fat and reduced-fat dairy products.
- 2 **J. Bogue** and D. Sorenson: An exploratory study of consumer attitudes towards health-enhancing foods.
- 3 **M. Brennan,** C. Ritson, M. Ness, S. Kuznesoff, L. Frewer and S. Miles: Communicating food risk uncertainty with the public.
- 4 **C. Golding,** J. Cade, C. Lawton and D. Greenwood: Comparison of low and high fat consumers in the UK women's cohort study.
- 5 **N.S. Coulson:** Adolescents' beliefs about the costs and benefits of additives and their presence in different foods.
- 6 A. Oenema and **J. Brug:** Social comparison and assumptions about personal dietary intake.
- 7 **V.B. Duffy,** M.N. Phillips, J.M. Peterson and L.M. Bartoshuk: Bitterness of 6-n-propylthiouracil (PROP) associates with bitter sensations and intake of vegetables.
- 8 **D.J. Snyder,** V.B. Duffy, K. Fast, J.M. Weiffenbach and L.M. Bartoshuk: PROP genetics interact with age and sex to influence food preferences.
- 9 **M.C. Goldsmith** and R.B. Kanarek: Genetically determined bitter perception influences human food preferences.
- 10 **N. Ullrich** and B.J. Tepper: Food adventurousness clarifies the influence of PROP taster status on food preferences.
- 11 **M.G. Tordoff:** The contribution of the number of choices to intake and preference.
- 12 **J.B. Elia,** K.A. Houpt and T.R. Houpt: Motivation of horses for fiber.
- 13 **E.L. Air,** D.J. Clegg, R.J. Seeley and S.C. Woods: Melanin concentrating hormone stimulates water intake independent of food intake.
- 14 **A.V. Azzara,** J. Sokolnicki and G.J. Schwartz: Melanocortin (MC) 3/4 receptor agonist MTII reduces meal size in rats.
- 15 **K. Bugarith,** S. Ritter, T.T. Dinh and D.A. Lappi: Injection of the peptide-toxin-complex neuropeptide Y (NPY)-saporin (NPY-sap) into the paraventricular nucleus of the hypothalamus (PVH) causes hyperphagia and obesity in rats.
- 16 **A.J. Jackman,** D.J. Clegg, J.A. Reed, R.J. Sheldon, S.C. Woods and R.J. Seeley: Interactions between melanin-concentrating hormone (MCH) and c-fos after agouti-related protein (AgRP) administration.
- 17 **J.A. Reed,** D.J. Clegg, A.J. Jackman, S.C. Woods and R.J. Seeley: Agouti-related protein (AgRP) increases melanocortin-concentrating hormone (MCH) expression in the lateral hypothalamus.
- 18 S.H. Choi, B.S. Kwon, H.T. Lee, D.G. Kim and **J.W. Jahng:** Chronic pCPA down-regulated gene expressions of CART in the rat hypothalamus.

- 19 **R.J. Bodnar**, J.A. Echo, N. Lamonte, T.F. Ackerman, C.K. Singh, V. Znamensky. Modulation of mu opioid agonist-induced feeding in the nucleus accumbens shell by excitatory and inhibitory amino acids.
- 20 A. Ster, **T. Kowalski**, M. Dube, S. Kalra and G. Smith: Evidence of increased hypothalamic NPY release in preweaning fa/fa rats.
21. **E. Rothe** and L. Rinaman: Central oxytocin (OT) activates hindbrain glucagon-like peptide-1 (GLP-1) neurons in rats.
- 22 **S. Markison**, H.J. Grill and J.M. Kaplan: Synergistic interactions with meal-taking underlie longer-term effects of urocortin..
- 23 **D.C. Sweet**, A.S. Levine and C.M. Kotz: Hypocretin-1 (orexin-A)-induced feeding is dependent on central opioidergic pathways.
- 24 **M. Tang-Christensen**, M. Hansen, A.W. Holst, L.K. Larsen, P.J. Larsen and N. Vrang: The distribution of GLP-1/GLP-2 neurones in the NTS projecting to the PVN and/or DMH.
- 25 **M.M. Wirth** and S.Q. Giraudo: Agouti-related protein and leptin in regulation of body weight.
- 26 **M.M. Vazquez** and A. Jones: The blocking effect of leptin activity on melanin concentrating hormone in regard to food consumption in rats.
- 27 **M.D. Wortman**, P. Rushing, S.C. Woods and R.J. Seeley: Neuroanatomical distribution of receptor activity modifying proteins 1-3 (RAMPs) in rat brain by in situ hybridization.
- 28 **S.F. Akana** and M.F. Dallman: Hyperphagia develops before shifts in corticosterone, insulin or leptin in mice with disruption of the serotonin 2C (5-HT_{2C}) receptor.
- 29 **S.F. Akana** and M. F. Dallman: Evaluation of endocrine function in mice with measurement of urinary hormones.
- 30 **M.E. Bell**, K.D. Laugero, S. Bhatnagar and M.F. Dallman: Spelling ‘stressed’ backwards: Plasma corticosterone (B) and sucrose drinking interact in a negative feedback loop during chronic stress.
- 31 **K.L.K. Tamashiro**, C. Markham, D.C. Blanchard, R.J. Blanchard, L.Y. Ma and R.R. Sakai: Chronic social stress effects on body weight.
- 32 M.C. Murphy, M. Bilodeau, J.M. Barranco and **L.A. Eckel**: Time course of estradiol’s inhibitory effect on meal size in female rats.
- 33 **M. Varma**, L. Zhang and M.M. Meguid: Effect of estradiol on hypothalamic dopamine and serotonin in Fischer –344 rats.
- 34 **E.G. Krause**, K.S. Curtis, L.M. Davis and R.J. Contreras: Estrogen influence on stimulated water intake by ovariectomized female rats.
- 35 **J-A.M. McQuade**, S.C. Benoit, M. Xu and R.J. Seeley: Alcohol consumption in the dopamine 3 receptor knockout mouse.
- 36 **S.C. Benoit**, J.A. McQuade, M. Xu and R.J. Seeley: Increased food intake in mice with targeted deletion of the dopamine 3 receptor.
- 37 **J. Overduin**, N. Sanchez, L. Hampton, J. Battey and J. Gibbs: Food intake in mice lacking

the bombesin receptor subtype-3.

- 38 **R.J. Davis** and S.E. Swithers: Regulation of gastric emptying of glucose in the preweanling rat.
- 39 **J.E. Cecil**, M.I. Friedman, H.J. Grill and J.M. Kaplan: Factors influencing the oral stimulation-induced inhibition of fat emptying from the stomach.
- 40 **M. Covasa** and R.C. Ritter: Inhibition of food intake by intestinal maltotriose requires oligosaccharide digestion but not glucose absorption.
- 41 **M. Covasa**, T. Bui, R.C. Ritter and G.A. Burns: NMDA participation in cholecystokinin-induced inhibition of food intake.
- 42 Withdrawn.
- 43 **J.E. Cox**, A. Randich, G.R. Kelm and S.T. Meller: Role of the vagus in feeding suppression produced by jejunal lipid infusions.
- 44 A. Randich, **J.E. Cox**, D.S. Spraggins, G.R. Kelm and S.T. Meller: Jejunal and portal vein infusions of lipids affect hepatic vagal afferent activity.
- 45 **P. Sanchez** and G.P. Smith: Common hepatic branch fibers to the liver are sufficient to mediate the satiating effect of endogenous CCK released by duodenal infusions of maltose.
- 46 **A-M. Torregrossa** and G.P. Smith: Effects of high-fat diets on the satiating potency of CCK-8.
- 47 **J.M. Brunstrom** and S. Higgs: Restrained eaters are insensitive to the flavor-flavor learning paradigm.
- 48 **B. Raudenbush** and B. Meyer: Disordered eating among college athletes: Influence of muscularity dissatisfaction and supplement use.
- 49 **R.L. Corwin** and J.J. Carman: Binge-type eating in rats is due to limited access, not food deprivation.
- 50 **M.L.S. Santos** and P.P.P. Machado: Food avoidance behaviour among female university students: association with Eating Disorder Inventory and its subscales scores. (New Investigator Awardee).
- 51 C.A. Schulz and **P. Wright**: Early eating experiences of women with eating problems.
- 52 E.J. Davidson and **P. Wright**: Selective processing of weight and shape related words in bulimia nervosa: use of a computerised stroop test.

17:30-18:30 **Business Meeting** – Meyerson B1

19:15- **Banquet** (included with registration) – Hall of Flags, Houston Hall

19:15-20:15 **Open Bar**

20:00-21:30 **Dinner and Presentations**

21:30- **Music and Dancing**

Saturday, June 30

- 07:30-14:00 **Check out** – Sansom West
- 07:30-08:30 **Breakfast** – Campus Dining
- 08:30-10:00 **Symposium 6: Satisfaction of Thirst** (Chair: M. Pelchat, Co-Chair: D. Booth) – Terrace Room
- 08:30-08:32 **M. Pelchat:** Introduction
- 08:32-08:49 **E.M. Stricker:** Multiple signals inhibit thirst.
- 08:49-09:06 **J.M. Brunstrom:** Effects of mouth wetting on the satiation of thirst.
- 09:06-09:23 **D.H. Passe, J. Stofan, M. Horn and R. Murray:** Impact of electrolytes, flavor and palatability on voluntary fluid intake during exercise.
- 09:23-09:40 **D.A. Booth:** Associative conditioning of the image of a can of cola by anticipatory thirst reduction and other refreshment.
- 09:40-10:00 General Discussion
- 08:30-10:00 **Oral Session 12: Small Molecule Neurotransmitters** (Chair: D.W. Gietzen) – Logan 17
- 08:30-08:45 **M.D. Lee, G.A. Kennett, C.T. Dourish and P.G. Clifton:** The 5-HT-1B/1D antagonist, GR 127,935, does not attenuate the hypophagic action of d-fenfluramine in mice: discrepancies between antagonist studies and transgenic models of 5-HT function.
- 08:45-09:00 **D.V. Coscina and G.C. Parker:** Antagonism of different serotonin (5HT) receptors in the basolateral amygdala (BLA): Effects on feeding.
- 09:00-09:15 **M.M. Meguid, Y. Qi and T. Tada:** Relationship of aminergic to peptidergic neuromodulators of paraventricular nucleus (PVN) in cancer anorexia.
- 09:15-09:30 **L. Zhang, M.M. Meguid and S.O. Fetissov:** Intra-VMN infusion of nicotine suppresses food intake and enhances dopamine (DA) and serotonin (5HT) release in both VMN and LHA.
- 09:30-09:45 **L. Thibault:** Dietary influence on plasma amino acids and central neurotransmitters in stress-susceptible pigs.
- 09:45-10:00 **D.W. Gietzen, B.G. Truong, J.E. Blevins and P.S. Teh:** Is glutamate in the Anterior Piriform Cortex-Lateral Hypothalamus pathway (APC-LH) involved in the anorectic responses to indispensable amino acid deficiency (IAAD)?
- 10:00-10:30 **Coffee Break** – Logan Foyer

- 10:30-12:00 **Oral Session 13: Hormones** (Chair: W. Langhans) – Logan 17
- 10:30-10:45 **R.D. Reidelberger**, L. Kelsey and D. Heimann: Dose-response effects of amylin-related peptides on food intake and gastric emptying in rats.
- 10:45-11:00 **P.A. Rushing**, R.J. Seeley, E.L. Air, T.A. Lutz and S.C. Woods: Acute 3rd-ventricular amylin infusion potently reduces food intake but does not produce aversive consequences.
- 11:00-11:15 **K.L. Teff**, S. Elliot, R. Townsend and P.J. Havel: High fructose meals reduce 24-hour circulating insulin and leptin concentrations and increase subsequent energy and fat intake in women.
- 11:15-11:30 **E. Beretta**, M.G. Dube, H. Dhillon, P.S. Kalra and S.P. Kalra: Central leptin gene therapy in prepubertal rats reduces weight gain, metabolic hormones, blood leptin and insulin levels but augments energy expenditure postpubertally for extended period.
- 11:30-11:45 **B.J. Hrupka** and W. Langhans: Is leptin involved in day-to-day regulation of energy intake?
- 11:45-12:00 **B. Selmaoui**, A. Oguine and L. Thibault: Effect of food access schedule and dietary composition on nocturnal levels of serum melatonin and pineal N-acetyltransferase activity.
- 10:30-12:00 **Oral Session 14: Learning and Reward** (Chair: T.A. Houpt) – Terrace Room
- 10:30-10:45 **Y. Oomura**, N. Hori, T. Shiraishi, S. Aou, X. Li, K. Fukunaga and K. Sasaki: Central mechanism of the effect of leptin on learning and memory.
- 10:45-11:00 **P.S. Schroy**, D.S. Wheeler and P.S. Grigson: Cocaine-induced avoidance of saccharin intake is associated with elevated circulating corticosterone levels at test when using a 5 min or a 30 min interstimulus interval (New Investigator Awardee).
- 11:00-11:15 **A.L. Tracy** and T.L. Davidson: Responding based on interoceptive signals produced by 2-DG depends on sucrose anticipation.
- 11:15-11:30 N. Wald and **M. Leshem**: Conditioning of flavor preference by NaCl after exercise is dependent on perspiration.
- 11:30-11:45 **T.A. Houpt**, D.W. Pittman, J.M. Barranco, J.W. Jahng and J.C. Smith: Circling behavior and conditioned taste aversion induced by high strength static magnetic fields.
- 12:00 **Adjourn**

Pattern recognition receptors in the brain. R. LANDMANN, K. FREI*, M. LETIEMBRE. Div. Infectious Diseases, University Hospital, Basel and *Dept. Neurosurgery, University Hospital Zürich, Switzerland Lipopolysaccharide (LPS), a membrane component of Gram-negative bacteria, induces anorexia via central nervous mechanisms. While LPS-induced TNF α and IL-1 have a defined role in anorexia, receptors for LPS in the brain have only recently been uncovered; the impact of their localization and function on anorexia has not been studied. LPS binds with LPS binding protein to the glyco-protein CD14 which exists either as a glycosylphosphatidylinositol (GPI)-anchored membrane molecule in myeloid cells or as a soluble protein. CD14 induces cell activation after interaction with the transmembrane heterodimer toll-like receptor 4 (TLR4)/MD-2. The intra-cellular tail of TLR4 is homologous to the IL-1 receptor. TLR4 signalling uses adaptor molecules and kinases homologous to those in IL-1 signalling and leads to NF κ B and cytokine gene expression. TLR2, although coexpressed with CD14 in some cells, does not recognize LPS, but is induced by LPS. CD14 mRNA is low in untreated rat and mice brain. In mice, CD14 mRNA and protein are induced in microglia by LPS, TNF α and bacterial infection; the choroid plexus and infiltrating leukocytes express it strongly. TLR4 mRNA is expressed in untreated rat and mice brain, in the former it colocalizes with CD14, but in contrast to CD14, it is downregulated by LPS. Weak constitutive brain TLR2mRNA is upregulated by LPS and bacterial infection. Protein expression of TLR2 and 4 in the brain is not known. The existence of LPS receptors in the brain indicates that they may participate in anorectic effect of LPS.

Biological mechanisms of the anorexia during infection. W. LANGHANS. Institute of Animal Sciences, Swiss Federal Institute of Technology (ETHZ), 8092 Zurich, Switzerland
The anorexia during infection is part of the host's defense mechanisms, and is initially beneficial. During chronic infections, however, anorexia is a major contributor to malnutrition and cachexia and can ultimately be deleterious. Microbial products presumably trigger the anorexia during infections by stimulating the production of pro-inflammatory cytokines, which serve as endogenous mediators. Several cytokines reduce food intake after parenteral administration, suggesting that they play a role in the anorexia during infection. As most microbes infect peripheral organs rather than the brain, cytokines presumably inhibit eating through activating peripheral to central neural and humoral pathways. Whereas those neural pathways mediate some of the defense reactions, they do not seem to be necessary for the anorexia during infection. Circulating cytokines can reach their central nervous system binding sites through circumventricular organs and through active or passive transport mechanisms, or they can act through receptors on endothelial cells of the brain vasculature and stimulate the release of subsequent mediators such as eicosanoids. Recent data in fact strongly suggest a role of the cyclooxygenase pathway in the anorectic effects of bacterial lipopolysaccharide (LPS). One important central mediator of LPS and IL-1 induced anorexia during infection appears to be serotonin, but increases in the production or turnover of other neurochemicals (e.g. dopamine, histamine, corticotropin releasing hormone, glucagon-like peptide-1) or a decrease in neuropeptide Y expression have been implicated as well. In sum, multiple interactions between various pleiotropic cytokines, and between cytokines and neurochemicals mediate the anorexia during infection. Understanding these biological mechanisms should help to design effective therapeutic strategies.

Mechanism of protein degradation in cancer cachexia. M.J. TISDALE. Pharmaceutical Sciences Research Institute, Aston University, Birmingham B4 7ET, United Kingdom. Loss of skeletal muscle mass in cachectic cancer patients results in weakness, immobility and eventually in death. The major factor contributing to protein loss is an increase in protein degradation initiated through an upregulation in the expression of the ubiquitin-proteasome pathway. We have isolated a sulfated glycoprotein of Mr 24kDa, produced by cachexia-inducing human and murine tumors that initiates direct protein catabolism in skeletal muscle. This material has been named proteolysis-inducing factor (PIF). In vivo studies show that PIF induces specific loss of skeletal muscle in mice, while visceral protein reserves are preserved. There is no effect on food or water intake despite massive weight loss (8% in 24h). PIF has been shown to induce protein degradation by increasing expression of both mRNA and protein of the key components of the ubiquitin-proteasome proteolytic pathway, in particular proteasome subunits and the ubiquitin-conjugating enzyme. The action of PIF can be completely attenuated by eicosapentaenoic acid (EPA). This effect is thought to arise from inhibition of intracellular signaling pathways induced by PIF leading to activation of proteolysis. EPA has been shown to downregulate the increased expression of the ubiquitin-proteasome pathway, and, combined with a nutritional supplement, to increase lean body mass in cachectic patients with pancreatic carcinoma.

Anorexia of Aging. J.E. MORLEY. GRECC, St. Louis VAMC, and Division of Geriatrics, Saint Louis University, St. Louis, MO. Numerous studies have demonstrated that food intake declines with aging in healthy persons. The reasons for this decline are multifactorial. It includes changes in taste and olfaction. However, the major reason is a decrease in compliance of the fundus of the stomach leading to reduced adaptive relaxation. This appears to be secondary to reduced nitric oxide release. Decreased adaptive relaxation results in more rapid antral filling and early satiation. Glucose in the duodenum enhances appetite in older persons. Lipid in the duodenum results in satiation. This is secondary to increased CCK release in older persons. Aging is also associated with an increased satiating effect of CCK. Males develop anorexia to a greater degree than females. This is due to the decrease in testosterone which leads to an increase in leptin and a reduction in CNS neuropeptide Y and nitric oxide. There is also evidence for a decline in opioid feeding drive with aging. Pathologically depression represents the major cause of anorexia in older persons. Some older persons develop anorexia tardive. While anorexia plays a role in the development of sarcopenia in older persons, other factors such as cytokines and hormone decline also play a key role. Cachexia is due to catabolism of muscle associated with disease in contrast to decreased muscle anabolism which results in sarcopenia.

Regulation of NaCl intake is disrupted by null mutation of the gene encoding KCNE1, a potassium channel regulator. R.B. PUCHALSKI, A.A. BACHMANOV, J. BARHANIN, M.G. TORDOFF. Monell Chemical Senses Center, 3500 Market St., Philadelphia, PA 19104, USA. The role of potassium channels in the regulation of NaCl intake has not been investigated previously. One channel, KCNQ1, and its regulator KCNE1, are expressed in salivary glands and kidneys, and KCNE1 null mutant mice are deficient in KCNQ1 potassium currents. To understand the role of the KCNQ1/KCNE1 channel in NaCl intake and taste, we compared the NaCl consumption of KCNE1 $+/+$, $+/-$, and $-/-$ mice, using two-bottle preference and lick tests. When fed a sodium-replete diet, KCNE1 $+/+$ and $+/-$ mice preferred 75-150 mM NaCl solutions but KCNE1 $-/-$ mice rejected them. This effect was observed in females only and required prior exposure to NaCl. There were no differences in initial rates of NaCl licking by naive mice, suggesting the KCNE1 $-/-$ mice learned to avoid NaCl. In response to dietary NaCl deprivation, 150 mM NaCl intakes and aldosterone concentrations were 3-fold higher and sodium excretion was 2-fold higher in KCNE1 $-/-$ relative to KCNE1 $+/-$ mice. In response to a NaCl load (i.p.), sodium excretion was 20% less in KCNE1 $-/-$ than KCNE1 $+/-$ mice on the first day but two times greater two days later. Thus, null mutation of the KCNE1 gene causes salt wasting. We conclude that the KCNQ1/KCNE1 potassium channel regulates NaCl intake by modulating sodium reabsorption.

Interaction of brain dopaminergic and peptidergic systems in salt seeking behavior. L.R. LUCAS, C.A. GRILLO, B.S. MCEWEN. Lab of Neuroendocrinology, The Rockefeller University, New York NY 10021 (USA) Acute sodium depletion by the combination of pharmacological natriuresis via furosemide (FURO) administration and a sodium deficient diet results in a strong induction of salt appetite in rats. We have previously shown that tachykinins (TKs) inhibit salt appetite since FURO treatment yields decreased TK-mRNA levels in many brain regions (i.e., limbic and striatal areas); and TKs reduce salt appetite if intracerebrally injected. Since acute FURO decreases dopamine (DA) uptake and TKs are known to be regulated by DA tone, we hypothesized that the DAergic motivational/attentional circuit in the brain is activated in this behavior. We found that FURO treatment (2 s.c. injections of 5 mg/rat, plus Teklad sodium deficient chow) induced decreased striatal DA transporter (DAT) levels and increased enkephalin (ENK) mRNA levels in the nucleus accumbens. In a separate set of experiments we pharmacologically blocked ENK or DA receptors 15 min. before FURO treated adult male Sprague-Dawley rats were allowed 2h access to 2% salt solution (2 bottle choice). We found that intracerebral infusion into the nucleus accumbens of naltrindole (*2 opioid antagonist, 1 μ g) or raclopride (D2R antagonist, 1 μ g) had no effect on salt intake whereas infusion of naltrindole into the ventral tegmental area induced a robust attenuation of salt intake. These data provide evidence that accumbal DA tone as mediated through ENK is increased in salt appetitive rats and that DA plays a permissive role in salt appetite behavior. Supported by MH43787

Actions of angiotensin and type-1 receptor specific non-peptide antagonists in different strains of rat, implications in sodium appetite. S.N. THORNTON*, C. FALCONETTI, M. CHAPLEUR, B. FERNETTE. EA 3114, Université Henri Poincaré, 38 rue Lionnois, Nancy, France. The AT-1 receptor mediates the effects of central administration of angiotensin II (AngII) on thirst and sodium appetite. Electrophysiological experiments using iontophoretic application of the AngII type-1 (AT-1) receptor specific non-peptide antagonists losartan and irbesartan do not give the same results in the forebrain of urethane anaesthetised male Wistar rats. DOCA pre-treatment changed the neuronal sensitivity to AngII and to the specific antagonists but not in the same direction. Here we have compared neuronal responses to AngII and the type-1 receptor specific antagonists in the preoptic/medial septum region using a 7 barrelled micro-iontophoretic electrode sealed to a recording electrode in urethane anaesthetised, male rats. Two groups were used, one that responds well to central AngII for sodium appetite, the Wistar, and the other that does not, the Fischer 344. As previously reported both losartan and irbesartan did not produce the same effects in both groups of rats. Neurons in the forebrain of the Fischer rats appeared less sensitive to the local application of AngII than those of the Wistar rats. Furthermore, the response of the neurons in both groups to local application of the two antagonists appeared to depend on the order in which they were applied. These results could be interpreted as evidence for a complex of AngII type-1 receptors with variable properties and it is the interaction of AngII on different (sub)types of these receptors that could be responsible for the induced sodium appetite in conscious behaving rats. (Support: MH 43787)

Hyperosmotic thirst in young pigs: the rapid drop in osmolality while drinking. T.R. HOUP. College of Veterinary Medicine, Cornell University, Ithaca, NY 14853, U.S.A. Plasma osmolality rises in young pigs deprived of drinking water with food available, and this hyperosmolality is then the primary stimulus to drinking. Previously it had been found that while the pig drank osmolality fell precipitously, by the end of the bout, to levels that would not stimulate further drinking. This decrease in osmolality could be due either to dilution of extracellular fluid (ECF) or removal of osmotically active particles. Movements of water or osmolytes into or out of the intestine seemed most likely as causes. Six pigs were fitted with intravenous and intraduodenal (ID) catheters. The pigs were deprived of drinking water for 6 hours, then given an intraduodenal infusion (20 ml/kg) of water or isotonic or hypertonic solutions of NaCl and sorbitol. Blood samples were taken and analyses were done for plasma osmolality, packed cell volume and plasma protein. Blood volume changes were estimated by calculation. The results suggest that water absorption from the gut after drinking was rapid and could account for much of the rapid plasma dilution. When water absorption was inhibited, plasma osmolality failed to fall as rapidly. If hypertonic NaCl solutions were infused, plasma osmolality tended to rise slightly, while isotonic NaCl infusion caused little or no change in osmolality. Isotonic sorbitol solution also failed to cause a fall in osmolality, indicating that movement of osmotically active particles into the gut was unlikely to be the cause of the fall of plasma osmolality. Changes in blood volume will also be considered.

Magnesium appetite in the rat. S.A. MCCAUGHEY, M.G. TORDOFF. Monell Chemical Senses Center, Philadelphia, PA 19104.

Rats modify their ingestive behavior to correct deficiencies of minerals such as sodium and calcium. The behavioral effects of magnesium deficiency have not been studied extensively, although in one prior experiment magnesium deprivation appeared to cause an aversion to magnesium. In the present work, we compared the drinking behavior of magnesium-deprived and replete male Sprague-Dawley rats. Subjects were maintained on a magnesium-free or nutritionally complete diet and were given either 3.2, 10, 32, or 100 mM MgCl₂; 32 mM CaCl₂; 32 mM NaCl; 10 mM HCl; or 2.5 mM NaSaccharin, and their intake was measured for 24 h in a two-bottle test with water. Within 5 min, magnesium-deprived subjects given 32 mM CaCl₂ or 3.2, 32, or 100 mM MgCl₂ drank significantly more of these solutions than did replete rats. Intake of NaSaccharin was reduced in the deprived subjects at 2 h. In a separate study, rats fed replete, magnesium-deficient, or calcium-deficient diets were given a choice between water, 32 mM MgCl₂, and 32 mM CaCl₂. The deficient rats preferred the solution that ameliorated their deficiency; for example, during the first 1 h, the magnesium-deficient rats drank 3.1 ± 0.5 ml MgCl₂ and 1.1 ± 0.4 ml CaCl₂. The calcium-deficient rats drank 1.8 ± 0.5 ml MgCl₂ and 3.9 ± 0.4 ml CaCl₂. Thus, magnesium deficiency leads to a compensatory appetite for magnesium, and the appetites for magnesium and calcium are distinct and specific. The rapid expression of magnesium appetite suggests that it may depend on innate, gustatory factors.

Diet manipulation has persistent effects in the weaning rat. A. DOERFLINGER, S.E. SWITHERS. Dept. of Psychological Sciences, Purdue University, West Lafayette, IN 47905. Observations from our laboratory have demonstrated differences in the developmental trajectories of pups reared by dams maintained on a high fat diet (HF pups) versus pups raised by a dam fed a "standard" chow diet (SC pups). On day 1, HF pups weigh less than SC pups, but rapidly surpass the body weight of SC animals. Data also suggests that HF pups show delayed responding to metabolic manipulations; HF pups do not respond to changes in fatty acid oxidation until a later age compared to SC pups (Swithers, Melendez, Watkins and Davis, 2001). To examine whether these differences in body weight and metabolic responding have persistent effects, we monitored body weight and behavioral changes through weaning in HF and SC pups. Body weights of pups were collected daily until day 12, then litters and dams were transferred to cages that allowed only pups to enter a pup-designated food and water compartment. Likewise, only dams could enter the dam-designated compartment. Pup and dam body weights, as well as food and water intake data, were collected every 12 hours (at lights on and lights off). Videotapes were made for 6 hr of the light and 6 hr of the dark cycle and behaviors of two focal pups were analyzed to determine suckling, ingestive, and general exploratory behavior. The results suggest that HF pups continue to show different developmental trajectories (e.g. body weight differences) through weaning and may have delayed initiation of independent ingestion compared to SC pups. Supported by NIDDK R01 55531.

Weight loss without hypophagia following overfeeding: Luxusconsumption lives. D.A. LEVITSKY, E. OBARZANEK, G. MRDJENOVIC. Division of Nutritional Sciences and Department of Psychology, Cornell University, Ithaca, NY. Twelve healthy young adults consumed all their food ad libitum from the Cornell Eating Laboratory where their intake was measured at each meal. Following a two week period (baseline), during which time the subjects maintained their weight constant, they were fed daily 133% of their baseline intake for 13 days. All subjects gained weight of which about 2/3 was fat and 1/3 lean body mass. The amount of weight gained and the change in body composition during overfeeding was consistent with literature values and showed little evidence of energetic inefficiency. However, on the first day following overfeeding, food intake of the subjects returned precisely to their baseline values. They maintained daily intake that was no different from baseline during the entire three weeks of recovery. Despite the lack of hypophagia, subjects lost most of the weight they gained during the overfeeding. To accomplish this weight loss, subjects must have expended 438 Kcal per day more than they ingested. The increase in energy expenditure relative to energy intake following the period of overfeeding and not detectable during overfeeding is consistent with animal studies (Almeida, Levitsky, and Strupp, 1996) suggesting that the ingestion of excess calories may suppress energy expenditure. Besides resurrecting the classic argument that the body can increase energy metabolism to defend weight gain (Luxusconsumption), these data raise seriously questions concerning the putative link between the regulation of body weight and the control of food intake.

Fasting plasma triglyceride levels predict weight gain in rats fed a high-fat diet. H. JI, M.I. FRIEDMAN. Monell Chemical Senses Center, Philadelphia, PA 19104, USA. Some rats gain more weight when eating a high-fat diet than do others. Such different sensitivities are thought to result from an interaction between genetic predisposition and environmental factors (i.e., dietary fat). We investigated whether there are any differences in fuel metabolism that could serve as a marker for the predisposition. Chow-fed male Sprague-Dawley rats (240-275 g) were deprived of food at the onset of dark period; tail blood was collected at 12, 18 and 24 hrs. A high-fat diet (40% fat, 40% carbohydrate and 20% protein by calorie) was then given ad libitum to the rats. Plasma prepared from the blood samples was analyzed for glucose, free fatty acids, triglycerides, ketones, glycerol, insulin and leptin. Only triglyceride levels at 18 hr of fasting consistently had a positive correlation ($r > 0.5$, $P < 0.05$) with weight gain during the subsequent 3-4 wk high-fat feeding period. Fast weight gainers (upper 50%) had higher triglyceride levels than slow gainers (bottom 50%; $P < 0.05$) at 18 and 24 hr of fasting, although their body weights were not different before and after the fast. The results suggest that the genetic predisposition to dietary-induced obesity (DIO) is reflected in elevated fasting triglyceride levels. Presumably, differences in fasting triglyceride levels result from differences in mobilization and metabolism of body fat even before the appearance of hyperinsulinemia or hyperleptinemia. These results suggest a new way to predict susceptibility to DIO and provide impetus for further investigation of the mechanism underlying these differences in fat metabolism. Supported by NIH grant DK53109

Environmental temperature during pregnancy and body mass index in late adolescence. L. VAN HANSWIJK DE JONGE. Department of Psychiatry, St. George's Hospital Medical School, University of London, Cranmer Terrace, London UK SW17 0RE, UK.

Extending on the literature from anorexia nervosa, recent research has demonstrated links between environmental temperature during the intrauterine period and eating attitudes in late adolescence. There is a comparable impact of environmental temperature on birth weight and ingestion in animal studies, but little evidence on the link between environmental temperature and weight in humans. The aim of the present study was to establish whether environmental temperature during different stages of pregnancy is associated with body mass index during late adolescence. Body mass index (BMI) was correlated with mean environmental temperature during the intrauterine period for a sample of 578 adolescents (aged 15-19), living in Philadelphia. There was the predicted positive association with temperatures in the second trimester, but only among overweight females (BMI > 25). Closer examination revealed that this effect was present among the African American women, but not among the Caucasians. This effect did not apply to any group of males. Therefore, it appears that environmental temperature during pregnancy can influence the degree to which African American females are overweight. Possible biological and dietary mechanisms for this finding are discussed, but it will be essential to rule out the role of potential social factors, such as differential levels of poverty.

Effect of hydroxycitrate (HCA) on food intake and body weight regain in rats after hepatic branch vagotomy or sham vagotomy. M. LEONHARDT, W. LANGHANS. Institute of Animal Sciences, Swiss Federal Institute of Technology, 8092 Zurich, Switzerland. The feeding suppressive effect of HCA is possibly due to an increased hepatic fatty acid oxidation and ATP production caused by a reduced formation of the carnitine palmitoyltransferase I inhibitor malonyl CoA. This might be signaled to the brain by vagal afferents. We therefore examined whether the hepatic branch of the vagus is involved in the suppressive effect of HCA on food intake. Adult male rats underwent hepatic branch vagotomy (HBV; n=23) or sham vagotomy (SHAM; n=24). After recovery from surgery, all rats were fed restrictively (10 g/day) for 10 days and then switched to 10 days of ad lib feeding, with 11 HBV and 12 SHAM rats receiving a diet (75% [w/w] carbohydrates, 12% fat, 9% protein) supplemented with 3% HCA. The remaining animals were fed the same diet without HCA. During the first four ad lib days, HCA reduced food intake in HBV and SHAM rats to the same extent (to about 87% of control intake). Thereafter, the feeding suppressive effect of HCA appeared to be attenuated in HBV rats. HCA rats' cumulative food intake from ad lib day 5 to the end of the experiment (ad lib day 10) was 97 and 88% of control intake for HBV and SHAM rats, respectively (HBV: 151±4 vs 156±4g; SHAM: 149±5 vs 168±5 g). The overall feeding suppressive effect of HCA was significant, but the interaction between diet and surgery was not. These results fail to clearly establish a role of the hepatic branch of the vagus in the feeding suppressive effect of HCA.

Etomoxir, an inhibitor of fatty acid oxidation, affects hepatic energy status but does not increase feeding or reduce liver ATP. C.C. HORN, H. JI, M.I. FRIEDMAN. Monell Chemical Senses Center, Philadelphia, PA 19104, USA. Studies suggest that methyl palmitoxirate (MP; an inhibitor of fatty acid oxidation) stimulates feeding by reducing liver ATP and lowering hepatic energy status. It is difficult to determine the stimulus that increases feeding after MP treatment because many metabolic parameters in the liver and blood are changed by the inhibitor. We discovered that etomoxir (Eto), which has a similar metabolic effect as MP (both inhibit carnitine palmitoyltransferase I, which transports long-chain fatty acids into mitochondria for oxidation), does not stimulate feeding in rats (0.5, 1, 5, 10, 20, and 30 mg/kg, p.o.). Eto (10 and 20 mg/kg), like MP, reduced hepatic glycogen and liver phosphorylation potential, and increased hepatic ADP. Eto also reduced blood levels of ketone bodies and glucose, and increased free fatty acids--a blood metabolic profile similar to that produced by MP. The only observed difference between the metabolic effects of Eto and MP was on liver ATP; Eto did not affect liver ATP whereas MP reduces it. This suggests that the lowering of liver ATP is the signal that stimulates feeding following MP treatment. [This work was supported by NIH grants DK02894, DK35109 and DK36339.]

Taste and visceral representation in the PBN. JP BAIRD, JB TRAVERS, SP TRAVERS. Ohio State University, Columbus, Ohio

The parabrachial nucleus (PBN) is thought to participate in feeding control through integration of oral and visceral signals. Classic neuroanatomical studies, however, suggest that this morphologically heterogeneous nucleus represents these afferent signals separately, with taste located caudal, in the waist region and adjacent ventral lateral and central medial subnuclei, and visceral function rostral in the central lateral and external subnuclei. Accumulating evidence suggests this classification may be oversimplified. Hajnal et al. ('99) recently showed that gustatory cells in PBN are modulated by duodenal intralipid. Similarly, our studies demonstrate that although a visceral/gustatory topography is evident along the rostral/caudal axis of PBN, responses to gastric distention intermingle, converge with, and modulate taste responses in the ventral lateral and central medial subnuclei. Preliminary observations likewise suggest overlap between gustatory and duodenal, and between duodenal and gastric afferent signals in this same classical "gustatory" region. Moreover, an earlier study (Halsell & Travers, '97) demonstrated intraoral sensory representation in the more rostral, traditionally "visceral" external lateral and medial subnuclei, which corroborated conclusions from Yamamoto et al. ('94), that were based on Fos immunohistochemistry. The functional overlap of taste and gastrointestinal inputs to the PBN is further substantiated by more recent double-labeling anatomical studies. Injections of different anterograde tracers into widely separated rostral (taste-responsive) and caudal (gastric-distention responsive) zones of NST demonstrated evidence of a systematic topography, but in addition, revealed substantial overlap of the terminal fields in PBN.

c-Fos induction in the rat parabrachial nucleus after visceral, gustatory, and magnetic stimulation. T.A. HOUPPT. Department of Biological Science, The Florida State University, Tallahassee, FL, USA.

The parabrachial nucleus (PBN) is a secondary relay for both taste (medPBN) and visceral sensation (latPBN) that is critical for normal ingestive behavior and conditioned taste aversion learning (CTA). By comparing the patterns of cellular activity after diverse stimuli using c-Fos, we can determine the convergence of multiple sensory pathways in the PBN. By examining c-Fos in rats with central or peripheral lesions, we can establish the functional connectivity of the PBN within the neural network mediating ingestive behavior. Little or no c-Fos has been observed in the medPBN, so c-Fos may be a poor marker of gustatory activation. The latPBN expresses c-Fos after treatment with many unconditioned toxic or visceral stimuli (such as LiCl or gut peptides) but not after expression of CTA (i.e. after rejection of the conditioned taste stimulus). This is consistent with behavioral lesion studies that have shown the PBN to be necessary for CTA acquisition but not expression (e.g. Grigson, Norgren et al.). Lesion studies have also shown that the induction of c-Fos in the latPBN by visceral stimuli (e.g. LiCl or gut peptides) is partially dependent on vagal input, but not on area postrema input (although AP lesions can block the behavioral consequences of LiCl). Recently we have found that c-Fos is induced in the latPBN by magnetic field exposure sufficient to mediate CTA acquisition, possibly dependent on vestibular input. These convergent lines of evidence support a necessary (but not sufficient) role for PBN activation in responses to visceral stimuli and CTA learning. Supported by NIDCD 03198.

Medial and lateral parabrachial nucleus lesions affect learned aversions and sodium appetite differentially. R. NORRGREN, B. LI, D. WHEELER. Dept. Beh. Science, College of Medicine, Pennsylvania State University, Hershey, PA 17033

Under electrophysiological guidance, bilateral ibotenic acid lesions (x) were centered in the medial, gustatory (n=7) or lateral, visceral afferent areas (n=7) of the parabrachial nuclei (PBN). Prior to surgery, experimental and control rats learned a conditioned taste aversion (CTA, n=21, LiCl UCS) or served as saline controls (n=12). After recovery, they were tested for retention with that CS (sucrose), for sodium appetite, and for acquisition of a second CTA (Polycose), a learned odor aversion (2% vanilla CS), and a learned oral trigeminal aversion (corn oil CS). During the presurgery CTA, all the rats given LiCl (ip) learned to avoid the CS; the saline controls did not. After surgery, the medial and lateral PBNx rats that learned the CTA, retained it. When tested with another CS, however, neither group of PBNx rats acquired the new CTA. The medial PBNx squad learned the odor and the oil aversions; the lateral PBNx rats barely learned to avoid vanilla, and oil not at all. During sodium appetite, the reverse occurred -- medial PBNx rats failed to exhibit an appetite, but the lateral lesions produced exaggerated intake of 0.51 M NaCl. These results confirm and extend prior observations of functional differences within the PBN. Supported by NIH DC00240, MH43787, and MH00563.

Serotonin and cholecystokinin in the lateral parabrachial nucleus in the control of sodium and water intake. J. I. F. DE GOBBI, L. A. DE LUCA Jr., A. K. JOHNSON, J. V. MENANI. Department of Physiology and Pathology, School of Dentistry, Paulista State University (UNESP), 14801-903 Araraquara, SP, Brazil and Departments of Psychology and Pharmacology and Exercise Science, and the Cardiovascular Center, University of Iowa, Iowa City, Iowa 52242-1407. Bilateral injections of the non-selective serotonergic (5HT) antagonist, methysergide, into the lateral parabrachial nucleus (LPBN) significantly increase 1.8% NaCl intake in a number of forms of experimentally-induced salt appetite, including icv angiotensin, systemic administration of a diuretic (furosemide) plus angiotensin converting enzyme inhibitor, sodium depletion, water deprivation and treatment with deoxycorticosterone, while the administration of a 5HT receptor agonist into the LPBN substantially reduces the salt intake. Injections of the cholecystokinin (CCK) antagonist proglumide into the LPBN also increase NaCl intake. In this study, we investigated interactions between 5HT and CCK into the LPBN to control water and NaCl intake. Male Holtzman rats with cannulas implanted bilaterally into the LPBN were treated with furosemide + captopril to induce water and NaCl intake. Bilateral LPBN injections of high doses of the 5HT antagonist methysergide (4 µg) or the CCK antagonist proglumide (50 µg) alone or combined produced similar increases in water and 1.8% NaCl intake. Low doses of methysergide (0.5 µg) + proglumide (20 µg) produced greater increases in NaCl intake than when they were injected alone. The 5HT_{2a/2c} agonist DOI (5 µg) into the LPBN reduced water and NaCl intake. Following proglumide (50 µg) + DOI treatment the intake was not different from vehicle treatment. CCK-8 (1 µg) alone produced no effect. CCK-8 combined with methysergide (4 µg) reduced the effect of methysergide on NaCl intake. The data suggest that functional interactions between 5HT and CCK in the LPBN may be important for exerting inhibitory control of NaCl intake. Supported by FAPESP and CNPq

Infusion of Neuropeptide FF (NPFF) into the parabrachial nucleus (PBN) inhibits feeding stimulated by the mu-opioid agonist DAMGO. K.J. SIMANSKY, D.M. NICKLOUS. Dept. Pharmacology and Physiology, MCP Hahnemann University, Philadelphia, PA. 19102 USA.

The pontine parabrachial nucleus (PBN) mediates regulatory processes including some involved in feeding. Previously, administration of the mu-opioid agonist DAMGO into the PBN increased food intake. Neuropeptide FF (NPFF) is an octapeptide from a family of morphine modulating peptides. NPFF displays antiopioid and pro-opioid actions and decreased feeding after injection into the lateral ventricle of the brain. By immunocytochemistry, we confirmed dense concentrations of NPFF and mu-opioid receptors within the lateral PBN (IPBN). We therefore tested whether infusing NPFF (0, 1.25, 2.5 and 5.0 nmol/0.5 ul unilaterally) into the IPBN would inhibit feeding stimulated by parabrachial infusion of DAMGO (2.0 nmol/0.5 ul). Ten adult male Sprague-Dawley rats were housed individually, maintained with ad libitum food and water, and tested during the light. Fresh pelleted chow was provided after two injections and intake measured at 30, 120 and 240 min intervals. DAMGO increased 4-hr intake from 0.7 + 0.1 g to 3.3 + 0.3 g (P < 0.01). Pretreatment with NPFF reduced intake to 2.1, 1.2 and 0.7 g (each dose, P < 0.01 cf. DAMGO). Inhibition occurred within the latter two periods; feeding was minimal under all conditions during the first 30 min. These data lead to the hypothesis that NPFF plays a physiological role in modulating the orexigenic role of opioid peptides in the PBN in feeding. Supported by MH 41987 to KJS.

Effects of urocortin, leptin and melanocortin receptor ligands delivered to the parabrachial nucleus. H.J. GRILL, J.M. Kaplan. Psychology and Neuroscience, University Of Pennsylvania, Philadelphia, Pa. USA

Attention has focused on the hypothalamus as the neural substrate that mediates the potent feeding effects of various peptides. The caudal brainstem has received much less attention, despite widespread distribution of peptide receptors across various brainstem structures of known relevance to energy balance. We have delivered MTII and SHU-9119 (MC3/4-R ligands), urocortin (CRH1/2-R agonist) and leptin to the 4th ventricle and have demonstrated dose-related feeding effects comparable to those seen with forebrain ventricular delivery. To assess the contribution of specific brainstem sites to the 4th icv effect we first explored the dorsal vagal complex (DVC). We have reported that MTII, SHU-9119, urocortin, and leptin, all at doses subthreshold for icv action, significantly affect 24 h food intake when delivered unilaterally to the DVC. Here, we determine whether the same treatments also affect intake when delivered to the PBN, an important structure at the crossroads of hypothalamic-brainstem interaction known to contain receptors for each of these ligands. PBN injections [0.5 ul] of MTII (10 pmol), SHU-9119 (63 pmol) or urocortin (0.1 ug), yielded intake and body weight effects quite similar to those we have reported for DVC injection. Results for leptin (0.1 ug) approached but did not achieve significance. Taken together the positive results argue that an integrated perspective on the action of feeding relevant peptides must address actions in hypothalamus and in brainstem regions including at least the PBN and DVC, as well as interactions between these regions. Supported by DK-21397 and DK-42284.

Maternal modeling and availability influences on young girls' beverage intakes. J.O. FISHER, D.C. MITCHELL, H. SMITCIKLAS-WRIGHT, L.L. BIRCH. Dept of Pediatrics, USDA Children's Nutrition Res Ctr, Baylor College of Medicine, Houston TX 77030 USA.

Previous research demonstrated that the trade-off between milk and soft drinks in 5-year-old girls' diets was related to mother-daughter similarities in milk intake. This research investigates whether such similarities reflect maternal modeling influences on girls' beverage intakes or maternal influences on milk availability. Participants were 191 7-year-old girls and their mothers. Mothers' and daughters' body mass index (BMI), and intakes of energy, milk, soft drink, and calcium were assessed. In addition, mothers indicated how often they served milk to daughters. Girls' preferences for milk were assessed. Four groups were created using maternal milk intake (low=0 serv/day vs high=1.3 serv/day) and milk availability at meals and snacks (low=rarely-sometimes vs high=sometimes-most always). Planned contrasts were used to compare girls' intakes in the high maternal intake/high availability group with those in all other groups and in the low intake/high availability group. All analyses controlled for girls' milk preferences. Mothers in the high/high group had daughters with higher milk and calcium intakes, and lower soft drink intakes than girls whose mothers were in the low intake/high availability group ($p<0.05$). Mothers in the high/high group had daughters who were almost twice as likely ($p<0.05$; 95% CI=1.02-3.51) to meet the DRI for calcium than were girls in all other groups. Girls' energy intake and BMI did not differ across groups. These findings indicate that the positive influence of maternal modeling on young girls' beverage and calcium intakes is separate and additive to maternal influences on milk availability at meals and snacks.

Cultural factors related to mother's child feeding practices. S.L. JOHNSON. University of Colorado Health Sciences Center, Denver, Colorado.

Costanzo & Woody (1985) theorize that parents who struggle with weight and employ Restraint to control their own food intake are more likely to control their children's eating. We investigated relations between mothers' adiposity, eating styles, child-feeding practices and 5-11 year-old children's adiposity in Caucasian and Hispanic families. We hypothesized that: 1) mothers' Restraint would be related to restriction and monitoring of children's eating; and 2) mothers would report more monitoring and restriction in response to increasing adiposity in their child. Mothers completed: 1) the Child Feeding Questionnaire (child-feeding practices and perceptions of child's weight status); 2) the Eating Inventory (Restraint and Disinhibition); and 3) self-reports of their own height and weight. Children's anthropometrics were measured during school. We performed multiple regression analyses to predict mothers' 1) child-feeding practices and 2) concern about child overweight. Our sample included 272 mothers (144 Caucasian, 128 Hispanic) and their children (147 girls, 135 boys). Mothers'; Restraint was significantly linked to mothers'; restriction and monitoring in feeding daughters ($p<.05$). Caucasian mothers'; Restraint and child-feeding practices were significantly related to daughters'; current weight status ($p<.05$) and Caucasian mothers'; concerns about their daughters'; future weight ($p<.05$). Mothers'; Restraint was also related to restriction and monitoring of sons'; eating ($p<.05$) with Hispanic mothers'; feeding practices relating to concerns about sons'; future weight but not their current weight ($p<.05$). In conclusion, mothers'; Restraint and dieting may influence child-feeding practices. In Caucasian families, mothers'; Restraint and feeding practices are linked to their concerns for their daughters'; current and future weight.

Relationship of dietary restraint and dietary disinhibition to the quality of food choices of women and their young children. I.R. CONTENTO, C.E. BASCH, P. ZYBERT. Department of Health and Behavior Studies, Teachers College, Columbia University, New York, NY 10027, USA The purpose of this study was to examine the association of dietary restraint and dietary disinhibition with quality of diets of women and their children. 187 Latina women ages 24-47 completed the Three Factor Eating Questionnaire and reported on the food intakes of themselves and their 5-7 year old child using the Willett food frequency questionnaire. Heights and weights were also obtained. Cognitive restraint in mothers was significantly positively related to their own BMI, intake of vitamin C, skim milk, 9 fruits, 7 vegetables, fish, and chicken, no skin; and negatively correlated with calories, saturated fat, cholesterol, icecream, and cookies. Restraint was positively correlated with their child's intake of vitamin C, orange juice, shellfish and tuna. Rigid restraint (Westenhoefer, 1994, 1999) was additionally positively correlated with mother's intake of more fruits and vegetables and negatively with sugar, vitamin B1, iron, & phosphorus. Mothers' dietary disinhibition was positively correlated to their own BMI, intakes of calories, sugar and % fat, 6 meats, fish, 8 cakes and pastries, potato chips, and other carbohydrate foods; and negatively correlated with squash and raisins. Disinhibition was significantly positively related to their child's intake of calories, fat, protein, phosphorus, hamburger, onion rings, and pies; and negatively related to intakes of vitamin A, 3 vegetables and 3 fruits. Cognitive restraint (particularly flexible control) in this low-income Latina population is associated with higher quality diets of self and child rather than pathological eating; and disinhibition is associated with overeating by self and child of high fat, high sugar foods.

Dieting in adolescence: biology, family, or the environment? A. DREWNOWSKI. Nutritional Sciences Program, School of Public Health and Community Medicine, University of Washington, Seattle, WA 98195. One in three adolescent women is reported to be on a restricted-calorie diet to lose weight. Survey studies of approximately 2,000 school girls between the ages of 10 and 18 y suggest that dieting begins shortly after puberty and is accompanied by a negative body image and a fear of uncontrollable weight gain. The prevalence of dieting increased with age, reaching a peak of 36% in the 16-18 y age group. Generally, young women wished to weigh 10-12 lb less than their current weight. The prevalence of dieting among surveyed mothers was also 36% and there was a concordance between the mothers' and the daughters' dieting. Daughters who engaged in extreme dieting practices were more likely to report being encouraged to diet by their mothers. The desire for thinness was also influenced by socioeconomic status (SES). Respondents in higher income brackets, both mothers and daughters, were thinner and expressed a desire for additional weight loss. Despite high rates of dieting, there were few survey responses consistent with a probable diagnosis of bulimia nervosa (<2% of the daughters). Dieting in adolescent women, precipitated by weight changes during puberty, is influenced by familial factors as well as by the social environment.

Influence of mild Gestational Diabetes on appetite for sweet foods in pregnancy. B.J. TEPPER. Department of Food Science, Rutgers University, New Brunswick, NJ 08901, USA. Gestational Diabetes Mellitus (GDM) is a form of carbohydrate intolerance that occurs during pregnancy. GDM is a significant public health problem for women because it increases their risk of obstetric and fetal complications and Type 2 diabetes later in life. Dietary compliance among women with GDM is poor. Changes in preference for sweet taste are reported in other forms of diabetes and are purported to occur in healthy pregnancy, as well. The combined effects of GDM and pregnancy on appetite for sweet foods have not been studied. Several measures of appetite for sweet foods were compared in pregnant women recently diagnosed with GDM (n=25) and those without GDM (n=16). Outcome measures included liking of glucose solutions (0-30%), food cravings (by questionnaire), food-frequency (by questionnaire) and nutrient intakes (by 24-hr recall). Women were tested at 28-32 wk gestational age and 6-wk postpartum. Plasma glucose in women with GDM was significantly correlated with both peak, liking ratings for glucose ($r=0.64$; $p<0.001$) and intake of simple carbohydrates as fruit/fruit juices ($r=0.45$; $p<0.02$). No significant correlations were found in women w/o GDM. Fruit/fruit juices were the most frequently craved foods during pregnancy, but women with GDM were not more likely to crave fruit/fruit juices than women w/o GDM. Women with GDM consumed less simple sugar during pregnancy than after delivery ($p<0.05$). These data suggest that liking for sweet taste is elevated in women with GDM and is related to the severity of glucose intolerance. However, this group of highly compliant women restricted their intake of simple sugars. The health implications of these findings will be discussed.

Psychosocial and life style determinants of weight gain and abdominal adiposity in women during transition to menopause. A. COLLINS, T. SPIEGEL. Department of Clinical Neuroscience, Karolinska Institute, 171 76 Stockholm, Sweden and Department of Gastroenterology, Cooper Medical Center, Camden, NJ, 08103 USA.

Life style, perception of work role and personality traits may affect changes in body weight and body composition in women at menopause. The aim of the study was to examine changes in body weight and fat distribution in relation to life style, menopausal status, blood lipids, self-rated health, personality traits and perception of work role during transition to menopause. A population based sample of 150 women were assessed annually over five years from pre- to postmenopause using health screening, blood sampling for assays of hormones and lipids, registration of body weight as well as waist and hip circumference. Body weight, body mass index (BMI) and waist-to-hip ratio (WHR) increased significantly over the five years, regardless of menopausal status. Multiple stepwise regression analyses showed that increases in body weight and BMI were significantly associated with low levels of high density lipoprotein (HDL), less attention paid to regularity of meals and types of food eaten, low self-rated health, monotony avoidance and low work role satisfaction. Increased abdominal adiposity as measured by waist circumference and waist-to-hip ratio was associated with irregular food habits, low work role satisfaction, hostility, monotony avoidance and smoking. In an ongoing cohort study increased body weight was associated with irregular food habits and low physical activity level. The results showed that perimenopausal women gain weight over time and that the changes in body weight and body composition are related to perception of work role, life style and enduring personality traits rather than to menopausal status.

Melanin-concentrating hormone (MCH)-induced hyperphagia is not blocked by naloxone, a non-specific opioid antagonist. D.J. CLEGG, E.L. AIR, S.C. WOODS, R.J. SEELEY. Department of Psychiatry, University of Cincinnati Medical Center, PO Box 670559, Cincinnati, OH 45267-0559. A single injection of a very low dose (0.01 nmol) of Agouti-related protein (AgRP) stimulates food intake for up to six days. AgRP induces an increase in fos-like-immunoreactivity (FLI) 24-hr after injection in the lateral hypothalamus (LH). The increase in FLI in the LH may indicate activation of orexigenic neurons co-expressed in the LH. One such orexigenic peptide, melanin-concentrating hormone (MCH), receives innervation from AgRP neurons, is highly expressed in the lateral hypothalamus, and is therefore thought to be functionally related to AgRP. The acute feeding effects of AgRP can be blocked by a subthreshold dose of the non-specific opioid receptor antagonist, naloxone; however, naloxone does not block the AgRP-induced hyperphagia when given 24-hrs later. Therefore, the initial effects of AgRP engage opioid receptors, but the long-term effects do not. We therefore determined if naloxone would also attenuate the hyperphagia induced by central MCH, which reliably stimulates food intake over 2 hours during the light phase. The same subthreshold dose of naloxone which blocks the acute orexigenic effect of AgRP had no effect on i3vt MCH (5 μ g/rat)-induced hyperphagia. This indicates that opioid receptor activation, which is necessary for both NPY and AgRP's stimulation of food intake, is not necessary for MCH's hyperphagic effect.

Obese binge eaters have more global stress and report more pain tolerance and hunger following a cold pressor test (CPT). M. GLUCK, A. GELIEBTER, A. GALVAN, E. YAHAV, D. HUI, J. HUNG, S. HASHIM. New York Obesity Research Center. St. Luke's/Roosevelt Hospital, Columbia University College of Physicians and Surgeons. New York, NY 10025. Global life stress has been reported in binge eating disorder (BED), and greater pain tolerance has been observed in other eating disorders. Compared to non-binge eaters, we predicted BED subjects would have greater global stress and experience greater pain tolerance and stress-induced hunger following CPT. Subjects were 31 overweight (18f, 6m) individuals (BMI = 30.7 + 6.5 [SD], age = 30.4 + 7.5). They completed a diagnostic questionnaire (QEW) to assess BED (n=7) and non-BED (n=24), and the Perceived Stress Scale (PSS) to assess global stress. Subjects immersed their hand in 0-4°C ice water for 2 minutes and indicated when they first felt pain. Visual analogue scales assessed pain and hunger. After controlling for gender, the BED group had higher PSS scores (p = .03) than normals. During CPT, BED subjects showed a trend to report initial pain after a longer time than normals (p = .08). Surprisingly, BED subjects reported twice as much initial pain as normals, even after controlling for time in water (p = .008). There was a trend for the BED group to report higher hunger ratings at 2 minutes (p = .06). In a subset of subjects (2 BED, 3 normal) baseline cortisol, although not differing between groups, predicted time until initial reported pain (p = .08) and pain ratings (p=.05). Thus, BED subjects demonstrated greater global stress and increased pain tolerance following CPT even though they reported more pain, which may be due to their greater perceived stress. NIH grant: DK 54318.

Pegylated human recombinant leptin causes additional weight loss and affects LH levels in energy-restricted overweight and mildly obese men. C.J. HUKSHORN, M.S. WESTERTER-PLANTENGA, L.A. CAMPFIELD, W.H.M. SARIS. Nutrition Research Institute NUTRIM, Maastricht University, P.O. Box 616, 6200 MD Maastricht, The Netherlands.

Starvation induces a complex neuroendocrine response in humans as a defence mechanism. It is suggested that the decrease in leptin is an important signal to trigger this adaptive metabolic response*. To explore this question, a randomized, double-blind, placebo-controlled study was executed to investigate whether elevated leptin levels using long-acting pegylated human recombinant leptin (PEG-OB) influenced adaptive physiological responses to semistarvation induced by a VLCD (2 MJ/day) in 24 healthy overweight and mildly obese male subjects (mean ± SEM; 34.8 ± 0.9 yr, 96.1 ± 1.8 kg, BMI 28.8 ± 0.3). PEG-OB was well tolerated based on adverse event reports, lab values and vital signs. Weekly s.c. administration of 80 mg PEG-OB led to significant (p<0.03) additional weight loss (14.6 ± 0.8 vs 11.8 ± 0.9 kg) and reduction in appetite (p<0.05) after 6 weeks of treatment and energy restriction but did not affect changes in body composition, Resting Metabolic Rate, glucose tolerance and plasma metabolites. Furthermore, a significant relationship between the change in mean LH levels and PEG-OB treatment when corrected for weight loss was demonstrated. Based on the measurements made, PEG-OB treatment did not reverse the fasting-induced changes in the thyroid, corticotropic, somatotrophic axes and sympathetic nervous system activity. These results support the hypothesis* that stimulating appetite and suppressing reproductive function during starvation are the main physiological functions of leptin in humans.

*Flier JS. 1998 J Clin Endocrinol Metab 83:1407-1413

Taste perception of free fatty acids, i.e. CLA in humans. M.M.J.W. KAMPHUIS, M.S. WESTERTER-PLANTENGA. Dept. of human biology, University of Maastricht, The Netherlands.

In rats, free linoleic acid (LA), but not oleic acid has been shown to stimulate taste receptor cells. Moreover, an inverse relationship between fatty acid taste perception and fat preferences were shown (Gilbertson et al., 1998)¹. We investigated possible LA perception in humans and its relation to satiety and energy intake. Subjects were identified as linoleic acid tasters (LAT, n=14) when they distinguished *9 samples and as linoleic-acid-non-tasters (LANT, n=6) with <9 samples when testing 10 samples of 10 µM linoleic acid against its solvent. A low concentration free LA as conjugated linoleic acid (CLA), oleic acid and no supplementation was added to a low energy ice cream and a high energy ice cream. One out of six ice creams was offered ad libitum each week; low energy without CLA (LE), with CLA (LEC) or with oleic acid (LEO); high energy without CLA (HE), with CLA (HEC) or with oleic acid (HEO). Food intake, hedonics and appetite were measured. LAT and LANT did not differ in bodyweight, BMI and age. No differences in hedonics, food intake and feelings of hunger and satiety were seen between LAT and LANT. In the LAT group, but not in the LANT group, there was a relationship between amount eaten (g) and *satiety for LEC (r²=0.6, p<0.001). Sensory specific satiety, expressed as *pleasantness of taste for LEC and HEC was higher for LANT than for LAT (p<0.05). In conclusion, linoleic acid tasters could be distinguished by a taste test of 10µM linoleic acid. CLA induced satiety in fatty acid tasters when it was added to a low energy food, but did not result in a difference in food intake. CLA affected sensory specific satiety in linoleic acid non-tasters. ¹Gilbertson, Ann N Y Acad Sci, 1998; 855:165

Does (-)-hydroxycitrate reduce de novo lipogenesis? E.M.R. KOVACS, M.S. WESTERTER-PLANTENGA. Department of Human Biology, Maastricht University, 6200 MD Maastricht, The Netherlands.

Introduction: (-)-Hydroxycitrate (HCA) might promote weight maintenance by inhibiting or limiting the capacity for de novo lipogenesis (DNL)¹. In humans DNL is not of quantitative significance, but a persistent excess of energy intake as carbohydrate will promote DNL. We investigated whether HCA may prevent or reduce DNL in humans. Methods: Subjects were 10 lean men (BMI: 22±2 kg/m²; age, 24±5 y). They performed a glycogen depletion exercise test followed by a 3-day high-fat diet (F/CHO/P, 60/25/15; 100% of EE) and a 7-day high-CHO diet (F/CHO/P, 85/10; 130-175% of EE; overfeeding). During overfeeding they ingested 3x500 mg/d HCA or placebo (PLA). During the last days of overfeeding the subjects stayed for 60 h in a respiration chamber. Results: Body weight increased during overfeeding in both treatments (PLA, 2.8±0.2 kg; HCA, 2.9±0.2 kg; ns). Respiratory quotient (RQ) was >1.00 in all subjects indicating that DNL was occurring. On day 9, 24-h EE was lower with HCA compared to PLA (p<0.05). On day 10, resting metabolic rate (RMR) was higher (p<0.01) and RQ over night tended to be higher (p<0.1) with PLA indicating higher DNL; activity-induced EE was higher with HCA (p<0.05) indicating the urge to produce the excess of energy ingested. No sign. difference was found in non-protein RQ, fat and CHO oxidation or fat balance between PLA and HCA. Conclusion: An experimental condition that results in DNL in humans was created. Lower DNL with HCA was indicated by a lower EE, RMR and a tendency for a lower RQ over night; excess of energy intake was spent on activity. ¹Sullivan et al. Arch Biochem Biophys 1972; 150: 183-190.

Elevated dietary salt suppresses renin release but not thirst evoked by hypotension. S.D. STOCKER, C. KIMBROUGH, E.M. STRICKER, A.F. SVED. Department of Neuroscience, University of Pittsburgh, Pittsburgh, PA 15260.

Hypotension increases plasma renin activity (PRA) and water ingestion in rats. The induced drinking has been attributed to increased activity of the renin-angiotensin-system (RAS), since pharmacological blockade of the RAS markedly attenuates the associated water intake. The present study sought to determine whether maintaining rats on a high NaCl diet, a non-pharmacological means to suppress PRA, attenuates hypotension-induced drinking. Rats were fed a diet containing either a standard (1%) or high (8%) NaCl content for two weeks. Then, rats were implanted with femoral artery and vein catheters. Two days later, hypotension was produced by injection of diazoxide (DZX; 25 mg/kg, iv). Water intakes were measured every 15 min for 1 h, and blood samples (0.5 ml) for PRA assay were taken at baseline and 30 min after DZX. In both groups, DZX produced comparable decreases in blood pressure (~110mmHg to ~80mmHg). Hypotension-evoked water intakes also did not differ between the two groups (1%: 8.1±1.4ml; 8%: 8.8±1.2 ml at 60 min; n=6), despite large differences in PRA between 1% and 8% rats at baseline (3.9±0.2 vs 0.8±0.2 ng/ml/min) and 30 min (47.3±10.4 vs 5.9±1.2 ng/ml/min). Nonetheless, pretreatment with captopril (100 mg/kg, sc) attenuated DZX-evoked drinking to the same extent in both groups (60-min intakes, 1%: 1.1±0.4 ml, 8%: 2.4±1.0 ml; n=5). Thus, hypotension-induced drinking in rats on a high NaCl diet is still dependent upon the RAS and suggests that elevated dietary NaCl intake markedly enhances the dipsogenic potency of angiotensin II in rats.

Effect of urocortin in the lateral septal area on food intake. C. WANG, C. KOTZ. Veterans Affairs Medical Center Research service and University of Minnesota, Dept of Food Science & Nutrition. Minneapolis, MN 55417, USA.

Urocortin (UCN), a new corticotropin-releasing hormone (CRH)-related peptide, has shown appetite-suppressing effects more potent than CRF after intracerebroventricular administration. In our previous studies, injection of UCN into the paraventricular nucleus decreased feeding induced by food deprivation and neuropeptide Y. Lesion and electrical stimulation studies indicate that the lateral septum (LS) is important to feeding regulation. UCN-like immunoreactivity and mRNA encoding CRF2 receptor, to which UCN binds with high affinity, are found at high levels in the intermediate part of the LS (LSi). In our first study, injection of 3, 10 and 30 pmol UCN into the LSi significantly decreased feeding in food-deprived rats at 0-1 and 0-2 hour, and 10 and 30 pmol UCN still decreased feeding at 24 hours after injection. In contrast, CRH at 3, 10 and 30 pmol significantly inhibited feeding only at 1 hour after injection. In the second study, we tested potentially aversive effects of UCN in the LSi. UCN at 100 pmol caused a conditioned taste aversion (CAT) to saccharine solution, whereas at 10 and 30 pmol no CAT was observed. In the third study, pretreatment with 1 µg -helical CRF (9-41) (CRF receptor antagonist) blocked UCN-induced feeding inhibition at 1, 2, and 4 hour after injection. In conclusion, UCN significantly inhibits deprivation-induced feeding at doses that do not produce a CTA; and UCN feeding inhibition is blocked by antagonism of the CRH receptor, suggesting mediation by CRH receptors in this region. These data suggest that the LSi may be an important site for UCN-induced anorexia.

Lewis and Fischer 344 rats exhibit different responses to natural rewards and drugs of abuse. R.A. WHEELER, D.S. WHEELER, P.S. GRIGSON. The Penn State University College of Medicine, Hershey, PA 17033. Lewis rats exhibit greater responsiveness for cocaine than Fischer 344 rats in conditioned place preference, self-administration, and saccharin avoidance designs. Although several laboratories have investigated strain differences in drug sensitivity, no one has investigated strain differences in responsiveness to gustatory stimuli using brief access periods. Therefore we tested intake of multiple concentrations of NaCl, sucrose, citric acid, quinine, Polycose, saccharin, and capsaicin in the gustometer (Spector et al., 1990). The results showed that Lewis and Fischer rats had similar taste responses to sucrose, citric acid, quinine, and Polycose. However, Lewis rats responded more than Fischer rats for the NaCl and saccharin stimuli and for the trigeminal stimulus, capsaicin. Further, we expanded the investigation of strain differences in cocaine-induced avoidance of a saccharin conditioned stimulus in a dose response study. Analyses indicated that Lewis rats readily avoided the saccharin cue when it was followed by an injection of a 7.5 mg/kg dose of cocaine, but Fischer 344 rats did not demonstrate saccharin avoidance until a 20 mg/kg dose of cocaine was used. Further, we investigated strain differences in acquisition of a LiCl-induced conditioned taste aversion and found no difference between the two strains. These data indicate that Lewis and Fischer rats exhibit different responses to the natural saccharin reward, and to the drug of abuse, cocaine, but not to the aversive agent, LiCl. Supported by DA05932, DA09815, and DA 12473.

Of human bondage: Craving, obsession, compulsion, and addiction. M.L. PELCHAT. Monell Chemical Senses Center, Philadelphia, PA, USA

Is it more than a linguistic accident that the same term, craving, is used to describe intense desires for both foods and for a variety of drugs of abuse? There is strong evidence for common pathways that are affected by most addictive drugs. As the other presenters in this session will indicate, a strong case can also be made for some shared substrates for food and drug rewards in animals. There has been less explicit work on this topic in humans but many lines of evidence support the common mechanism view: As in the animal literature, opioid peptides seem to influence food palatability for humans. There is mounting evidence for comorbidity between drug/alcohol abuse and excessive craving or liking for sweets. Anecdotally, elderly individuals tend to "age out" of drug abuse, and the elderly also experience markedly fewer food cravings with age. If we focus on the compulsive aspects of food and drug cravings, there is also evidence for overlap: For example, activity in orbitofrontal cortex is associated with cocaine and alcohol craving. This area is also implicated in the pathology of obsessive-compulsive disorder. Although there is no direct evidence of orbitofrontal involvement in food cravings, there is indirect evidence such as higher than expected co-occurrence of obsessive-compulsive behavior and eating disorders.

Opioid modulation of taste hedonics within the ventral striatum. A.E. KELLY, M. ZHANG, M. WILL, S.N. HABER, T. STEININGER. Department of Psychiatry, University of Wisconsin-Madison, 6001 Research Park Blvd., Madison, WI 53705 There is a long-standing interest in the role of endogenous opioid peptides in feeding behavior and in particular in the modulation of food reward and palatability. Since drugs such as heroin, morphine, and alcohol activate this system, the question arises as to the interaction between food and drug reward with regard to the opioid system. We have studied in detail the functional role of enkephalinergic neurons and mu receptors within the nucleus accumbens and ventral striatum. This system appears to be a critical modulator of the affective response to food ("liking" in the system of Robinson and Berridge). We have conducted microinjection mapping studies of the structures efferent to the ventral striatum that may be involved in the opioid-mediated feeding response elicited from the ventral striatum, based on Fos studies. We have found that lateral hypothalamus, dorsomedial hypothalamus, ventral tegmental area, and nucleus of the solitary tract are all necessary for expression of this response. In additional recent studies we have found that daily exposure to a highly palatable food (chocolate Ensure) strongly decreases preproenkephalin (PPE) gene expression in the rat striatum and accumbens. Since chronic morphine has been found to have a similar effect on PPE gene expression, it may well be that repeated ingestion of sweet, calorie-rich food and certain aspects of opiate addiction may share a common molecular substrate. We have found that lateral hypothalamus, dorsomedial hypothalamus, ventral tegmental area, and nucleus of the solitary tract are all necessary for expression of this response. In additional recent studies we have found that daily exposure to a highly palatable food (chocolate Ensure) strongly decreases preproenkephalin (PPE) gene expression in the rat striatum and accumbens. Since chronic morphine has been found to have a similar effect on PPE gene expression, it may well be that repeated ingestion of sweet, calorie-rich food and certain aspects of opiate addiction may share a common molecular substrate.

Augmentation of drug reward by chronic food restriction: behavioral evidence and underlying mechanisms. K.D. CARR. Department of Psychiatry, New York University School of Medicine, New York, New York, USA. Chronic food restriction and maintenance of animals at low body weight increases the self-administration of most drugs of abuse. Research in this laboratory indicates that this phenomenon may be due to increased sensitivity of the neural substrate for drug reward. First, rewarding effects of centrally administered psychostimulants, opioids, and NMDA antagonists, indexed by their ability to lower threshold for lateral hypothalamic self-stimulation (LHSS), were shown to be greater in food-restricted than ad libitum fed rats. Second, the motor activating effects of these compounds were also shown to be enhanced by food restriction. Third, using c-fos immunohistochemistry, the cellular activating effects of centrally administered amphetamine and MK-801 were shown to be augmented in several dopamine terminal areas. Should the modulatory effect of food restriction be exerted within the dopamine synapse, it seems more likely to be exerted postsynaptically than presynaptically because rewarding effects of the direct D-1 agonist, A77636, and motor-activating effects of the direct D-2 agonist, quinpirole, were also augmented by food restriction. Because the augmenting effect of food restriction is reversed within several days of restored ad libitum access to food, humoral factors that vary dynamically with depletion and repletion of adipose stores may be involved in the regulation of drug reward sensitivity. Results of studies aimed at elucidating mechanisms underlying the behavioral and cellular effects of food restriction will be presented. Supported by DA00292 and DA03956 from NIDA/NIH.

Neurophysiological Analysis of Cocaine Self-Administration vs. "Natural" Reinforcement. R.M. CARELLI. Department of Psychology, The University of North Carolina at Chapel Hill, Chapel Hill, NC 27599 The nucleus accumbens (Acb) is a neural substrate crucially involved in mediating the reinforcing properties of 'natural' reinforcers such as food and water, and drugs of abuse such as cocaine. Research in my laboratory focuses on understanding how this structure processes reward-related information by using electrophysiological recording procedures in behaving animals. The multi-neuron recording technique involves surgical implantation of microelectrode arrays (16 wires) into the Acb, and the extracellular recording of Acb neurons in rats trained to press a lever for water or food reinforcement, or for an intravenous infusion of cocaine (i.e., drug self-administration). Using this approach, we recently completed a series of studies that examined the activity of the same Acb neurons in rats responding on multiple schedules for either two distinct natural reinforcers (water and food), or one of those natural reinforcers and the intravenous self-administration of cocaine. The results showed that the majority of neurons tested exhibited similar, overlapping neuronal firing patterns across the two natural reinforcer conditions. In contrast, the majority of neurons examined (> 90%) exhibited differential, nonoverlapping firing patterns relative to operant responding for water (or food) vs. cocaine reinforcement. These findings indicate that in the well-trained animal, cocaine activates a neural circuit in the Acb that is largely separate from the circuit that processes information about food and water reward. Ongoing studies are examining Acb cell firing relative to operant responding for a highly palatable sweet substance (sucrose) vs. cocaine reinforcement.

Beta-endorphin-induced feeding: modulation by opioid receptor and G-protein antisense oligodeoxynucleotides. R.J. BODNAR, R.M. SILVA, M.M. HADJIMARKOU, H.C. GROSSMAN, P. LIU, G.C. ROSSI, G.W. PASTERNAK. Queens College, CUNY and Memorial Sloan-Kettering Cancer Center, New York, NY. The classic opioid peptide, beta-endorphin stimulates feeding following ventricular and intracerebral administration in rats. Selective opioid antagonist studies implicate the mu, and secondarily delta and kappa opioid receptors in this response. The use of antisense oligodeoxynucleotide probes allow for the identification of specific exons of cloned receptors in mediating specific behavioral responses. The present study examined whether beta-endorphin-induced feeding was selectively altered by antisense probes directed against opioid receptors or G-protein alpha sub-units in rats. Beta-endorphin-induced feeding was significantly and selectively reduced by antisense probes directed against exons 1, 3 and 4 of the MOR-1 gene, and exon 1 of the DOR-1 gene; antisense probes directed against exons of the KOR-1 or KOR-3 genes were ineffective. These data confirm the importance of mu opioid receptors in this ingestive response. Beta-endorphin-induced feeding was significantly reduced by antisense probes directed against the alpha subunits of G₁₁ and G_{s/12}, and was significantly potentiated by antisense probes directed against the alpha subunits of G₁₂, G₁₃ and G_s. This pattern differs substantially from feeding elicited by other mu-selective opioid agonists such as morphine and its active metabolite, morphine-6beta-glucuronide. This suggests that beta-endorphin-induced feeding displays a different signal transduction profile from these traditional mu-selective opioid agonists.

Acute reduction of food intake by intracerebroventricular insulin. E. AIR, K. BLAKE, S. WOODS. Department of Psychiatry, University of Cincinnati, Cincinnati, OH 45267

When administered chronically into the 3rd-cerebral ventricle over several days (i3vt), the adiposity-signaling hormone, insulin, reduces food intake and body weight. To assess more acute effects of insulin, we have developed a paradigm in which rats are habituated to a schedule on which food (chow) is removed for 4 hr/day in the middle of the light cycle. At the end of the 4-hr deprivation they are given 30-min access to a 15% sucrose solution, and then chow is returned until the following day. After a week on the schedule, body weight is normal and daily sucrose intake is stable. I3vt insulin administered 3 hr prior to sucrose access significantly reduces both sucrose intake and chow intake over the entire day relative to i3vt saline. At a dose of 2 mU, sucrose intake was reduced by 60% and chow intake over the subsequent day was reduced by 42%, as compared to saline. Body weight was also reduced 24 hrs after doses of 2 mU and 4 mU of insulin. This paradigm allows investigation of the acute actions of i3vt insulin, and permits determining possible interactions with other acutely acting anorexic and orexigenic compounds.

Hyperphagia on a high-fat diet is attributed, in part, to a rise in circulating triglycerides and hypothalamic galanin and a reduction in insulin and leptin. S.F. LEIBOWITZ, P. PAMY, N. LEVENKOVA, J. DOURMASHKIN. The Rockefeller University, New York, N. Y. 10021. Rats given access to a high-fat diet (HFD) exhibit overeating during the first 1-2 weeks on the diet. To determine various parameters that may contribute to this hyperphagia, measures of the hormones insulin and leptin, of circulating triglycerides, and the peptide galanin (GAL) were taken in rats given exposure to a HFD (50% fat), as compared to a control diet (COND. 25% fat), for periods of 2 hours, 1 day, 5 days and 3 weeks. At each time interval except for 3 weeks, the rats on a HFD exhibit hyperphagia. In addition, the expression and immunoreactivity of GAL are both elevated in response to the HFD, even after a 2-hour meal, specifically in the paraventricular nucleus (PVN) but not the arcuate nucleus (ARC). Thus, this peptide, which is known to stimulate feeding behavior, may be a factor that contributes to the HFD-induced hyperphagia. An additional parameter involved in this behavior may be the hormone, insulin. This is suggested by the finding that central insulin injection inhibits the expression and production of GAL in the PVN, while inhibiting fat ingestion, and a HFD at all intervals tested reduces circulating levels of insulin. Whereas the adipocyte hormone, leptin, also inhibits GAL in the PVN but not the ARC and reduces fat intake, the evidence does not support a role for this hormone in chronic hyperphagia. That is, leptin generally rises with body fat and GAL in rats maintained on a HFD. This hormone, however, may have a short-term function in controlling meal size, since it is actually reduced by consumption of a high-fat meal, while both PVN GAL and meal size are enhanced. At each of the 4 time intervals tested, consumption of a high-fat diet also causes a rise in circulating triglycerides. The importance of this dietary lipid in stimulating GAL, and consequently meal size, is indicated by strong, positive correlations invariably seen between this lipid, PVN GAL and caloric intake on a HFD. Thus, it is concluded that the overeating invariably associated with consumption of fat-rich food may be attributed to elevated triglycerides and GAL in addition to a reduction in insulin and leptin.

Central neuropeptide Y (NPY) and peripheral cholecystokinin (CCK) have opposing, independent, and summative effects on food intake in rats. S. AJA, K.J. MILLS, T.H. MORAN. Dept. Psychiatry, Johns Hopkins Univ. Sch. Med., Baltimore, MD 21205.

During feeding, meal-related signals are produced that exert negative feedback on ongoing food intake. Food intake is also influenced by hypothalamic signaling systems involved in and reflecting overall energy balance. Hypothalamic signals may influence food intake partly by modulating responsiveness to meal-related negative feedback. For example, icv leptin potentiates the satiety response to CCK, a peptide released from the gut during a meal. Leptin may reduce feeding, in part, by reducing the production of NPY, an orexigenic peptide, from the arcuate hypothalamic nucleus. Under conditions of hypoleptinemia, such as fasting, NPY production is elevated, likely contributing to an increased drive to eat. To determine if NPY reduces the hypophagic response to CCK, we injected rats icv with 1 nmole NPY or saline vehicle in the lateral ventricle two hours before, and i.p. with doses of CCK-8 (0, 0.32, 1, 3.2 nmole/kg) in saline just prior to lights out and chow access. CCK reduced 30-min chow intake dose-dependently. NPY increased 30-min and 4-h chow intake. There was no significant interaction between the NPY and CCK effects. Thus, relative to the different baselines with and without NPY, CCK's ability to reduce food intake as measured by grams consumed was not altered. These results suggest that NPY and CCK might act independently to affect food intake, and do not provide a clear suggestion that NPY influences the satiety response to CCK in a specific or mechanistic manner. Supported by DK19302.

Suppression of gastric emptying, but not of sucrose ingestion, by hindbrain cocaine- and amphetamine regulated transcript (CART) peptide 55-102 is dependent on corticotropin-releasing factor receptors. U. SMEDH, T.H. MORAN. Dept. of Psychiatry and Behavioral Science, Johns Hopkins School of Medicine, Baltimore, MD, USA. Cocaine- and amphetamine regulated transcript (CART)-derived peptides (CARTp) have been shown to suppress food intake and gastric emptying when injected into the lateral cerebral ventricle. Accumulating anatomical and functional evidence suggest that CARTp may interact with corticotropin-releasing factor (CRF) to produce some of its effects, which may involve brainstem neuronal targets involved in gastric controls and food intake regulation. We investigated whether CRF receptors are involved in the control of brainstem-elicited suppression of food intake and/or gastric emptying by CARTp 55-102. Rats equipped with chronic guide cannulas aimed at the fourth ventricle, some of which were bearing chronic intragastric fistulas were used. For gastric emptying assessment, the rats received intragastric infusions (1.0 ml/min) of 12 ml 12.5% glucose and gastric samples were withdrawn immediately after the gastric infusion to reflect emptying during gastric fill. CARTp (0.5 microg and 1.0 microg, but not 0.1 microg) injected fourth i.c.v. suppressed gastric emptying during gastric fill ($p < 0.01$). The effect of 1.0 microg CART given 4th i.c.v. on gastric emptying, but not on sucrose intake, was blocked by pretreatment with the CRF antagonist alpha-helical CRF9-41 (10 nmol). This demonstrates that CARTp suppresses gastric emptying via a CRF-dependent brainstem mechanism whereas CARTp-induced suppression of food intake is independent of the integrity of brainstem CRF receptors. (Supported by DK 19302 and the Wenner-Gren Foundation.)

The nonpeptidic ORL1 receptor agonist, Ro 64-6198, reverts restraint stress- and CRF-induced anorexia in rats. R. CICCOCIOPPO, M. BIONDINI, L. ANTONELLI, J. WICHMANN*, F. JENCK*, M. MASSI. Department of Pharmacological Sciences and Experimental Medicine, University of Camerino, 62032 Camerino, Italy; *Pharma Division, Preclinical Research, F. Hoffmann-La Roche Ltd., CH-4070 BASEL, Switzerland. Nociceptin/orphanin FQ (NC), the endogenous ligand of the opioid receptor-like1 (ORL1) receptor, exhibits antistress and hyperphagic properties and has been shown to antagonize hypophagia induced by stress or central administration of corticotropin releasing factor (CRF) in rats. The present study evaluated whether Ro 64-6198, a nonpeptidic ORL1 receptor agonist, reduces restraint stress- or CRF-induced anorexia. Twenty-h food deprived rats received intraperitoneal (IP) Ro 64-6198, 10 min before 60-min body restraint or 20 min before intracerebroventricular (ICV) injection of CRF (200 ng/rat). Feeding was markedly reduced by body restraint or CRF. IP pretreatment with Ro 64-6198 reverted hypophagia induced by stress or CRF; the effect was statistically significant at 0.3, 1 or 2.5 mg/kg. The same doses did not reduce the anorectic effect of E. coli lipopolysaccharide, suggesting that the effect is selective for stress- or CRF-induced anorexia. The effect of Ro 64-6198 on CRF-induced anorexia was significantly reduced by ICV pretreatment with [NPhe1]NC(1-13)NH₂, a selective ORL1 receptor antagonist. In freely feeding rats, Ro 64-6198 significantly increased feeding at 2.5, but not 0.3 or 1 mg/kg; thus, the effect on stress- or CRF-induced anorexia can be evoked at doses not hyperphagic, suggesting that it may be related to the antistress properties. Diazepam was unable to reduce the anorectic effect of CRF at the anxiolytic dose of 0.3 mg/kg, and partially reduced it at the hyperphagic dose of 1 mg/kg. Present results confirm those obtained with NC and suggest that Ro 64-6198 may represent an interesting tool for treatment of stress-induced anorexia.

Intermittent fenfluramine administration to rats suppresses food intake despite chronic brain serotonin suppression. S. CHOI, E. JONAK, L. SIMPSON, V. PATIL, J.D. FERNSTROM. UPMC Weight Management Center, University of Pittsburgh Medical Center.

The mechanisms by which fenfluramine suppresses food intake and body weight have been linked to its ability to enhance transmission across serotonin synapses in brain. This drug initially lowers body weight and suppresses food intake, yet after chronic administration food intake soon returns to normal and body weight no longer decreases. Fenfluramine also reduces brain serotonin levels, which might account for the loss of appetite suppression. We assessed if intermittent, chronic fenfluramine administration could suppress food intake during each treatment period, and if so, whether such an effect occurs in the presence of greatly reduced brain serotonin levels. Rats were injected once daily with 10 mg/kg d,l-fenfluramine for 5 days, and then maintained 5 days without injections. This 10-day sequence was repeated 5 more times. During each period of fenfluramine administration, daily food intake dropped markedly the first 1-2 days of treatment, but returned to pretreatment values by day 5. Body weight dropped modestly during each period of fenfluramine administration, and rose during each subsequent period when injections had ceased. Serotonin levels and synthesis rates in several brain regions were markedly reduced throughout the experiment. Despite the long-term reduction in brain serotonin, fenfluramine continues to reduce food intake and body weight. This suggests that serotonin receptors involved in reducing food intake are unaffected by a long-term suppression of neuronal serotonin pools, and that the food intake reduction may result from an action of the drug that does not involve its promotion of serotonin release or blockade of presynaptic serotonin reuptake.

Distribution of Serotonin (5-HT) 1B Receptors in the Rat Diencephalon. I.G. MAKARENKO^{1,2}, M.M. MEGUID¹, M.V. UGRUMOV². ¹Neuroscience Program, Surg. Metab./Nutrition Laboratory, SUNY Upstate Medical University, NY 13210, ²Institute Developmental Biology RAS, 117808 Moscow, Russia. Introduction: 5-HT_{1B} receptors are involved in many pathophysiological processes including eating disorders, obesity and anorexia, in that they may control the release of other neurotransmitters in the brain. Morphological investigations of 5-HT_{1B} receptors have focused mostly on cortex, striatum and basal ganglia. No data exist about their distribution in the diencephalon, areas of great interest in relation to control of food intake. Method: We visualized 5-HT_{1B} receptors immunocytochemically on cryostat sections of paraformaldehyde fixed Fisher rat brain by PAP method using specific primary antibodies generated to the third large intracellular loop of the 5-HT_{1B}-receptor protein (Chemicon) diluted 1:2000. Results: Immunostaining revealed an abundance of neurons with 5-HT_{1B} reaction in the cytoplasm of cell bodies and proximal dendrites. 5-HT_{1B} immunoreactive (IR) neurons were highly specifically distributed in the hypothalamus and thalamus. The most intense IR was observed in the hypothalamic magnocellular nuclei (suoroaptic, paraventricular, retrochiasmatic) and parvicellular arcuate nucleus. Prominent group of large and medial neurons with different 5-HT_{1B}staining pattern are located in the lateral hypothalamus with specific distribution in rostrocaudal direction. Medium and light staining was observed in neurons of anterior periventricular, ventromedial and dorsomedial hypothalamic nuclei. In thalamus neurons expressed 5-HT_{1B} receptors are localized in anterodorsal and medial habenular, paraventricular, anterodorsal, reticular nuclei and in zona incerta. Conclusion: These data serve as a morphological background for postulating the role of 5-HT_{1B} receptors in the brain and their involvement in the processes of neural and humoral control of food intake.

Effects of the 5-HT_{1A} receptor agonist 8-OH-DPAT on food intake in obese Zucker rats of different ages. J.-P. VOIGT, H. HÖRTNAGL*, B. BERT, H. FINK. Institute of Pharmacology and Toxicology, School of Veterinary Medicine, Freie Universität Berlin, Koserstr. 20, D-14195 Berlin, Germany. *Institute of Pharmacology and Toxicology, Medical Faculty (Charité) of the Humboldt University at Berlin, D-10098 Berlin, Germany. Changes in brain serotonin (5-HT) metabolism as reported in Zucker rats may also alter feeding or satiety responses to serotonergic drugs. We investigated the effect of the 5-HT_{1A} receptor agonist 8-OH-DPAT on feeding in 3, 6, and 10 months old obese Zucker rats. In addition, we determined 5-HT content and 5-HIAA/5-HT ratio in various brain regions. At the age of 3 months, 8-OH-DPAT (100 µg/kg) stimulated feeding in lean control rats as expected, but failed to do so in obese Zucker rats. In contrast, the higher dose of 300 µg/kg 8-OH-DPAT inhibited food intake in the obese rats, but had no effect in lean controls. This pattern of action did not change in 6 months old rats. At 10 months of age, 8-OH-DPAT lost its inhibitory activity in Zucker rats, but still significantly stimulated feeding in lean controls (300 µg/kg). At the age of 3 months, 5-HT levels were significantly higher in the hypothalamus, the frontal cortex, and the parietal cortex of obese Zucker rats and were associated with a lower 5-HIAA/5-HT ratio. In the hypothalamus and the parietal cortex the difference was still significant in 6 months old rats, whereas no significant differences were observed in 10 months old rats. The data indicate persistently different feeding responses to 8-OH-DPAT when obese Zucker rats and lean controls are compared. This finding may be related to changes in brain 5-HT metabolism in the obese Zucker rats, but also to changes at pre- and/or postsynaptic 5-HT_{1A} receptor sites.

Sham-fed sucrose increases accumbens dopamine in a concentration- dependent manner: a microdialysis study in behaving rats. A. HAJNAL, G.P. SMITH*, R. NORGRÉN. Dept. Behavioral Science, College of Medicine, The Pennsylvania State University, Hershey, PA, 17033; *Bourne Laboratory, Dept. Psychiatry, New York Hospital-Cornell Medical Center, White Plains, NY, 10605

There is considerable evidence that central dopamine (DA) mediates the positive reinforcing effect of sapid sucrose on eating (Smith, 1995). To test this idea further, we investigated the relationship between the intensity of orosensory stimulation by sucrose during 20 minutes of sham-feeding and the release of DA from the nucleus accumbens shell (NAc) using chronic microdialysis and HPLC. Rats (n=14) were maintained on food ad libitum, water deprived overnight, and trained to sham-feed 3 sucrose solutions (0.03, 0.1, 0.3M). Sucrose stimulated ingestion was a linearly increasing function of concentration (0.03M: 18.84±1.01ml; 0.1M: 30.41±1.52; 0.3M: 39.64±1.48ml; ANOVA: F(2,42)=19.07; p<0.0001; r=0.69, F(1,43)=38.96; p<0.0001, n=45). sucrose also stimulated the release of DA from the NAc as a function of concentration (0.03M: 120.76±2.6%; 0.1M: 140.28±7.8%; 0.3M: 146.27±5.05%; ANOVA: F(2,39)=6.57, p<0.01). There was a positive correlation between sucrose concentration and DA release (r=0.41, F(1,54)=10.901, p<0.002) and between total intake and DA release (r=0.37, F(1,54)=8.76, p<0.01). These results demonstrate that sham-feeding of sucrose activates the NAc DA system as a linear function of stimulus concentration, and they provide further evidence for the hypothesis that central DA mediates the positive reinforcing effect on eating of orosensory stimulation by sucrose. Supported by NIH grants DC00240, DC04751, MH40010, MH00653.

The effect of centrally administered lithium chloride on conditioned taste aversion and c-fos in rats. J.M. BARRANCO, J. LORCH, T.A. HOUPPT. Department of Biological Science, The Florida State University, Tallahassee, FL, USA.

Pairing an intraperitoneal injection of LiCl with a novel tastant leads to the acquisition of a conditioned taste aversion (CTA) in rats. The site of lithium's action is unknown. We determined whether central administration of LiCl is sufficient for CTA acquisition. We also examined c-fos -like immunohistochemistry (c-fos) to determine which brain regions were activated by central injections independent of behavioral responses. Rats were implanted with intracerebroventricular (ICV) cannulas. After one week, cannula placement was verified by increased water intake after angiotensin injection. Rats were placed on a deprivation schedule with six hour access to water daily. After a week, rats were given 30 minute access to 5% sucrose, and then received LiCl or NaCl ICV (5 ul, 0.15 or 0.6 M, n= 5-8/group). CTA expression was tested by 30 minute access to sucrose 48 hours after the pairing of sucrose and ICV LiCl. Sucrose intake was significantly decreased in rats that received 0.6 M LiCl, but not 0.15 M LiCl, compared to NaCl injected rats. One week later, the rats were given another ICV injection of LiCl or NaCl and perfused one hour later. Brains were processed for c-fos. 0.15 M LiCl induced c-fos only in the supraoptic n. 0.6 M LiCl induced c-fos in multiple brain regions, including amygdala and cortex. We conclude that 0.15 M LiCl is sufficient to induce c-fos but not CTA, and 0.6 M LiCl induces c-fos expression and CTA. Thus, LiCl does have direct central effects. Supported by NIDCD03198.

Effect of deprivation condition and inter-solution interval on anticipatory contrast obtained with sucrose-sucrose and saccharin-sucrose pairings. M.H. LESZCZUK, T. ALLEN, S. JUDGE, C.F. FLAHERTY. Department of Psychology, Rutgers University, Piscataway NJ 08854 USA.

Allowing rats daily brief access (3 min) to a 2% sucrose solution or 0.15% saccharin solution prior to brief access (5 min) to a 32% sucrose solution results in suppression of intake to the first solution compared to control groups that receive 2% sucrose or 0.15% saccharin as both the first and second solutions. This anticipatory negative contrast (ANC) effect diminishes as the inter-solution interval (ISI) increases. The present experiment examined the joint effect of ISI (0 seconds or 5 minutes) and deprivation condition (free-fed or 82% body weight) in rats given daily pairings of 0.15% saccharin and 32% sucrose or 2% sucrose and 32% sucrose. With a 0-sec ISI large and equivalent ANC effects occurred in 0.15-32 and 2-32 groups. However, ANC was considerably smaller in free-fed rats in that ANC occurred in only the last three blocks (days 9-14) in the 0.15-32 group and there was no reliable ANC in the 2-32 group. Thus, deprivation enhanced the relative suppression of the initial substance and more so with 2% sucrose than with 0.15% saccharin. Preliminary data obtained with a 5-min ISI suggest that contrast will develop with 0.15-32 pairings in both free-fed and deprived rats. However, contrast develops more slowly or not at all in both deprived and free-fed rats given the sucrose pairings (2-32). This result suggests that a 5-min period between first and second solutions negates the devaluation of a sucrose solution, but not of a saccharin solution, that normally occurs in the ANC procedure.

Sucrose intake in Sprague-Dawley and Lewis rats in two consummatory contrast procedures. C.F. FLAHERTY, M. MCCOOL, M.H. LESZCZUK. Department of Psychology, Rutgers University, Piscataway NJ 08854 USA.

Male Sprague-Dawley (SD) and Lewis rats were tested in two consummatory contrast situations - successive negative contrast (SNC) and anticipatory negative contrast (ANC). In the SNC procedure, shifted rats (32-4) were given a 10-day period of access to 32% sucrose for five min per day and then shifted to 4% sucrose for four days. Unshifted control rats (4-4) received 4% sucrose for all 14 days. During the preshift period the Lewis rats consumed more 32% sucrose than the SD rats, but consumption of the 4% solution was equivalent in both strains. In the postshift period, both strains showed equivalent SNC effects (rats shifted from 32% to 4% sucrose consumed less 4% than unshifted 4% rats). In the ANC procedure the contrast group was given daily 3-min access to 4% sucrose followed by 3-min access to 32% sucrose, whereas the control group was given two access periods to the 4% solution. The Lewis rats consumed more of both the bottle 1 and bottle 2 solutions than did the SD rats. Both Lewis and SD rats showed suppressed intake of the 4% solution when it preceded 32% sucrose (ANC effect). Contrast tended to develop earlier in the Lewis rats than the SD rats, but was terminally larger in the SD rats. The difference in contrast was due primarily to greater suppression in the contrast group (Group 4-32) group in the SD rats rather than to strain differences in consumption in the 4-4 control group.

The insular cortex and the prefrontal cortex are involved in the association between taste and odor in the rat. N. SAKAI, S. IMADA. Department of Psychology, Hiroshima Shudo University, 731-3195 JAPAN.

It is suggested that the flavor perception is formed through the association between the taste and the odor. However, the brain mechanisms of the flavor perception have not been fully understood yet. Thus, we aimed to investigate whether the rats can acquire the association between the taste and the odor. First, the rats were presented the pairings of the odor (coffee flavor or grape flavor) and the 0.3 M NaCl solution. Then, the rats were received the furocicide to develop the sodium deficiency. The sodium-deprived rats ingested the water flavored with the odor that had been paired with NaCl. On the other hand, the normal rats avoided ingesting the water flavored with the odor that had been paired with NaCl. The rats with lesions in the insular cortex (including the gustatory cortex) or in the prefrontal cortex (including the orbitofrontal cortex) could not show preference and aversion to the water flavored with the odor that had been paired with NaCl, although these rats could show sodium appetite. Thus, we concluded that the rats could acquire the association between the taste and the odor, and that this association was controlled by the insular or the prefrontal cortices.

Anticipatory contrast effects are disrupted by a local context change both before and after the CS-US pairing. P.S. GRIGSON, V. SANCHEZ, S.M. BALLARD. The Penn State University College of Medicine, Hershey, PA 17033 Intake of a palatable saccharin conditioned stimulus (CS) is disrupted if it comes to predict the future availability of a highly preferred sucrose unconditioned stimulus (US) following once daily pairings. This reduction in CS intake is referred to as an anticipatory contrast effect (ACE, Flaherty & Checke, 1982). Although ACEs are typically robust, Experiment 1 confirmed that the ACE is greatly disrupted when using a "delay/cue procedure" in which each saccharin-sucrose pairing is preceded by onset of the house light 30 sec into a 60 sec delay period and then followed by offset of the house light. The results of Experiment 2 verified that the disruption to the ACE in Experiment 1 was due to the change in lighting both before and after the CS-US pairings, not to the 60 sec delay period that preceded CS access. Finally, the results of Experiment 3 showed that the disruptive effect of the delay/cue procedure can be attenuated if the animals are habituated to the change in lighting prior to the first CS-US pairing. Taken together the data show that the development of ACE depends upon the establishment of the CS-US association, which is greatly impaired if the presentation of the CS-US pairing is preceded and followed by a change in lighting in the chamber. Supported by DA09815 and DA12473.

The suppressive effects of cocaine self-administration on saccharin vs. malic acid intake: Acquisition and reinstatement. R.C. TWINING, P.S. GRIGSON. Penn State College of Medicine, Hershey, PA 17033.

The suppressive effects of iv cocaine on intake of a saccharin or a malic acid conditioned stimulus (CS) were evaluated in two experiments. In Experiment 1, water-deprived rats were given 5 min access to 0.15% saccharin. This bottle was then retracted and an empty spout advanced for 1 hour and every 10 licks led to an iv infusion of 0.2 ml of saline (n=17) or 0.33 mg cocaine (n=18). There was one such taste-drug pairing a day for 13 days. Following 30 days to 6 months of abstinence, the rats were exposed to the same regimen for one test day, but no drug was delivered. The results showed that greater avoidance of the saccharin cue was associated with greater cocaine self-administration and that this pattern was still evident following 1 to 6 months of abstinence. In Experiment 2, using the same conditioning procedure, we directly compared the suppressive effects of cocaine self-administration when 0.15% saccharin (n=18) or 0.01 M malic acid served as the CS (n=18). Following 30 days of abstinence, self-administration behavior was extinguished, and then reinstated following presentation of saccharin or malic acid (n=4 or 5 per cell). The results showed that the nature of the CS did not affect acquisition of either the reduction in CS intake or self-administration of the cocaine. However, during reinstatement, the malic acid gustatory cue was more effective at reinstating drug seeking behavior than was saccharin. Supported by NIH grants DA 09815 and DA 12473.

Acquisition of conditioned trigeminal aversions in rats with parabrachial nucleus lesions. P.L. SMITH¹, R. NORRGREN², G. SCALERA³, J.C. SMITH¹, P.S. GRIGSON². ¹Department of Psychology, Florida State University, Tallahassee, FL 32306-1270; ²Department of Behavioral Science, College of Medicine, Pennsylvania State University, Hershey, PA 17033; ³Department of Biomedical Science, University of Modena, 41100 Modena, Italy. It has been shown that rats with bilateral parabrachial nucleus (PBN) lesions fail to acquire an aversion to a gustatory conditioned stimulus (CS) when it is paired with a LiCl injection. The same rats can learn an aversion to oral trigeminal stimuli like capsaicin or 100% corn oil. Thus, the PBN appears to play an essential role in a standard conditioned taste aversion (CTA), but it may be less critical for learning aversions to trigeminal stimuli. In a series of three experiments, it was determined whether PBNx rats were able to learn an aversion to three different "trigeminal stimuli (Purina wet mash, water at 10°C, pure corn oil)." In each of the experiments, 28 rats (14 SHAM surgery, 14 PBNx) were given access to a particular CS. Half of the rats in each surgery group were then injected interperitoneally with 0.15 M saline or 0.6 M LiCl. A 2-choice preference test was conducted 24 hrs later, between the CS and a CS- (a stimulus not paired with an injection). In the two experiments using wet mash and 10°C water, further post-conditioning tests were conducted. The results showed that while the SHAM and the PBNx rats acquired an aversion to all three stimuli, the aversions to the wet mash and 10° water were rapidly extinguished for the PBNx-LiCl group. These results clearly demonstrate that PBNx rats can associate trigeminal stimuli with LiCl-induced illness. Once acquired, however, the extinction data show that this association is more transient than that acquired by the SHAM rats.

Estradiol does not affect sham feeding of sucrose in ovariectomized rats. M. MANGIARACINA, N. GEARY. Bourne Laboratory, NY Presbyterian Hospital-Weill Cornell Medical College, White Plains, NY 10605 USA. Estradiol (E_2) reduces meal size (MS) both tonically and cyclically, during estrus. Part of the mechanism involves increases in the potencies of postingestive controls of feeding. The possibility that E_2 also changes orosensory controls of feeding has not been extensively investigated. Therefore, we tested the effects of a physiological, cyclic regimen of E_2 treatment on intake and lick microstructure in rats sham feeding 0.2, 0.4 and 0.8 M sucrose. Female Long-Evans rats were bilaterally ovariectomized and implanted with gastric sham feeding cannulas. Half the rats were treated with E_2 (one SC injection of 2 μ g E_2 benzoate in 100 μ l sesame oil every fourth day) and half with oil alone. Tests began after five treatment cycles, when E_2 -treated rats weighed on average 37 g less than untreated rats ($p < 0.001$). Sham feeding tests (45 min) were done after overnight food deprivation (1) just prior to E_2 treatment, modeling early diestrus, and (2) on the second day after treatment, modeling estrus. Rats were adapted on each sucrose concentration until sham intakes stabilized, and data from the last tests were analyzed by ANOVA. There was a significant, although modest, increase in sham intake with increasing sucrose concentration ($p < 0.003$), but there were no significant effects of E_2 on sucrose intake or lick microstructure on either treatment cycle test day for any sucrose concentration. These results extend previous evidence that E_2 does not affect the processing of the positive feedback control of meal size produced by sweet taste. Supported by NIH DK 54523.

Flavor preferences reinforced by the postingestive effects of glucose but not galactose in preweanling rats. K.P. MYERS, A. SCLAFANI. Department of Psychology, Brooklyn College of CUNY, Brooklyn, NY 11210, USA. Adult rats acquire robust preferences for flavors that have been paired with the postingestive effects of nutrients. Little is known of the ontogeny of these learning capacities. In a series of experiments, infant rats were removed from the dam each afternoon from P16-P19 and given two oral infusions (1.2 ml/8 min) separated by 1 hr. The two infusions contained different cue flavors mixed in either a carbohydrate solution or a .05% saccharin solution. Following weaning, preferences were measured in two-bottle intake tests with both flavors now mixed in .05% saccharin. In Exp. 1, pups preferred a flavor previously mixed in glucose over a flavor previously mixed in saccharin. Exp. 2 implicated postingestive reinforcement in this learning, as pups learned to prefer a flavor paired with glucose over a flavor paired with sucrose, which is sweeter but relatively non-nutritive for pups (due to low intestinal sucrase levels). In Exp. 3, pups failed to acquire a preference for a flavor paired with galactose (which is converted to glucose postabsorptively), nor did they exhibit a conditioned avoidance of the galactose-paired flavor as adult rats do. These studies demonstrate an early competency to acquire conditioned flavor preferences based on postingestive factors. The apparent inability of galactose to reinforce conditioned preferences provides information regarding potential postingestive sites of action of nutrient reinforcement. (Supported by NIDCD DC00444 and NIDDK DK-31135)

Conditioned changes in evaluation of a flavor paired with the postingestive effects of glucose: A taste reactivity analysis. K.P. MYERS, A. SCLAFANI. Department of Psychology, Brooklyn College of CUNY, Brooklyn, NY 11210, USA.

Rats learn to prefer a flavor paired with intragastric (IG) nutrient infusions over a flavor paired with non-nutritive infusions. It has been suggested that such conditioned preferences are based on a learned shift in the hedonic evaluation of the nutrient-paired cue flavor. The present study tested this hypothesis using the taste reactivity (TR) test to infer nutrient-conditioned changes in flavor palatability. Rats were trained in a series of 20 hr/day and then 30 min/day sessions, during which intake of a flavored saccharin solution (the CS+) was paired with IG infusion of 16% glucose, and a differently flavored saccharin solution (the CS-) was paired with IG water infusion. Rats exhibited a strong preference for the CS+ flavor over the CS- flavor in two-bottle tests. When tested with brief intraoral infusions of the CS flavors, rats exhibited significantly more ingestive TR responses to the CS+ flavor than to the CS- flavor, which indicates an enhanced hedonic evaluation. Subsequent 30 min/day choice tests revealed that the CS- flavor was equally preferred to 3% fructose, whereas the CS+ flavor was equally preferred to 16% fructose. In addition, the TR responses to 3% and 16% fructose paralleled the TR responses to CS- and CS+, respectively. These findings suggest that pairing a flavored saccharin solution with IG glucose conditions an increase in palatability comparable to that produced by increasing the concentration of a fructose solution. (Supported by NIDCD DC00444 and NIDDK DK-31135)

Conditioned flavor preferences as a function of sugar concentration. A. SCLAFANI. Department of Psychology, Brooklyn College of CUNY, Brooklyn, NY 11210 USA.

In confirmation of prior work, rats given 1-bottle training (30 ml/day) with Kool-Aid flavored 5% and 30% sucrose solutions (CS+5, CS+30) strongly preferred the CS+5 when both flavors were presented in intermediate (17.5%) sucrose solutions. The CS+5 preference has been attributed to a conditioned satiety response to the CS+30 flavor, but a bout pattern analysis did not support this view. To determine if sweetness differences between training and test solutions contributed to the CS+5 preference, new rats were trained and tested with isosweet flavored 10% sucrose solutions. One flavor (CS+5) was paired with matched IG water infusions (= net 5% solution) and another flavor (CS+30) was paired with matched IG infusions of 50% sucrose (= net 30% solution) during 1-bottle training. In 2-bottle tests the rats showed no preference for the CS+5 or CS+30 with both paired with IG infusions of 25% sucrose (= net 17.5% solution). Following additional training, the rats significantly preferred the CS+30 to the CS+5 (both paired with IG 25% sucrose in test). These data indicate that sweetness differences can affect results obtained when sugars are consumed orally. The delayed CS+30 preference with IG training may be due to the taste of the 10% sucrose CSs overshadowing IG reinforcement and/or the satiating effect of the 50% sugar infusion limiting its reinforcing potency. (Supported by NIH DK31135 and MH00983)

Postingestive nutrient conditioning of flavor preferences does not require food deprivation. K. ACKROFF, A. SCLAFANI. Department of Psychology, Brooklyn College of CUNY, Brooklyn, NY 11210 USA.

The role of deprivation state in flavor preference conditioning by nutrients was investigated. Rats fitted with intragastric (IG) catheters were adapted to drink 0.2% saccharin under both food restricted (FR) and food ad libitum (AL) conditions. Two groups were then formed and trained either FR or AL to drink 7 ml of flavored saccharin solutions (CS+, CS-) paired with 7-ml IG infusions of 16% maltodextrin or water (30 min/day sessions). In subsequent two-bottle tests (CS+ vs. CS-), the FR group drank more than the AL group, but both groups showed similar preferences for the CS+ flavor (81-85%). Switching the groups' deprivation states shifted total intakes but did not alter their CS+ preferences. Both groups continued to prefer the CS+ flavor following IG maltodextrin preloads that reduced their test intakes. In a second experiment, the same rats were trained while water-restricted (WR) or food-restricted (FR) with new CS+ and CS- flavors paired with IG maltodextrin and water infusions. Both groups developed a CS+ preference but the preference was stronger in FR than WR rats (82% vs. 62%). The FR and WR data show that flavor conditioning is sensitive to hydration state, but the FR and AL data indicate that neither its acquisition nor expression require repletion of an energy deficit state. Thus, just as the "taste" of carbohydrate is reinforcing to nondeprived rats, so are its postingestive effects. (Supported by NIH DK31135 and MH00983)

Carbohydrate-conditioned preference for the flavor of ethanol. K. ACKROFF, A. SCLAFANI. Department of Psychology, Brooklyn College of CUNY, Brooklyn, NY 11210 USA.

The unpalatable flavor of ethanol is thought to limit its consumption by rats; naive animals typically accept moderate concentrations but reject ethanol at higher concentrations. We determined if ethanol flavor aversion, like bitter or sour taste aversion, can be reversed by intragastric (IG) carbohydrate conditioning. 5% ethanol (E) and a similarly acceptable flavor (0.05% citric acid + 0.5% maltodextrin, CM) were offered to ad lib fed rats on alternate days. For control rats postingestive effects were equated: when they drank one solution they were infused IG with the other. Conditioned rats were also infused with 5% E when they drank CM, but when they drank 5% E they were infused with CM + 16% maltodextrin. In choice tests, only the conditioned rats preferred ethanol to CM (91%, vs. 56% for controls); both groups preferred 5% E to water (82-90%). When the ethanol concentration was increased to 10%, conditioned rats still preferred it to water (83%) but controls did not. The conditioned rats continued to prefer 10% E to water when the IG maltodextrin concentration was gradually reduced to 0.5%. Oral ethanol concentration was then gradually increased to 25%. Ethanol (vs. water) preference declined from 48% to 30% in the control rats, and from 84% to 50% in the experimental rats. Thus, the initial aversive response to ethanol flavor can be reversed or reduced by postingestive nutritive conditioning. Such conditioning may combine with the pharmacological effects of ethanol to produce the acquired appetite for the flavor of alcoholic beverages. (Supported by NIAAA AA11549)

Conditioned taste aversion and c-Fos expression in cholestatic rats. E. NA, D.A. FITTS. University of Washington, Seattle, WA USA.

Past studies have demonstrated that the symptoms accompanying a ligation of the bile duct (BDL), a model of cholestasis and cirrhosis, may produce a conditioned taste aversion (CTA) in rats. However, these experiments used only fluid-intake tests. The present experiment uses a taste reactivity test to determine if a CTA is acquired. Rats fitted with intraoral catheters were administered 3 ml of a 0.15% saccharin solution for 10 minutes. The rats in the paired condition were then subjected to a sham surgery or a ligation surgery. Rats in the non-paired condition were subjected to surgeries 48 hours after their first infusion. Daily intakes of 0.06% saccharin and water were recorded until a second infusion of 0.15% saccharin was administered 7-9 days post surgery. All BDL animals avoided saccharin in the preference test, but the paired BDL rats showed an intense aversion and were the only group actively to reject the stimulus during the second infusion of saccharin. Eight control rats received either a BDL or sham surgery but were not fitted with an intraoral cannula or exposed to saccharin. Analysis of the area postrema (AP) and the nucleus of the solitary tract (NTS) revealed that all three BDL groups had significantly more c-Fos-like immunoreactivity in both nuclei, but this was not increased by the presentation of a conditioned stimulus. The activity in these nuclei may reflect the illness associated with the unconditioned stimulus.

Evidence for extinction of a conditioned flavor preference. N.L. TARNER, J. FRIEMAN, R. MEHIEL, E.M. DOUGLASS. Kansas State University, Manhattan, Kansas.

Eighteen rats were conditioned to prefer one flavor over another by pairing the one flavor with sucrose and the other flavor with saccharin. After 10 conditioning trials, the rats were given a two-bottle preference test. Results showed that the rats consumed more of the sucrose-paired flavor than the saccharin-paired flavor. Following conditioning the rats were matched on the amount of sucrose consumed and placed into one of four groups during extinction. The two experimental groups were Group SUC and Group SUC/SACC. Group SUC received their sucrose-paired flavor and Group SUC/SACC received both the sucrose- and saccharin-paired flavor, but on alternating days. After ten extinction trials, the rats were given a two-bottle preference test. Results showed that the rats in the two experimental groups decreased their consumption of the sucrose-paired flavor, thus demonstrating extinction. The rats in the two control groups maintained their preference for the sucrose-paired flavor. The results found in the current experiment demonstrate for the first time extinction of a conditioned flavor preference.

Conditioned Food Aversion in the Terrestrial Hermit Crab (*Coenobita clypeatus*). L.J. NOLAN, B.A. SOUSA, A.M. SIEGEL. Department of Psychology, Wagner College, Staten Island, NY 10301.

Terrestrial hermit crabs are omnivorous scavengers that must make choices among a variety of available foods in order to obtain nutrients and avoid toxins. Like vertebrate omnivores, it would be advantageous for them to be able to modify their selection based on experience. This study was conducted to determine whether these animals can learn food aversions. 11 hermit crabs were trained to eat apple pieces in a glass fingerbowl. Five were then randomly assigned to consume ground beef and then injected with LiCl (in cheliped joint with 0.70 M solution) to determine whether a conditioned food aversion (CFA) to beef would be formed. The other 6 (controls) were injected with equivolume distilled water after consumption of beef. On each day following the injection, all animals were offered beef and apple and the number of animals that consumed each food was counted each day until all CFA animals consumed beef on 3 consecutive days. The results of a chi square analysis indicated the formation of a CFA that was specific to beef in the CFA animals. Extinction of the CFA was also observed. It was concluded that terrestrial hermit crabs are capable of CFA learning and that it may play a role in their food selection. Invertebrate models of food-related learning may be useful in studies of the neuroscience of eating.

Acute Effects of Corticosterone on LiCl-Induced Rapid Gustatory Conditioning in Rats: Examining Patterns of Licking Behavior. Acute Effects of Corticosterone on LiCl-Induced Rapid Gustatory Conditioning in Rats: Examining Patterns of Licking Behavior. W.D.T. KENT, S.K. CROSS-MELLOR, M. KAVALIERS, K.-P. OSSENKOPP. Neuroscience Program and Department of Psychology, University of Western Ontario, London, Ontario, CANADA, N6A 5C2. Acute treatment with corticosterone (Cort) has been shown to have rapid effects on learning and memory. We observed (NeuroReport 2000, 11:3903-08) that in a taste reactivity test (forced exposure procedure) rats treated with both LiCl and Cort showed enhanced aversive responding and reduced ingestive responding. These results indicated Cort enhanced learning when rats were subjected to toxin-induced rapid gustatory conditioning. In the present study we used a lickometer to examine the effects of acute Cort administration on changes in patterns of voluntary licking of a sucrose solution when treated with LiCl or NaCl. Four groups of male rats ($n = 8/\text{group}$) were adjusted to a water deprivation schedule and trained to drink water from a spout in a Plexiglas test box. On each of 3 conditioning days rats were given intraperitoneal (ip) injections of either LiCl (0.75 mEq) or saline control (NaCl; 0.9%) and 10 min later received a second ip injection of either Cort (5 mg/kg) or cyclodextrin vehicle. Rats were then placed in the test boxes for 20 min and allowed to lick a sucrose solution (0.3 M). Licking behavior was analyzed using Quick Lick software. LiCl significantly decreased total number of licks as well as meal duration and this effect was significantly enhanced with Cort. Cort also increased the number of lick clusters in the NaCl group and decreased the number of clusters in the LiCl group relative to the NaCl-vehicle group. These results provide convincing evidence of within meal toxin-induced conditioning that is enhanced by acute Cort treatment. (Supported by grants from NSERC)

Nutritional intake differences in food neophobics and neophilics. B. RAUDENBUSH, N. CORLEY, N.R. FLOWER, A. KOZLOWSKI, B. MEYER. Wheeling Jesuit University, Department of Psychology, Wheeling, WV, USA.

Past research addressing differences between food neophobics (those individuals reluctant to try new foods) and food neophilics (those individuals particularly willing to try new foods) indicates that neophobic individuals have significantly lower body weights. The present study explored the dietary basis of this weight difference. One hundred people were asked to complete a food diary, listing the type and amount of all items consumed over a randomly assigned 3 day period. Basic components, vitamins, and minerals were calculated for each participant. Groups were then formed by a data tri-split based upon scores on the Food Neophobia Scale (FNS), thus forming groups of neophobics and neophilics, and an average group. Neophobics tended to consume fewer total calories, and showed decreased fat and cholesterol intake than either neophilics or the average group. This decrease in fat consumption among neophobics may account for their decreased weight. However, few to no other differences were found among the groups on 45 other measures of nutritional adequacy. These results support the notion that there are relatively no detrimental nutritional effects related to food neophobia.

Cephalic phase salivary response differences characterize level of food neophobia. B. RAUDENBUSH, N. CORLEY, N.R. FLOWER, A. KOZLOWSKI, B. MEYER. Wheeling Jesuit University, Department of Psychology, Wheeling, WV, USA. Cephalic phase responses are rapid physiological processes, stimulated by the sensory properties of foods, that are believed to prime the body to better absorb and utilize ingested nutrients. The pre-ingestive flow of saliva following sensory stimulation provides a quantitative index of responsiveness to foods. Past research addressing differences between food neophobics (those individuals reluctant to try new foods) and food neophilics (those individuals particularly willing to try new foods) suggests differences may exist in cephalic phase salivary responses between these groups. The saliva flow of 90 participants was measured for a 3-minute period following exposure to either a lemon, funistrada (a fictitious food) or a non-food control stimulus. Participants were divided into 3 groups (data tri-split) based on level of food neophobia, as measured by the Food Neophobia Scale (FNS). Regardless of the stimulus, neophobics showed decreased salivation and neophilics showed increased salivation in relation to the average group. Only the average group showed salivation differences based on stimulus type, with low salivation in the control condition, moderate stimulation in the funistrada condition, and high salivation in the lemon condition.

Operant conditioning utilizing mice in a foraging paradigm. C.H. VAUGHAN, N.E. ROWLAND. Department of Psychology, University of Florida, Gainesville, FL 32611 The main purpose of this study is to discover parameters for studying meal patterning in mouse foraging behavior using an operant paradigm. Four female C57BL/6J mice were housed in operant chambers with a 12-h light: 12-h dark cycle. Food pellets (20 mg Noyes pellets) were available at all times but were received only through consecutive pressing of a procurement then a consumatory lever. The mice received visual light cues signaling the appropriate lever. The procurement lever delivers the food rewards. Each pellet's delivery time was recorded. This measurement allowed a number of variables to be calculated including meal size and intermeal intervals. A series of fixed ratios (FR) were introduced to the mice and the consequent meal characteristics were noted. As the fixed ratios progressed meal patterning became more evident for each mouse. Total daily pellet intake decreased as fixed ratios increased. Average total intake consumed per day for FR-10 was 3.49 ± 0.41 g and for FR-120 the average was 2.75 ± 0.19 g. The average number of meals per day at FR-10 and FR-120 was 8.17 ± 1.76 and 4.55 ± 1.93 respectively. Overall, bout size, meal size, meal frequency and intermeal intervals also showed a moderate decrease as fixed ratios increased. The intermeal relationship shows that the higher the cost, the more time is spent between lever pressing for meals. Meals occurred mostly in the dark cycle. This study hopes to set standards for other comparison studies using genetic and physiological manipulations.

Training in Acquisition of Odor-Cued Fasting-Anticipatory Satiety in Rats. Y. YIIN, L. THIBAUT. School of Dietetics and Human Nutrition. Macdonald Campus of McGill University, Montreal, Canada. Animals can learn to associate food's orosensory characteristics with its postingestive effects and therefore use orosensory cues to adjust meal size to meet energy requirement. Two studies were conducted to test if rats could acquire anticipatory satiety through odor-fasting duration conditioning. In the first study, adult male Sprague-Dawley rats were given a test meal (grounded chow) odorized with peanut butter flavor for 1 hour prior to a 10-hour fast and with strawberry flavor prior to a 3-hour fast. The rats went through a pseudo-random sequence of ten duplicates of each odor-fast pairing, followed by an odor preference test. Result suggested that the rats did not acquire anticipatory satiety possibly due to unlearned preference for strawberry odor. Thereafter, a pre-test was conducted to test odor preference among eight odors presented in a casein-based meal instead of strongly smelling chow; vanilla and chicken odors were similarly preferred by rats. A second study was undertaken in which thirteen male and eleven female adult Sprague-Dawley rats were conditioned in a similar fashion to the first study except that the casein-based test meals were odorized by either vanilla or chicken, the length of test meal was expanded to 1.5 hours, and that of the short and long fasts to 4 and 12 hours respectively. In addition, odor-fasting duration pairing were counter-balanced. We found that female rats learned to have greater meal intake prior to a long fast than prior to a short fast but not male rats. These results suggest that postprandial fasting duration can be cued by odor, and gender differences may influence this conditioning. Key words: rats, odors, anticipatory satiety Acknowledgement: this research was supported by a grant from the National Science and Engineering Research Council of Canada (NSERC) and by a NSERC fellowship (Y.Yiin)

The effects of clozapine, olanzapine and haloperidol on the microstructure of ingestive behaviour in the rat. A.W. HARTFIELD, N.A. MOORE, P.G. CLIFTON. Experimental Psychology, University of Sussex, Falmer, Brighton, BN1 9QG. UK.

Antipsychotic drugs, particularly the newer atypical compounds, may induce rapid weight-gain in a clinical setting, however, thus far there are few animal models producing reliable hyperphagia correlating with the human weight-gain liability of these drugs. Here we report that olanzapine and clozapine, but not haloperidol, significantly increased intake of a palatable, high-calorie fat emulsion in rats. Microstructural analysis revealed that both olanzapine and clozapine significantly elevated the number of clusters of licking whilst maintaining the mean cluster size suggesting a delayed postingestive satiety signal with unchanged palatability. Conversely, haloperidol exerted a hypophagic response as a consequence of stable cluster number and reduced mean cluster size analogous to unaltered postingestive negative feedback signals and decreased palatability. The results are discussed in relation to the development of a possible animal model of antipsychotic-induced weight-gain and the mechanisms that might be responsible.

Influence of nibblings and strains on body weight gain and food intake of rats, Wistar vs Dark Agouti. C. LARUE-ACHAGIOTIS, M.L. NAVIS, A. BENSALD, M. LACROIX, G. FROMENTIN, N. GAUSSERES*, D. TOME. INA P-G, INRA Paris Cedex 05. *Danone Vitapole, France. This study aims to investigate the effects of 2 extra-meals or 2 nibblings on body weight (BW) and food intake on 2 strains of rats: Wistar, prone to obesity and Dark Agouti (DA): which are more resistant to obesity. Rats (8 weeks) were accustomed to a 3 meal feeding schedule during one week (a breakfast=48.5kJ, and a one hour ad lib lunch and dinner). Thereafter, 3 groups of rats were determined for each strain : the first remained on this pattern (3R), the 2nd received 2 fixed extra-meals every day at the same time (17.1kJ = 3R+2F) and a 3rd had, two, at random, extra-nibbling (17.1kJ, 3R+2A). Results : During the 3 weeks, in Wistar, the 3R+2F and 3R+2A groups gained more BW than the 3R, this difference was clear during the first week, thereafter there was no significant difference. In DA no difference were observed in total BW gain, however, during the 1st week of treatment, 3R+2A had a higher BW gain. Wistar rats ingested more energy in 3R+2F and 3R+2A groups, this was due mainly to the highest intake of the 1st and 3rd weeks: those rats did not decrease their lunch ingestion, whatever the week, but they reduced it for dinner during the 1st and 2nd weeks. In DA, there was neither difference in the total intake, nor in per week. They reduced their lunch and dinner intakes, the extra-ingestions are compensated in DA whatever the time of intake, while Wistar did not compensate at all during the 1st week and slightly thereafter. The 3R+2A group seems to be the most affected.

Microinfusion of D-CPP into the brainstem reticular formation suppresses ingestion and rejection in the awake rat. Z. CHEN, S.P. TRAVERS, J.B. TRAVERS. Ohio State University, Columbus OH.

The brainstem reticular formation (RF) plays an essential role in orchestrating the oromotor components of ingestion and rejection. Evidence from electrical brain stimulation in acute preparations suggests that different classes of glutamate receptors in the medullary RF play distinct roles in jaw movement during rhythmic oral activity (Inoue et al. 1994). To further examine the role of these receptors in more naturally occurring ingestive behavior, we infused an NMDA receptor antagonist into the lateral medullary RF and measured its effects on jaw opening and tongue protrusion during ingestion (licking) and rejection (gaping) in awake, freely-moving rats. Compared to saline controls, bilateral infusions of D-CPP (100 nl: 0.198 - 1.98 mmol) reduced the amplitude of muscle contractions as well as the rate of rhythmic activity from the geniohyoid (tongue protruder) and anterior digastric (jaw opener) muscles during licking and gaping induced by intraoral sucrose or QHCl stimulation. Responses returned to baseline within approximately 3 hr. The amplitude and rate of appetitive licking from a bottle containing 0.3 M sucrose was similarly reduced by D-CPP infusions. We conclude that NMDA receptors on neurons in the lateral medullary RF form part of the excitatory drive to oromotor neurons active during consummatory responses, regardless of whether these responses terminate appetitive behavior or are induced by direct oral stimulation. Supported by DC00417. .

Subjective recognition of a narrow glycemia range after training. M. CIAMPOLINI, M. VAN WEEREN*, B. DE PONT*, W. DE HAAN*, L. BORSELLI. Department of Pediatrics, University of Florence, Italy & *AMC, Amsterdam, The Netherlands. Adults may be trained to presume their glycemia at the threshold emergence of depletion feelings (bearable hunger). 44 healthy adults, 19 male and 25 female between 18 and 50 years trained themselves by measuring glycemia at first perception of depletion feelings. Over time, the repetition of the same feeling with the same blood glucose level indicated the completion of the training. The recognition of this level served to start the meal. Two months after the training period, a laboratory investigation was made after an overnight fast. The subjects declared the presumed glycemia, which was then measured by the hexokinase method in the hospital laboratory. A presumption that was higher (or lower) than the measurement by a 20% or more was assumed to be a deception and was separately listed. The coefficient of variation was calculated in the remaining glycemia presumptions (prevalence trend). RESULTS: Four subjects presumed values that were 20% higher than the measured value. Three of the 4 subjects declared Yes hunger, the measurement was between 3.11 and 3.78mmol/l, and the deception inconsequential. The fourth subject presumed 5.0mmol/l glycemia with No hunger, and measured 3.77mmol/l. The presumed glycemia was 4.41 ± 0.52 and the measured one was 4.49 ± 0.66 in the further 40 laboratory observations. The SD of the differences showed the approximation in the presumption that was 0.33mmol/l (7.5% of the measured value) in the 40 laboratory observations. Healthy adults are able to subjectively recognize the narrow glycemia range between 4.7mmol/l and 3.3mmol/l by feelings of depletion after adequate training.

Recognition of depletion manifestations (bearable hunger) in infants by trained caregivers and lower fecal energy loss. M. CIAMPOLINI, V. GIANNELLINI, N. BUTTE. Department of Pediatrics and Pharmacological Sciences, University of Florence, Italy and Houston TX, USA

Malnutrition recovered after training caregivers to recognize the depletion manifestations (food request) in a controlled investigation on 9 infants. The fecal energy emission may decrease in the infants of trained caregivers. 34 infants with chronic non-specific diarrhea were randomly assigned to either an intervention or a control group in the second year of life, and 26 were followed for 5 months. The controls consisted of 7 males and 5 females, the intervention group of 9 males and 5 females. The caregivers trained themselves to subjectively predict the glycemia level between 4.7mmol/l and 3.3mmol/l in their infants by the manifestations of food request (depletion). The recognition of this level served to start the meal. Three or more months after complete diarrhea remission, the caregivers made a 7-day home food-diary and a 3-day weighed collection of feces. A sample of mixed material was dried and analyzed by the bomb calorimeter. RESULTS: The energy content in a gram of dry stool was 5.24 ± 0.5 in the control group and 4.72 ± 0.45 kcal in the intervention group ($P < 0.02$). The fiber intake was 9.1 ± 5.1 g (SD) and 19.7 ± 7.2 g ($P < 0.001$), the wet stool weight 67.2 ± 26.9 g and 111.0 ± 51.3 g ($P < 0.02$), the dry stool weight 19.1 ± 8.6 g and 20.5 ± 9.7 g, the stool energy loss 101.6 ± 50.0 and 97.9 ± 49.6 kcal per day in the control and intervention groups, respectively. The intervention saved about 40 kcal a day from fiber as compared to 'ad libitum' eating, and this amount became available for metabolism probably by colonic fermentation.

Employing an evaluation framework to increase the effectiveness of a nutrition and physical activity intervention in a Latino community in Los Angeles. J.C. GUSTAVSON, J. ASARIAN-ANDERSON, N. TAYAG, C. YOUNG, A. GUSTAVSON. County of Los Angeles, Department of Health Services, Chronic Disease Prevention, and Health Promotion—Nutrition Program, Los Angeles, CA, USA. A community and public health partnership combined Promotoras (lay health educators), professional health and nutrition staff, and a research analyst to deliver a nutrition and physical activity curriculum and document program effectiveness, using process, outcome, and impact evaluation methods. Ten experienced Promotoras were trained to deliver cultural and language sensitive nutrition and physical activity classes to families who are isolated by language, economic and cultural barriers in their northeast Los Angeles County community. The classes were designed to help decrease diet related chronic disease. By using both qualitative and quantitative evaluation methods at various stages of program planning and implementation, the program could be modified to be more effective in changing dietary behavior in the target community. Qualitative methods used at various stages of the program, identified cultural beliefs and attitudes about food. This information helped modify the classes to be more effective at reaching our goal of increasing fruit and vegetable consumption by one serving daily in this community. Forty-four randomly chosen class participants completed a series of eating habit surveys over 3 months. Fruit and vegetable consumption was evaluated using a t-test for repeated measures. Mean number of fruit and vegetable servings consumed increased significantly from 3.55 prior to education, to 4.53 after training ($t(43) = 2.78, p = .007$). Without knowledge of the food beliefs of this community obtained from qualitative evaluation methods, this program may have not been as successful.

The stigmatization of obese peers among 5th- and 6th- grade children: comparison with a 1961 investigation. J.D. LATNER, A.J. STUNKARD. Department of Psychology, Rutgers University, 152 Frelinghuysen Road, Piscataway, NJ 08854, U.S.A. A greater acceptance of diversity in recent years has been accompanied by an increased emphasis on thinness and disapproval of excess weight. The present study aimed to identify which of these two opposing trends has prevailed among children judging their obese peers – acceptance of these peers despite their different physical appearance or bias against them. In a replication of a 1961 study, 458 5th- and 6th-grade children ranked 6 drawings of same-sex children with obesity, various disabilities, or no disability, according to how well they liked each child. Children in the present study liked the obese child least, and even less than in 1961 ($t(1053) = 5.12, p < 0.001$). The thin, non-disabled child was ranked highest, and even more highly than in 1961 ($t(1053) = 5.07, p < 0.001$). Compared to 1961, the greater acceptance of children with facial abnormalities ($t(1053) = 9.98, p < 0.001$) may reflect an increased acceptance of different facial appearances (which often accompany ethnic differences). The greater dislike of a child in a wheelchair ($t(1053) = 6.51, p < 0.001$) may indicate higher current expectations for physical fitness and a rejection of children who do not meet these standards. Girls liked the obese child less ($t(413) = 3.08, p < 0.001$), and the child in a wheelchair more ($t(413) = 3.41, p < 0.001$), than boys did. These findings suggest that stigmatization of obesity has increased over the last 40 years despite progress in reducing bias against other disabilities.

Exploring the changeroom effect: females' perceptions of bodily size and attractiveness. Y. MARTINS, P. PLINER. Department of Psychology, University of Toronto at Mississauga, 3359 Mississauga Road North, Mississauga, Ontario, Canada, L5L 1C6. This study was conducted to explore females' perceptions of the body size and attractiveness of both themselves and others. Upon arrival at the lab, females (with BMI's between 19 and 26) changed into form-fitting clothing provided by the experimenters. Next, headless instant photographs were taken of their bodies. The participants were then given the photo of themselves and the photo of the previous participant, one at a time, in a counterbalanced order and rated each, one in the "self" role and one in the "other" role. For each photograph, participants were asked to identify the body silhouette most closely resembling the body in the photograph and to rate the attractiveness of each body, as it currently looks, the way it would look after losing 10 pounds, and the way it would look after gaining 10 pounds. Paired t-tests were conducted to assess differences in ratings between "self" photos and "other" photos for women high and low in appearance self-esteem. Results demonstrated that women low in appearance self-esteem rated the "other" body, after it had gained 10lbs., as smaller in size and more attractive than their own body, after it had gained 10lbs. No differences between ratings of "self" and "other" photos were found for women high in appearance self-esteem. Implications of these findings are discussed in relation to previous research in the areas of body image and effects of body size on individuals' perceptions of others.

Do children regulate energy intake? G. MRDJENOVIC, D.A. LEVITSKY. Division of Nutritional Sciences, Cornell University, Ithaca, NY. Our goal was to determine how well preschool children regulate food intake under natural conditions. Twenty-four hour food intakes of 16 preschool children 4 to 6 years of age were measured for seven consecutive days. Children's intake at meals was significantly negatively correlated with the amount and energy intake at the previous meal ($r = -0.13, p = 0.02$). However, the amount served at a given meal was also negatively correlated with the amount of the food served and consumed at the previous meal ($r = -0.16, p = 0.001$). Multiple regression analysis indicated that the amount of food served was the major determinant of food intake at each meal (40-60% variance explained). What was consumed at a meal was not influenced by any dietary variable of the previous meal. Amount of food consumed and nutrient content of the diet served were the major determinants of children's energy intake at a meal. Higher was the dietary fat content, higher was the energy intake. Children appeared to adjust the amount food they consumed to the energy density of a meal. However, after adjusting for the amount served this apparent regulation disappeared: the amount of food consumed remained constant across all density levels ($p > 0.05$). The present results indicate that under natural conditions, the amount and composition of the foods served to children are the best predictors of how much they consume.

Caloric compensation in college students during lunchtime meals. J. WILSON, H. GWINN, S. FERNANDO, S. JAMES. Wittenberg University, Springfield, OH, U.S.A. Preschool children consume significantly more energy at a meal when offered chocolate milk with the meal than they do when served plain milk (Wilson, 1991, 1994, 2000). They drink large quantities of the sweetened beverage without decreasing their intake of other food items available at the lunchtime meal. To examine the eating behavior of men and women offered chocolate-flavored or plain milk at lunch, food consumption by 46 undergraduate college students was measured. One menu (consisting of pasta with vegetarian sauce, cheese, green beans, whole wheat bread, brownie, and applesauce) was served two times during a two-week period, being presented once with chocolate-flavored milk and once with plain milk. The order of milk-beverage presentation was varied systematically. The type of milk served had a significant effect on the consumption of other food items at that meal. Subjects consumed significantly more chocolate milk (Mean = 234.8 kcal) than plain milk (Mean = 158.5 kcal) during meals, $F(1,44) = 14.59, p < .001$. However, the total energy intake was nearly identical for meals served with chocolate milk (Mean = 775.2 kcal) and those served with plain milk (Mean = 773.7 kcal), $F(1,44) = 0.00, n.s.$ No significant differences were detected between men and women in any of the statistical analyses conducted. These results suggest that adults are able to reduce the intake of other food items at a meal to compensate for the increased energy intake that results from increased consumption of chocolate-flavored milk.

Hydrochlorothiazide induced NaHCO_3 intake by rats in a simulated foraging paradigm. C.L. COLBERT, N.E. ROWLAND. Department of Psychology, University of Florida, Gainesville, Florida 32611.

Collier and colleagues have shown that if rats are required to press a lever to procure food (or water) then the number of ingestive bouts per day declines sharply as procurement fixed ratio (PFR) increases. The objective of the present study is to examine whether the consumption of 0.3 M NaHCO_3 solution by male long evans rats, that are given hydrochlorothiazide (HCZ), is affected when a PFR is imposed. Four rats were housed in 2-lever operant chambers; powdered sodium deficient chow was available ad libitum from a jar remote from the levers. Operation of a predetermined PFR (1, 80 or 300 in each phase of the study) caused either distilled water or 0.3 M NaHCO_3 to become available via a drinking spout that moved into a slot above the respective lever. Licks were recorded by contact closure. In one phase, water and NaHCO_3 were available on the same PFR, while in a second phase, NaHCO_3 PFR increased relative to water. During baseline, the rats showed a decrease in number of daily water drinking bouts (from 10-20/day at PFR1 to 3-4/day at PFR80), and a compensatory increase in bout size. Furthermore, NaHCO_3 bouts were reduced from 10-14/day at PFR1 to 0/day at PFR80. Following these studies, rats were given the diuretic thiazide, HCZ in their food (HCZ, .6g/kg food). Salt appetite was manifest by increased intake of 0.3 M NaHCO_3 with HCZ treatment. Results were compared with the free access paradigm.

Sodium appetite in DBA/2J mice. S. EYLAM, M. GARCEA, A.C. SPECTOR. Department of Psychology, University of Florida, Gainesville, FL

The amiloride-sensitive sodium taste transduction pathway has been shown to be necessary and sufficient for Na^+ -taste recognition in rats. Nerve recordings show that this pathway appears missing in the taste receptors of the anterior tongue in DBA/2J (D2) but not in C57BL/6J (B6) mice. Thus, we set out to investigate sodium appetite in D2 mice using a brief-access taste test (5-sec trials) and a 24-h intake test (n=5-6/group). To our knowledge, this is the first study to investigate sodium appetite in this strain. Although these mice demonstrated motivation to lick when water-deprived in the brief-access paradigm, there was tremendous variability and limited licking of the various sodium and non-sodium salts (0.1 and 0.5 M) when mice were Na^+ -deficient, in contrast to the significant increases in sodium licking seen in rats under similar conditions. The few Na^+ -deficient mice that did lick, exhibited enhanced responsiveness specifically to the sodium solutions when the salts were dissolved in water but not when they were dissolved in amiloride (100 μM), similar to rats. Sodium output and urine volume measurements indicated that the natriuretic treatment (through Na^+ -deficient chow and furosemide injections) was successful. As expected, there was a significant elevation in NaCl intake in the 2-bottle test of the Na^+ -deficient but not of the control mice, whether the solutions were adulterated with amiloride or not. We are currently in the process of repeating these procedures with B6 mice in order to identify possible strain differences. Supported by NIDCD grant R01-DC-04574.

Neuronal activity in rat insular cortex after sodium depletion: manner of sodium delivery. C.V. PASTUSKOVAS, A.K. JOHNSON, R.L. THUNHORST. Department of Psychology and Pharmacology and the Cardiovascular Center, University of Iowa, Iowa City, IA 52242, USA.

Insular cortex (IC) receives inputs from multiple sensory systems that monitor body electrolyte and fluid balance, including taste, and is postulated to process cognitive information about salt taste. This work analyzed metabolic activity of IC neurons when sodium is presented in different ways and in different volumes after sodium depletion. Male Sprague Dawley rats were depleted of sodium by injections of the diuretic, furosemide (10 mg/kg, bw) followed 10 min later by injections of the angiotensin-converting enzyme inhibitor, captopril (5 mg/kg, bw). Rats had either no access, ad libitum access, or restricted (0.5 ml) access to water and 0.3 M NaCl, or 0.5 ml saline introduced into the mouth by intraoral infusion. The results showed that the pattern of increased c-fos activation found in the subregions of IC (i.e., granular, dysgranular, and agranular areas) following sodium depletion depended both on the volume of sodium ingested (i.e., about 4.0 ml ad libitum vs 0.5 ml restricted) and the manner of delivery (i.e., freely ingested vs infused). The results suggest that factors related to volume of sodium ingested, duration of sodium taste in the mouth, or the behavioral act of drinking sodium are responsible for the differential increases in c-fos activation in subregions of IC following induction of salt appetite after sodium depletion.

Behavioral sensitization to sodium (Na) depletion is correlated with increased dendritic length and spines in nucleus accumbens neurons. M.F. ROITMAN, G. ANDERSON, M.T. KOH, T.A. JONES, I.L. BERNSTEIN. Princeton University, Princeton NJ 08544 USA.

An acute challenge of Na depletion sensitizes behavior directed at Na - there is enhanced Na intake following subsequent depletion episodes. Physiological changes underlying this sensitization have yet to be determined. The delivery of drugs of abuse (i.e. cocaine, amphetamine) sensitizes behavior as well. Recently, drug sensitization has been correlated with persistent alterations in the dendritic morphology of nucleus accumbens (NAc) neurons. We investigated whether similar neuronal alterations would be detected following multiple Na depletions in rats. The morphology of NAc neurons were analyzed in Golgi stained material from rats sacrificed after their third Na depletion. These rats displayed enhanced Na intake following their second Na depletion (10.33 \pm 0.98ml) relative to their first (7.8 \pm 1.02ml; $p < .05$). Total dendritic length of NAc neurons from these Na depleted rats was significantly greater compared to that observed in rats with no depletion history (depleted: 789 \pm 27 mm; control: 584 \pm 76 mm, $p < .05$). This difference was primarily due to significantly longer higher order dendritic branches (third order and higher). In addition, third order terminal dendrites of multiple depleted rats had significantly more spines than those of non-depleted controls (depleted: 99.37 \pm 8.17; control: 64.75 \pm 11.09; $p < .05$). These results provide striking evidence that acute Na depletion is associated with dendritic growth and the development of spines in the NAc. The observed alterations are remarkably similar to those observed after drug sensitization and suggest that sensitization to a challenge, in general, is associated with morphological changes in the NAc.

Long-term food restriction and area postrema lesions: effect on heart rate in rats. K.S. CURTIS, E.G. KRAUSE, R.J. CONTRERAS. Department of Psychology and Program in Neuroscience, Florida State University, Tallahassee, FL USA 32306-1270

Heart rate (HR) is decreased after area postrema lesions (APX) in rats; however, hypophagia and weight loss also occur after APX and may contribute to the decreased HR. We evaluated the contribution of decreased food intake and body weight to decreased HR in rats with APX. Adult male Sprague-Dawley rats were implanted with telemetry devices to record mean arterial blood pressure (MAP) and HR. After 2wk recovery, baseline MAP and HR were recorded for 1wk. APX then were produced by vacuum aspiration. Rats with sham APX (APS) served as controls. Rats with APS were fed ad libitum (APS-a, n=4), or were food restricted (APS-r, n=5) to match their weights to those of rats with APX (n=8). MAP and HR were recorded for 6wk. Rats with APS-a gained weight during the 6wk, whereas rats with APX and with APS-r lost weight comparably. Weight decreased to 85% of preoperative weight by D21, then increased slowly to 95% of preoperative weight by 6wk. MAP did not change in any group during the 6wk. HR remained stable in rats with APS-a, but decreased both in rats with APX and with APS-r. HR decreased more rapidly in rats with APX (-31.9 beats/min by D5) than in rats with APS-r (-19 beats/min by D5), and remained consistently lower in rats with APX throughout the 6wk. Thus, the decreased HR in rats with APX is, in part, a consequence of long-term food restriction/weight loss; however, additional factors contribute to the decreased HR in rats with APX.

Immunotoxin lesion of norepinephrine (NE) and epinephrine (E) neurons innervating the medial hypothalamus elevates basal expression of and attenuates glucoprivation induced increases in agouti gene related-protein (AGRP) mRNA. G.S. FRALEY, T.T. DINH, S. Ritter. Programs in Neuroscience, Washington State University; Pullman, WA 99164-6520.

Norepinephrine and E innervation of medial hypothalamic structures is necessary for glucoprivation-induced feeding, glucocorticoid secretion and Fos expression in the paraventricular (PVH) and arcuate nuclei of the hypothalamus. In this experiment, we tested the hypothesis that the 2-deoxy-D-glucose (2DG)-induced increase in AGRP mRNA expression (reported recently by Sergeev et al., 2000) also requires NE/E neurons. NE and E neurons innervating the medial hypothalamus were lesioned using the toxin, saporin, targeted for selective entry into NE/E neurons by conjugation with a monoclonal antibody against dopamine beta hydroxylase. This toxin (DSAP), or unconjugated saporin (SAP) control solution, was microinjected into the PVH. Approximately 4 wks later, DSAP rats with confirmed 2DG-induced feeding deficits and SAP controls were injected with 2DG (250 mg/kg), or saline (0.9%, 1 ml/kg) and maintained for 2 hrs without food. Hypothalami were harvested and subjected to Northern blot analysis of AGRP mRNA. Blot analysis (expressed as Relative Density Units, or RDU) revealed that 2DG increased mRNA expression in SAP controls (2DG: 1.41 ± 0.21 RDU; saline: 1.01 ± 0.08 RDU, $p < .05$), but not in DSAP lesioned rats (2DG: 1.49 ± 0.20 ; saline: 1.41 ± 0.02). In addition, basal AGRP mRNA expression was significantly elevated in DSAP-lesioned rats compared to SAP controls ($p < .05$). These data suggest that basal AGRP gene expression is controlled by hindbrain NE/E neurons and that increased expression induced by glucoprivation requires these neurons. Furthermore, increased basal AGRP mRNA expression in DSAP-lesioned rats may be causally related to the mild obesifying effect we reported previously to be associated with this immunotargeted lesion.

Hypoglycemia-associated autonomic failure does not involve diminishing of hypothalamic norepinephrine release. M.G. DE VRIES, L.M. ARSENEAU, B.L.M. FRENKEL, J.L. BEVERLY. University of Illinois at Urbana-Champaign, IL 61801, USA

In diabetic humans hypoglycemia-associated autonomic failure often causes attenuated autonomic and hormonal responses to multiple episodes of hypoglycemia. This experiment is designed to investigate whether that impaired hormone release is mediated by reduced noradrenergic activity in the hypothalamus. Rats received daily saline injections for three consecutive days, followed by four days of insulin (0.5, 1, 1, 0.5 U/kg on respective days). Rats were fitted with a jugular vein catheter and a guide cannula to the ventromedial (VMH) or paraventricular (PVN) area of the hypothalamus. Blood samples were withdrawn to measure changes in blood glucose, catecholamines, glucagon, and corticosterone. Dialysate samples (flow rate = 1.5 mL/min) were collected to monitor extracellular norepinephrine in the hypothalamus. The maximum increase in plasma epinephrine during the first episode of hypoglycemia was 450 ± 195 pg/mL after 15 min, and was blunted to 265 ± 48 pg/mL ($p < 0.01$) during the fourth episode. The maximum responses of glucagon ($t = 15$ min, $p < 0.01$ compared to saline) and corticosterone ($t=30$ min, $p < 0.05$) were not significantly affected by repeated hypoglycemia. The bimodal increase of norepinephrine in the VMH ($t=5,35$ min; $P < 0.01$) and the trend towards an increase in the PVN during hypoglycemia were not diminished by repeated episodes. In Conclusion, blunted compensatory hormone responses to repeated hypoglycemia are not mediated by reduced norepinephrine release in the hypothalamus.

JCR:LA-cp rats show improved lipid profiles in response to diets containing chromium picolinate and biotin. J.R. KOMOROWSKI, J. DE LA HARPE, W.T. CEFALU*, X.H. ZHANG*, Z.Q. WANG*, D. GREENBERG. Nutrition 21, Inc. Purchase, NY 10577 and *University of Vermont College of Medicine, Burlington, VT 05405.

Obesity and insulin resistance are strongly associated with an increased risk of cardiovascular disease. JCR:LA-corpulent rats are obese, insulin-resistant and known to develop hyperlipidemia and atherosclerosis. There is evidence that supplementation with chromium picolinate reduces total cholesterol in blood and that biotin is involved in carbohydrate and lipid metabolism. In order to determine if there was a synergistic effect of these nutrients on blood lipids, we compared the effects of diets supplemented with chromium picolinate and biotin, separately or in combination, on blood lipids and body weights of JCR:LA-cp rats. Rats (n=4 to 6/group) were maintained on diets supplemented with chromium picolinate and/or biotin for 3 months. Blood lipids were measured at baseline and at 1, 2 and 3 months. Daily doses of chromium picolinate (8 mcg/kg or 80 mcg/kg) and/or biotin (30 mcg/kg or 300 mcg/kg) were used. All supplementation except the lowest dose of chromium picolinate alone, significantly raised HDL levels. The combination of the high dose of chromium picolinate and high dose biotin elevated HDL levels to a significantly greater extent than when these nutrients were given separately [$F(9,141)=5.8$; $p < .0001$]. No effects were seen on the body weights of these rats. We conclude that chromium picolinate and biotin can act synergistically to improve blood lipid profiles in an animal model of insulin resistant obesity. These results suggest that this nutrient combination could be beneficial for human populations that are insulin resistant and subject to cardiovascular disease. Supported by: Nutrition 21, Inc.

Effect of repetitive etomoxir administration on 24h substrate oxidation and satiety in humans. W. LANGHANS¹, V.B. HINDERLING¹, P. SCHRAUWEN², M.S. WESTERTERPLANTENGA². ¹Institute of Animal Sciences, Swiss Federal Institute of Technology, Zurich, Switzerland, ²Department of Human Biology, Nutrition and Toxicology Research Institute Maastricht (NUTRIM), University of Maastricht, The Netherlands. A single administration of the carnitine palmitoyl transferase I (CPT-1) inhibitor etomoxir (ETO) acutely stimulated eating in fat-consuming subjects. We therefore examined the effects of repeated ETO administration on substrate oxidation and satiety on a high fat diet. We fed 10 healthy men (mean age 25.6y; mean BMI 21.8kg/m²) twice a high fat diet for three days at home and subsequently for 36h in energy balance in a respiration chamber and administered ETO or placebo (PLAC) 5 times in 36 h (600mg ETO in total). Blood samples were taken and appetite profiles determined in the morning of days 4 and 5. ETO increased 24h RQ (ETO: 0.833±0.014 vs PLAC: 0.814±0.006, p <0.05). -hydroxybutyrate concentration correlated with differences in overnight fasted satiety ratings between ETO and PLAC (r²=0.44; p=0.052). These results show that repetitive ETO administration inhibits 24h whole body fat oxidation in healthy subjects and provides indirect evidence for a role of hepatic fatty acid oxidation in satiety.

Glycemic, insulinemic and appetitive responses to meals differing in protein content and glycemic index: a pilot study in obese adults. K.J. MELANSON, M.R. CARPENTER, S.J. KNIFE, J. DELL'OLIO, D.P. BERLIN, J.M. RIPPE. Rippe Lifestyle Institute, 21 North Quinsigamond Ave, Shrewsbury MA 01545 USA.

Glycemic and insulinemic responses to meals varying in macronutrient composition and glycemic index (GI) have been implicated in appetite regulation. This possibility was examined in 9 obese (BMI 31.0±2.4 kg/m²; (x±sd)) adults (41.3±5.8 years, 5F, 4M) who consumed two test meals on separate days in randomized order. Both meals were 1087 kJ, matched for energy density, but the low and high GI meals contained 25.5% and 13.2% protein respectively. Blood samples and VAS appetite ratings were obtained at baseline, and 15, 30, 45, 60, 90, 120, 150 and 180 minutes postprandially. Volunteers recorded their ad libitum food intake for the remainder of the day. Following consumption of the low GI, high protein meal, peak glucose (p<0.0001), peak insulin (p<0.05), and areas under the glucose (p<0.01), insulin (p<0.01) and hunger (p<0.05) curves were significantly lower than following consumption of the high GI, low protein meal. Changes in satiety over 180 minutes were 8.7±6.5 (x±se) for the low GI, high protein meal and 2.9±8.5 for the high GI, low protein meal (ns). Differences in hunger ratings during the test did not correspond with subsequent differences in recorded energy or macronutrient intake for the remainder of the day. In these obese adults, although consumption of breakfasts differing in protein content and GI produced different glycemic and insulinemic responses and acute differences in hunger suppression, no differences in subsequent ad libitum food intake resulted. Future work is needed, examining acute and chronic effects in obese adults, as well as physiological and non-physiological factors involved.

Glucoprivic feeding, but not other glucoregulatory responses, is attenuated in dehydration anorexia. D. SALTER, A.G. WATTS. Neuroscience Graduate Program, University of Southern California, Los Angeles, California 90089-2520. Progressively dehydrated animals actively curtail their food intake. This anorexia persists until robustly reversed after access to water is restored. The neural circuits responsible for inhibiting food intake in this circumstance are unknown. We have used the glucoprivic response to the antimetabolic glucose analog, 2-deoxy-D-glucose (2DG), to help characterize the central locus of dehydration-induced anorexia. Behavioral, physiological, and neural responses to 2DG were evaluated in euhydrated non-anorexic (EU) and dehydrated anorexic (DE) male Sprague Dawley rats. Food intake was measured for four hours in a repeated measure design following injections of saline or 2DG (200 mg/kg, 250 mg/kg). Drinking water was replaced with 2.5% saline for five days, which reduced nocturnal food intake by 63%. After five days, the feeding response to 2DG was re-tested. In another group, using the same design, rats were injected with saline or 2DG (200 mg/kg) and multiple blood samples collected for glucose and corticosterone determination. In a third group, EU and DE rats were injected with saline or 2DG (200mg/kg) and sacrificed after two hours to determine brain Fos immunoreactivity (Fos-ir). Dehydration reduced 2DG-induced food intake to that of baseline levels but did not significantly change either the blood glucose or corticosterone response to 2DG. Fos-ir present in DE-2DG injected rats appeared to be additive of that present in DE-saline and EU-2DG injected rats. The present findings indicate the neural circuits activated by DE specifically inhibit feeding, but not other glucoregulatory responses, following glucoprivation.

Brain mechanism of the recognition for glucose utilization in rats. K. TORII. Central Research Laboratories, Ajinomoto Co., Inc. 1-1 Suzuki-cho Kawasaki-ku Kawasaki-shi Kanagawa, Japan. Diabetic animals display a strong sugar preference reflecting lower glucose utilization. Glucose intake in diabetics depends on its physiological needs. The brain mechanisms for the recognition of glucose peripheral utilization were studied using a functional MRI. When young male rats were treated with 25 or 50 mg of Streptozotocin (STZ), both preference for glucose and overnight fasting plasma glucose concentration remain within normal levels but rats with high STZ displayed strong preference for glucose and hyperglycemia beyond 350mg/dL. Therefore rats with 60mg of STZ/kg BW were employed as a model for insulin dependent diabetes mellitus (IDDM). Anesthetized rats were placed in a 40-cm bore of a 4.7 tesla MRI. Their plasma glucose at fasting was above 400mg/dL and normalized 100 min after subcutaneous insulin injection (20 U/kg BW). The brain flow-weighted image was visualized chronologically by the rapid gradient echo pulse method. After insulin, signal changes were observed at 20 min in the hippocampus, 30 min in the paraventricular nucleus, 40 min in the thalamus and dorsomedial hypothalamus, and 100 min in the ventromedial hypothalamus recovering after 120 min. Saline injection did not cause brain changes in IDDM rats. The brain mechanisms recognizing sweetness, glucose intake and utilization in peripheral tissues play quite important roles due to controls for glucose homeostasis and energy metabolism.

Selective immunotoxin lesions of hindbrain norepinephrine/epinephrine (NE/E) neurons impair feeding and glucocorticoid responses to insulin-induced hypoglycemia. N.M. SANDERS, T.T. DINH, S. RITTER. Washington State University, Pullman, WA 99164.

The targeted immunotoxin, anti-dβh-saporin (DSAP), can be used to selectively destroy subpopulations of hindbrain NE/E neurons. Using this technique, we have previously shown that hindbrain NE/E neurons projecting to the hypothalamus are required for the feeding response to 2DG-induced glucoprivation while those that project spinally are required for the adrenal medullary response. In this study, we injected DSAP or unconjugated saporin (SAP) control into the paraventricular nucleus of the hypothalamus to determine the role of hypothalamic projecting NE/E neurons in mediating the feeding and corticosterone (CORT) responses to insulin-induced hypoglycemia (IIH). Insulin administration (1.5U/kg) in the absence of food, reduced blood glucose to similar levels in the DSAP and SAP rats (15 and 17 mg/dl, respectively). In contrast, the CORT response in the DSAP rats was severely impaired, peaking at only 123% of pre-insulin levels, compared to 353% in the SAP rats. DSAP injections also abolished the feeding response to IIH. DSAP rats ate only 0.9 g of food during IIH while the SAP control rats ate 6.1 g of food. These findings further demonstrate the necessity of hindbrain NE/E neurons in communicating glucoprivic signals, generated not only by 2DG-induced glucoprivation, but also by IIH, to hypothalamic effector nuclei that coordinate feeding and hypothalamic-pituitary-adrenal responses.

Diet-induced hyperphagia: role of fat content and caloric density. Z.S. WARWICK. Dept. Psychology, Univ. Maryland Baltimore County, Baltimore, MD 21250.

Diets high in fat typically elicit greater kcal intake and weight gain than low-fat diets in both humans and rats. High-fat and low-fat diets differ on multiple dimensions, e.g. taste, caloric density, and postingestive processing. The development of liquid high-fat (HF) and low-fat (LF) diets (Warwick and Weingarten, *Am. J. Phys.*, 1995) has enabled a systematic evaluation of the independent and interactive contribution of these variables to high-fat diet-induced hyperphagia. Most recently, these diets have been used to explore the dose-response relationship between dietary fat content and caloric intake/weight gain. Ratio combinations of HF and LF yielded five diets with fat contents ranging from 17-60% kcal (all 2.3 kcal/ml). Diets were fed to separate groups (n=9) for 16 days, and results indicated a threshold effect (non-linear). No differential response was observed across the range of 17-49% dietary fat; however, the 60% fat diet elicited significantly greater intake and weight gain. The role of diet caloric density was investigated by formulating HF and LF at three caloric densities: 2.3; 1.15; and 0.575 kcal/ml. These six diets were fed to separate groups (n=9-10) for 16 days. Results of this study will be presented.

Refeeding induced expression of neuronal nitric oxide synthase in the rat hypothalamic paraventricular nucleus. J.W. JAHNG, T.A. HOUPTI, G.T. KIM, D.G. KIM. Department of Pharmacology and BK 21 Project for Medical Science, Yonsei Univ. College of Medicine, Seoul, Korea. 1.Department of Biological Science, Florida State University, Tallahassee, FL., U.S.A.

Nitric oxide(NO) is an important biological messenger, especially in the hypothalamic paraventricular nucleus(PVN) of the brain NO modulates a large number of neuronal, autonomic, and endocrine functions. The expression of neuronal nitric oxide synthase(nNOS), the enzyme producing NO in the PVN, is down-regulated by fasting, and we previously reported that the adrenal glucocorticoids were involved in the fasting induced down-regulation of nNOS in the PVN. In this study, we investigated if the fasting induced decrease of nNOS in the PVN recovered by re-intake of food, and if so, how fast it did. Rats were subjected to 48 hrs of fasting after 5 days of habituation with sweetened milk, then transcardially perfused with 4% of paraformaldehyde at different time points after refeeding (0, 0.5, 6, 24 hrs after the milk onset and a fed control) for the histochemical studies on mRNA, protein and enzyme activity of nNOS in the PVN. In our results, nNOS in the PVN recovered within 30 min of refeeding after 48 hrs of food deprivation (e.g. from 67 % of nNOS immunopositive cells to 82 %, compared to fed control), and returned to its original level by 24 hrs of refeeding. However, dexamethasone, a synthetic glucocorticoid, given 30 min prior to the onset of the food supply abolished the refeeding induced nNOS expression, although dexamethasone pretreatment did not alter the amount of food intake during the refeeding. Our results suggest that there may be a fast regulatory system for the induction of nNOS expression by refeeding, and the plasma glucocorticoids may be involved in this process.

Hypothalamic c-fos expression in normal and weight-reduced rats during acute inflammation T.A. LENNIE, College of Nursing, Ohio State University, Columbus, OH, USA

Reductions in body weight prior to induction of acute inflammation can attenuate the anorexia typically associated with a systemic inflammatory response. To determine potential feeding-related sites that may be involved in this phenomenon, c-fos expression is being compared in four groups of rats: two ad-lib fed normal-weight groups and two groups in which body weights were reduced by 12% over one week. Inflammation was induced by subcutaneous turpentine injections at the end of the weight reduction week in a normal-weight and a weight-reduced group, while the remaining groups received saline injections. Preliminary examination of hypothalamic c-fos expression shows that, at four hours postinflammation, both the normal and weight-reduced inflammation groups have strong immunoreactivity in the medial and ventral parvocellular regions of the paraventricular nucleus (PVN). The weight-reduced rats appear to have greater c-fos expression in the rostral parvocellular PVN and supraoptic nucleus. In contrast, only minimal c-fos immunoreactivity is visible in the arcuate nucleus, median eminence, lateral hypothalamus, and ventromedial hypothalamus of both inflammation groups at the four hour time point. Examination of c-fos expression in the brainstem and at different time points is currently underway.

A comparison of satiety measures. E.P. MERRILL, F.M. KRAMER, A. CARDELLO, H. SCHUTZ. Behavioral Sciences Division, U.S. Army Natick Soldier Systems Command, Natick, MA 01670-5020, U.S.A. A variety of measures have been used to quantify human satiety and hunger in studies of human eating behavior. The purpose of this study was to compare five such measures, consisting of 100mm visual analogue scales: 1) bipolar hunger-fullness scale, 2) unipolar hunger scale, 3) unipolar fullness scale, 4) unipolar "amount-could-eat" scale, and 5) 7-pt, equal labeled interval, bipolar scale. Nineteen subjects were served 240 kcal samples of four foods (yogurt, bread, croissants, oatmeal) varying in satiating efficacy. Each food was consumed on one of 4 consecutive days. Subjects rated their satiety on each scale before eating and every 10 minutes thereafter for one hour. Testing was repeated one week later to assess test-retest reliability. ANOVA showed no main effect of replication for any scale. Correlations among the scales indicated that the "amount-could-eat" scale was least consistent with the other scales. In addition, ANOVA of the average area under the curve showed this scale to produce significantly lower satiety scores. ANOVA with post hoc tests showed the number of mean differences between foods to be greatest for the bipolar hunger-fullness scale and lowest for the unipolar fullness scale. There were significant differences in rated satiety within and between the four foods for all 5 scales. These data suggest that reliability differences among satiety scales may be minor, but sensitivity differences may be large. Future research will focus on the identification of an optimal tool for assessing satiety including the development and testing of a labeled magnitude scale.

Using forward and reverse genetics to study hypothalamic regulation of body weight. G. BARSH, C. KAELIN, T. GUNN, L. HE, K. CLEMENT. Dept. of Pediatrics and Genetics, and HHMI, Stanford University School of Medicine, Stanford CA 94305-5323 USA.

Pleiotropic effects of melanocortin signaling were first described nearly 100 years ago when mice carrying the lethal yellow (Ay) allele of the Agouti coat color gene were recognized to develop increased growth and adiposity. Work from our laboratory and others over the last several years has demonstrated that the non-pigmentary effects of Ay are caused by ectopic expression of Agouti protein, a paracrine signaling molecule whose normal function is to inhibit signaling through the melanocortin 1 receptor (Mc1r), but which can mimic the effects of Agouti-related protein (Agrp), a homologous neuropeptide produced in the medial portion of the arcuate nucleus that acts as a potent antagonist of the Mc3r and Mc4r. Regulation of Agrp lies at the intersection of leptin and the HPA axis, and is likely to play a major role in normal and abnormal patterns of feeding behavior. Using comparative sequence analysis we have identified Agrp 5' flanking sequences that direct expression of a heterologous reporter gene to Agrp neurons, and that respond to food deprivation in the same way as Agrp. Molecular analysis of Agrp regulation should clarify the mechanisms that connect peripheral nutrient sensing to the engagement of central effectors, and may provide tools with which to perturb those pathways in transgenic animals.

Chronic hyperphagia, hyperactivity and late-onset obesity in serotonin 5-HT_{2C} receptor mutant mice. L.H. TECOTT, K. NONOGAKI, E. GOULDING, L. ABDALLAH, J. WADE. University of California, San Francisco Department of Psychiatry, San Francisco, California.

The serotonin 5-HT_{2C} receptor subtype contributes substantially to the serotonergic inhibition of feeding. Accordingly, animals bearing a targeted null mutation of the 5-HT_{2C} receptor gene display hyperphagia, evident by 5 weeks of age. Studies were performed to characterize the neuropsychological processes underlying this hyperphagia. The mutants did not display evidence of enhanced sucrose preference in two-bottle tests. Meal pattern analysis revealed a hyperphagia associated with abnormal organization of ingestive behavior bouts; a pattern that differed from that of hyperphagic ob/ob mutant mice. Interestingly, despite chronic hyperphagia, body weights of 5-HT_{2C} receptor mutants do diverge from wild type levels until 5-6 months of age. Studies were performed to evaluate compensatory mechanisms maintaining normal adiposity levels young adult mutants. Analysis of locomotor activity revealed a chronic home cage hyperactivity in the 5-HT_{2C} receptor mutants that may contribute to this compensation. The hyperactivity was tightly correlated with visits to the feeder, indicating a tight association of locomotion and feeding. A 24-hr fast further elevated locomotor activity levels in the mutants, without altering activity levels in wild type mice. During the fast, activity remained highly correlated with visits to the feeder, raising the possibility of enhanced food-seeking in the mutants. Developmental studies are underway to determine mechanisms underlying the late onset obesity of 5-HT_{2C} receptor mutants. To date, these studies reveal that late-onset obesity development is not attributable to age-dependent increases in food intake or decreases in locomotor activity. Thus, other age-dependent physiological changes that may account for late-onset obesity development in these animals are under investigation.

Melanin Concentrating Hormone and Energy Homeostasis in Transgenic and Knockout Mice. E. MARATOS-FLIER. Research Division, Joslin Diabetes Center, 1 Joslin Place, Boston, MA Physiologic and pharmacologic data indicate that melanin concentrating hormone (MCH) plays an important role in feeding behavior. MCH mRNA increases with fasting and is increased in leptin deficient ob/ob mice. When injected into the lateral ventricle of rats, MCH induces a rapid increase in feeding and treated animals eat 2-3 fold more than animals injected with artificial CSF. Genetic analysis using a knockout mouse model reveals that mice lacking the MCH gene are lean and have decreased adiposity, reflected by low leptin levels. MCH ablation appears to attenuate the obesity seen in Agouti mice. In contrast, mice overexpressing MCH eutopically demonstrate mild obesity which is strain dependent and also show increased susceptibility to diet induced obesity and hyperinsulinemia. On the C57Bl background, increased obesity is seen in both males and females. Increased weight is due to a small increase in food intake (6%) as both activity and oxygen consumption are normal. However, when fasting, MCH-OE mice show an increase in stereotypic behavior. In addition they appear to be more interested in olfactory stimuli. In summary data obtained from the study of mouse models either lacking or overexpressing MCH confirms the role of this neuropeptide as an important regulator of energy homeostasis.

Leptin and neurocytokine gene transfer to probe the etiology of hypothalamus based obesity and energy expenditure. S.P. KALRA, P.S. KALRA. McKnight Brain Institute, College of Medicine, University of Florida, Gainesville, FL 32610, USA

Adeno-associated virus, a non-immunogenic and non-pathogenic vector, can efficiently deliver target genes into neurons. We have developed recombinant adeno-associated viral vectors (rAAV) that encode either leptin (rAAV-leptin) or the cytokines, ciliary neurotrophic factor (rAAV-CNTF) and leukemia inhibitory factor (rAAV-LIF), to evaluate their efficacy in circumventing leptin resistance and to reinstate hypothalamic control on appetite, energy expenditure, and secretion of adipocyte leptin and pancreatic insulin. A single administration of rAAV-leptin vector either intracerebroventricularly or into relevant hypothalamic sites suppressed weight gain, markedly reduced adiposity and hyperinsulinemia for extended periods in outbred Sprague-Dawley rats fed ad libitum either rat chow or a high-fat (45% Kcal) diet. Further, depending upon the viral infection dosage, rAAV-leptin suppressed weight gain either by augmenting energy expenditure alone through increased thermogenesis, or along with diminution in food intake. These effects were correlated with appropriate shifts in gene expression of orexigenic and anorexigenic neuropeptides in the hypothalamus. On the other hand, rAAV-LIF and rAAV-CNTF suppressed weight gain with small reductions in blood leptin and insulin levels, and no effect on energy expenditure. Thus, central delivery of rAAV vectors encoding genes involved in weight control can circumvent leptin resistance and curb weight gain efficiently through differential modulation of hypothalamic mechanisms that regulate appetite, energy expenditure and metabolic hormone secretions. (Supported by NIH DK37273 and NS32727).

Hypothalamic-Pituitary-Adrenal Axis in the Night Eating Syndrome. G.S. BIRKETVEDT, J. SUNDSFJORD, J. FLORHOLMEN. Institute of Clinical Medicine, 9037, University of Tromsø, Norway. We have previously described the typical neuroendocrine characteristics of the night eating syndrome as changes in the circadian rhythm. Birketvedt et al described in 1999 (JAMA, 282, 657-63) these changes as attenuation in the nocturnal rise of the plasma concentrations of melatonin and leptin and increased circadian secretion of cortisol. In this study we have tested the hypothesis that night eaters have an overexpressed hypothalamic-pituitary-adrenal axis with attenuated response to stress. Five female subjects with the night eating syndrome and 5 sex- and weight-matched controls performed a 120 min corticotropin releasing hormone (CRH) test (100 ug i.v.). Blood samples were drawn intravenously for measurements of the serum concentrations of adrenocorticotropin-releasing hormone (ACTH) and cortisol. We found slightly increased basal serum concentrations of ACTH and cortisol in the night eaters compared to the controls and the night eaters showed reductions in the CRH-induced ACTH and cortisol response to 50% and 33% respectively of that observed in the control group. We therefore conclude that in the night eating syndrome there are disturbances in the hypothalamic-pituitary-adrenal axis with an attenuated ACTH and cortisol response to CRH.

The interactive effects of stress, dietary restraint, and disinhibition, on eating in a multi-item meal. C. HAYNES, M. LEE, M.R. YEOMANS. Lab. of Expt. Psych., Univ. of Sussex, Brighton, BN1 9QG, UK. Previous research has indicated that individuals categorized as high restraint (HR), according to the Revised Restraint Scale, will increase food intake in response to stress (disinhibited eating). However, when categorized according to the Three Factor Eating Questionnaire (TFEQ), several studies have not found disinhibited eating in HR individuals in response to stress. In the present study women without an eating disorder were categorized according to both TFEQ restraint (high, HR or low, LR) and disinhibition (high, HD or low, LD), generating four groups of ten women in each experimental condition. Subjects ate a multi-item meal following a Stress (S) or No-stress (N) procedure. There was an overall increase in negative affect in S compared to N, which was significantly higher in the HD groups compared to LD ($p < 0.05$). There was a trend towards a full interaction ($p = 0.056$) on total intake (Kcal). In N, the LR-HD women consumed significantly more than HR-HD. In S, there were no significant differences in intake between the R-D groups. A comparison of intake between conditions indicated that HR-HD and LR-LD increased their food intake in S compared to N, LR-HD consumed less in S, and HR-LD showed no difference between conditions. HD women were more likely than LD to report binge eating problems ($p < 0.005$). These results suggest that HD groups are more responsive to a stressor, and report more disordered eating than LD groups.

Effects of energy density with and without nutrition information on food intake in lean women. T.V.E. KRAL, L.S. ROE, B.J. ROLLS. Nutrition Department, The Pennsylvania State University, University Park, PA 16802 U.S.A.

Previous studies have shown that energy density of foods (ED; kcal/g) affected energy intake when participants were not aware of the ED of the experimental foods. It is possible that the provision of nutrition information, as in "real-life" situations, may influence the effect of energy density on intake. In the present study, lean women reported to the laboratory for breakfast, lunch and dinner on three experimental days which were separated by one week. The main entrée of each meal varied in ED (1.25 kcal/g, 1.50 kcal/g, 1.75 kcal/g), but not in macronutrient content or palatability. The ED of all entrées was held constant during one experimental day. Subjects consumed the main entrées ad libitum. Participants were divided into two groups (information and no-information). The information group received training on the topic of energy density, and at each meal they were provided with information about the ED of the main entrée in the form of a nutrition label. The no-information group was unaware of the ED of the meals throughout the course of the study. Results showed that the provision of nutrition information did not affect intake. Both groups ate a similar weight of food across the three levels of ED. Women consumed approximately 20% less energy in the lower-ED condition than in either the medium-ED or the higher-ED condition ($P < 0.05$). These findings suggest that ED has a strong influence on intake whether or not individuals are aware of a food's energy density.

Effects of test meal palatability on responses to intragastric nutrient preloads. T.M. ROBINSON¹, S.J. FRENCH¹, M.D. LEE², R.W. GRAY², M.R. YEOMANS². ¹Centre for Human Nutrition, University of Sheffield, Sheffield, S5 7AU; ²Experimental Psychology, Sussex University, Brighton, BN1 9QG, UK.

This study investigated the effects of test-meal palatability on feeding responses to intragastric infusions of fat or carbohydrate (CHO). Male volunteers consumed either a bland or palatable pasta lunch 30 minutes following intragastric infusion of either a low (63 kcal) or high (360 kcal) energy soup. Effects on food intake and rated appetite during the meal were assessed by use of a computer and disguised balance system. Preload energy content had a significant influence on overall food intake ($p < 0.01$), with the CHO infusion reducing intake to a greater extent than fat. Increasing the palatability of the test meal elevated food intake ($p < 0.01$) with the greatest effect being seen following fat infusion. High energy preloads reduced pre-meal hunger ratings regardless of meal palatability ($p < 0.01$), however, there was a tendency for subjects to report lower pre-meal fullness ratings when presented with a palatable meal. These data support previous suggestions that palatability may counteract the satiating effects of fat and CHO. The observation that palatability-enhanced food intake is less following a CHO preload, however, suggests that differing interactions between palatability and macronutrient-specific satiety may exist.

Gastric capacity and test meal intake in obese binge and non-binge eaters. A. GELIEBTER, E. YAHAV, D. HUI, M. GLUCK, S. HAQ, S.A. HASHIM. New York Obesity Research Center, St. Luke's/ Roosevelt Hospital, Departments of Medicine and Psychiatry, Columbia University College of Physicians and Surgeons, New York, N.Y. 10025. Because one function of the stomach is as a food reservoir, gastric capacity may limit the amount of food ingested and thereby influence satiety. We previously showed that obese and normal-weight bulimics have a larger than normal capacity. We hypothesized that in a subset of obese individuals, those with binge eating disorder (BED) may be responsible for the increased gastric capacity in the obese. We compared gastric capacity in two groups of obese women: BED ($n = 7$) and non-BED ($n=18$). Their ages, 30 ± 8 SD, and BMI's did not differ, 35.7 ± 5.6 SD. Following a 12 h overnight fast, gastric capacity was estimated by filling a gastric balloon with water at 100 ml/min, with 1 min pauses for measuring intragastric pressure. One estimate was based on the maximum volume the subject could tolerate. Another estimate was based on the volume required to produce a 5 cm rise of intragastric pressure. On another day following a fast, subjects were asked to consume a liquid nutritionally complete meal by straw from a covered opaque container until extremely full. The gastric capacity based on tolerated volume (1071 ml + 91 SE) was significantly larger for BED subjects ($p = .04$), than non-BED subjects (755 ml + 91). Similarly based on intragastric pressure, the gastric volume showed a trend ($p = .06$) to be larger for BED (777 ml + 192) than non-BED subjects (427 ml + 99). Likewise, test meal intake showed a trend ($p = .10$) to be larger for BED subjects (1053 ml + 97) than non-BED subjects (854 ml + 83). Thus the larger gastric capacity previously observed in obesity may be due the contribution of BED subjects. NIH Grant: DK 54318

Energy intake regulation and habitual meal frequency in time-blinded men. M.S. WESTERTERP-PLANTENGA, E.M.R. KOVACS, K.J. MELANSON. Department of Human Biology, University of Maastricht, PO-box 616, 6200 MD Maastricht, The Netherlands.

Daily energy intake is regulated more accurately in nibblers than in gorgers (1). Thus, we assessed a possible relationship between habitual as well as introduced meal frequency, blood glucose pattern, macronutrient and energy intake (EI) in men. A time-blinded within-subject design comparing iso-energetic (1MJ) iso-volumetric high fat and simple carbohydrate (CHO) preloads, was applied in twenty healthy men (18-31yrs; BMI: 22.8 ± 1.9 kg/m²), to assess energy intake regulation in spite of intervention. Introduced meal frequency, continuous blood glucose levels and patterns, macronutrient and energy intake, appetite ratings and taste perception from the two test-days were determined. Habitual meal frequency was determined from controlled 3-day food intake diaries. The difference in 24h EI on the two test-days was inversely related to habitual meal frequency ($r^2=0.56$; $p < 0.001$). Habitual and introduced meal frequency was positively correlated with the number of transient and dynamic blood glucose declines ($r^2=0.74$; $p < 0.0001$). Habitual, but not introduced meal frequency was positively correlated with average blood glucose level at baselines ($r^2=0.44$; $p < 0.01$), sweetness perception of the preload, percentage energy from CHO and inversely correlated with percentage energy from fat, hunger suppression during preload consumption, and EI during the test-days (all: $0.76 < r^2 < 0.84$; $p < 0.0001$).

Habitual meal frequency is of greater significance in energy intake regulation in healthy young men than introduced meal frequency.

1. Westerterp-Plantenga MS et al., *Appetite* 1994; 22:173-182.

Metabolic sensors: viewing glucosensing neurons from a broader perspective. B.E. LEVIN. Neurology Service (127C), VA Medical Center, E. Orange, NJ 07018, USA and Department of Neurosciences, NJ Medical School, Newark, NJ 07103, USA.

The brain maintains the body's energy homeostasis using specialized metabolic sensor neurons which receive and integrate afferent neural and metabolic signals conveying information about the body's energy status. These neurons are located in brain areas involved in homeostatic functions. Unlike most neurons that use glucose as an energy substrate, metabolic sensing neurons utilize glucose as a signaling molecule to control the rate of cell firing and neurotransmitter release. Glucose responsive (GR) neurons increase, while glucose sensitive (GS) neurons decrease their firing rate as brain glucose levels rise. Little is known about the mechanism by which GS neurons sense glucose. GR neurons appear to function like pancreatic β -cells where ATP production from glucose is regulated by glucokinase and determines the activity of an ATP-sensitive K⁺ (KATP) channel which controls firing rate. The KATP channel is composed of 4 pore-forming units and 4 sulfonylurea binding sites. NPY and POMC neurons in the hypothalamic arcuate nucleus are critical components of the energy homeostasis pathways in the brain. Both express Kir6.2, sulfonylurea receptors and glucokinase as well as leptin receptors. Both leptin and insulin act on the KATP channel to oppose the actions of glucose in these neurons which also receive visceral neural and intrinsic neuropeptide and transmitter inputs. Thus, metabolism-related signals summate upon the KATP channel activity to alter membrane potential, neuronal firing rate and peptide/transmitter release. GR efferents are integral components of effector systems that regulate energy homeostasis. Thus, arcuate NPY and POMC neurons are likely prototypes of this important class of sensor-integrator-effector neurons.

Brain glucosensing: It's all in how you slice it. V.H. ROUTH. Departments of Physiology & Pharmacology and Neurosciences, New Jersey Medical School (UMDNJ), Newark NJ 07103. Glucose is the preferred fuel of the brain. Therefore, the brain has a vested interest in the regulation of glucose homeostasis. Neurons within the hypothalamus change their firing rate in response to changes in extracellular glucose levels. These neurons exist in nuclei such as the arcuate (ARC) and ventromedial hypothalamic nucleus (VMN) which are important for the regulation of food intake and energy balance. However, early studies of these neurons used very non-physiologic levels of extracellular glucose. This raises concern about the physiologic relevance of these neurons. We have found that neurons within the ARC and VMN do respond to physiologic changes in extracellular glucose. Moreover, VMN glucosensing involves a complex convergence of pre- and postsynaptic mechanisms. That is, there are 2 subtypes of VMN glucosensing neurons which intrinsically sense glucose. These neurons, as well as non-intrinsically glucosensing VMN neurons, receive presynaptic input from other glucosensing neurons. The exact origin of these presynaptic influences remains unknown, however they must originate within the brain slice itself. Thus, it is likely that glucosensing neurons from adjacent hypothalamic nuclei (e.g., ARC) regulate VMN glucosensing neurons. In addition, these glucosensing neurons may also serve as "metabosensors" by responding to other indices of metabolic status such as insulin and leptin. Finally, both pre- and postsynaptic glucosensing mechanisms in the VMN are dysfunctional in a rodent model of diet-induced obesity (DIO) and type II diabetes in which central glucosensing is abnormal. This suggests that these mechanisms are important for the central regulation of glucose homeostasis.

POMC neurons integrate diverse metabolic signals including leptin, glucose and ghrelin. M.A. COWLEY¹, J.L. SMART¹, S. DIANO³, M. RUBINSTEIN², M.J. LOW¹, T.L. HORVATH³, R.D. CONE¹. ¹The Vollum Institute, OHSU, Portland OR 97201-3098 USA. ²Ingebi, Conicet, University of Buenos Aires, Buenos Aires, Argentina. ³Dept of Ob/Gyn, Yale Medical School, New Haven, CT 06520 USA. We have previously shown that leptin activates proopiomelanocortin (POMC) neurons in the arcuate nucleus of the hypothalamus. Leptin directly depolarizes the POMC neurons, and leptin reduces a tonic inhibition from nearby neuropeptide Y (NPY) neurons. Using the same model system, transgenic mice expressing GFP under the control of the POMC promoter, we have investigated the role of other metabolic regulators in the control of POMC neurons. We have found that insulin and glucose have similar effects on POMC neuronal activity to those which leptin has. Interestingly, the stomach peptide ghrelin inhibits POMC neurons, demonstrating that the activity of POMC neurons can be increased and decreased. Thus, we show that POMC neurons are sensitive to diverse metabolic signals; furthermore, it is possible that the POMC neurons serve to integrate diverse, peripheral signals of energy homeostasis. Supported by DK51730, DK55807, HG00201, RO1 MH59847 and TW01233.

Relative roles of central and peripheral glucose sensors in the response to hypoglycemia. R.N. BERGMAN, A. HEVENER, C. DONOVAN. University of Southern California, Los Angeles 90033. Glucose is a critical fuel for the central nervous system. In normal individuals the concentration of glucose remains within a restricted window, despite vicissitudes in fuel availability and energy demands. Traditional physiology teaches that plasma glucose is sensed by the hypothalamus. In the face of glucopenia of CNS glucose sensitive cells efferent signals excite neurohormonal responses which act ensemble to activate glucose production by liver, and restrict glucose utilization by non-CNS cells. In other vegetative regulatory systems sensors exist outside the brain monitoring conditions in the peripheral tissues. We devised the "local irrigation technique" which allowed us to test for the existence of peripheral glucose sensors. Systemic hypoglycemia in dogs was induced by insulin infusion; glucose was infused directly into carotids to minimize central hypoglycemia. Counterregulation was not prevented suggesting the existence of peripheral glucose sensors. Irrigation of the abdominal portal circulation suppressed the catecholamines response by 2/3 despite central glucopenia. To localize receptors in the abdominal region, during hypoglycemia glucose was infused into the portal vein (prevented counterregulation) or into the hepatic artery (counterregulation not prevented). These experiments localized sensors in the portal circulation, rather than in the liver itself. Insulin-induced counterregulation could be prevented by a) irrigation of the portal vein by glucose; b) denervation of the portal vein. The question of the relative roles of the two populations of sensors (CNS versus portal) were examined. Threshold for response is at a higher absolute glucose concentration for the portal (~65 mg/dl) than the central (~55 mg/dl) sensors. The former threshold is similar to that for counterregulation; the latter is for hypoglycemia awareness (for normal subjects). Thus we hypothesize that peripheral sensors mediate counterregulatory responses to moderate hypoglycemia; brain glucose sensors become important when hypoglycemia awareness is triggered and conscious behavioral changes may be induced (e.g., food-seeking behavior).

Decreased influence of estradiol on CCK-induced satiation and brainstem c-Fos expression in estradiol receptor- α null mice. N. GEARY, ¹ L. ASARIAN, ² K. KORACH, ³ D. PFAFF, ³ S. OGAWA. ¹ Bourne Laboratory, NY Presbyterian Hosp.-Weill Cornell Medical College, White Plains, NY 10605; ² Lab. Reprod. Develop. Toxicol., NIEHS, Res. Triangle Park, NC 27709; ³ Lab. Neurobio. Behav., Rockefeller Univ., NY NY 10025. An increase in the potency of the peripheral cholecystokinin (CCK) satiation-signaling pathway mediates part of estradiol's inhibitory effect on feeding in female rats. Here we tested the role of expression of the classical estrogen receptor (ER) in CCK satiation. Wild type (WT) mice and mice with null mutations of ER α (α ERKO) were ovariectomized and maintained on estradiol (75 pg/d) or vehicle. IP injections of 250 μ g of the CCKA receptor antagonist devazepide increased 3-h food intake in estradiol-treated WT mice, but not in vehicle-treated WT mice or either group of α ERKO mice. IP injections of 4 μ g/kg CCK-8 increased the number of cells in the nucleus of the solitary tract (NTS) expressing c-Fos immunoreactivity in estradiol-treated WT mice more than any other group. The NTS is the first central relay of the vagal afferent neurons that carry negative-feedback information controlling meal size from CCK's site of action in the gut. Thus, ER α is necessary for the normal behavioral and neural responsiveness of the CCK satiation-signaling system to estradiol in mice, ER β alone is not sufficient for these effects, and estradiol may act in the NTS to decrease the representation of the CCK-satiation signal. It is important to investigate whether such actions of E on CCK satiation may have roles in the pathogenesis, course, or treatment of disordered eating in women. Supported by NIH DK54523 (N.G.) and HD-05751 (D.W.P.) and NSF IBN-9728579 (S.O.).

Relationships between drug self-administration and sucrose intake. B. GOSNELL. Department of Basic Sciences, Neuropsychiatric Research Institute, Fargo ND 58107 USA

Animal and human studies indicate a positive relationship between the intake of sucrose or saccharin and the self-administration of a number of drugs. There is some indication that this relationship may be attributable to the actions of palatable foods and drugs on a common substrate, the mesolimbic dopaminergic system. This overlapping of mechanisms may partially explain the frequent observations of a high rate of co-morbidity between eating disorders and substance abuse. Further, it suggests the possibility that dietary assessments and/or manipulations may be useful in efforts to predict, prevent or treat substance abuse. Results from studies with animal models of drug abuse and relapse will be presented that examine the ability of sucrose to influence cocaine self-administration or cocaine-seeking. Preliminary data on the use of sweets to attenuate alcohol craving in humans will also be discussed.

Substance abuse among patients with eating disorders. J.E. MITCHELL. Department of Neuroscience, Neuropsychiatric Research Institute, Fargo ND 585107 USA

Studies examining the comorbidity for other psychiatric disorders among patients with eating disorders reveal higher than expected rates of substance abuse among patients with bulimia nervosa and, in some studies, among the subgroup of binge/purge anorexia nervosa patients. Because of this, some researchers have speculated that eating disorders are another example of addictive disorders, and some have attempted to treat patients with eating disorders using traditional addiction treatment models. This paper will review the similarities and differences between substance abuse and eating disorders. The paper will also review literature on atypical substances of abuse commonly used by patients with eating disorders. These include laxatives, diuretics, diet pills, the emetic drug Ipecac®, as well as other compounds. The majority of patients with eating disorders will, at least intermittently, abuse one or more of these compounds. Their use represents an important problem in terms of potential medical complications and treatment.

Possible role of accumbens dopamine and acetylcholine in sugar withdrawal and behavioral depression. B.G. HOEBEL, D. CHAU, R.A. KOSLOFF, J.L. TAYLOR, P. RADA. Dept of Psychology, Princeton University, Princeton NJ 08544, USA.

Prior research suggests that intermittent intake of large meals of sugar leads to D1 and M1 receptor up-regulation in the nucleus accumbens (NAc). Subsequent injection of naloxone i.p. causes behavioral signs of anxiety. There is also a decrease in extracellular dopamine (DA) coupled with an increase in acetylcholine (ACh) in the NAc, like that seen during opiate withdrawal. This demonstrates sugar dependency. New results suggest that this DA/ACh imbalance may be a feature of depression as modeled in the Porsolt swim test. To investigate the role of ACh as a cause of behavioral depression, a muscarinic agonist (arecholine, 40 and 80 ug) or saline (in counterbalanced order) was injected bilaterally in the NAc on Day 2 or 3 of the swim test when rats have learned to give up escape attempts and tread water instead. The muscarinic agonist decreased swimming even further ($p < 0.01$). A muscarinic M1 receptor antagonist (pirenzepine, 17.5 and 35 ug), on the other hand, acted like a classic antidepressant drug by increasing swimming in attempts to escape ($p < 0.001$). Thus the onset of immobility may be related to cholinergic activation of M1 receptors in the accumbens. The implication is that withdrawal from sugar, which releases ACh when DA is low, may cause certain aspects of depression. Thus ACh in the NAc may have some common role in drug withdrawal, sugar withdrawal and behavioral depression.

Disturbances of gastric function in bulimia nervosa. B.T. WALSH, E. ZIMMERLI, H.R. KISSILEFF, J.L. GUSS, M.J. DEVLIN. Department of Psychiatry, College of Physicians & Surgeons, Columbia University, and St. Lukes/Roosevelt Medical Center, New York, NY. There is compelling though largely circumstantial evidence that patients with bulimia nervosa have an acquired disturbance in the development of satiety. Extensive research, largely based on animal models, has demonstrated that the stomach and upper gastrointestinal tract are involved in the development of satiety. Therefore, we have examined the functioning of this portion of the gut in patients with bulimia nervosa. This presentation will describe the results of two recently completed studies of gastric function. In one study, a barostat was utilized to examine the gastric relaxation response in 16 patients with bulimia nervosa and 16 controls. Patients showed diminished relaxation (29.7 ± 24.4 (SE) ml vs 105 ± 25.8 ml, $p < .04$). In a separate study, the rate of gastric emptying of a liquid meal (Ensure Plus, 1.5 kcal/g) over 90 minutes was examined in another group of 16 patients and 16 controls. The emptying rate was significantly slower in patients compared to controls (2.32 g/min $\pm .09$ vs 3.07 g/min $\pm .09$, $p < .001$, but improved significantly after treatment (by 0.69 g/min ± 0.20 , $p < .03$). These data indicate that patients with bulimia nervosa have functional abnormalities of the stomach which may normalize with improvement in eating behavior. It is possible that these gastric abnormalities contribute to a disturbance in the development of satiety during a meal.

Trait-related serotonin disturbances in anorexia and bulimia nervosa: PET imaging studies with radioligands. W. KAYE, G. FRANK, C. MELTZER, J. PRICE, C. MATHIS, W. DREVETS, C. MCCONAHA, K. SKOVIRA. UPMC, Western Psychiatric Institute & Clinic, Pittsburgh PA USA.

Individuals with bulimia nervosa (BN) and anorexia nervosa (AN) have persistent disturbances of brain serotonin (5HT) neurotransmission after recovery. Recent family, twin, and physiologic studies show that AN and BN are cross transmitted in families, are highly heritable, and share common etiologic vulnerabilities such as restrictive eating, obsessive perfectionism, anxiety, and harm avoidance that could be related to a common 5HT alteration. In contrast, other factors distinguish subgroups; behavioral over-control and restricted eating occur in AN whereas alterations between over- and under-control as well as restricted and binge eating occur in BN. Positron Emission Tomography (PET) imaging with selective radioligands has the potential for increased understanding of previously inaccessible brain neurotransmitter function and its relationship with human behaviors. Preliminary data show that REC ED subjects had an increase in binding of [¹¹C]WAY100635 in the raphe nucleus (pre-synaptic 5HT_{1A} autoreceptors) and post-synaptic 5HT_{1A} receptors. Binding in cingulate-temporal cortical regions was positively correlated with anxiety. We hypothesize that increased post-synaptic 5HT_{1A} receptor binding is an attribute that is common to all subtypes of EDs and is associated with anxious, harm avoidant traits which make people vulnerable to develop an ED. Regional differences in [¹⁸F]altanserin binding may distinguish ED subgroups after recovery. Data suggest that REC BN women had a substantial reduction of [¹⁸F]altanserin 5HT_{2A} receptor binding localized to the orbital frontal cortex. In contrast, REC AN had diminished [¹⁸F]altanserin cingulate-temporal cortex binding. Orbital frontal 5HT circuits in BN may contribute to a vulnerability for imprecise and poorly modulated self-control whereas cingulate-temporal alterations in AN may contribute to impaired motivation and integration of cognition and mood.

Chemiluminescent in situ hybridization of the melanocortin-4 receptor in adrenalectomized lean and obese Balb/c-Avy mice. L.M. BROWN, C.T. HANSEN, R.L. ESKAY, T.W. CASTONGUAY. Department of Nutrition and Food Science, University of Maryland, College Park MD 20742

The melanocortin receptor MC4-R is important in the control of body weight. The MC4-R knock out mouse is obese and has many of the features of the yellow obese syndrome. High levels of glucocorticoids have been reported in several models of genetic obesity, including the agouti mouse. Lean and obese Balb/c mice were either adrenalectomized (ADX) or sham operated. Food intake and body weights (BW) were measured daily for 30 days. At sacrifice, brains were dissected and preserved for in situ hybridization. Plasma was assayed by RIA for ADX verification. MC4-R mRNA was measured by chemiluminescent (horseradish peroxidase) in situ hybridization. Densitometry was used to estimate the OD volume of labeled MC4-R mRNA in the basal hypothalamic area. ADX decreased BW in both lean and obese mice. On day 30 lean ADX mice weighed 28.3 ± 1.9 g. By comparison, lean sham controls weighed 34.6 ± 1.4 g ($p < 0.05$). Similarly, obese sham controls weighed significantly more than obese ADX mice (40.4 ± 1.7 g vs. 35.3 ± 1.4 g; $p < 0.05$). The ADX lean and obese groups lost BW during the experiment (-3.5 ± 1.3 g and -5.5 ± 1.8 g respectively). Conversely, the lean and obese sham controls gained weight during the experiment (0.9 ± 0.4 g and 0.7 ± 0.8 g respectively). ADX failed to change MC4-R expression in the basal hypothalamic area in either lean or obese groups. These results do not replicate our observations on the role of the adrenal on MC4-R expression in C57-Avy mice.

Melanocortin Regulation of Feeding: A cellular model. J.D. ROTH, D.K. YEE, J. HINES, S.J. FLUHARTY. Dept. Animal Biology, University of Pennsylvania, Phila, PA 19104, USA.

A good in vitro model within which to investigate molecular interactions between feeding-relevant neuropeptide systems has been lacking. We recently reported that N1E-115 neuroblastoma cells are an attractive model system because they contain mRNA for a variety of key systems, including the long-form leptin-receptor, insulin receptor, neuropeptide Y1 receptor, melanocortin 3 receptor (MC3R) and agouti-related protein (AgRP). We have begun to explore regulation and signal transduction of the MC system within this cellular model. Results from radioimmunoassay demonstrate that mRNA for AgRP (MCR antagonist) does encode for the protein and that levels of AgRP increase with differentiation of N1E-115 cells into the neuronal phenotype. Additionally, treatment of these cells with MC agonists (e.g., -MSH) produce characteristic, dose-dependent cAMP increases, implying the presence of functional MC3R protein. Investigations into the effects of MCR antagonism at these receptors are ongoing. Because these players are expressed endogenously (rather than transfected), these cells may be amenable for studying the mechanisms of up- and down-regulation of MC3Rs as well as regulation of AgRP in response to chronic MCR stimulation or antagonism. Supported by MH43787

Immunohistochemical evidence of the physiologic role for alpha-melanocyte-stimulating hormone (alpha-MSH) in the regulation of ingestive behavior. P.K. OLSZEWSKI, M.M. WIRTH, T.J. SHAW, M.K. GRACE, C.J. BILLINGTON, A.S. LEVINE. Minnesota Obesity Center, V. A. Medical Center, Minneapolis, MN 55417

The majority of data suggesting that alpha-MSH is involved in the control of feeding and energy storage comes from injection studies. Centrally-administered alpha-MSH decreases feeding, which suggests the role for this peptide in the mediation of satiety. As alpha-MSH supports the acquisition of conditioned taste aversions (CTAs) under certain conditions, the nature of its involvement in the regulation of ingestive behavior, i.e. whether it is related to aversion and/or satiety, remains unclear. In the current experiments, we applied immunostaining, including that for c-Fos – a marker of "neuronal activation", to further substantiate the physiologic role for alpha-MSH in the control of consummatory behavior. We found that an increase in a number of Fos-positive alpha-MSH neurons present in the arcuate nucleus (ARC) coincided with meal termination. Administration of a powerful aversive substance, lithium chloride, did not have any effect on activation of these neurons, but did stimulate oxytocin (OT) and vasopressin (VP) cells, which are thought to be the final elements of circuitry mediating taste aversion. Fewer activated OT/VP neurons were observed following alpha-MSH injection into the lateral ventricle or into the hypothalamic paraventricular nucleus, treatments that induce mild or no CTA, respectively. The degree of Fos immunoreactivity of OT/VP neurons paralleled the magnitude of aversive responses induced by a given treatment. These results support the hypothesis that ARC alpha-MSH acts as a satiety mediator independent from aversive mechanisms.

Failure of melanocortin-3/4 receptor agonism or antagonism to affect estradiol-induced inhibition of feeding in ovariectomized rats. C. POLIDORI, N. GEARY. Bourne Laboratory, NY Presbyterian Hospital-Weill Cornell Medical College, White Plains, NY 10605 USA. Meal size, food intake and weight gain increase in untreated ovariectomized (OVX) rats, but not in OVX rats maintained on a physiological regimen of estradiol (E₂) treatment. We investigated the hypothesis that the melanocortin 3 or 4 (MC3/4) receptors mediate estradiol's inhibitory actions on feeding. Female Long-Evans rats were bilaterally ovariectomized and implanted with injection cannulas in the lateral cerebral ventricle. Half the rats were treated with E₂ (one SC injection of 2 µg E₂ benzoate in 100 µl sesame oil every fourth day) and half with oil alone. Tests began after six treatment cycles, when E₂-treated rats weighed 37 ± 3 g less than untreated rats (p < 0.001). Tests were done on the second day after oil or E₂ treatment, i.e., the day modeling estrus, when E₂'s decreased feeding significantly. First, intracerebroventricular injections (2 µl) of aCSF and 0.5 nmol of the MC3/4 receptor agonist MTII were done just before dark onset in 18h food-deprived rats, and food intake was measured 2, 4, and 24 h later. MTII decreased food intake identically in E₂-treated and untreated OVX rats. Then, aCSF, 0.5, 0.125 and 0.0125 nmol of the MC3/4 receptor antagonist SHU9119 were similarly tested. SHU9119 elicited identical dose-related increases in food intake in E₂-treated and untreated rats. These results indicate that the MC3/4 receptor system is not involved in E₂'s inhibitory action on feeding in OVX rats. Supported by NIH DK 54523 and NIH MH 18390.

Meal pattern effects of central melanocortin receptor activation and blockade. T.H. MORAN, S. KNIPP, S. AJA, E.E. LADENHEIM. Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, 21205, USA. Central administration of alpha-melanocyte stimulating hormone (alpha-MSH) or the melanocortin 3,4 receptor agonist MT11 reduces food intake whereas administration of agouti-related protein (AgRP) or the melanocortin receptor antagonist SHU9119 increases intake. The actions of both the natural peptides or synthetic ligands are long-lasting, resulting in changes in food intake extending beyond a 24 hrs. In the current study, we began to assess the behavioral mechanisms through which melanocortin actions on feeding are expressed by monitoring the occurrence of feeding in meals. Male Sprague-Dawley rats were equipped with lateral ventricular cannulas. Doses of MT-11 (0, 0.01, 0.032 and 0.1 µg) or SHU9119 (0, 0.32, 0.56 or 1.0 µg) were administered icv 1 hr prior to dark onset and food intake in 45 mg pellets was monitored for the subsequent 20 hr. The time that each pellet was taken by the rat was computer monitored and meal pattern analyses were performed on the data. All doses of MT11 significantly reduced intake, with a maximum reduction of 57%. MT11-induced reductions were expressed as significant changes in both meal number and meal size. SHU9119 significantly increased food intake beginning at a dose of 0.56 µg with a maximum increase of 95%. In contrast to the effect of MT11, SHU9119 did not significantly alter meal frequency but specifically increased meal size. These data are consistent with melanocortin induced alterations in the efficacy of within meal satiety signaling as a mechanism of action for their feeding effects. Supported by DK19302.

Food restriction in the hours after injection reduces long-term hyperphagic effects of SHU9119. D.L. WILLIAMS, H.J. GRILL, J.M. KAPLAN. Psychology Department, University of Pennsylvania, Philadelphia, PA 19104, USA.

A single icv injection of SHU9119, a melanocortin 3/4 receptor antagonist, results in profound hyperphagia that persists for up to 4 days. Explanations for this long-duration effect may involve pharmacokinetics, genomic changes, and interactions with peripheral events associated with feeding state. We provide evidence for the latter, first by demonstrating that the long-term hyperphagia is abolished if rats are food-deprived for 24 h following 4th-icv SHU9119 injection. We then asked whether this reversal is due to the 24 h food deprivation, itself, or related to a lack of feeding at some critical time soon after drug injection. When rats were denied access to food for only 6 h after injection of SHU9119, the long-term effect was significantly smaller than that seen under ad lib feeding conditions. In the reverse situation, rats were injected and then given 6 h of access to food, followed by food deprivation for the next 18 h. Here, preliminary data indicate that SHU9119 caused long-duration increases in feeding equivalent to that seen when animals were fed ad lib. The results thus far suggest that feeding in the first several hours after SHU9119 injection is necessary and sufficient for the treatment's long-duration effects. Supported by NSF fellowship, NIH DK-42284, and -21397.

Marital careers and food choices. J. SOBAL, M.M. CONNORS, C.M. DEVINE, C.A. BISOGNI. Nutritional Sciences, Cornell University, Ithaca, NY 14853. Marital status is an important influence on food choices. Marital careers involve the adoption and enactment of marital identities, transformations between marital identities, and continuation of past and consideration of future marital identities. This analysis examined marital careers and food choices using qualitative data from three in-depth interviews with 86 adults. Food choice issues in three marital identities were analyzed: Unmarried, married, and previously married. Food choice issues expressed by unmarried people focused on eating and living alone, sharing cooking and eating with parents or roommates, and negotiating about food with boyfriends/girlfriends (with unmarried people with children emphasizing child feeding responsibilities). Food choice issues discussed by married people (and cohabitators) included adapting to marriage, awareness of spousal food preferences, mutual spousal influences, responsibilities for food tasks, negotiations about food choices, and feeding the family. Food choice issues voiced by previously married people included stress from spousal loss, eating alone, influences of prior partners, and (for some) developing new relationships. These findings suggest that people enact their marital identities in the process of making food choices during their marital careers. Common marital food choice issues occurred within each marital identity. Interpretations of current eating patterns were made using contrasts with other prior and potential identities in their marital careers.

Motivations for fruit and vegetable consumption in the UK Women's Cohort Study. J. POLLARD, D. GREENWOOD, J. CADE. Nutrition Epidemiology Group, Nuffield Institute for Health, University of Leeds, LS2 9PL UK.

A four-day food diary and a questionnaire, including the Food Choice Questionnaire (FCQ) and a measure of stage of change (SOC) for fruit and vegetable consumption, were administered to females, aged 35-69 years, participating in the UK Women's Cohort Study (n = 998). From the diary data fruit and vegetable intake was calculated and then averaged over the four-day period. Motivation scores from the FCQ were compared with fruit and vegetable intake and SOC data. Median daily fruit and vegetable consumption was 6.7 portions. Within these women health, sensory appeal, natural content and weight control were the strongest determinants of general food choice. In a multiple linear regression model, including age, education level and all FCQ motivations, the strongest motivations affecting specifically fruit and vegetable intake were health and natural content. It was found that for a one point increase (measured on a scale of 0-4) in health and natural content scores, fruit and vegetable consumption increased by 1.11 portions (95% C.I. 0.53 - 1.70) and 0.84 portions (95% C.I. 0.44 - 1.25) respectively (p<0.01). The SOC evaluation showed significant associations with portions of fruit and vegetables consumed (p<0.01). Women who reported having eaten at least five servings of fruit and vegetables daily for more than six months, were classed as maintenance phase and were found to score higher on the health, natural content, weight control and ethical concern factors (p<0.01). These women also scored lower on convenience questions (p<0.01). These results may have important implications for future health promotion strategies.

Consumer health consciousness and the organic foods boom: fact or fiction? K. BRUNSØ, J. SCHOLDERER. Centre for Market Surveillance, Research and Strategy for the Food Sector (MAPP), The Aarhus School of Business, Denmark.

Sales of organic foods have tremendously increased over the last years. The conclusion seems obvious: European consumers have become more health-conscious. Or have they? In fact, it is not quite clear from previous research whether rising market shares reflect changes in consumer attitudes, changes in the supply structure, or changes in the pricing of foods. Five scales from the Food-Related Lifestyle instrument (FRL) were used in replicated consumer surveys in Germany in 1993 (N = 1000) and 1996 (N = 1042), France in 1994 (N = 1000) and 1998 (N = 1000), and the UK in 1994 (N = 1000) and 1998 (N = 1000). The five scales (three items each) assessed the importance of organic foods, healthiness, freshness, novelty, and the price/quality relation to consumers' food choices. Trends in the importance of these aspects were modeled using multi-sample confirmatory factor analysis with structured means. Results indicate that, contrary to widespread expectations, the importance of healthy/unprocessed foods, organic foods, and fresh foods has been declining in all three countries since the early 1990s. The pattern suggests that the actual consumer trend to organic foods already peaked several years ago, and that the current boom is likely to be a mere short-term consequence of changes in pricing and distribution.

Perceived health and environmental consequences as predictors of consumer attitudes and behaviour towards organic foods. M.K. MAGNUSSON, A. ARVOLA, U.-K. KOIVISTO HURSTI, L. ÅBERG*, P.-O. SJÖDÉN. Department of Public Health and Caring Sciences, Section of Caring Sciences, University of Uppsala, Uppsala Science Park, S-751 83 Uppsala, Sweden and *Department of Psychology, University of Uppsala, P. O. Box 1225, S-751 42 Uppsala, Sweden.

The overall objective of the present study was to investigate the importance of perceived environmental and human health consequences for consumer attitudes and purchase of organic foods. More specifically, the aims were to investigate: 1) consumer perceptions of the likelihood of occurrence and importance of three types of consequences of their choice of organic foods: environmental, human health consequences and the well-being of domestic animals; 2) the possibility to predict consumer attitudes and behaviour related to organic foods on the basis of such perceptions; and 3) on the basis of self-reported recycling and other environmentally friendly behaviours. A questionnaire was mailed to a random nation-wide sample of 2000 subjects (response rate 58%), ages 18-65 years. Most of the questions were focused on four target foods: milk, meat, potatoes and bread. The majority considered it likely and important that their choice of organic foods would result in positive environmental and human health consequences, and the improved well-being of domestic animals. Perceived human health consequences and the performance of environmentally friendly behaviours (e.g. "purchase environmentally friendly labelled products" and "save electricity") were the most important predictors of attitudes, the importance of the criterion "organically produced", purchase intention and purchase frequency of the investigated foods. The results suggest that health concerns is a stronger motive for purchasing organic foods than is concern for the environment.

Changing public perceptions of genetically modified foods: Effects of consumer information and direct product experience. J. SCHOLDERER, T. BECH-LARSEN, K.G. GRUNERT. Centre for Market Surveillance, Research and Strategy for the Food Sector (MAPP), The Aarhus School of Business, Denmark.

Previous research concerning public perception of GM foods indicates that European consumers hold firm negative attitudes to GM foods. These attitudes, however, are not based on risk-benefit evaluations of particular products. Rather, they seem to be a function of general sociopolitical attitudes and values. Two policies can be adopted in such a situation: (a) consumers can be actively informed regarding the risks and benefits and (b) consumers can be given the opportunity to evaluate products on the basis of direct experience. The effectiveness of both policies was tested in two experiments. In Experiment 1, attitude change experiments were conducted with consumers from Denmark, Germany, Italy and the UK (N = 1650). Different information strategies were tested against a control group for their ability to change consumers' attitudes and their influence on product choice. Results indicate that no attitude change occurred. Instead, all strategies seemed to bolster pre-existing attitudes, thereby significantly decreasing consumers' preferences for GM products. The effect did not occur when consumers only saw a labeled product example. In Experiment 2, we tested the effects of direct experience on consumers' attitudes and product preferences. Preferences for different cheeses were elicited under different tasting conditions from Danish, Finnish, Norwegian and Swedish consumers (N = 753) and scaled by means of conjoint analysis. Results indicate that direct tasting experience had a positive effect on consumers' attitudes and preferences for GM foods. The effect was particular strong when the product offered additional health benefits.

More apples for the young: Can a means-end based framework explain intentions to increase apple consumption? K.G. GRUNERT, J. SCHOLDERER. MAPP, The Aarhus School of Business, Denmark.

The means-end framework for analysing consumer behaviour has achieved considerable popularity also in the food area, because it is intuitively appealing and combines the cognitive with the motivational. However, the major underlying assumptions have rarely been put to empirical test, especially since the major data collection method usually used, the laddering interview, enforces a data structure in line with the theoretical assumptions. We report a study attempting to explain Danish adolescents' intent to eat more apples based on a means-end framework. Qualitative input from a laddering study was transformed to verbal formulations of links between attributes, consequences and values, which respondents in a subsequent survey had to rate in terms of agreement. We group value-consequence links and consequence-attribute links into a number of latent constructs and test the hypothesis that V-C links determine intention mediated by C-A links. Employing structural equation modelling, we test this model (which follows from the assumptions of means-end theory) against competing models.

Use of a chimeric receptor strategy reveals commonalities and dissimilarities in the molecular mechanisms within the angiotensin II receptor family. D.K. YEE, J. HINES, S.J. FLUHARTY. Department of Animal Biology and Institute of Neurological Sciences, University of Pennsylvania, Philadelphia, PA. 19104.

Angiotensin II (AngII) is a key regulator of salt appetite and thirst. The peptide hormone exerts its biological effects by binding to cell surface receptors, of which two subtypes (termed AT₁ and AT₂) have been identified. Much of the current research on elucidating AngII receptor structure-function relationships, i.e. identifying amino acids within the receptor protein that confer ligand binding and functional properties, has focused on the AT₁ subtype. Receptor mutagenesis data have identified AT₁ structural elements responsible for ligand binding and receptor activation, thereby leading to the development computer models illustrating possible molecular mechanisms that underlie ligand-receptor interactions. In contrast, analogous efforts on the AT₂ receptor have lagged behind. Furthermore, the extent that current AT₁ models are applicable to AT₂ receptors is unclear due to the surprisingly low homology (34%) shared between the two subtypes. Consequently, we are using a chimeric receptor strategy, i.e. creating mutational combinations of the AT₁ and AT₂ subtypes, to probe the molecular structure of both receptors. Our investigations have revealed that AT₂ receptor activation mechanisms may be functionally similar to the AT₁ counterpart despite drastic differences in sequence homology. Moreover, these studies are the first to identify binding determinants for the AT₂-selective ligand CGP 42112A. These experiments represent our continuing efforts to identify the structural determinants defining AT₁ and AT₂ receptor binding and function. By comparing and contrasting AT₁ and future AT₂ models, a greater understanding of the molecular properties of the entire AngII receptor family may be established. Supported by NIH grants HL58792 and MH43787.

Activation of the different signal transduction pathways of the AT₁ receptor requires different structural determinants. J. HINES, S.J. FLUHARTY, D.K. YEE. Dept. of Pharmacology and Animal Biology, University of Pennsylvania, Philadelphia, PA 19104-6046.

The ingestive behaviors classically associated with the neuropeptide angiotensin II (AngII) -- increased thirst and salt appetite -- are mediated by centrally-located angiotensin II type 1 (AT₁) receptors. This receptor subtype has been demonstrated to signal through (1) phosphoinositide turnover, which controls cellular calcium mobilization, and (2) activation of the p42 and p44 mitogen-activated protein kinases (MAP kinases), intracellular effectors which control gene expression and cell proliferation. Conversely, the enigmatic AT₂ receptor subtype is incapable of activating either of these intracellular effectors. A series of chimeric AngII receptors was constructed by replacing transmembrane-spanning and intracellular domains of the AT₁ receptor with the corresponding domains from the AT₂ receptor. Like the wild type AT₁ receptor, the chimeric receptors bound [¹²⁵I]-angiotensin II and mediated AngII-dependent activation of MAP kinases. However, one of the chimeric receptors was incapable of signaling through phosphoinositide turnover, despite evidence that it was coupled to G proteins. Both G_q- and G_i-mediated activation of MAP kinases have been reported for other G protein-coupled receptors. While the AT₁ receptor is capable of coupling to both G_q and G_i, the signaling pathway(s) utilized to activate MAP kinases has not been completely determined for this receptor. The present results do not support a role for G_q in AT₁ receptor-mediated activation of MAP kinases. Furthermore, the results indicate that the structural determinants for signaling through MAP kinases and phosphoinositide turnover are partially distinct in the AT₁ receptor. Supported by HL58792 and MH43787.

Lack of a 2-deoxy glucose-induced feeding response in mice lacking the dopamine 3 receptor. J.-A.M. MCQUADE¹, D.J. CLEGG¹, M. XU², R.J. SEELEY¹, S.C. WOODS¹. Departments of Psychiatry¹ and Cell Biology², University of Cincinnati, Cincinnati, OH 45220.

The non-metabolizable glucose analogue, 2-deoxy-D-glucose (2DG), causes decreased intracellular glucose utilization by disrupting glycolysis. The administration of 2 DG dramatically stimulates food intake in animals, and central injections are efficacious at doses that are ineffective when given peripherally, supporting the hypothesis that glucoprivic feeding is centrally mediated. Administration of 2 DG either systemically or centrally elicits dopamine release in the hypothalamus, suggesting that dopamine may be an important mediator of 2 DG-induced glucoprivic feeding. To date, neither Dopamine 1 (D1 and D5) nor Dopamine 2 (D2, D3 and D4) class receptors have been implicated in this effect. We administered 2DG intraperitoneally (250-750 mg/kg) to wild-type and dopamine 3 receptor (D3) knockout mice and monitored food intake for 4 hours post injection. 2DG elicited robust eating in wild-type mice. In contrast, D3 receptor knockout mice ate significantly less. As an example, after the 500 mg/kg dose, at the 4 hour time point, wild-type mice ate 27.5 g/kg whereas knockouts ate 15.1 g/kg (P < 0.005). These results imply that the D3 receptor mediates feeding in response to 2DG.

Microinjection of recombinant adeno-associated virus vector encoding rat leptin gene (rAAV-leptin) in discrete hypothalamic sites suppressed weight, adiposity and plasma insulin for extended period in adult rats. M. BAGNASCO, M.G. DUBE, H. DHILLON, P.S. KALRA, S.P. KALRA Department of Neuroscience, Department of Physiology, McKnight Brain Institute, University of Florida, Gainesville, FL 32610-0244, USA

We have previously shown that a single intracerebroventricular injection of rAAV-leptin to outbred male and female Sprague-Dawley (SD) rats suppressed the age-related weight gain and adiposity for extended periods. In view of the reports that leptin targets for energy homeostasis are widely distributed in the hypothalamus, rAAV-leptin or the control vector (rAAV-UF5) was microinjected bilaterally (10^{13} particles/ml) into either the paraventricular nucleus (PVN, 0.3 μ l/ injection) or ventromedial nucleus (VMN, 0.5 μ l/ injection) of adult female rats maintained on ad libitum rat chow and water for 8 weeks. Leptin mRNA over-expression in the microinjected hypothalamic sites was confirmed by RT-PCR. Results showed that at 8 weeks post-injection rAAV-leptin microinjection either into the PVN or the VMN decreased weight by 27-29%, and food intake by 17-22% vs controls. Serum leptin levels were reduced by 82-97% along with 90% reduction in insulin. Glucose and non-esterified fatty acids were reduced by 29% and 35% respectively. In addition, UCP-1 mRNA in brown adipose tissue was increased in rAAV-leptin microinjected rats, thereby implying increased energy expenditure through thermogenesis. In rats pair-fed to the level of rAAV-leptin treated rats, weight was reduced by 4-10% only and other parameters, including leptin and UCP-1 were unchanged. Thus, increased leptin availability at targets in either the PVN or VMN by leptin transgene, prevented the age-related gain in weight, adiposity and plasma insulin levels in outbred SD rats, the responses attributable to concomitant decreases in energy intake and increases in energy expenditure. (Supported by NIH DK37273)

Central leptin gene therapy blocks high fat diet-induced weight gain, hyperinsulinemia, hyperleptinemia and leptin resistance in rats. M.G. DUBE, E. BERETTA, H. DHILLON, S.P. KALRA, P.S. KALRA. Departments of Physiology and Neuroscience, McKnight Brain Institute and University of Florida College of Medicine, Gainesville, Florida.

Rats fed a high fat diet show increased adiposity due to development of resistance to peripheral leptin. We tested the hypothesis that central leptin insufficiency may contribute to excessive weight and fat deposition in these rats. Adult male rats were injected intracerebroventricularly (5 μ l) with a recombinant adeno-associated viral vector encoding either rat leptin (rAAV-Lep, 10^{10} particles) or green fluorescent protein (rAAV-GFP, 10^{11} particles) and then maintained on a high fat (45 Kcal%) diet. An additional group of unoperated control rats was maintained in parallel on standard laboratory chow (11 Kcal%) for 9 weeks. Cumulative food consumption (Kcal) was similar in the three groups. The results showed that rAAV-Lep injection prevented the weight gain seen in rAAV-GFP controls (4.8% vs 44.7%, $p < 0.05$) and weight was maintained below that seen in chow fed controls. This was accompanied by blockade of both hyperinsulinemia (0.13 vs 1.52ng/ml, $p < 0.05$) and hyperleptinemia (0.19 vs 4.74ng/ml, $p < 0.05$) seen in rAAV-GFP controls. Serum glucose levels were within the normal range for all groups. These results show, for the first time, that increased availability of leptin produced by leptin transgene locally in the hypothalamus, readily overcomes the high fat diet-induced weight gain, hyperinsulinemia, hyperleptinemia and leptin resistance, and support our hypothesis that despite peripheral hyperleptinemia, leptin insufficiency at hypothalamic target sites may underlie increased adiposity, hyperinsulinemia and excessive weight gain in response to a high fat diet. (Supported by NIH NS32727 and DK37273)

Positional cloning of *Sac* (*Gpr98*), a gene controlling sweetener ingestion. M.G. TORDOFF, A.A. BACHMANOV, X. LI, D.R. REED, S. LI, Z. CHEN, P.J. DE JONG, C. WU, D.R. WEST, A. CHATTERJEE, D.A. ROSS, J.D. OHMEN, G.K. BEAUCHAMP. Monell Chemical Senses Center, Philadelphia, PA 19104, USA .

We have identified a gene responsible for the avidity for sweeteners (e.g. sucrose, saccharin, D-phenylalanine). The gene was isolated by (a) genetic mapping, using a C57BL6/ByJ x 129P3/J mouse strain intercross and a 129.B6-Sac segregating partially congenic strain, (b) physical mapping, based on a bacterial artificial chromosome (BAC) contig that was generated by screening the RPCI-23 mouse BAC library, and (c) sequencing of BAC 118E21, followed by gene identification. The gene encodes an 858-amino acid protein with a seven-transmembrane motif that is moderately homologous to G-protein coupled receptors, including T1R1 and other putative taste receptors. The gene product is expressed in mouse and human tongue (and other organs). Several single nucleotide polymorphisms (SNPs) in the gene cosegregate with sweetener preference. One SNP results in substitution of isoleucine for threonine at the 60th amino acid. Mouse strains with isoleucine here have high sweetener preference whereas those with threonine have low sweetener preference. We therefore believe we have identified a gene responsible for sweet taste detection and, consequently, the avidity for sweeteners. This is the first time that positional cloning of a quantitative trait locus (*Sac*) has been used to identify a gene (*Gpr98*) underlying an ingestive behavior.

Neurocircuitry and neurochemistry of the dorsomedial hypothalamic nucleus. N. VRANG, P.J. LARSEN, M. TANG-CHRISTENSEN. Laboratory of Obesity Research, CCBR, Ballerup Byvej 222, 2750 Ballerup, Denmark.

Pioneering work by Bernardis and Bellinger in the early 1970's first pointed to an important role of the dorsomedial hypothalamic nucleus (DMH) in the regulation of growth and body-weight. Since then, a vast number of anatomical and behavioral studies have substantiated the DMH as one of the key components in the hypothalamic circuitry that regulates and controls body energy homeostasis. Our understanding of DMH function in appetite and body-weight regulation is in large part dependent on the degree to which we understand the intrinsic and extrinsic anatomy of the DMH. The cytoarchitectural organization of the DMH is rather complex and the nucleus consists of several subdivisions. Although the neurochemistry of the majority of the DMH neurons is unknown, a number of neurotransmitters implicated in appetite regulation have been localized to cell bodies and/or fibers in the DMH. Recent data suggest that the DMH contains a population of NPY expressing neurons that are sensitive to metabolic manipulations. Also, our laboratory has shown the existence of a GLP-2 containing pathway linking the brainstem with the DMH and implicated in the regulation of feeding behavior. The interconnectivity between the DMH and other hypothalamic nuclei, forebrain and brainstem areas will be discussed in light of the known cytoarchitecture and neuropharmacology of the nucleus.

The Dorsomedial Hypothalamic Nucleus (DMN), and its role in feeding behavior; lessons learned from lesioning studies. L.L. BELLINGER, L.L. BERNARDIS. Dept. of Biomed. Sci., The Texas A&M Univ. Syst. Hlth. Sci. Ctr., Dallas, TX and VA Western New York Health Care System and SUNY at Buffalo, NY. Starting in 1963 numerous papers have shown that bilateral electrolytic DMN lesions (DMNL) [stainless steel or platinum-iridium electrodes and anodal/cathodal current] produced a hypophagic, hypodipsic, lighter and shorter rat, with notably normal body composition (Proc Soc Exptl Biol Med 218:284, 1998). Body weight (BW) reduction prior to DMNL produced a hyperphagic rat that gained BW until it reached the BW of the non-restricted DMNL control group. DMNL rats will defend their lower than normal BW. If ovariectomized or given palatable diets the DMNL rats increase their food intake (FI) and BW gain. While DMNL rats are hypodipsic they show normal responses to intra- and extracellular thirst challenges. Intermediary-metabolism parameters are normal following DMNL, whereas pair-fed control rats show many alterations. These above findings suggested that the DMNL changed the rat's BW 'set point' without altering their responsiveness to changes in their body fat 'set point'. DMNL rats don't respond normally to glucoprivic challenges, i.e., 2DG, glucose or insulin, and CCK. However, DMNL rats do meter 24h caloric intake; generally have normal meal patterns; and make normal macronutrient selections, which suggests that compensation for short-term regulatory deficits occurs. Alternately, DMNL rats can over-consume highly palatable foods and become obese. DMNL rats have normal ultradian secretion of growth hormone prior to establishing their lower BW. Circadian concentrations of growth hormone, insulin-like growth factor-1, thyroxine, triiodothyronine and insulin are normal in the growth-retarded DMNL rat. Excitotoxin lesions showed that damage to DMN cell bodies was responsible for the majority of the previous findings. Naloxone didn't suppress FI of DMNL rats suggesting the DMN contained an opioid system that increased FI by inhibiting a satiety system. DMNL would remove this inhibition and leave the satiety system uninhibited and produce hypophagia. Also not damaging feeding systems may explain the ability of the DMNL rat to increase their intake under certain challenges. Knife-cut experiments showed that loss of pathways leaving-entering the DMN from posterior and ventral routes are important in producing hypophagia and reduced BW and that the DMN is important in the early recognition of imbalanced amino acid diet consumption. The DMN contains neurotransmitters and receptors, e.g., leptin and orexin that may also affect feeding behavior.

The DMH NPY system: lessons learned from the OLETF rats. S. BI, E.E. LADENHEIM, T.H. MORAN. Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205.

Neuropeptide Y (NPY) is a hypothalamic orexigenic peptide involved in the regulation of food intake and energy balance. The arcuate nucleus (ARC) contains a major population of NPY neurons that are involved in leptin signaling. NPY mRNA expression in these neurons is increased by acute food deprivation and decreased in response to leptin administration. The dorsomedial hypothalamus (DMH) also contains a significant NPY containing neuronal population. The regulation of NPY mRNA expression in these two populations appears to be differentially mediated. Characterization of the relative responses of ARC and DMH NPY to acute food deprivation reveals deprivation duration induced increases in ARC NPY but not in DMH NPY. Recent data from Otsuka Long-Evans Tokushima Fatty (OLETF) rats lacking cholecystokinin A (CCK-A) receptors, also suggests an alternative role for NPY in the dorsomedial hypothalamus (DMH) in the controls of food intake and energy balance. DMH but not ARC NPY mRNA expression is significantly increased in both young preobese OLETF rats and in OLETF rats pair fed to amounts consumed by LETO controls. In intact rats, NPY and CCK-A receptors are co-localized in DMH neurons. Thus, central CCK acting through CCK-A receptors in the DMH may signal the DMH NPY pathway to limit food intake. A disruption in this signaling pathway may play a causative role in the hyperphagia and obesity of OLETF rats. These data suggest that hypothalamic NPY may be differentially regulated in the ARC and the DMH to control food intake and energy balance (Supported by DK57609).

Neuropeptide Y (NPY) Neurons in the Dorsomedial Hypothalamic Nucleus (DMH): Implications in the Development and Regulation of Hypothalamic Feeding Circuits. K.L. GROVE, P. CHEN, M.S. SMITH. Division of Neuroscience. Oregon Region Primate Research Center. Oregon Health Sciences University. Beaverton, OR 97006. In the normal adult rodent NPY neurons in the hypothalamus are limited to the arcuate nucleus (ARH), with low levels in the compact zone of the DMH (cDMH). However, in specific models of obesity or hyperphagia, NPY neurons have been reported in the noncompact zone of the DMH (ncDMH). Very little is known about the role of the DMH-NPY neurons in the regulation of food intake or energy balance. We are investigating the regulation of NPY gene expression in the DMH in two natural models of perceived negative energy balance: postnatal development and lactation. During development, the rat pup displays high levels of NPY mRNA in the both the cDMH and ncDMH, with the expression peaking around postnatal (P) day 15-16, and then declining to levels seen in the adult by P30. While fasting for 36 h at P10 or P15 significantly increases NPY mRNA expression in the ARH, NPY gene expression in the ncDMH is decreased by this treatment. The functional significance of NPY neurons in the DMH during postnatal development is unknown, but they may be important for regulation of energy balance prior to innervation of the DMH by ARH-NPY neurons. During lactation, the suckling stimulus activates not only ARH-NPY neurons but also ncDMH-NPY neurons that project to the paraventricular nucleus. However, blocking suckling-induced prolactin release inhibits the activation of the ncDMH-NPY neurons, but not those in the ARH. Furthermore, prolactin receptor expression in the DMH corresponds with the distribution of NPY neurons in this region, suggesting that prolactin may directly activate ncDMH-NPY neurons.

The General Labeled Magnitude Scale provides valid measures of genetic variation in taste and may be a universal psychophysical ruler. L.M. BARTOSHUK, V.B. DUFFY, K. FAST, B.G. GREEN, D.J. SNYDER. Department of Surgery, Yale University School of Medicine, New Haven, CT.

Since we cannot share experiences, how can we obtain valid across-group comparisons? Borg and Teghtsoonian proposed that the strongest sensations are equal across modalities and individuals, making absolute comparisons possible. We present two experiments that question this. First, subjects (N=26) rated common sensations including the strongest remembered from a variety of sensory modalities; there were significant differences across them. Next, subjects (N=247) inexperienced with PROP (6-n-propylthiouracil) rated the strongest sweet, salty, sour and bitter sensations remembered, then rated PROP bitterness; taste ratings correlated with PROP bitterness (ie, significant differences occurred across individuals). Although our data do not support the Borg/Teghtsoonian model, the strongest of all sensations of any kind might be approximately equal to everyone and thus could serve as the upper limit of a general scale of intensity. We generalized the Labeled Magnitude Scale (LMS, Green et al) by placing "strongest imaginable sensation of any kind" at its top (gLMS). Comparison with magnitude matching suggests that the gLMS produces valid measures of genetic taste variation. This work owes much to Borg, Moskowitz, Gracely, Schutz and Cardello who produced similar adjective spacings on scales with ratio properties. We conclude that intensity adjectives have veridical relationships to one another but do not denote absolute intensity. Adjective-labeled scales are like rulers that can stretch or compress to fit any domain. By stretching the scale to a maximum that encompasses all intensity domains, the gLMS may have promise as a universal psychophysical ruler; it may be useful for the study of ingestive behavior.

Testing the effects of personalised feedback to influence dietary intake awareness. A. OENEMA, J. BRUG. Department of Health Education and Promotion, Maastricht University. Testing the effects of personalised feedback to influence dietary intake awareness. Purpose: Intake levels of fat, fruit and vegetables are not in accordance with recommendations for most people. Nevertheless many people do not intend to change to healthier diets. Lack of awareness of personal intake is thought to be a major barrier in motivating people to behaviour change. Based on the Precaution Adoption Process personalised feedback on intake levels is proposed as a strategy to influence awareness. The present study examined two personalised feedback interventions: a web-based computer-tailored nutrition education program and a written self-test. The interventions were tested against a control condition in which general nutrition information was provided. Methods: A randomised controlled trial, with a pre-test post-test design, was conducted. Three hundred adults participated in the study. Outcome measures were awareness and intention to change and appreciation of the intervention. Results: Anovas and Ancovas showed significant differences in awareness and intentions between the tailored intervention group and both the self-test and control groups. There were no significant differences between the self-test group and the control group. Furthermore, the tailored intervention was appreciated best. Conclusions: It was concluded that a web-based tailored nutrition education program is a promising means to make people aware of their dietary intake levels and thus motivating them to change to a healthier diet. Self-tests did not outperform the control condition, therefore, self-tests seem to be not more effective in influencing awareness and motivation to change than general nutrition information.

Strategies for increasing statistical power in studies on human energy intake. C.L. PELKMAN, L.S. ROE, B.J. ROLLS. Nutrition Department, The Pennsylvania State University, University Park, PA 16803. Determination of statistical power is especially important in research on human ingestive behavior where the costs of conducting well-controlled experiments can be prohibitive. In this presentation we will focus on the concept of effect size in the determination of statistical power. Effect size is determined by simply dividing an expected difference between means by a measure of its variation. Increasing the effect size in an experiment decreases the number of subjects needed to obtain adequate statistical power (0.80). Using data from our laboratory and from others, we will demonstrate how aspects of research design and characteristics of the participants can affect the numerator or denominator in the calculation of effect size. For example, in a preloading experiment we found that reducing the variance in lunch intake, by using a within- subjects rather than a between-subjects design, increased the effect size and reduced the number of subjects required from 40 to 17 per group. Effect size can also be increased by increasing the numerator in the equation (i.e. the expected difference in energy intake between groups). We found that for women, soup reduced lunch intake more (134 kcal) than a large-volume, milk-based drink (54 kcal). The larger effect size in the soup study resulted in a smaller required sample size (n=12) than the milk preload study (n=83). These examples, as well as others we will provide, demonstrate some practical aspects of research design for the investigator to consider in the planning of efficacious studies on human ingestive behavior.

Words used by patients with bulimia nervosa and healthy controls to express sensations associated with different types of meal. J.L. GUSS, H.R. KISSILEFF, B.T. WALSH, D.A. BOOTH. St. Luke's/Roosevelt Hospital Center and Columbia University, New York, NY 10025, USA. This study was undertaken to determine, in relation to eating: 1) whether words chosen by investigators for rating scales match those used by subjects, and 2) whether this vocabulary differs between patients with Bulimia nervosa (BN) and healthy controls. Eighteen BN patients and 52 controls each recorded up to four words to describe their feelings in the following scenarios: after eating "a meal that disagreed with you", "the largest meal ever", "a normal dinner" and "a small snack". Words chosen by subjects were then categorized based on their literal definition and grammatical root. Of the 80 words recorded by patients and the 328 by controls, 94% fell into the following five categories: illness, satisfaction, anxiety, filled and lethargic. Words used most frequently to describe 'illness' were 'nausea' for controls (35/84 words) and 'sick' for patients (14/27 words), and words most frequently used to describe 'filled' were 'full' for controls (38/92 words) and 'bloated' for patients (8/15 words). Across scenarios, words expressing negative affect (illness, anxiety) were used more frequently by patients (42/67 words) than by controls (93/317 words), whereas words expressing positive affect (satisfaction, filled) were used more frequently (224/317 words) by controls than by patients (25/76 words); $X^2(1) = 27, p < 0.0001$. We conclude: 1) Investigators should consider modifying their frequent usage of "full" and "sick" on standardized scales, because subjects only used these words about half the time; 2) Following meals, the predominant mood of BN patients appears to be malaise rather than pleasure.

Effect of Rating Scales for feelings in imagined eating situations by patients with Bulimia Nervosa and Normal Controls. H.R. KISSILEFF, J.L. GUSS, L. BARTOSHUK, B.T. WALSH. St.Luke's/Roosevelt Hospital Center and Columbia University, New York, NY 10025 USA. Labeled magnitude scales are more sensitive to taste intensity differences at high concentrations of bitter solutions in tasters than is a 9 point category scale. In order to determine whether LMS scales might also produce more accurate assessment of fullness and other variables than the traditional VAS by reducing ceiling effects, six patients with bulimia nervosa (BN) and seven controls rated six feelings (hunger, full, sick, thirst, wanting a meal, and wanting dessert) for six imagined eating situations including the "largest meal" and a "normal dinner" on the VAS and LMS. Scales were 150 mm lines anchored by "not at all" and extremely for the VAS, and by "barely detectable" and "strongest imaginable situation of any kind" for the LMS (which also included four other labeled marks along the scale). Controls rated fullness significantly lower after a normal dinner on the LMS scale (45.7 mm) than on the VAS (104.1 mm), whereas patients with BN did not differ in their ratings on the two scales (LMS = 34.5, VAS = 32.2 mm). The difference between these differences (group x scale interaction = 60.7 mm \pm 28.2 SE was significant, $p = .03$). There was no significant difference in fullness between scales between patients and controls for their largest meal, and there were no other significant group x scale interactions for other situation-feeling combinations. These results suggest that with an actual meal the VAS may be a better instrument than LMS for measuring differences in fullness at intermediate, rather than excessive, intakes.

Appetitive ratings during a test meal measure effects of CCK on sickness and satiety. D.M. PRESS, J.L. GUSS, C. HAYNES, X. PI-SUNYER, H.R. KISSILEFF. This study was done to quantify the effect of CCK in combination with a varying preload on sensations of satisfaction or discomfort as a function of how much is eaten in a meal. Three normal-weight men were given a tomato soup preload (50g or 640g) and a slow intravenous drug infusion (saline or CCK at 168ng/min, for 30 min) on four separate occasions followed by a strawberry yogurt shake test meal. Eating was interrupted at 75g increments for subjects to make ratings on 150 mm line scales, anchored by "not at all" and "extremely", of feelings and sensations including "sickness" and "fullness". The combination of CCK with the large preload increased "fullness" during the latter stages of the test meal (mean = 94 and 93mm at 600g and 675g increments respectively) in comparison to the other three conditions, which were not different from one another (mean = 66.8 and 63.6 mm; drug by preload interaction, $p < 0.02$). With the 640 g preload, initial rate of fullness as a function of amount eaten was 8.7 mm/100 g (1.6 SED) higher than after saline whereas with the 50 g preload there was a non significant -2.2 mm/100 g (± 1.6 SED) difference, resulting in a significant interaction between CCK and preload (10.9mm/100g, $p = 0.003$). CCK compared to saline did not produce significant increases in 'sickness'. These results suggest that previous findings that CCK reduces total food intake may be due to increases in sensations of satisfaction rather than discomfort.

The Integration of Market and Sensory Information to Explore Consumers' Preferences for Full-fat and Reduced-fat Dairy Products. J. BOGUE. Department of Food Business and Development, University College, Cork, Ireland. Consumer food preference is an extremely complex phenomenon with many interacting variables influencing food preferences. Understanding consumer preferences for intrinsic and extrinsic product attributes has a significant role to play in successful food marketing. The aim of this research was to understand consumer preferences for lighter foods through the integration of market and sensory information. The research methodology consisted of 6 focus groups interviews and sensory analysis was carried out on a range of full-fat and reduced-fat milks, Cheddar-type cheeses and strawberry yoghurts. Quantitative Descriptive Analysis (QDA) was used to describe the sensory character of the products for flavour, texture and appearance attributes based on an agreed vocabulary by 12 assessors. Analysis of variance found significant differences between the products obtained from Principal Components Analysis of the sensory data. A total of 200 consumers expressed their preferences for the products on a nine-point hedonic scale. Internal preference mapping showed individual consumers' preferences for the products. Three segments of consumers with similar preferences for products were identified through Hierarchical Cluster Analysis (HCA). Consumer expectation of lighter foods was low and consumers had a higher preference for lighter milks and lighter strawberry yoghurts than lighter Cheddar-type cheeses. Socio-economic data obtained from the consumers highlighted interesting relationships between socio-economic variables and consumer preferences. Overall, the research illustrated how market and sensory information could be used to determine consumer preferences for food products. Effective integration of information from both techniques can be used to correctly position products and target products at specific consumer groups.

An Exploratory Study of Consumer Attitudes Towards Health-Enhancing Foods. J. BOGUE, D. SORENSON. Department of Food Business and Development, University College, Cork, Ireland. Health-enhancing foods have an important role to play in the alleviation, treatment and prevention of diseases. The objective of this study was to explore consumer attitudes and perceptions towards health-enhancing foods and their role in a healthy diet. Five focus groups were conducted with consumers from different socio-economic groups. Consumer knowledge of the importance of a healthy diet was not reflected in their dietary behaviour. Social, lifestyle, educational, preferences and situational factors exerted influences on food choice. Knowledge and awareness of the disease prevention concept, and its influence on dietary practices was greater for negative foods (low-fat), than for positive foods (health-enhancing foods). Awareness of specific health messages relating to fat and sodium intake explained certain health-related dietary behaviours. Participants preferred to consume dietary supplements over nutrient-enriched health-enhancing foods. There was limited awareness and knowledge of the health benefits of fish, fruit and vegetables. The majority of discussants were unaware of the health benefits of probiotics, fibre and fish oils, although fish oils were considered beneficial for health. Participants were unfamiliar and unaware of the health benefits of functional ingredients such as plant sterols, probiotics, Omega-3 oils and wholegrains. Consumers were sceptical of the health claims that appeared on a selection of food labels. Coherent and accessible dietary-related health messages communicating the benefits of health-enhancing foods, could have a positive influence on consumer purchase behaviour. Successful marketing and positioning of health-enhancing foods depends on differentiating health-enhancing foods from conventional foods, through communicating health claims that consumers understand and consider beneficial.

Communicating Food Risk Uncertainty with the Public. M. BRENNAN, C. RITSON, M. NESS, S. KUZNESOFF, L. FREWER*, S. MILES*. Department of Agricultural Economics and Food Marketing, University of Newcastle Upon Tyne, UK. *Institute of Food Research, Norwich, UK.

The concepts of risk and uncertainty have long been researched within the academic community. To date, the research has mainly concentrated on developing definitive characterisations of risk and uncertainty. Few consumer based studies have been conducted which examine lay concepts of food risk and uncertainty with respect to Food, whether they find it acceptable and how they wish it to be communicated to them. The aim of this project was to specifically examine lay concepts of risk and uncertainty when related to food information in particular. The project involved four main sections, combining a variety of both qualitative and quantitative methods, including a nationally representative questionnaire (N=1092). The research findings have been fourfold. It is clear that the UK public can articulate what the term uncertainty means to them, accept that uncertainty exists in some situations and can deal with the presence of uncertainty. It was also evident that the only effective method for communicating food risk and uncertainty to the public is through a policy of transparency and openness. Where uncertainty exists, the public want to be provided with all relevant information as soon as it is available and provided with reassurances from those regulating the industry that actions are being taken to investigate and deal with the uncertainty.

Comparison of low and high fat consumers in the UK Women's Cohort Study. C. GOLDING¹, J. CADE¹, C. LAWTON², D. GREENWOOD¹. ¹Nutrition Epidemiology Group, Nuffield Institute for Health, University of Leeds, Leeds, LS2 9PL, U.K. and ²School of Psychology, University of Leeds, Leeds, LS2 9JT, U.K.

Participants of the UK Women's Cohort Study (aged 35-69 years) have completed a baseline 217-item Food Frequency Questionnaire, which covers all the major sources of fat in the UK diet, including reduced-fat products (n=11,935). Low-fat consumers (LF) (94g fat/day). More LF had modified their diet due to being overweight (14%) and concern over healthy diets (12%) compared to HF (10% and 11% respectively). A greater percentage of LF believed they consumed small portions of food (17%) or were following low-fat diets (34%) and slimming diets (10%) compared to HF (15%, 19% and 6% respectively). A greater proportion of LF never drank alcohol (15%) compared to HF (12%). Body Mass Index and age were both higher in LF. Comparison of socio-demographic characteristics showed fewer LF lived with their partners (67%) compared to HF (74%). More LF had no academic qualifications (17%) compared to HF (12%) and fewer had professional occupations. A multiple logistic regression model was performed to determine predictors of low-fat consumption. The strongest predictors identified were typical portion size, currently following low-fat and slimming diets, marital status and socio-economic group. All differences described above were statistically significant at the 5% level. The findings of this study have important implications for future Public Health Policy and may be further used to achieve dietary change in high-fat consumers.

Adolescents' beliefs about the costs and benefits of additives and their presence in different foods. N.S. COULSON. Institute of Behavioural Sciences, University of Derby, Mickleover, DE3 5GX, England, UK. A total of 572 high school students aged 12-15-years-old completed an anonymous questionnaire concerned with the use of additives in food. Participants were asked to rate the frequency of consumption of 16 everyday food items and the amount of colourings, flavourings, preservatives and pesticide traces thought to be present in these foods. In addition, they also rated the importance of 8 factors (e.g. health, taste) in their food choices, and provided comparative evaluations of the concepts 'food with additives' and 'food without additives' in terms of these same eight attributes. The results indicated that the adolescents could discriminate between the 16 foods in terms of the chemicals they believed they contained, but these ratings were not affected by gender or age. When these estimates were weighted by the adolescents' own reported consumption, females scored lower than males on a measure of the assumed additive content of their diet. Own additive consumption was significantly associated with ratings of factors important in food choice as well as the comparisons of food with/without additives. However, the form of this association did not support standard expectancy-value formulations of attitude-behaviour relationships. Food with additives was seen overall as preferable in terms of ease of preparation and duration of freshness, but more problematic in terms of health and safety.

Social comparison and assumptions about personal dietary intake. A. OENEMA, J. BRUG. Department of Health Education and Promotion, Maastricht University. Social comparison and assumptions about personal dietary intake. Lack of awareness of personal dietary habits is a major barrier in contemplating dietary change. Optimistic bias in comparison with others was found to predict lack of awareness. To encourage people to change to healthier diets it is important to make them aware of their dietary risk behaviour. In order to make people aware it is important to change the way they compare themselves with others where diet is concerned. The present study was conducted to gain more insight into the process of diet-related social comparison. Specific research questions were: (1) who do people compare themselves with, (2) what characteristics of others are important in diet-related social comparison, and (3) which factors predict different social comparison patterns. Two telephone surveys were conducted, one for fat and one for fruit and vegetables. 384 respondents aged 18 to 67 participated in the surveys. Two thirds were female and one third were male. Response rate was 50%. Descriptive statistics and regression analyses were used to find social comparison patterns and their determinants. Descriptive statistics showed that partner, family and friends were the most common persons to compare with. People in the supermarket were important persons to compare with for fat, but not for fruit and vegetables. Eating habits, food purchase patterns, appearance and cooking patterns were the most important characteristics for comparison.

Bitterness of 6-n-propylthiouracil (PROP) associates with bitter sensations and intake of vegetables. V.B. DUFFY, M.N. PHILLIPS, J.M. PETERSON, L.M. BARTOSHUK. School of Allied Health, University of Connecticut, Storrs, CT, USA. Bitterness of PROP is one marker of genetic variation in taste. Those who taste PROP as exceptionally bitter (ie, supertasters) also taste a number of bitter compounds as more intense than do those who taste PROP as weakly bitter (ie, nontasters). As part of The Genetic Taste and Dietary Behavior Study, we are investigating the influence of PROP tasting on bitter sensations and preference for and intake of foods and beverages that may have bitter qualities. Seventy-five subjects who reported low cognitive restraint over eating used the general labeled magnitude scale (Green et al, 1993; Bartoshuk et al, in press) to rate: 1) bitterness and degree of liking/disliking of strong black coffee and grapefruit juice; and 2) bitterness of .0032 M PROP and .001 M quinine hydrochloride (QHCl). Vegetable intake was assessed by five, nonconsecutive food records and an interviewed food frequency survey. Individuals who tasted more PROP bitterness also tasted greater bitterness from QHCl, coffee and grapefruit juice and reported less liking for both beverages. Those who tasted greater PROP bitterness also reported lower intake of vegetables over five days (food records) and less frequent intake of green vegetables over one-year (frequency survey). These data support that PROP tasting influences bitter sensations and this in turn may influence preference for and intake of foods and beverages with bitter qualities. The correlation between PROP tasting and vegetable intake is nutritionally important, as greater intake of vegetables can reduce the risk of chronic diseases. (NRCGP/USDA 9603745 and NIH DC00283 supported).

PROP genetics interact with age and sex to influence food preferences. D.J. SNYDER, V.B. DUFFY, K. FAST, J.M. WEIFFENBACH, L.M. BARTOSHUK. Department of Surgery, Yale University School of Medicine, New Haven, CT. Food preferences predict diet, which contributes to overall health. Because many health problems are associated with aging, we are assessing how and why food preferences change across the lifespan. Using the general Labeled Magnitude Scale (gLMS; Green et al., 1993; Bartoshuk et al., 2000) adapted for hedonic measurement (Duffy et al., 1999), subjects (N=2690) rated preferences for 26 foods; factor analysis produced four groups showing age and sex effects. Preferences for high-fat foods increased between ages 15-40; younger women liked fat less. Meanwhile, sweet preferences declined with age for women but remained stable for men, even though sweet perception is constant across age. Men also maintained constant preferences for salty foods; these were elevated in women aged 20-60. Finally, bitter preferences rose with age, presumably since bitter taste perception declines. Interestingly, PROP (6-n-propylthiouracil) perception influenced food preferences by interacting with age and sex. All PROP supertasters liked bitters the least. Older nontaster women liked sweet foods the least, while middle-aged supertasting men liked high-fat foods the most. Overall, these effects may impact diet-mediated cardiovascular or cancer risk. Preference data for specific foods were idiosyncratic; some foods showed trends incongruent with their statistical food group, while many showed consistent effects at different ages that were in fact more robust. For example, preferences for milk chocolate dropped sharply in supertasting women after age 50, but declined more modestly in supertasting men after age 30. We conclude that the gLMS is a powerful tool for analyzing the complex interactions that characterize food preferences.

Genetically determined bitter perception influences human food preferences. M.C. GOLDSMITH, R.B. KANAREK. Tufts University, Medford, MA, USA. Previous research found individual differences in bitter flavor perception. Early studies linked this with dominant and recessive alleles. More recent work further separates individuals into categories of supertasters, medium tasters and nontasters. Supertasters rate sweet and capsaicin solutions as more intense than nontasters. The object of this experiment was to determine the affect of bitter perception on human food preferences. Seventy-two college students participated in the experiment yielding 22 bitter supertasters and 18 bitter nontasters. Bitter perception was determined by the detection of 6-n-propylthiouracil in liquid samples of increasing concentrations and taste bud densities observed by digital photographs of the subject's tongue. Intensity, preference and forced choice ratings were obtained on a set of foods and liquids. Dietary preferences and consumption frequency information was gathered via questionnaires. The results showed supertasters made greater distinctions between highly rated foods and the merely palatable compared to nontasters. The difference in the mean number of tolerable foods between the two groups was significant. Supertasters demonstrated negative reactions to a greater number of foods than nontasters. It is hypothesized that the enhanced perception of flavor for supertasters provides a greater saliency of stimuli for developing strong food preferences. Nontasters experience weaker sensations from food such that these individuals find more foods palatable and therefore have more diverse food preferences.

Food adventurousness clarifies the influence of PROP taster status on food preferences. N. ULLRICH, B.J. TEPPER. Department of Food Science, Rutgers University, New Brunswick, NJ USA. Taste sensitivity to the bitterness of 6-n-propylthiouracil (PROP) is inherited. PROP tasters are more sensitive to bitterness, sharpness and oral pungency, and show lower acceptance of bitter fruits and vegetables, strong cheeses, dark beers and chili peppers. However, reported differences in most studies are small, and inconsistent findings are common. Thus, other factors might influence the relationship between PROP status and food preferences. We studied the influence of 'food adventurousness' on food preferences of PROP tasters (n=135) and non-tasters (n=67). Subjects completed a 68-item food preference checklist. Food adventurousness was defined as the willingness to try new foods (1=never to 4=most of the time). The foods were separated into general categories (fruits, vegetables, condiments and alcohol) and factor analysis was used to create subgroups within these general categories based on liking. Separate analyses were conducted for tasters and non-tasters. The models accounted for 59-83% of the variance in the data, and the factor groupings were similar for tasters and non-tasters. The mean number of foods 'liked' for each subgroup was calculated for each subject and differences between those reporting high or low food adventurousness were examined. Tasters reporting high food adventurousness liked all the food subgroups more than tasters reporting low food adventurousness ($p < 0.05-0.001$), except for one subgroup, olives. Greater food adventurousness had no effect on non-tasters. These data suggest that food adventurousness had different effects on liking of foods by tasters and non-tasters and only tasters reporting low food adventurousness showed the characteristic rejection of strong tasting foods.

The contribution of the number of choices to intake and preference. M.G. TORDOFF. Monell Chemical Senses Center, Philadelphia, PA 19104, USA. Since the 1930's, the standard method of measuring taste solution acceptability in animals has been the two-bottle preference test, which involves a choice between a taste solution and water. Intake of the taste solution is believed to reflect physiological and taste-related hedonic factors. We were surprised to find that mice drank significantly more taste solution (saccharin, NaCl, citric acid, or quinine) when given two bottles of taste solution and one of water than two bottles of water and one of taste solution. In follow-up work, mice always received 6 bottles, with 1, 2, 3, 4, or 5 containing 75 mM NaCl and 5, 4, 3, 2, or 1 containing water, respectively. There was a monotonic increase in NaCl intake as the number of bottles containing NaCl increased. Preference ratios for NaCl ranged from ~50% when mice received 1 bottle of NaCl and 5 of water to >95% when they received 5 bottles of NaCl and 1 of water. This experiment was replicated using 10% ethanol as the taste solution. In related work, we examined the macronutrient intakes of rats given separate cups of protein, carbohydrate and fat. Rats given three "extra" cups of carbohydrate or fat ate more of these nutrients at the expense of the others. Thus, the availability of a nutrient can profoundly influence its intake. Intake in "taste" and "self-selection" experiments involves cognitive, as well as physiological and hedonic factors.

Motivation of horses for fiber. J.B. ELIA, K.A. HOUPPT, T.R. HOUPPT. Department of Biomedical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, New York, USA. The natural diet of free-ranging horses is grass, which is typically high in fiber and calorically dilute, however diets for high performance domestic horses are often low in fiber and calorically dense. To determine the motivation of horses for fiber a progressive ratio technique was used. The horses were placed in a special stall and operantly conditioned to press a panel with their nose to open a door, which allowed them access to a food reward. Six mares were each tested under two dietary conditions: ad lib. grass hay (30% fiber), and a complete pelleted feed (CPF) (Bonanza®, 24%). Each trial lasted three weeks, with a one-week transition between diets. The mares were tested twice weekly in the stall with a 100g food reward of either the CPF or hay. They worked through a progressively increasing ratio (1,2,4,7,11...) until they extinguished or refused their food reward when released. When fed CPF the horses worked for 750±405 (SE)g CPF and when fed hay they worked for 1505±367g CPF. When fed hay the horses worked for 0±0g hay and when fed CPF they worked for 822±226g hay. These results are indicative of a greater motivation for fiber when fed a low fiber diet as well as a greater motivation for a novel than a familiar diet. The horses were also observed to determine dietary induced differences in behavior.

Melanin Concentrating Hormone Stimulates Water Intake Independent of Food Intake. E.L. AIR, D.J. CLEGG, R.J. SEELEY, S.C. WOODS. Department of Psychiatry, University of Cincinnati, Cincinnati, OH 45267.

Melanin Concentrating Hormone (MCH)-producing neurons are located primarily in the lateral hypothalamus. Mice with targeted deletion of MCH are hypophagic and lean, and exogenous intraventricular administration of MCH elicits acute increases in food intake. After administering 5 µg of MCH i3vt, we observed a significant increase in food intake (400%, $p < 0.05$). In addition, copious water drinking accompanied the increase in food intake in MCH-treated rats, and the ratio of water intake/gram of food intake was elevated in rats treated with MCH compared to both vehicle-treated rats and rats treated with the orexigenic compound, AgRP. To determine whether the increase in water intake is secondary to the increase in food intake, we administered MCH to rats with access to water but no food for 2 hours. During the first hour following injection, rats that received MCH consumed 3.0 mL, while those that received saline consumed 0.8 mL ($p < 0.05$). When food was returned two hours later, 60-min food intake was increased in MCH-treated rats (not significant). There was no difference in water intake during this same period. When AgRP was given in the same paradigm, there were no differences in water intake between AgRP and saline-treated animals during the food deprivation period. These data imply that, in contrast to AgRP, MCH elicits increased water intake independent of food intake. These results may help explain historical data linking activity of the LH with water as well as food intake.

Melanocortin (MC) 3/4 receptor agonist reduces meal size in rats. A.V. AZZARA, J. SOKOLNICKI, G.J. SCHWARTZ. WMC Cornell Univ. Bourne Lab, White Plains, NY 10605 USA. Central administration of MC3/4 receptor agonists inhibit feeding, but their effects on meal size and pattern in the overall reduction of food intake have not been assessed. To characterize MC3/4R—agonist induced feeding suppression and to evaluate the potential for MC interaction with small intestinal meal-related negative feedback signals, we measured meal patterns and feeding microstructure after third intracerebroventricular (i.c.v.) administration of sub and suprathreshold feeding suppressive doses (0.01, 0.1 nmol/3 microliter) of the MC3/4 agonist MTII in male Sprague Dawley rats. We also examined ingestive behavior during a 60 min daytime access to 12.5% glucose, following 4.4 ml/10 min duodenal 12.5 % glucose preloads alone or in combination with i.c.v. MTII. In overnight pellet tests MTII (0.1 nmol) suppressed total intake, reduced meal size, and increased meal number. During glucose test meals: 1) duodenal glucose preloads reduced the initial lick rate, total intake, and cluster size, 2) MTII alone had no effect, and 3) MTII did not increase the effect of duodenal glucose. These data demonstrate that: 1) MTII does not potentiate the feeding suppressive effects of duodenal glucose, and 2) MTII suppresses food intake by reducing meal size, suggesting that melanocortin system modulates the processing of alimentary tract signals generated by the consumption of food. Supported by NIH-DK47208.

Injection of the peptide-toxin-complex neuropeptide Y (NPY)-saporin (NPY-sap) into the paraventricular nucleus of the hypothalamus (PVH) causes hyperphagia and obesity in rats. K. BUGARITH, S. RITTER, T.T. DINH, D.A. LAPPI*. Programs in Neuroscience, Washington State University, Pullman, WA 99164-6520 USA. * Advanced Targeting Systems, San Diego, CA USA. Animal studies have shown NPY to be one of the most potent stimulators of feeding when administered into the brain. We injected either NPY-sap or unconjugated saporin (sap) bilaterally into the PVH and subsequently tested rats for feeding behaviour and body weight. NPY-sap is a conjugate of the peptide NPY and saporin, a plant toxin that inactivates ribosomes. It is proposed that NPY-sap specifically binds to and lesions cells containing the NPY receptor, but studies are currently being done to verify this proposed mechanism. At surgery there was no difference in body weights between the groups. Three weeks after the injection the NPY-sap rats (501.33g ± 15.84g) were much heavier than the sap rats (371g ± 11.79g). However, there was no difference in 2DG-induced, baseline or overnight food deprivation-induced feeding between the groups. Ten weeks after the injection, the NPY-sap rats (654.3g ± 39.04g) were even heavier than the sap rats (410.6g ± 15.29g). Further, daytime (0800 – 1700H) food intake was approximately twice as much in NPY-sap (9.53g ± 0.996g) as in sap (5.74g ± 0.476g) rats. Overnight feeding (1700 – 0800H), 2DG-induced and overnight food deprivation-induced feeding was not different between the groups. However, NPY-sap rats now ate significantly more than sap animals during baseline feeding (6.57g ± 0.873g and 3.08g ± 2.97g respectively). Results indicate that the lesion produced by NPY-sap results in hyperphagia and obesity in rats.

Interactions between melanin concentrating hormone (MCH) and c-fos after agouti-related protein (AgRP) administration. A.J. JACKMAN¹, D.J. CLEGG², J.A. REED², R.J. SHELDON¹, S.C. WOODS², R.J. SEELEY². Procter & Gamble Pharmaceuticals, Cincinnati, OH¹; University of Cincinnati, Cincinnati, OH².

AgRP is an endogenous antagonist of the CNS melanocortin 3- and 4-receptors (MC3/4R) and is expressed in a discrete neuronal population in the arcuate nucleus (ARC). Exogenous AgRP potently increases short-term food intake, and the hyperphagia from a single bolus injection to the 3rd-cerebral ventricle (i3vt) injection lasts for up to 6 days. While the precise mechanism for this effect is unclear, it has been reported that c-fos protein is increased in the lateral hypothalamus (LH) 24 hours after injection of AgRP. We hypothesized that AgRP signaling through projections from the ARC to the LH drives changes in gene expression in the LH, including upregulation of c-fos and potential target genes that mediate orexigenic effects, such as melanin-concentrating hormone (MCH). To assess this, adult male Long-Evans rats were injected i3vt with 1 nmole AgRP one hour before dark, allowed to feed for 17 hours, and fasted for 6 hours. Brain tissue was then harvested for Northern blot analysis and immunohistochemical staining of whole hypothalamus. MCH mRNA was increased in AgRP-treated compared to saline-treated rats, while c-fos mRNA was unchanged. Immunohistochemical staining for MCH was observed in the LH of AgRP-treated rats. To determine whether the observed MCH expression is mediated by AgRP-induced c-fos, we will dual-label for MCH and c-fos at several timepoints in the fed and fasted states.

Agouti-related protein (AgRP) increases melanocortin-concentrating hormone (MCH) expression in the lateral hypothalamus. J.A. REED, D.J. CLEGG, A.J. JACKMAN, S.C. WOODS, R.J. SEELEY. Department of Psychiatry; University of Cincinnati; Cincinnati, Ohio.

Agouti-related protein (AgRP) signaling arising from neurons localized in the arcuate nucleus is known to stimulate an orexigenic response. Exogenous administration of a single bolus injection of AgRP into the 3rd-cerebral ventricle (i3vt) markedly increases food intake in rats for up to six days. While orexigenic peptides in the hypothalamus are thought to contribute to both the short- and long-term response to AgRP, little is known about downstream pathways mediating the hyperphagic response. Adult male Long-Evans rats received 1 nmole AgRP or saline i3vt 1 hour prior to onset of the dark. Animals were allowed 17 hours of access to food, fasted for the next 6 hours, and then killed. Brains for RNA analyses were immediately harvested and frozen. RNA samples were isolated from whole hypothalamus, and 20 mg total RNA was electrophoresed for Northern blotting. The blot was sequentially probed for pro-opiomelanocortin (POMC), neuropeptide Y (NPY), and melanocortin-concentrating hormone (MCH) mRNA species. There were no differences in POMC or NPY mRNA accumulation between AgRP- and saline-treated rats. In contrast, MCH mRNA was increased in AgRP-treated rats compared to the saline-treated group. Immunohistochemistry for MCH in sections from perfused rat brains identified positively-stained regions within the lateral hypothalamus (LH). These findings suggest that MCH signaling in the LH plays a role in the long-term hyperphagic response to AgRP signaling in the arcuate nucleus.

Chronic pCPA down-regulated gene expressions of CART in the rat hypothalamus. S.H. CHOI¹, B.S. KWON¹, H.T. LEE¹, D.G. KIM², J.W. JAHNG². ¹Department of Animal Science, Konkuk University, Seoul, Korea. ²Department of Pharmacology, BK21 Project for Medical Science, Yonsei University College of Medicine, Seoul, Korea.

It was reported that the mice intracerebroventricularly injected with cocaine-amphetamine-regulated transcript(CART) showed behavioral changes resembled with the typical behavioral alterations found in the mice carrying disorders in the brain serotonergic (5-HT) system. To find a molecular correlation between CART and 5-HT, the mRNA levels of CART after the injections of para-chlorophenylalanine(pCPA, 300mg/kg i.p., single injection or daily for three consecutive days) were examined in the rat brains by in situ hybridization using the rat CART cDNA probe cloned in our laboratory. Systemic administrations of pCPA, a potent inhibitor of tryptophan hydroxylase(TPH), the rate limiting enzyme of 5-HT biosynthesis, acutely depletes the brain 5-HT in the hypothalamus and decreases the mRNA level of serotonin transporter(5-HTT) in the dorsal raphe nucleus(DRN), which reuptakes terminal 5-HT. In our results, the mRNA levels of CART significantly decreased in the arcuate nucleus, paraventricular nucleus, and lateral hypothalamic nucleus by three days of daily injection with pCPA, and there was not a noticeable change detected 24 hrs after the single injection. The message levels of 5-HTT in DRN decreased by both the single and three days of injections. These results suggest that there maybe a molecular correlation between CART and 5-HT in responding to the stimuli, probably in the same direction each other.

Modulation of mu opioid agonist-induced feeding in the nucleus accumbens shell by excitatory and inhibitory amino acids. R.J. BODNAR, J.A. ECHO, N. LAMONTE, T.F. ACKERMAN, C.K. SINGH, V. ZNAMENSKY. Queens College, CUNY, New York, NY. The nucleus accumbens shell is a central site of action for ingestive responses elicited by agonists and antagonists of endogenous opioid peptides, excitatory amino acids and GABA in rats. The selective mu opioid receptor agonist, DAMGO, significantly increases food intake following administration into the nucleus accumbens shell which is reduced by pretreatment with selective opioid and dopaminergic receptor antagonists. The present study examined whether pretreatment with selective GABA_A (bicuculline) or GABA_B (saclofen) antagonists or selective excitatory amino acid agonists (NMDA, AMPA) altered DAMGO-induced feeding in the nucleus accumbens shell. Bicuculline significantly and dose-dependently enhanced DAMGO-induced feeding in the nucleus accumbens shell without altering intake per se. In contrast, saclofen significantly and dose-dependently reduced DAMGO-induced feeding in the nucleus accumbens shell with altering intake per se. Both GABA_A (muscimol) and GABA_B (baclofen) agonists induced feeding in the nucleus accumbens shell which were selectively blocked by their corresponding antagonists; naltrexone pretreatment blocked GABA_A, but not GABA_B agonist-induced feeding. Both NMDA and AMPA dose-dependently stimulated feeding in the nucleus accumbens shell which was blocked by naltrexone pretreatment. DAMGO-induced feeding was significantly enhanced by co-treatment with AMPA, but not NMDA in the nucleus accumbens shell; this selective enhancement was blocked by pretreatment with general and mu opioid antagonists. These data indicate important and differential reliance of opioid-induced feeding upon both inhibitory and excitatory amino acid receptors in the nucleus accumbens shell.

Evidence of increased hypothalamic NPY release in preweaning fa/fa rats. A. STER, T. KOWALSKI, M. DUBE, S. KALRA, G. SMITH. Bourne Laboratory, Weill Med. College of Cornell Univ. and NY-Presbyterian Hosp. White Plains, NY 10605.

Of the many consequences of the decreased leptin action produced by the mutation of the leptin receptor in the Zucker (fa/fa) rat, increased action of hypothalamic NPY has been proposed as a major factor in the pathophysiology of the syndrome. Using a developmental strategy to test this hypothesis, we showed that significantly more arcuate n. NPY was expressed in fa/fa pups than in +/+ pups on postnatal day (P) 2 and throughout the preweaning period (Kowalski et al. *Physiol. Behav.* 67: 521, 1999), and that hyperphagia first appeared on P 12 (Kowalski et al *Am J Physiol* 275: R1106, 1998). To test the hypothesis further, we used a specific radioimmunoassay to measure the concentration of hypothalamic NPY peptide in fa/fa and +/+ pups on P 9, 10, and 12. The major results were that the concentration of NPY was not significantly different between the genotypes on any day, but there was a significant decrease in NPY with age in fa/fa pups (NPY (pg/ug protein) on P9, P10 and P12 in +/+ = 22±5, 23±5, 22±4; in fa/fa = 29±5, 23±5, 14±2). The combination of increased NPY message and decreasing concentration of NPY peptide in fa/fa pups with age is consistent with, but does not prove, increased release of hypothalamic NPY in fa/fa pups before the emergence of hyperphagia on P12. These results provide further support for the importance of hypothalamic NPY in the phenotypic expression of the hyperphagia of the fa/fa genotype.

Central oxytocin (OT) activates hindbrain glucagon-like peptide-1 (GLP-1) neurons in rats. E. ROTHE, L. RINAMAN. Department of Neuroscience, University of Pittsburgh, Pittsburgh, PA 15260. Intraventricular administration of either OT or GLP-1 potently inhibits feeding in rats. Results from recent pharmacological and behavioral studies suggest that the anorexigenic effects of OT may occur, at least in part, through activation of central GLP-1 signaling pathways. Indeed, light microscopy suggests that GLP-1 neurons receive direct synaptic input from OT-positive fibers. The present study sought to determine whether central administration of OT activates GLP-1-positive neurons in the caudal medulla. Male Sprague-Dawley rats were anesthetized and implanted with chronic indwelling lateral ventricular guide cannulas. After 2 weeks of recovery and handling acclimatization, rats received intraventricular infusion of 5 microL sterile vehicle containing 0, 1, or 5 microG of synthetic OT (Bachem). Rats were anesthetized 60-90 min later and perfused transcardially with fixative containing 4% paraformaldehyde and 2% acrolein. Brains were cryoprotected, frozen, and cut coronally at 30 microns. Sections were incubated in rabbit anti-cFos (provided by Dr. Philip Larsen; 1:50K), and blue-black nuclear cFos labeling was produced using Elite Vectastain reagents and a nickel-DAB reaction product. Sections were then incubated in rabbit anti-GLP-1 (Peninsula, 1:10K), and reacted with plain DAB to produce brown cytoplasmic immunolabeling. Quantitative analysis of dual GLP-1 and cFos immunolabeling demonstrated that OT caused a dose-related activation of hindbrain GLP-1 neurons. These data are consistent with the view that central administration of OT produces its anorexigenic effects, in part, by activating endogenous central GLP-1 signaling pathways. Supported by MH59911.

Synergistic interactions with meal-taking underlie longer-term effects of urocortin. S. MARKISON, H.J. GRILL, J.M. KAPLAN. Department of Psychology, University of Pennsylvania, 3815 Walnut Street, Philadelphia, PA 19104, USA.

Urocortin (UCN), a CRH receptor agonist, delivered to the 4th ventricle suppresses ingestive behaviors including intraoral glucose intake and 24-h chow intake. We had suggested that the long-term intake effect of UCN might be attributed in part to an interaction between short-term drug effects and inhibitory signals arising from meals ingested shortly after drug delivery. We tested this idea using a novel two-meal intraoral intake paradigm that allows precise control of meal initiation in non-deprived rats. In an earlier experiment, we used a relatively short experimental time frame (2 h) and found no such interaction. In the current experiment, we expanded the time frame to 6 h. When rats were tested 30 min after treatment, UCN (3 µg) reduced intraoral intake from the vehicle baseline (30 to 19 ml, respectively). The UCN effect at 6 h, however, depended on whether an initial meal was delivered. When rats were tested twice, 30 min and 6 h after UCN treatment, intake was suppressed by ~11 ml, at each time point, relative to vehicle treatment. When rats were tested just once 6 h after UCN treatment, the drug effect was not significant. These results suggest that UCN produces a relatively short-lived anorectic effect that can be amplified and/or carried forward in time by an intervening meal. The nature of the meal-related signals that synergize with UCN are not known. Supported by: DK-42284 and DK-21397

Hypocretin-1 (orexin-A)-induced feeding is dependent on central opioidergic pathways. D.C. SWEET, A.S. LEVINE, C.M. KOTZ. Minnesota Obesity Center, Veterans Affairs Medical Center, Minneapolis, MN 55417.

We have previously shown that lateral hypothalamic (LH) injection of hypocretin-1 (Hcrt-1) stimulates feeding in rats. Others have shown that a neuropeptide-Y (NPY) blockade suppresses Hcrt-1-induced feeding, and a central opioid blockade suppresses NPY-induced feeding. We examined the deductive hypothesis that Hcrt-induced feeding is opioid-dependent, and that opioid inhibition would therefore suppress Hcrt-1-induced feeding. To test our hypothesis, rats received vehicle or naltrexone (NTX, non-specific opioid antagonist) either peripherally (i.p., 5.00 mg/kg), intracerebroventricularly (i.c.v., 200 mg), or into the LH (25 mg). Rats were then injected with vehicle or Hcrt-1 (1.00 nmol) in the LH. Pretreatment with i.p. NTX suppressed Hcrt-1-induced feeding at 2 h (1.0 ± 0.3 g compared to 2.2 ± 0.3 g when treated with Hcrt-1 alone, p = 0.002, and 1.0 ± 0.3 when injected with vehicle alone, p = 0.879). Pretreatment with i.c.v. NTX partially suppressed Hcrt-1-induced feeding at 2 h (2.2 ± 0.6 g compared to 3.9 ± 1.1 g when treated with Hcrt-1 alone, p = 0.056, and 0.5 ± 0.5 when injected with vehicle alone, p = 0.062). LH pretreatment with NTX did not affect Hcrt-1-induced feeding. To verify that the inhibition was due to central opioid blockade, vehicle or naloxone methiodide (NLX-MeI, non-specific opioid antagonist, does not cross the blood-brain barrier) was injected i.p. (5 mg/kg), and then vehicle or Hcrt-1 was injected into the LH. NLX-MeI did not affect Hcrt-1-induced feeding. We conclude that an opioid receptor blockade at a central site other than the LH decreases Hcrt-1-induced feeding.

The distribution of GLP-1/GLP-2 neurones in the NTS projecting to the PVN and/or DMH. M. TANG-CHRISTENSEN, M. HANSEN, A.W. HOLST, L.K. LARSEN, P.J. LARSEN, N VRANG. Laboratory of Obesity Research, CCB, 2750 Ballerup, Denmark. The pre-proglucagon derived peptides, Glucagon-Like Peptide-1 and Glucagon-Like Peptide-2 (GLP-1 and GLP-2) are both involved in a wide variety of peripheral functions, such as glucose homeostasis, gastric emptying, insulin secretion and the regulation of food intake. In the central nervous system, processing of pre-proglucagon is similar to that of the gut yielding glicentin, GLP-1 and GLP-2. The Glucagon-Like Peptides are exclusively produced in the non-catecholaminergic, leptin-receptor positive cells of the nucleus of the solitary tract. We have previously shown that GLP-1 constitutes a widespread multi-targeting transmitter system whereas the GLP-2 system constitute a distinct projection system connecting the nucleus of the solitary tract with the dorsomedial hypothalamic nucleus (DMH) being the only hypothalamic nucleus expressing GLP-2 receptor mRNA. In a subsequent set of experiments we have mapped the distribution of the GLP-2 receptor throughout the rat brain, which will be presented at the meeting. Furthermore, we have by use of double retrograde tracing experiments examined the GLP containing pathways innervating the paraventricular and dorsomedial hypothalamic nuclei. These studies reveal a partial overlap between the GLP containing neurones that innervate the PVN and DMH respectively. Currently we are examining if this population of neurones are activated, by use of c-Fos, after ip administration of LiCl.

Agouti-related protein and leptin in regulation of body weight. M.M. WIRTH, S.Q. GIRAUDO. Minnesota Obesity Center, V.A. Medical Center and University of Minnesota. Recent studies have suggested that the regulatory effects of leptin on food intake and body weight are mediated in part by the melanocortin agonist a-MSH. Agouti-related protein (Agrp) has been shown to play an important role in food intake and body weight by inhibiting the melanocortin receptors. It is therefore conceivable that Agrp can antagonize leptin action by this mechanism. We examined the effects of Agrp administered into the dorsal medial hypothalamus (DMH) on the satiety effects of leptin. Following 16 h food deprivation, rats received (via cannula) 0.9% saline or 100 pmol Agrp along with an i.p. injection of 0.5 mg/kg body weight of leptin or saline. After injection the animals had free access to pre-weighed chow. Food intake and body weight measurements were collected for 24 h. Co-injection of Agrp abolished the leptin-induced decrease in food intake relative to control rats. Body weight regain was inhibited significantly in rats treated with leptin. Treatment with Agrp reversed the leptin-induced inhibition of body weight change. These results suggest that Agrp can also block the leptin-induced increase in energy expenditure. The present results suggest that Agrp is able to antagonize leptin action by blocking the hypothalamic a-MSH/MCR signaling activated by leptin. In addition, Agrp seems to decrease energy expenditure because its effects on body weight cannot be completely accounted for by its effects on food intake. Supported by the Dept of Veterans Affairs and NIDDK DK 50456.

The blocking effect of leptin activity on melanin concentrating hormone in regard to food consumption in rats. M.M. VAZQUEZ, A. JONES. Joint Science Department, The Claremont Colleges, Claremont, California.

Previous studies have shown that the central administration of leptin, the obesity gene product synthesized in adipose tissue, reduces food consumption in rats. The central administration of melanin concentrating hormone (MCH), a target of leptin signaling, has been confirmed to be an orexigenic agent and has been proposed as a central stimulator of food intake. MCH has also been known to decrease food intake by decreasing the level of melanin concentrating hormone. The present study attempts to block the effects of leptin activity on MCH regarding food consumption in rats. Based on the hypothesis that co-administration of leptin and MCH injections will reverse the appetite suppressant effects of leptin and lead to normal food consumption in rats, eight rats were given injections of leptin, and leptin/MCH on two separate days. Food consumption was measured and compared to normal food consumption (baseline). The results indicate that MCH can indeed block the effects of leptin and cause relatively normal food consumption.

Neuroanatomical distribution of Receptor Activity Modifying Proteins 1-3 (RAMPs) in rat brain by in situ hybridization. M.D. WORTMAN, P. RUSHING, S.C. WOODS, R.J. SEELEY. Department of Psychiatry, University of Cincinnati College of Medicine, Cincinnati, OHIO 45220. Members of the calcitonin gene-related peptide (CGRP) family include peptides that have roles in the regulation of a variety of physiological processes from vasodilatation to food intake. Historically, receptors for peptides in this family had been studied in in vivo and in vitro preparations because isolation of specific receptors eluded investigators. It had been known for some time that both the calcitonin receptor (CTR) and the calcitonin receptor-like receptor (CRLR) were involved in CGRP-family signaling. In 1998 the three known RAMPs were first described and it was demonstrated that specific combinations of RAMPs and CTR/CRLRs led to specific receptors for the CGRP-family peptides. Because peptides from the CGRP family including amylin, adrenomedullin, salmon calcitonin and others have described influences on food and water intake, we decided to map the neuroanatomical distribution of the three RAMPs in rat brain. RESULTS: RAMP1 and 3 have an identical pattern of distribution with the highest level of expression in the dorsal thalamus and lateral geniculate. RAMP2 has a completely different pattern of distribution with highest, but still relatively low, expression in the hippocampus. All three RAMPs have distribution throughout the brain and extend into the brainstem.

Hyperphagia develops before shifts in corticosterone, insulin or leptin in mice with disruption of the serotonin 2C (5-HT_{2C}) receptor. S.F. AKANA, M.F. DALLMAN. Department of Physiology, University of California San Francisco, San Francisco, CA 94143-0444.

Mice with disruption of the 5-HT_{2C} receptor (KO) express late-onset hyperphagia with subsequent hyperinsulinemia and hyperleptinemia resulting in mid-life obesity compared to their wildtype (WT) controls (Tecott, 1995). We were interested in determining whether the hyperphagia or alterations in metabolic hormones were the first manifestation of the phenotype. We found that the KO phenotype is very sensitive to stress and developed a protocol to measure urinary peptide hormones to enable noninvasive, repeated measurement of hormones. We designed a metabolism cage study of WT and KO male mice (n=7/grp) at a prehyperphagic age (9-10 weeks) and again when hyperphagia was first expressed (13-14 weeks). Daily body weight, food intake and urinary measures of corticosterone, insulin and leptin were determined over a three-day basal period following a 72-hour adaptation interval at both the prehyperphagic and hyperphagic stage. Body weight and caloric efficiency were identical between WT and KO at both ages whereas there was a trend towards increased food intake in the KOs at 9-10 weeks (p=0.09) and hyperphagia was established at 13-14 weeks (p=.0053). By contrast, there were no genotype differences in urinary corticosterone, insulin or leptin at either age, no increase of corticosterone with age, but an increase of insulin and leptin with age. We conclude that in 5-HT_{2C} receptor KOs, the first manifestation of the phenotype is a hyperphagia, which is not accompanied by changes in caloric efficiency or shifts in corticosterone, insulin or leptin. (supported by MH34149 and DK28172.)

Evaluation of endocrine function in mice with measurement of urinary hormones. S.F. AKANA, M.F. DALLMAN. Department of Physiology, UCSF, San Francisco, CA 94143-0444.

We tested whether urinary hormones are useful measures of stress responses in mice by measuring corticosterone, testosterone, insulin, leptin and ACTH before, during and after the metabolic stress of 24 hours starvation and refeeding. Male, 9-week-old C57Bl/6 mice were allowed 72h to adapt to metabolic cages with daily measurement of body weight, food and water consumption and collection of urine. After a further 24h baseline measurement (fed), mice were fasted by removing all food for 24h (fast); this was followed by reintroduction of food for 24h (refed). Fasted mice lost a mean of 3.93 grams (p=0.0001) from an initial weight of 24.70 ± 0.47 grams; this weight was regained with return of food (p=0.0001). When permitted to refeed, mice ate 47% more food than on their control fed day. Consequently, caloric efficiency increased in the refed state. Mice responded to fasting with increases in 24 hour urinary corticosterone (p=0.0001), a nonsignificant increase in ACTH excretion, and decreases in insulin, leptin and testosterone excretion (all p=0.0001). Upon refeeding, corticosterone, insulin, leptin and testosterone excretion returned to basal values. All the urinary hormones respond in a profile appropriate to known secretory responses to fasting and resupply upon refeeding. The measurement of urinary hormones in mice provides an effective method of multiple endocrine system assessment that does not require handling, blood sampling or death of the animal. Moreover, this assessment can be repeated across time, allowing documentation of age-related changes in the same normal or genetically manipulated mice. (supported by MH34149 & DK28172).

Spelling 'stressed' backwards: Plasma corticosterone (B) and sucrose drinking interact in a negative feedback loop during chronic stress M.E. BELL, K.D. LAUGERO, M.F. DALLMAN. University of California San Francisco, California, USA
Chronic cold stress alters food intake and increases appetite for sucrose. We investigated the interaction of drinking sucrose and response to chronic stress. Male rats were adrenalectomized (ADX), implanted with 30mg (LowB, n=14) or 100mg(HighB, n=9) B pellets, and given 0.5%NaCl to drink. 18 rats were sham ADX. A second bottle containing 30% sucrose or tap water was offered immediately after surgery (d1-d5), removed for 3d, and returned when all animals were placed in cold (4°C; d8-12). All intakes were normalized to body weight. All rats offered sucrose drank it and simultaneously decreased food intake. At RT, all groups with sucrose drank similar amounts and caloric intake was increased in sucrose drinkers compared to tap drinkers. In the cold, HighB and shams increased, but LowB maintained sucrose intake; food intake was decreased in sucrose drinkers and in LowB+tap in cold. Caloric intake in cold in LowB +sucr was similar to intake in shams, and greater than in LowB+tap rats. Body weight gain and caloric efficiency in the cold were decreased in LowB+tap rats compared to all other groups. As previously reported in unoperated rats, plasma B levels were significantly lower in Sham+sucr compared to Sham+tap. This suggests that sucrose drinking and B may interact in a negative feedback loop wherein sucrose drinking decreases plasma B and plasma B increases sucrose drinking during chronic stress. Our results support recent reports that dietary selection is one method for coping with chronic stress (Nutrition 16:886-893). Supported by NARSAD and DK28172.

Chronic social stress effects on body weight. K.L.K. TAMASHIRO1, C. MARKHAM2, D.C. BLANCHARD2, R.J. BLANCHARD2, R.R. SAKAI1. 1Dept. of Psychiatry, Univ. of Cincinnati Medical Center, Cincinnati, OH 45267, USA. 2Bekey Laboratory of Neurobiology, Univ. of Hawaii, Honolulu, HI 96822, USA.

The Visible Burrow System (VBS) allows for mixed sex rat colonies (4 males and 2 females each) to be housed for extended periods of time during which animals form a dominance hierarchy. This dominance hierarchy provides a variety of behavioral and physiological characteristics that is consistent with psychosocial stress. Subordinate rats (SUB) housed in the VBS exhibit a significant decrease in body weight compared with dominant (DOM) and control animals (CON). This decrease in body weight begins immediately following colony formation and is maintained throughout housing in the VBS. Once removed from the VBS, SUB animals increase their body weight but do not recover to levels comparable to DOM and CON. This body weight difference is accentuated when the animals are subjected to three repeated cycles of VBS housing followed by a recovery period. Neuroendocrine measures indicated that SUB had high levels of plasma corticosterone and low levels of plasma testosterone compared to DOM and CON. In addition, carcass analysis revealed a significantly low percentage of body fat in SUB compared to CON that positively correlated with plasma leptin levels. These data suggest that neurochemical changes associated with chronic social stress influence systems involved in food intake and body weight regulation. Supported by: NSF, The Guggenheim Foundation, and NARSAD.

Time course of estradiol's inhibitory effect on meal size in female rats. M.C. MURPHY, M. BILODEAU, J.M. BARRANCO, L.A. ECKEL. Program in Neuroscience, Florida State University, Tallahassee, FL 32306.

Estradiol decreases food intake (FI) in female rats by a selective decrease in meal size (MS). We investigated the time course over which estradiol exerts this effect. Beginning on diestrus 1, meal patterns were examined in 8 female rats for 2 ovarian cycles. Rats were then ovariectomized (OVX) and, beginning 21 days later, a within-subjects counterbalanced design was used to administer varying doses of estradiol (0, 2, 4 µg). Meal patterns were monitored in OVX rats prior to and after hormone replacement. During estrus, rats displayed a 34% decrease in dark MS and a non-compensatory increase in dark meal number (MN). Light phase meal patterns were not influenced by the ovarian cycle. OVX rats displayed a stable 36% increase in dark MS and a 25% increase in light MS. Within 17 days, a compensatory decrease in MN decreased daily FI to pre-surgical levels. Estradiol treatment produced a dose-dependent decrease in dark MS. Light MS was unaffected. Both doses of estradiol reduced MS beginning 48 h after hormone treatment, however, MS remained suppressed for 5 days after the 4 µg dose and for 1 day after the 2 µg dose. These data demonstrate that 1) estradiol decreases FI by a selective decrease in dark MS; 2) the absence of estradiol permanently increases both dark and light MS; 3) the inhibitory effects of exogenous estradiol on MS are only expressed 48 h after hormone treatment. We conclude that estradiol acts via a genomic mechanism to reduce MS in female rats.

Effect of estradiol on hypothalamic dopamine and serotonin in Fischer -344 rats. M. VARMA, L. ZHANG, M.M. MEGUID. Neuroscience Program, Department of Surgery, SUNY, Upstate Medical University, 750 East Adams Street, Syracuse NY 13210. Eating disorders are more common in females in whom estrogens modulate body weight, meal number (MN) and meal size (MZ) whose product equals food intake (FI) $FI = MN \times MZ$. We previously have shown that changes in hypothalamic dopamine (DA) and serotonin (5HT) influence MN, MZ and hence FI via their effect on lateral (LHA) and medial hypothalamus (VMN). We proposed that estrogens effect FI via changes in hypothalamic monoamines and its receptors. Methods: We measured hypothalamic DA, and 5HT via in-vivo microdialysis, and DA receptors D1 and D2 via RTPCR in ovariectomized (ovx, n=7) and sham operated pro-estrous (n=7) female Fischer -344 rats. Guide cannulas for microdialysis were implanted in ipsilateral VMN and contralateral LHA in all rats along with either ovx or sham operation. After 10 days of recovery in-vivo microdialysis was done to measure DA and 5HT (pg/20ul) in LHA and VMN. Subsequently rats were sacrificed and LHA and VMN harvested for receptor analysis. Data calculated as mean + SE and analyzed by t test $*=p<0.05$, significant. Results: In ovx rats, LHA DA, LHA 5HT and food intake were all greater than in the intact proestrous rats. There was no significant difference between the VMN DA and 5HT between the groups. Further dopamine D1 receptors were seen only in the intact rats and were absent in the ovx rats. OvX rats have a higher food intake with increased LHA DA and absent D1 receptor. In contrast high estradiol during proestrous decreases food intake with lower LHA DA and serotonin.

Estrogen influence on stimulated water intake by ovariectomized female rats. E.G. KRAUSE, K.S. CURTIS, L.M. DAVIS, R.J. CONTRERAS. Florida State University, Program in Neuroscience, Tallahassee, FL 32306-1270.

Stimulated water intake is attenuated during the estrous phase of the female rat reproductive cycle. Elimination of estrogen via ovariectomy (OVX) abolishes this attenuation. To further understand how estrogen modifies thirst, we examined stimulated water intake by adult female Sprague-Dawley rats in the presence or absence of estrogen. Female rats underwent OVX and were allowed one week to recover. OVX rats were then treated with estrogen (.10ug/1ml oil) or vehicle (1ml oil) on Day1 and Day2. On Day3, OVX rats were subjected to 24h food and water deprivation. On the following day rats were given access to water in graduated drinking tubes and intake was measured every 30 min for 2h. Estrogen significantly decreased water intake when compared to that by vehicle treated animals following 24h water deprivation. A separate group of adult female rats underwent OVX and were allowed a week to recover. OVX rats were then treated with estrogen (.10ug/1ml oil) or vehicle (1ml oil) on Day1 and Day2. On Day4 of the schedule OVX rats were given subcutaneous injections of hypertonic saline (1ml of 2M NaCl). Following the injections rats were given access to water in graduated drinking tubes and intake was measured every 30min for 2h. Estrogen had no effect on water intake when compared to that by vehicle treated animals following injection of hypertonic saline. The results suggest that estrogen may specifically modulate water intake elicited by volemic dehydration because osmotically-elicited drinking was similar under estrogen and vehicle conditions.

Alcohol consumption in the dopamine 3 receptor knockout (D3 -/-). J.-A.M. MCQUADE¹, S.C. BENOIT¹, M. XU², R.J. SEELEY¹. Department of Psychiatry¹ and Cell Biology², University of Cincinnati, Cincinnati, OH 45220

There is a large body of evidence suggesting a role for the mesolimbic dopaminergic system in ethanol consumption and reinforcement. Ethanol consumption, in rats, has been demonstrated to stimulate dopamine release in the nucleus accumbens. Moreover, microinjection of dopamine antagonist into the nucleus accumbens will block ethanol self-administration. It is well established that dopaminergic signaling is mediated by two-subtypes of receptors. The D1 class (D1 and D5) and the D2 class (D2, D3, and D4) which activate and inhibit the release of cAMP, respectively. The role of each receptor subtype in ethanol consumption is still controversial. Reports utilizing the D1 R and D2 R deficient mice have demonstrated a lack of ethanol consumption in a home cage access paradigm. Despite being heavily expressed in the nucleus accumbens, little data exists focusing on the D3 R and its potential role in mediating voluntary consumption of ethanol and the reward associated with ethanol consumption. We sought to investigate the role the D3 R plays in reward associated with ethanol consumption by using mice with a targeted deletion of the D3 R. Mice were given access to increasing concentrations of ethanol in a two-bottle free choice paradigm in addition to a restricted 1 hour acute access paradigm. Once the mice reached maximum preferred intake at 6 %, they were administered a variety of DA receptor agonists and antagonists. Thus, the present study assessed acute and chronic ethanol consumption in the D3 -/- mouse.

Increased food intake in mice with targeted deletion of the dopamine 3 receptor. S.C. BENOIT, J.-A. MCQUADE, M. XU, R.J. SEELEY. Depts. of Psychiatry and Cell Biology, University of Cincinnati, Cincinnati, OH 45220

We recently reported that mice with targeted deletion of the dopamine 3 (D3) receptor gene (D3^{-/-}) have increased adipose stores relative to controls, and that this phenotype is exacerbated when animals consume a high fat diet. However, short-term analyses of food intake revealed no differences between wild-type controls and D3^{-/-} mice. The present experiments were conducted to elucidate possible mechanisms for increased adiposity in D3^{-/-} mice. Using indirect calorimetry, we assessed energy expenditure in both genders of D3^{-/-} and control mice, but found no differences among groups. In addition, D3^{-/-} and control mice had similar responses to food restriction and refeeding. Finally, we assessed three-month cumulative food intake on mice consuming either a high fat diet or standard chow. D3^{-/-} mice consumed significantly more food than wild type controls. Hence, the elevated level of body fat is due to a small but steady increase in food intake.

Food intake in mice lacking the bombesin receptor subtype-3. J. OVERDUIN, N. SANCHEZ, L. HAMPTON, J. BATTEY, J. GIBBS. Bourne Laboratory, Weill Medical College of Cornell University, White Plains, New York 10605, USA. The bombesin receptor subtype-3 (BRS-3), located on the X chromosome, has affinity for bombesin and mammalian bombesin-like peptides (Gorbulev et al, 1992). Mice lacking BRS-3 develop obesity – a 46% increase in body weight compared to wild-type littermates by 40 weeks of age (Ohki-Hamazaki et al, 1997). In that report, the only one to date, solid food intake at 20-24 weeks of age was significantly increased (by 9%) in BRS-3-deficient mice compared to wild-type littermates. We are tracking body weight, food intake, meal patterns, and the microstructure of feeding in this gene-deleted strain. Here, we report body weights and food intakes for Week 12 (the first measurement period) in BRS-3-deficient mice and their wild-type littermates. Fifteen male BRS-3-deficient (-/Y) and 15 wild-type littermate controls (+/Y) were maintained on a diluted, sweetened condensed milk diet (1.4 kcal/ml; the only source of food) and water, both ad-libitum. Mean body weights at Week 12 were 24.5 +/- 0.3 g for -/Y and 22.9 +/- 0.3 g for +/Y (p<0.001). Mean food intakes averaged over 7 days were 12.4 +/- 0.3 g/day for -/Y and 11.2 +/- 0.2 g/day for +/Y (p<0.002). These data replicate the findings of Ohki-Hamazaki et al, and extend them to consumption of a palatable liquid food. Supported by NIDDK RO1 DK33248 and by the NIDCD.

Regulation of Gastric Emptying of Glucose in the Preweanling Rat. R.J. DAVIS, S.E. SWITHERS. Dept. of Psychological Sciences, Purdue University, West Lafayette, IN 47907

During development, a behavioral change in independent ingestion in response to the content of a gastric preload has been described. For example, 9-day-old pups that receive a gastric preload of glucose consume less than pups that receive a saline preload; glucose loads also produce termination of ingestion with larger gastric volumes (Swithers & Hall, *Physiol. & Behav.* 46(5) 873-879, 1989). Thus, it appears that an alteration of gastric emptying in response to some property of a glucose load may contribute to the termination of ingestion. To investigate this hypothesis, we examined gastric emptying of a glucose diet in 6 and 15 day old pups. Pups received an oral infusion of 0, 1.2, 2.4, 4.8 or 9.6% glucose via a posterior oral cannula and diets were made osmotically equivalent by the addition of NaCl; phenol red was added to the diet to measure gastric emptying. By 6 days of age, gastric emptying of the highest concentration of glucose was slower than gastric emptying of saline; a similar pattern was observed in 15-day-old pups. These results suggest that pups as young as 6 days of age are capable of regulating gastric emptying of a glucose solution by a mechanism not related to osmotic properties. Supported by NIDDK RO1 55531

Factors influencing the oral stimulation-induced inhibition of fat emptying from the stomach. J.E. CECIL¹, M.I. FRIEDMAN¹, H.J. GRILL², J.M. KAPLAN². ¹Monell Chemical Senses Center, Philadelphia, PA, 19104; ²Dept. Psychology, University of Pennsylvania, Philadelphia, PA, 19104, USA.

Recent data indicate that gastric emptying of fat is slower after oral than after intragastric delivery, whereas carbohydrate empties from the stomach at a similar rate regardless of delivery route. To further investigate this delivery route effect for fat, experiments were conducted to determine: (1) the amount of oral stimulation by fat required to inhibit gastric emptying and (2) to establish whether chemical properties of chyme associated with ingestion underlie this effect. Rats received intraoral (IO), intragastric (IG) or concurrent (IO+IG: 50% + 50%, 25% + 75%) infusions of corn oil-water emulsion (1:1) delivered at a net infusion rate of 1.0ml/min. Infusions containing any oral component significantly slowed gastric emptying compared with rates obtained with IG infusions alone. However, when the IO component of concurrent infusions was reduced to 10% of the total amount infused (IO+IG: 10% + 90%), the delivery route effect disappeared. Together, these data establish a threshold proportion of oral delivery, between 10 and 25% of the total load, below which no inhibition of gastric emptying is obtained. In a further experiment, IG infusion of gastric contents recovered from donor rats, previously given IO corn oil emulsion, yielded emptying rates no lower than that obtained when unadulterated emulsion was delivered IG. This result shows that the delivery route effect does not depend on orally stimulated secretions or facilitated hydrolysis of the emulsion, and suggests a neurogenic underpinning of this cephalic phase response.

Inhibition of food intake by intestinal maltotriose requires oligosaccharide digestion but not glucose absorption. M. COVASA, R.C. RITTER. Department of VCAPP, Washington State University, Pullman, WA 99164-6520, USA. Intestinal infusion of maltotriose, a product of starch digestion, inhibits food intake. This inhibition of food intake by intestinal maltotriose depends upon activation of capsaicin-sensitive vagal sensory neurons. Since the neuronal processes do not penetrate into the lumen, they must either be sensitive to absorbed carbohydrate (glucose), or be activated secondarily by an action of the carbohydrate in the intestinal lumen. However, the roles of digestion and absorption in inhibition of food intake by maltotriose, or other carbohydrates, have not been systematically investigated. Therefore, we examined glucose absorption and inhibition of food intake following intestinal maltotriose infusion in rats that were treated with acarbose, to inhibit the digestion of maltotriose to glucose, or with phlorizin, to inhibit the intestinal transport of glucose by the sodium dependent glucose transporter, SGLT-1. Intestinal infusion of 180 mM maltotriose markedly inhibited food intake and significantly elevated plasma glucose concentrations by 30 min post infusion, indicating that maltotriose-induced inhibition of food intake was accompanied by hydrolysis of the oligosaccharide and absorption of glucose. Pretreatment of the intestine with acarbose prevented post-infusion hyperglycemia, and attenuated maltotriose-induced inhibition of food intake, indicating that hydrolysis of maltotriose to glucose is necessary for inhibition of food intake. On the other hand, pretreatment with phlorizin prevented elevation of blood glucose following maltotriose infusion, but did not attenuate reduction of food intake, indicating that glucose absorption is not necessary for inhibition of food intake. We conclude that reduction of food intake by intraintestinal maltotriose results from an intraluminal action of glucose generated by maltotriose hydrolysis. Supported by NS-20561.

NMDA participation in cholecystokinin-induced inhibition of food intake. M. COVASA, T. BUI, R.C. RITTER, G.A. BURNS. Department of VCAPP, Washington State University, Pullman, WA 99164-6520, USA. Several investigators have demonstrated that the NMDA receptor antagonist, MK-801, increases food intake and attenuates reduction of intake by exogenous CCK. MK-801 does not increase food intake by triggering meal initiation after a meal has terminated. Rather, MK-801 delays meal termination only if injected prior to or soon after meal initiation. These results suggest that NMDA receptor blockade interferes with the development of satiation signals, but does not reverse satiation signals once they have occurred. To determine whether MK-801-induced attenuation of CCK-induced reduction of food intake is sensitive to the time of MK-801 administration, we examined the effect of MK-801 on food intake when the antagonist was administered either prior to or following injection of CCK. When MK-801 (100 µg/kg, IP) was administered prior to CCK (2 µg/kg, IP) injection, reduction of food intake was attenuated. When CCK was injected 5 min before MK-801 injection, reduction of food intake by CCK also was reversed. These results indicate that, while MK-801 and CCK produce opposing effects on meal size, it is unlikely that MK-801's effect is due to direct antagonism of CCK-induced satiation. Supported by NIDDK-52849 and NS-20561.

Role of the vagus in feeding suppression produced by jejunal lipid infusions. J.E. COX, A. RANDICH, G.R. KELM, S.T. MELLER. Department of Psychology, University of Alabama at Birmingham, Birmingham, AL and Procter and Gamble, Health Care Technology Division, Mason, OH. We have previously observed that jejunal infusions of linoleic acid reduce total caloric intake, body weight, and body fat. A role for celiac vagal fibers was indicated by increased activity of celiac afferents in response to linoleic acid infusions and attenuation, though not elimination, of the feeding suppression by selective celiac vagotomy. In the current experiment, rats with total subdiaphragmatic vagotomy (TVx; N=7), hepatic-branch vagotomy (HVx; N=6), and sham vagotomy (N=8) underwent four tests with jejunal linoleic acid matched with four saline tests, as well as two tests with infusions of corn oil matched with two saline tests. Infusions (0.2 ml/h; 11.5 kcal total) were 7 h in duration, beginning 1 h before the start of the dark cycle. Liquid diet (vanilla Boost, Mead-Johnson) was available starting 1 h after onset of infusions. TVx produced results very similar to what we have previously observed with celiac vagotomy: suppression of cumulative intake was attenuated by approximately 50% at 3, 6, and 23 h. HVx did not significantly reduce linoleic acid-induced suppression at any time. On the other hand, when corn oil was infused, suppression was similar in the TVx and HVx groups, that is, significantly less than in controls at 3 and 6 h. Thus, our results support a role for the hepatic vagus in suppression produced by infusion of a triglyceride but fail to point to a similar conclusion regarding suppression by a long-chain fatty acid. In addition, the presence of a vagally-independent mechanism is indicated by significant residual suppression after transection of all subdiaphragmatic vagal branches.

Jejunal and portal vein infusions of lipids affect hepatic vagal afferent activity. A. RANDICH, J.E. COX, D.S. SPRAGGINS, G.R. KELM, S.T. MELLER. Department of Psychology, University of Alabama at Birmingham, Birmingham, AL. Procter and Gamble, Health Care Technology Division, Mason, OH. Jejunal infusion of lipids has been shown to both suppress food intake and activate celiac vagal afferents. Celiac vagotomy attenuates this effect of lipid administration on suppression of food intake but does not eliminate it. We examined whether either jejunal or portal vein infusions of lipids affected hepatic vagal afferent activity as a possible alternative substrate for the behavioral effects. Jejunal infusion (1 ml) of either linoleic acid, corn oil, or caprylic acid significantly increased hepatic vagal afferent activity during 2 hour recording periods, whereas saline infusions were not effective. The magnitude of response was greatest with linoleic acid and corn oil, and the response latency was approximately 30 minutes. The response to caprylic acid was delayed by approximately one hour. Portal infusions (.5 ml/h - 1.5 mg) of either linoleic acid, Liposyn II, or caprylic acid significantly increased hepatic vagal afferent activity during 1 hour recordings, whereas 5% albumin/phosphate buffer vehicle was ineffective. The magnitude of response was greatest with either linoleic acid or Liposyn II. These data show that either jejunal or portal infusions of lipids affect activity of hepatic vagal afferents, and can potentially serve as an alternative substrate to celiac vagal afferents in mediating the effect of jejunal infusions of lipids in suppressing food intake.

Common hepatic branch fibers to the liver are sufficient to mediate the satiating effect of endogenous CCK released by duodenal infusions of maltose. P. SANCHEZ, G.P. SMITH. Bourne Laboratory, Weill Medical College of Cornell University, White Plains, NY, 10605 USA.

Duodenal infusions of maltose decrease food intake through a paracrine action of endogenous CCK (Brenner et al., 1993). We recently demonstrated that the common hepatic branch (CHB) of the abdominal vagus is sufficient to detect and transduce the inhibitory effect of CCK released by duodenal infusion (di) of 20% maltose (Sanchez et al., 2000). Because the CHB consists of fibers to the liver (H) and to the gastroduodenal region (GD), we investigated whether the inhibitory effect of 20% maltose (di) could be mediated by H fibers alone. Rats with H fibers alone (n=9) were prepared by sectioning the GD fibers and all of the other abdominal vagal branches except the CHB. Their 30-min intakes of 10% sucrose after 20% maltose (di) were compared to intakes in sham-operated rats (n=6). 20% maltose (di) decreased intake significantly ($p<0.05$) and equally in sham ($25 \pm 9\%$) and H rats ($34 \pm 11\%$). When a CCKA antagonist was given before 20% maltose (di), H rats ate $84 \pm 34\%$ more than after 20% maltose alone ($p<0.05$). Because exogenous CCK-33 inhibited intake significantly in H rats ($38 \pm 8\%$; $p<0.05$), but equimolar CCK-8 did not ($-5 \pm 17\%$), we suggest that 20% maltose (di) decreased intake by releasing CCK-33 that acted on afferent CCKA receptors on the H fibers. Supported by: NIH grant MH40010d by NIDDK RO1 DK33248 and by the NIDCD.

Effects of high-fat diets on the satiating potency of CCK-8. A-M. TORREGROSSA, G.P. SMITH. Bourne Laboratory, Weill Medical College of Cornell University, White Plains, NY, 10605 USA

Covasa et al. (1998) recently demonstrated that a high-fat diet (34% fat, by weight) decreased the satiating potency of low doses of CCK-8 ($<1\mu\text{g}/\text{kg}$), but not of higher doses, on deprivation-induced test meals. In an attempt to replicate this phenomenon, we maintained rats on the low-fat (5% fat) and high-fat diets (34% fat) used by Covasa et al.. Male Sprague Dawley rats were adapted to the diets for a minimum of 2 weeks before testing. In protocol 1 rats (n=12) were tested daily; in protocol 2 rats (n=14) were tested on alternate days. In both protocols rats were injected, IP, with five doses of CCK-8 (0.125-2.0 $\mu\text{g}/\text{kg}$) or saline (1 ml/kg) 5 minutes before access to their maintenance diet after a 17-hour food deprivation. Intake was measured after 30 minutes. We found no significant difference in the potency of CCK-8 in rats maintained on 5% fat or 34% fat diets. Furthermore, when rats in protocol 1 were maintained on a 60% fat diet, the satiating potency of four doses of CCK-8 (0.125-1.0 $\mu\text{g}/\text{kg}$) increased significantly ($p=0.003$) compared to rats on the 5% fat diet. Thus, we failed to replicate Covasa et al.'s report of reduced satiating potency of low doses of CCK-8 by a 34% fat diet, despite using identical diets and experimental conditions. In addition, we found that a 60% fat diet significantly increased the potency of CCK-8. Supported by MH 40010

Restrained eaters are insensitive to the flavor-flavor learning paradigm. J.M. BRUNSTROM¹, S. HIGGS². ¹Human Sciences, Loughborough University, Loughborough, Leicestershire, LE11 3TU, UK. ²School of Psychology, University of Birmingham, Edgbaston, Birmingham, B15 2TT, UK.

Flavor preference learning in 21 restrained and 21 unrestrained females was explored using an evaluative conditioning paradigm. Each participant was exposed to an adapted version of the procedure used by Johnsrude et al., (1999, *Learning & Motivation*, 30, 250-264). During training, participants sampled 10 instances of three novel flavors presented in a semi-randomized order. After sampling a flavor they were instructed to eat a sweet according to three different flavor-reinforcement contingencies. One flavor was accompanied by a sweet on 90% of trials and was presented alone on 10% of the trials, while the remaining flavors were rewarded at ratios 50% and 10%: 90%, respectively. The training phase occurred in conjunction with a counting task requiring continuous use of working memory, and was immediately followed by the participants making hedonic ratings of each flavor. Very few participants showed awareness of the purpose of the experiment or the specific reward contingencies. Despite this, the ratings given by the unrestrained eaters were highly correlated with the reward ratio experienced during training. In contrast, restrained eaters exhibited no evidence for evaluative learning. These findings may explain the equivocal nature of results from previous studies of positive flavor-flavor learning and may offer a novel theoretical context within which to study dietary restraint.

Disordered eating among college athletes: Influence of muscularity dissatisfaction and supplement use. B. RAUDENBUSH, B. MEYER. Wheeling Jesuit University, Department of Psychology, Wheeling, WV, USA.

Mixed findings have been reported regarding body satisfaction in males, with some studies reporting males are satisfied with their body image and others reporting that they are not. One of the confounds of past research assessing body image satisfaction is that it typically employs figure drawings which vary in level of body fat. The present study assessed body image satisfaction using figures varying in level of muscularity among college athletes participating in intercollegiate track/cross-country, soccer, basketball, swimming and lacrosse. In addition, level of disordered eating and mass/muscle gain supplement use was assessed for each sports team. Players in all sports chose significantly different figure drawings to best represent their current, ideal and attractive to the opposite sex figures. For each sport, their current figure was significantly less muscular than their ideal figure. Also for all sports, their current figure was significantly less muscular than the figure they thought was attractive to the opposite sex. Soccer and lacrosse players chose an ideal figure larger than the figure they thought was attractive to the opposite sex, while swimmers chose an ideal figure smaller than the figure they thought was attractive to the opposite sex. Lacrosse players wished to gain the greatest amount of musculature. Those athletes using mass gain supplements engaged in more hours of weight workouts per week, and had greater eating pathology. They also viewed their current figure as larger than those athletes not using mass gain supplements. Of all athletes, swimmers indicated the greatest eating pathology. The present results further validate the desire of athletes to gain musculature, possibly to the extent of abusing weight/mass gain supplements and providing the foundation for eating disorder development.

Binge-type eating in rats is due to limited access, not food deprivation. R.L. CORWIN, J.J. CARMAN. Nutrition Dept., Penn State Univ., University Park, PA 16802.

Limiting access to an optional source of shortening induces binge-type behavior in non-food-deprived rats. Under these conditions, rats consume more energy than controls on shortening days, and less on non-shortening days. As a result, rats enter the shortening access (binge) sessions in a self-imposed food-deprived condition. Thus, the bingeing could be attributed to the previous day's reduced intake rather than to limited access per se. The present research addressed this issue. Forty-two adult male Sprague-Dawley rats were assigned to four groups: Control-no shortening access; RSA7-shortening access for 2-h/day, 7 days/week; RSA3-shortening access for 2-h/day, 3 days/week (regular weekly schedule); ISA-shortening access for 2-h/day (irregular weekly schedule). Across the 4-wk study RSA3 and ISA both had 12 binge sessions. However, the last binge sessions of RSA3 and ISA were separated by 1 and 4 day(s), respectively. All rats had continuous access to chow throughout the study. RSA3 and ISA consumed significantly more than RSA7 during the binges ($p<0.05$). 24-h energy intake on the days immediately prior to the last two binges in the ISA group did not differ from Control intakes. Intakes of RSA3, however, were less than Control and ISA ($p<0.05$). Even though energy intake prior to the binge sessions differed between RSA3 and ISA, consumption during the binges did not differ between these two groups. These data demonstrate that bingeing under limited access conditions in rats can be induced by limited access alone, and is not dependent upon undereating on the previous day. Supported by NIMH.

Food avoidance behaviour among female university students: association with Eating Disorder Inventory and its subscales scores. M.L.S. SANTOS, P.P.P. MACHADO. Instituto de Educação e Psicologia, Universidade do Minho, Campus de Gualtar, 4710-057 BRAGA, Portugal.

Young female university students (18-30 years old; $N=437$) filled in the Eating Disorders Inventory (EDI) and were asked to indicate on a list of 70 common foods which were those they usually avoided and the respective reason for such behaviour. About 16% of the women ($n=68$) had an EDI score higher than 43, therefore being at a higher risk of developing an eating disorder (at-risk group) when compared with others scoring 43 or below (normal group). More of the women at-risk than the normal ones avoided foods/beverages, most of them with a high energy density. Significant differences between the two groups of women among the diversity of reasons were observed for the avoidance of 27 foods (mostly with a high energy density too). Reasons under the category "fattening" were pointed out more by women at-risk, while unpleasant sensory and/or unhealthy aspects were referred to more by normal women. The same procedure, taking into account the different cut off points, was also applied to all EDI subscales. The above findings were also found for one or more subscales more related to eating disorders: Drive for thinness, Bulimia and Body dissatisfaction. High scores for the remaining EDI subscales, connected to more general psychological traits, did not have a significant association with foods and/or motives for avoidance. The present research confirms that the EDI and at least three of its subscales consist of a robust instrument for detecting eating disorders and the associated food avoidance behaviour.

Early eating experiences of women with eating problems. C.A. SCHULZ, P. WRIGHT. Department of Psychology, University of Edinburgh, UK. Research and clinical reports have stressed the importance of the family environment for the development of eating disorders. The aim of the present study was to examine the influence of early experiences of food and eating within the family on different forms of eating problems. Method: To allow a comparison we assessed anorexic, bulimic and morbidly obese women ($n=57$) and a control group of women without a history of eating problems ($n=20$). Questionnaires about current eating behaviour (BITE, EAT, EDI), childhood relationship with parents (PBI) and being teased about appearance (PARTS) were used. Additionally an extensive semi-structured interview to explore their early socialisation in regards to food was carried out. A statistical analysis was undertaken and put into context with the results of a qualitative analysis of the interview data. Results: Despite such different eating pathology surprisingly few differences regarding early eating experiences could be found. One of the key areas where differences emerged was in regards to early body image. The eating disordered groups had a more negative body image as a child and experiencing more extensive teasing. The groups with eating problems were also more likely to have negative memories of meal situations and described their parents as generally more controlling particularly in regard to food. This study also highlights the complementary nature of results from quantitative and qualitative analyses.

Selective Processing of weight- and shape-related words in bulimia nervosa: use of a computerised Stroop test. E.J. DAVIDSON, P. WRIGHT. Dept of Psychology, University of Edinburgh, Scotland UK Objective: A computerised Stroop colour naming task was used to measure concerns about weight, shape and eating in bulimia nervosa. Method: Two versions of the computerised Stroop were compared, a voice-activated and a button-pressing-activated programme. Results: Bulimia nervosa patients were significantly slower in colour naming shape- and weight-related words than their female age-matched controls. The button-pressing computerised Stroop was both more sensitive and more accurate at measuring colour naming speeds than the voice-activated version. When the bulimia nervosa group were divided according to their EAT scores, those who showed extreme pathological attitudes to weight and shape were significantly slower in colour naming size words and in food disruption scores, than those with a lesser degree of psychopathology. Discussion: The computerised Stroop might be useful as a diagnostic tool and in the assessment of the effectiveness of therapy for the individual patient.

Multiple signals inhibit thirst. E.M. STRICKER. Department of Neuroscience, University of Pittsburgh, Pittsburgh, PA 15260.

Three signals are known to stimulate thirst in rats and other animals: blood-borne osmolytes that affect forebrain osmoreceptors, neural input to the brain stem from cardiovascular baroreceptors, and a blood-borne endocrine stimulus detected by angiotensin II (AngII) receptors in the subfornical organ. However, excitatory signals are not the only stimuli that control thirst. For example, anticipatory processes provide early, preabsorptive inhibition of further drinking. In dogs, apparent oral metering of ingested fluid provides satiety irrespective of which fluid is consumed, whether that fluid is absorbed, and what its ultimate impact is on the osmolality of systemic blood. In rats, early inhibitory signals result from a somewhat different process, because the mere act of drinking does not confer satiety. Thus, whereas consumption of water by dehydrated rats confers satiety before the osmolality of systemic plasma has been reduced, consumption of isotonic saline has no such inhibitory effect on fluid ingestion (as it does in dogs). An additional inhibitory signal results from osmotic dilution of systemic blood; it potentially reduces water consumption induced by hypovolemia and AngII despite the continued presence of excitatory signals. A third inhibitory signal results from increases in arterial blood pressure, which reduce water intake similarly regardless of whether thirst is stimulated by hyperosmolality, hypovolemia, or AngII; this inhibitory effect appears to be mediated by arterial baroreceptors. The same excitatory and inhibitory signals that affect thirst appear also to control the pituitary secretion of vasopressin, which complements water intake in thirsty animals.

Effects of mouth wetting on the satiation of thirst. J.M. BRUNSTROM. Human Sciences, Loughborough University, Loughborough, Leicestershire, LE11 3TU, UK.

In humans, the association between mouth dryness and thirst has been examined in a variety of contexts. Typically, studies show that drinking behavior produces a concomitant reduction in unpleasant dry-mouth sensations. Evidence is reviewed for a mechanism that induces satiety by metering this change. Drinking behavior causes a progressive increase in parotid saliva flow. Thus, one possibility is that satiety can result from a decrease in the reward associated with mouth wetting during a drinking episode. Several studies have shown that beverages differ in their satiating ability. This variability is reviewed in relation to this 'mouth-wetting hypothesis'. In particular, two beverage characteristics may be particularly important, temperature and acidity. In relation to these factors, evidence is assessed for an association between thirst satiety, preference, and changes in mouth-state.

Impact of electrolytes, flavor and palatability on voluntary fluid intake during exercise. D.H. PASSE, J. STOFAN, M. HORN, R. MURRAY. Gatorade Sports Science Institute, 617 West Main Street, Barrington, IL, U.S.A.

Previous research suggests that adding flavor to water can increase its palatability and can increase voluntary fluid intake during exercise. Current dose-response studies suggest that increasing flavor intensity results in an increase in beverage palatability, in an inverted u-shaped function. Varying the concentration of a carbohydrate-electrolyte flavor system in water produced more salient changes in perceived flavor intensity and sweetness intensity than in perceived saltiness intensity. Systematically increasing sodium concentration did not increase palatability. Sodium beyond 40 mEq/L, in a 6% carbohydrate electrolyte rehydration beverage, was associated with a decrease in overall palatability. While there were statistically significant correlations between overall beverage palatability and voluntary fluid intake, palatability accounted for less than 20% of the variation in fluid intake in these studies. Methodological differences may account for the relatively low correlations observed. Directly manipulating beverage palatability as an independent variable produced substantial effects on voluntary fluid intake. Subjects drank more of a beverage previously identified as being highly palatable, than of plain water.

Associative conditioning of the image of a can of cola by anticipatory thirst reduction and other refreshment. D.A. BOOTH. Food Quality & Nutritional Psychology Research Group, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK. To round off laboratory and field data on the slaking of thirst, longstanding theory of physiologically and socially reinforced Gestalten of satiating stimuli is applied to the amounts that individuals drink of familiar liquids. Whether or not (as previously argued) hunger and thirst themselves are usually conditioned responses to sensorily familiar ingestates in particular bodily states and social contexts, there is good reason and evidence for such learning of normal control of the volumes of foods and fluids consumed by our and other vertebrate species. The evidence is indirect, however, in the case of 'long' beverages such as cola, beer, tea (if not coffee), fruit juices and water itself. Brown drinks are rated as thirst-quenching in USA and UK but purple drinks even more so in the UK: blackcurrant flavouring may have this image primarily because of association of the end of the glass or pack of drink with increased residual wetting of the mouth after stimulation by acid. Perhaps brands of cola have broader refreshing images largely by association with the sight of young people enjoying themselves. Sociable mood and intellectual confidence are conditioned to drink breaks by longer delayed reinforcers such as diffuse neural excitation by caffeine and, some claim, cognitive enhancement by action of glucose on the brain. These learnt responses are also cued by anticipatory need states. The collection of key evidence is therefore a challenging task that has yet to be understood in or out of industry.

The 5-HT_{1B/1D} antagonist, GR 127,935, does not attenuate the hypophagic action of d-fenfluramine in mice: discrepancies between antagonist studies and transgenic models of 5-HT function. M.D. LEE¹, G.A. KENNETT², C.T. DOURISH², P.G. CLIFTON¹. ¹Experimental Psychology, Sussex University, Brighton, BN1 9QG. ²Vernalis Research Ltd, Wokingham, RG51 5UA, UK.

The importance of 5-HT_{1B} receptors in the control of ingestive behaviour in mice has been emphasised by two recent studies demonstrating that mice lacking 5-HT_{1B} receptors are less sensitive to the hypophagic action of d-fenfluramine (Kennett et al. *Abstr Neurosci* 26:145.11, 2000) and racemic fenfluramine (Lucas et al. *J Neurosci* 18:5537, 1998). Here we characterise the effect of the selective 5-HT_{1B} agonist, CP-94,253, on food intake and prandial behaviours in mice. We also challenge the hypophagic action of CP-94,253, and that of d-fenfluramine with the 5-HT_{1B/1D} antagonist GR 127,935. Non-deprived 5-HT_{1B} knockout (KO) mice and matched wild-type (WT) controls (N=12/genotype) were given CP-94,253 (0, 5, 10, 20 mg/kg IP) 30 min prior to a 40-min test meal of palatable wet mash. CP-94,253-treatment reduced the consumption of mash in WT mice at 10 and 20 mg/kg (Pd-fenfluramine (10mg/kg). Although these findings support the involvement of 5-HT_{1B} receptors in the modulation of feeding behaviour in mice, they are not compatible with the postulated role of 5-HT_{1B} receptors in d-fenfluramine-induced hypophagia suggested by the knockout studies. Supported by BBSRC-LINK award 85/LKD12007.

Antagonism of different serotonin (5HT) receptors in the basolateral amygdala (BLA): Effects on feeding. D.V. COSCINA, C.M. PARKER. Department of Psychology, Wayne State University, Detroit, MI 48202 USA. A variety of work has shown that enhancing 5HT neurotransmission in brain can inhibit food intake in rats. The ability of suppressing 5HT brain function to drive overeating has been less well studied, particularly in brain structures outside the hypothalamus. Data will be presented showing that bilateral infusions of the 5HT_{1/2/7} antagonist, metergoline (MET), are effective in enhancing short-term intake of lab chow or the more preferred sweet food, Froot Loops cereal, in otherwise satiated female rats. These feeding effects appear to be relatively independent of the animals' phase of estrous cycling. In contrast to these results, BLA infusions of the 5HT_{1B} antagonist, SB 216641, or the 5HT_{2C} antagonist, RS 102221, in doses equimolar to MET do not elicit overeating, despite clear evidence that both receptors reside in this brain region. The implications of this work will be discussed in the context of other anatomical and neurochemical systems that appear to interact with the BLA in modulating feeding as well as the types of processes they may help control. This novel research holds promise for improving our basic understanding of potential etiological factors that drive overeating in such troubling clinical entities as bulimia nervosa and binge eating disorder.

Relationship of aminergic to peptidergic neuromodulators of paraventricular nucleus (PVN) in cancer anorexia. M.M. MEGUID, Y. QI, T. TADA. Neuroscience Program, Surgical Metabolism & Nutrition Lab., University Hospital, SUNY Upstate Medical University, Syracuse, NY 13210, USA.

INTRODUCTION: Studies demonstrate that increased serotonin (5HT) and decreased dopamine (DA) in VMN, and decrease of NPY in PVN exist in cancer anorexia of tumor bearing (TB) rats. Intra-PVN injection of 5HT antagonist increases NPY and food intake (FI). We hypothesized that 5HT, DA and NPY are abnormal in PVN of TB rats, and that these revert to normal with increase in FI after tumor resection. METHODS: 48 male rats were randomized: i) TB, inoculated with MCA sarcoma (n=16); ii) Non-tumor bearing (NTB, n=16) and iii) NTB pair-fed (PF, n=16). Feeding pattern was measured via rat eater meter. With anorexia, 8 rats/gp were sacrificed. Remaining TB rats had tumor resected (TB-R) while NTB and PF had sham operation. When FI in TB-R rats normalized, all rats were sacrificed. Measured were: PVN-NPY (RIA), DA and 5HT (HPLC). Data tested via ANOVA. RESULTS: When anorexia developed, PVN-5HT increases (12.2 \pm 0.5 to 14.9 \pm 0.8 pg/ug protein, p < 0.01), and NPY decreases (23.5 \pm 4.3 to 13.6 \pm 1.4 pg/ug protein, p < 0.05) in TB vs NTB. After tumor resection, 5HT, DA and NPY in PVN reverted to normal levels. CONCLUSION: At onset of anorexia a 5HT increase and a decrease in DA and NPY with a concomitant decrease in FI occurred in PVN. These neuromodulators and food intake normalized after tumor resection. Data link aminergic to peptidergic neuromodulators in PVN in normal and cancer anorexia.

Intra-VMN infusion of nicotine suppresses food intake and enhances dopamine (DA) and serotonin (5HT) release in both VMN and LHA. L. ZHANG, M.M. MEGUID, S.O. FETISSOV. Neuroscience Program, Surgical Metabolism and Nutrition Laboratory, University Hospital, SUNY Upstate Medical University, Syracuse, NY 13210, USA.

INTRODUCTION: Nicotine reduces appetite and body weight. Because of the involvement of the ventral median nucleus (VMN) and lateral hypothalamic area (LHA) neurotransmitters in food intake control, we hypothesized that nicotine may increase activity of DA and/or 5HT in the VMN and LHA, in relation to nicotine-induced hypophagia. METHODS: Adult male Fischer rats had microdialysis guide cannulas implanted in ipsilateral VMN and contralateral LHA. Either 4mM nicotine (n = 8) or vehicle (n = 8) was administered via reverse microdialysis into the VMN of overnight food-deprived rats for 60 min. Then food was provided for 40 min. Simultaneous measurement of DA and 5HT in VMN and LHA using HPLC was done at 20-min intervals before, during, and after nicotine administration. Data are mean + SE, and analyzed by ANOVA and t-test. RESULTS: Food intake during the 40-min re-feeding period was significantly lower during nicotine perfusion vs. control vehicle (4.1 \pm 0.3g vs. 2.6 \pm 0.4, p<0.05). Relative to baseline, continuous nicotine perfusion into the VMN for 60 min increased VMN-DA (213 \pm 37%, p<0.05) and 5HT (823 \pm 324%, p<0.05) concentrations. After cessation of nicotine perfusion the increase in VMN-5HT (210 \pm 21%, p<0.05) persisted for another 60 min. Concomitantly, an increased LHA-5HT (150 \pm 21%, p<0.05) was also measured. CONCLUSION: Data indicate that intra-VMN nicotine enhances simultaneously dopaminergic and serotonergic activity in both VMN and LHA, and that enhanced VMN-5HT, VMN-DA and LHA-5HT activity contribute to nicotine-induced hypophagia.

Dietary influence on plasma amino acids and central neurotransmitters in stress-susceptible pigs. L. THIBAUT. School of Dietetics and Human Nutrition. Macdonald Campus of McGill University, Montréal, Québec, Canada. Central neurotransmitters are involved in behaviours such as feeding, drinking and the stress response. The present study was performed in pigs varying in their susceptibility to stress to assess if feeding with a casein-based diet influences plasma amino acids profile and central neurotransmitters levels, compared to the traditional western Canadian cereal-based diet. Fifty-eight 8-week old pigs of both sexes representing each of the three genotypes for stress susceptibility (nn, Nn, NN) had blood sampling prior to dietary adaptation, and after 2 and 5 weeks of dietary adaptation. Serotonin (5-HT) and 5-HIAA, DA, NA and their metabolites were measured by HPLC in various brain areas dissected from half of the animals prior to dietary adaptation and in the other half following 5 weeks. There was a greater feed consumption by pigs fed the cereal-based diet than pigs fed the casein diet, but feed intake was not affected by sex or genotype. Diet adaptation to the casein diet resulted in greater essential amino acids tryptophan, lysine, threonine, methionine and arginine plasma concentrations compared to cereal-based diet. Among effects of dietary casein on central neurotransmitters, pigs from the nn genotype displayed a higher 5-HIAA/5-HT ratio in the pons and increased 5-HT and 5-HIAA levels in the raphe nuclei and the hypothalamus, but decreased NE levels in the pons. Dietary protein's influence on central neurotransmitters and their dietary precursors might impact on various aspects of farm animals behaviour. Acknowledgements: This study was made possible by funds from the Canada-Québec subsidiary agreement Agri-Food development. The author wishes to thank Drs. A.C. Murray and A.L. Schafer from the Agriculture and Agri-Food Canada Research Center in Lacombe (Alberta) for their technical expertise.

Is glutamate in the Anterior Piriform Cortex-Lateral Hypothalamus pathway (APC-LH) involved in the anorectic responses to indispensable amino acid deficiency (IAAD)? D.W. GIETZEN, B.G. TRUONG, J.E. BLEVINS, P.S. TEH. Department of VM Anatomy, Physiology and Cell Biology, UC Davis, CA, USA. Animals fed a diet causing IAAD become hypophagic within 1hr. The brain area that senses IAAD may be in the APC, which projects directly to the LH. This projection may be important in the anorectic responses to IAAD. Aja (1999) showed that single axons from the IAAD sensitive site project directly to the magnocellular cells of the dorsolateral LH (MCLH). Monda et al. (1997) showed increased firing rates in the cells of the LH 1/2hr after injection of threonine into the APC of threonine deficient rats. The primary output cells of the APC are glutamatergic, so we examined the effects of glutamate agonists and antagonists both in the APC and in the LH. In the APC, the NMDA antagonist, AP5 (1.6 ug) caused a 31% increase in IAAD intake that lasted 24 hr, while the AMPA antagonist, NBQX (1.0ug) resulted in a 58% increase at 6 hr that also remained elevated at 43% to 24 hr (Truong, 1999). In contrast in the LH, NMDA had no effect on IAAD intake, but AP5 caused a 30% decrease in IAAD intake. Microdialysis for glutamate in the MCLH suggests that glutamate release in the extracellular spaces around the APC terminals is increased (153% of baseline) at 20 min after IAAD, but also after the corrected control diet. Thus, the glutamate system is clearly implicated in the APC in these responses, but further study of the APC-LH pathway in IAAD feeding is needed. Supported by NIH: NS33347, DK50347, DK35747, DK07355, & USDA: NRI 97-3500-4477 & 2000-1049.

Dose-response effects of amylin-related peptides on food intake and gastric emptying in rats. R.D. REIDELBERGER, L. KELSEY, D. HEIMANN. Department of Veterans Affairs Medical Center, Omaha, NE, 68105.

Amylin is a 37 amino acid peptide that is secreted with insulin in response to a meal. When administered systemically or into the brain of rats, amylin potently reduces food intake and body weight. This study compared the dose-dependent effects of IV infusion of the amylin-related peptides amylin, rat calcitonin gene-related peptide (rCGRP), salmon calcitonin (sCT), and rat calcitonin (rCT) on food intake and gastric emptying. Non-food-deprived rats (n=9-14) received a 3-h IV infusion of peptide (0.1-100 pmol/kg/min) or vehicle beginning 15 min before dark onset. Food intake was recorded for 17 h after onset of infusion. In gastric emptying experiments rats (n=8-12) received a 15 min IV infusion of peptide (1-500 pmol/kg/min) or vehicle beginning 10 min before gastric instillation of 3 ml saline containing phenol red. Five min later gastric contents were recovered and the amount of saline emptied was determined. sCT, amylin, and rCGRP dose-dependently inhibited 3-h food intake with mean effective doses (ED₅₀s) of 0.5, 8, and 26 pmol/kg/min and maximal inhibitions of 91, 78, and 90%, respectively. sCT produced a prolonged suppression of food intake: 0.3, 1, 3, and 10 pmol/kg/min doses decreased 17-h intake by 8, 20, 55, and 85%, respectively. sCT, amylin, rCGRP, and rCT dose-dependently inhibited gastric emptying with ED₅₀s of 1, 2.9, 130, and 730 pmol/kg/min and maximal inhibitions of 60, 60, 66, and 44%, respectively. The amylin-related peptides inhibited food intake and gastric emptying with a similar rank order of potency: sCT > amylin > rCGRP > rCT.

Acute 3rd-ventricular amylin infusion potently reduces food intake but does not produce aversive consequences. P.A. RUSHING, R.J. SEELEY, E.L. AIR, T.A. LUTZ, S.C. WOODS. Department of Psychiatry, University of Cincinnati, USA; Institute of Veterinary Physiology, Zürich, Switzerland.

Although data indicate that the pancreatic hormone amylin provides a signal to the brain to reduce food intake, it is also possible that amylin produces aversive consequences that secondarily reduce intake independently of the normal regulation of energy balance. Thus, we used a conditioned taste aversion (CTA) paradigm to assess this possibility. Rats received 1-h daily access to water for 7 days to ensure stable fluid intake during the 1-h period. On day 8, rats were given 1-h access to a 0.15 % saccharin solution (rather than water) followed immediately by treatment. One group (n = 7) received 100 pmol amylin (i3vt in 3 µl volume), a 2nd group (n = 7) received i3vt CSF as control, a 3rd group (n = 7) was given LiCl (0.15 M LiCl given ip in a volume of 2% of body weight), and a 4th group (n = 7) was given ip saline as control for ip LiCl. After 2 intervening days of 1-h access to water, rats were tested for acquisition of a CTA. In a two-bottle choice test, rats were simultaneously presented with water and saccharin for 1 h. As a quantitative measure of the formation of a CTA, the preference ratio of saccharin intake to total volume of fluid consumed was calculated. The LiCl rats exhibited a marked aversion to the saccharin. In contrast, no evidence of a CTA was observed in the rats that received i3vt amylin. One week after CTA assessment, we tested the ability of the 100 pmol i3vt amylin dose to reduce food intake in the same group of rats. Consistent with what we have previously reported, both short-term (1-h) and long-term (24-h) food intake were significantly reduced by amylin. In summary, these data are consistent with the conclusion that acute i3vt amylin infusion does not reduce food intake by producing aversive consequences.

High fructose meals reduce 24-hour circulating insulin and leptin concentrations and increase subsequent energy and fat intake in women. K.L. TEFF^{1,2}, S. ELLIOT³, R. TOWNSEND², P.J. HAVEL³. ¹Monell Chemical Senses Center, ²University of Pennsylvania Health System and ³University of California, Davis, CA. Leptin secretion is dependent on insulin-mediated glucose utilization. Since fructose does not stimulate insulin secretion, we hypothesized that meals high in fructose would produce lower leptin concentrations compared to meals containing the same amount of glucose. Nine women (BMI = 23.5±3.3 kg/m²) were studied on two separate visits to the GCRC during which they consumed 3 meals containing 55% of total kcal as carbohydrate, 30% as fat, and 15% as protein with 30% of the energy as either fructose (HFr) or glucose (HG) beverage. Blood samples were collected every 30-60 minutes during the first 24-h of the study. On the subsequent day, subjects were allowed to select their meals ad libitum. Postprandial glycemic responses were reduced by 50% (p< 0.025) and meal-induced insulin secretion was 60% lower (p< 0.0001) on the HFr day compared with the HG day. The 24 h area under the curve (AUC) for circulating leptin was 28±12% (p< 0.05) smaller on the HFr compared to the HG day. On the day following the HFr day, the subjects consumed an average of 220±521 kcal more energy and the quantity of fat ingested was ~30% greater (39.6 ± 12.3 g) compared with the HG day (30.3±6.9 g, t= 2.23, P< 0.025) and meal-induced insulin secretion was 60% lower (p< 0.0001) on the HFr day compared with the HG day. The 24 h area under the curve (AUC) for circulating leptin was 28±12% (p< 0.05) smaller on the HFr compared to the HG day. On the day following the HFr day, the subjects consumed an average of 220±521 kcal more energy and the quantity of fat ingested was ~30% greater (39.6 ± 12.3 g) compared with the HG day (30.3±6.9 g, t= 2.23, P<0.05). In summary, HFr meals resulted in lower 24-h circulating glucose, insulin, and leptin concentrations and a subsequent increase in ad libitum energy and fat consumption. Decreases of circulating insulin and leptin could contribute to weight gain during long-term consumption of diets high in fructose.

Central leptin gene therapy in prepubertal rats reduces weight gain, metabolic hormones, blood leptin and insulin levels but augments energy expenditure postpubertally for extended period. E. BERETTA, M.G. DUBE, H. DHILLON, P.S. KALRA, S.P. KALRA. Department of Neuroscience, Department of Physiology, McKnight Brain Institute, University of Florida, Gainesville, FL 32610-0244 USA

Increased incidence of pediatric obesity and its ramifications on postpubertal health is a major clinical concern. We investigated the effects of a single intraventricular administration of a recombinant adeno-associated viral vector encoding rat leptin (rAAV-leptin, 10¹² particles in 3 µl) or green fluorescent protein (rAAV-GFP in 3 µl) in 24 days old, prepubertal female rats on weight gain, adiposity and various metabolic hormones for 11 months postpubertally. The results showed that rAAV-leptin reduced the rate of weight gain by 36%, food intake by 23% and adiposity, as reflected by 85% reduction in serum leptin. Likewise, the metabolic parameters, insulin, non-esterified fatty acids (NEFA), insulin-like growth factor (IGF-I), thyroxine (T4) and glucose were significantly reduced. These long-term effects were produced by a local increase in leptin in the hypothalamus, as shown by enhanced leptin mRNA expression analyzed by RT-PCR and by the observation that, unlike the rAAV-leptin treated rats, pair-fed (PF) rats did not display similar marked reductions in blood leptin and other metabolic hormones in association with reduced rate of weight gain. In addition, UCP1 mRNA in brown adipose tissue was significantly augmented in rAAV-leptin (19%) but not in PF rats, thereby implying increased energy expenditure through thermogenesis driven by rAAV-leptin. In summary, central rAAV-leptin therapy prepubertally is a potential therapeutic modality to reduce weight gain, adiposity and attendant metabolic hormonal disorders for extended periods postpubertally. (Supported by NIH HD 08634, DK37273 and NS 32727).

Is leptin involved in day-to-day regulation of energy intake? B.J. HRUPKA, W. LANGHANS. Institute of Animal Sciences, Swiss Federal Institute of Technology, 8092 Zurich, Switzerland.

Plasma leptin levels are not constant, and can change dramatically within 1 d of fasting or high fat feeding. We examined the possibility that leptin plays a role in integrating day-to-day energy intake, possibly by acting as an integrative signal of energy influx and efflux from adipose tissue, and altering subsequent food intake accordingly. Fourteen lean (Fa/?) and 14 obese (fa/fa) Zucker rats were used in each study (7 ad libitum-fed controls/genotype). Rats were adapted to either standard rat chow (Exp. 1) or a high fat diet (18% w/w, Exp. 2). In both experiments, half the rats/genotype were food deprived for 24 h. Regardless of diet composition, Fa/? rats increased their food intake the day after food deprivation, while fa/fa rats did not (genotype x treatment, both both P<0.001). In Experiment 3, rats were switched from ad libitum food access to a 12 hr schedule-feeding paradigm with dark phase food access. Schedule-fed Fa/? rats ate 95% of control rats'; food intake from d 2 onward. Schedule-fed fa/fa rats adapted significantly slower, and took until d 4 to consume 95% of control rats'; food intake (P<0.005). Under a 6 hr schedule-feeding paradigm with early dark phase food access (Exp. 4), Fa/? and fa/fa rats adapted similarly (P>0.4). Both groups consumed 48% of control rats'; food intake on d 1, and slowly increased their food intake to 78% by d 7 (P<0.0001). These results further support the hypothesis that leptin is involved in regulating day-to-day energy fluctuations.

Effect of food access schedule and dietary composition on nocturnal levels of serum melatonin and pineal N-acetyltransferase activity. B. SELMAOUI, A. OGUINE, L. THIBAUT. School of Dietetics and Human Nutrition, Macdonald Campus of McGill University, 21,111 Lakeshore Road, Ste Anne de Bellevue, QC, Canada, H9X 3V9 This study investigated the effect of dietary composition and food access schedule on the rhythmicity of serum melatonin and pineal N-acetyltransferase (NAT) activity. Wistar rats maintained on a 12:12h light: dark cycle were assigned to two dietary groups: a group fed rat chow and a group fed a choice between a protein-rich and a carbohydrate-rich diet. Each dietary group was further divided based on feeding schedule, with food available between 0800 and 1600 h or ad libitum access to food. Regardless of dietary condition, total food and carbohydrate intake of rats having free access to food was higher than under the restricted food access schedule. Protein intake of rats fed the dietary choice was lower with the restricted access than in the free access. In rats fed the dietary choice, melatonin levels and NAT activity were significantly decreased with restricted access compared to free access. Such results were not found in rats offered chow. This study suggests that the rhythms of melatonin secretion and NAT activity can be altered by dietary composition. Key words: Circadian rhythm, Rats, Protein-rich diet, Carbohydrate-rich diet, Daytime feeding. Acknowledgements: This research was supported by a grant from the Natural Sciences and Engineering Research Council of Canada, and by a Postdoctoral Fellowship from the Service de la coopération internationale-programme Québécois de bourses d'excellence, Québec, Canada (B. Selmaoui).

Central mechanism of the effect of leptin on learning and memory. Y. OOMURA, N. HORI, T. SHIRAIISHI, S. AOU, X. LI, K. FUKUNAGA, K. SASAKI. Dept. Physiol. Kyushu Univ. Fukuoka, 812-0054, Tokai Univ. Ishizaki, 259-1100, Kumamoto Univ. Kumamoto, 860-0811, Toyama Univ. Toyama, 930-8555, Japan. Leptin increases in rat plasma by about 2.4×10^{-10} M during eating and enters into CSF less than 1/1000. Iv applications of leptin 5 - 50 fEq/kg (approximate concentration in CSF, 10-12 - 10-11M) facilitated dose-dependently emotional and spatial learning and memory and that of 500 fEq/kg impaired them. In vitro hippocampal CA1 synapses, 10-12M leptin application for 15 min enhanced long-term potentiation (LTP) and 10-10M suppressed it. The amount of presynaptic transmitter release measured by the paired pulse facilitation and miniature excitatory postsynaptic current methods decreased under 10-12M leptin and increased under 10-10M. The postsynaptic membrane response for the transmitters did not change by 10-12M leptin and decreased slightly in 10-10M. The $[Ca^{2+}]_i$ in CA1 neurons reached to about 300nM and 700nM in 10-12M and 10-10M leptin, respectively. The above pre-and postsynaptic events could be explained by the $[Ca^{2+}]_i$ results. More than 500 fEq/kg increase in $[Ca^{2+}]_i$ deactivates calmodulin protein kinase II (CaMKII) activity and activates calcineurin, a phosphatase. Actually, CaMKII activity in the CA1 neurons increased in 10-12M leptin after tetanization, but decreased in 10-10M leptin. The increase in CaMKII activity is closely related to the facilitation of LTP and of behavioral performance. On the other hand, Zucker rats and db/db mice who have not normal leptin receptors showed learning and memory impairment and no LTP. Moreover, CaMKII activity did not change after tetanization. These evidences indicate an importance of the physiological role of leptin affecting on the higher brain function.

Cocaine-induced avoidance of saccharin intake is associated with elevated circulating corticosterone levels at test when using a 5 min or a 30 min interstimulus interval. P.L. SCHROY, D.S. WHEELER, P.S. GRIGSON. The Penn State College of Medicine, Hershey, PA 17033.

Despite the same number of saccharin-morphine pairings, some rats (the Small Suppressers) exhibit a small suppression of conditioned stimulus (CS) intake, and others (the Large Suppressers) a large suppression of CS intake. The present experiment tested whether similar individual differences would be evident when using cocaine as the unconditioned stimulus (US) and a 5 min or a 30 min interstimulus interval (ISI). During testing, water-deprived male Sprague-Dawley rats were given 5 min access to 0.15% saccharin and, after a 5 or 30 min ISI, were injected with either saline ($n = 8/\text{cell}$) or cocaine ($n = 16/\text{cell}$). There were 7 CS-US pairings followed by one CS only test. Plasma corticosterone (CORT) was evaluated 15 min after CS access both before the first, and after the last, trial. The results showed that cocaine suppressed CS intake more when using a 5 min, rather than a 30 min, ISI. CORT levels were elevated in both the Small and Large Suppressers in the 5 min condition, but only the Large Suppressers in the 30 min condition. In each case (i.e., when using either the 5 or 30 min ISI), however, greater cocaine-induced suppression of CS intake was associated with higher CORT levels at test (5 min ISI: $r = -0.466$, $p < 0.01$; 30 min ISI: $r = -0.64$, $p < .0001$). Taken together, the data show that, like morphine, greater avoidance of the saccharin CS following saccharin-cocaine pairings is associated with higher levels of circulating CORT at test. Supported by NIH grants DA09814 and DA12472.

Responding based on interoceptive signals produced by 2-DG depends on sucrose anticipation. A.L. TRACY, T.L. DAVIDSON. Department of Psychological Sciences, Purdue University, W. Lafayette, IN, USA.

Previous research indicates that the glucose antimetabolite 2-deoxy-D-glucose (2-DG) not only increases food intake in rats, but also produces interoceptive properties that are similar to those of 24-hr food deprivation (Benoit & Davidson, 1996). This study used a deprivation intensity discrimination procedure in which rats were trained to anticipate a mild shock based on high or low levels of food deprivation. In the current study, we examined if the same effect would be seen in animals trained to perform this discrimination, but receiving food, rather than shock, as the unconditioned stimulus (US). Further, we wanted to determine if this effect would be dependent on the macronutrient composition of this training US (i.e. 100% carbohydrate vs. 100% fat), as 2-DG-treated animals show a preferential increase in carbohydrate intake. In an extinction test, animals trained with a carbohydrate US did show generalization to 24-hr food deprivation when given 2-DG (350 mg/kg). Animals trained to anticipate a fat US failed to show this generalization and, in fact, showed no appetitive responding in the training context, regardless of their deprivation state US contingency during training. These results suggest that separate representations can be formed for foods with different macronutrient compositions and that these representations can be selectively activated by particular interoceptive deprivation signals.

Conditioning of flavor preference by NaCl after exercise is dependent on perspiration. N. WALD, M. LESHEM. Department of Psychology, University of Haifa, Israel 31905. micah.leshem@psy.haifa.ac.il We hypothesized that NaCl after exercise-induced sodium loss may have increased rewarding properties, as it does in sodium depleted rats. To evaluate the rewarding value of post-ingestive physiological changes induced by the salt, and to avoid assumptions about the temporal pattern of these changes as well as cognitive attitudes to salt, we used taste conditioning for a novel drink by concurrent untasted NaCl after 90min exercise (aerobic or basketball). Participants drank 100 ml root beer, and swallowed a capsule of NaCl either empty (placebo) or containing 200, 400 or 600mg NaCl (20 participants in each dose group). There were 4 conditioning trials each 3-7 days apart. We found that the variable that best predicted a salt-conditioned taste preference was individual sweat loss. Participants that lost substantial sweat and received 200-600mg NaCl increased their preference for the root beer. Exercisers that sweated minimally and received 600mg NaCl showed preference below placebo levels, suggesting a conditioned taste aversion. This reduced preference was not related to reported side effects (which were very few, and bore no relationship to any measure of preference conditioning or NaCl dose). The results suggest that i) in humans, as in rats, sodium loss increases the rewarding value of NaCl, ii) that providing sodium after exercise-induced sweating may induce a rewarding physiological state and iii) that conditioned taste preference may provide a useful measure of the benefits of nutrients in sports drinks. Arguably, the experiment provides further evidence for the physiological determinants of human salt appetite.

Circling behavior and conditioned taste aversion induced by high strength static magnetic fields. T.A. HOUP, D.W. PITTMAN, J.M. BARRANCO, J.W. JAHNG, J.C. SMITH. Program in Neuroscience, The Florida State University, Tallahassee, FL, USA and Department of Pharmacology, Yonsei University College of Medicine, Seoul, Korea.

Advances in magnetic resonance imaging are driving the development of more powerful and higher resolution machines with high-strength static magnetic fields. The behavioral effects of high-strength magnetic fields are largely uncharacterized, although restraint within a 9.4T magnetic field is sufficient to induce a conditioned taste aversion (CTA) and induce brainstem expression of c-Fos in rats. In order to determine if the behavioral effects of static magnetic fields are dependent on field strength, duration of exposure, and orientation with the field, rats were restrained within the bore of 7T or 14T superconducting magnets for variable durations. Behavioral effects were assessed by scoring locomotor activity after release from the magnetic field, and by measuring CTA acquisition after pairing intake of a palatable glucose and saccharin solution (G+S) with magnetic field exposure. Magnetic field exposure at either 7T or 14T suppressed rearing and induced tight circling. The direction of the circling was dependent on the rats' orientation within the magnetic field: if exposed head-up, rats circled counter-clockwise; if exposed head-down, rats circled clockwise. CTA was induced after 3 pairings of G+S and 30-min 7T exposure, or after a single pairing of G+S and 1-min 14T exposure. These results suggest that magnetic field exposure has graded effects on rat behavior. We hypothesize that restraint within high-strength magnetic fields causes vestibular stimulation resulting in locomotor circling and CTA acquisition. Supported by NIDCD03198 and BK21 Project.

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Notes:

Layfayette

Elsevier 1

Elsevier 2

Mini Mitter 1

Mini Mitter 2

Coulbourn 1 insert

Coulbourn 2 insert

Coulbourn 3 insert

SSIB and how to join

SSIB is a professional organization of research scientists who study eating and drinking behavior. The Society's main goal is to advance the study of ingestive behavior by facilitating communication among its members. "Intake" is the Society's periodic newsletter.

SSIB membership information and applications are available at the meeting registration desk, at the SSIB web site (<http://www.ssib.org>), or by writing to the Central Office (address below).

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Stephen Woods, Ph.D.
Gary J. Schwartz, Ph.D.
Randall Sakai, Ph.D.
Janet Guss
Harvey J. Grill, Ph.D., *ex officio*

Tuesday, June 26

13:00-17:00 Registration – Sansom P West
13:00-17:30 Penn Symposium
15:00 Beverage Break – Meyerson Lobby
17:30 Open Bar (provided by the University of Pennsylvania) - Bodek Lounge, Houston Hall
19:00-21:00 Welcome Reception (Beer, Wine and Snacks) – Bodek Lounge, Houston Hall

Wednesday, June 27

07:30-08:30 Breakfast – Campus Dining
08:20-08:30 Opening: T.R. Scott – Logan 17
08:30-10:00 Symposium 1: Biological Mechanisms of Clinical Cachexias (Chair: G.J. Schwartz) – Logan 17
08:45-10:00 Oral Session 1: Mineral and Fluid Balance – (Chair: M.G. Tordoff) – Terrace Room
10:00-10:30 Coffee Break– Logan Foyer
10:30-12:00 Oral Session 2: Metabolic Controls (Chair: M.I. Friedman) – Terrace Room
10:30-12:00 Oral Session 3: The Parabrachial Nucleus – Role in Ingestion and Beyond (Chair: K.J. Simansky) – Logan 17
10:30-12:00 Oral Session 4: Social and Physiological Aspects of Food Intake in Females (Chair: T.A. Spiegel) – Meyerson B1
12:00-13:30 Free Time for Lunch (lunch not provided)
13:30-15:30 New Investigator Presidential Symposium (Chair: T.R. Scott) – Meyerson B1
15:30-16:00 Coffee Break– Meyerson B1 and Logan Foyer
16:00-17:30 NIDA-sponsored Symposium 2: Like Drugs for Chocolate...(Chair: P.S. Grigson, Co-Chairs: M. Lynch, S. Volman) – Logan 17
16:00-17:30 Oral Session 5: Central Peptides (Chair: T.H. Moran) – Terrace Room
19:00-22:00 Poster Session I – Bodek Lounge, Houston Hall

Thursday, June 28

07:30-08:30 Breakfast – Campus Dining
08:30-10:00 Symposium 3: Transgenic Models & Genetic Approaches...Food Intake Control and Obesity (Chair: S.P. Kalra) – Logan 17
08:30-10:00 Oral Session 6: Human ingestion (Chair: B.J. Rolls) – Terrace Room
10:00-10:30 Coffee Break – Logan Foyer
10:30-12:00 Symposium 4: The Developing Story of Glucoreceptor Neurons (Chair: B.E. Levin, Co-Chair: H.J. Grill) – Logan 17
10:30-12:00 Oral Session 7: Physiologic Mechanisms of Disordered Eating (Chair: B.T. Walsh) – Terrace Room
12:00-13:30 Free Time for Lunch (lunch not provided)
13:30-15:00 Keynote Lecture (Chair: H.J. Grill) – Meyerson B1. J.S. Flier: Leptin and the regulation of body weight...
15:00-16:00 Women's Forum – Terrace Room
16:00-17:00 New Investigators' Forum – Terrace Room
15:30-18:00 Long Range Planning Committee Meeting - Bishop White Room, Houston Hall
18:00-20:00 Reception at the Monell Chemical Senses Center
20:00-22:00 Board Meeting – Bishop White Room, Houston Hall

Friday, June 29

07:30-08:30 Breakfast – Campus Dining
08:30-10:00 Oral Session 8: Melanocortins (Chair: J.M. Kaplan) – Logan 17
08:30-10:00 Oral Session 9: Influences on Selections among foods (Chair: D.A. Booth) – Meyerson B1
08:30-10:00 Oral Session 10: Genetic and Molecular Approaches (Chair: S.J. Fluharty) – Terrace Room
10:00-10:30 Coffee Break – Logan Foyer
10:30-12:00 Symposium 5: The Dorsomedial Hypothalamus Revisited (Chair: M. Tang-Christensen) – Logan 17
10:30-12:00 Oral Session 11: Scaling of Sensations & Intake Measurements (Chair: H.R. Kissileff, Co-Chair: L. Bartoshuk)–Terrace Rm.
12:00-13:30 Free Time for Lunch (lunch not provided)
13:30-14:30 Award Lectures (Chair: R. Norgren) – Meyerson B1
13:30-14:00 Early Career Award Lecture, R. Seeley: When worlds collide: GLP-1 and the response to visceral illness.
14:00-14:30 Johnson & Johnson Career Award for Research in Ingestive Behavior Lecture, A.J. Stunkard: Eating disorders& the brain.
14:30-17:30 Poster Session II – Bodek Lounge, Houston Hall
17:30-18:30 Business Meeting – Meyerson B1
19:15- Banquet (included with registration) – Hall of Flags, Houston Hall
19:15-20:15 Open Bar
20:00-21:30 Dinner and Presentations
21:30- Music and Dancing

Saturday, June 30

07:30-14:00 Check out – Sansom West
07:30-08:30 Breakfast – Campus Dining
08:30-10:00 Symposium 6: Satisfaction of Thirst (Chair: M. Pelchat, Co-Chair: D. Booth) – Terrace Room
08:30-10:00 Oral Session 12: Small Molecule Neurotransmitters (Chair: D.W. Gietzen) – Logan 17
10:00-10:30 Coffee Break – Logan Foyer
10:30-12:00 Oral Session 13: Hormones (Chair: W. Langhans) – Logan 17
10:30-12:00 Oral Session 14: Learning and Reward (Chair: T.A. Houpt) – Terrace Room

Next SSIB Meeting

**August 7-11, 2002
University of California Santa Cruz
Santa Cruz, CA USA**