PROGRAM

2007 SSIB Annual Meeting

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Steamboat Springs, Colorado, USA

July 24-29, 2007

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WELCOME

Dear Colleagues,

Welcome to the 15th Annual Meeting of SSIB, the Appetite Society. I hope your travel was not to stressful and you find our meeting site enjoyable. Try to forget the often hectic and sometimes stressful life being a scientist and relax in this beautiful oasis in the Rocky Mountains.

Our Executive Coordinator, Marianne Van Wagner has worked very hard and diligently to make the meeting experience enjoyable, and the Program Committee with Tim Moran at its helm has prepared an outstanding and stimulating program.

The highlights of the program include favorites such as the New Investigator Symposium, the Master Foods Lecture Series, and no less than eight Symposia focusing on many timely and important topics. In addition we will enjoy other "old favorites" such as the stimulating and cutting edge Oral Presentations and the lively Poster Sessions.

We also have three new program items. An Industry Symposium is intended to facilitate the dialogue and collaboration between SSIB and industry researchers. Two representatives from different branches of the National Institute of Health will help us to be more successful in writing grants and obtaining research funds. Finally, the new Presidential Symposium will feature selected topics by investigators that have just made the critical step to independence in their own laboratories.

We should have lots of fun, whether listening to and discussing with speakers and friends, engaging in physical activity on a mountain hike or on the dance floor, indulging in the delicacies of the local cuisine, or just simply lay back and relax in a light breeze of pure mountain air.

It is my sincere hope that you all can go back with that good feeling of being scientifically invigorated that comes from having learned something new, engaged in vigorous discussions, and made new friends.

We welcome your participation, criticism and suggestions. Enjoy!

Hans-Rudi Berthoud

President of SSIB, The Appetite Society, 2006-2007

Long Range Planning Committee

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On behalf of the entire membership of the Society for the Study of Ingestive Behavior (SSIB), The Board and the Organizing Committee of the 2007 Annual Meeting sincerely thank the following organizations for their generous support of the Society:

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We also sincerely thank all the individual SSIB members who made personal contributions to the Society this year.

Distinguished Career Award

JACK D. DAVIS
Listening to the tongue

Alan N. Epstein Research Award

STEPHEN C. BENOIT, University of Cincinnati, USA
A poker enthusiast's guide to central insulin and the regulation of energy balance

New Investigator Awards

Listed alphabetically here and marked in the program with #.

Lucy Caroline Chambers, Sussex University, UK

Jon Franklin Davis, University of Cincinnati, USA

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Margriet A.B. Veldhorst, Maastricht University, The Netherlands

Linda A.W. Verhagen, Rudolf Magnus Institute of Neuroscience, The Netherlands

Tuesday, July 24, 2007

8:00-2:00 ***SSIB Board Meeting*** – Location: Twilight Room

3:00-5:30 ***Registration*** – Please use this time to pick up your name badges, receipts and program. Location: Foyer

5:30-7:30 ***Welcome Reception for all Participants**** (hors d'oeuvres and cash bar) – Location: Poolside Tent

7:30-9:00 ***Students Only--Get Acquainted Social Event**** (food and beverages will be provided) – Location: Aspen Room

Wednesday, July 25, 2007

NOTE: Presenting authors are indicated by **BOLD** print throughout the program.

7:30-8:15 CONTINENTAL BREAKFAST/EXHIBITORS' DISPLAY – FOYER

8:15 Greeting – ***HANS-RUDI BERTHOUD***, President

Symposium 1 - 8:30-10:30
Location: *Storm Peak*
Latest Research Developments from the Drug and Food Industries in the Battle against Obesity
Chair: ***A. GELIEBTER***

8:30 ***E.M.R. KOVACS (Unilever):*** Weight control functionality in commercial foods: From 'R' to 'D'.

9:00 ***P.A. TATARANNI (Sanofi-Aventis):*** Selective CB1 receptor antagonism for the management of cardiometabolic risk factors.

9:30 ***N. ERONDU (Merck):*** Neuropeptide Y5 receptor antagonism in overweight and obese adults.

10:00 ***C. MACK (Amylin):*** Integrated neurohormonal therapy for obesity: A novel approach aimed at harnessing the interaction between adipocyte-, islet-, and gut-derived signals in energy homeostasis.

Oral Session 1 - 8:30-10:30
Location: *Mt. Werner*
Ingestion - Developmental Factors
Chair: ***A. WELLER***

8:30 ***F. RUTTERS***[#]*, A.G. NIEUWENHUIZEN, N. VOGELS, F. BOUWMAN, E. MARIMAN AND M.S. WESTERTERP-PLANTENGA:* Genetic, parental and behavioral factors, plus leptin-adiposity relationship changes are involved in the development of body-weight in a Dutch children cohort.

8:45 ***T.V.E. KRAL****, A.J. STUNKARD, R.I. BERKOWITZ, V.A. STALLINGS, M.S. FAITH:* Associations between eating frequency, energy intake, and body weight among 6-year-old children.

9:00 ***J. ARGUELLES****, J.J. DIAZ, I. MALAGA, C. PERILLAN, S. MALAGA, M. VIJANDE:* Salt intake and preference in different groups of Spanish children and adolescents.

9:15 *C. PAQUET,* ***C. HUET****, L.THIBAULT, L. DUBÉ:* Objective sensory dimensions of food as predictor of nutrient intake in the elderly.

9:30 ***K.A. SCOTT****, Y. YAMAZAKI, R. YANAGIMACHI, S.C. WOODS, R.R. SAKAI, K.L.K. TAMASHIRO:* Mice generated by assisted reproductive techniques exhibit altered glucose metabolism.

9:45 *C. MORENS, P. NIVOIT, A. PIERSMA, B. REUSENS, C. REMACLE, F.A. VAN ASSCHE:* Development of metabolic syndrome can be programmed in utero by maternal diet induced obesity.

10:00 ***A.K. PORTELLA****, P.P. SILVEIRA, J. DIORO, M.J. MEANEY:* Maternal care programs appetite and weight gain in rats.

10:15 ***C.M. PATTERSON****, B.E. LEVIN:* Short-term post-weaning exercise increases central leptin sensitivity in diet-induced obese rats fed a high-energy diet even after exercise termination.

10:30-11:00 BREAK/EXHIBITORS' DISPLAY - FOYER

11:00-12:00 Masterfoods Keynote Lecture Series *- Introduction: W. LANGHANS.*
MARTIN G. MYERS, JR.: Molecular and neural mechanisms of leptin action and leptin resistance. Location: Storm Peak/Mt. Werner

Free Afternoon

New Investigator Symposium - 4:00-6:00
Location: Storm Peak/Mt. Werner
Chair: ***H-R. BERTHOUD***

Supported by educational grants from NIH and Takeda Pharmaceuticals North America

4:00 ***G. FINLAYSON***[#]*, A. ARLOTTI, N. KING, J. BLUNDELL:* Sub-clinical binge eating tendency associated with increased BMI, weakened satiety response, increased liking for high-fat sweet food and enhanced explicit but not implicit wanting for food.

4:15 ***B.M. GEIGER***[#]*, L.E. FRANK, A.D. CALDERA-SIU, L. STILES, E.N. POTHOS:* Deficiency of central dopamine in multiple obesity models.

4:30 ***C.M. MATHES***[#]*, M. FERRARA, D. SURESH, A. ANDREASEN, C. HASKELL-LUEVCANO, N.E. ROWLAND:* Effect of level of dysfunction of the melanocortin-4 receptor (MC4R) on overconsumption and binge-like eating of a palatable dessert in mice.

4:45 ***M.L. SMITH***[#]*, A. KIECHLER, S.Z. GOODIN, A.D. STRADER:* The hyperphagic effect of the selective melanocortin-4 receptor antagonist HS014 is gender specific.

5:00 ***M. RUITER***[#]*, T. DUFFY, S. SIMASKO, R.C. RITTER:* Hindbrain leptin injections induce forebrain as well as hindbrain signaling.

5:15 ***J.A. TESKE***[#]*, C.M. KOTZ:* Orexin A effects on wakefulness in obesity prone and obesity resistant rats.

5:30 ***E.G. KRAUSE***[#]*, J.F. DAVIS, L.Y. MA, R.R. SAKAI:* Angiotensin type 1 receptor antisense in the subfornical organ attenuates the drinking and corticosterone response to isoproterenol.

Poster Session 1 – 6:00-8:00
Location: Sunshine Peak/Twilight – WINE & CHEESE

1. ***G.L. EDWARDS****, D.R. GADDAM, K.G. FREEMAN:* Cannabinoid agonist antagonizes cFos induced by intraperitoneal cholecystokinin.

2. ***A.N.A. VERTY****, W.M. BOON, B.J. OLDFIELD:* The impact of CB1 cannabinoid receptor antagonist SR 141716 (rimonabant) on hypothalamic peptides mediating energy balance.

3. ***A.N.A. VERTY****, B. J. OLDFIELD:* The effect of the cannabinoid receptor antagonist, rimonabant (SR 141716) on energy expenditure.

4. ***D.L. CHOI****, J.F. DAVIS*[#]*, D.J. CLEGG, S.C. BENOIT:* Mice exhibit enhanced macronutrient-specific conditioned place preference under hypocaloric conditions.

5. ***J.P. BAIRD****, R. MCINTYRE & K.R. THEIM:* Effects of dieting history saliency on self-esteem and perceived body image in college women.

6. ***N.T. BELLO****, J.L. SETHNESS, M.H. KEMM, T.H. MORAN:* A history of binge-type feeding leads to alterations in forebrain dopamine and hindbrain c-Fos immunoreactivity in rats.

7. ***S. IMADA****, H. SETOYAMA, T. HASEGAWA, N. SAKAI:* The relationship between eating problems and personality of Japanese college students.

8. ***G. MARTÍNEZ****, A. LÓPEZ-ESPINOZA, A. GALINDO, V. AGUILERA, A. GONZALEZ, C. DE LA TORRE-IBARRA:* Effects of glucose and sucralose solutions on water and food intake: Binge drinking response in albino rats.

9. ***C.M. MATHES***[#]*, M. FERRARA, N.E. ROWLAND:* Differences in binge-like eating patterns of a palatable dessert and body weight gain in young and old female rats.

10. *K. BLACKER, R. DRAKE, A. REED, J. ALMEIDA,* ***B. RAUDENBUSH****:* Body image satisfaction among intercollegiate female athletes using a scale of muscularity.

11. ***C. PELKMAN****, R. POHLE, J. NAVIA:* Dietary restraint status modulated satiety responses in women.

12. ***S. TAZAKI****, S. IMADA, T. MORI:* The relationships among the domain of self-esteem, drive for thinness, and restraint eating in Japanese adolescent females.

13. ***L.M. FLANAGAN-CATO***, *S.J. FLUHARTY, D.R. LABELLE:* Effects of a high-fat diet (HFD) on dendritic morphology in the male rat hypothalamic ventromedial nucleus (VMH).

14. ***R.I. GEDDES***, *L. HAN, P.S. GRIGSON:* Lesions of the gustatory thalamocortical loop block drug-induced devaluation of a natural saccharin reward cue, while leaving instrumental responding for the drug intact.

15. ***J.U. HEIMAN***, *J.F. DAVIS[#], A.L. TRACY, R.J. MOORE, J.M. ZIGMAN, D. CLEGG, S.C. BENOIT:* Mice lacking the ghrelin receptor (GHS-R -/-) exhibit impaired hippocampal-dependent learning.

16. *C.H.* ***REVELLE***, *Z.S. WARWICK:* Time course of acquisition of flavor-nutrient learning with fat vs. carbohydrate calories.

17. ***S. JARVANDI***, *D.A. BOOTH, L. THIBAULT:* Learnt anticipatory eating in rats reinforced by a single length of fast.

18. ***S. MOBINI***, *M. LEITCH, N. GOULD, M.R. YEOMANS:* Differential hedonic, sensory and behavioural changes associated with flavour-nutrient and flavour-flavour learning.

19. ***I.M.T. NIJS***, *I.H.A. FRANKEN, P. MURIS:* Food cue-elicited brain potentials in obese and healthy-weight individuals.

20. ***H. ZHENG***, *L.M. PATTERSON, H.-R. BERTHOUD:* Food reward: orexin-signaling in ventral tegmental area contributes to high-fat intake induced by accumbens opioid stimulation.

21. *N.S. NAIR,* ***I.M. BRENNAN***, *T.J. LITTLE, D. GENTILCORE, T. HAUSKEN, K.L. JONES, J.M. WISHART, M. HOROWITZ, C. FEINLE-BISSET:* Day-to-day reproducibility of, and relationships between, energy intake, gastric emptying and plasma CCK and GLP-1 in healthy lean males.

22. ***K. DIEPVENS***, *D. HÄBERER, M. ARNOLD, M.S. WESTERTERP-PLANTENGA:* Different relationships with respect to satiety between exogenous and endogenous peptides.

23. ***M.J. DONOVAN***, *H.E. RAYBOULD:* Interaction between the ghrelin and CCK1 receptors and its effect on meal patterns.

24. ***C.M. LO***, *D. ZHANG, K. PEARSON, L. MA, R.R. SAKAI, W.S. DAVIDSON, S.C. WOODS, P. TSO:* Interaction of apolipoprotein AIV with cholecystokinin on food intake.

25. ***C. PHILES***, *R.C. RITTER:* Paracrine-like cytoarchitecture and proximity to nerve fibers in a subpopulation of GLP-1 and CCK immunoreactive enteric mucosal cells.

26. *P.K. CHELIKANI, A.C. HAVER,* ***R.D. REIDELBERGER****:* Effects of intraperitoneal versus intravenous infusion of anorexigenic compounds on food intake in freely-feeding rats.

27. ***E.B. RÜTTIMANN****, M. ARNOLD, N. GEARY, W. LANGHANS:* Brief, meal-contingent intravenous infusion of glucagon-like peptide-1 (GLP-1) decreases spontaneous meal size in rats.

28. ***T. WOLDEN-HANSON****:* ICV ghrelin increases food intake in senescent male Brown Norway rats.

29. ***A-M. TORREGROSSA****, A.V. AZZARA, M.D. DEARING:* CCK-8 does not have a differential effect on plant-toxin ingestion in the white-throated woodrat (Neotoma albigula).

30. *S.J. RABOIN, J.R. REEVE, JR., G.M. GREEN, J. E. COX, J. GIBBS, L.J. PEREZ, M.S. COOPER, J. OVERDUIN,* ***A.I. SAYEGH****:* Differential effects of exogenous CCK-8 and CCK-58 on food intake.

31. ***P.Y. WIELINGA****, B. ALDER, T.A. LUTZ:* Effects of amylin and salmon calcitonin on energy expenditure and body temperature.

32. *D.B GUARD,* ***M. COVASA****, R.C. RITTER, and G.A. BURNS:* Increased food intake evoked by DVC injection of NR2B, NR2C/NR2D preferring NMDA receptor antagonists.

33. ***K. CZAJA****, G.A. BURNS, R.C. RITTER:* Hindbrain glutamatergic control of meal size: Evidence for participation of specific N-methyl-D-aspartate receptor (NMDAR) phenotypes on myelinated vagal afferent neurons.

34. *R.L. TOWNSEND, H. ZHENG, L.M. PATTERSON,* ***H-R.BERTHOUD****:* Food intake suppression induced by fourth ventricular CCK and MTII but not oxytocin and bombesin is partially mediated by activation of the ERK intracellular signaling cascade.

35. ***J.P. BAIRD,*** *C.J. GUENTHNER:* d-Amphetamine enhances gustatory responses in brief access tests, but withdrawal does not produce gustatory anhedonia.

36. ***L.M. BARTOSHUK****, S.E. MARINO, D.J. SNYDER:* Age and hormonal effects on sweet taste and preference.

37. ***M. COVASA****, N.L. NOWAK, A. HAJNAL:* Increased intake of oil in obese OLETF rat is driven by caloric deficit.

38. ***A. HAJNAL****, P. KOVACS, M. COVASA, R.N. COONEY:* Gastric bypass surgery restores altered neuronal coding for sweet taste in obese OLETF rats.

39. *E. ANDERZHANOVA, M. COVASA,* ***A. HAJNAL****:* Altered basal and stimulated accumbens dopamine release in obese OLETF rats: a quantitative microdialysis study.

40. ***C.T. KING***, *M. GARCEA, A.C. SPECTOR:* Effects of cross-wiring the lingual taste nerves on quinine-stimulated fos-immunoreactivity in the rat parabrachial nucleus.

41. ***A. SCLAFANI****:* Carbohydrate and fat preferences in the "Tasteless" P2X2/P2X3 Double Knockout Mouse.

42. ***J.M. STRATFORD***, *K.S. CURTIS, R.J. CONTRERAS:* Lingual co-application of sodium and linoleic acid affects chorda tympani nerve electrophysiological responses.

43. ***B.P. SHAH***, *T. YU, D.R. HANSEN, T.A. GILBERTSON:* Fatty acid responses in an enteroendocrine cell line: chemosensory cues for dietary fat.

44. *R. BAYLEY, L. MATTHEWS, E. STREET, J. ALMEIDA,* ***B. RAUDENBUSH****:* Ability of gum flavors to distract participants from painful stimuli: Differential effects of retronasal vs. orthonasal scent administration.

Thursday, July 26, 2007

7:30-8:30 CONTINENTAL BREAKFAST/EXHIBITORS' DISPLAY – FOYER

Symposium 2 - 8:30-10:30
Location: Storm Peak
Energy Expenditure in Relation to Food Intake and Body Weight Regulation
Chair: ***P. WIELINGA***

Supported by GlaxoSmithKline

8:30 ***H. GRILL****, T. BARTNESS, R. HARRIS:* Distributed control of energy expenditure: contributions from the isolated caudal brainstem.

9:00 ***D. RICHARD****:* The control of the sympathetic nervous system (SNS) - mediated uncoupling-protein 1 (UCP1) activity.

9:30 ***K. WESTERTERP****:* Determinants of energy expenditure and energy balance.

10:00 ***D. BESSESEN***: Effects of overfeeding in obesity prone and obesity resistant rats and humans

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Oral Session 2 - 8:30-10:30
Location: Mt. Werner
Eating Disorders
Chair: ***M. BOGGIANO***

8:30 *N.M. AVENA, M. BOCARSLY, P. RADA, A. KIM,* ***B.G. HOEBEL****:* Sugar withdrawal induced by fasting in rats.

8:45 ***L.A.W. VERHAGEN***[#]*, M.C.M. LUIJENDIJK, J.J.G. HILLEBRAND, R.A.H. ADAN:* Food-anticipatory activity in activity-based anorexia: the involvement of dopamine.

9:00 *E. STICE, J.A. COOPER, D.A. SCHOELLER, K. TAPPE,* ***M.R. LOWE****:* Are dietary restraint scales valid measures of longer-term dietary restriction? Objective biological and behavioral data suggest not.

9:15 ***L. CHAMBERS***[#]*, M.R. YEOMANS:* Low fasting PYY3-36 levels in healthy women with high dietary restraint.

9:30 *E.M. FORMAN, K.L. HOFFMAN, K.B. MCGRATH, J.D. HERBERT, L.L. BRANDSMA,* ***M.R. LOWE****:* The Power of Food Scale predicts chocolate cravings and consumption and response to a cravings intervention.

9:45 ***C.O. OCHNER***[#]*, M.R. LOWE, J. KOUNIOS, D. GREEN, J. VAN STEENBURGH:* Binge eating, appetitive responsiveness, and disinhibition: relation to asymmetrical activation in the human brain.

10:00 ***J.A. NASSER****, S. M. EVANS, A. GELIEBTER, F. X. PI-SUNYER, R. W. FOLTIN:* Using a computer-based operant task and progressive ratio to estimate eating independent of hunger in obese women with binge eating disorder.

10:15 ***A.J. STUNKARD****, K.C. ALLISON, J.D. LUNDGREN, J.P. O'REARDON:* Serotonergic mechanisms in the night eating syndrome.

10:30-11:00 BREAK/EXHIBITORS' DISPLAY - FOYER

11:00-1200 Masterfoods Keynote Lecture Series *- Introduction: A. SPECTOR.*
MICHAEL G. TORDOFF*:* Genetics of Calcium Appetite. Location: Storm Peak/Mt. Werner

Oral Session 3 - 1:30-3:30
Location: Storm Peak
Taste/Water-NaCl Ingestion
Chair: ***A. SCLAFANI***

1:30 ***C.L. GROBE****, A.C. SPECTOR:* Application of an operant generalization paradigm to characterize the perceptual quality of common taste compounds in a rat model.

1:45 ***C.A.F. ANDRADE****, G.M.F. ANDRADE, L.A. DE LUCA JR, D.S.A. COLOMBARI, J. V. MENANI:* Interactions between serotoninergic, alpha2-adrenergic, gabaergic and opioidergic mechanisms of the lateral parabrachial nucleus in the control of NaCl intake.

2:00 ***L.A. DE LUCA JR.****, D.T.B. PEREIRA, J.V. MENANI, R.C. VENDRAMINI:* Damage to the central amygdala (CeA) induces differential c-fos expresssion in the parabrachial and paraventricular nucleus in the water deprivation-partial rehydration protocol.

2:15 ***F.W. FLYNN****, G.E. HALEY, D.D. JENSEN, K. SCHAMBER:* Central injections of tachykinin NK3 receptor agonists inhibit salt appetite and cause translocation of the NK3 receptor to the nuclei of neurons in the paraventricular nucleus of the hypothalamus.

2:30 ***H.R. KISSILEFF****, S.J. MCNALLY, R. GORDON, M. GONDEK-BROWN, J. FARKAS, A. SCLAFANI:* Measuring food reward value in humans.

2:45 ***R.M.A.J. RUIJSCHOP****, A.E.M. BOELRIJK, M.J.M. BURGERING, J.A. DE RU, C. DE GRAAF, M.S. WESTERTERP-PLANTENGA:* Engineering satiating liquid food products by altering the extent of flavour release.

3:00 ***C.D. DOTSON****, A.E.T. ELSON, H. SHAW, X. SHI, C.M. DAMCOTT, A. NAJ, S. SNITKER, N.I. STEINLE, S.D. MUNGER:* Taste receptor polymorphisms in the Old Order Amish: Associations with obesity and related traits.

3:15 ***A.J.P.G. SMEETS****, M.P.G.M. LEJEUNE, M.S. WESTERTERP-PLANTENGA:* The effect of oral fat perception compared to fat ingestion on energy expenditure and appetite profile.

Oral Session 4 - 1:30-3:30
Location: Mt. Werner
GI Peptides and Satiety I
Chair: ***C. FEINLE-BISSET***

Supported by Research Diets, Inc.

1:30 ***M.A. CORNIER****, S.S. VON KAENEL, D.H. BESSESEN:* The effects of short-term overfeeding on ad libitum energy intake.

1:45 ***N. ZIJLSTRA****, M. MARS, R.A. DE WIJK, M.S. WESTERTERP-PLANTENGA, C. DE GRAAF:* The effect of viscosity on ad libitum food intake and satiety hormones.

2:00 ***K.T. BORER*** *, E. WUORINEN, C. BURANT:* Associations of plasma ghrelin, leptin and cholecystokinin (CCK) with sensations of hunger and fullness during manipulations of energy balance by meal size, exercise and intravenous nutrient replacement.

2:15 ***M. BALDINGER****, R. RUBIN, N. WRIGHT, L. FLANCBAUM, A. GELIEBTER:* Change in hunger, fullness, ghrelin, pyy and glp-1 in relation to a fixed test meal pre and post rouen-Y gastric bypass (RYGBS).

2:30 ***I.M. BRENNAN****, T.J. LITTLE, K.L. FELTRIN, M. HOROWITZ, C. FEINLE-BISSET:* Dose-dependent effects of cholecystokinin (CCK-8) on pyloric motility, appetite and energy intake.

2:45 ***S.F. LEIBOWITZ****, V. GAYSINSKAYA, O. KARATAYEV:* Acute hyperphagia on a high-fat diet: relation to circulating triglycerides and orexigenic peptide.

3:00 ***M.R. HAYES****, H.J. GRILL:* The caudal brainstem is sufficient to mediate inhibition of gastric emptying by peripheral glucagon-like-peptide-1 receptor activation.

3:15 ***J. ATHANACIO*** *, T. COFFEY, C. MACK, D. PARKES, J. ROTH:* Combination therapy with amylin and PYY(3-36) in diet induced obese rats: A response surface analysis.

3:30-4:00 BREAK/EXHIBITORS' DISPLAY - FOYER

NIH Sponsored Symposium – 4:00-6:00
Location: Storm Peak/Mt. Werner
Neurobiology of Anorexia Nervosa, Bulimia Nervosa and Related Behaviors
Chair: ***K.A. HALMI***

4:00 ***K.A. HALMI****, W.H. KAYE, PRICE FOUNDATION COLLABORATORS:* Susceptibility genes for anorexia and bulimia nervosa.

4:30 ***W.H. KAYE****, U.F. BAILER, A. WAGNER, G.K. FRANK:* Brain imaging studies: New insights into puzzling symptoms in anorexia nervosa.

5:00 ***A. GELIEBTER****:* Peripheral mechanisms in bulimia nervosa and binge eating disorder.

5:30 ***J.J.G. HILLEBRAND****, C.E. DERIJKE, L.A.W. VERHAGEN#, M. DEKROM, A.A. VAN ELBURG, H.W. HOEK, M.J.H. KAS, R.A.H. ADAN:* Neurobiological parameters and anorexia nervosa: findings from animal studies.

Poster Session 2 – 6:00-8:00
Location: Sunshine Peak/Twilight – WINE & CHEESE

45. ***K. ACKROFF****, A. SCLAFANI:* Dietary fat content and flavor reinforcement by intragastric sucrose and corn oil in rats.

46. *D. HÄBERER, K. DIEPVENS, N. GEARY,* ***W. LANGHANS****:* Intragastric infusion of pea protein hydrolysate reduces food intake more than pea protein.

47. *N.L. KEIM,* ***W.F. HORN****:* Hunger and fullness ratings in response to meals based on whole grains vs. refined grains.

48. ***T. IMADA****, H. ZAI, M. KUSANO, S. FUJITA, M. KAWAI, A. OKIYAMA, Y. SHIMOYAMA, M. MAEDA, A. NAGOSHI, T. HIGUCHI, S. KURIBAYASHI, O. KAWAMURA, M. MORI, T. TANAKA, H. UNEYAMA:* Effect of dietary free glutamate on gastric emptying rate and postprandial sensation for protein-rich liquid diet.

49. ***A. LLUCH****, E. BOELSMA, S. VINOY, D. L'HEUREUX-BOURON, H.F.J. HENDRIKS:* Enrichment of yoghurt with protein and fibre reduces appetite.

50. *A. GONZALEZ,* ***A. LÓPEZ-ESPINOZA****, A. G. MARTÍNEZ, V. AGUILERA, A. GALINDO, C. DE LA TORRE-IBARRA:* Effects of food supplements on feeding behavior and body weight in rats.

51. ***F. MCKIERNAN****, J.H. HOLLIS, R.D. MATTES:* Short-term dietary compensation in free-living adults.

52. ***J.A.H. VAN VUGHT****, A.G. NIEUWENHUIZEN, R.J. BRUMMER, M.S. WESTERTERP-PLANTENGA:* The effects of oral ingestion of soy protein on the somatotropic axis.

53. ***S.E. LA FLEUR****, A.J. VAN ROZEN, M.C.M. LUIJENDIJK, LJ.M.J. VANDERSCHUREN, R.A.H. ADAN:* Maladaptive changes in response to a choice diet with fat and sugar.

54. ***B.A. CASSADY****, J.H. HOLLIS, R.D. MATTES.* The effect of mastication on appetite and lipid bioaccessibility.

55. *A. REED,* ***B. RAUDENBUSH****:* Effects of green tea on cognition, perceived workload, mood, and endurance.

56. *R. DRAKE, D. FELBAUM, C. HUNTLEY, A. REED, L. MATTHEWS,* ***B. RAUDENBUSH****:* Effects of chocolate consumption on enhancing cognitive performance.

57. ***A.L. TRACY****, J.F. DAVIS*[#]*, J.U. HEIMAN, J.D. SCHURDAK, D.J. CLEGG, S.C. BENOIT:* Effect of high-fat diet induced obesity on the efficacy of food and drug reinforcers.

58. ***A. MANSOURI****, M.D. KOSS, N. GEARY, W. LANGHANS, M. LEONHARDT:* Mercaptoacetate's feeding stimulatory effect is not increased in rats fed a medium-chain triglyceride-rich diet.

59. ***M. H. EMOND****, J. FERGUSON:* The effects of a conditioned taste aversion on food intake and flavor preference in the presence of a stressor in female rats.

60. ***G.J. GOLDEN****, T.A. HOUPT:* Intraoral intake and c-Fos induction in rats with conditioned flavor taste preferences.

61. ***T. INUI****, T. SHIMURA, T. YAMAMOTO:* The GABAergic system in the ventral pallidum is involved in conditioned taste aversion in rats.

62. ***B.S. KWON****, T.A. HOUPT:* Expression of c-Fos-LacZ transgene after LiCl administration.

63. ***L. MARINI****, J. TRUSHENSKI, D.L. WENDT, A.D. STRADER:* Acute and long-term metabolic consequences of chronic consumption of dietary sweeteners.

64. ***J.T. ALEXANDER-CHACKO****, D.K. SINDELAR:* Characterization of the alcohol-preferring P Rat on normal chow and a high-fat diets.

65. ***M.L. PELCHAT****, E.V. IZBICKI, D. OSLIN:* The sweet taste of excess.

66. ***R.B. KANAREK****, W. FOULDS-MATHES:* Activity-induced anorexia in rats augments the development of tolerance to morphine.

67. *T.W. HAHN, J.M. WIEBELHAUS, R.L. THAM, T.R. SMITH, E.E. EWAN, M.K. GRACE, A.S. LEVINE,* ***D.C. JEWETT****:* Effects of opioids in subjects trained to discriminate 22 from 2 hours food deprivation.

68. ***A. MITRA****, K.L. ROBERTSON, K. ALVERS, N.E. ROWLAND:* Effect of prenatal or postnatal high fat diet on physiology and behavior of borderline hypertensive rats (BHR).

69. ***M. SCHROEDER****, Y. LAVI-AVNON, O. ZAGOORY –SHARON, T.H. MORAN, A. WELLER:* Maternal and offspring's involvement in the development of pre-obesity in infant OLETF rats.

70. ***M. SCHROEDER****, O. ZAGOORY-SHARON, T.H. MORAN, S. BI, A. WELLER:* Food restriction from weaning to puberty reduces long term food intake, adipocyte area and obesity in male but not in female OLETF rats.

71. ***S.B. YOO****, V. RYU, S. LEE, J.W. CHANG, J.W. JAHNG:* The arcuate expression of NPY, POMC and CART responding to food deprivation was exaggerated by experience of maternal separation in female rats.

72. ***M. DAILEY****, C.H. VAUGHAN, T.J. BARTNESS:* Fasting-induced increases in food hoarding are elevated in adult hamsters treated neonatally with monosodium glutamate (MSG).

73. ***K.P. MYERS****, C. PICKERING:* Sensory variety during nursing increases young rats' acceptance of novel flavors after weaning.

74. *M. VICTORIANO, A. DIANE, G. FROMENTIN,* ***D. TOME****, C. LARUE-ACHAGIOTIS:* High protein diet from sukling to adulthood induces hyperinsulinemia later in life in adult rats.

75. ***V. AGUILERA****, A. LÓPEZ-ESPINOZA, A. G. MARTÍNEZ, A. GONZALEZ, A. GALINDO, C. DE LA TORRE-IBARRA:* Sex and running: Eating behavior differences.

76. ***J.B. CHAMBERS****, J.D. SCHURDAK, S.C. BENOIT, D.J. CLEGG:* Behavioral and metabolic phenotyping of GPR30 and neuronal estrogen receptor-alpha knockout mice.

77. ***A. LÓPEZ-ESPINOZA****, C. DE LA TORRE-IBARRA, V. AGUILERA, A. GALINDO, A. G. MARTÍNEZ, A. GONZALEZ:* Sex and social interaction on novel food consumption.

78. ***H.M. RIVERA****, S.C. NORRBIN, K.S. CURTIS, L.A. ECKEL:* Intake of dilute sweet solutions is influenced by stage of the rat's estrous cycle: evidence for a shift in palatability.

79. ***J. SANTOLLO****, L.A. ECKEL:* Estradiol decreases the orexigenic effect of NPY, but not AgRP, in ovariectomized rats.

80. ***C.J. KEMP****[#], J. CALDWELL-LAWSON, D.J. CLEGG, S.C. BENOIT:* Interaction of MMP-3 and sex hormones in the control of energy balance.

81. *S. THAMMACHAROEN, T.A. LUTZ, N. GEARY,* ***L. ASARIAN****:* Anomalously rapid effect of the estrogen receptor- a agonist PPT on food intake in ovariectomized rats.

82. ***N.E. ROWLAND***, *M.A. CHANEY, C.H. VAUGHAN:* Food intake and meal patterns of mice under various schedules of food reinforcement.

83. ***N.E. ROWLAND***, *K.L. ROBERTSON, A. BARDIN, E. COONS:* Food intake and meal patterns of Lewis rats with continuous access to nicotine self-administration.

84. *D. ATALAYER,* ***N.E. ROWLAND****:* Comparison of lever press and nose poke operants for acquiring the demand function for food intake and meal patterns in mice.

85. ***S.J. MELHORN***, *E.G. KRAUSE#, J.D. JOHNSON, R.R. SAKAI:* Meal patterns and hypothalamic gene expression following chronic social stress.

86. ***L.L. BELLINGER***, *P.J. WELLMAN, P.R. KRAMER:* Nicotine (NIC) reduces body weight (BW) by decreasing food intake (FI).

87. ***A-M. TORREGROSSA***, *A.V. AZZARA, M.D. DEARING:* Caching as a mechanism for detoxification of plant toxins in herbivorous rodents (genus Neotoma).

88. ***C.N. OCHNER#***, *A. GELIEBTER, C.L. BAUER, S.A. HASHIM:* Effects of strength and aerobic training on metabolic syndrome, insulin, and testosterone levels in dieting obese subjects.

Friday, July 27, 2007

7:30-8:30 CONTINENTAL BREAKFAST/EXHIBITORS' DISPLAY – FOYER

Symposium 3 - 8:30-10:30
Location: Storm Peak
Early Life Events and Long Term Obesity Risk
Chair: ***T.L. BALE***

8:30 *K.M. CARLIN,* ***T.L. BALE****:* A mechanistic examination of the impact of maternal high fat diet on long-term offspring obesity risk.

9:00 ***K.L.K. TAMASHIRO****, J. HYUN, J.I. KOENIG, T.H. MORAN:* Prenatal stress and nutrition effects on metabolic phenotype of offspring.

9:30 *K.W. WHITAKER,* ***T.M. REYES****:* In utero programming of metabolic dysregulation: Focus on CNS mechanisms.

10:00 ***P.P. SILVEIRA****:* Early life events and the programming of feeding behavior.

Oral Session 5 - 8:30-10:30
Location: Mt. Werner
Macronutrient Effects
Chair: ***K.P. KINZIG***

8:30 ***N.V. DIPATRIZIO****, K.J. SIMANSKY:* Cannabinoid CB1 receptors of the parabrachial nucleus in selective modulation of various diets: a comparison with opioids.

8:45 ***B. BOUTER****, N. GEARY, W. LANGHANS, L. ASARIAN:* Diet- and obesity-independent improvements in insulin sensitivity in mice genetically deficient in tumor necrosis factor-a (TNF-a KO).

9:00 ***K.L. FELTRIN****, T.J. LITTLE, J.H. MEYER, T. RADES, M. HOROWITZ, C. FEINLE-BISSET:* Comparative effects of the free fatty acids, lauric acid and oleic acid, on antropyloric motility and energy intake in healthy young men.

9:15 ***S. JARVANDI****, L. THIBAULT, D.A. BOOTH:* Obesity from high-fat diet weakened learning of anticipatory eating in rats.

9:30 *M. POTIER, A. MARSSET-BAGLIERI, C. MORENS, A. BENSAID,* ***D. TOME****, G. FROMENTIN:* Decrease in food intake during a meal following a protein preload could be related to acute hyperleptinemia.

9:45 ***E.J. BERTENSHAW****, A. LLUCH, M.R.YEOMANS:* Increasing the protein content of a beverage reduces subsequent intake at the next meal.

10:00 ***M.P.G.M. LEJEUNE****, A.J.P.G. SMEETS, M.S WESTERTERP-PLANTENGA:* Effects of a high-protein diet with or without monosodium-glutamate in combination with inosine-monophosphate-5 on 24-h energy metabolism and appetite profile.

10:15 ***M.A.B. VELDHORST***[#]*, A.G. NIEUWENHUIZEN, A. HOCHSTENBACH-WAELEN, K.R. WESTERTERP, M.P.K.J. ENGELEN, R.J. BRUMMER, N.E.P. DEUTZ, M.S. WESTERTERP-PLANTENGA:* Effects of high or normal casein-, soy-, or whey with or without GMP- protein breakfasts on satiety, 'satiety' hormones, and plasma amino acid responses

10:30-11:00 BREAK/EXHIBITORS' DISPLAY - FOYER

11:00-1200 Masterfoods Keynote Lecture Series *- Introduction: H-R. BERTHOUD.*
ERIC RAVUSSIN: Effect of Caloric Restriction on Biomarkers of Longevity in Humans: Physiological and Molecular Responses. Location: Storm Peak/Mt. Werner

NIH Grant Workshop 1:30-2:30. Location: Storm Peak/Mt. Werner

1:30-2:00 ***MICHAEL SELMANOFF***, Ph.D. Scientific Review Administrator, BRS and NNB Study Sections, Integrative, Functional and Cognitive Neuroscience IRG, Center for Scientific Review, National Institutes of Health.

2:00-2:30 ***JUDITH PODSKALNY***, Ph.D. Director, Research Fellowship & Career Development and Digestive Disease Centers Programs, Division of Digestive Diseases and Nutrition, National Institute of Diabetes & Digestive & Kidney Diseases, National Institutes of Health

Presidential Symposium – 4:00-6:00
Location: Storm Peak/Mt. Werner
Chair: ***H-R. BERTHOUD***

Supported by educational grants from NIH and Takeda Pharmaceuticals North America

4:00 ***S. PECINA:*** Hedonic hotspots in the brain.

4:30 *C.L. WHITE, M.J. BARNES, A. WHITTINGTON, Z. WANG, W.T. CEFALU, G.A. BRAY,* ***C.D. MORRISON****:* PTP1B: A molecular mediator of leptin resistance.

5:00 *M. FAOUZI, G.M. LEINNINGER, G. LOUIS, R. LESHAN, J.C. JONES, C.J. RHODES,* ***H. MÜNZBERG****:* Leptin signaling in the dorsomedial hypothalamus and its role in feeding circuits.

5:30 ***D. SANDOVAL****:* CNS GLP-1 Regulation of peripheral glucose homeostasis.

Poster Session 3 – 6:00-8:00
Location: Sunshine Peak/Twilight – WINE & CHEESE

89. ***J. BLUNDELL****, H. GREEN:* Appetite regulation and functional food claims: Experimental methodology and physiological targets.

90. ***K. DIEPVENS****, J. STEIJNS, P. ZUURENDONK, M.S. WESTERTERP-PLANTENGA:* Short-term /effects of a novel fat emulsion on appetite and food intake in different weight and age groups.

91. ***G. FINLAYSON****[#], N. KING, J. BLUNDELL:* Implicit wanting in relation to explicit liking and wanting for food: implications for appetite control.

92. ***L.J. NOLAN****, L.B. HALPERIN, A. GELIEBTER:* Emotional appetite questionnaire: Construct validity and relationship with BMI.

93. ***J.A. NASSER****, E. BOO, F.X. PI-SUNYER A. GELIEBTER:* Interaction of food deprivation state and food intake recall in humans.

94. *T. CESSNA,* ***B. RAUDENBUSH****, A. REED, R. HUNKER:* Effects of video game play on snacking behavior.

95. *W. NEFTI, N.I. DARCEL, G. FROMENTIN,* ***D. TOMÉ*** *:* Long term exposure to high protein diet or high fat diet have opposite effects on vagal afferent sensitivity to luminal macronutrients, ip cholecystokinin and Serotonin.

96. ***C. CHOTIWAT****, C. SHARP, K. TEFF, R.B.S. HARRIS:* Feeding a high fructose diet induces leptin resistance in rats.

97. *B.D. HUDSON, M.F. WIATER,* ***S. RITTER****:* Chronic central leptin treatment increases protein intake in rats.

98. *D. HUDSON, M. F. WIATER,* ***S. RITTER****:* Hindbrain catecholamine neurons contribute to control of daily food intake during chronic leptin treatment.

99. ***L. YANG****, J. HYUN, T.H. MORAN, S. BI,:* Voluntary running activity is not sufficient for preventing the obesity of Koletsky (fak/fak) rats.

100. *E.W. KELSO,* ***R.B.S. HARRIS****:* In vitro inhibition of adipocyte lipogenesis in adipocytes exposed to serum from obese hyperleptinemic rats.

101. ***D.M. PENN****[#], R.B.S. HARRIS:* The effect of leptin on adipocyte metabolism in leptin-responsive, partially leptin-resistant, and leptin-resistant animals.

102. ***J.P. BAIRD**, A.L. PECORA:* Effects of neuropeptide Y and melanocortin 3/4R antagonist SHU9119 injections into the lateral parabrachial nucleus on intake and licking for water and sucrose solutions.

103. ***J.P. BAIRD**, P.V. HOLMES, K. WICKWIRE, R. DAILEY, S.Q. GIRAUDO:* Forebrain agouti-related protein (AgRP) injections increase appetitive but not consummatory feeding behaviors in the rat.

104. ***J.P. BAIRD**, C. RIOS, J.L. LOVELAND, J. BECK:* Effects of hindbrain melanin-concentrating hormone and neuropeptide Y administration on licking for water, saccharin, and sucrose solutions.

105. *S. WAN, K. N. BROWNING, R. A. TRAVAGLI, H. ZHENG,* ***H-R. BERTHOUD**:* Melanocortinergic modulation of food intake in the medulla: Evidence for presynaptic MC4-receptors on vagal afferents.

106. ***S.Z. GOODIN**, A.R. KIECHLER, M.L. SMITH[#], D.L. WENDT, A.D. STRADER:* Differential effects of centrally-administered agouti-related peptide in male and female rats.

107. ***K. TUCKER**, J.M. OVERTON, D.A. FADOOL:* Kv1.3 gene-targeted deletion reduces fat deposition and total body weight in melanocortin 4 receptor (MC4R)-null mice; a model of hypothalamic-driven late-onset obesity.

108. ***C.H.VAUGHAN**, C.K. SONG, E. KEEN-RHINEHART, T.J. BARTNESS:* Effects of central melanocortin administration on brown adipose tissue (BAT) thermogenesis.

109. ***S. BI**, K.A. SCOTT, K.L. TAMASHIRO, J. HYUN, T.H. MORAN:* Knockdown of dorsomedial hypothalamic NPY gene expression prevents hyperphagia and obesity of OLETF rats lacking CCK1 receptors.

110. ***V. RYU**, J.-H. LEE, S.B. YOO, J.W. JAHNG:* Dysregulation in NPY expression and feeding behavior in adolescent rats by neonatal maternal separation.

111. *J.H. KIM, H.Y. JOUNG, S.A. KANG, K.H. PYUN,* ***I. SHIM**:* Ginsenoside Rb1 as a suppressor in central modulation of feeding in the rat.

112. ***E.E. LADENHEIM**, R.R. BEHLES, S. BI, T.H. MORAN:* Neuropeptide Y Y1 and Y5 receptors are co-expressed with GRP mRNA in the rat hypothalamic paraventricular nucleus.

113. *K. SCHILLER, L. SUCHOR, F. SMITH, D. PORTER, L. MURT, J. ALLEN, A.C. SMITH,* ***L.A. CAMPFIELD**:* Program ENERGY: An Extension Focusing on Brain and Nervous System Science for 6th Grade Elementary School Children.

114. *D. PORTER, F. SMITH, J. ALLEN, A.C. SMITH,* ***L.A. CAMPFIELD**:* Program ENERGY: Successful Dissemination to an Isolated High Risk Rural Location, Cortez, CO.

115. ***L.A. CAMPFIELD****, F.J. SMITH, D. PORTER, K. SCHILLER, L. MURT, J. ALLEN, L. SUCHOR, A.C. SMITH:* Program ENERGY: Scientists and Students in the Classroom Tackling Type 2 Diabetes and Obesity in Elementary Schools.

116. ***F.J. SMITH****, D. PORTER, A. HOLLIDAY, S. DURHAM, L. MURT, J. ALLEN, K. SCHILLER, A. C. SMITH, L.A. CAMPFIELD:* Program ENERGY: Scientists and Students in the Classroom Tackle Type 2 Diabetes and Obesity in Elementary Schools in Three States.

117. ***R. NORGREN****, S. PECKINS, S. DAYAWANSA:* Asymmetric lesions of the parabrachial nuclei and lateral hypothalamus block sodium appetite in rats.

118. *M.L. HOFFMANN,* ***E.M. STRICKER****:* Gastric emptying of ingested 0.15 M NaCl solution by rats with thirst and/or salt appetite.

119. ***M.P. LAWLER****, M.G. TORDOFF:* Food, water and NaCl consumption by 14 strains of rats.

120. *S. MCBRIDE,* ***F.W. FLYNN****:* Brain vasopressin involvement in behavioral sensitization to amphetamines and drinking hypertonic salt solutions.

121. ***A. SCLAFANI****, K. ACKROFF, N. ABUMRAD:* Fat preference and acceptance in the CD36 knockout mice.

122. ***R. SHIBATA****, M. KAMEISHI, T. KONDOH, K. TORII:* Mesolimbic dopaminergic system relates to sucrose intake, but not intake of salt, umami compounds or lysine in rats.

123. ***G. SCALERA****, C. BENASSI, A. BIGIANI:* Sapid solutions and food intake in repeated dehydration and rehydration periods in rats.

124. ***A. GALINDO****, A. LÓPEZ-ESPINOZA, A. G. MARTÍNEZ , V. AGUILERA, A. GONZALEZ, C. DE LA TORRE-IBARRA:* Glucose and sucrose intake affects feeding behavior: a parametric analysis.

125. ***C. LIN****, N. BOSAK, X. LI, M. L. THEODORIDES, D.R. REED, G. K. BEAUCHAMP, A. A. BACHMANOV:* Genetic control of sucrose intake by mice.

126. ***L. RINAMAN****, A. SCLAFANI, R.R. VOLLMER, J. MIEDLAR, J.A. AMICO:* Oxytocin knockout mice overconsume palatable carbohydrate solutions, but not palatable lipid solutions.

127. ***J.F. DAVIS***[#]*, E.G. KRAUSE*[#]*, S.C. BENOIT, R.R. SAKAI:* Social stress attenuates motivation for food reward.

128. ***J. HUBERT****, D. WEINBERG, C.P. SHEN, T. FONG, A.M. STRACK, D.E. MACINTYRE, S.J. LEE,:* The effects of a brain penetrant neurotensin analog, NT-2, on food intake, body weight and activity in the diet induced obese (DIO) mouse.

129. *E. LONDON, G. LALA, R.BERGER, A. PANZENBECK, A. KOHLI, M.RENNER, A.JACKSON, T. RAYNOR, K. LOYA ,* ***T.W. CASTONGUAY****:* Sucrose access and 11 ß-hydroxysteroid dehydrogenase-1 message in liver and adipose tissue in rats.

130. ***M.P. LAWLER****, D.R. REED, M.G. TORDOFF:* How many genes control body weight?

131. ***K-S. KIM****:* The effects of pentoxifylline on human preadipocyte, adipocyte, keratinocyte and lung fibroblast.

132. ***D.H. MORALEJO****, J.M. FULLER, P.M. TREUTING, T.D. TUPPLING, C.T. HANSEN, Å. LERNMARK:* Introgression of Leptin receptor (lepr) mutation from Koletsky rat renders obesity and diabetes in the Bio-Breeding (BB) rat.

133. ***B.C. DE JONGHE****, C.C. HORN:* Common hepatic branch vagotomy reduces anorexia and pica produced by a cancer chemotherapy agent in the rat.

134. ***M.J. BARNES****, S.D. PRIMEAUX, C. BLACKMON, C. WEDGEWORTH, G.A. BRAY:* The preference for a high fat diet in food deprived animals is attenuated by ß-Funaltrexamine.

Saturday, July 28, 2007

7:30-8:30 CONTINENTAL BREAKFAST/EXHIBITORS' DISPLAY – FOYER

Symposium 4 - 8:30-10:30
Location: Storm Peak
Human Thirst and Sodium Appetite
Chair: ***J. ARGUELLES***

8:30 ***A.K. JOHNSON****:* Why do we love salt?

9:00 ***R.D. MATTES****:* The relationship between human thirst and feeding.

9:30 ***M. LESHEM****:* Human sodium appetite, habit and homeostasis.

10:00 ***M.J. MCKINLEY****, T. BOWALA, G.E. EGAN, M.J. FARRELL, P. FOX, M.L. MATHAI, P.A. PHILLIPS, R. SHADE, D.A. DENTON*. Age-related changes in thirst and associated neural activity in human subjects.

Oral Session 6 - 8:30-10:30
Location: Mt. Werner
GI Peptides and Satiety II
Chair: ***M.A. COWLEY***

8:30 ***D.L. WILLIAMS****, D.G. BASKIN, M.W. SCHWARTZ:* The role of intestinal glucagon-like peptide 1 in satiety.

8:45 *S. EISEN, P.A. RUSHING, N. GEARY,* ***G.P. SMITH****:* The satiating effect of CCK-33 is produced by hormonal and paracrine mechanisms, but CCK-8 acts through only a paracrine mechanism in the rat.

9:00 ***D.A. LEVITSKY****, G.T. HUNG:* Long term lack of compensation of energy intake for meal replacements at lunch.

9:15 ***C. SCOTT****, W. PASMAN, J. HEIMERIKX, C. RUBINGH, R. VAN DEN BERG, M. O'SHEA, L. GAMBELLI, H. HENDRIKS, L. MENNEN, A. EINERHAND:* PinnoThin suppresses appetite in overweight women.

9:30 ***S.E. KANOSKI****, E.K. WALLS, J.J. FIELDS, T.L. DAVIDSON:* Interoceptive signals and postingestive consequences produced by regulatory peptides.

9:45 ***J.M. BRUNSTROM****, L.L. WILKINSON:* Conditioning expectations about the satiating quality of food.

10:00 *T.J. LITTLE, A. RUSSO, M. HOROWITZ, J.H. MEYER, D. SMYTH, K.L. JONES, J. WISHART, M. BELLON,* ***C. FEINLE-BISSET****:* The effects of free fatty acids on gastric emptying, plasma cholecystokinin (CCK) and peptide YY (PYY), appetite and energy intake in humans are more potent than those of triglycerides.

10:15 ***R.D. REIDELBERGER****, P.K. CHELIKANI, A.C. HAVER:* Effects of chronic intermittent intraperitoneal infusion of salmon calcitonin on food intake and adiposity in diet-induced obese rats.

10:30-11:00 BREAK/EXHIBITORS' DISPLAY - FOYER

11:00-1200 MasterFoods Keynote Lecture Series *- Introduction: R. RITTER.*
HELEN E. RAYBOULD: How does your gut taste? Nutrient detection in the gut wall. Location: Storm Peak/Mt. Werner

Symposium 5 - 1:30-3:30
Location: Storm Peak
The Social Psychology of Eating: Schacter's Legacy.
Chair: ***M. PELCHAT*** *and* ***P. PLINER***

1:30 ***P. PLINER****, S. SALVY, T. LEONE, D. JARRIN:* Eating and social influence: effects of multiple models and free-eating situations on intake.

2:00 ***C.P. HERMAN****:* The role of external cues in the control of food intake in humans: A tangled history.

2:30 ***J. POLIVY****, J. COELHO, D. HARGREAVES, A. FLEMING, C.P. HERMAN:* The effects of external cues on eating and body weight: Another look at obese humans and rats.

3:00 ***S. HIGGS****, A.C. WILLIAMSON, A.S. ATTWOOD:* Cognitive control of food intake: The effects of manipulating memory for recent eating.

Oral Session 7 - 1:30-3:30
Location: Mt. Werner
Ingestion - Central Mechanisms
Chair: ***S.C. BENOIT***

1:30 ***B.S. KOPF***[#]*, N. GEARY, W. LANGHANS, L. ASARIAN:* Intraperitoneal (ip) bacterial lipopolysaccharide (LPS) elicits rapid, graded increases in c-Fos expression in the raphe pallidus nucleus (RPa) and central nucleus of the amygdala (CeA) in male rats.

1:45 ***E.M. GALARCE***[#]*, M.A. MCDANNALD, P.C. HOLLAND:* BLA but not CEA is involved in the control of meal interruption cues on feeding behavior.

2:00 ***M. MASSI****, A. FEDELI, D. ECONOMIDOU, S. BRACONI, A. CIPPITELLI, R. CICCOCIOPPO:* Interactions between neuropeptide S and corticotropin releasing factor in the control of palatable food intake in rats.

2:15 ***R.J. MARTIN****, X. XI, C.D. MORRISON:* In vivo and in vitro manipulation of AGRP expression and carbohydrate response element binding protein by nutrient status.

2:30 *S. MICHEL, T. LUTZ,* ***T. RIEDIGER****:* Nutrients modulate amylin's effect on c-Fos expression in the area postrema and on food intake.

2:45 ***M.S. BYERLY****, L.A. COGBURN, T.E. PORTER:* Hypothalamic BDNF gene expression is modulated by thyroid hormones in vivo.

3:00 *W. ABPLANALP,* ***D.J. CLEGG****:* Translocation of PKC theta by fatty acids causes hypothalamic insulin resistance.

3:15 *M. OSTO, P.Y. WIELINGA, B. ALDER, N. WALSER,* ***T.A. LUTZ****:* Central modulation of the feeding response to amylin by leptin and insulin.

3:30-4:00 BREAK

4:00 ***Award Announcements*** - Storm Peak/Mt. Werner

Chair: ***H-R. BERTHOUD***
Gerard P. Smith Award for the best graduate student presentation
TestDiet Jackson Lab Scholarship for the best post-doc presentation

4:05 ***Alan N. Epstein Research Award***
Chair: ***T.L. DAVIDSON***
STEPHEN C. BENOIT*:* A poker enthusiast's guide to central insulin and the regulation of energy balance.

4:30 ***Distinguished Career Award:***
Chair: ***G.P. SMITH***
JACK D. DAVIS*:* Listening to the tongue

5:30 Please move to foyer so hotel can convert room for our banquet.

7:00 Cash Bar - FOYER

7:30 Banquet, entertainment and dancing

Abstracts for the 2007 SSIB meeting appear in *Appetite* 49: 272-341, 2007. Abstracts not published in *Appetite* per the authors' request are printed below in alphabetical order.

Increasing the protein content of a beverage reduces subsequent intake at the next meal. E.J. Bertenshaw*, A. Lluch** & M.R.Yeomans*, *University of Sussex, Brighton, UK. **Danone Research, RD 128, 91767 Palaiseau Cedex, France. Protein is widely considered to be more satiating than carbohydrate. However, it is unclear whether this rule applies equally to drinks as solid foods. Discrepancies in the literature may indicate a critical level of protein (PRO) is required in drinks, to obtain differences in appetite between CHO and PRO. In this repeated measures, cross-over design study, 28 lean male volunteers (18-35 yrs) ate a standard breakfast in the laboratory and 210 minutes later consumed one of four preloads 30 minutes prior to an ad. libitum pasta meal. Three of the preloads were isocaloric (~278kcal) mixed composition dairy fruit drinks (300g) of low (12.5% energy PRO/87% energy CHO), medium (25% energy PRO/75% energy CHO) and high (50% energy PRO/50% energy CHO) protein content. The control drink was a low energy (78kcal) alternative (12.3% energy PRO/84.3% energy CHO). ANOVA linear contrasts indicated a dose response effect of preload protein level on intake (g) at the ad. libitum meal, ($F(1,24) = 16.15$, $p<0.001$). Subsequent intake in each condition was: control (637.5g±39.7), low (596.9g±40.5), medium (546.9g±34.7), and high protein (533.6g±42.3). Participants did not compensate fully at lunch for the additional energy in the test drinks, however in the high PRO condition alone, total energy intake was not significantly different from the control condition. There were no differences in ratings of hunger and fullness across conditions. Our findings support the view that increasing the protein composition of beverages could be of interest to prevent short-term positive energy balance.

Effects of Overfeeding in Obesity Prone and Obesity Resistant Rats and Humans. D. Bessesen, Department of Medicine, University of Colorado at Denver and Health Sciences Center, Denver, CO. To maintain energy balance, nutrient flux must be sensed and when imbalances occur, adaptive responses made. Weight maintenance is possible only if these processes of nutrient sensing and adaptive responding occur relatively quickly following the development of energy or nutrient imbalance. Following the introduction of a high fat diet, both obesity prone (OP) and obesity resistant (OR) rats overconsume the diet for 1 day. Over the next 4 days, OR rats exhibit comprehensive adaptive responses to the period of overfeeding that restore energy balance. As compared to OP rats, OR rats reduce food intake, have higher rates of dietary fat oxidation and increased energy expenditure. To see if similar responses occur in OP versus OR human subjects we examined the effects of 3 days of overfeeding a mixed diet (40% above basal energy) in human subjects who self report difficulty gaining weight, reduced obese individuals, and subjects who come from families where obesity is a problem. Outcome measures included hunger and satiety measured with visual analogue scores, dietary fat oxidation measured with tracers, insulin sensitivity measured with the hyperinsulinemic euglycemic clamp, energy expenditure measured with whole room indirect calorimetry and physical activity monitors, post-meal excursions of a number of hormones and metabolites known to be related to food intake and regional brain activity measured with fMRI. The results of these studies suggest that OR humans also respond to passive overfeeding with comprehensive adaptive responses that promote weight maintenance. The results support the notion that the trafficking of dietary fat towards oxidizing tissues such as skeletal muscle and liver allow for more accurate nutrient sensing.

The effect of mastication on appetite and lipid bioaccessibility. B.A. Cassady, J.H. Hollis, R.D. Mattes. Purdue University, West Lafayette, IN, 47907, US. Mastication contributes to satiety by multiple mechanisms, one of which may entail release of compounds like lipids that elicit release of gut peptides. In this study, almonds were chosen due to their satiating property and evidence that the bioaccessibility of their lipid is largely dependent on mechanical fracture of their cell walls. Healthy adults participated in this three-arm, cross-over design study. For each test day, they reported to the laboratory after an overnight fast, underwent a baseline blood draw, and completed an appetite questionnaire. In random order, they chewed 11, 5g portions of almonds either 10, 25 or 40 times before swallowing. Further blood draws were taken and appetite was reassessed at specified times for 4 hours. Over the following 4 days, participants consumed all meals in the laboratory. All feces were collected during this controlled feeding period and appetite sensations were recorded each waking hour. Glucose and insulin concentrations did not differ by chewing treatment. Analysis of fecal samples showed that with fewer chews, more energy was lost in the feces. This suggests decreased lipid bioavailability.

There were no significant differences in fullness or hunger ratings over the 4 hours post-almond consumption between chewing treatments. Future analysis will examine cholecystokinin (CCK), glucogon-like-peptide-1 (GLP-1), ghrelin, and leptin to explore the implications of differences in lipid bioavailability.

Common hepatic branch vagotomy reduces anorexia and pica produced by a cancer chemotherapy agent in the rat. B.C. DE JONGHE, C.C. HORN. Monell Chemical Senses Center, Philadelphia, PA 19104, USA. Anticancer chemotherapies, such as cisplatin, induce nausea, vomiting and anorexia. Cisplatin acts on vagal afferents of the gut to produce emesis but little is known about how this drug and other chemotherapies generate anorexia and nausea. Electrophysiological data from our laboratory indicate activation of vagal afferents of the common hepatic branch (CHB) by cisplatin. The goal of this study was to assess the effect of CHB lesion on anorexia and pica produced by cisplatin in the rat. We also examined the effects of the dopamine-D2 agonist apomorphine, which induces emesis via a central mechanism. Although rats lack a vomiting response, they ingest kaolin clay (a pica response) when made sick by toxins, and this behavior can be blocked by anti-emetic drugs. Our results show that cisplatin-induced pica was markedly reduced in CHB-vagotomized animals. Similarly, suppression of daily food intake and body weight following cisplatin treatment was also blunted by CHB ablation. CHB lesion had no effect on apomorphine-induced pica. The results indicate that the CHB, which innervates primarily the upper duodenum, plays an important role in cisplatin-induced malaise. These data also suggest that pica has sensory pathways similar to emetic systems in other species since a vagal lesion inhibited cisplatin-induced pica but had no effect on apomorphine-induced pica. In summary, this investigation is important because it helps to define neural systems involved in malaise, which can significantly impact feeding behavior in patients with chronic disease, such as cancer and AIDS, who receive potent drug treatments. Supported by NIH-DK065709 and NIH-DC000014.

Neuropeptide Y5 receptor antagonism in overweight and obese adults. N. ERONDU, Clinical Research, Metabolism, Merck & Co, Inc. Neuropeptide Y (NPY) is an orexigenic peptide, and antagonism of NPY Y1 and NPY Y5 receptors (NPYxR) is considered a potentially important anti-obesity drug target. We tested the hypothesis that blockade of the NPY5R will lead to weight loss in humans using MK-0557, a potent, highly selective, orally active NPY5R antagonist. A multiple-dose positron-emission tomography study and a 12-week proof-of concept/dose ranging study, suggested an optimal MK-0557 dose (1 mg/day). The hypothesis was next evaluated in a 52-week randomized, double-blind, placebo-controlled trial involving 1661 overweight and obese patients. Although statistically significant at 52 weeks, the observed drug-related weight loss was not clinically meaningful. These observations provide important specific clinical insights into the human NPY-energy homeostatic pathway.

BLA but not CEA is involved in the control of meal interruption cues on feeding behavior. E.M. Galarce, M.A. Mcdannald, P.C. Holland. Johns Hopkins University, Baltimore, MD. Environmental cues paired with meal interruption in hungry rats stimulate feeding when they are sated. In the current experiment we explored the involvement of the central nucleus (CEA) and basolateral amygdala (BLA) in this form of learning. Rats first received bilateral neurotoxic or sham lesions of BLA or CEA. Behavioral training consisted of two parts: a) rats received 70 two-minute cued 8% sucrose trials; b) 83% of the next 168 trials were interrupted with an auditory stimulus at random times between 30 and 90 seconds after trial onset. In this way, when a trial was interrupted the presentation of the food cue as well as the delivery of food were suspended until the following trial. Training was followed by a week of ad-libitum chow availability. Testing consisted of three 10-min sessions with unlimited access to experimental food and presentation of cues paired with food (CS+), meal interruption signals (SC) or no cue. Sham and CEA animals showed cue induced potentiation of feeding during both CS+ and SC tests, while BLA lesioned rats failed to show this effect. These results are discussed in the context of learning, allostasis and possible brain pathways.

Distributed control of energy expenditure: Contributions from the isolated caudal brainstem. H. GRILL. University of Pennsylvania, Philadelphia, Pa; T. BARTNESS. Georgia State University, Atlanta, GA; R. HARRIS, University of Georgia, Athens, GA. Data are reviewed that support the hypothesis that the neural control of energy expenditure is distributed among several levels of the neuraxis. This view contrasts with that expressed most commonly in literature, that hypothalamic neurons, whether they are those of the preoptic anterior hypothalamus, dorsal medial hypothalamus or acuate nucleus, are the critical sites for processing relevant afferent input and issuing commands that control energy expenditure in response to energy deficit or surfeit, or exposure to cold environments. Caudal brainstem neurons are part of the effector pathways for energy expenditure and thermoregulation. Caudal brainstem neurons also receive afferent input – e.g., skin thermal afferents, leptin and ghrelin receptors. Two experiments are discussed that

assess whether afferent processing by caudal brainstem neurons that have been neurally isolated from the hypothalamus is sufficient to engage local effectors that produce responses that yield compensatory adjustments in energy expenditure. COLD EXPOSURE. Heart rate (HR), Tc, and activity responses of chronic decerebrate (CD) rats were compared with those of control rats exposed for 6 h to 4°, 8° or 12° C. Other CD and controls were exposed to 4° C and 23° for 4 h and tissues was processed for norepinephrine turnover (NETO), a proxy of sympathetic drive. FOOD DEPRIVATION. Oxygen consumption, respiratory quotient and core temperature responses of CD and comparably maintained control rats were examined under baseline gavage fed conditions and over the course of a 48 h period where rats received water but not food. Data from both studies demonstrate that when neurally isolated from the hypothalamus, afferent input drives caudal brainstem neurons that engage or inhibit sympathetic energetic responses that are comparable in most respects to energetic responses of comparably maintained intact rats. Differences between the responses of CD and control rats will also be described. Supported by NIH grant SCRO - DK-21397 (HG,TB,RH).

Learnt anticipatory eating in rats reinforced by a single length of fast. S. JARVANDI[1], D.A. BOOTH[2], L. THIBAULT[1]. [1]School of Dietetics and Human Nutrition, McGill University – Macdonald Campus, 21,111 Lakeshore Road, Ste-Anne-de-Bellevue, Quebec, Canada H9X 3V9. [2]School of Psychology, University of Birmingham, UK. Short-term intake is influenced by learning from subsequent physiological requirements, not only by current need. Previous work on anticipatory eating trained each rat on two durations of food deprivation. This experiment used only one length of fast. Eight male rats were presented odourized solutions of 33% maltodextrin for 1 h after 3 h without food in the dark phase. In each of ten 2-day training cycles, a rat had one odour followed by one length of fast, with maintenance diet on intervening days. Anticipatory eating was measured as the difference at each cycle between intake of CHO having the odour predictive of the longer fast (TL) and intake of CHO with an odour cuing the shorter fast (TS). Learnt anticipatory eating was observed as an increase in TL minus TS over cycles. The temporal pattern of differences between groups was very similar to that between successive training periods switching odour and fast length. Mean TL - TS rose from near zero to a peak of 7.42 at Cycle 6 in first training and 4.00 at Cycle 5 in the second period, declined to 1.82 and -0.05 and then increased again to 6.10 and 5.08 at Cycle 9 in first and second training periods, respectively. These findings replicated delay in avoidance learning by repletion-conditioned preference and self-extinction of avoidance by prevention of hunger reinforcement, and showed for the first time an expected re-learning of anticipatory eating. They also show that this learning does not depend on contrast between longer and shorter lengths of fast.

Obesity from high-fat diet weakened learning of anticipatory eating in rats. S. JARVANDI[1], L. THIBAULT[1], D.A. BOOTH[2]. [1]School of Dietetics and Human Nutrition, McGill University – Macdonald Campus, 21,111 Lakeshore Road, Ste-Anne-de-Bellevue, Quebec, Canada H9X 3V9. [2]School of Psychology, University of Birmingham, UK. Cognitive functioning may be impaired by a diet high in fat (Chambers, 2006). Rats' integration of controls of ingestion includes learning to increase amount eaten of a food that cues absence of food when next hungry (Thibault & Booth, 2006). Acquisition of this anticipatory eating was studied in eight female rats with obesity induced and maintained by high-fat diet. Odourised training/test food was presented for 1.5 h after withholding food for 3 h from the start of the dark phase. On 10 training days, postprandial fasts were imposed in pseudorandom sequence of 3h after one odour or 10h after another odour, with maintenance food on days between. In experiments on normal-weight rats, amount eaten of food with odour predicting the longer fast (TL) increased relative to amount of food cuing animals to the shorter fast (TS), in competition with a decrease attributed to repletion-conditioned preference. The obese rats showed this cubic trend in TL minus TS, with the most negative value at Cycle 3 (mean = -1.48 g) and a peak at Cycle 5 (TL - TS = 1.13 g, although only $P < 0.1$). In these obese rats, however, the greater length of fast did not produce a higher intake in the first hour of access to maintenance diet (2.53 g) than after the shorter fast (3.31 g). High-fat diet or obesity may weaken learning anticipatory eating to avoid hunger by reducing the flow of energy out of lean tissues (Booth, 1978) and its negative reinforcement signal.

The effects of pentoxifylline on human preadipocyte, adipocyte, keratinocyte and lung fibroblast. K.S. Kim, I.K. Lee. The Catholic University of Korea, Seoul, Korea. Subcutaneous injection technique, so called 'mesotherapy' using pentoxifylline has been being used for local fat tissue reduction in the clinical fields. But there are no scientific experimental studies on it`s safety and efficacy as of now. So we carried out this study to evaluate the effects of pentoxifylline on local fat tissue. We cultured preadipocyte using the fat tissue taken from liposuction-operated patient. Human fibroblast and keratinocyte were also cultured in conventional method. Each cell`s media are mixed with pentoxifylline by each concentration. For measuring the cell viability, we performed CCK-8 assay. For verifying the apoptosis, we performed DNA fragmentation, western blotting, Hoechst and Propidium Iodide(PI) staining and FACs

analysis, etc. 1. Pentoxifylline was well-dissolved on media, there were no pH changes of dissolved media according to various pentoxifylline concentration. 2. The cell viabilities of all of adipocytes, preadipocytes, keratinocytes and fibroblasts were inhibited significantly at 20 mM of pentoxifylline. 3. Apoptosis was not induced at less than 20 mM of pentoxifylline, but induced by p53 mechanism at more than 40 mM of pentoxifylline in all of adipocytes, preadipocytes, keratinocytes and fibroblasts. 4. In adipocytes, significant lipolytic effects were shown at 20 mM of pentoxifylline. In conclusion, further studies including in vivo study about the usefulness of pentoxifylline for reduction of local fat tissue are required.

Weight control functionality in commercial roods: From 'R' To 'D', E. M.R. KOVACS, Unilever North America, Englewood Cliffs, NJ, USA. Commercial food approaches to weight control range from more traditional reduced energy technologies to the use of "functional" ingredients with putative effects on appetite control or energy metabolism. The potential value of different approaches, the link between research and product claims, and efficacy testing of weight control foods and ingredients will be briefly considered, with some specific examples. In many cases, however, the application of technologies in foods is not limited by scientific support, but by a range of other product and commercial feasibility issues that must be satisfied to get from the laboratory to the marketplace. These aspects are often poorly recognized or underestimated by academic researchers. A better understanding of these issues, and awareness of commercial needs and opportunities for weight control through foods, can improve the ability of academics to influence and work effectively with industry to deliver proven benefits to consumers.

Genetic control of sucrose intake by mice. C. LIN, N. BOSAK, X. LI, M. L. THEODORIDES, D.R. REED, G. K. BEAUCHAMP, A. A. BACHMANOV. Monell Chemical Senses Center, Philadelphia, PA 19104, USA. Inbred mouse strains differ in voluntary consumption of sucrose solutions. In long-term two-bottle tests, mice from the C57BL/6ByJ (B6) strain drink more sucrose than mice from the 129P3/J (129) strain. To conduct genetic analyses of sucrose intake, we produced hybrids of the second filial generation (F2) between the B6 and 129 strains, measured sucrose intake of F2 mice, and examined genetic markers on all chromosomes. A genome scan of the B6 x 129 F2 identified several quantitative trait loci (QTL) for sucrose intake. One of them, on chromosome 4, corresponds to the Tas1r3 gene (formerly Sac, saccharin preference locus) encoding a sweet taste receptor, T1R3. The Tas1r3 locus had stronger effect on intake of 120 mM sucrose than on intake of 300 mM sucrose. Relative contributions of other QTLs also changed with sucrose concentration. Sucrose intake is influenced by both sensory and postingestive mechanisms. Our data suggest that that genes involved in taste play more important role in intake of weaker sucrose solutions (e.g., 120 mM), and genes involved in postingestive mechanisms may be more important for intake of stronger sucrose solutions (e.g., 300 mM).

Integrated neurohormonal therapy for obesity: A novel approach aimed at harnessing the interaction between adipocyte-, islet-, and gut-derived signals in energy homeostasis, C. MACK, Amylin Pharmaceuticals Inc, San Diego, CA, USA. The development of anorexigenic anti-obesity agents has historically focused on small molecular agents to target individual CNS signaling pathways, an approach that has yielded modest efficacy, and has been repeatedly hampered by safety concerns. Adipocyte-, islet- and gut-derived neurohormones are known to play an important role in the complex physiological regulation of food intake and body weight and may offer a promising alternative approach to obesity pharmacotherapy. Studies in diet-induced obese (DIO) rodents have shown marked additive or synergistic interactions between amylin, PYY_{3-36}, and leptin. Co-administration of these three hormones at low doses led to a ~50% reduction in food intake and to complete reversal of obesity in DIO rats. Clinical research studies testing various combinations of amylin-, leptin-, and PYY_{3-36} -agonists are underway to investigate to what extent these findings will translate to human obesity. This innovative, integrated neurohormonal approach to obesity harnesses naturally-occurring synergies between adipocyte-, islet-, and gut-derived signals.

Short-term dietary compensation in free-living adults. F. MCKIERNAN, J.H. HOLLIS, R.D. MATTES. Purdue University, W Lafayette, IN, 47907, US. Evidence from children indicates that intra-individual variation in 24-hour energy intake is smaller than variation at individual eating occasions. This indicates children self-regulate energy intake on a short-term basis. A within-subject design study was used to explore this behavior in 50, weight-stable, obese and non-obese adults (11m, 39 f; age 30 Â±11 yrs; BMI 26.3Â±5.9). 24-hour dietary recalls were kept for 7 consecutive days. Each 24-hour period was divided into 7 eating occasions. The coefficient of variation (standard deviation divided by the mean) for energy intake was calculated for each adult for each eating occasion and over each 24-hour period. Sub-group variability was assessed by gender, age, BMI, level of dietary restraint and frequency of consumption of

calorically-sweetened beverages. The mean coefficient of variation for energy intake across the 7 eating occasions was 110.5%, compared to 28.9% for the day as a whole. A similar pattern of greater variation in energy intake at individual eating occasions than over the 24-hour period was noted for all sub-groups. Significantly greater variation in energy intake was noted for snacks compared to meals ($P < 0.0001$). These data suggest that adults regulate energy intake over a 24-hour period more closely than they do at individual eating occasions, similar to the pattern previously observed in children. Longitudinal research is warranted to determine whether this pattern and magnitude of variability changes over longer time intervals and how it relates to energy balance and body weight.

Introgression of Leptin receptor (lepr) mutation from Koletsky rat renders obesity and diabetes in the Bio-Breeding (BB) rat. D.H. MORALEJO, J.M. FULLER, P.M. TREUTING, T.D. TUPPLING, C.T. HANSEN and Å. LERNMARK. Department of Comparative Medicine and R.H. Williams. Laboratory Medicine, University of Washington, Seattle, WA. USA. Type 1 and 2 diabetes (T1DM and T2DM) and obesity are complex interacting polygenic diseases. The (BB.GTP/ase immune-associated protein 5 (Gimap5-/-)) lymphopenic rat is a model of T1DM, while (BB.+/+) is non-lymphopenic and diabetes resistant. The aim of the study was to further investigate the Lepr and Gimap5 mutated genes in the 1) delays of diabetes and/or 2) susceptibility to diabetes in the lymphopenic and non-lymphopenic rats. We secured introgression of the Lepr gene onto the BB.(+/+) rat through a series of N15 marker assisted backcrosses. The double congenic line BB.(lepr-/-, gimap5-/-) was generated by intercrosses. The following parameters were measured until 150 days of age: body weight and blood glucose daily, and at the endpoint of the study peripheral blood, serum (cytokines analyses) and tissues (histology of pancreas) were collected. At 150 days of age the body weight and blood glucose level of the BB.(lepr-/-) was (37.6 % increased and 5.6 fold increased) compared with the BB.(+/+) male rats, respectively. The diabetes onset of the BB.(lepr-/-, gimap5-/-) was at (140 ± 10 days of age) compared with the BB.(gimap5-/-) (58 ± 4 days of age) female rats. This data demonstrates that introgression of Lepr gene: 1) Onto BB.+/+ non-lymphopenic rat produces an extremely obese rat with susceptibility conferred only to male for the T2DM development. 2) Onto BB.(gimap5-/-) lymphopenic rat delays the onset of spontaneous diabetes in the female rats. Our data suggest a sex different (hormonal) effect in the diabetes development.

Emotional Appetite Questionnaire: Construct Validity and Relationship with BMI. L.J. Nolan, L.B. Halperin, A. Geliebter. Department of Psychology, Wagner College, 1 Campus Rd., Staten Island, NY 10301 and New York Obesity Research Center, St. Luke's-Roosevelt Hospital Center and College of Physicians and Surgeons, Columbia University, New York, NY 10025 USA. The Emotional Appetite Questionnaire (EMAQ) is based on ratings of tendency to eat in response to both positive and negative emotions and situations. To provide construct convergent validity, the responses of 98 male and female college students to the EMAQ subscales were correlated with scores on the emotional subscale of the Dutch Eating Behavior Questionnaire (DEBQ). Secondarily, EMAQ subscale scores were correlated with the restrained and external eating subscales of the DEBQ. We also correlated EMAQ scores with body mass index (BMI). Several significant relationships were found between EMAQ and DEBQ scores. Convergent validity was demonstrated by a significant positive relationship ($r=.539$, $p<.001$) between the combined negative emotions and situations scores of the EMAQ and the emotional eating DEBQ score that is largely based on negative emotions. Discriminant validity was demonstrated especially in the women by a negative relationship of EMAQ combined positive emotion (EMAQ-P) and situation scores ($r= -.247$, $p=.038$) with the DEBQ emotional eating score in contrast to the positive correlation with combined negative scores (EMAQ-N) ($r=629$, $p<.001$). Indeed, for the whole sample there was a significant negative correlation between EMAQ-P and EMAQ-N ($r=-.227$, $p=.024$). The negative EMAQ score was also significantly positively correlated with BMI ($r=.211$, $p=.039$) replicating the finding by Geliebter and Aversa (2003) while the emotional eating score of the DEBQ was not ($r=.051$, $p=.624$). Thus, the EMAQ was shown to have construct validity – both convergent and discriminant, and the combined negative emotions and situations score EMAQ-N was significantly correlated with BMI.

Asymmetric lesions of the parabrachial nuclei and lateral hypothalamus block sodium appetite in rats. R. NORGREN, S. PECKINS, and S. DAYAWANSA. Department of Neural and Behavioral Science, College of Medicine, The Pennsylvania State University, Hershey, PA 17033 <p>Bilateral lesions of the parabrachial nuclei (PBN) disrupt the expression of Na appetite in rats. Bilateral damage to the lateral hypothalamus (LH) has a similar effect. Because the PBN projection to the ventral forebrain is primarily ipsilateral, we tested the functional connection between these areas by making PBN lesions on one side and LH lesions on the other (Ibotenic acid, 20μg/ml, 0.2μl PBN, 0.5μl LH, n=13). Six rats had identical but ipsilateral PBN and LH lesions. Four rats served as surgical controls. After acclimating to water and 0.5M NaCl, trials consisted of removing the NaCl, injecting furosemide (100 mg/kg, sc), and providing low sodium chow overnight. Water was available continuously. The following morning, the 0.5 M NaCl was

replaced and intake measured at 15, 30, 60, 120 min, and 24 h. This regimen was repeated 5 times at weekly intervals. On trial 4, all the rats received an injection of saline instead of furosemide. The controls and the rats with ipsilateral PBN and LH damage displayed a robust Na-appetite, 4.3±0.76 ml and 4.5±0.62 ml of 0.5M NaCl in the first 15 min, respectively. At 24 h, the comparable figures were 16.3±1.80 ml and 16.4±2.04 ml. The rats with asymmetric PBN and LH damage ingested 0.7±0.23 ml of 0.5M NaCl at 15 min; 3.05±0.56 ml at 24 h. At both times, the differences between the asymmetric group and both sets of controls were significant [15 m - $F(2,20)=27.8$, $p<0.0001$; 24 h – $F(2,20)=19.4$, $p<0.0001$]. Supported by DC005435 and DC008937.

Paracrine-like cytoarchitecture and proximity to nerve fibers in a subpopulation of GLP-1 and CCK immunoreactive enteric mucosal cells. C. PHILES and R. C. RITTER. Programs in Neuroscience and Dept. of VCAPP, Washington State University, Pullman, WA 99164-6520. Cholecystokinin (CCK) and glucagon like peptide-1 (GLP-1) are secreted by enteroendocrine cells of the intestinal mucosa. Both peptides appear to participate in control of food intake by ingested nutrients. Furthermore, vagal afferents mediate reductions of food intake by CCK and have been implicated in reduction of food intake by native GLP-1. Nevertheless, the manners in which these peptides access nerve afferent receptors are unclear. Convergent lines of evidence suggest that reduction of food intake by some intestinal nutrients may be mediated by paracrine release of CCK. However, immunoreactive CCK has not been observed in mucosal cells exhibiting a paracrine-like phenotype, and proximity of nerve fibers or terminals to CCK-secreting I-cells does not suggest a uniquely paracrine relationship. Therefore, we have reexamined the rat small intestinal mucosa for the presence of peptide immunoreactive cells with paracrine-like cytoarchitecture. Here we report that the distal half of the small intestinal mucosa contains a substantial subpopulation of cells exhibiting paracrine-like cytoarchitecture. Specifically, we find that both CCK and GLP-1 immunoreactivities are present not only in typical A-bottle- or A-spindle- shaped enteroendocrine I cells and L cells, but also are expressed in cells that exhibit an elongated process that extends beneath the bases of the enterocytes in a paracrine fashion. Some of these cells are immunopositive for CCK or GLP-1, with a subpopulation that is immunopositive for both peptides. Finally, in preparations stained for the neuronal marker, PGP 9.5, nerve fibers appear to run parallel and in close proximity to CCK- and GLP-1-immunoreactive paracrine-like processes.

Time course of acquisition of flavor-nutrient learning with fat vs. carbohydrate calories. C.H. REVELLE, Z.S. WARWICK. Department of Psychology, University of Maryland, Baltimore County, Baltimore, Maryland. Flavor-nutrient (F-N) learning occurs when the post-ingestive consequences of a food are associated with its flavor. Learned flavor cues thereby provide a mechanism for animals to anticipate calories based on a food's flavor and can contribute to regulation of intake during a meal prior to experiencing post-ingestive signals. F-N associations based on fat kcals require higher nutrient concentrations and experimental conditions that are not needed to train carbohydrate (cho)-based associations. This study compared the time course of acquisition of cho- and fat-based associations. Rats were trained to associate distinctive flavors with high-density (3.2kcal/ml) and low-density (0.2kcal/ml) solutions consumed orally. Half were trained with fat (corn oil emulsion), half who cho (sucrose). For each nutrient, both within- and between-groups designs were used to assess (via two-bottle test) whether F-N learning occurred after 2, 4, or 6 trial pairs. Preliminary results indicate that rats trained with cho demonstrated F-N learning after only 2 training trial pairs; rats trained with fat showed no learning after 2 trial pairs but did demonstrate learning after 4 trial pairs. These findings suggest that the longer latency to acquire F-N associations may be another mechanism by which high-fat foods promote overeating. Supported by NIDDK 55367

Serotonergic Mechanisms in the Night Eating Syndrome. A.J. Stunkard, K.C. Allison, J.D. Lundgren, J.P. O'Reardon. University of Pennsylvania. This review describes new findings regarding the Night Eating Syndrome (NES), a distinct disorder that is precipitated by stress most persons who suffer from it. It is manifested by a delayed circadian rhythm of food intake (evening hyperphagia, nighttime awakenings with ingestions and morning anorexia) with an intact circadian sleep rhythm. It is readily diagnosed by calibrated diagnostic instruments and has been widely reported in the medical literature. NES has genetic determinants and is more prevalent than the traditional eating disorders. New findings suggest that it represents a dysregulation of serotonin function. Thus, SPECT (single positron emission computerized tomography) has shown greatly elevated serotonin transporter binding in the midbrain of night eaters. This appears to impair postsynaptic serotonin transmission in areas mediating circadian rhythms and food intake. Restoration of serotonergic function with SSRIs (selective serotonin reuptake inhibitors) in four clinical trials has resulted in a great decrease in night eating behaviors and significant weight loss among overweight and obese night eaters.

Selective CB1 receptor antagonism for the management of cardiometabolic risk factors. P. A. TATARANNI, Sanofi-Aventis, Bridgewater, NJ, USA. The endocannabinoid (EC) system is involved in regulating energy homeostasis and glucose and lipid metabolism through the action of endogenous ligands on CB1, a transmembrane G-protein-coupled receptor expressed in the brain, adipose tissue, skeletal muscle, and liver. Preclinical findings show that overactivation of the EC system leads to excessive food and fat intake, exacerbation of both glucose intolerance and the atherogenic lipid profile, and excessive accumulation of intra-abdominal fat. The RIO (Rimonabant In Overweight/Obesity) program with four double-blind, placebo-controlled, Phase III clinical trials evaluated the efficacy and safety of Rimonabant, the first in a new class of selective CB1 blockers, in 6,600 patients, of whom about 1000 were type 2 diabetics. Treatment with Rimonabant (20 mg) was well tolerated and markedly improved multiple cardiometabolic risk factors including abdominal obesity, lipid and glucose metabolism, and insulin resistance, and in type 2 diabetics, HbA1c as well. Selective CB1 blockade shows promise for treating cardiometabolic risk factors associated with abdominal obesity.

The effects of oral ingestion of soy protein on the somatotropic axis. J.A.H. VAN VUGHT[1,2], A.G. NIEUWENHUIZEN[1,2], R.J. BRUMMER[2], M.S. WESTERTERP-PLANTENGA[1,2]. [1]Dept. of Human Biology, P.O. Box 616, 6200 MD, Maastricht, The Netherlands, [2]Top Institute Food and Nutrition, P.O. Box 557, 6700 AN, Wageningen, The Netherlands. Growth hormone (GH) is an important regulator of growth and body composition. Low levels of GH are likely to be involved in the pathogenesis of obesity, metabolic syndrome, sarcopenia and growth retardation. It has been shown that GH release can be promoted by administration of various amino acids, especially arginine and lysine, that are ample present in soy protein. The aim of the study was to compare the effects of hydrolysed soy protein and complete soy protein on GH-secretion. In this study, eight healthy women (BMI=19-26 kg/m^2; 19-36 years) received a drink solution containing either placebo, hydrolysed soy protein or complete soy protein (0.6g protein per kg bodyweight), in a randomized, single blind crossover design. Plasma GH, insulin and somatostatin were determined every 20 minutes for 5 hours. GH-responses, as determined by area under the curve (AUC), were significantly higher after hydrolysed and complete soy protein compared with placebo ($p<0.05$). Peak values of GH were significantly higher after complete soy protein compared with hydrolysed soy protein ($p<0.05$). Insulin responses (AUC) were significantly higher after complete and hydrolysed soy protein, compared with placebo ($p<0.05$). No significant difference was seen in plasma somatostatin concentrations. In conclusion, soy hydrolysate and soy protein stimulate GH release. Dietary protein therefore may affect physiological regulation of GH secretion.

Effects of amylin and salmon calcitonin on energy expenditure and body temperature. P.Y. WIELINGA, B. ALDER, T.A. LUTZ. Institute of Veterinary Physiology and Zurich Center for Integrative Human Physiology, Vetsuisse Faculty University of Zürich, Winterthurerstrasse 260, 8057 Zürich Switzerland. The pancreatic B-cell hormone amylin is known to be involved in the regulation of meal ending satiation and it also shares typical features of adiposity signals. Chronic amylin administration has recently been shown to increase energy expenditure under certain conditions. Here we investigate the acute effect of peripheral administration of amylin or its agonist salmon calcitonin (sCT) on energy expenditure and RQ. First, rats were injected with amylin (5 μg/kg IP) or saline just before dark onset. Despite significantly decreased food intake in amylin-treated rats compared to controls until 2 h post-injection ($p<0.05$), amylin did not influence energy expenditure or RQ. Reduced food intake, which reduces energy expenditure, may have confounded a stimulatory effect of amylin on energy expenditure. Therefore, in the second experiment, amylin (1, 5 and 10 μg/kg IP) or saline was injected in the middle of the light phase (t=0h) without access to food during 3 h post-injection. Amylin had no significant effects on energy expenditure or RQ. In a similar paradigm, the effect of sCT (0.1, 1.0 and 5.0 μg/kg IP) was tested. During food restriction, 1.0 and 5.0 μg/kg sCT significantly stimulated energy expenditure compared to control ($p<0.05$). Subsequent to refeeding at t=3h, energy expenditure was decreased compared to control from t=6h until t=12h after 5.0 μg/kg sCT, probably due to sCT's strong anorectic action. Amylin may prevent the compensatory decrease in energy expenditure normally seen in animals that eat less. The longer acting sCT stimulated energy expenditure in animals without access to food. Ongoing studies focus on the effect of chronic infusion of amylin and sCT on energy expenditure.

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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 contacting the Central Office.

Central Office

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SUMMARY SCHEDULE

Tuesday, July 24, 2007

8:00-2:00	SSIB Board Meeting – Location: Twilight Room
3:00-5:30	Registration – Location: Foyer
6:00-7:30	Welcome Reception for All Participants – Location: Poolside Tent
7:30-9:00	Students Only--Get Acquainted Social Event – Location: Aspen Room

Wednesday, July 25, 2007

7:30-8:30	Continental Breakfast/Exhibitors' Display – Foyer
8:30-10:30	Symposium 1 - Location: Storm Peak - Latest Research Developments from the Drug and Food Industries in the Battle against Obesity
8:30-10:30	Oral Session 1 - Location: Mt. Werner - Ingestion - Developmental Factors
10:30-11:00	Break/Exhibitors' Display - Foyer
11:00-12:00	Masterfoods Lecture - Martin G. Myers, Jr. - Location: Storm Peak/Mt. Werner
4:00-6:00	New Investigator Symposium - Location: Storm Peak/Mt. Werner
6:00-8:00	Poster Session 1 – Location: Sunshine Peak/Twilight

Thursday, July 26, 2007

7:30-8:30	Continental Breakfast/Exhibitors' Display – Foyer
8:30-10:30	Symposium 2 - Location: Storm Peak - Energy Expenditure in Relation to Food intake and Body Weight Regulation
8:30-10:30	Oral Session 2 - Location: Mt. Werner - Eating Disorders
10:30-11:00	Break/Exhibitors' Display - Foyer
11:00-1200	Masterfoods Lecture - Michael G. Tordoff: Location: Storm Peak/Mt. Werner
1:30-3:30	Oral Session 3 - Location: Storm Peak - Taste/Water-NaCl Ingestion
1:30-3:30	Oral Session 4 - Location: Mt. Werner - GI Peptides and Satiety I
3:30-4:00	Break/Exhibitors' Display - Foyer
4:00-6:00	NIH Sponsored Symposium – Location: Storm Peak/Mt. Werner - Neurobiology of Anorexia Nervosa, Bulimia Nervosa and Related Behaviors
6:00-8:00	Poster Session 2 – Location: Sunshine Peak/Twilight

Friday, July 27, 2007

7:30-8:30	Continental Breakfast/Exhibitors' Display – Foyer
8:30-10:30	Symposium 3 - Location: Storm Peak - Early Life Events and Long Term Obesity Risk
8:30-10:30	Oral Session 5 - Location: Mt. Werner - Macronutrient Effects
10:30-11:00	Break/Exhibitors' Display - Foyer
11:00-1200	Masterfoods Lecture - Eric Ravussin - Location: Storm Peak/Mt. Werner
1:30-2:30	NIH Grant Workshop: Michael Selmanoff / Judith Podskalny - Location: Storm Peak/Mt. Werner
4:00-6:00	Presidential Symposium – Location: Storm Peak/Mt. Werner
6:00-8:00	Poster Session 3 – Location: Sunshine Peak/Twilight

Saturday, July 28, 2007

7:30-8:30	Continental Breakfast/Exhibitors' Display – Foyer
8:30-10:30	Symposium 4 - Location: Storm Peak - Human Thirst and Sodium Appetite
8:30-10:30	Oral Session 6 - Location: Mt. Werner - GI Peptides and Satiety II
10:30-11:00	Break/Exhibitors' Display - Foyer
11:00-1200	Masterfoods Lecture - Helen E. Raybould - Location: Storm Peak/Mt. Werner
1:30-3:30	Symposium 5 - Location: Storm Peak - The Social Psychology of Eating: Schacter's Legacy.
1:30-3:30	Oral Session 7 - Location: Mt. Werner - Ingestion - Central Mechanisms
3:30-4:00	Break
4:00	Award Announcements - Storm Peak/Mt. Werner Gerard P. Smith Award / TestDiet Jackson Lab Scholarship
4:05	Alan N. Epstein Research Award - Stephen C. Benoit
4:30	Distinguished Career Award - Jack D. Davis
7:00	Cash Bar - Foyer
7:30	Banquet, Entertainment and Dancing – Storm Peak/Mt. Werner/Sunshine Peak/Twilight

Next SSIB Meeting

July 15-20, 2008

Paris, France
Local Organizer: Daniel Tome