Is food addiction real?

Robert H. Lustig, M.D., M.S.L.

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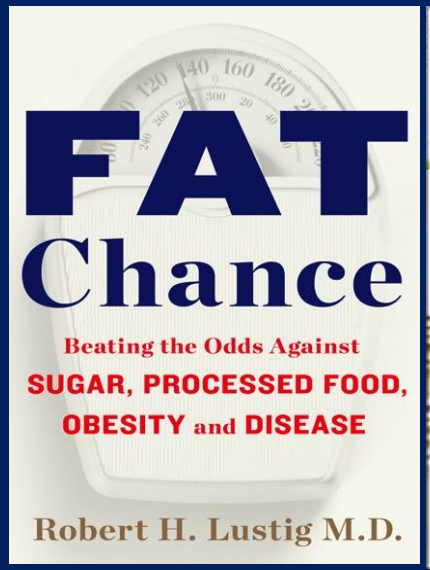
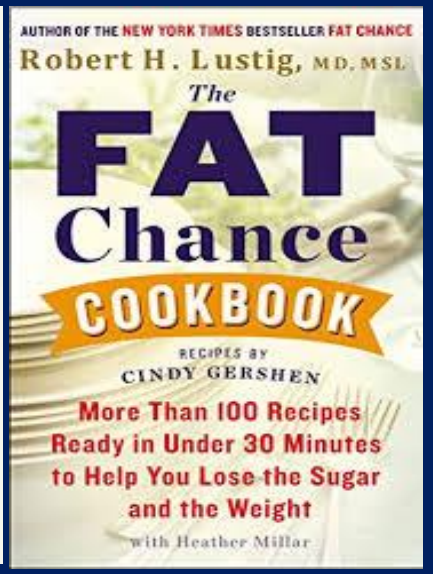
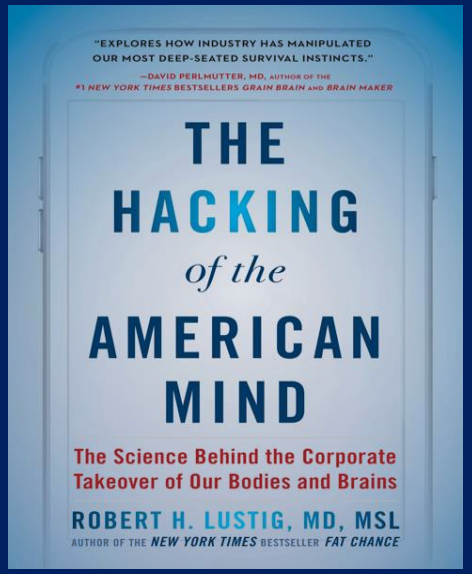
University of California, San Francisco

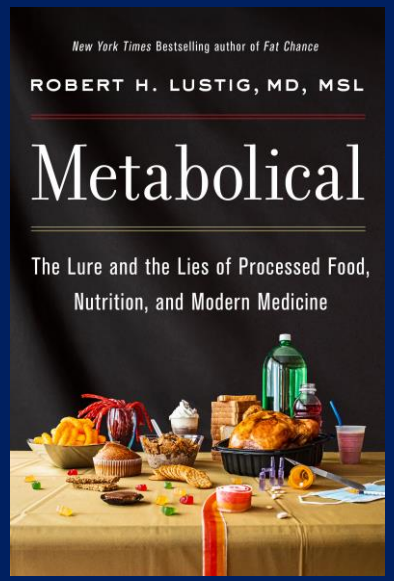
Nutrition Network, 2022

Disclosures









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Kalin Health
BioLumen
Foogal
Perfact

Paid Advisor:
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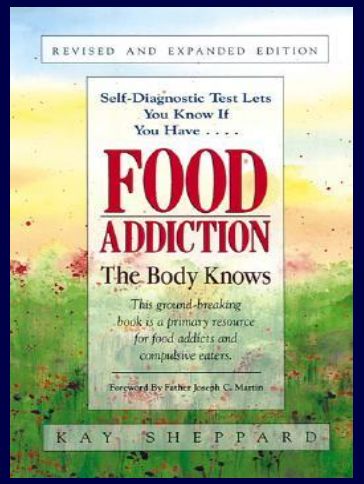
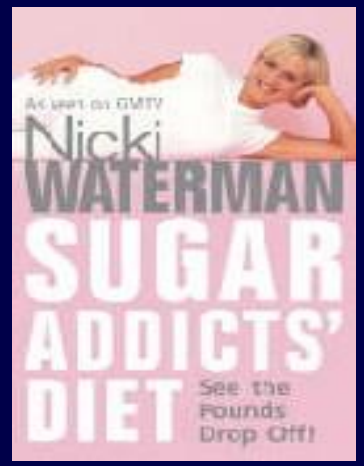
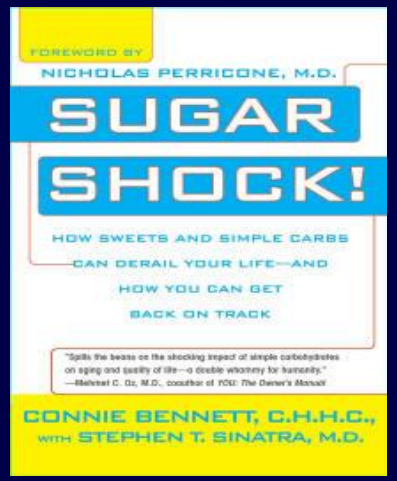
ReadOut Health
Simplex Health
Levels Health

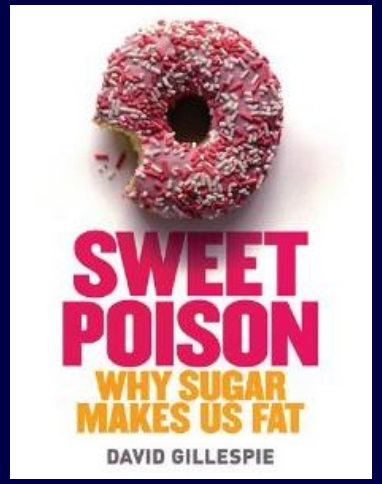
Unpaid Advisor:
Kuwaiti Danish Dairy

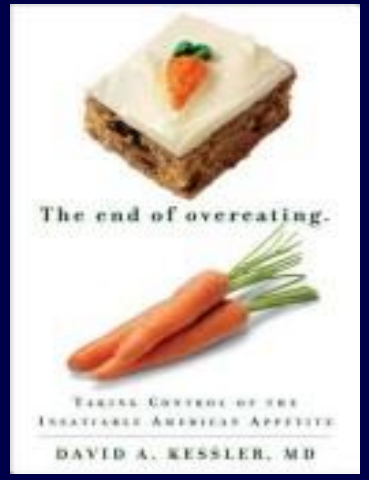
Is food addiction real?
The lay public seems to know….

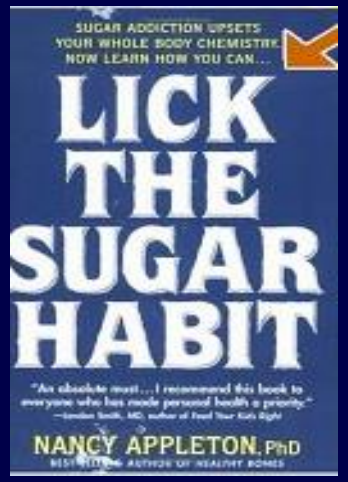








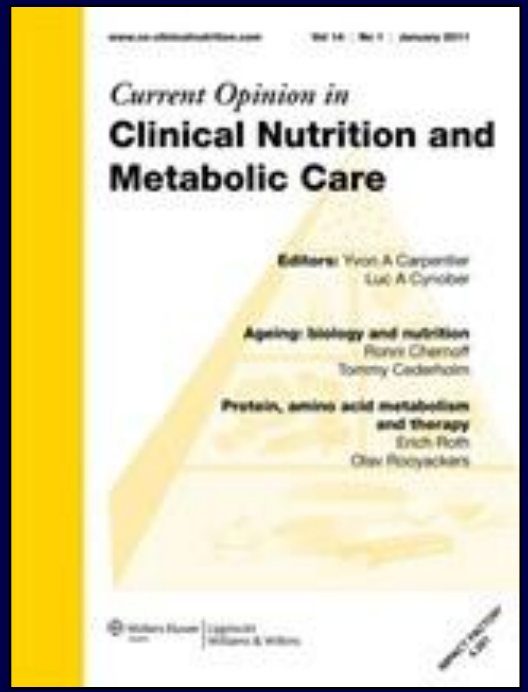




The evolution of “food addiction”

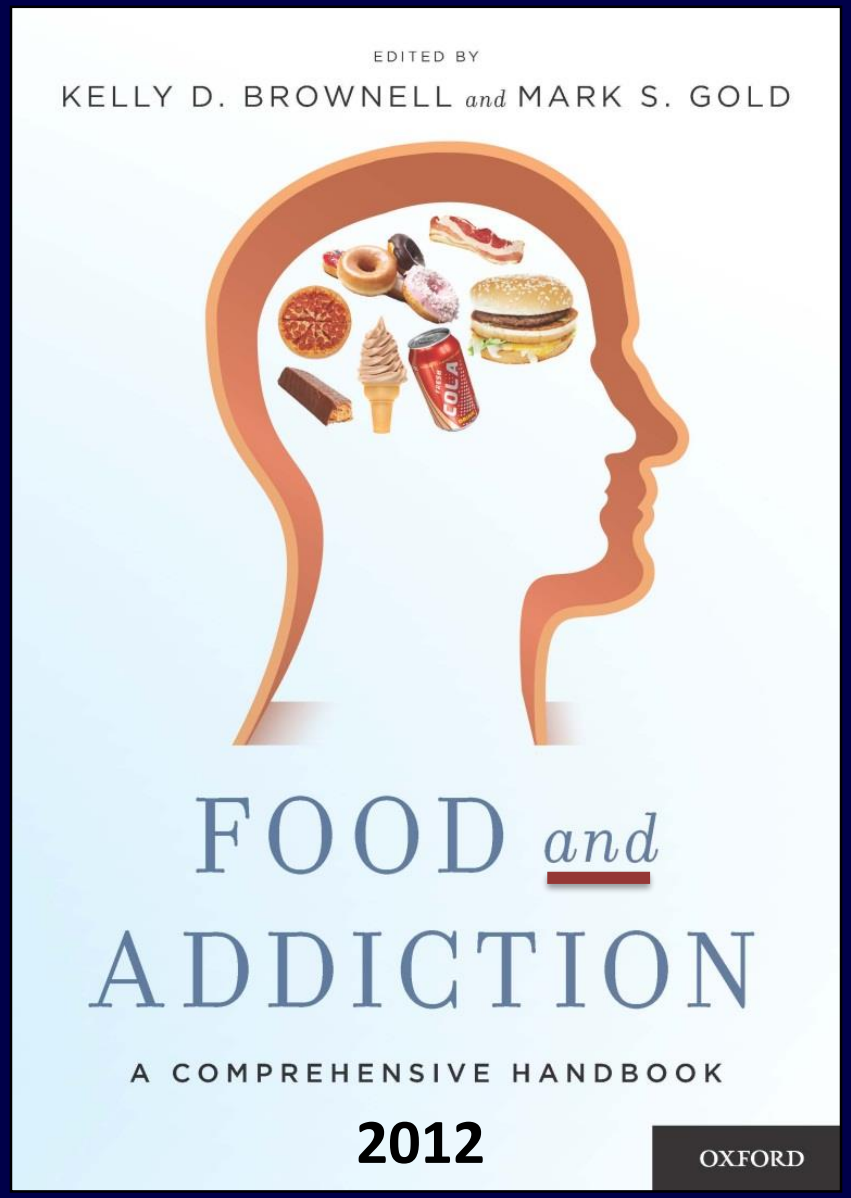
2009

EDITOR: Mark S. Gold
SPECIAL ISSUE: FOOD ADDICTION



2010

Daniel M. Blumenthal and Mark S. Gold
NEUROBIOLOGY OF FOOD ADDICTION


EDITED BY
KELLY D. BROWNELL and MARK S. GOLD
FOOD and ADDICTION
A COMPREHENSIVE HANDBOOK
2012
OXFORD

OPINION

Obesity and the brain: how convincing is the addiction model?

Hisham Ziauddeen, I. Sadaf Farooqi and Paul C. Fletcher

Abstract | An increasingly influential perspective conceptualizes both obesity and overeating as a food addiction accompanied by corresponding brain changes. Because there are far-reaching implications for clinical practice and social policy if it becomes widely accepted, a critical evaluation of this model is important. We examine the current evidence for the link between addiction and obesity, identifying several fundamental shortcomings in the model, as well as weaknesses and inconsistencies in the empirical support for it from human neuroscientific research.

Nature Rev Neurosci 13:279, 2012

CORRESPONDENCE

Tossing the baby out with the bathwater after a brief rinse? The potential downside of dismissing food addiction based on limited data

Nicole M. Avena, Ashley N. Gearhardt, Mark S. Gold, Gene-Jack Wang and Marc N. Potenza

Nature Rev Neurosci 13:514, 2012

CORRESPONDENCE

Food addiction: is there a baby in the bathwater?

Hisham Ziauddeen, I. Sadaf Farooqi and Paul C. Fletcher

Nature Rev Neurosci 13:514, 2012

Neuroscience and Biobehavioral Reviews 47 (2014) 295–306

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review

"Eating addiction", rather than "food addiction", better captures addictive-like eating behavior

Johannes Hebebrand[a], Özgür Albayrak[a], Roger Adan[b], Jochen Antel[a], Carlos Dieguez[c,d], Johannes de Jong[b], Gareth Leng[e], John Menzies[e,*], Julian G. Mercer[f], Michelle Murphy[f], Geoffrey van der Plasse[b], Suzanne L. Dickson[g]

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Puts the onus on the individual, not the food

NeuroFAST
www.neurofast.eu

CONSENSUS

NeuroFAST consensus opinion on food addiction

- **Current evidence does not allow us to conclude that a single food substance** via a single specific neurobiological mechanism (e.g. specific brain receptors or specific neuronal pathways) can account for the fact that people overeat and develop **obesity**.
- **In humans, there is no evidence that a specific food, food ingredient or food additive causes a substance-based type of addiction** (the only currently known exception is caffeine which via specific mechanisms can potentially be addictive).

NeuroFAST
www.neurofast.eu

CONSENSUS

NeuroFAST consensus opinion on food addiction

- Within this context **we specifically point out that we do not consider alcoholic beverages as food**, despite the fact that one gram of ethanol has an energy density of 7 kcal.

- **Addictive (over)eating is clearly distinct from substance use disorders** that cause addiction via specific mechanisms (e.g. nicotine, cocaine, cannabinoids, opioids, etc).

So, NeuroFAST exempts both alcohol and caffeine, even though both are in food

Great Debates in Nutrition

David S. Ludwig, Section Editor

The concept of "food addiction" helps inform the understanding of overeating and obesity: YES

Ashley N Gearhardt[1] *and Johannes Hebebrand*[2]

[1]Department of Psychology, University of Michigan, Ann Arbor, MI, USA; and [2]Department of Child and Adolescent Psychiatry, Psychosomatics, and Psychotherapy, University Hospital Essen, University of Duisburg-Essen, Duisburg, Germany

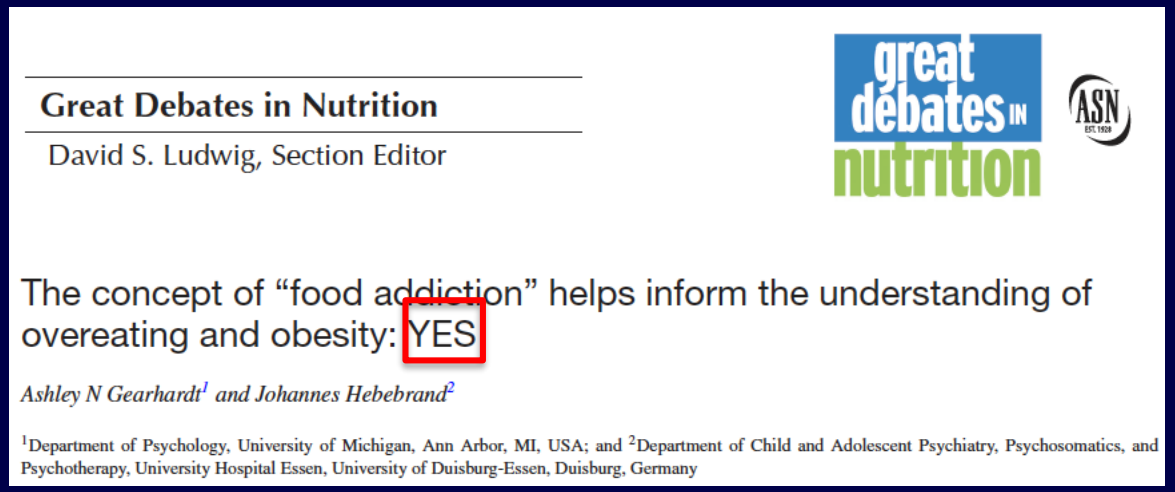
Great Debates in Nutrition

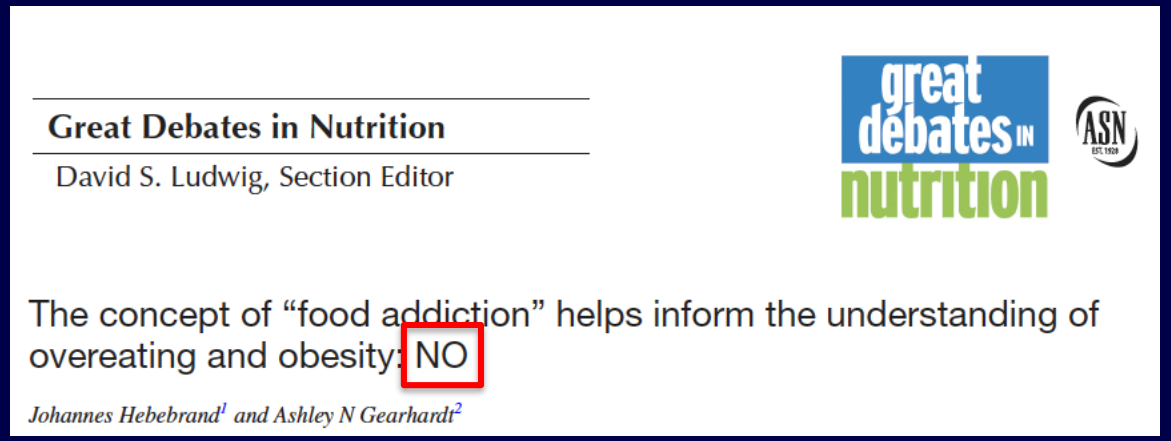
David S. Ludwig, Section Editor

The concept of "food addiction" helps inform the understanding of overeating and obesity: NO

Johannes Hebebrand[1] *and Ashley N Gearhardt*[2]

Am J Clin Nutr Feb 2021





Hebebrand: "Evidence that specific food ingredients are key determinants of addictive-like eating behavior is lacking."

Gearhardt: "Highly processed foods are complex substances developed through engineering by combining reinforcing ingredients (i.e., refined carbohydrates, fat) and additives (e.g., salt) to deliver unnaturally heightened levels of reward."

Am J Clin Nutr Feb 2021

If:

— it’s about obesity; or

— it’s about eating addiction; or

— no specific foodstuff is addictive,

then the food industry has “carte blanche”;
and there is no option for societal intervention

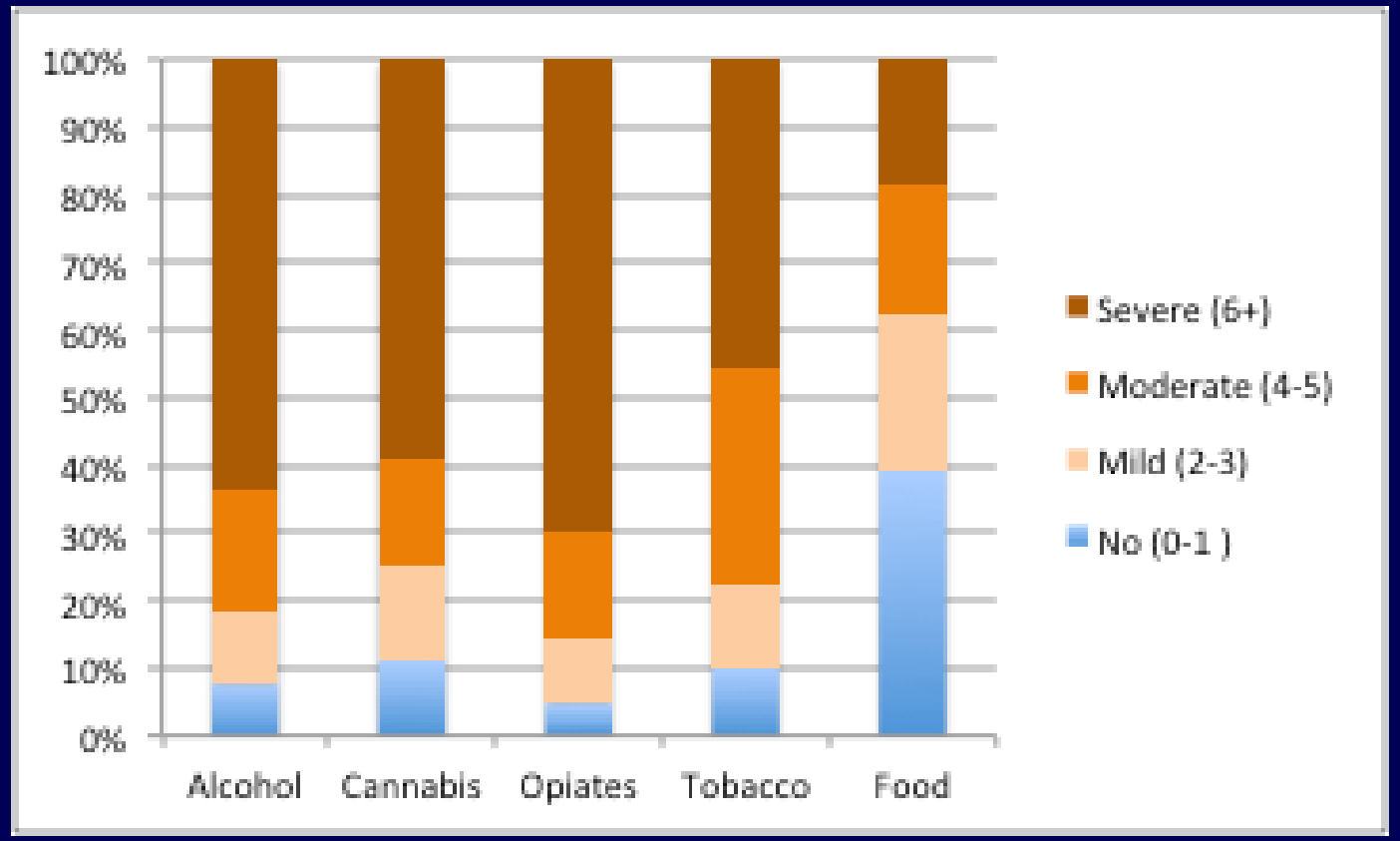
Aquitaine Addiction Cohort Study

Objective

- To examine severity and discrimination of DSM-5 criteria for Food Addiction adapted from SUD criteria
- To compare with SUD criteria (n = 875)
  - Alcohol, tobacco, cannabis, opiates

Denis et al. CPDD 2016

Results – DSM-5 Diagnosis



- Mostly severe use disorder
  - >90% of the sample for SUD
- More variability for food addiction

Denis et al. CPDD 2016

Criteria endorsement

	Alcohol	Food	Opiates	Cannabis	Tobacco
Tolerance	61.7	28.8	80.9	61.6	43.8
Withdrawal	44.8	33.8	86.6	55.4	70.3
Large amount	80.5	71.6	70.7	55.0	72.1
Unsuccessful cut down	60.5	49.6	73.2	48.0	68.7
Time spent	49.0	14.0	68.1	51.4	38.2
Given up activities	51.4	11.8	64.5	47.7	17.2
Psy or phy. pbl	54.6	48.2	60.5	50.3	60.4
Failure fulfill roles	50.7	17.4	33.3	38.7	9.3
Hazardous Use	72.5	16.1	58.7	67.5	21.6
Social pbl	59.2	15.7	51.4	51.0	29.1
Craving	65.2	69.8	79.5	68.0	78.8

- Similar pattern of criteria endorsement across substances and Food Addiction criteria

Denis et al. CPDD 2016

Item response theory (IRT) model

- Discrimination estimates across groups ranged from 0.88 to 5.12
- FA criteria exhibited the highest discrimination estimates

	Alcohol	Opiates	Cannabis	Tobacco	Food
Tolerance	1.52	1.70	1.94	1.31	3.09
Withdrawal	1.42	1.71	2.04	1.97	4.04
Large amount	1.48	1.71	1.75	1.59	3.99
Unsuccessful cut down	1.52	1.69	1.76	1.49	5.12
Time spent	1.22	1.72	1.79	1.17	3.84
Given up activities	2.00	1.74	1.92	1.17	3.15
Psychological or physical pbl	1.41	1.65	1.64	1.09	4.13
Failure fulfill roles	1.31	1.48	1.41	1.35	2.96
Hazardous Use	1.17	1.53	1.45	0.88	2.33
Social pbl	2.24	1.67	1.97	0.97	3.86
Craving	2.26	1.70	2.84	2.56	4.20

Denis et al. CPDD 2016

Yale Food Addiction Scale

- In 2009, the Yale Food Addiction Scale (YFAS) was created to study food addiction by applying the DSM-IV criteria for substance dependence to eating behaviors

Sample items:

- "I find myself continuing to consume certain foods even though I am no longer hungry"
- "I eat to the point where I feel physically ill"
- "I find that when I start eating certain foods, I end up eating much more than planned"

Gearhardt et al. Arch Psychiatr 2009

YFAS correlates with neuroimaging

- YFAS related to greater responsivity of reward regions (caudate, ACC, medial OFC, amygdala) and lower responsivity of an inhibitory control region (lateral OFC)

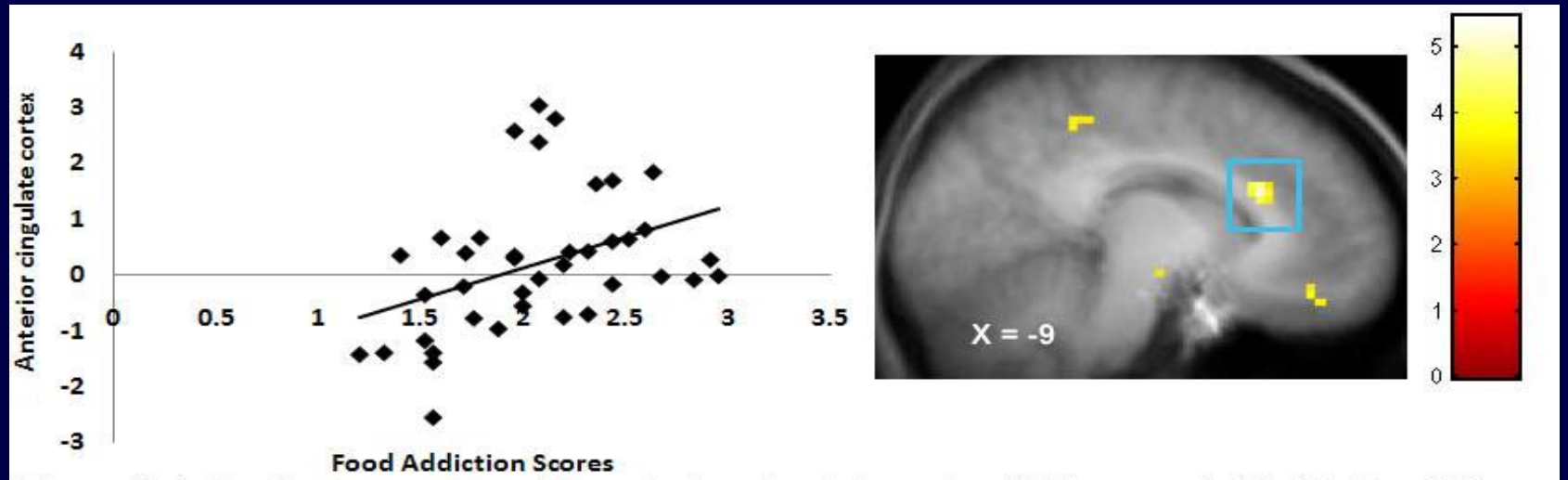


Figure 2. Activation in a region of the anterior cingulate cortex (MNI x,y,z: -9, 24, 27, Z = 4.64; pFDR <.001) during milkshake cues vs tasteless solution cues as a function of Yale Food Addiction Scale scores, with the graph of parameter estimates from that peak.

Palatable food cues trigger these areas as well

- Greater reward region response to palatable food cues predicts future weight gain
- Similar effects for substance use onset

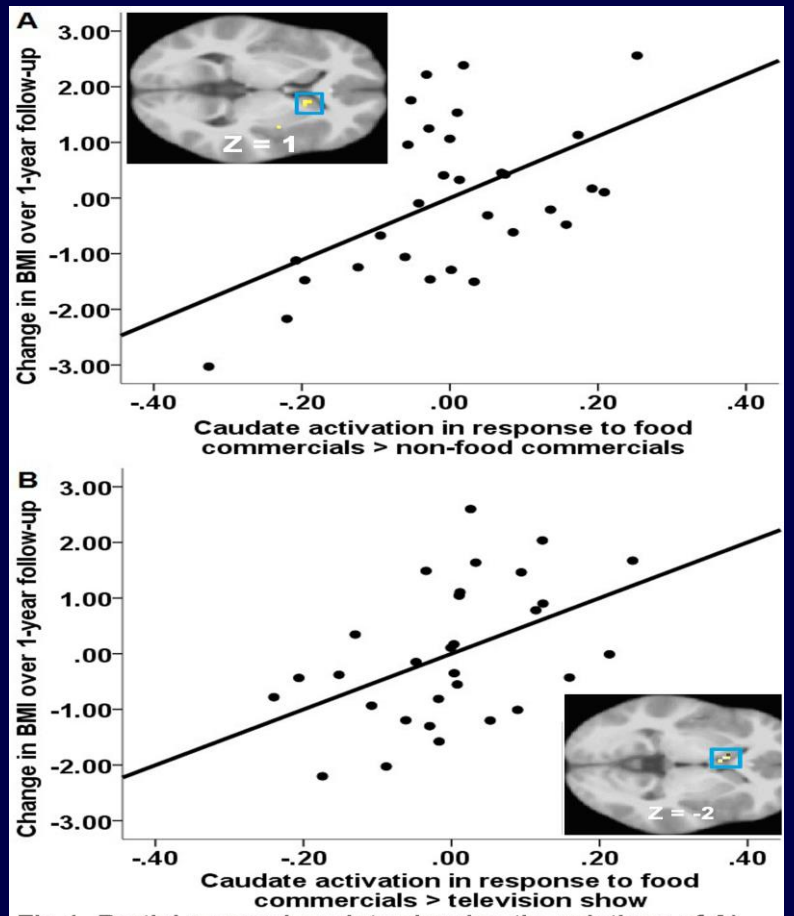
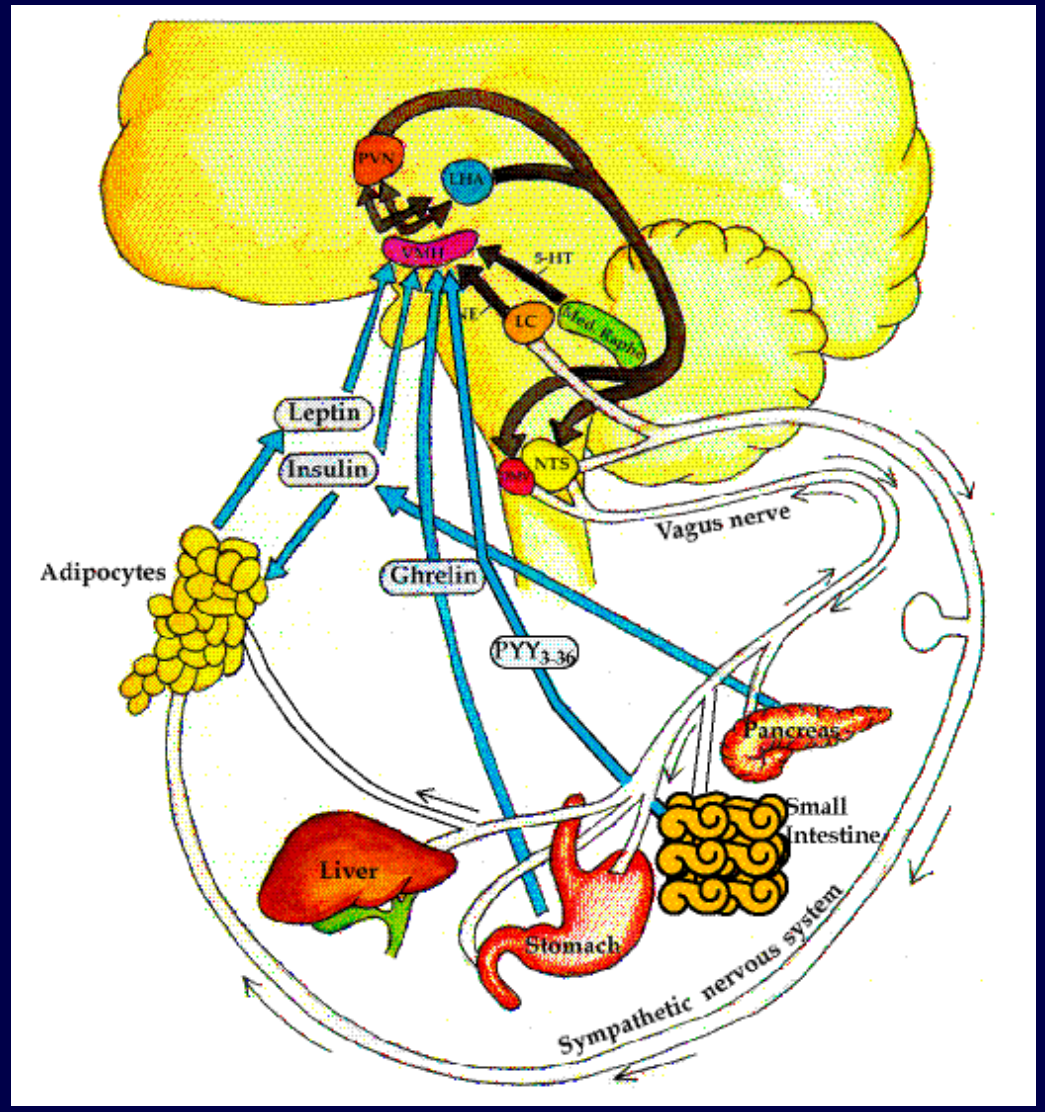


Fig 1. Partial regression plots showing the relations of **A)** activation in the caudate (MNI coordinates: 12, 14, 1) in response to food commercials > non-food commercials and **B)** activation in the caudate (MNI coordinates: -9, 14, -2) in response to food commercials > television show to change in BMI over 1-year follow-up

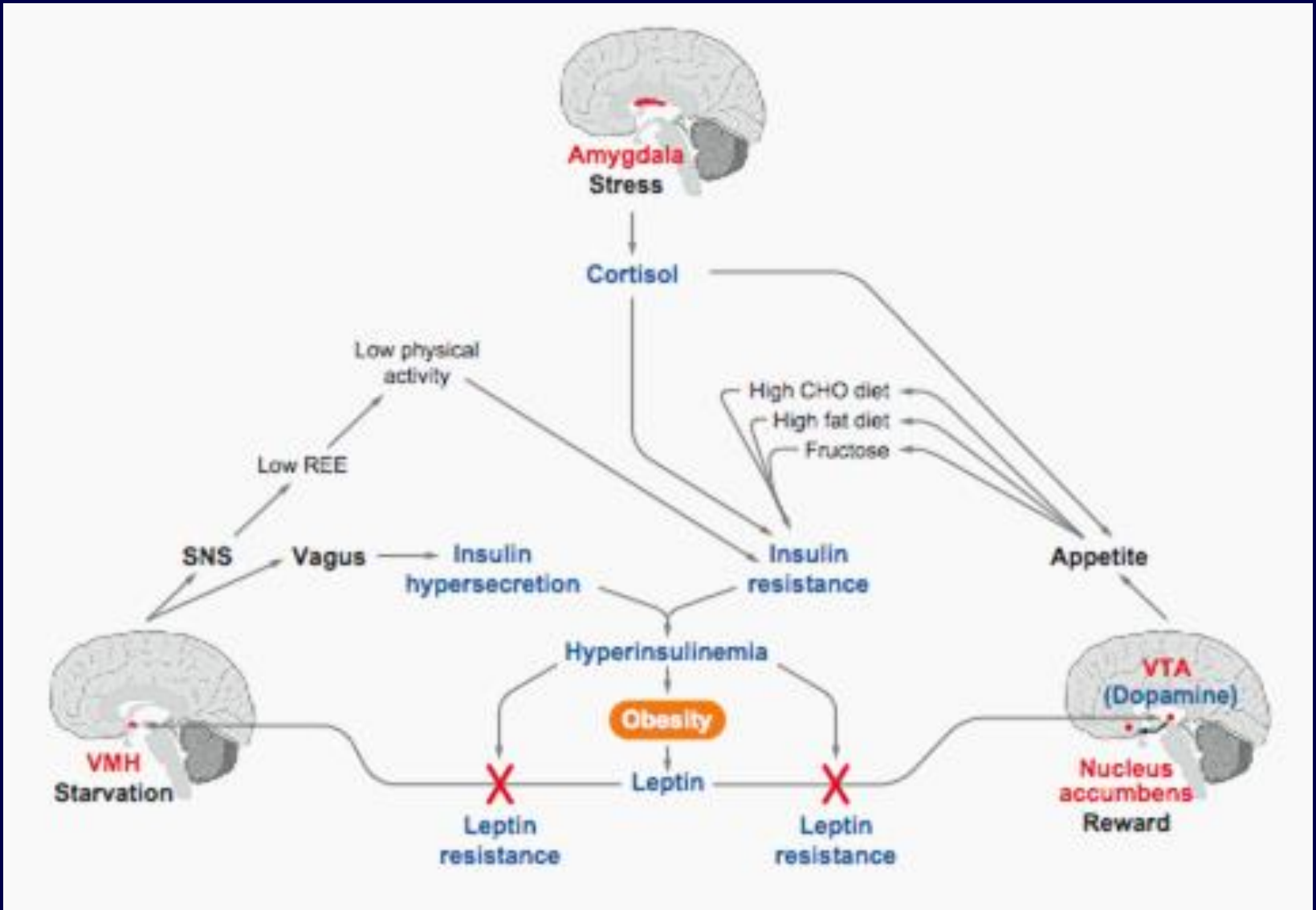
Chouinard et al. 2010; Demos et al. 2012; Stice et al. 2010; Yokum et al. 2011; Stice et al. 2013

The neuroendocrinology of energy balance



Lustig, Endo Clin NA, 2001

The “limbic triangle”

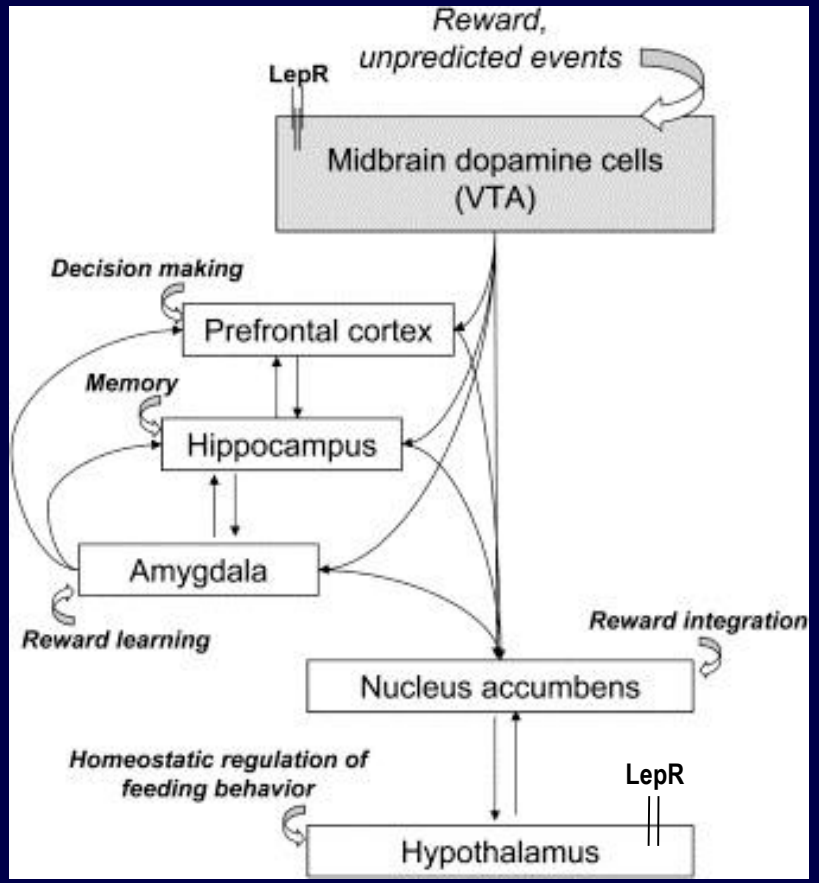


Mietus-Snyder and Lustig, Ann Rev Med 59:147, 2008

Indirect effects on the reward system:

Leptin and Insulin

The integration of the starvation and addiction pathways



Cota et al. Neuron 51:678, 2006

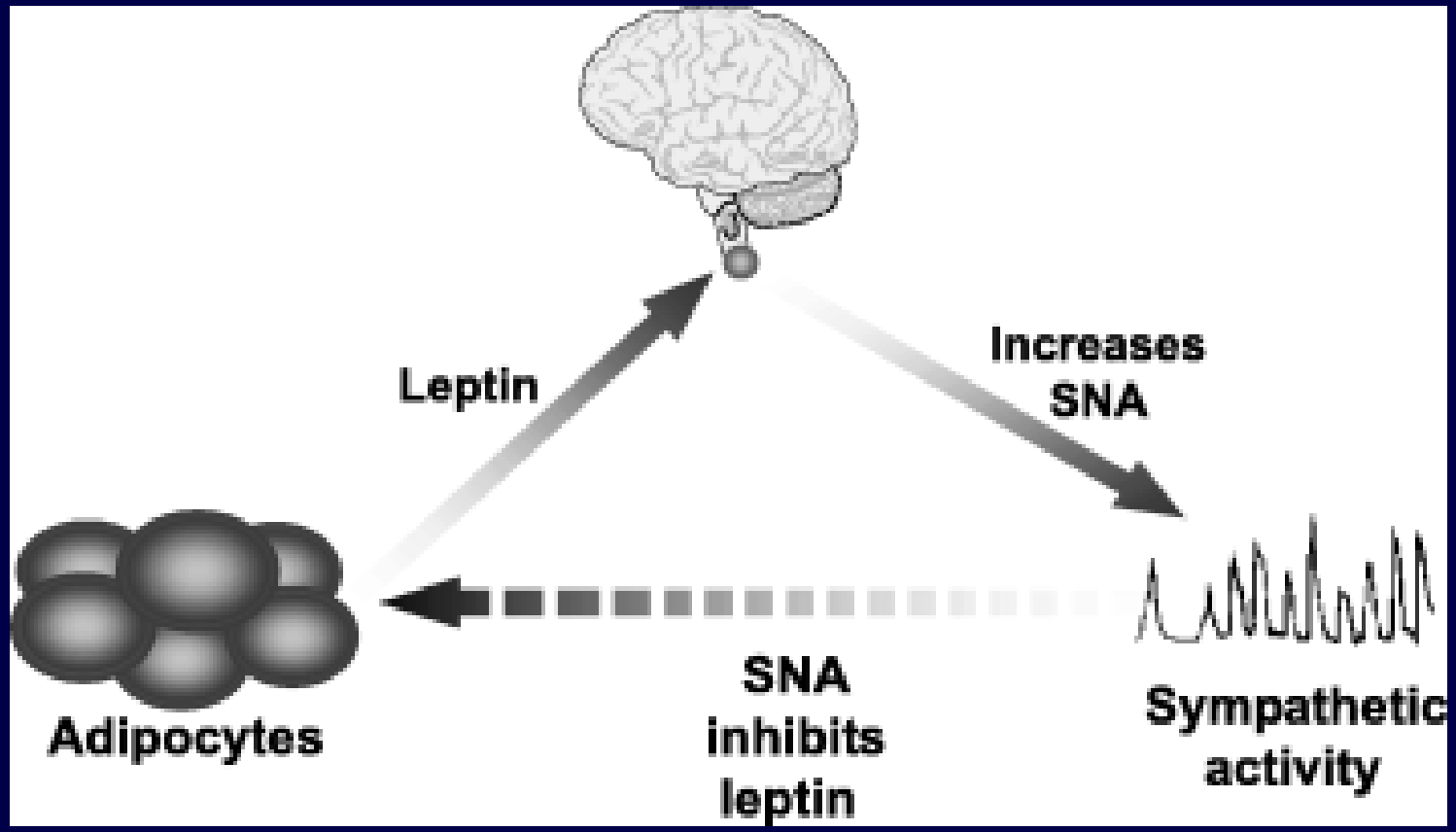
PARADOX:

If you give a 5 year old kid a cookie:

PARADOX:

If you give a 5 year old kid a cookie:

Leptin is supposed to keep us in energy balance



Mark et al. Acta Physiol Scand 177:345, 2003

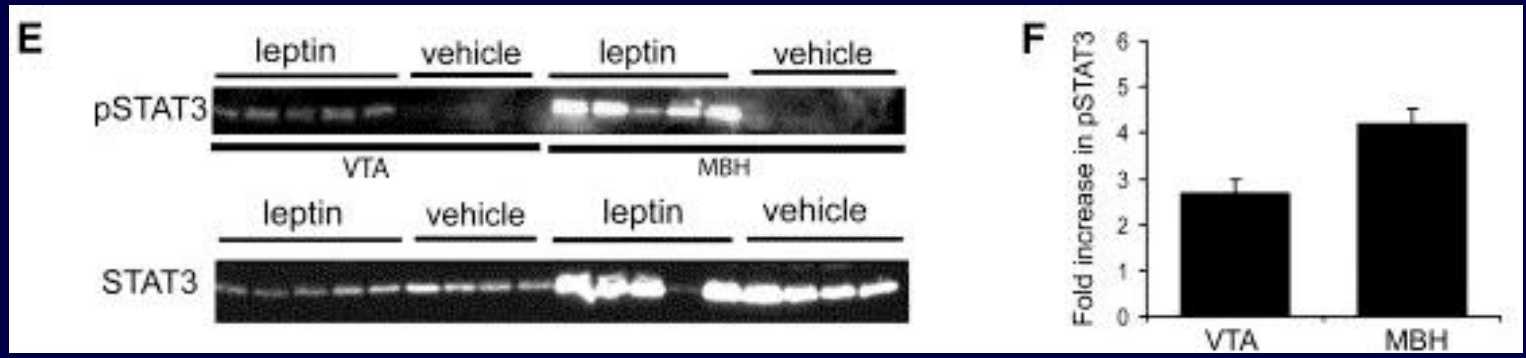
PARADOX:

But if you give a 5 year old obese kid a cookie:

PARADOX:

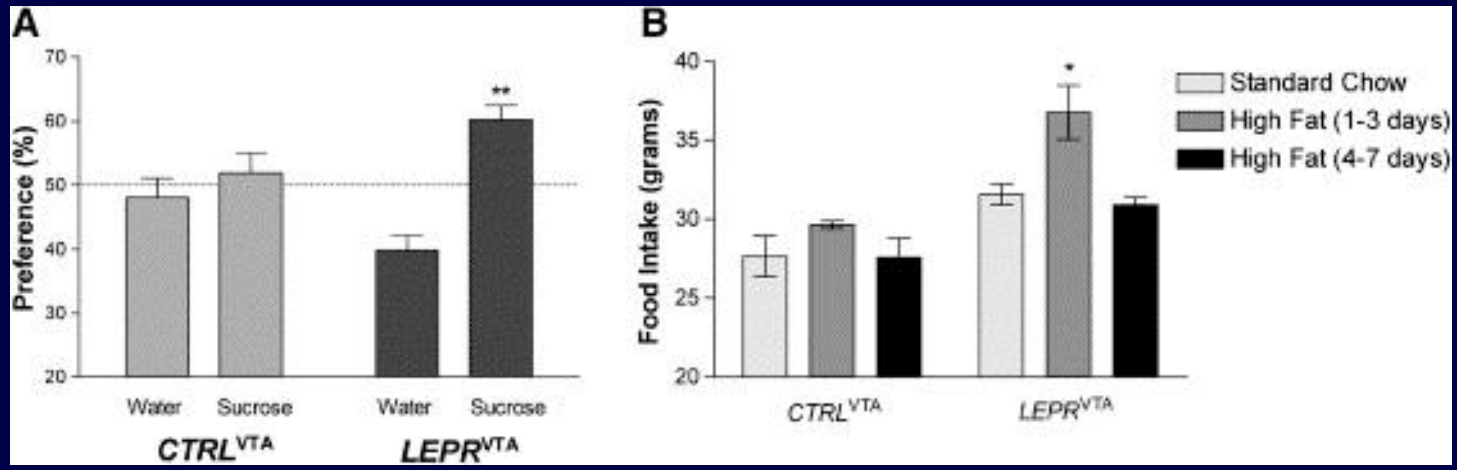
But if you give a 5 year old obese kid a cookie:

Leptin upregulates pSTAT-3 in the VTA and in the hypothalamus



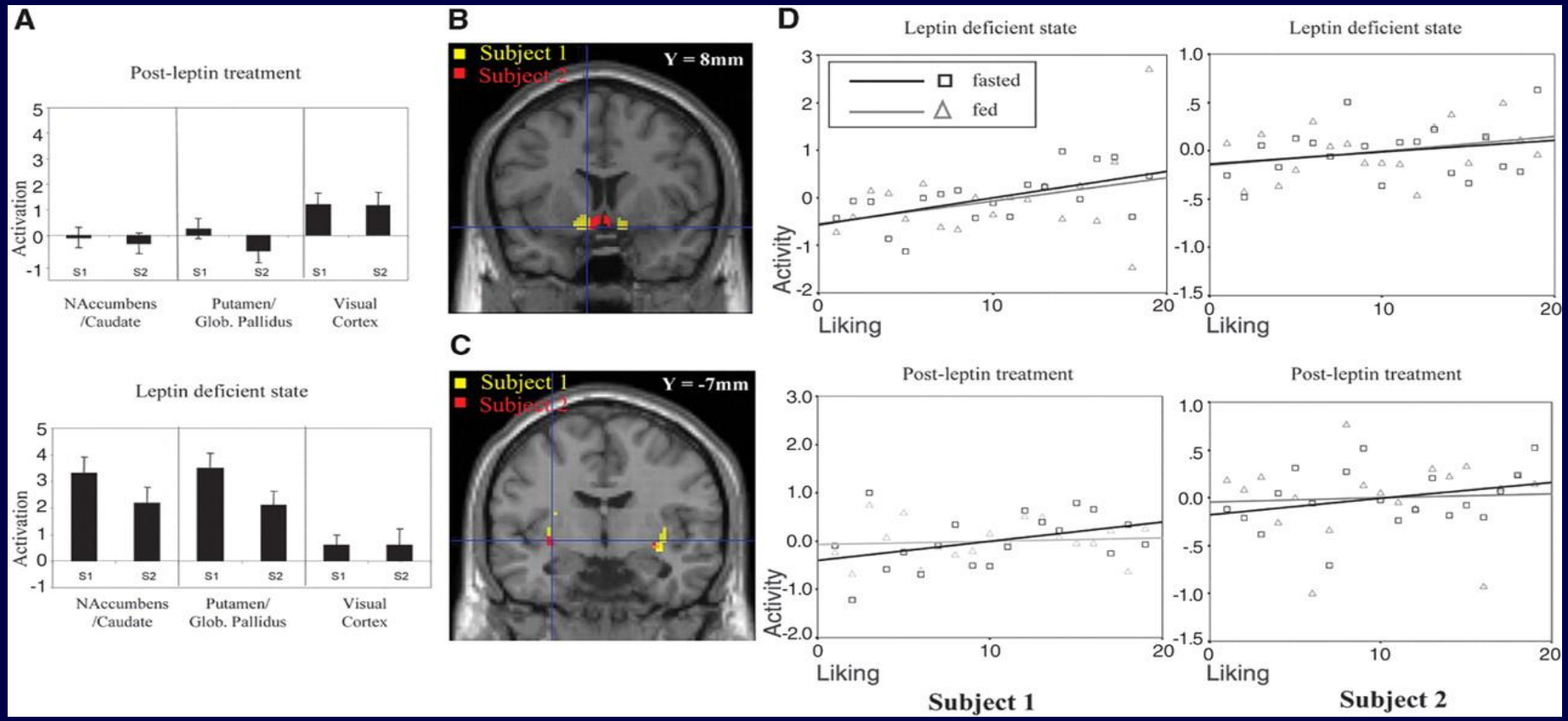
Fulton et al. Neuron 51:811, 2006

RNA-i mediated knockdown of leptin receptor in the VTA increases palatability of sucrose and fat



Hommel et al. Neuron 51:801, 2006

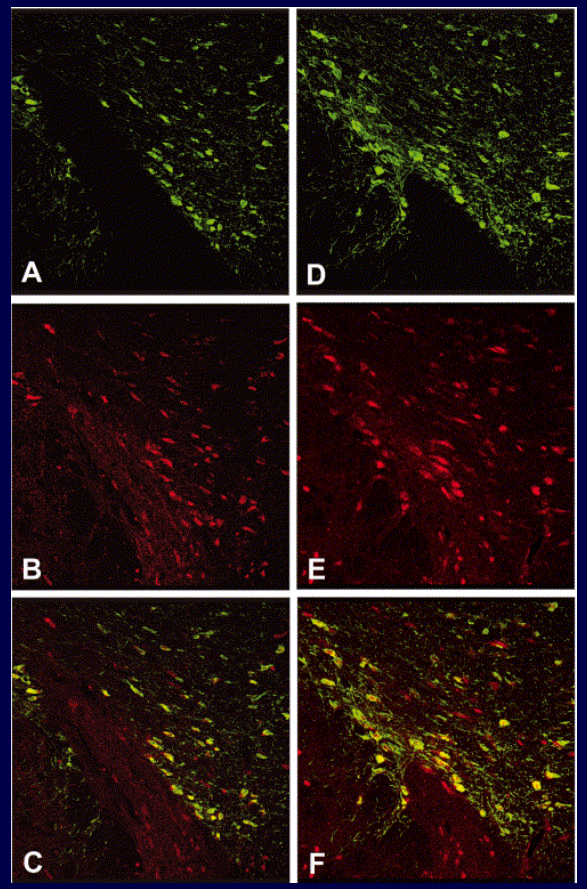
Leptin regulates brain responses to food images



Farooqi et al., Science 317:1355, 2007

Insulin and leptin receptors in dopaminergic neurons of the Ventral Tegmental Area (VTA)

Leptin receptor



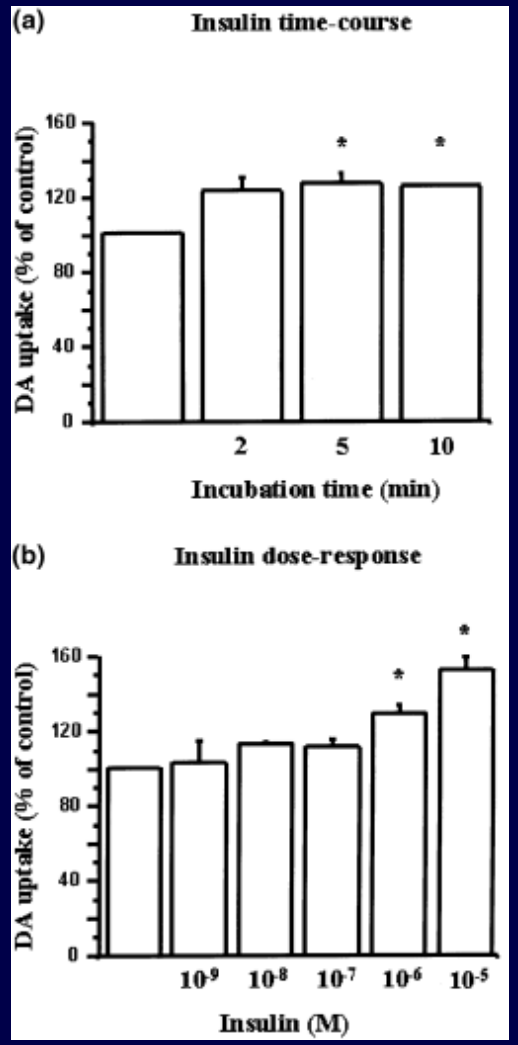
Tyrosine hydroxylase
(enzyme that makes dopamine)

Insulin receptor

Co-localization

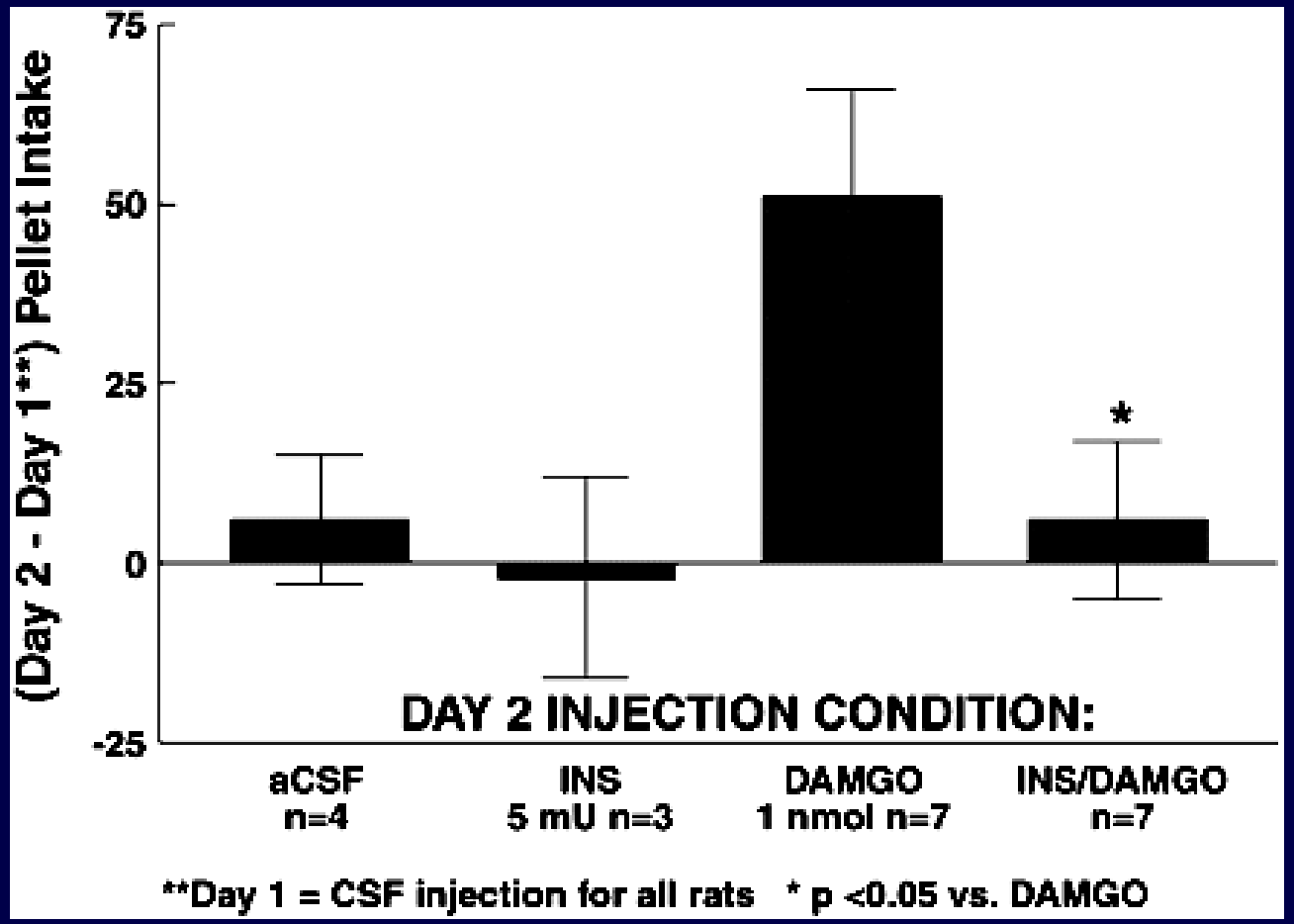
Figlewicz et al. Brain Res 964:107, 2003

Insulin stimulates [^{3}H]dopamine uptake in FLAG-hDAT cells



Carvelli et al. J Neurochemistry 81:859, 2002

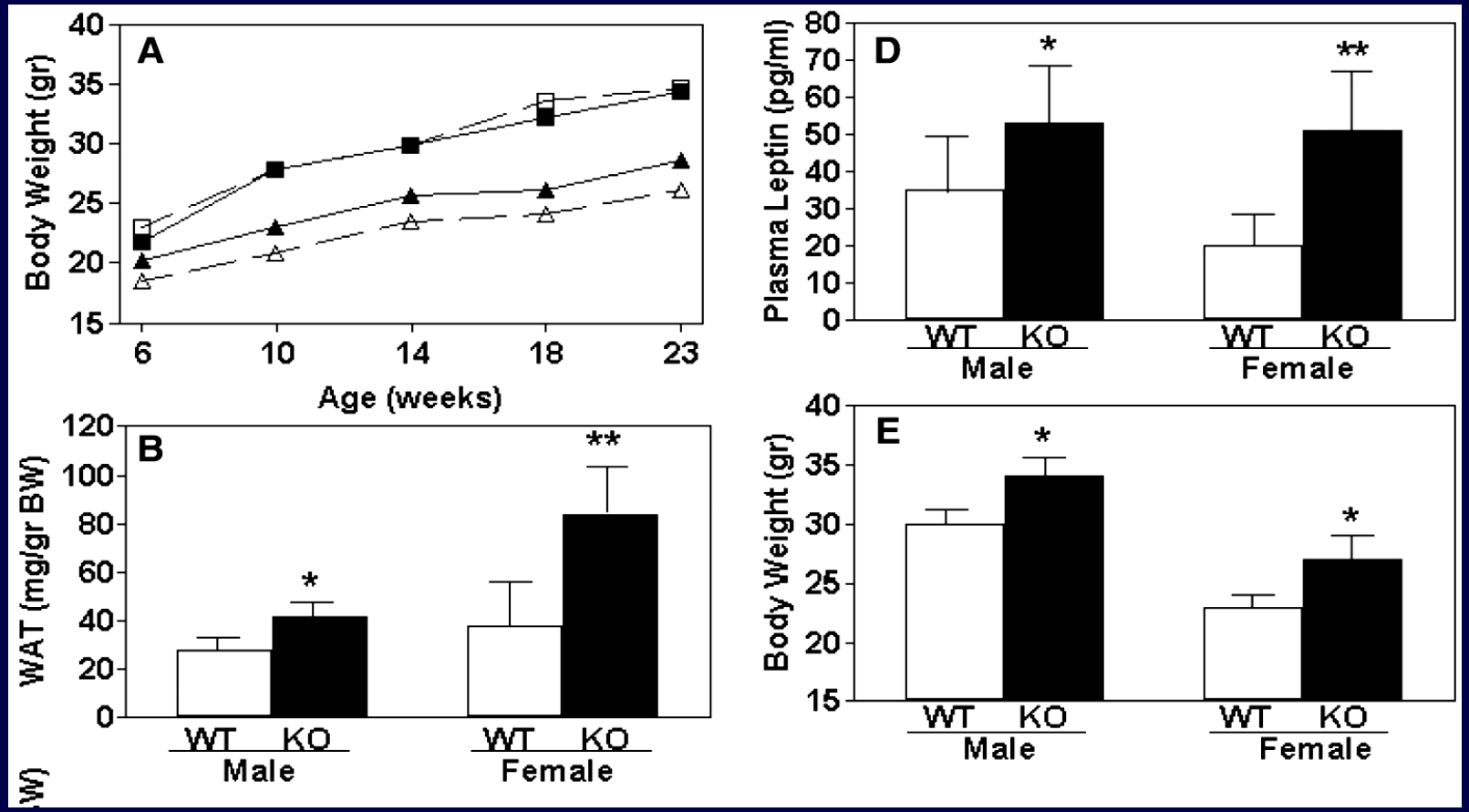
Insulin infusion into the Ventral Tegmental Area (VTA) blocks acute opiate effects on food intake



Figlewicz et al. Am J Physiol 284:R882, 2003

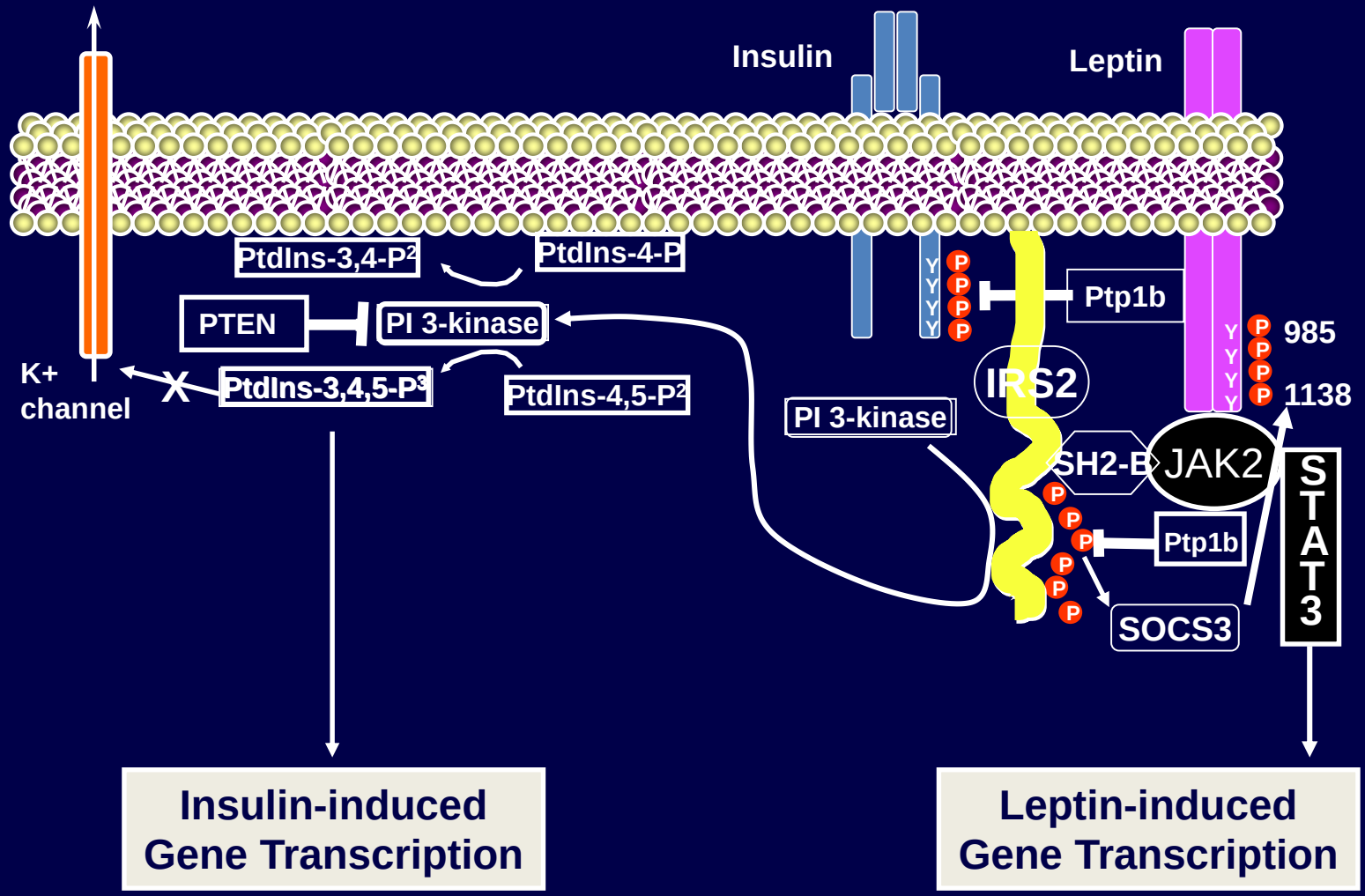
What does CNS insulin resistance do to reward?

The NIRKO (Brain Insulin Receptor Knockout) Mouse



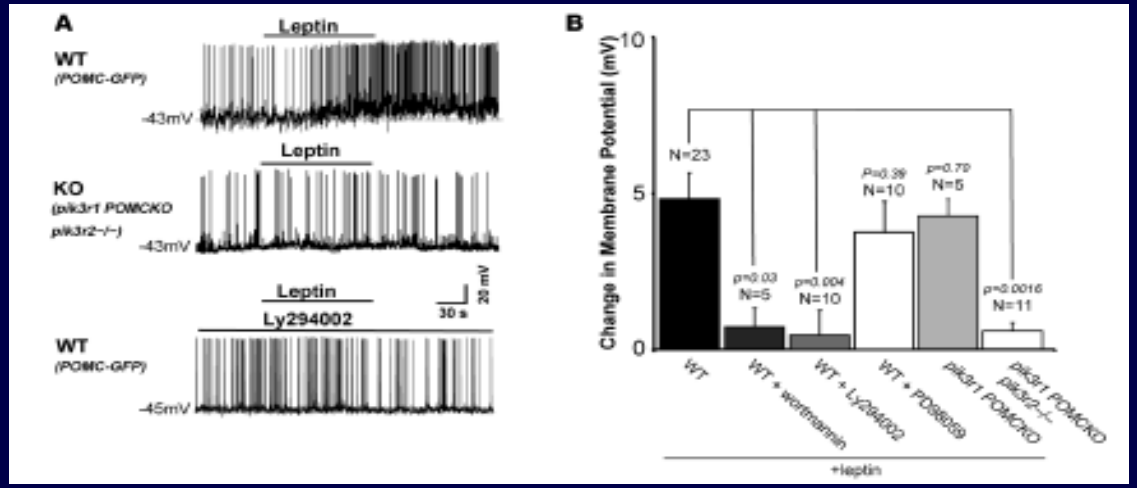
Brüning et al. Science 289:2122, 2000

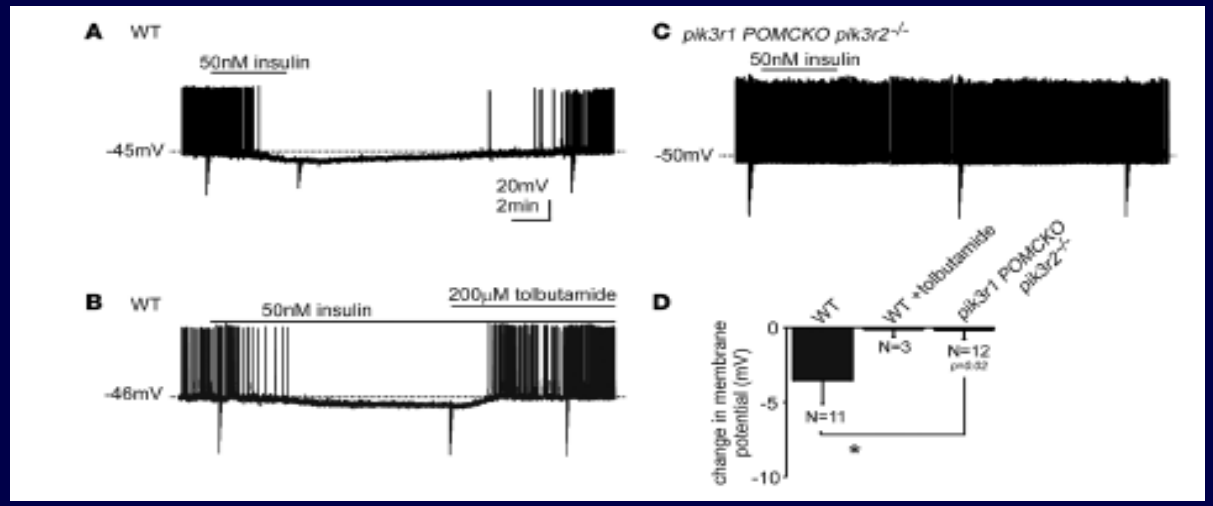
Knockout studies of leptin resistance



Lustig, Nature Clin Pract Endo Metab 2:447, 2006

Leptin depolarizes, while insulin hyperpolarizes POMC neurons through a PI3K-mediated mechanism





Hill et al. J Clin Invest 118:1796, 2008

Hyperinsulinemia blocks leptin signaling

Insulin inhibits leptin receptor signalling in HEK293 cells at the level of janus kinase-2: a potential mechanism for hyperinsulinaemia-associated leptin resistance

M. Kellerer[1], R. Lammers[1], A. Fritsche[1], V. Strack[1], F. Machicao[1], P. Borboni[3], A. Ullrich[2], H. U. Häring[1]

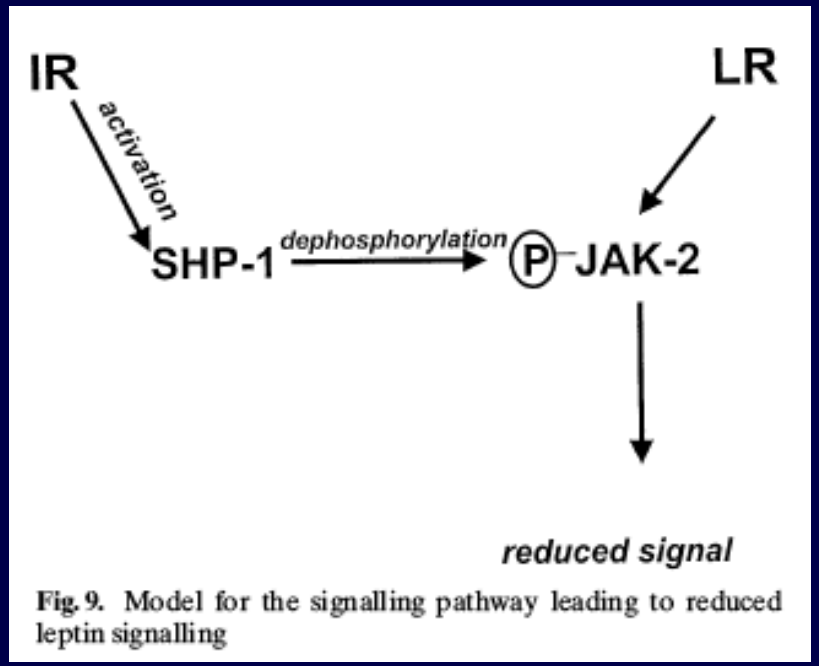
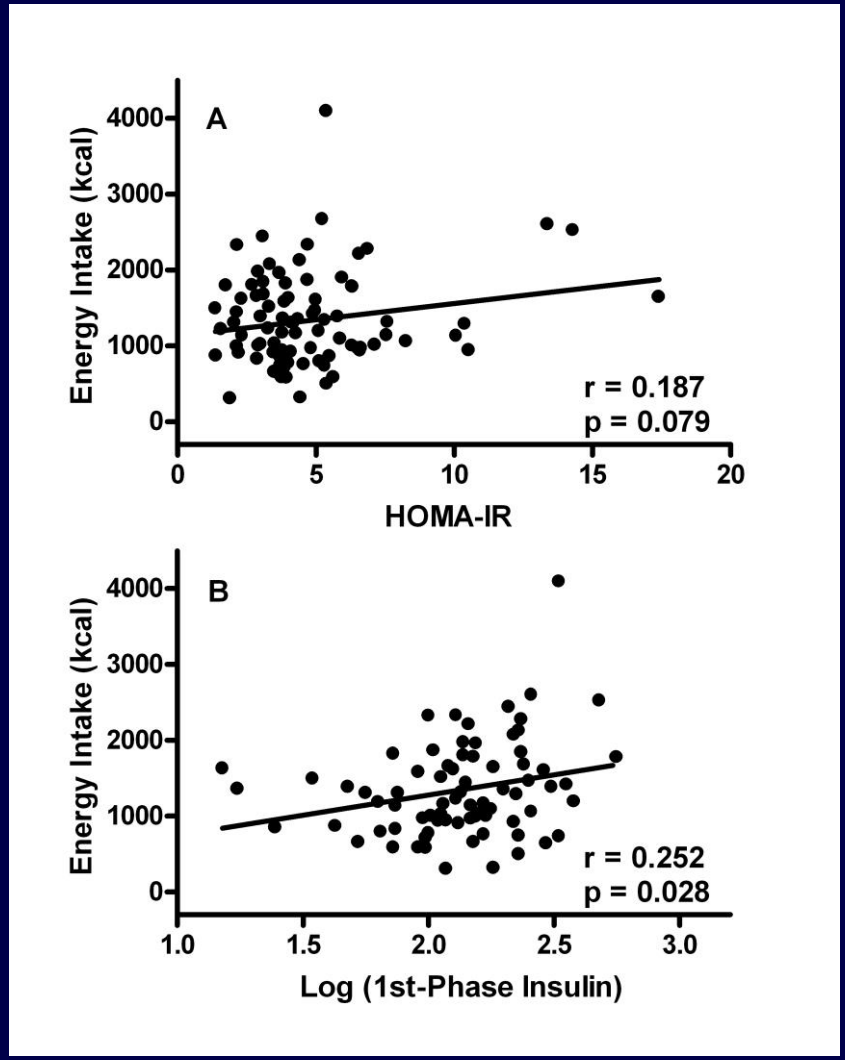


Fig. 9. Model for the signalling pathway leading to reduced leptin signalling

Kellerer et al. Diabetologia, 44:1125, 2001

Hyperinsulinemia correlates with energy intake in obese children

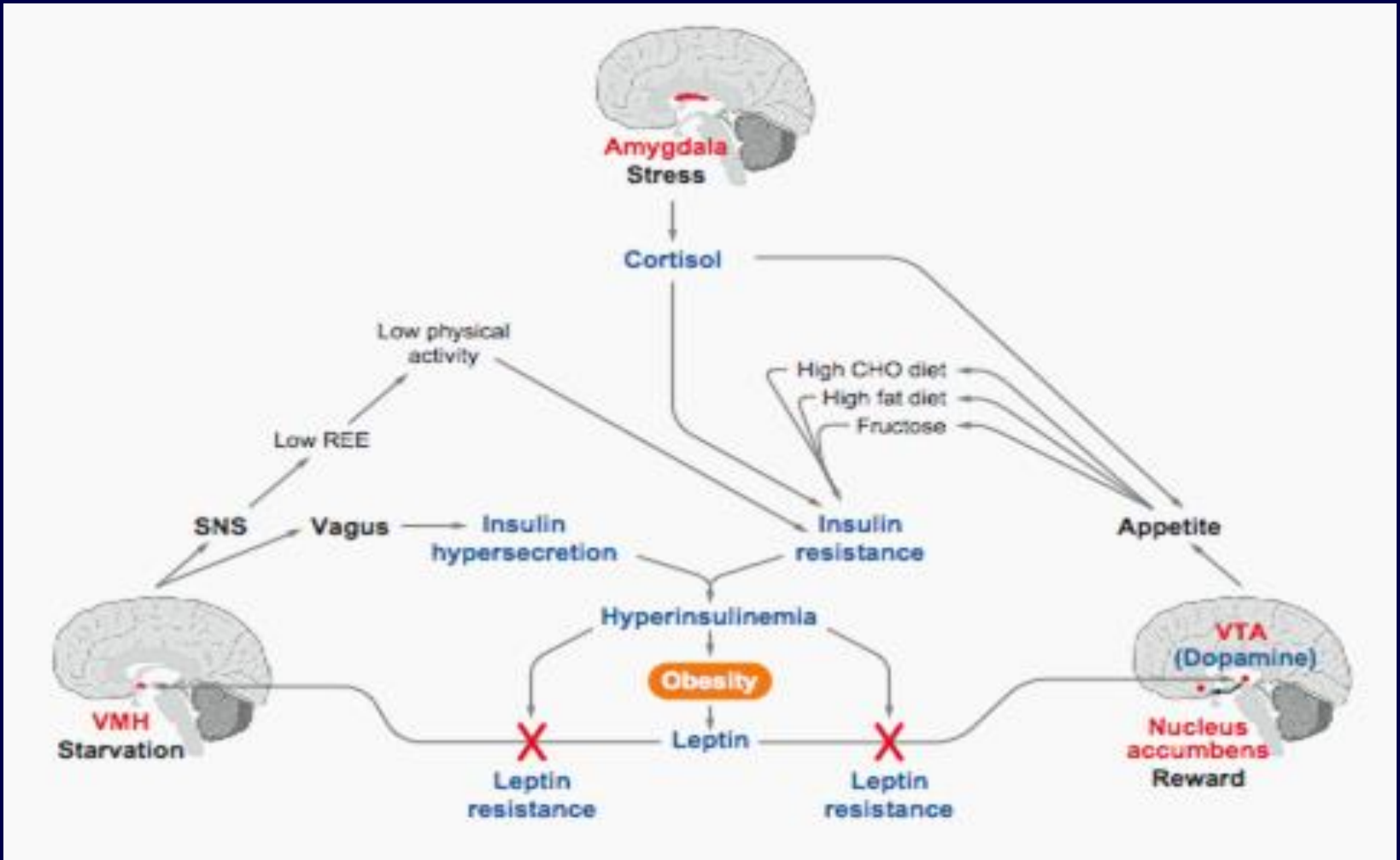


Han et al. J Pediatr 152:612, 2008

Chronic hyperinsulinemia promotes obesity by:

- driving energy into adipose tissue
- interfering with leptin signaling in the VMH (starvation)
- interfering with leptin extinguishing of dopamine clearance in the NA (addiction)

The “limbic triangle”



Mietus-Snyder and Lustig, Ann Rev Med 59:147, 2008

Direct effects on the reward system:

- Controlled by the Ventral Tegmental Area and Nucleus Accumbens

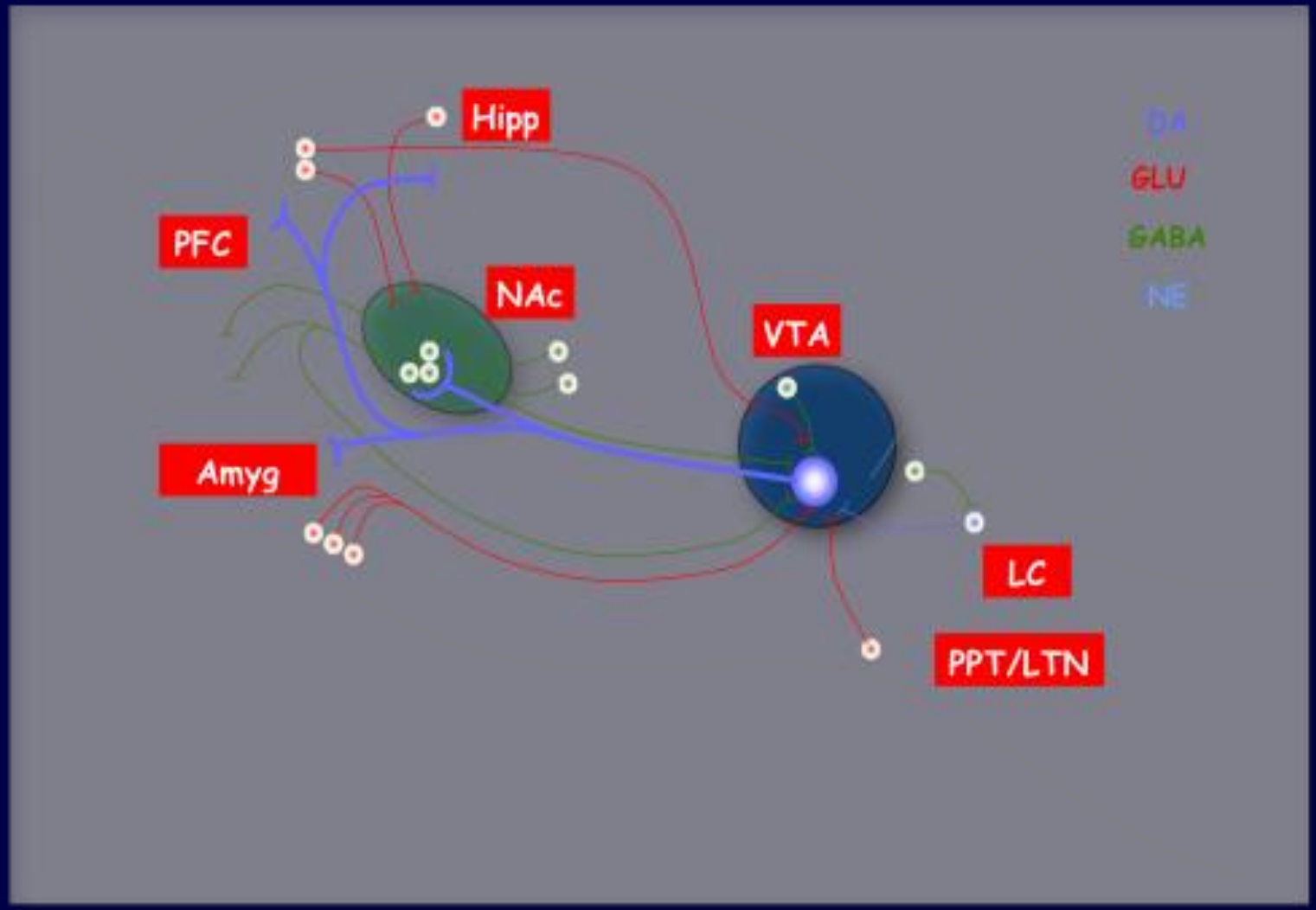
- dopamine

- endogenous opioids (mu and delta receptors)
- acetylcholine
- stress

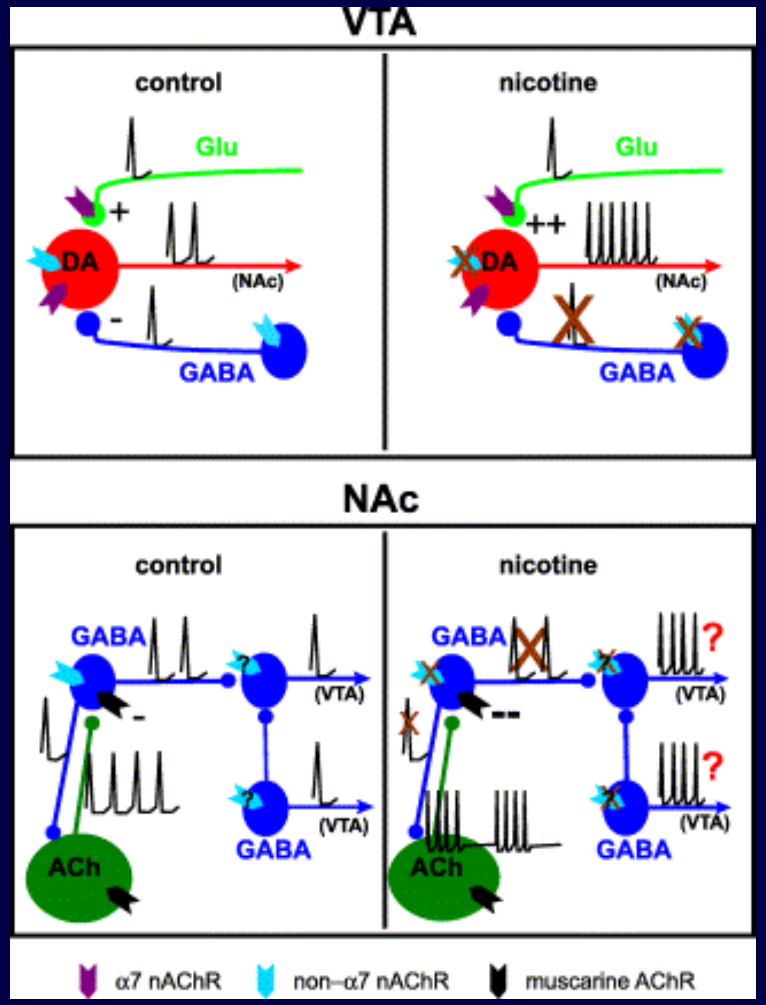
all equally important, but will not be discussed

The mesolimbic reward system in rodents

(Luscher, 2004)

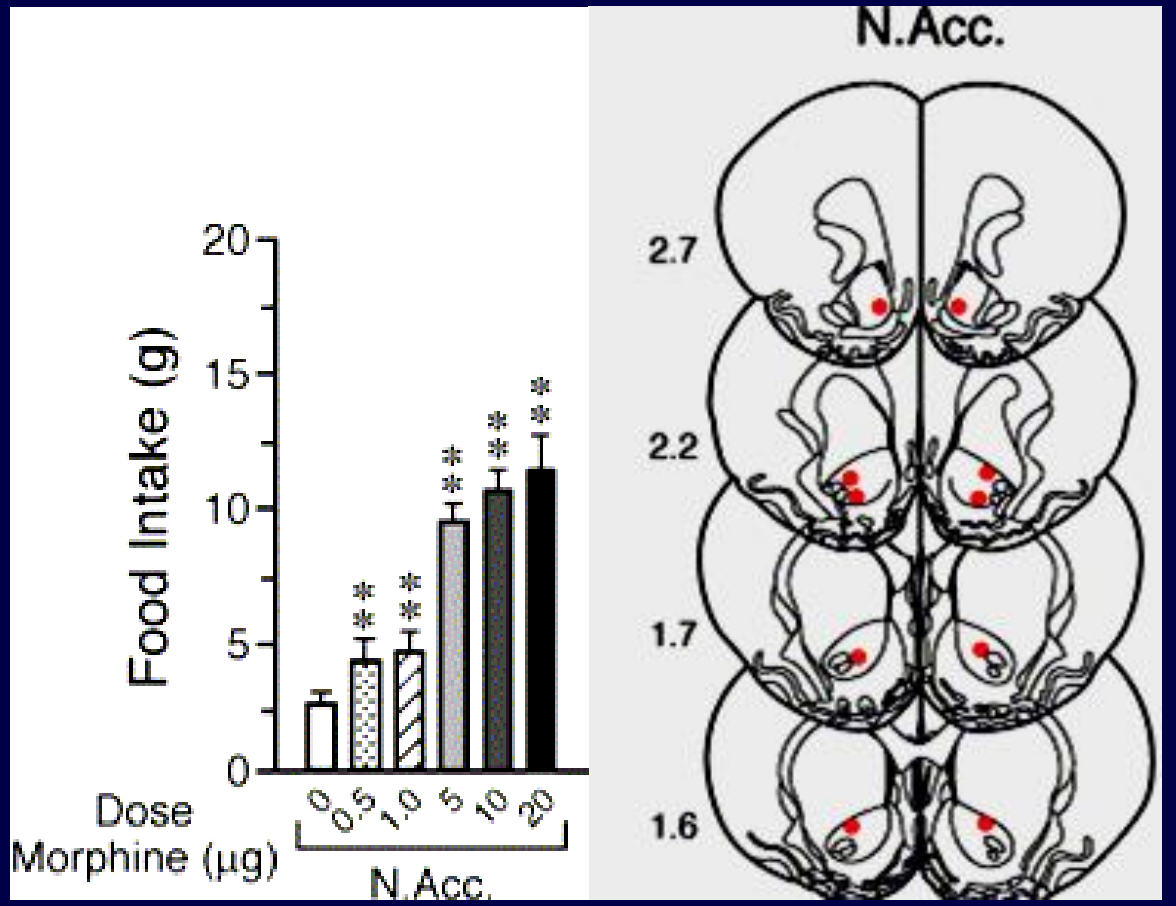


The Ventral Tegmental Area and the Nucleus Accumbens: Sites of opiate and nicotine effects on reward



Mansvelder et al. Eur J Pharmacol 480:117, 2003

The Ventral Tegmental Area and the Nucleus Accumbens: Sites of opiate and nicotine effects on reward



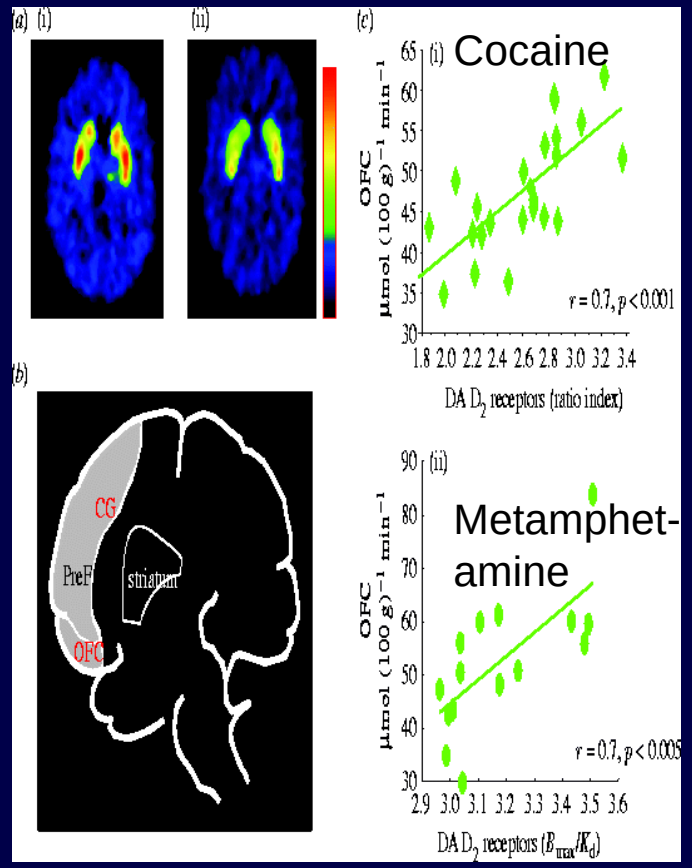
Kelly et al. Physiol Behav 76:365, 2002

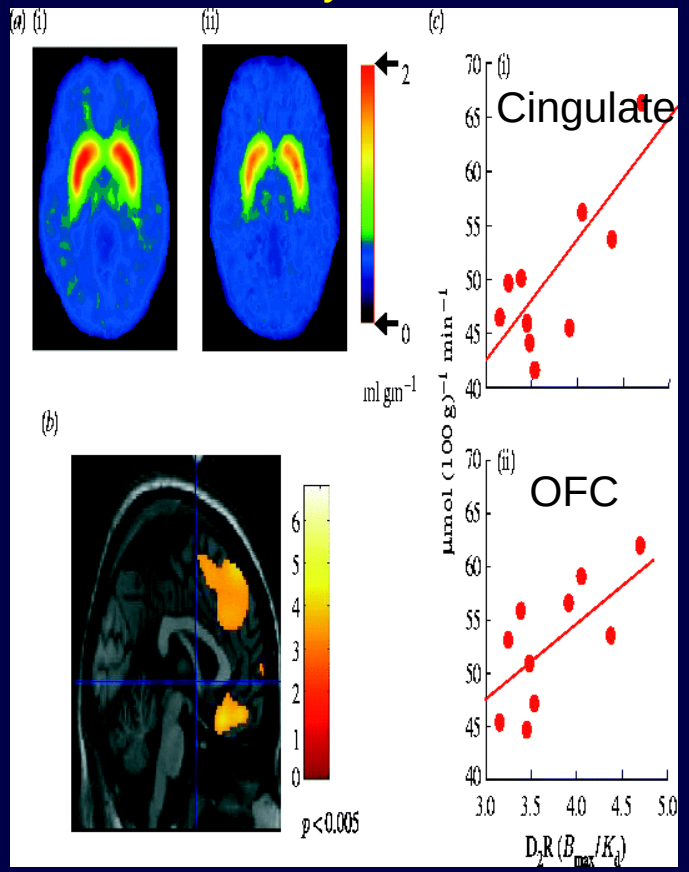
D_2 receptor binding correlates with glucose metabolism both in drug addiction and obesity

CTL Cocaine

CTL Obesity

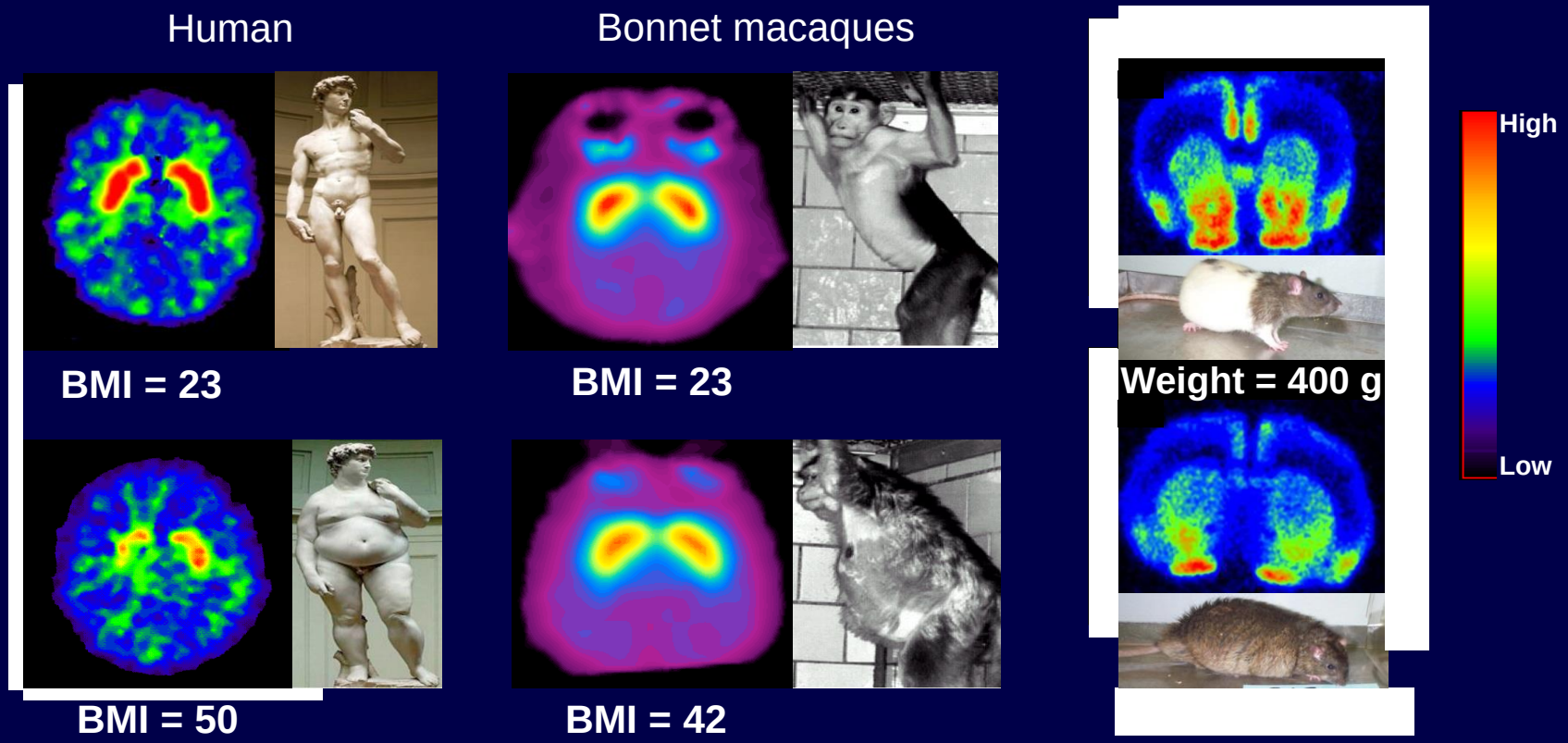
D2 receptors





Volkow et al. Philos Trans R Soc Lond B Biol Sci. 2008 363:3191, 2008

Decreased D_2 Receptors in Obese Human, Monkey and Rodent



Wang et al. J Nucl Med. 49(Suppl 1):208P, 2008; Thanos et al. Synapse. 62:50, 2008

Evidence of down-regulation of D_2 receptors

- Women who gained weight showed a reduction in striatal response to “sweet” vs. women who were weight stable or weight losers

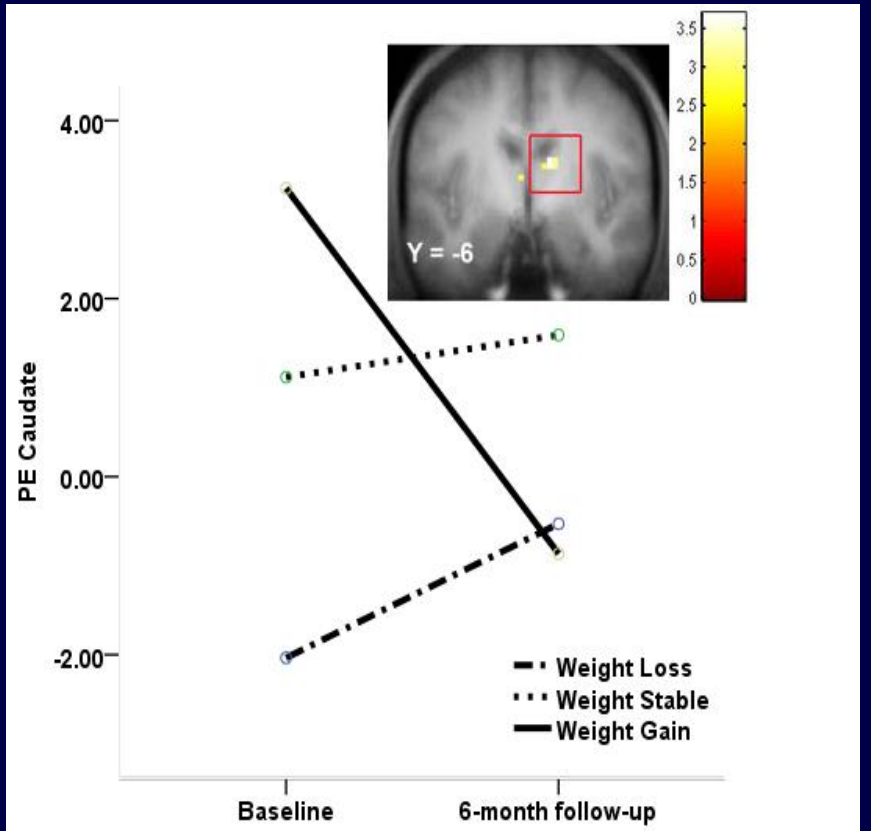
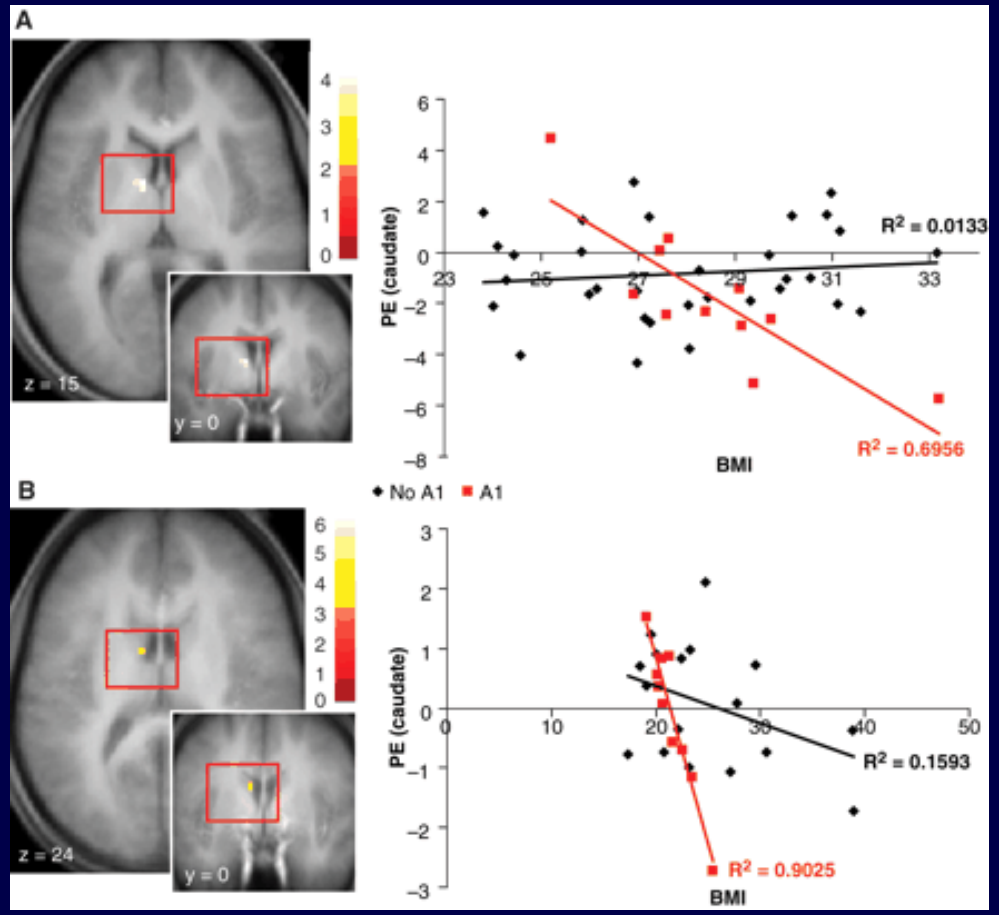


Figure 1. Less activation in the caudate (12, -6, 24, Z = 3.44, pFDR = 0.03, *k* = 3) in the weight gain group versus the weight stable group during milkshake receipt - tasteless receipt at 6-month follow-up compared to baseline.

Stice et al., J Neurosci. 2010 30:13105,.2010

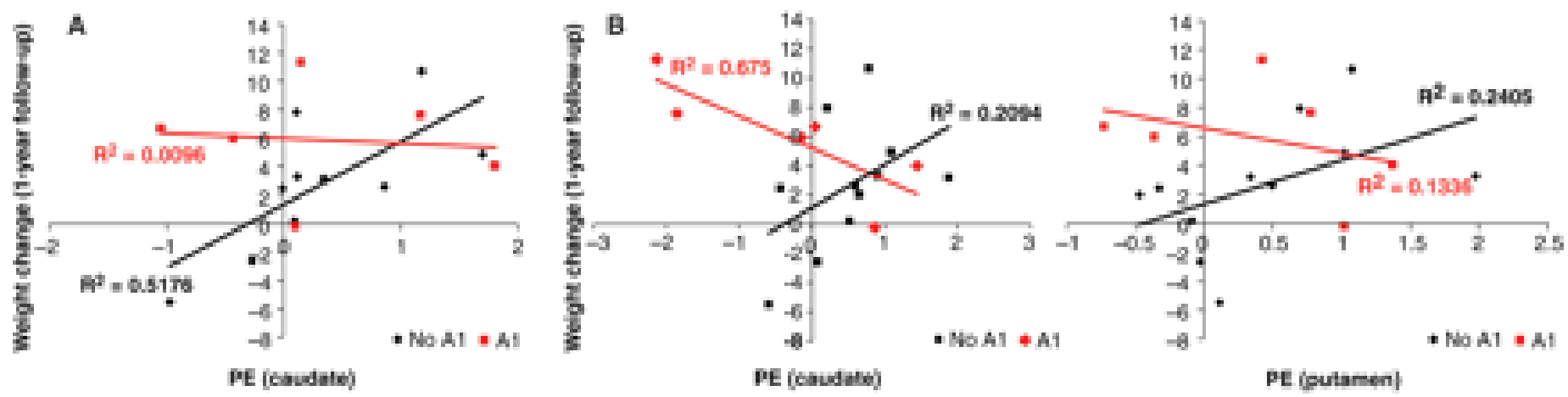
Blood oxygen level-depdendent fMRI: hypofunctioning dopaminergic activity in caudate, esp. with the Taq 1A allele (assoc. with low D2 receptors)



Stice et al. Science 322:449, 2008

Genetics of D_2 receptors and weight gain

Weight gain over one year correlated negatively with DA activity in those with the TaqA1 allele, and positively in those without the A1 allele

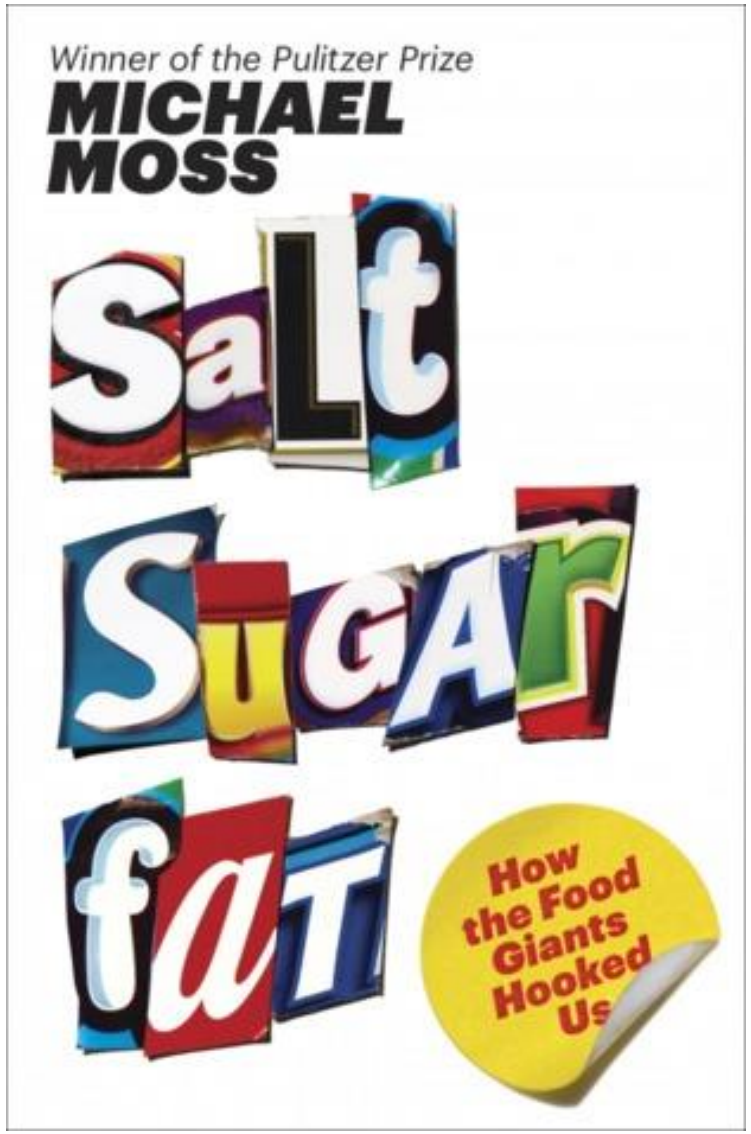


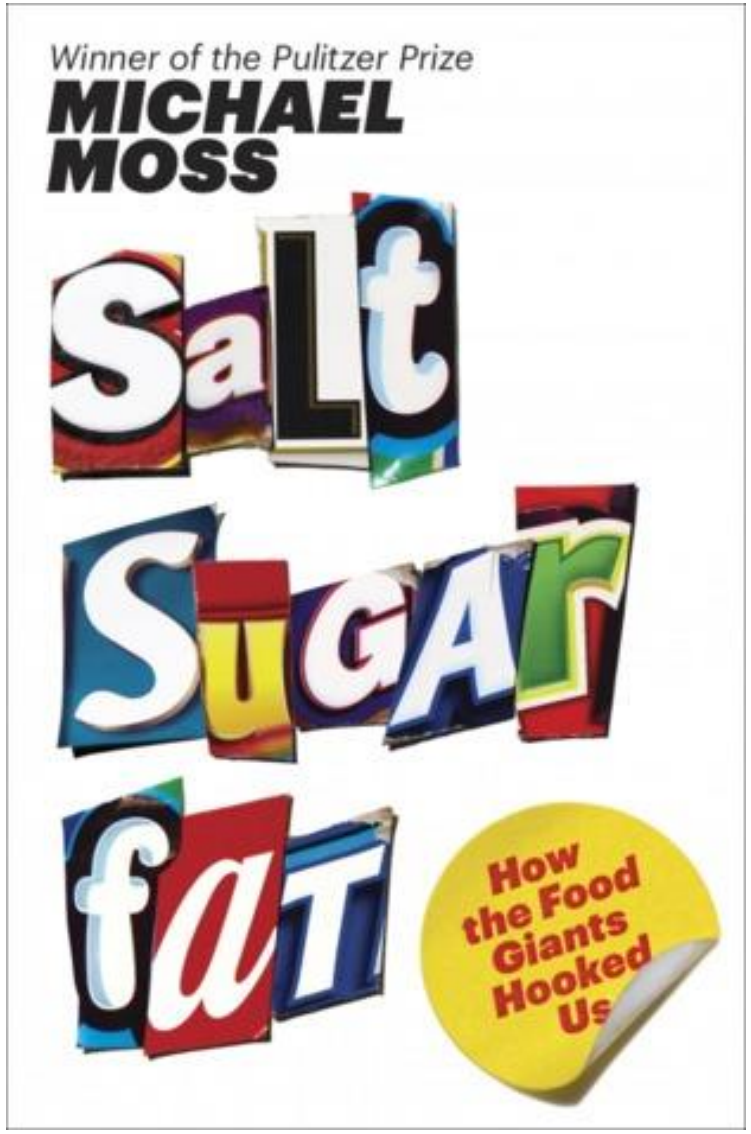
Stice et al. Science 322:449, 2008

Direct effects on the reward system:

Is fast food addictive?

Garber and Lustig, Curr Drug Abuse Rev 4:190, 2011


Winner of the Pulitzer Prize
MICHAEL MOSS
SALT
SUGAR
FAT
How the Food Giants Hooked Us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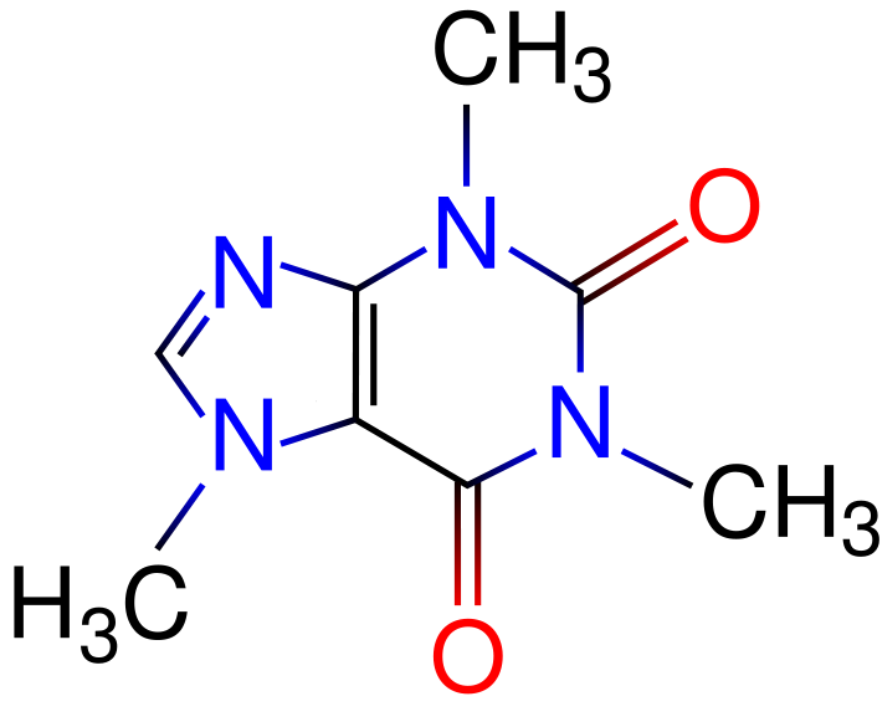
CH3
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CH3
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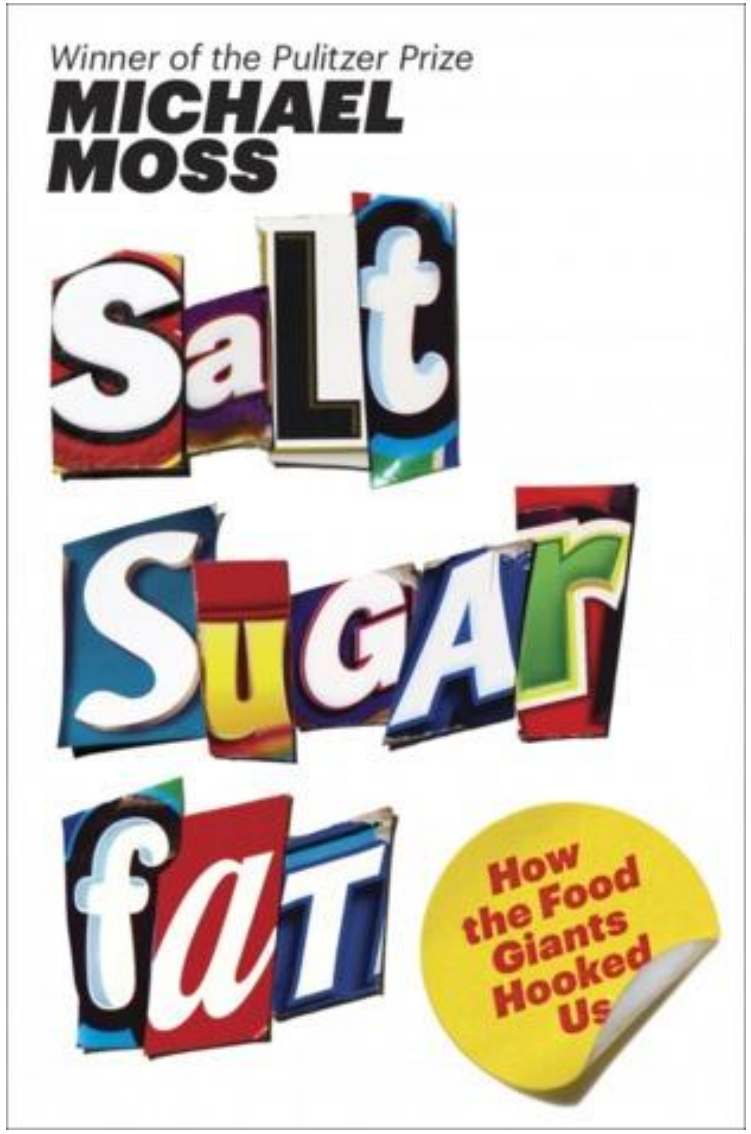
Caffeine

Salt

- In rodents, dopamine signaling (reward) in response to salt, bingeing, cross-sensitization with amphetamines

- In humans,
  - Lower threshold physiologically fixed
  - Higher levels attributed to “preference”, can retrain
  - Salt-losing congenital adrenal hyperplasia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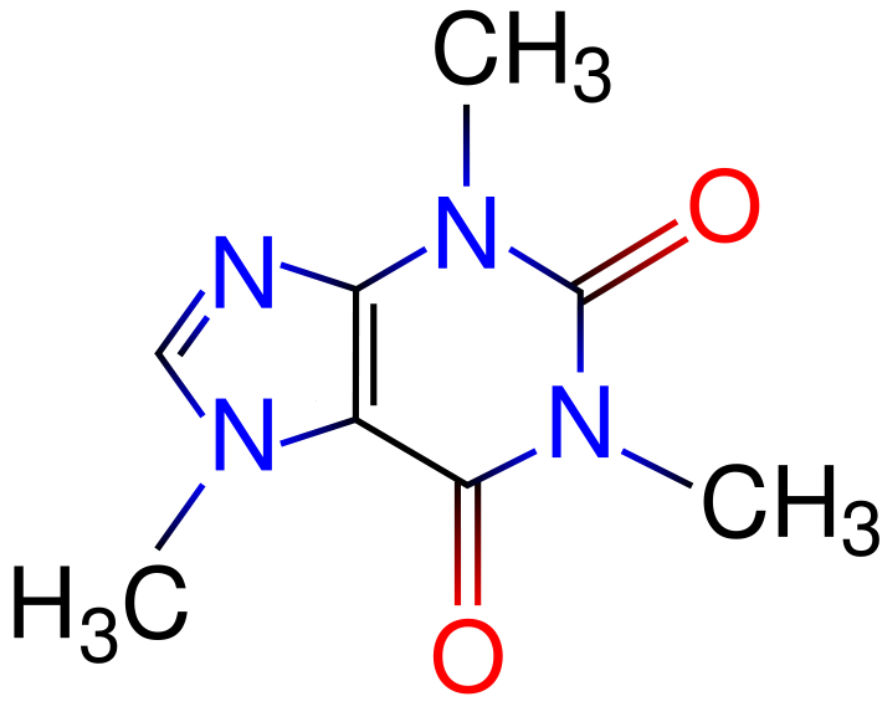
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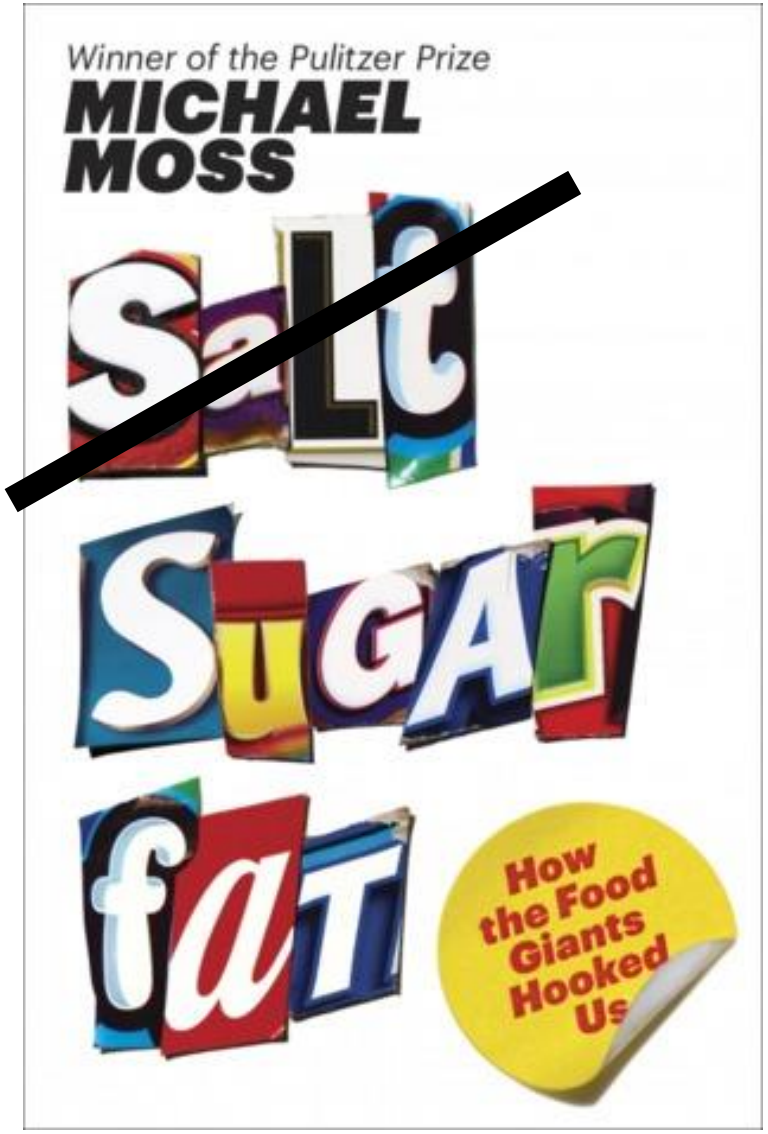
Caffeine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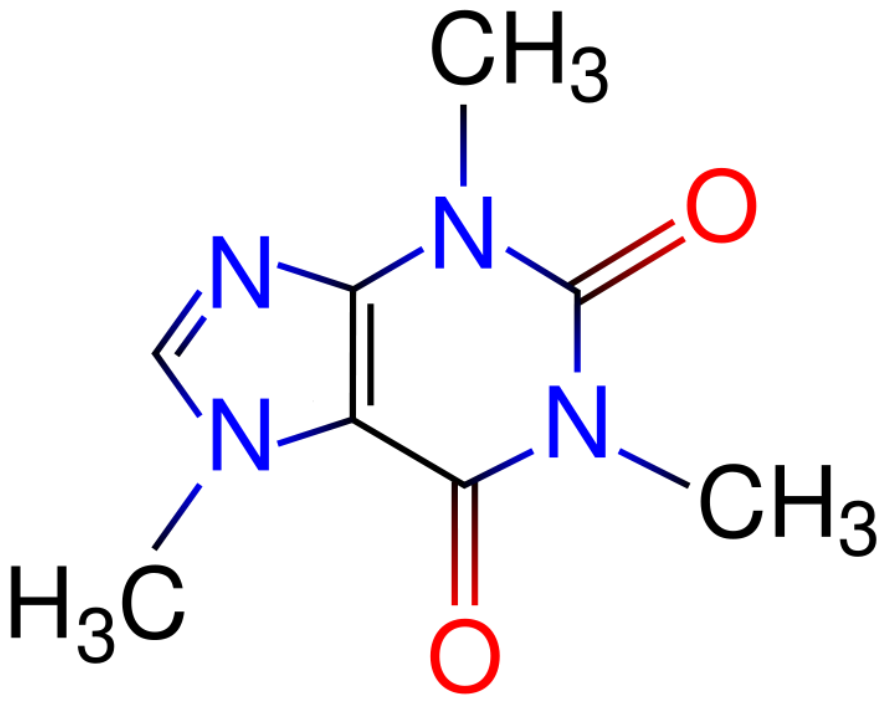
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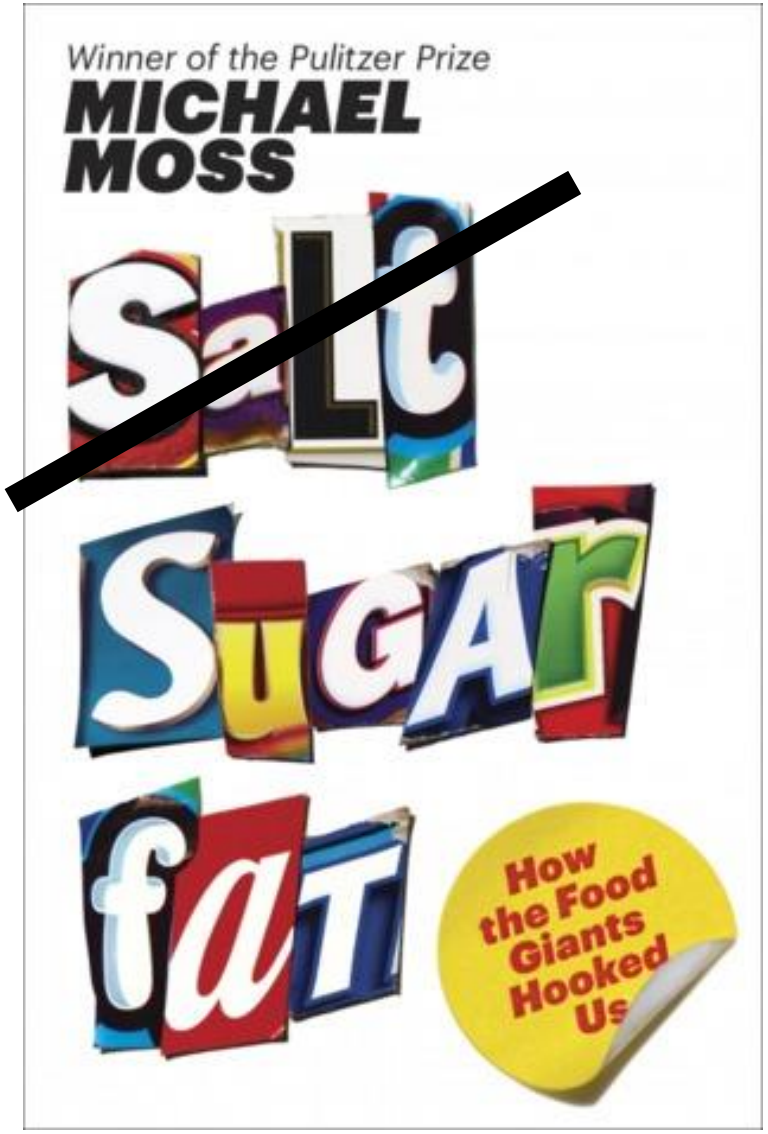
Caffeine

Fat

- Rodents binge but no signs of dependence
- In humans, binge foods are high fat but also high carb/sugar (e.g. pizza, ice cream)
  - Likely synergy, adding sugar increases preference for fatty foods [Drewnowski et al.]
- Atkins diet does not show dependence
- Energy density: stronger association with obesity, metabolic syndrome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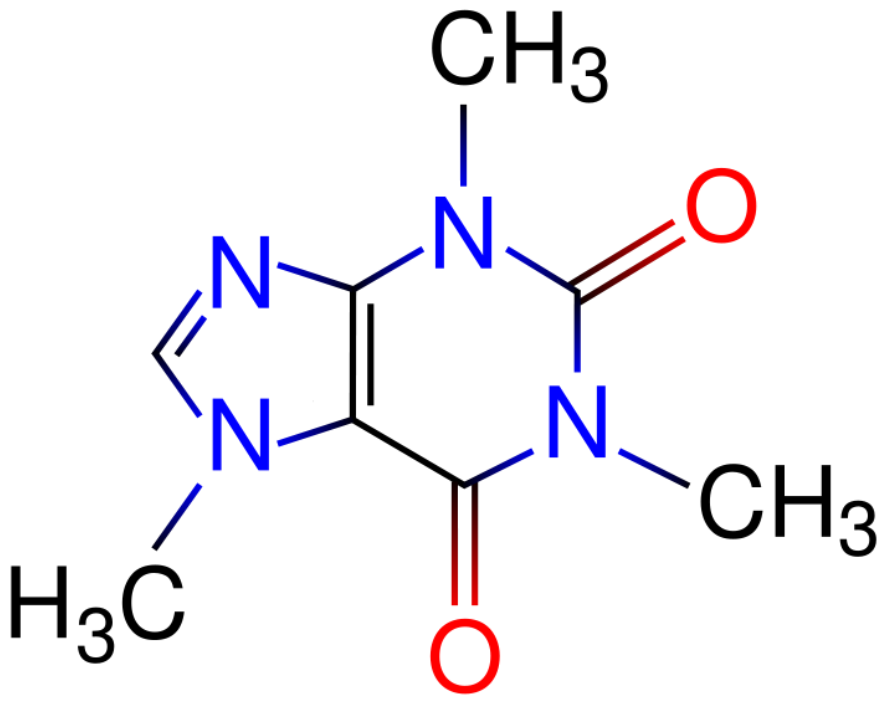
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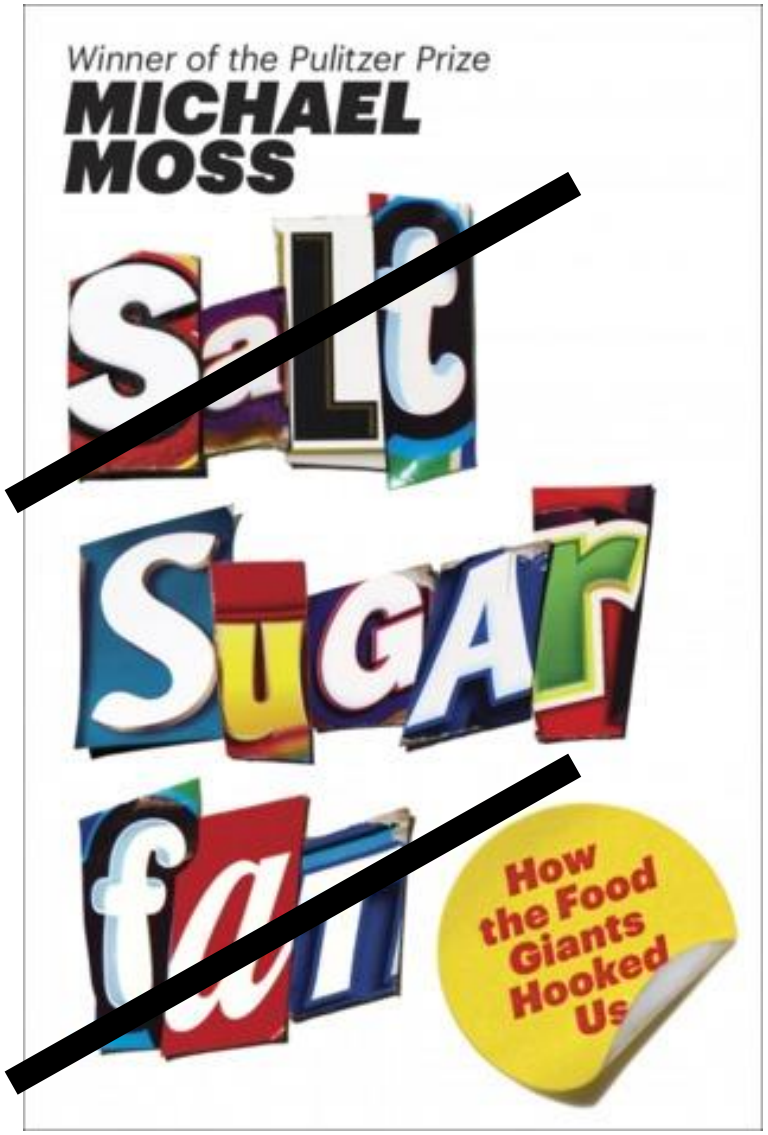
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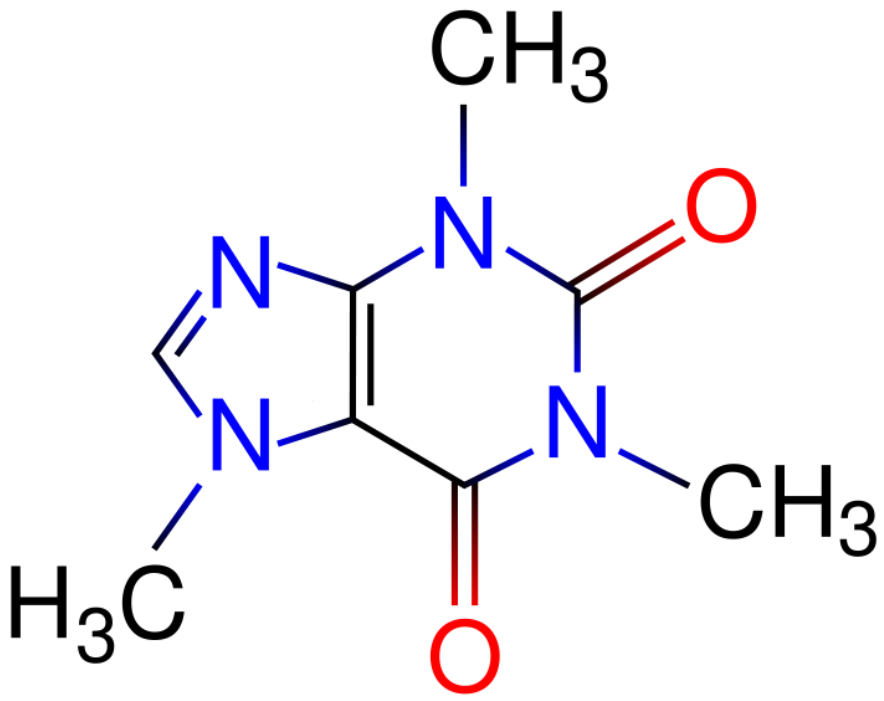
How the Food Giants Hooked Us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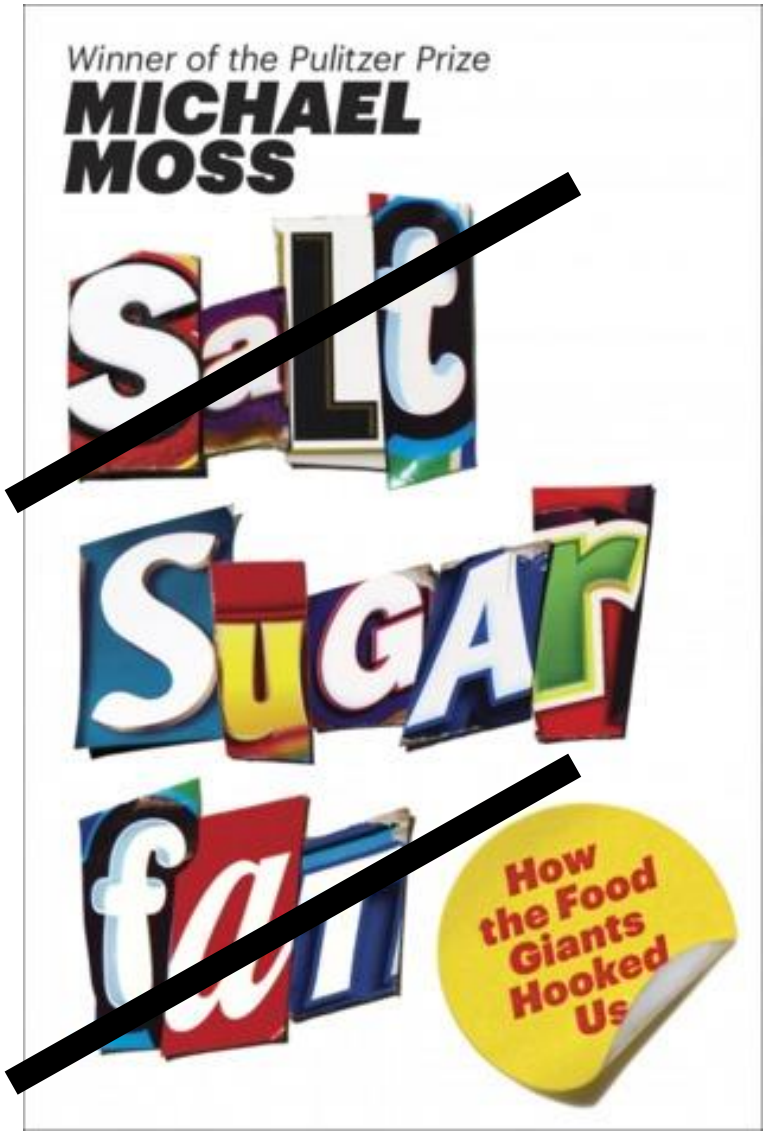
Caffeine

Caffeine

- “Model drug” of dependence
- In humans, dependence shown in children, adolescents and adults
  - 30% who consume it meet DSM criteria for dependence
  - Physiologic addiction established: headache (increased cerebral blood flow). Impaired task performance, fatigue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SALT
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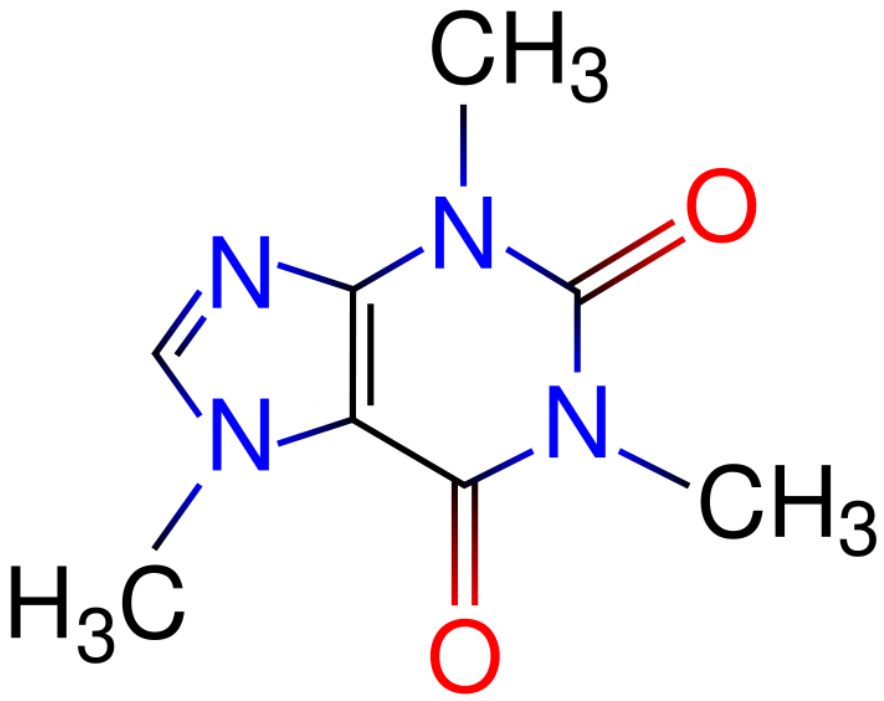
FAT
How the Food Giants Hooked Us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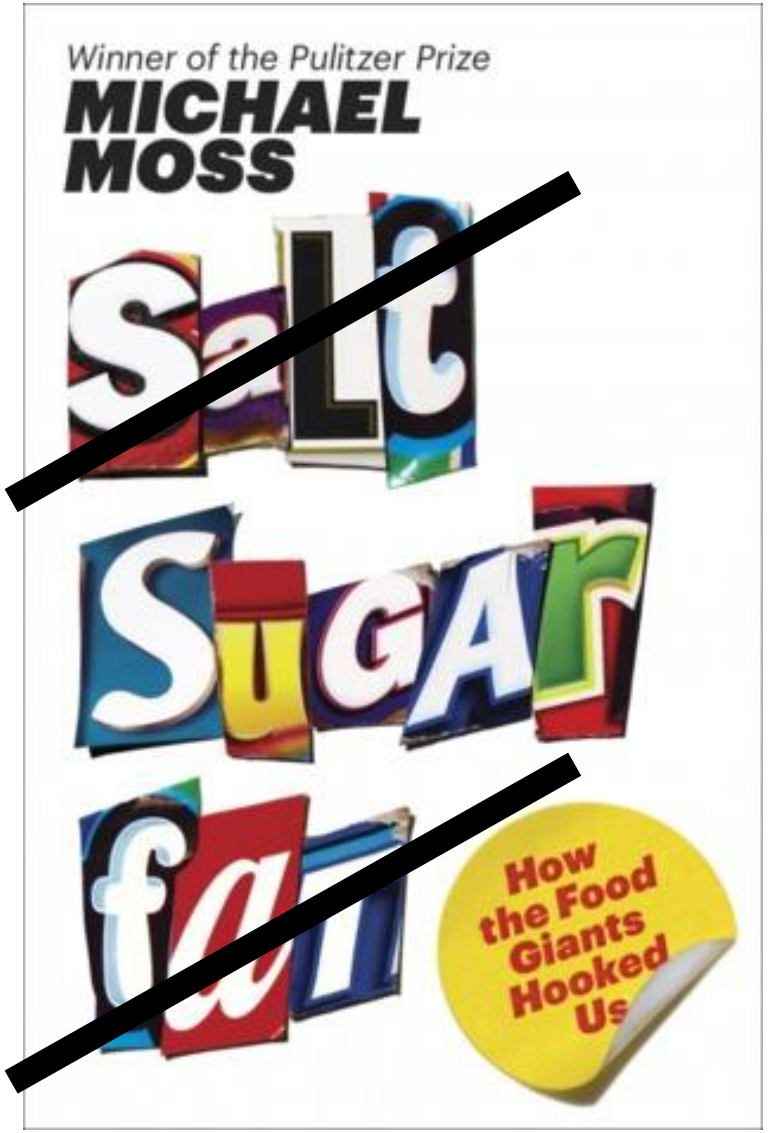
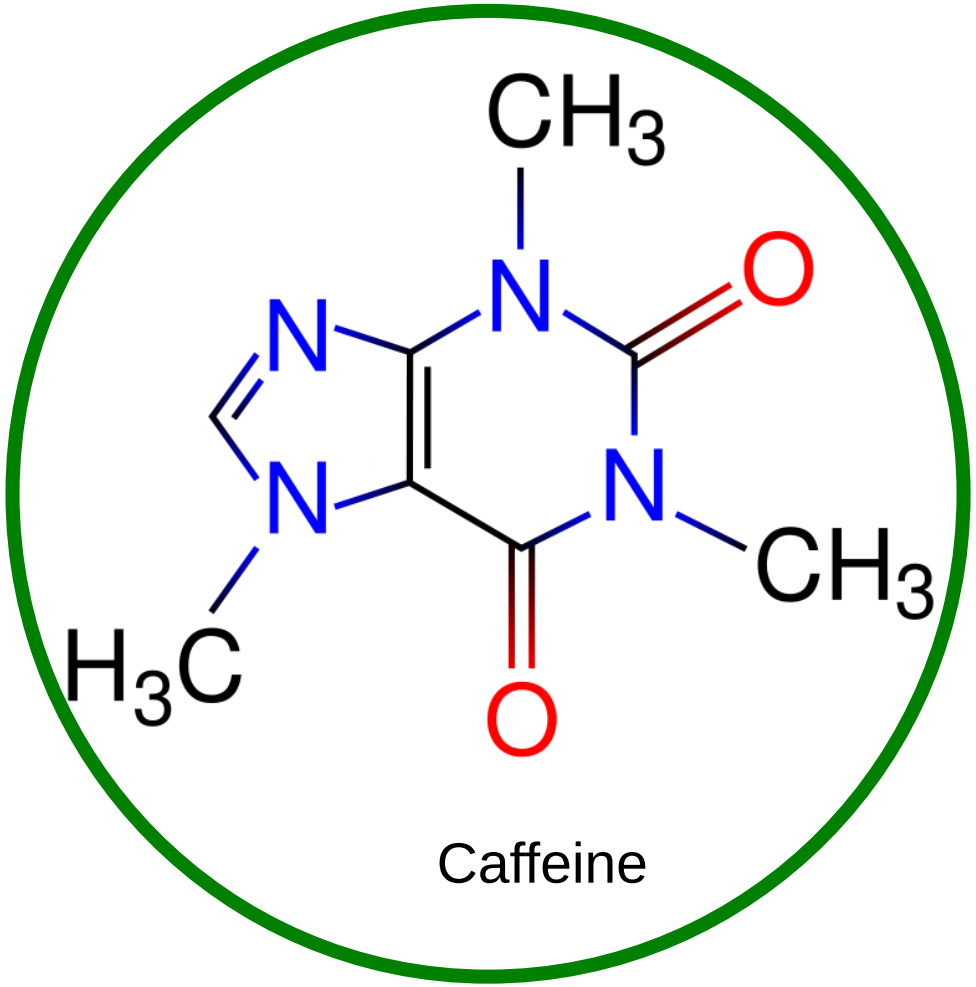
Caffeine


Winner of the Pulitzer Prize
MICHAEL MOSS
Salt
Sugar
fat
How the Food Giants Hooked Us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Caffeine

Direct effects on the reward system:

Is sugar (fructose) addictive?

Garber and Lustig, Curr Drug Abuse Rev 4:190, 2011

Sugar 'not addictive' says Edinburgh University study

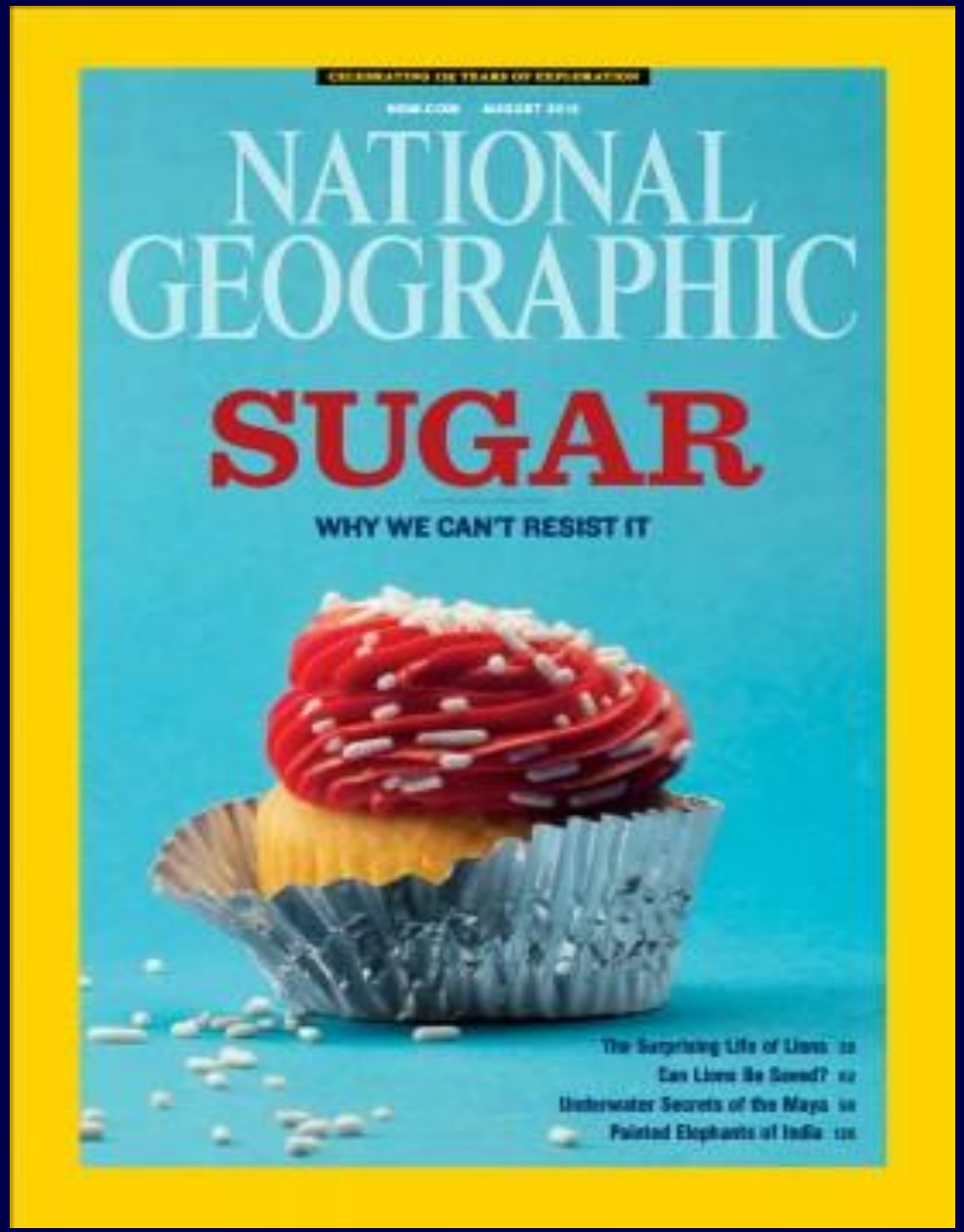
9 September 2014 | Edinburgh, Fife & East Scotland

Hebebrand et al. 2014
Neurofast (a review, not a study)

The research suggested people don't become addicted to individual foods but rather the act of eating

Sugar and opioids

Sweet-Ease increases endogenous opioids to reduce pain,
Even in neonates


NATIONAL GEOGRAPHIC
SUGAR
WHY WE CAN'T RESIST IT
The Surprising Life of Lions
Can Lions Be Saved?
Underwater Secrets of the Maya
Painted Elephants of India

Is there really such a thing as sugar addiction?

Need to look for similarities to drugs of dependence

- nicotine
- morphine
- amphetamine
- cocaine
- cannabis
- **ethanol**

What makes a milkshake so rewarding?

- Normal weight young adult subjects, fMRI
- Milkshakes with graded doses of fat vs. sugar
- The fat stimulated the somatosensory cortex (e.g. mouthfeel)
- Only sugar stimulated the nucleus accumbens
- Adding more fat was not additive to the effect of sugar on reward

Stice et al. Am J Clin Nutr 98:1377, 2013

RESEARCH ARTICLE

Dissociable Behavioral, Physiological and Neural Effects of Acute Glucose and Fructose Ingestion: A Pilot Study

Bettina Karin Wölnerhanssen[1☯*], Anne Christin Meyer-Gerspach[1☯], André Schmidt[2,3], Nina Zimak[1], Ralph Peterli[4], Christoph Beglinger[1], Stefan Borgwardt[2,3]

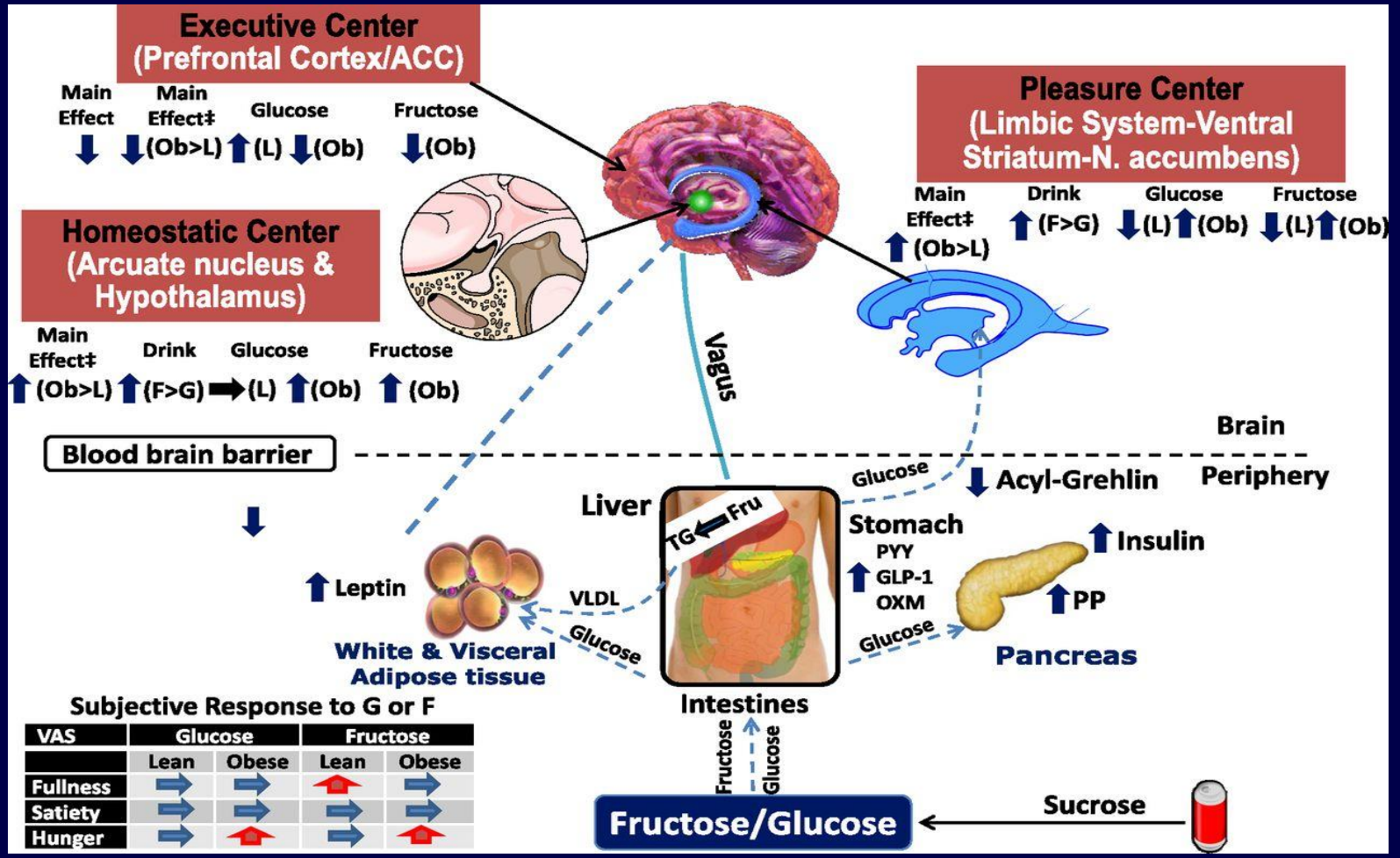
1 Department of Gastroenterology, University Hospital of Basel, Basel, Switzerland, 2 Medical Image Analysis Center, University Hospital of Basel, Basel, Switzerland, 3 Department of Psychiatry, University Hospital of Basel, Basel, Switzerland, 4 Department of Surgery, St. Clara Hospital, Basel, Switzerland

No satiety or fullness with fructose compared with glucose
No insulin rise with fructose compared with glucose

fMRI:
Glucose: caudate, putamen, precuneus, lingual gyrus
Fructose: amygdala, hippocampus, parahippocampus, orbitofrontal cortex
precentral gyrus

PLoS One 10(6):e0130280, 2014

Effects of fructose and glucose on the brain in normal and obese adolescents



Jastreboff et al. Diabetes 65:1929, 2016

How about humans?
The DSM-V criteria for addiction

2 of the 11 following criteria within a 12-month period:

1. Tolerance
2. Withdrawal

Physiologic

3. Craving or a strong desire to use
4. Use resulting in a failure to fulfill major role obligations (work, school, home);
5. Recurrent use in physically hazardous situations (e.g. driving);
6. Use despite social or interpersonal problems caused or exacerbated by use;
7. Taking the substance in larger amounts or over a longer period than intended;
8. Attempt to quit or cut down;
9. Time spent seeking or recovering from use;
10. Interference with life activities;
11. Use despite negative consequences.

Psychologic (Dependence)

NeuroFAST
www.neurofast.eu

CONSENSUS

NeuroFAST consensus opinion on food addiction

•**In humans, there is no evidence that a specific food, food ingredient or food additive causes a substance-based type of addiction** (the only currently known exception is caffeine which via specific mechanisms can potentially be addictive).

• Within this context **we specifically point out that we do not consider alcoholic beverages as food**, despite the fact that one gram of ethanol has an energy density of 7 kcal.

Alcohol and caffeine are really "food additives"

Is sugar a “food”?

FDCA: 321.201(f) The term "food" means (1) articles used for food or drink for man or other animals, (2) chewing gum, and (3) articles used for components of any such article.

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Webster: a material consisting essentially of protein, carbohydrate, and fat used in the body of an organism to sustain growth, repair, and vital processes and to furnish energy; also: such food together with supplementary substances (as minerals, vitamins, and condiments)

Is sugar a “food”?

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Webster: a material consisting essentially of protein, carbohydrate, and fat used in the body of an organism to sustain growth, repair, and vital processes and to furnish energy; also: such food together with supplementary substances (as minerals, vitamins, and condiments)

Sugar provides only energy, but that should make it a food, right?

Can you name an energy source that is:

Can you name an energy source that is:

Not necessary for life

Can you name an energy source that is:

Not necessary for life

There is no biochemical reaction in the body that requires it

Can you name an energy source that is:

- Not necessary for life
- There is no biochemical reaction in the body that requires it
- Is not nutrition

Can you name an energy source that is:

- Not necessary for life
- There is no biochemical reaction in the body that requires it
- Is not nutrition
- When consumed in excess it is toxic

Can you name an energy source that is:

- Not necessary for life
- There is no biochemical reaction in the body that requires it
- Is not nutrition
- When consumed in excess it is toxic
- We love it anyway, and it’s addictive

Can you name an energy source that is:

Not necessary for life

There is no biochemical reaction in the body that requires it

Is not nutrition

When consumed in excess it is toxic

We love it anyway, and it’s addictive

Answer: Ethanol

American Dietetic Association

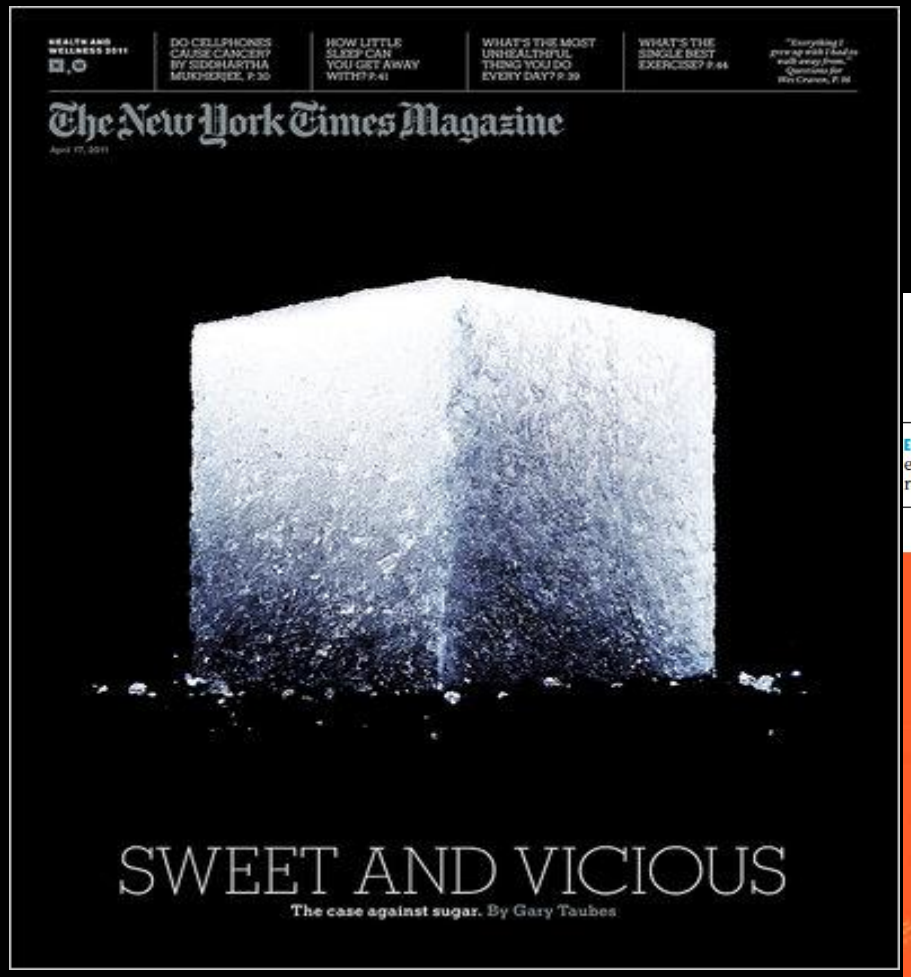
RESEARCH

Review

Fructose: Metabolic, Hedonic, and Societal Parallels with Ethanol

ROBERT H. LUSTIG, MD

Lustig, J Am Diet Assoc 110:1307, 2010



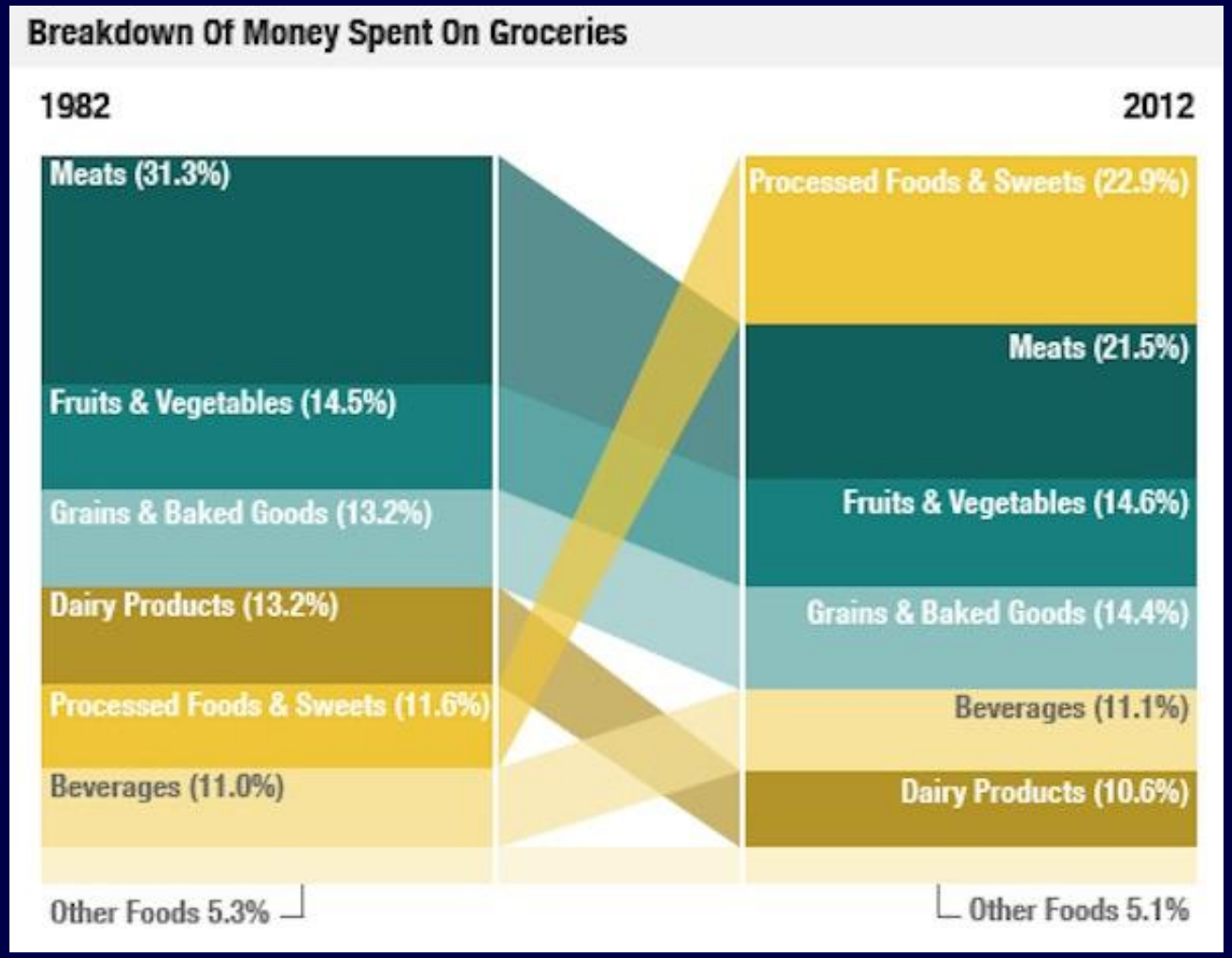
New York Times, April 17, 2011

Nature 487:27-29, Feb 1, 2012

Addictive and hazardous to your health

Addictive and hazardous to your health

How our food dollars have been reallocated



Philpott, Mother Jones 2012 (from Bureau of Labor Statistics)

Of the 600,000 items in the American food supply,

74% have added sugar (sucrose, HFCS)

56% of sugar is in ultra-processed foods

Ng et al. J Acad Nutr Diet 2012; Srour et al. BMJ 365:I1451, 2019

nutrients

MDPI

Article

Ultraprocessed Food: Addictive, Toxic, and Ready for Regulation

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Received: 27 September 2020; Accepted: 23 October 2020; Published: 5 November 2020

check for updates

Summary

- The only items in “junk” food that are addictive are sugar and caffeine; but they are food additives
- Fat and salt increase the “salience” of food, but are not themselves addictive
- Sugar increases insulin, inhibiting leptin signaling, which indirectly inhibits the extinguishing of reward
- Sugar directly stimulates the nucleus accumbens
- Just because something has calories doesn’t make it a food; it can be a “food additive” (e.g. ethanol, trans-fats)
- Sugar’s the payload; ultraprocessed food is the vehicle
- Food addiction is a misnomer; it’s really ”food additive” addiction

Collaborators

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San Francisco General Hospital Depts. of Medicine and Radiology

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Stanton Glantz, Ph.D.
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Columbia University

Nicole Avena, Ph.D.

University of Michigan

Ashley Gearhardt, Ph.D.