The Obesogen Model as a Unifying Theory for the Global Rise in Obesity

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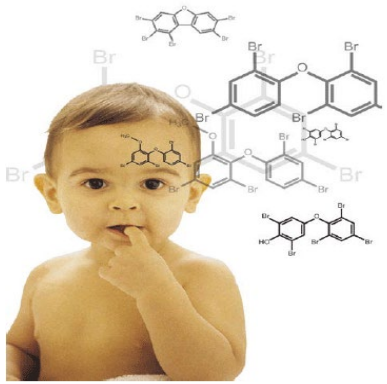
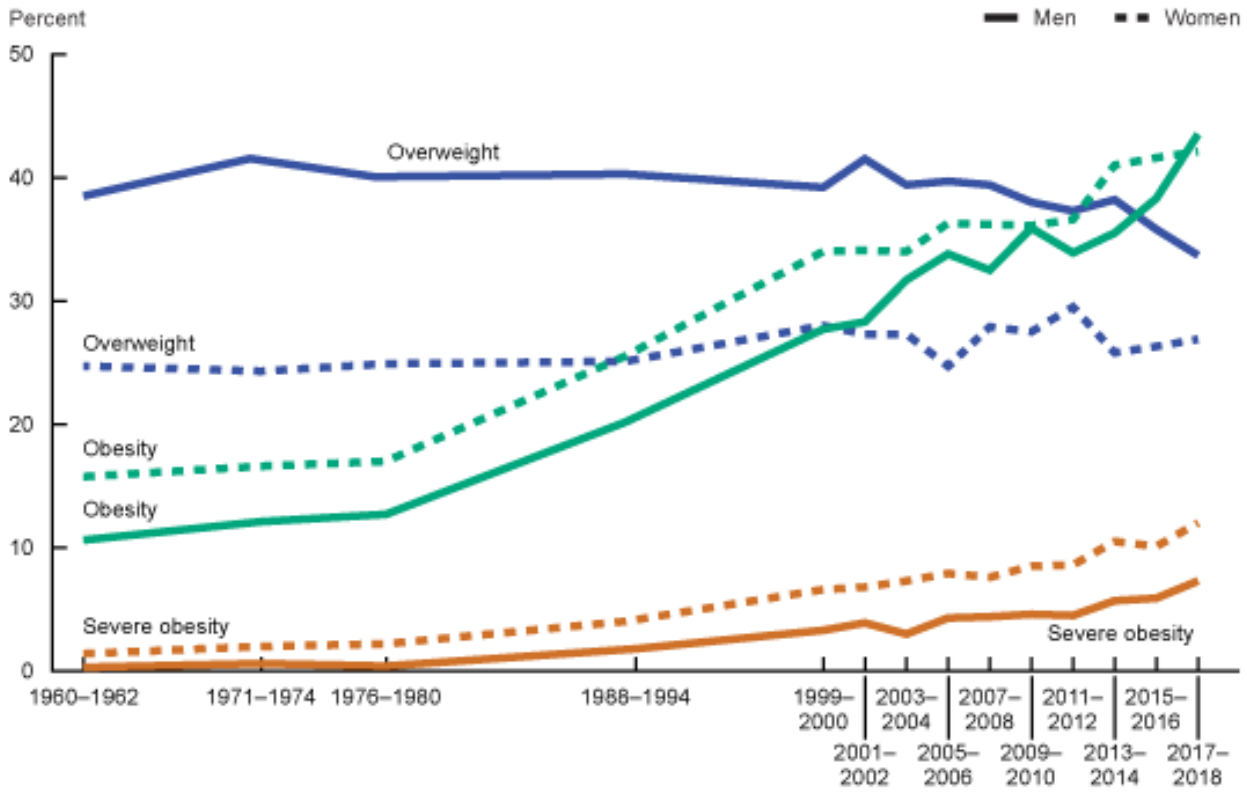


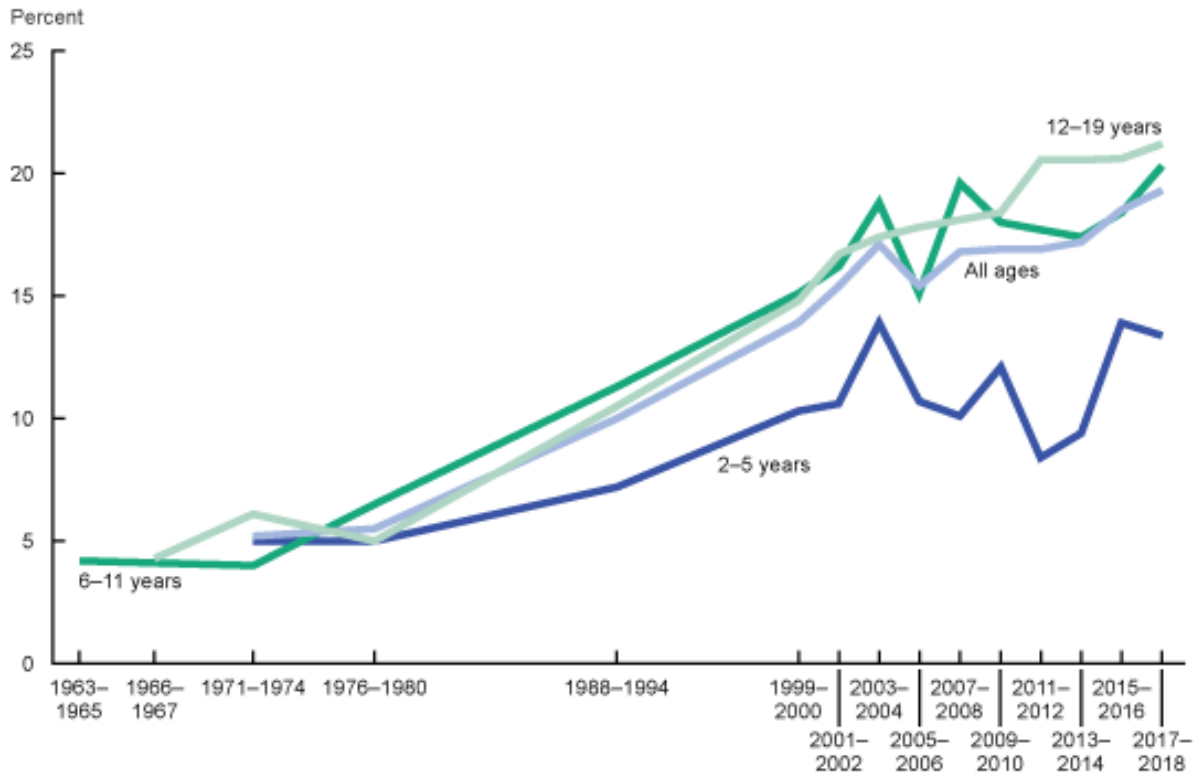
HEEDS
A Program of Environmental Health Sciences

Trends in Obesity in the U.S. 1963-2018

Adults



Children and Adolescents



Source: NHANES, 1963 - 2018

Questions for Today:

What causes obesity?

When does obesity start?

Current models of obesity.

What data support the role of environmental chemicals (obesogens)?

REDOX model of obesity.

How can all these models be integrated to explain obesity?

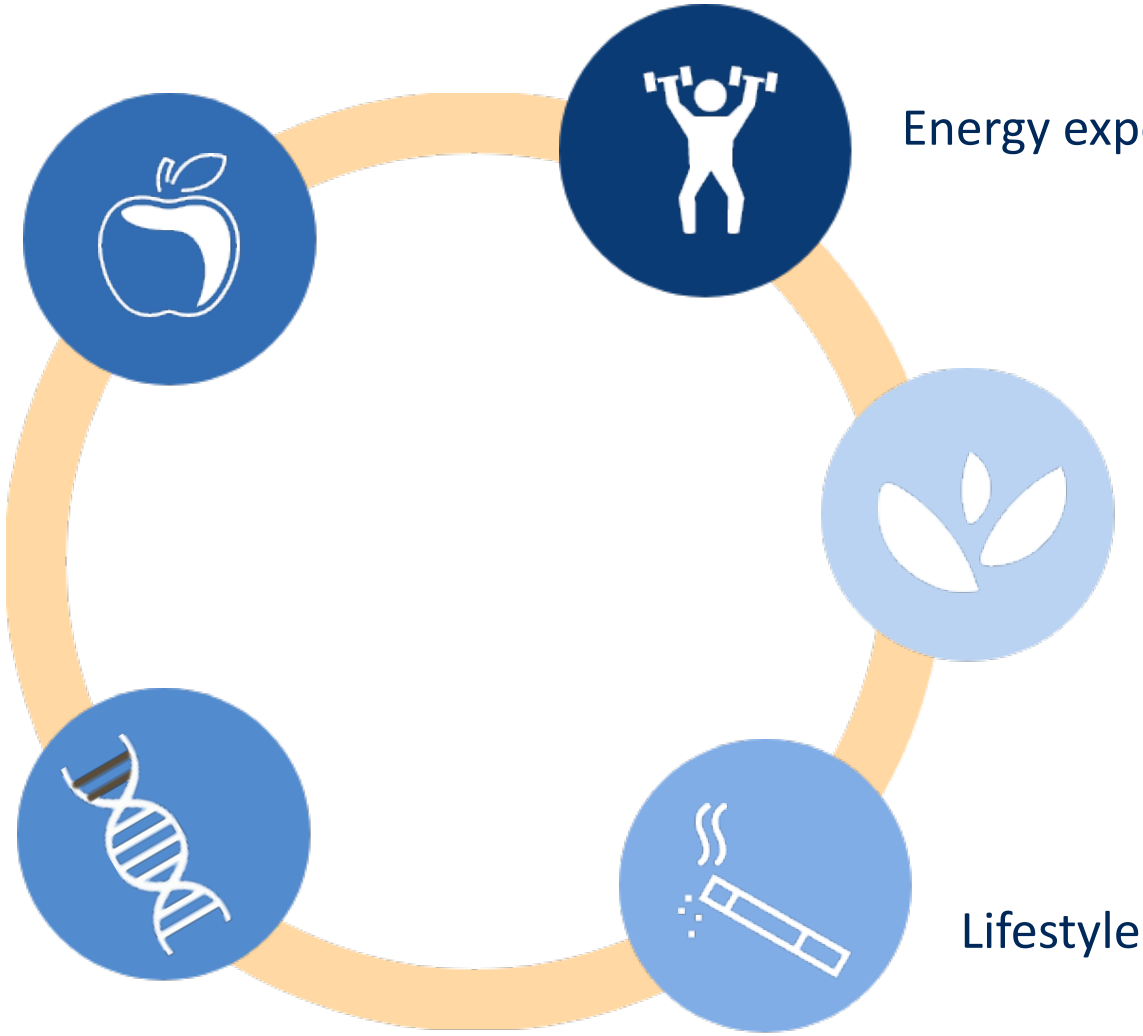
The Causes of Obesity are Not Understood

- An Endocrine Society Scientific Statement noted, "**The current lack of consensus regarding obesity pathogenesis has resulted in competing and poorly justified claims both from within and outside of the scientific community.** These inconsistencies erode public trust and confidence in the scientific process as it pertains to obesity and its treatment, which only further supports nonscientific ideologies and products." (Schwartz, Seeley et al. 2017)

- A recent perspective noted that **we don't have a clear explanation for the obesity epidemic**. "This lack of adequate attention and investment in understanding of the root causes of the obesity epidemic-one of the most rapid and widespread alterations of human health in history-may at least partly owe to the belief that the foundational causes are already known." (Mozaffarian 2022)

- There is currently a Lancet Commission on Clinical Obesity engaged in **defining the disease** with a report expected later this year.

Obesity - A Complex Problem



Drugs
Built Environment
Environmental chemicals
Stress
Viruses
Antibiotics

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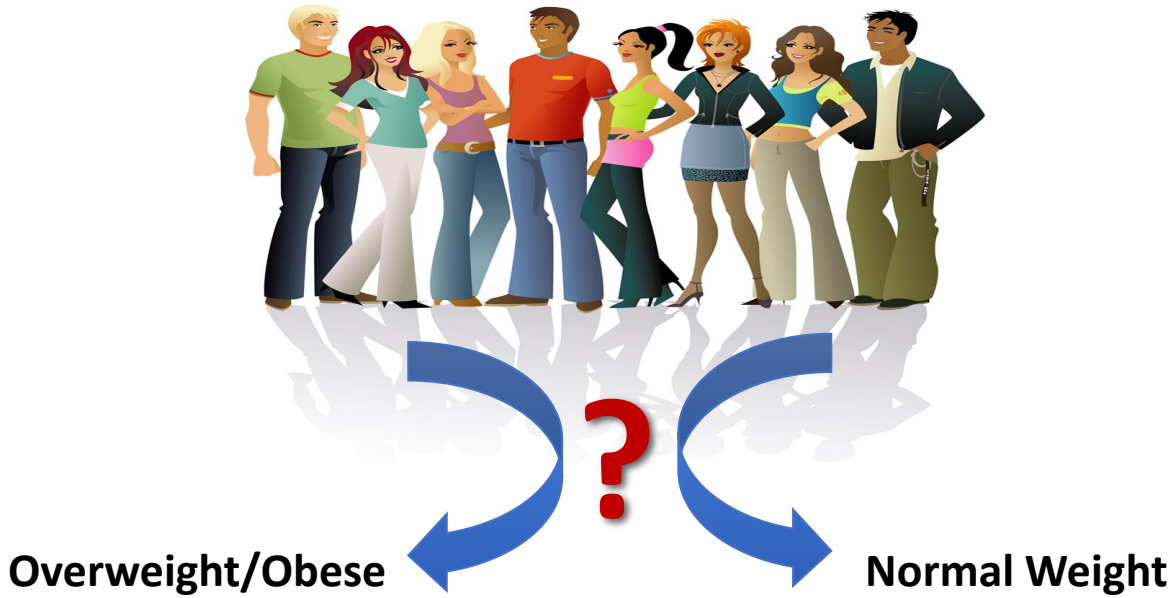

Obesity in Infants

- **Even 6-month-old babies are becoming obese**

  Kim et al, Obesity 15:1107, 2006

- **Even newborns are affected**

200 gm increase in neonatal body weight (DEXA: all fat) over the last 25 years (U.S., South Africa, Israel, Russia)

Lustig et al., Biochem Pharmacol, 199:115012 , 2022

Diet and exercise cannot explain obesity in infants.

Any hypothesis that attempts to explain the obesity epidemic must be able to explain why infants are also affected.

Critical Periods Must be Accounted for

- While nutrition, stress, and environmental chemicals can affect weight gain across the lifespan...

- Development (*in utero* and first years of life) is most sensitive to their effects.

Developmental Origin of Adult Disease: Barker Hypothesis

- 1989 David Barker: inverse relationship between birth weight and death from heart disease in England and Wales
- "Dutch Hunger Winter": food supply to the Netherlands was cut off by Nazis
- Individuals born during this time had increased insulin resistance as adults

Fetal Origin of Adult Disease (FEBAD) confirmed for:

- Coronary heart disease
- Hypertension
- Type II diabetes
- Obesity

Table 1. Hazard ratios for coronary heart disease according to body size at birth[a]

	Hazard ratio (95% CI)	No. of cases/ No. of men
Birthweight (g)		
<2500	3.63 (2.02–6.51)	24/160
–3000	1.83 (1.09–3.07)	45/599
–3500	1.99 (1.26–3.15)	144/1775
–4000	2.08 (1.31–3.31)	123/1558
>4000	1.00	21/538
P for trend	0.006	
Ponderal index (kg m^{-3})		
<25	1.66 (1.11–2.48)	104/1093
–27	1.44 (0.97–2.13)	135/1643
–29	1.18 (0.78–1.78)	84/1260
>29	1.00	31/578
P for trend	0.0006	

D. Barker, Trends in Endocrinology and Met. (2010)

The Disease Paradigm:
Developmental Origins of Health and Disease (DOHaD)

- The environment during development.. stress, nutrition, infections, drugs and environmental chemicals:
  - All cells contain the same DNA, During development hormones and growth factors turn genes on and off to form specific cells and tissues: a process called epigenetic programming
  - Environmental agents alter the epigenetic programming of cell and tissue differentiation

A bad start…lasts a lifetime!

changes").
  - "Functional" changes lead to a tissue that "looks" normal but is metabolically abnormal, permanently altering how it functions.
  - Changes persist throughout life.
  - Effects occur across the lifespan and may require a "second hit."
  - Programming changes lead to increased susceptibility to disease across the lifespan.

Disease Risk Increased by Developmental Exposures

Environmental Chemicals

Reproductive/ Endocrine

- **Breast/prostate cancer** (BPA)
- **Endometriosis** (Dioxin, PCBs)
- **Infertility** (Phthalates, Estrogens, Pesticides)
- **Diabetes/metabolic syndrome** (BPA, PCBs)
- **Early puberty** (Estrogens, BPA)
- **Obesity** (BPA, Tributyltin, Organochlorine pesticides)

Brain/Nervous System

- **Alzheimer's disease** (Lead)
- **Parkinson's disease** (Pesticides)
- **ADHD/learning disabilities, IQ** (PCBs, Lead, Ethanol, Organochlorine Pesticides)

Immune/Autoimmune

- **Susceptibility to infections** (Dioxin, Perfluorinated compounds, Arsenic)
- **Autoimmune disease** (Dioxin)

Pulmonary/Cardiovascular

- **Asthma** (Air Pollution)
- **Heart disease/hypertension** (BPA)
- **Stroke** (PCBs)

Nutrition

Diseases/Dysfunction

- **Maternal Obesity**
- **Gestational Diabetes**
- **Low birth weight/catch up growth**
- **High birth weight**
- **Sensitivity to high sugar/high-fat diet**
- **Obesity**
- **Diabetes**
- **NAFLD**
- **Cardiovascular disease**

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Redox model of obesity.
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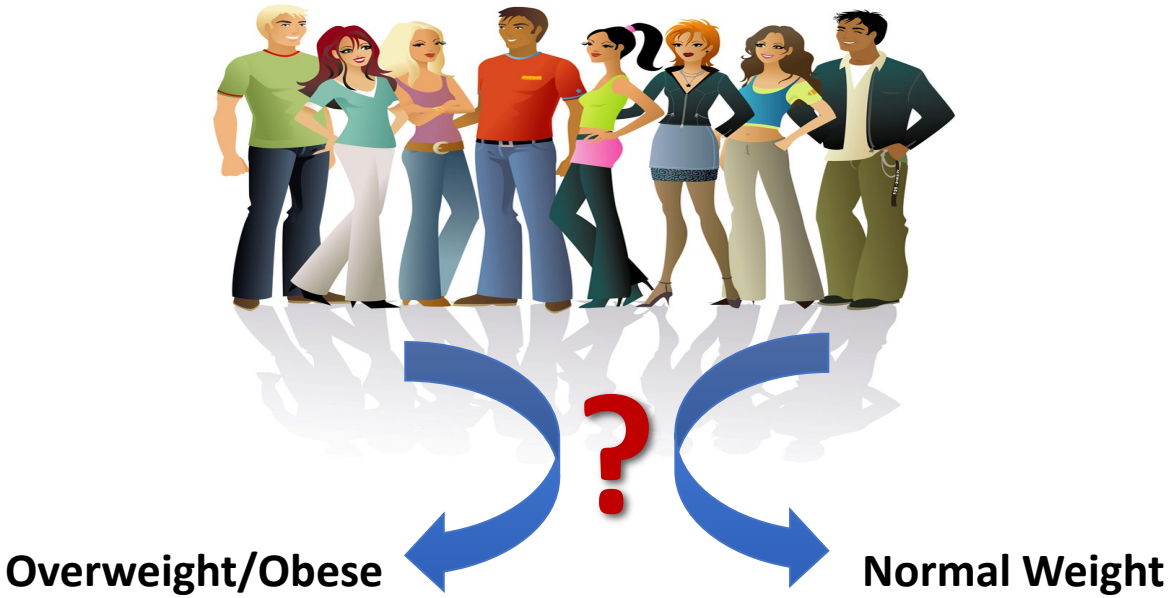


Current Models of Obesity

Energy Balance Model (EBM)

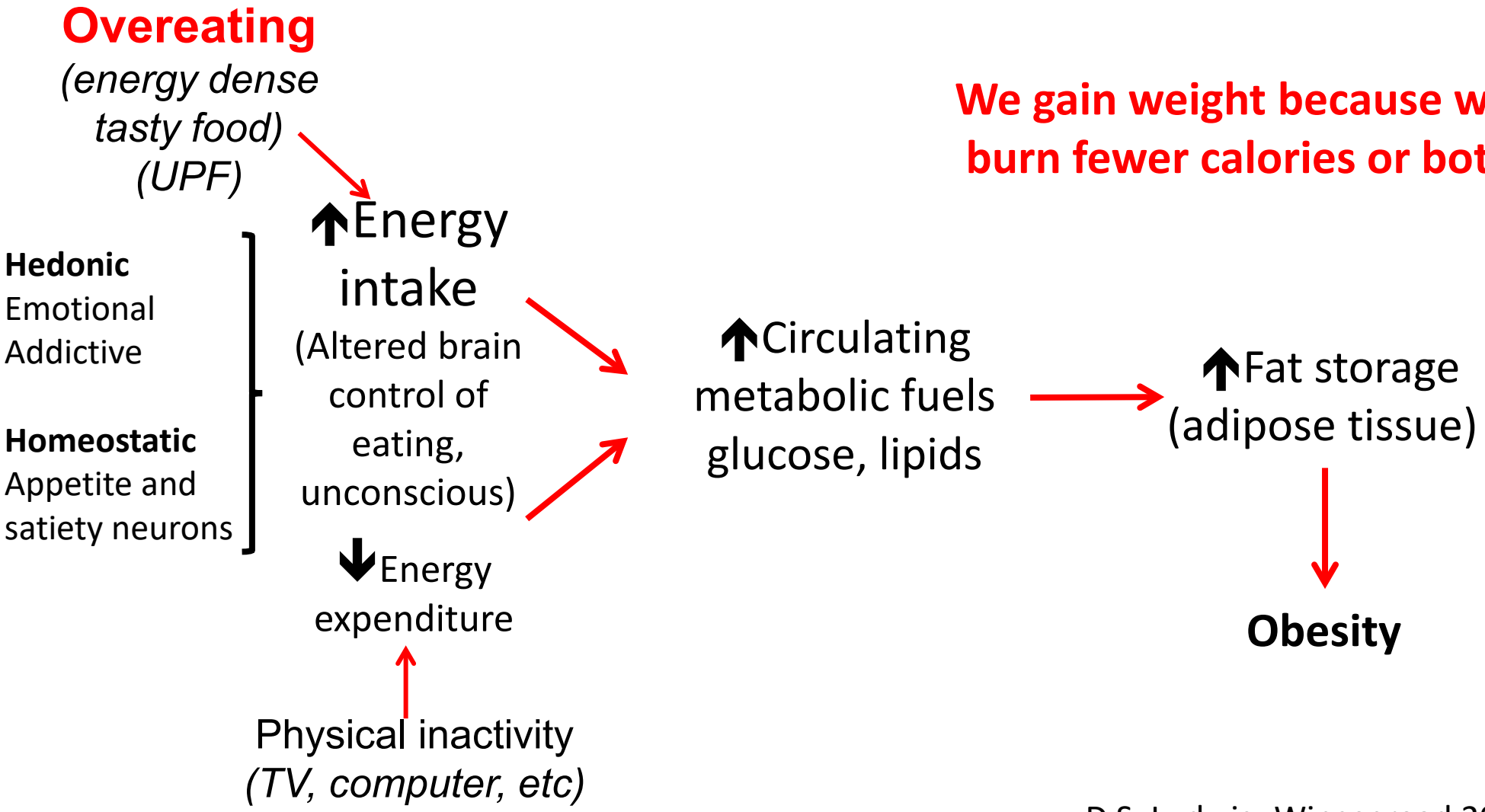
- Obesity is due to overeating; a calorie is a calorie.
- The brain is responsible for controlling weight gain.

Carbohydrate-Insulin Model (CIM)

- High glycemic index carbohydrates (sugar!) stimulate insulin secretion.
- Insulin stimulates fat storage and cell growth and division.

Energy Balance Model: EBM

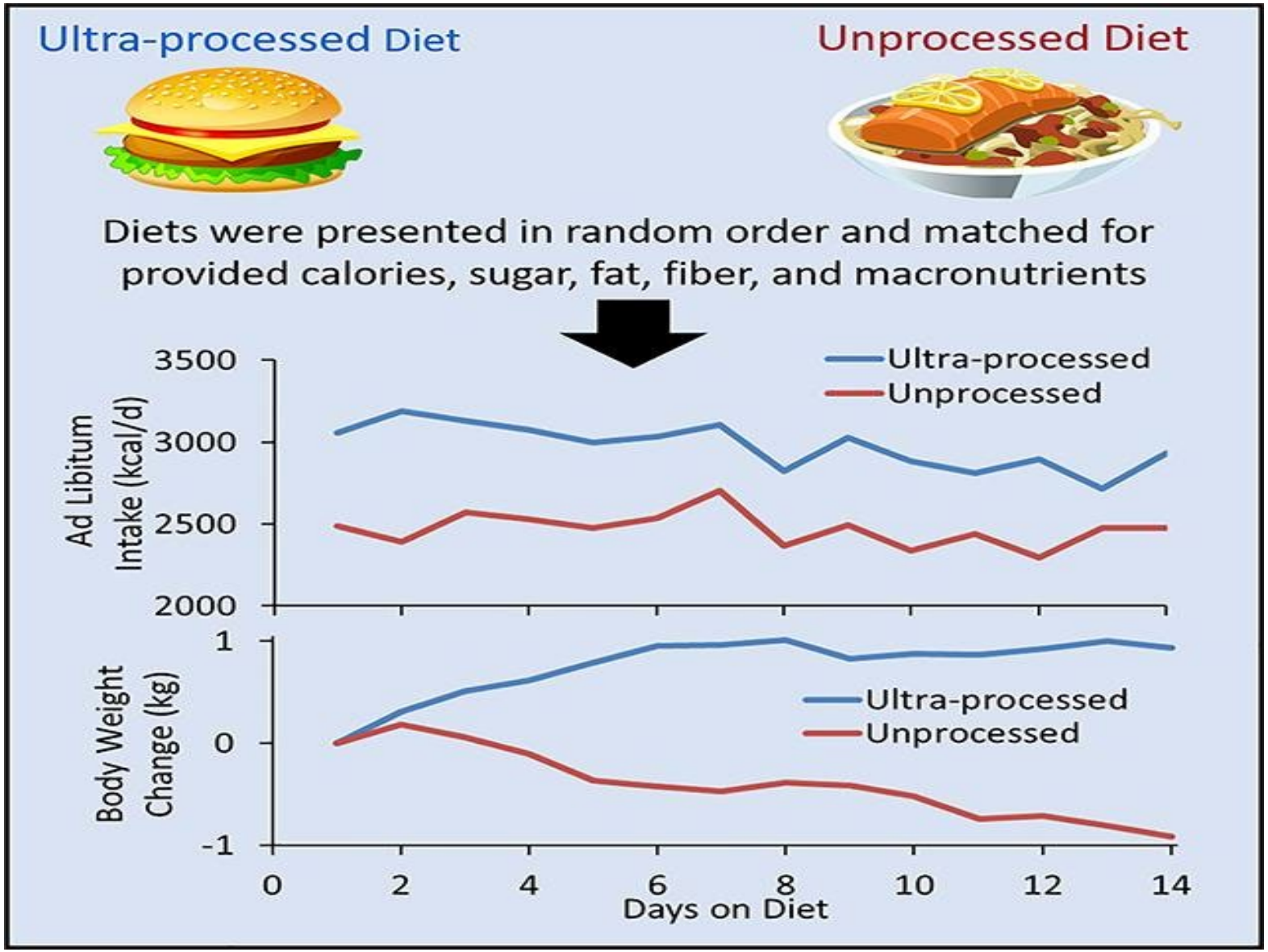
Obesity, a failure to control energy balance



We gain weight because we eat more, burn fewer calories or both.

D.S .Ludwig, Wingspread 2022

Diets High in Ultra-processed Foods Cause Weight Gain



K.D. Hall et al Cell Metab 2021

NOVA Food Groups

Unprocessed or minimally processed foods (1)

Breakfast egg sandwich	Other fresh fruit	Sweet potato	Other noodles	Other chicken dishes	Other real juice
Other eggs	Cantaloupe, in season	Other potatoes	Beef	Oysters	Iced tea
Cooked Cereal	Strawberries, in season	Green salad	Ribs	Shellfish	Water
Milk on cereal	Watermelon, in season	Tomatoes	Pork	Fried fish	Coffee
Yogurt	Broccoli	Other vegetables	Veal, lamb	Other fish	Hot tea
Bananas	Carrots	Refried beans	Liver	Rice	Bean soup
Apples or pears	Corn	Other beans	Menudo	Nuts	Milk
Oranges or tangerines	Green beans	Vegetable stew	Other beef dish	Tomato juice	Total fruits
Grapefruit	Spinach	Fried chicken			
Peaches, raw	Greens	Spaghetti with meat sauce	Roast chicken	Real orange juice	Total vegetables

Processed culinary ingredients (2)

Butter	Fat/oil in cooking

Processed foods (3)

Cheese	Vegetable soup	Other soup	Tofu	Tacos
Tuna	Tortillas (flour)	Corn bread	Peanut butter	Chips
Mustard	Beer	Wine		

Ultra-processed foods (4)

Breakfast sausage	Bacon	Pancakes	Cold cereal	Canned fruit	French Fries
Cole slaw	Salad dressing	Pizza	Mac N Cheese	Meat substitutes	Hamburger (cheeseburger)
Hot dogs	Lunch Meats	Meat loaf	Pigs feet, variety meats	Biscuits	Burger rolls
Bagels, English muffins	White bread	Margarine	Power bars	Breakfast bars	Crackers
Jelly	Mayonnaise	Ketchup (catsup)	Donuts	Cake	Cookies
Ice cream	Chocolate syrup	Pumpkin Pie	Other pie	Chocolate candy	Other candy
Slimfast	Hi C	Drinks with some juice	Kool aid	Sodas	Liquor

Support and Challenges to the EBM

Support:

- It is possible to lose weight on various diets…calories count.
- Consumption of ultra-processed food causes weight gain via overeating due to altered brain control of eating.

Challenges:

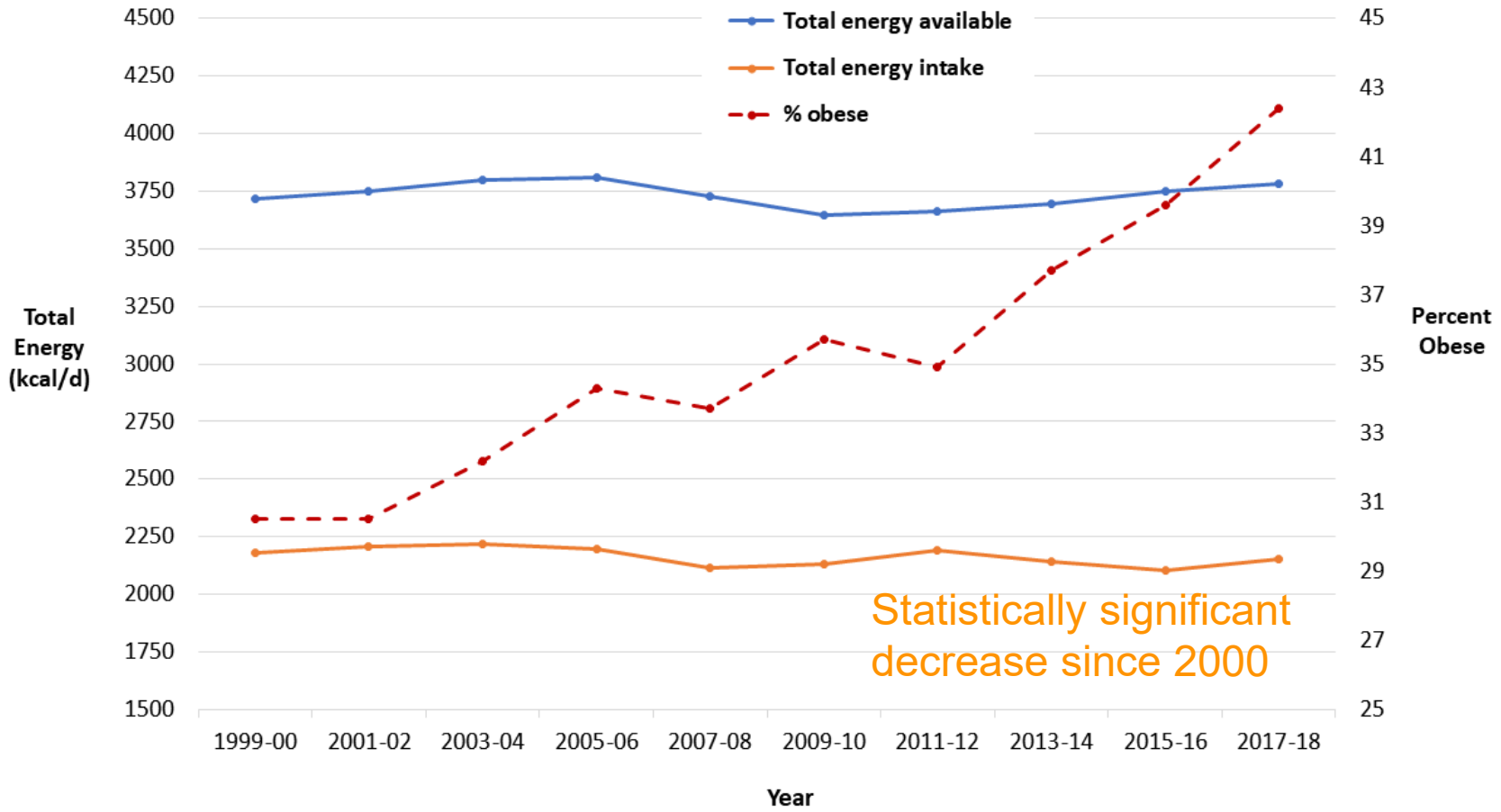
- Why have animals also gained weight?
- What about the increased weight of babies?
- It does not address the role of developmental programming affecting weight gain.
- No mechanism to explain the altered control of food intake.

D.S. Ludwig, Wingspread 2022

Leibel. NEJM 1995;332:621-8

Challenges to the EBM

Secular trends challenge the primary role of "overeating"



D.S.Ludwig, Wingspread 2022

Mozaffarian. AJCN 2022 Apr 23:nqac075

Insulin & Body Weight

A dominant anabolic hormone

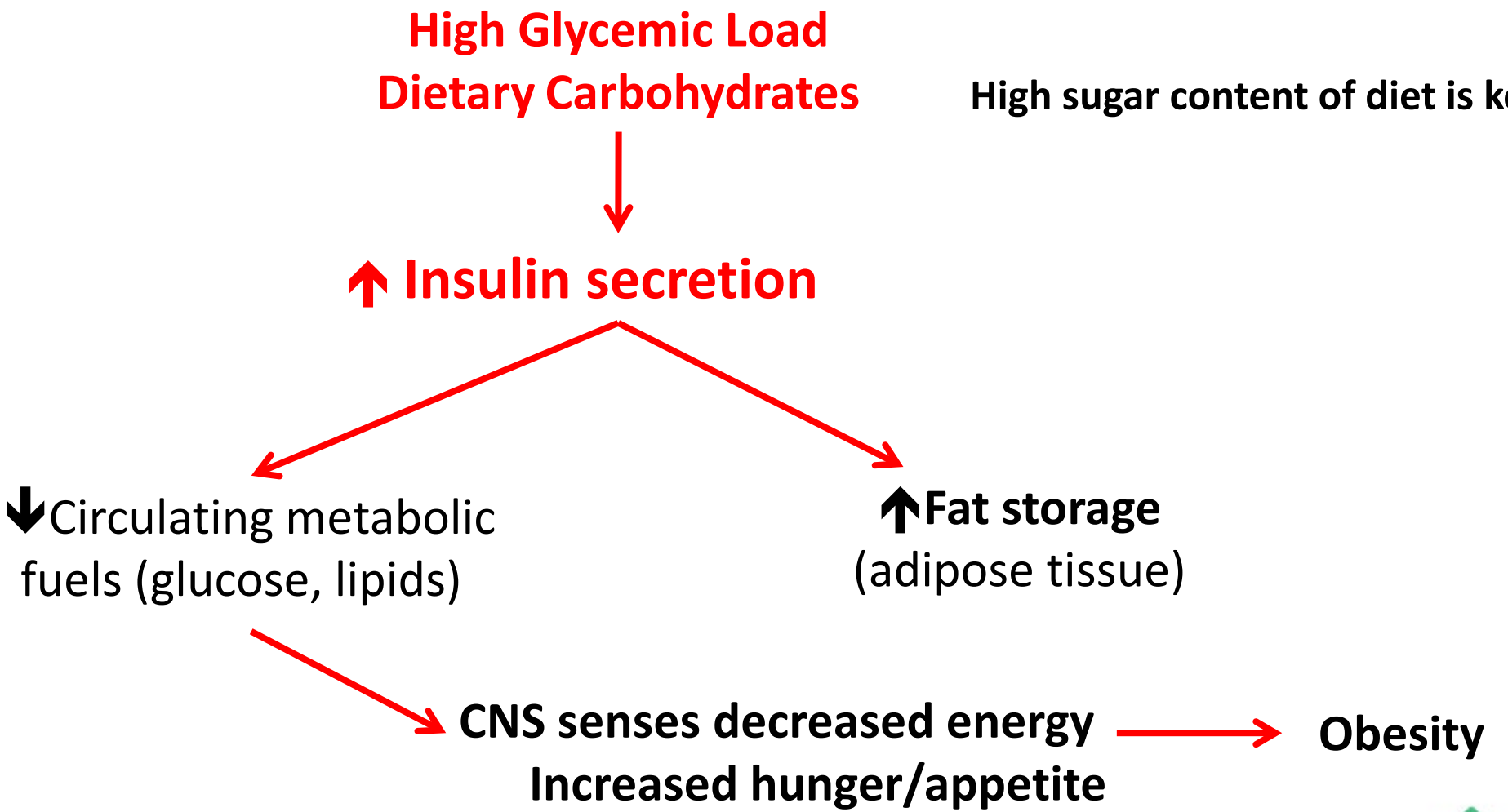
- **Regulates storage of all metabolic fuels**
  - Stimulates glucose uptake into cells.
  - Stimulates fat synthesis and deposition.
  - Inhibits fat release and oxidation.

- **Excess insulin causes weight gain**
  - Increased insulin makes you hungry.
  - Excessive insulin treatment in diabetes.
  - Insulin-secreting tumors.
  - Human genetic variants affecting insulin secretion.
- **Decreased action causes weight loss**
  - Under-treatment of type 1 diabetes.
  - Adipocyte Insulin receptor knock-down models but not muscle receptor knock-down.

UKPDS Lancet 1998, 352:837; Carlson. Diabetes 1993, 42:1700; Le Stunff, Nat Gen 2000, 26:444-6; Lustig, IJO 2006, 30:331-41; Templeman. J Endo 2017, 232:R173-83

Carbohydrate-Insulin Model: CIM

A metabolic disorder of fat storage



D.S.Ludwig, Wingspread 2022

Support and Challenges to the Carb-Insulin Model

Support:

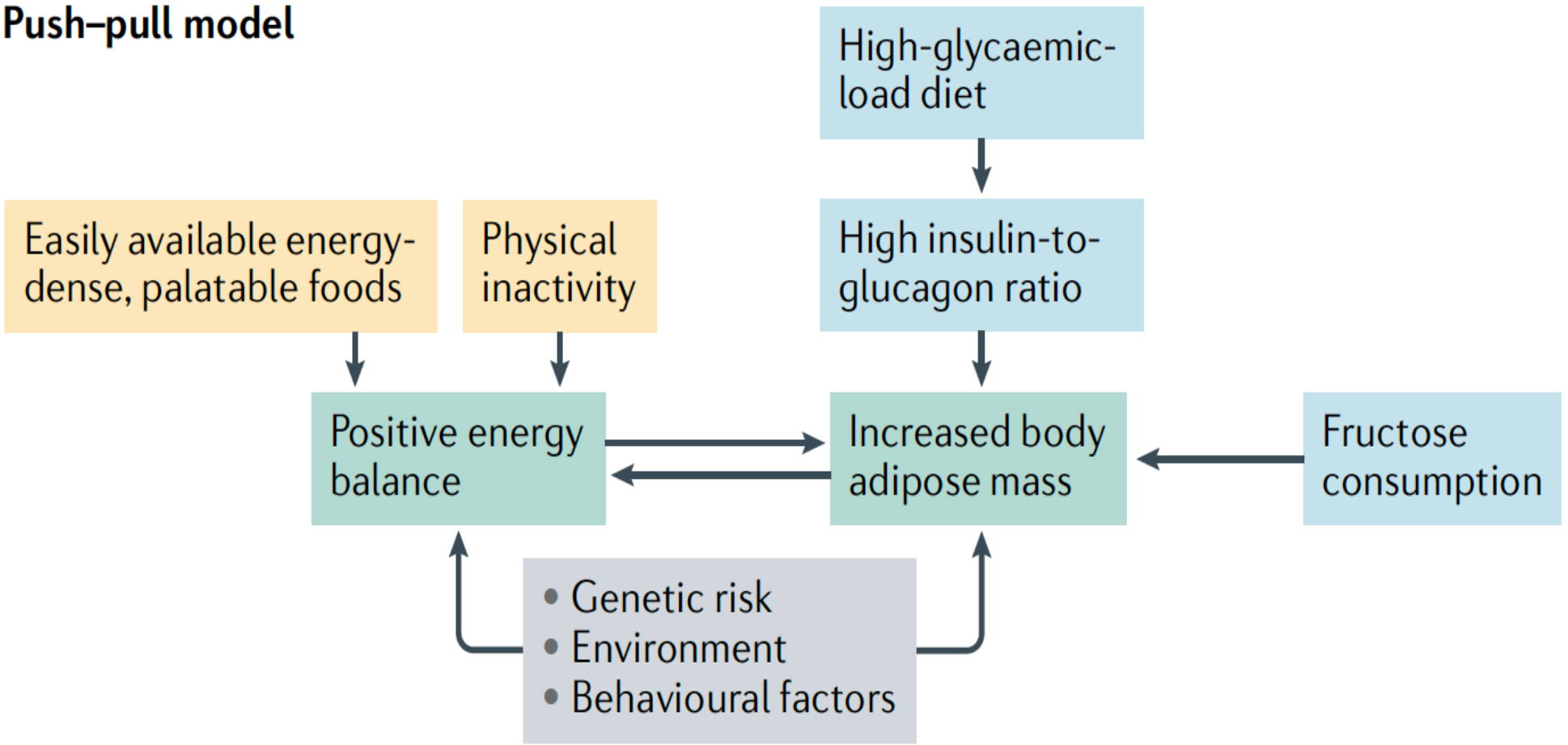
- Ketogenic and low-carbohydrate diets promote weight loss more effectively than low-fat diets.
- Inhibition of insulin secretion increased weight loss in dieting obese patients.
- The current Western diet is insulinogenic.
- May contribute to obesity in a subset of individuals.

Challenges:

- There is no focus on the role of developmental programming.
- High glycemic index processed foods contain many new ingredients that may stimulate insulin.

D.S. Ludwig, Wingspread 2022 Alemzadeh 1998

An Integrated Model of Obesity (EBM and CIM)



Ludwig, Sørensen. Nat Rev Endocrinol 2022, 18:261-262

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Obesogens

- Obesogens are environmental agents that alter energy metabolism.
- They disrupt **signaling pathways** (e.g., hormone receptors and transcription factors) in various cell types and tissues that regulate energy intake and expenditure, nutrient handling, and adiposity.
- Obesogens can be:
  - natural (e.g., metals, viruses),
  - anthropogenic (prescription drugs),
  - environmental (insecticides, plastics, household chemicals, particulate matter),
  - food components (fructose, *trans*-fats, preservatives, emulsifiers).
- **The majority of obesogens are endocrine disruptors**.

Endocrine Disruptors

An **endocrine disruptor** is an exogenous chemical, or a mixture of chemicals, that **interferes with any aspect of hormone action** (Zoeller et al. Endocrinology, 2012).

A chemical designed for a specific purpose but with a side effect of mimicking or antagonizing hormone action.

>300,000 chemicals in commerce

Some % are toxic, via

Alterations of DNA

mutagens

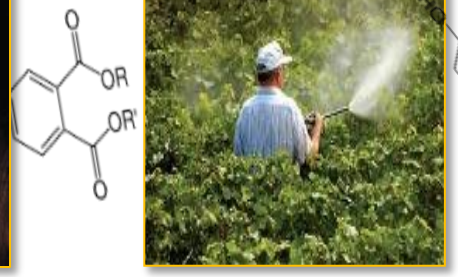
or general toxicity

Some % are toxic because they interfere with some aspect of the endocrine system

Endocrine Disruptors

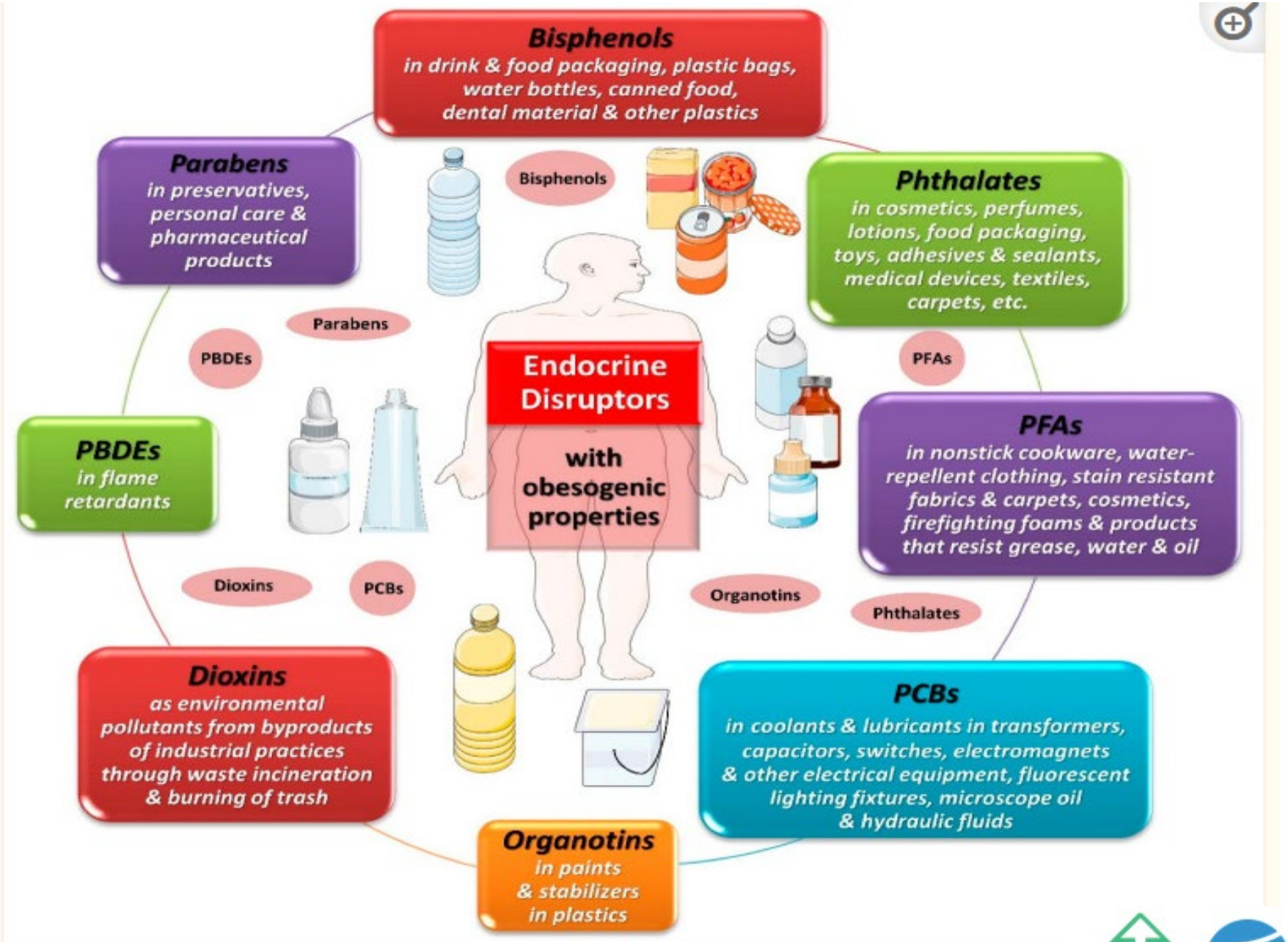
Some are toxic because they cause an increase in fat

Obesogens



Obesogenic Endocrine-disrupting chemicals

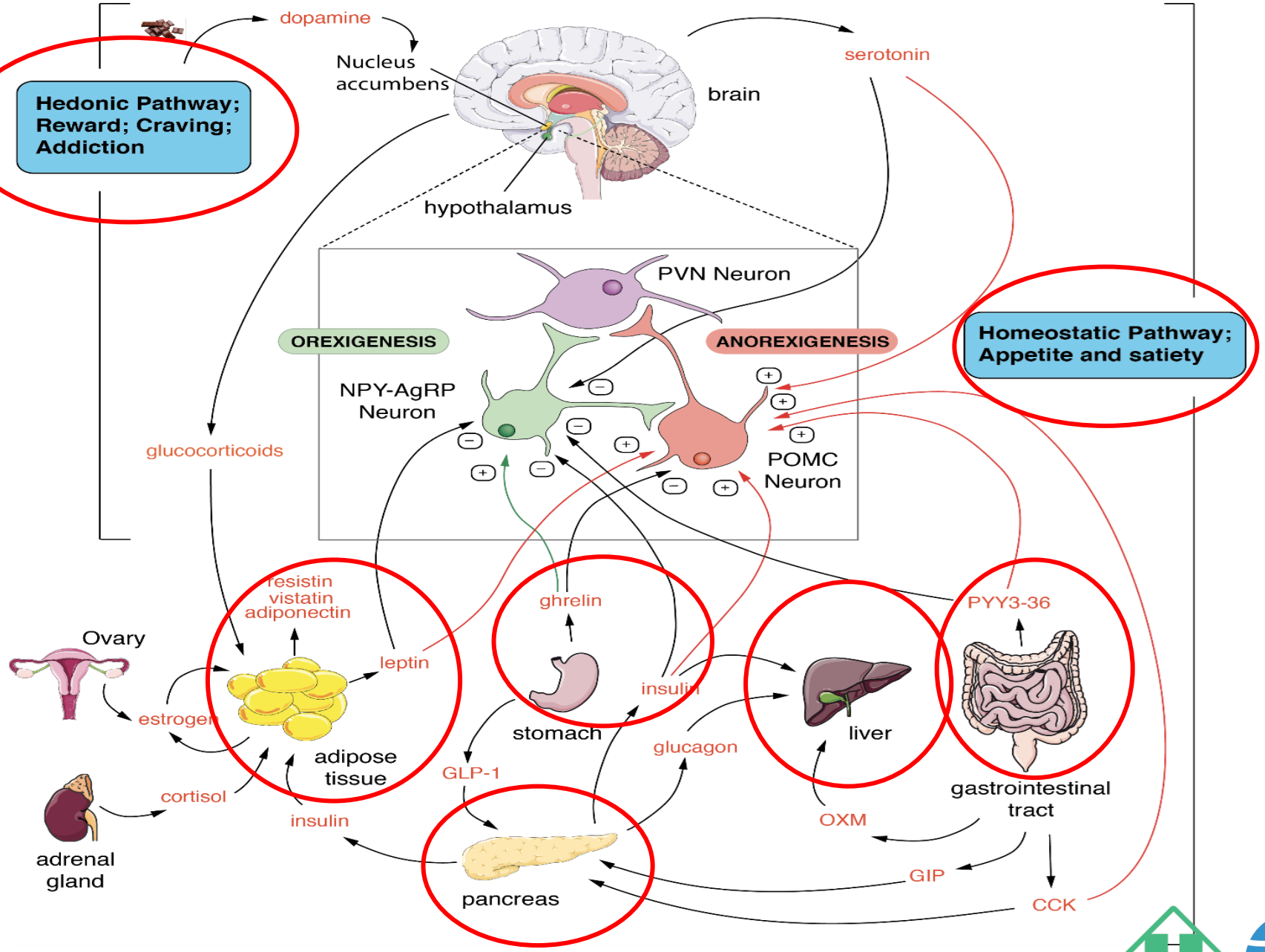
We are born pre-polluted!



M. Dalamaga et al Int J Mol Sci 2023

Endocrine Control of Metabolism

All hormones and hormonal pathways are potential targets for obesogens.



Obesogens

Maternal Smoking (nicotine)

Air Pollution (PAHs, particulate matter (PM))

DDT

Perfluorinated compounds (PFAS)

Bisphenols (A, S, F, AF…)

Phthalates (DEHP, DBP, DiBP)

Tributyltin

Flame retardants (PBDEs, OPFRs)

Dioxin and polychlorinated biphenyls (PCBs)

Fructose

Non-nutritive sweeteners

- Aspartame
- Sucralose
- Saccharin

Agricultural chemicals

- Chlorpyrifos
- Diazinon
- Neonicotinoids
- Permethrin
- Tolyfluanid

Food preservatives/additives/emulsifiers

- Methyl and butylparaben
- Tween 80/carboxymethylcellulose
- 3-tert-butyl-4-hyroxyanisole (3-BHA)
- Dioctyl sodium sulfosuccinate (DOSS)
- Monosodium Glutamate (MSG)

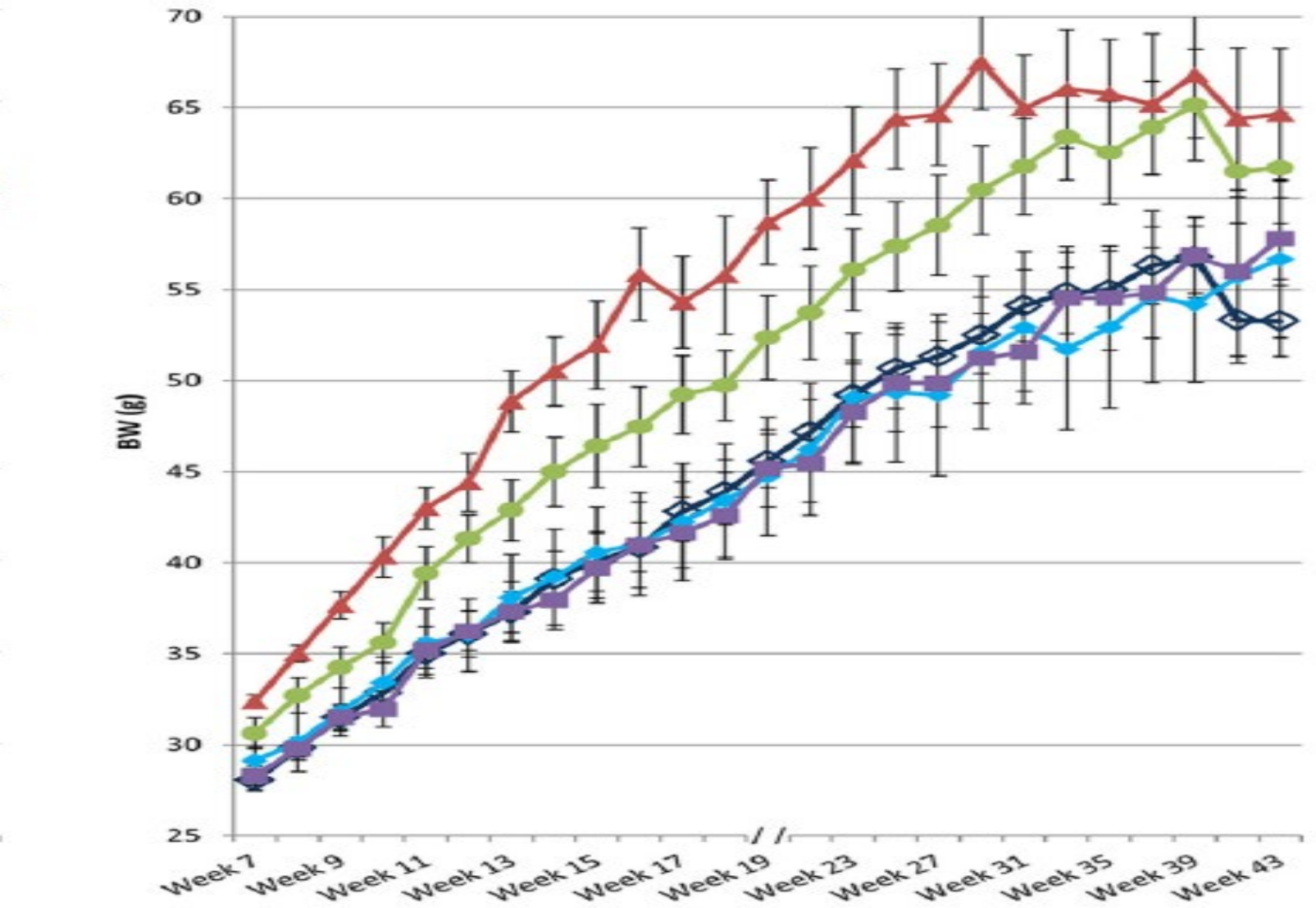
Cadmium

Arsenic

Dibutyltin

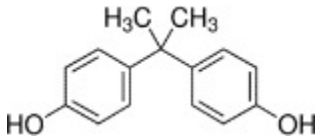
Microplastics

Animal Studies: BPA is an Obesogen



Perinatal and peripubertal exposure

Developmental exposure to BPA in mice increases body weight in female offspring.



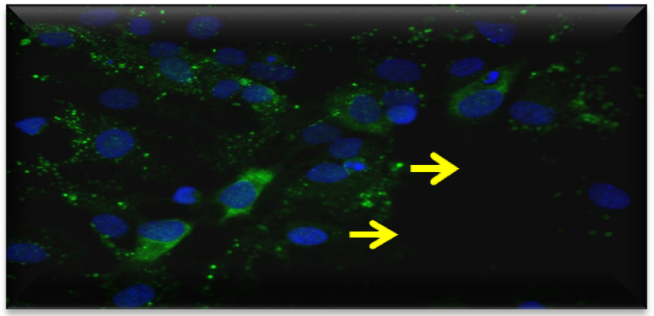
Rubin et al. Reprod Toxicol 2017

Bisphenol A is an Obesogen

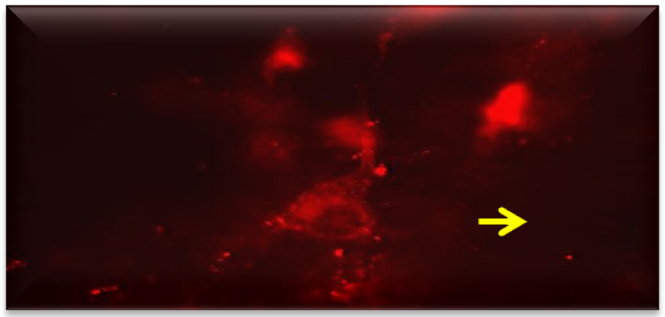
Developmental Exposure
SD Rat model
Increased weight
Increased food intake

BPS and BPF are obesogens

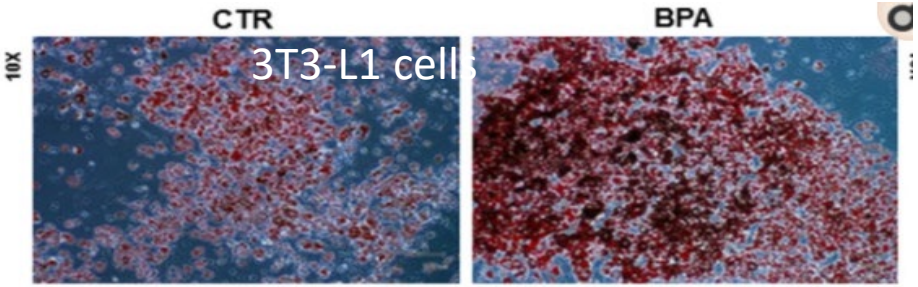
Lipid accumulation
in HepG2 cell



Appetite (NPY) neurons



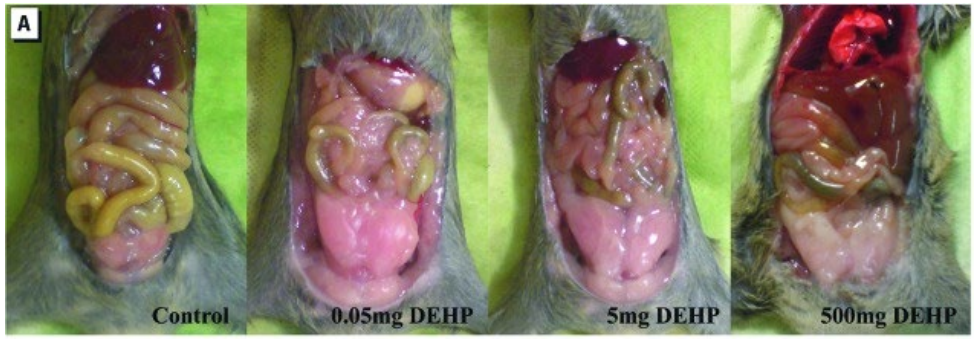
Satiety (POMC) neurons

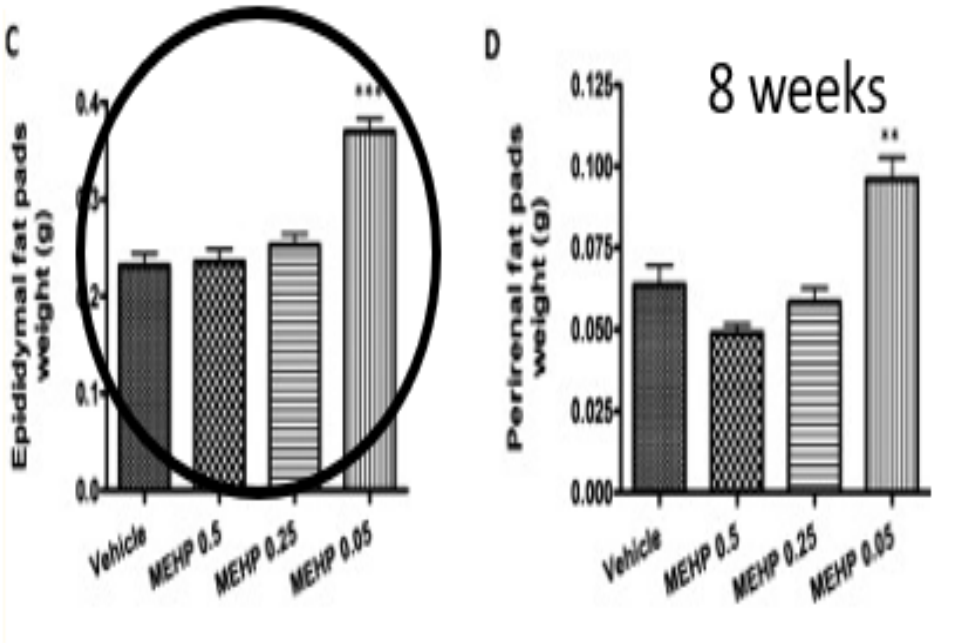


Strong *in vitro* and animal data
Inconsistent human data

Ross and Desai, 2018

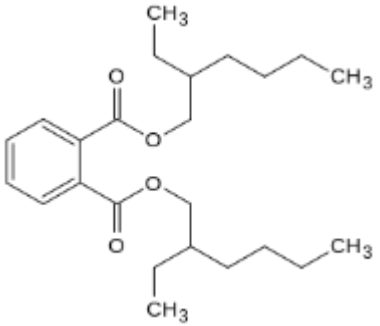
Animal Studies: Phthalates Are Obesogens





Developmental exposure C57/B6 mice

(A) **Adult mice** exposed to environmentally-relevant levels of di(2-ethylhexyl) phthalate (DEHP) for 8 weeks developed:



- higher body weight
- increased food intake
- excess visceral fat
- larger fat cells

(C/D) **Developmental exposur**e to DEHP and assess weight at 8 weeks of age:

- higher body weight
- excess visceral fat

Schmidt et al., EHP, 2012 Ho et al Biosci Rep 2012

Human Studies: EDCs as Obesogens

- A 2020 **meta-analysis** identified 18 studies showing an overall positive association between exposure to **Phthalates** and measures of excess weight or adiposity in adults and children *(Ribeiro, 2020).*
- **Meta-analysis**s showed an overall positive association between **BPA** exposure and obesity and abdominal obesity in children and adults (Ribeiro, 2020).
- A **review of bisphenols** and obesity finds "**BPS** median urinary concentrations … were associated with the development of obesity." (Alharbi et al. Int J Environ Res Public Health. 2022).
- In a **pooled analysis**, **air pollutants,** including PM2.5, PM10, and NO2 were positively associated with obesity in children (Parasin, 2021).
- A 2021 **systematic review and meta-analysis** identified 33 studies assessing prenatal OC exposure and obesity in early life**. DDE (DDT) and HCB (hexaclorobenzene)** were positively associated with BMI z-score in childhood (2-9 years) (Cano-Sancho 2017).

The Western Diet (Ultra-processed) is Obesogenic

Western Diet

- High fat
- High sugar
- High salt
- Low fiber
- Inadequate fresh fruit and vegetables
- Micronutrient deficient
- **High in ultra-processed food**
- **Addictive**

Contains Obesogens

- Bisphenol A
- Phthalates
- PFAS
- Fructose
- Non-nutritive sweeteners
- Methyl and butyl parabens
- Tween80/carboxy cellulose
- 3-tert-butyl-4-hydroxyanisole (3-BHA)
- Monosodium glutamate (MSG)
- Red coloring 40*
- Yellow coloring 5 and 6*
- Pesticides (PFAS)

* Potential obesogens

Contents lists available at ScienceDirect

Clinical Nutrition

journal homepage: http://www.elsevier.com/locate/clnu

Original article

Consumption of ultra-processed foods associated with weight gain and obesity in adults: A multi-national cohort study

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Ultra-processed Foods and Diseases

Ultra-processed food is associated with an increased risk of Crohn's disease: a cross-sectional and prospective analysis of 187,154 participants in the UK Biobank

- Association of ultra-processed food intake with risk of inflammatory bowel disease: prospective cohort study

Asthma and Ultra-Processed Food

Associations of ultra-processed food consumption with cardiovascular disease and all cause mortality: UKbiobank

Ultra-processed food and risk of type 2 diabetes: a systematic review and meta-analysis of longitudinal studies

Ultra-processed food targets bone quality via endochondral ossification

Consumption of ultra-processed foods and cancer risk: results from NutriNet-Santé prospective cohort

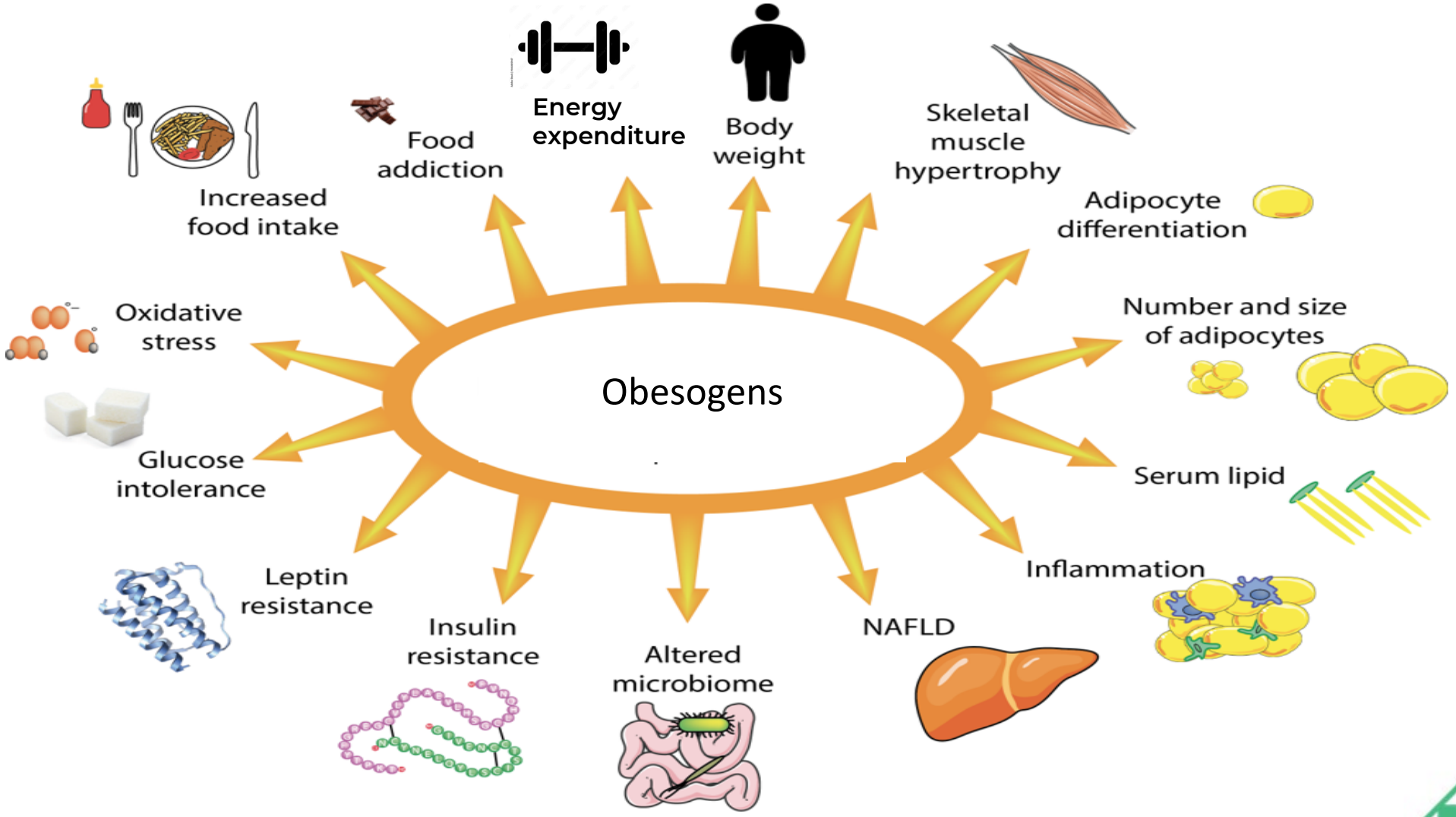
Ultra-Processed Food Availability and Noncommunicable Diseases: A Systematic Review

Association of Ultra-processed Food Consumption with risk of Dementia, a prospective cohort study

Ultra-Processed Food Consumption and Mental Health: A Systematic Review and Meta-Analysis of Observational Stu

Ultra-processed food consumption and excess weight among US adults

Obesogens Can Alter Many Metabolic Endpoints



Obesogens Affect Multiple Endpoints

Altered microbiome

- BPA, BPS
- Phthalates
- PFAS
- PBDE
- TCDD
- PCBs
- PAH (Air pollution)
- Methyl paraben
- Triclosan

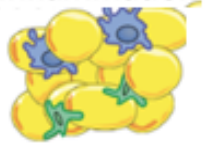
Increased food intake

- BPA
- DEHP
- TBT
- OPFR (flame retardant)

Larger effect with HFD

- BPA
- Butyl benzyl phthalate
- DEHP
- TBT
- Nicotine
- Chlorpyrifos
- Imidacloprid
- DDT
- Permethrin
- Atrazine

Inflammation



BPA
TBT
DEHP
PBDEs
Triclosan
CMC
$PM_{2.5}$
Western diet

NAFLD

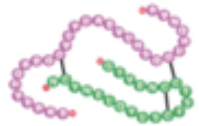
- BPA
- DEHP
- TBT
- DDT
- PCBs
- BaP
- Triclosan

Dysfunctional Adipocyte

- BPA
- DEHP
- TBT

TCP

Insulin resistance



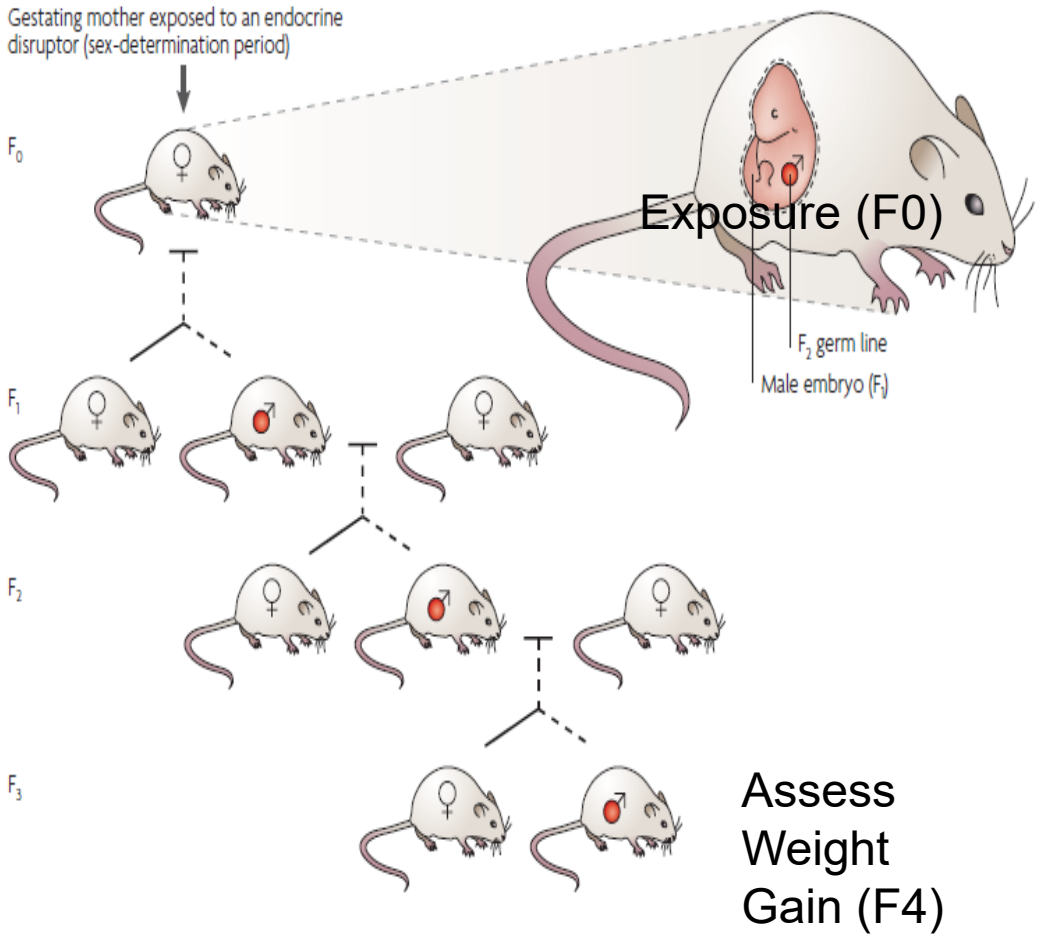
- BPA
- DEHP
- $PM_{2.5}$
- PFOS
- Atrazine
- Cadmium
- Permethrin
- Western diet
  - OPFRs
  - Chlorpyrifos

Increase ROS

- BPA
- TBT
- BBP
- DEHP
- PFAS
- Atrazine
- Cadmium
- Chlorpyrifos
- {CBs
- PBDE
- Air pollution

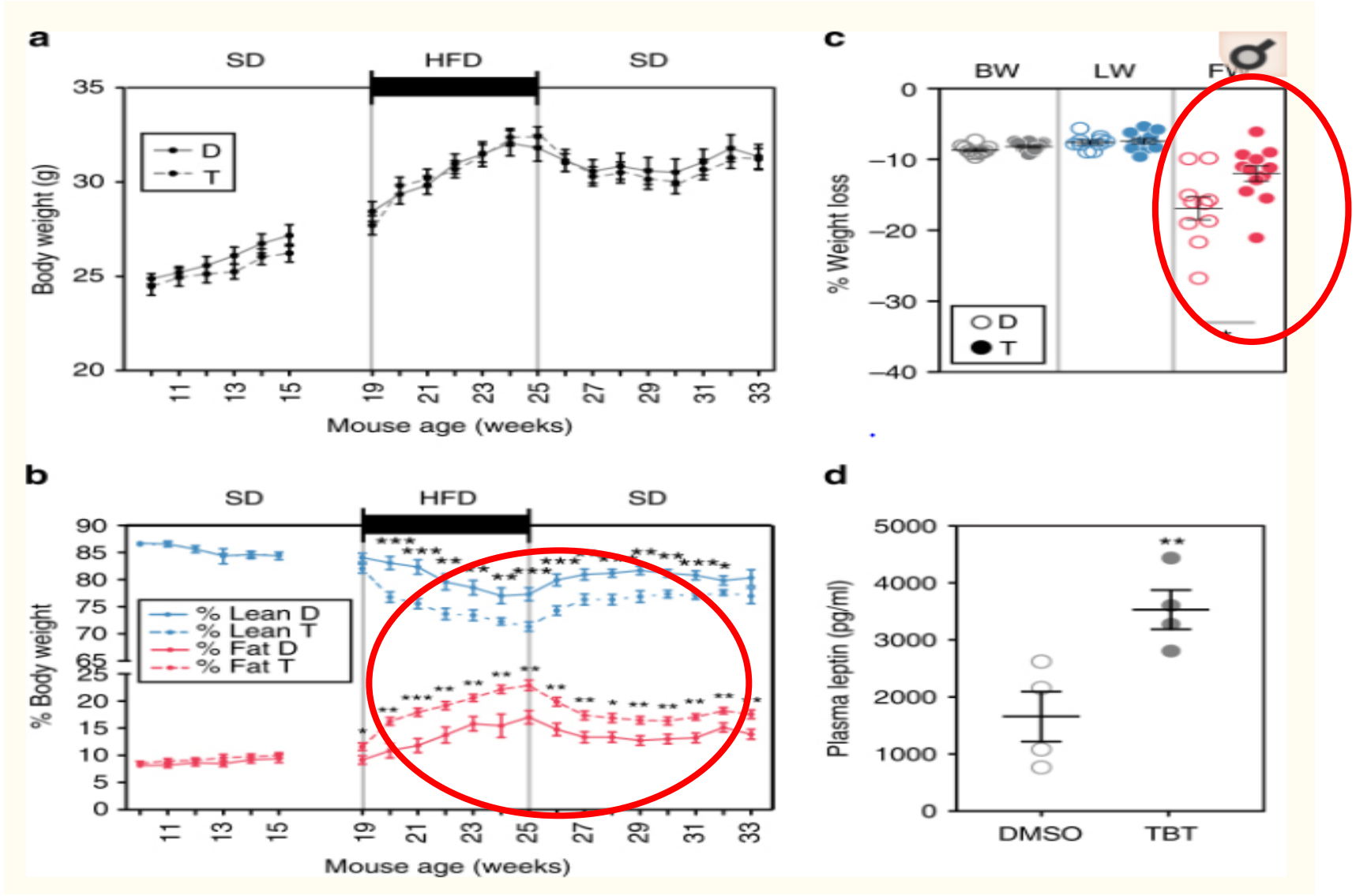
Transgenerational Epigenetic Inheritance of Obesity

- **Positive results**
  - **Tributyl tin** (M and F)
  - **DDT** (M and F)
  - Hydrocarbon mixture (jet fuel) (M and F)
  - **BPA DEHP, DBP** (M and F)
  - **BPA**
  - **Nutrition**
- **Negative results**
  - Permethrin/DEET mixture
  - Vinclozolin
  - Dioxin



Prenatal Exposure to Tributyl Tin: Higher Fat Diet Causes Obesity in F4 Males

Chamorro-Garcia et al., Nature Comm 2017

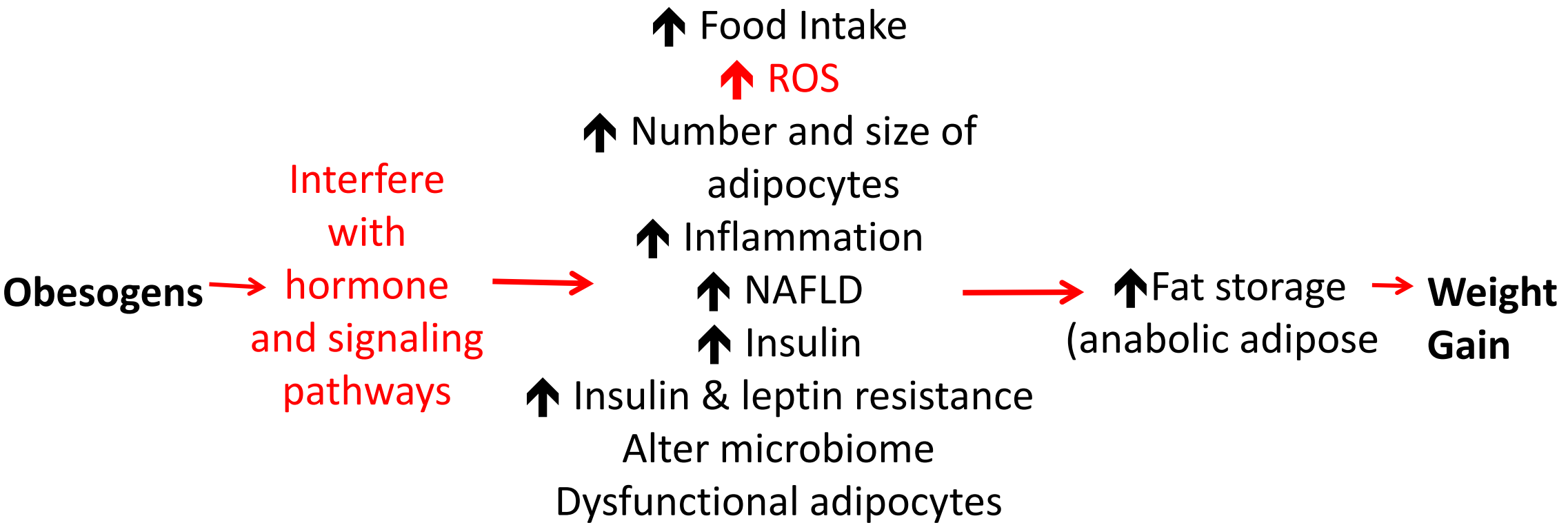


Resistant to fat loss!

Gain weight more easily and harder to lose weight!

Obesogen Model: OBS

A multiorgan disruption of metabolism



Can Obesogens Explain Why Diets Fail?

- POUNDS Lost study: two-year randomized clinical trial compared four diets on body weight.
- Healthy diet
- High protein/low glycemic index
- High protein/high glycemic index
- Low protein/low glycemic index
- Low protein/high glycemic index
- Most weight loss occurred in the first six months, followed by weight regain through 24 months, with no differences between diet regimens.
- **Perfluorinated chemicals (PFAS) were associated with:**
  - **Greater weight regain**
  - **Greater decline in basal metabolic rate during weight loss**
  - **Less increase in basal metabolic rate during the weight regain**

Liu et al Plos Medicine 2018

Grandjean et al Obesity 2023

Support and Challenges to the OBS Model

Support:

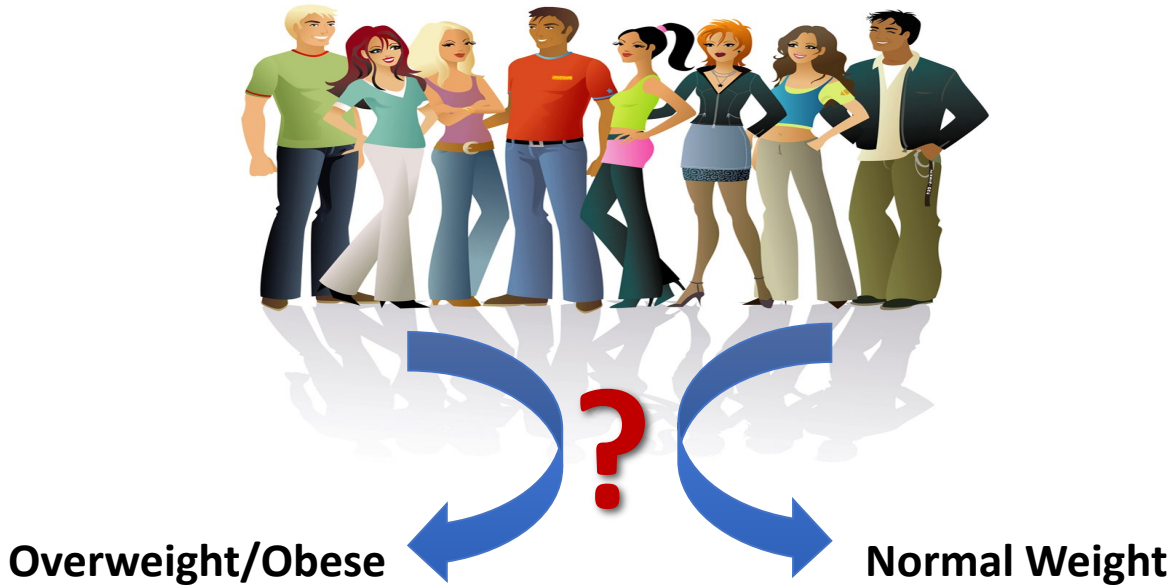
- In vitro, animal and human studies.
- Developmental and adult exposures cause weight gain.
- Affects endpoints considered part of EBM and CIM.
- Human exposure to a variety of obesogens across the lifespan.

Challenges:

- Lack of developmental biomarkers that indicate susceptibility to weight gain later in life.
- Lack of data showing the effects of obesogens in clinical-type studies.
- Lack of data showing a decrease in exposure results in lower weight gain.

Questions for Today:

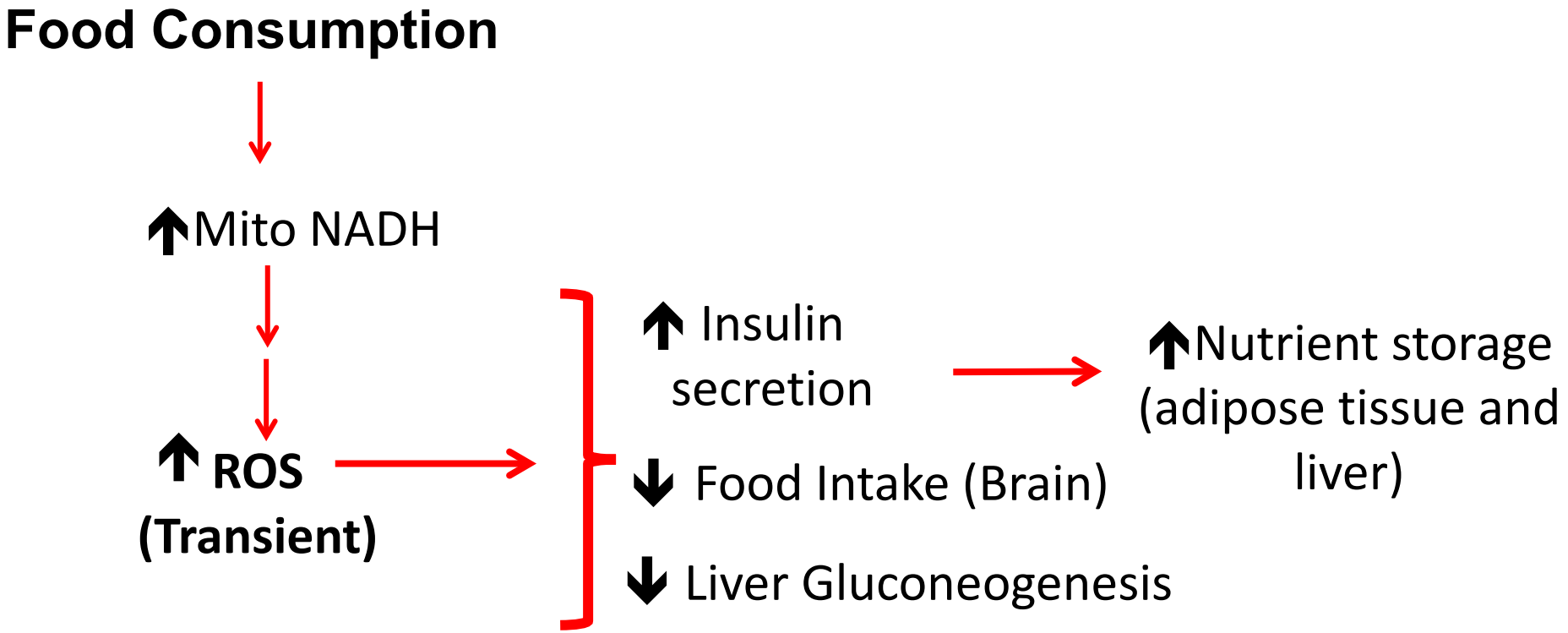
What causes obesity?
When does obesity start?
Current models of obesity.
What data support the role of environmental chemicals (obesogens)?
Redox model of obesity.
How can all these models be integrated to explain obesity?



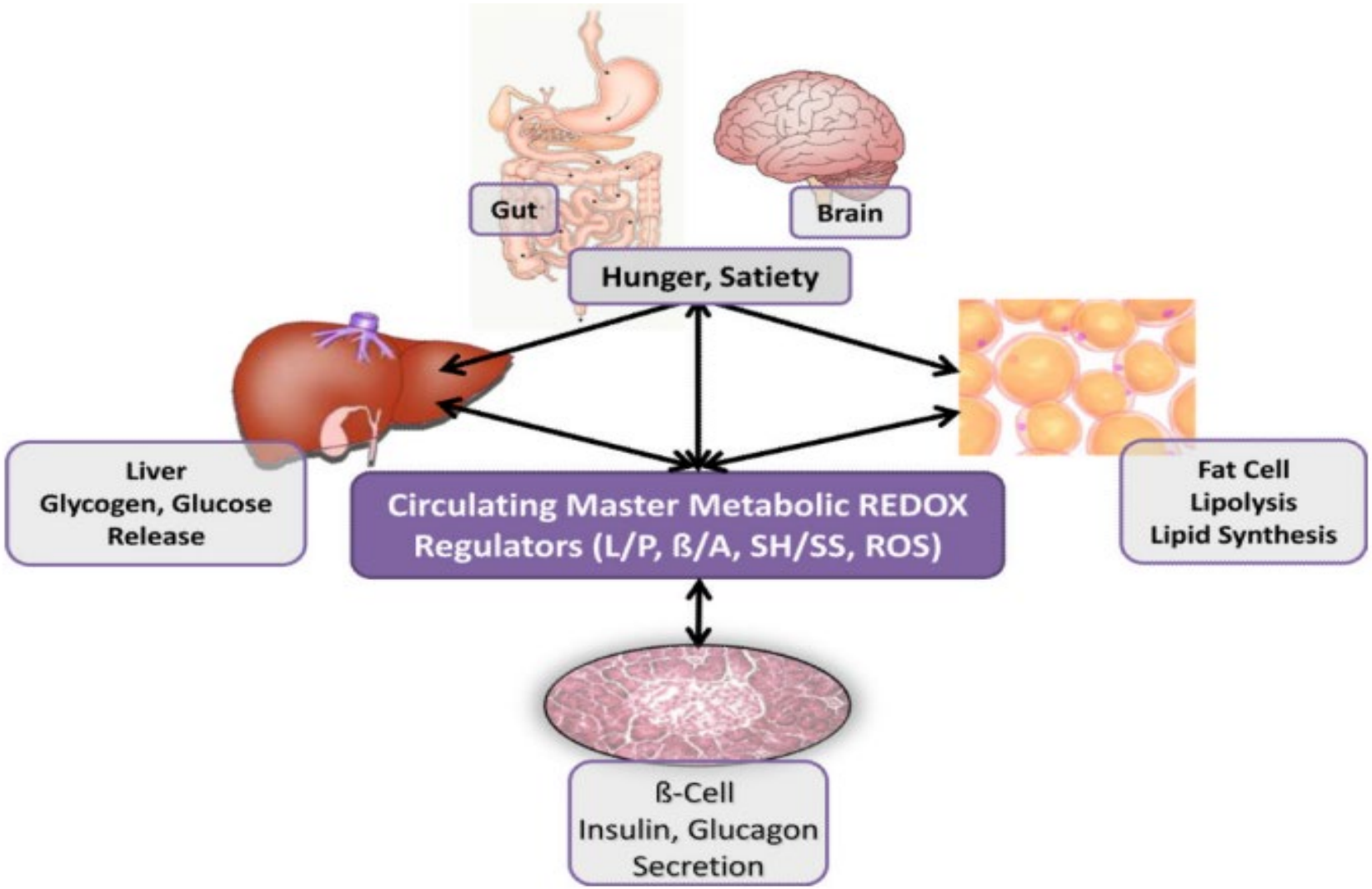
Redox Model of Obesity

- REDOX is an energy-responsive communication system that changes with fuel needs.
- Normal response to fuel is to increase reactive oxygen species (ROS) transiently.
- ROS signals to store fuel by increasing insulin, altering brain control of food intake, and stimulating triglyceride synthesis in adipose tissue.
- A ROS signal is inappropriate when fuel is normal… but stimulated by an outside force.

Energy Reduction-Oxidation Model: REDOX



Redox Sharing Among Metabolic Organs via the Bloodstream



B. Corkey and O. Shirhai, Trends Endocrinol Metab 2012

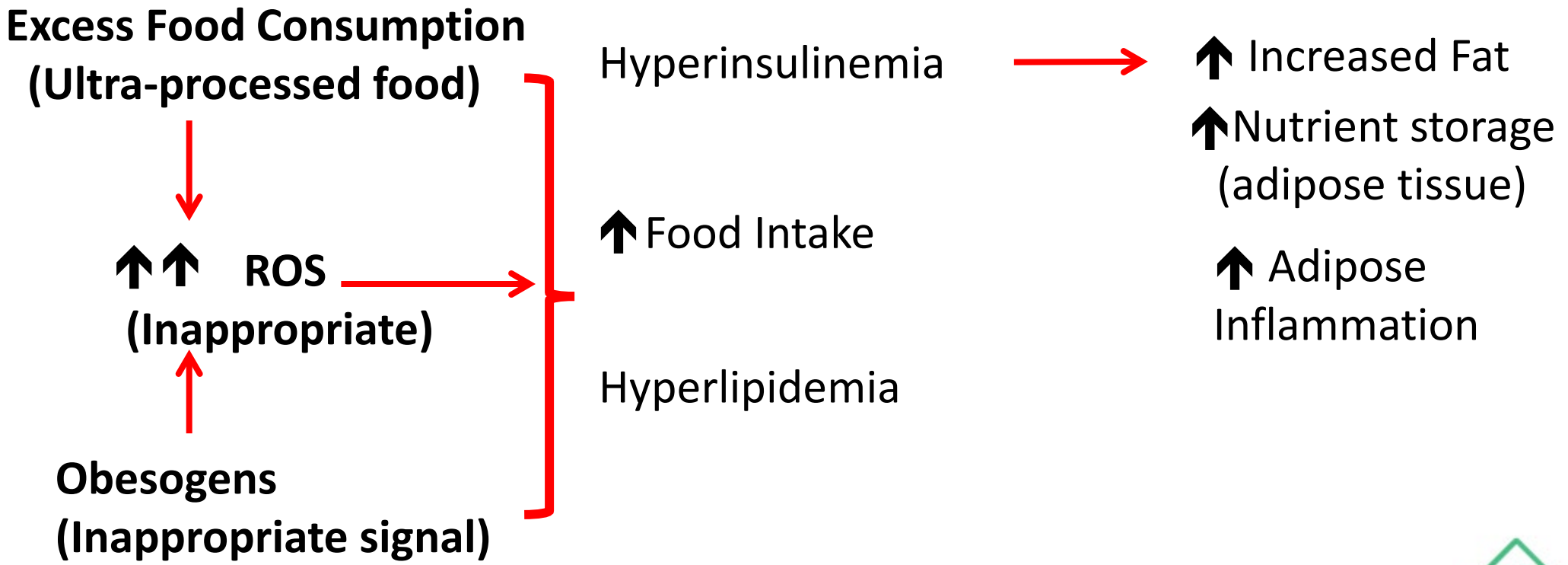
Is Obesity a Metabolic Disorder of the Control of Redox?

What has changed in the last ~50 years that is responsible for inappropriate increases ROS?

- ↑ Environmental Chemicals (Obesogens) that generate inflammation (ROS)
- ↑ Ultra-processed foods that contain Obesogens
- ↑ Ultra-processed carbohydrates
- ↑ Inadequate ROS scavenging capacity to meet the excess demand

Energy Reduction-Oxidation Model: REDOX

A metabolic disorder due to obesogen-mediated ROS



Summary and Implications (OBS-REDOX)

1. Ultra-processed food (especially carbohydrates) and obesogens change redox and generate ROS shared among all organs.
2. Such ROS constitutes a misleading signal.
3. Redox couples are transported among cells via circulation, thus interconnecting all organs.
4. ROS and redox changes impact function in an organ-specific manner.
5. The increase in inappropriate ROS causes hyperinsulinemia, hyperlipidemia, increased appetite and fat disposition.

Support and Challenges to REDOX Model

Support:

- The circulating redox state becomes more oxidized with age and disease.
- The changes in redox response to fuel consumption and obesogens are well documented.
- ROS impacts cell function in an organ-specific manner.

Challenges:

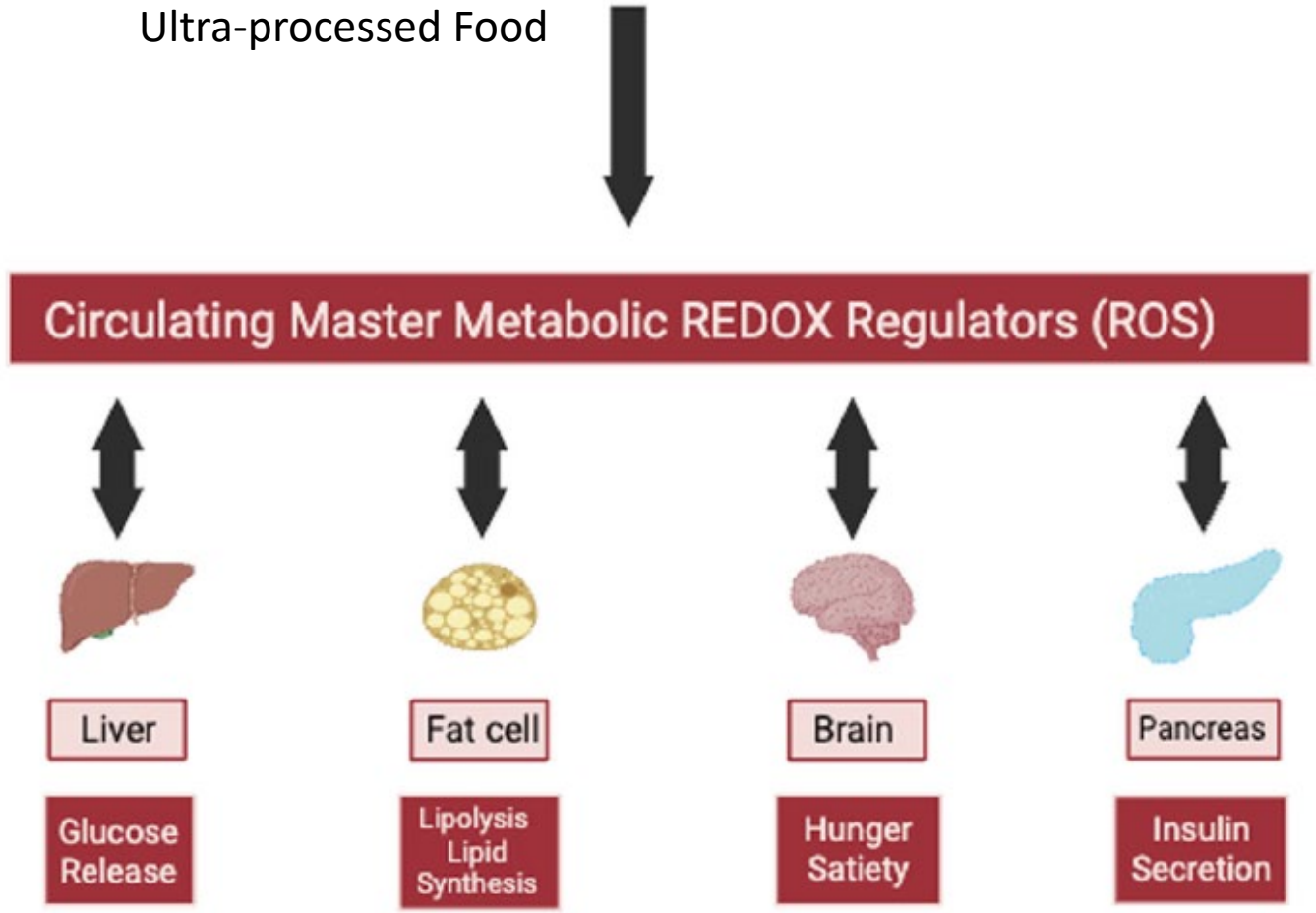
- The redox model is largely hypothetical.
- Strong evidence requires identifying the specific chemicals in human circulation and testing their ability to generate ROS.
- Documentation that removal of relevant chemicals can alter the redox state.

D.S. Ludwig, Wingspread 2022 Alemzadeh 1998

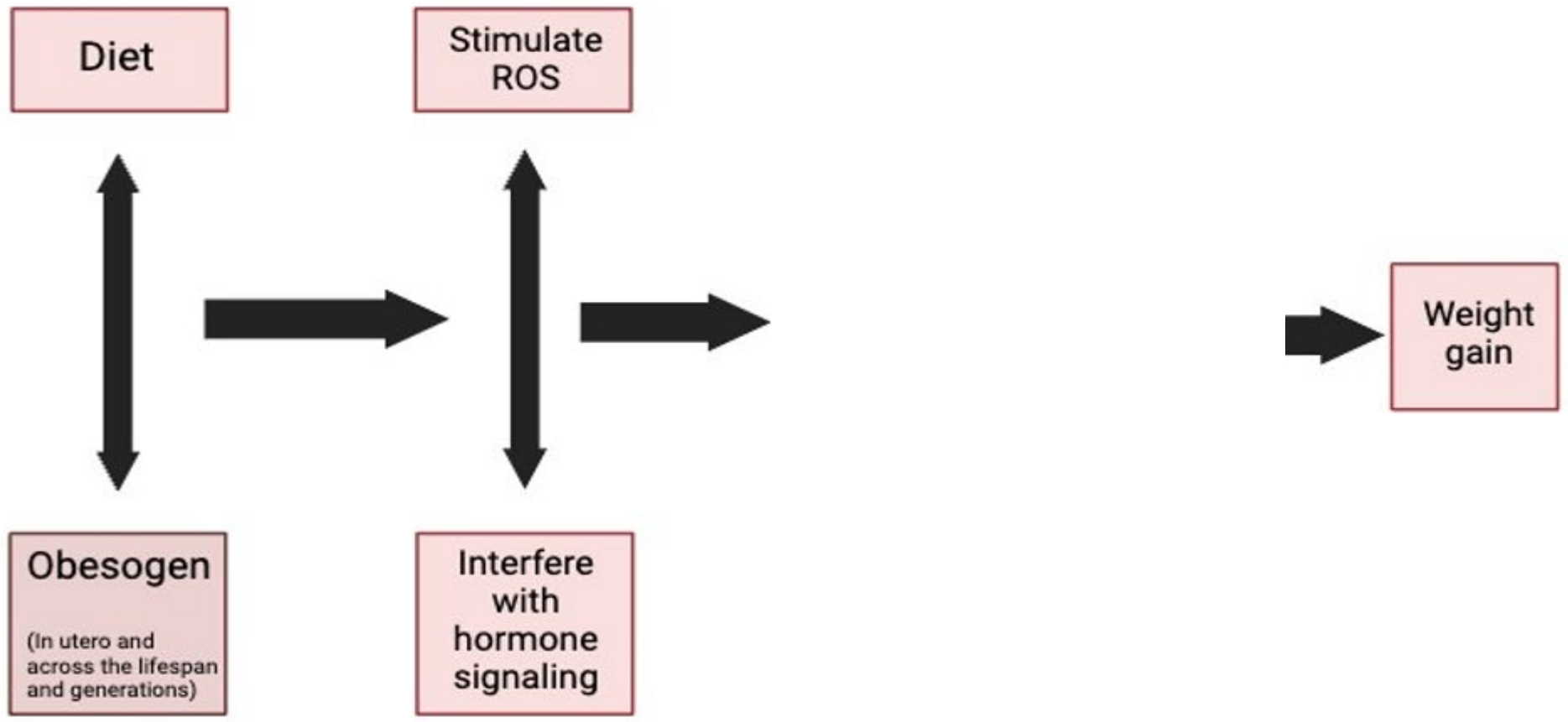
Integrating REDOX and OBS Models

Diet & Obesogens

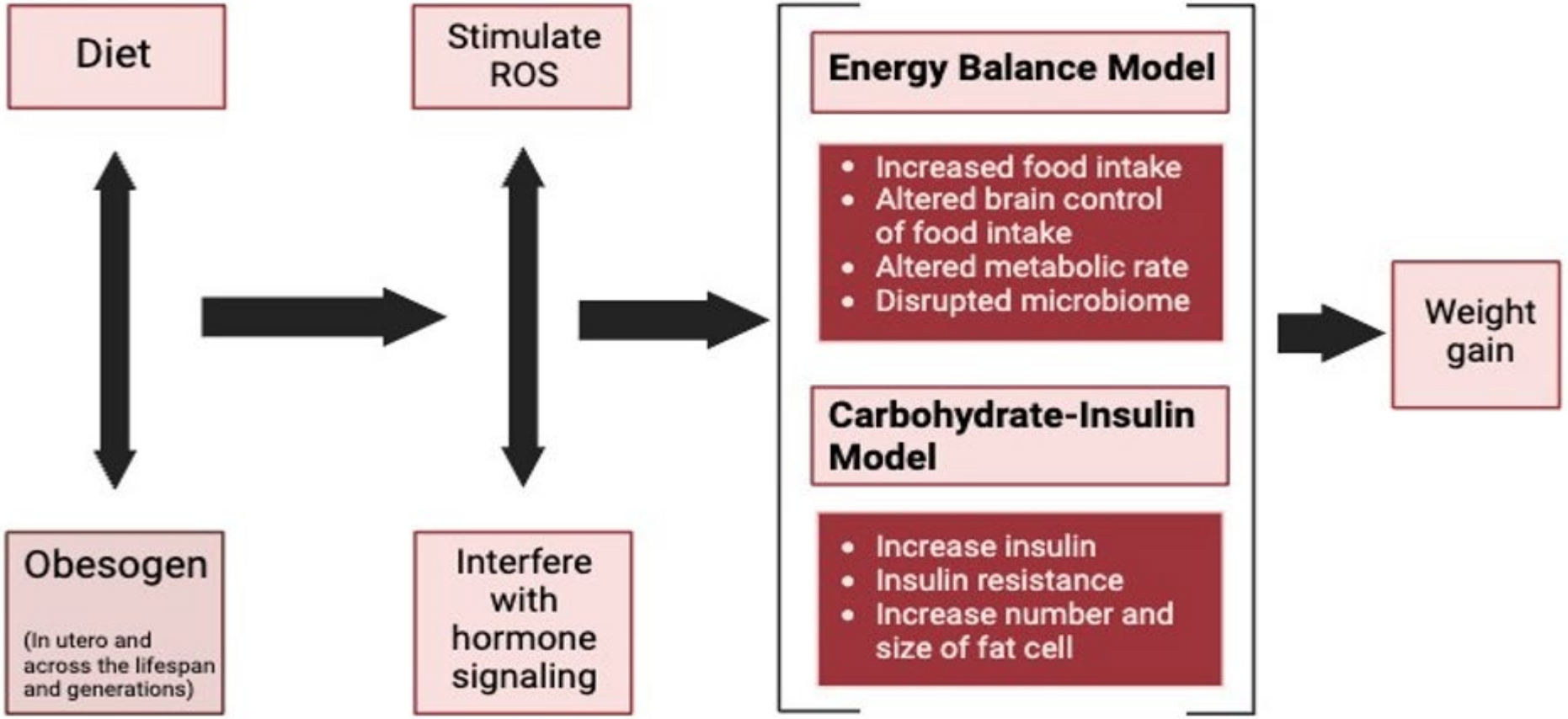
Abnormal Increase ROS: TBT, BBP, DEHP, PFAS, Atrazine, BPA, Cadmium, Chlorpyrifos, PCBs. PBDE, Air Pollution
Ultra-processed Food



The OBS/REDOX Model Contributes to a Unifying Theory for the Global Rise in Obesity



The OBS/REDOX Model Contributes to a Unifying Theory
A Focus on Obesogens



Key Points

- The cause of obesity is unknown…but it is multifactorial.
- There are four major models.
- Each model makes essential contributions to understanding the pathogenesis of obesity.
- We propose an integrated model that can explain all the major changes that are responsible for weight gain.
  - Developmental programming
  - Increased food intake
  - Weight gain without increased food intake
  - Increased insulin secretion
  - Altered microbiome
  - NAFLD
  - Increased number and size of adipocytes
  - Inflammation
  - Increased ROS
- Our model puts a focus on obesogens.
- Acceptance of this integrated model will focus on preventing obesity by reducing exposure to obesogens.

Good News: A Focus on Obesogens Changes the Narrative to Prevention

There are personal changes that can help.

- Reduce **exposure to environmental chemicals before** and during pregnancy
  - Filtered drinking water
  - Organic household products, cleaners, pesticides
  - Eliminate plastics/canned food (ultra-processed food)
  - Careful with cosmetics/sunscreens
- Watch nutrition, increase exercise, and decrease chemical exposures throughout life…to reduce the impact of second hits.

Obesity is a public health problem!

- **Provide affordable, sustainable, healthy food**
- **Reduce exposures to obesogens via policy and regulatory changes**

Not the end but just the end of the beginning of solving the obesity pandemic!

Poster Contest by HRIDAY with support from WHO SEARO