

Empty Calories: Obesity and the Composition of Dietary Fat

Tyler Ransom

*University of Oklahoma,
IZA & GLO*

Tucker Goodrich

Independent Researcher

Presented at San Diego State University

Aug 27, 2026

Obesity and Calories

- Health Econ Literature → declining price of calories is main driver of obesity (Cutler, Glaeser, and Shapiro, 2003; Philipson and Posner, 2003; Lakdawalla and Philipson, 2009)

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Heterogeneity of food is a natural place to look, but on which margin?

1. Intake: cheaper, tastier, more tempting → eat more (Strulik, 2019; Dragone and Ziebarth, 2017)
2. **Expenditure**: same total calories, **but different calories out**

Economics has exclusively followed #1

Can Dietary Composition Explain the Rise in Obesity?

The food-to-body conversion rate is variable

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Testing this requires holding calories fixed over a long horizon

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Identification Assumption: Mechanisms in animal experiments generalize to human biology (Dettmer et al., 2026, JPE)

Preview of Findings

- Ample animal evidence showing high-LA diet → obesity
- Human clinical evidence corroborates endocannabinoid mechanism
 - Rimonabant (blocks endocannabinoid receptors): weight-loss drug
 - GLP-1 RA drugs operate downstream of the ECS
- LA tracks obesity where the usual suspects (calories, sugar) do not
 - 1900–1960: obesity rate ↑4x, LA ↑1.7x, calorie supply ↓4%
 - 1980–2022: obesity rate ↑2.7x more, LA ↑1.7x more, sugar ~↗
 - 2000–2022: obesity rate ↑30%, LA ↑37%, calorie supply ~↗, sugar ↓16%
 - Same pattern in every anglophone country (reliable intake data)
- Our model predicts the BMI **distribution** better than a **calories-only** benchmark

Background: The Four Different Types of Fat

Fatty Acids

Chains of Hydrogen & Carbon atoms w/ Oxygen at end

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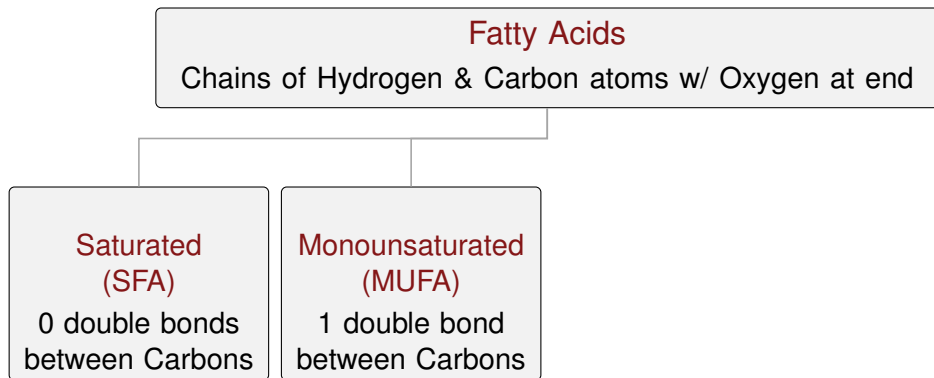
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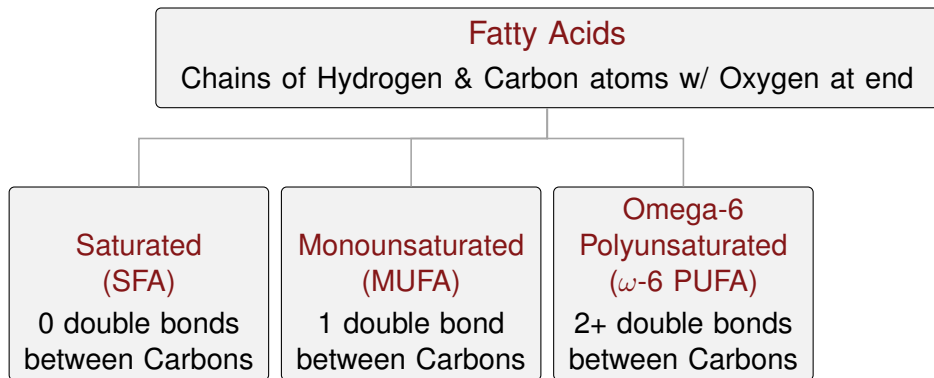
Saturated (SFA)

0 double bonds
between Carbons

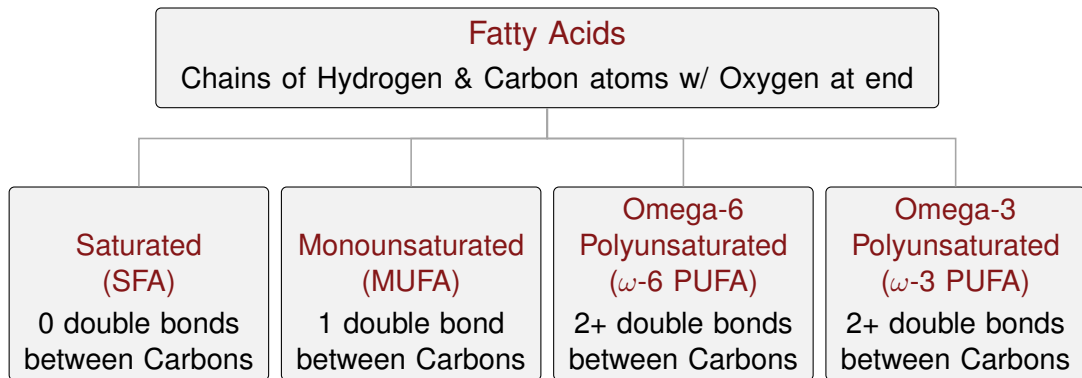
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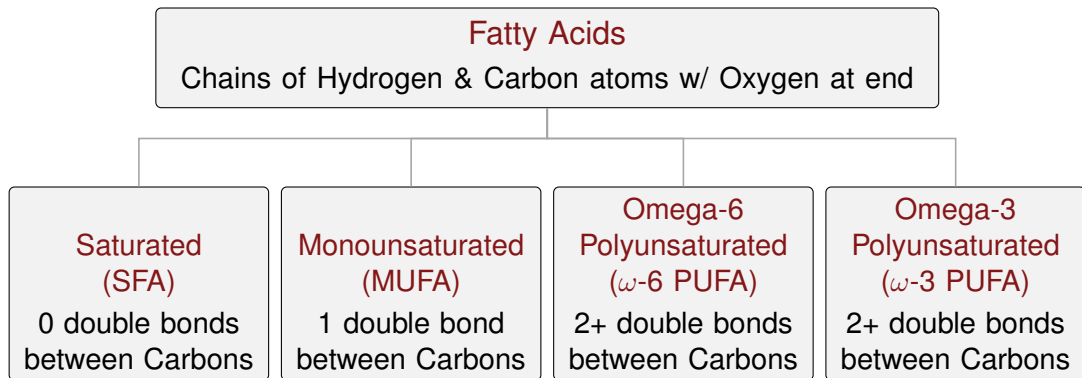
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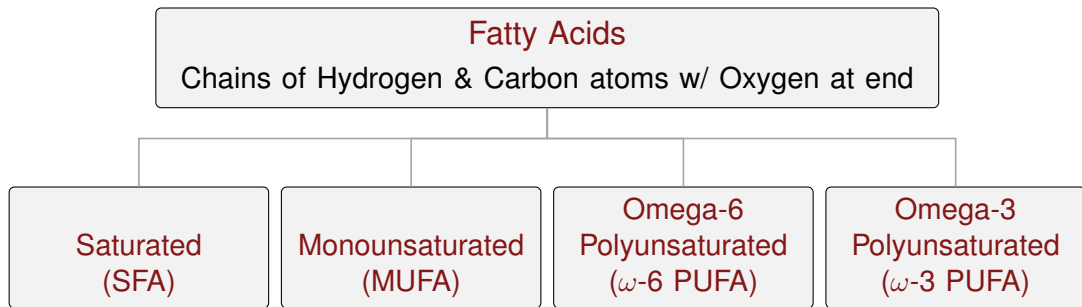
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Instability (peroxidation potential) increases in number of double bonds



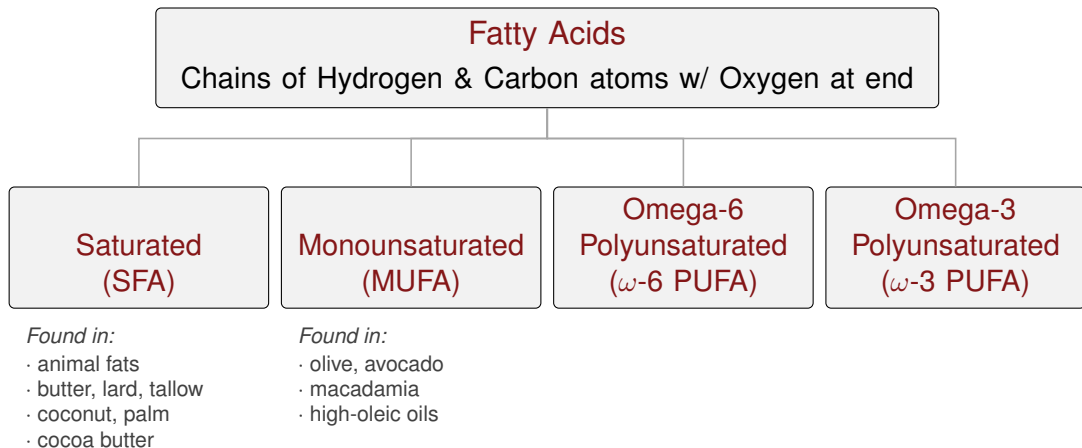
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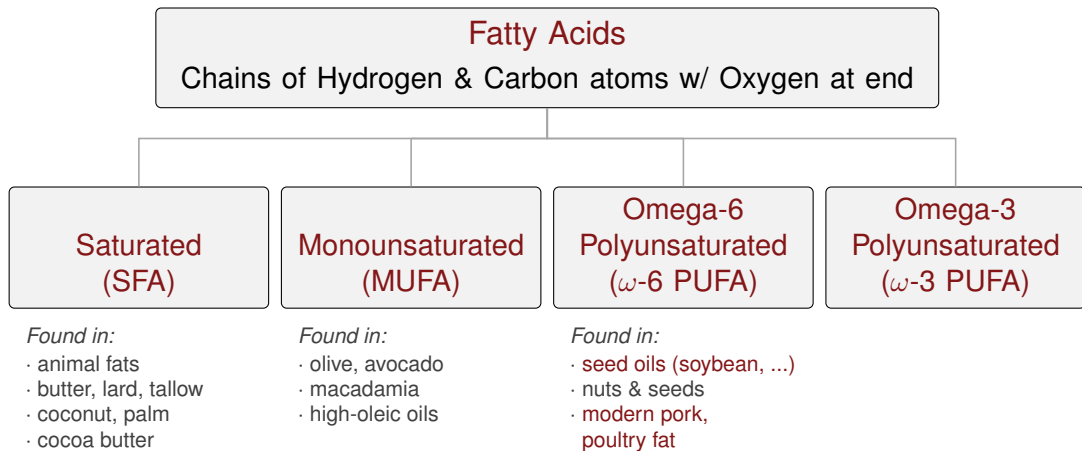
Found in:

- animal fats
- butter, lard, tallow
- coconut, palm
- cocoa butter

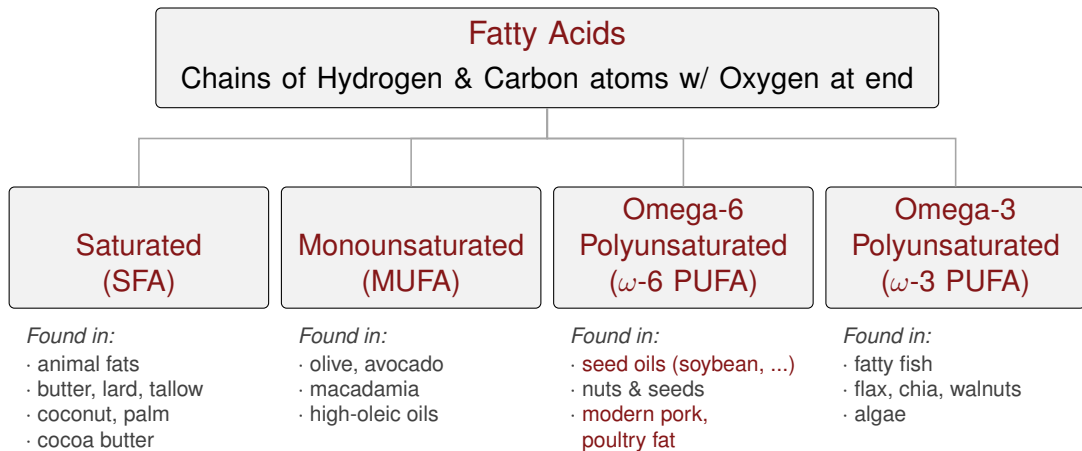
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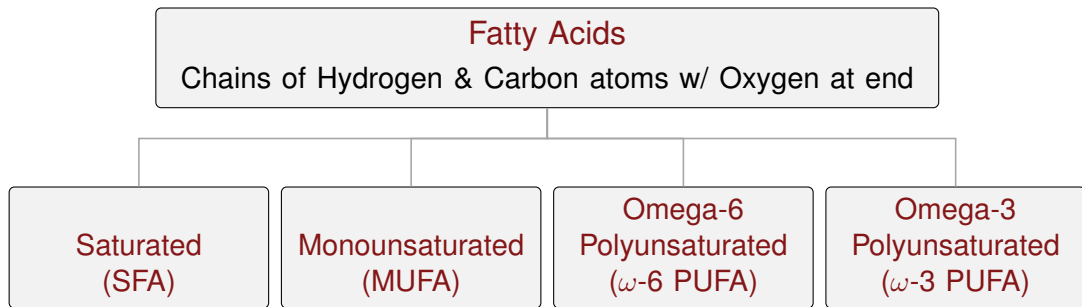
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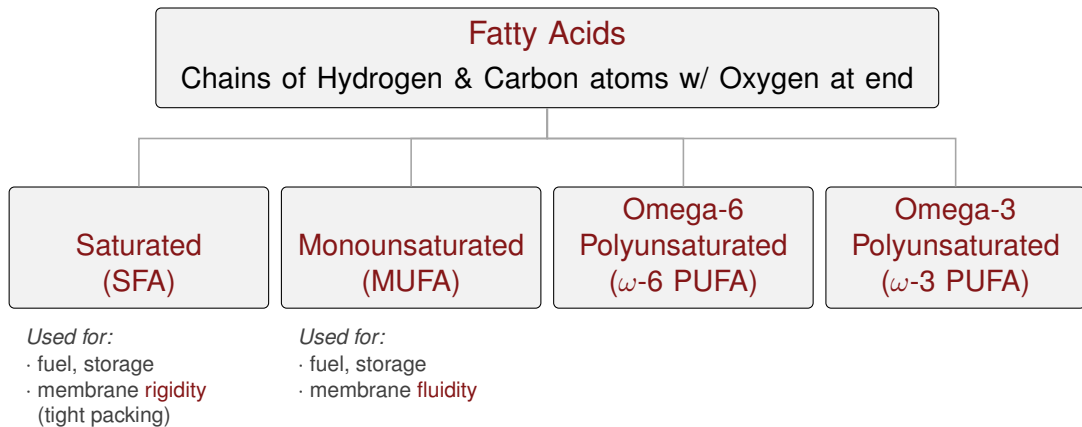
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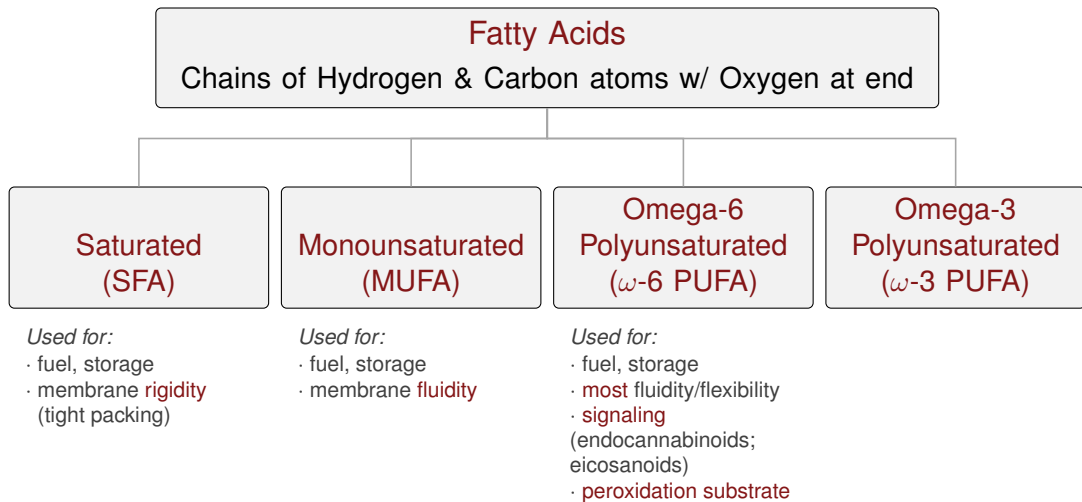
Used for:

- fuel, storage
- membrane rigidity
(tight packing)

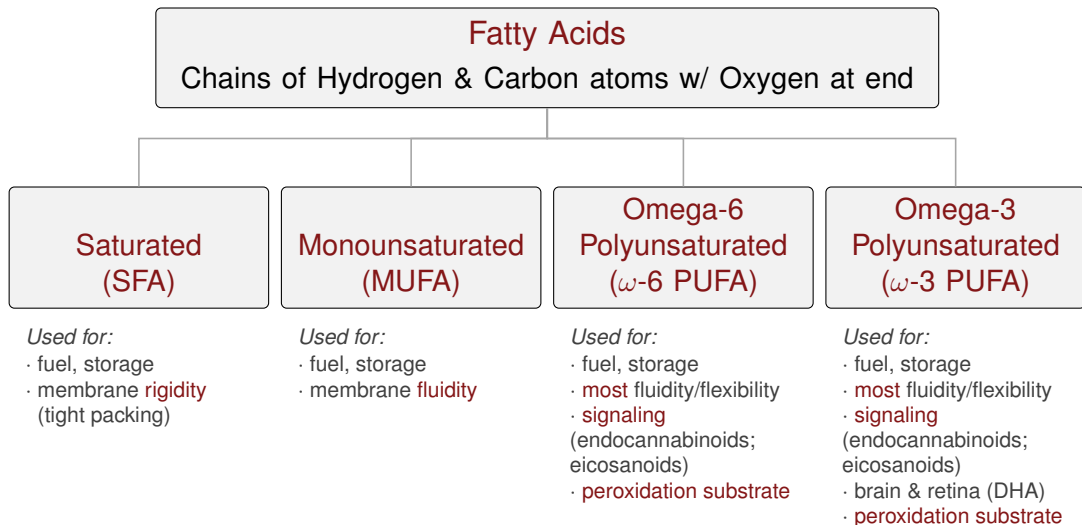
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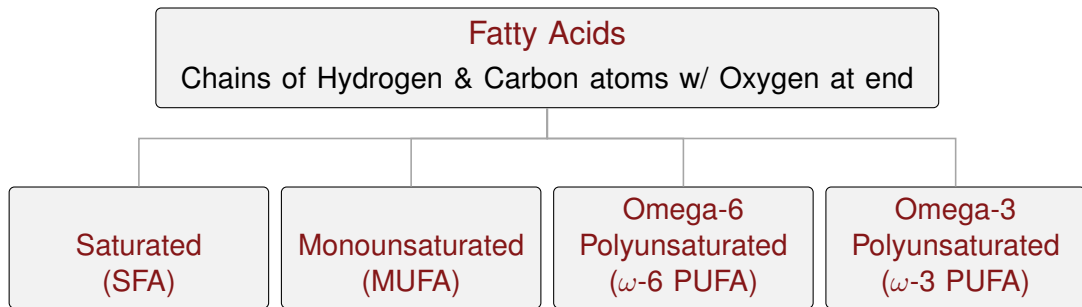
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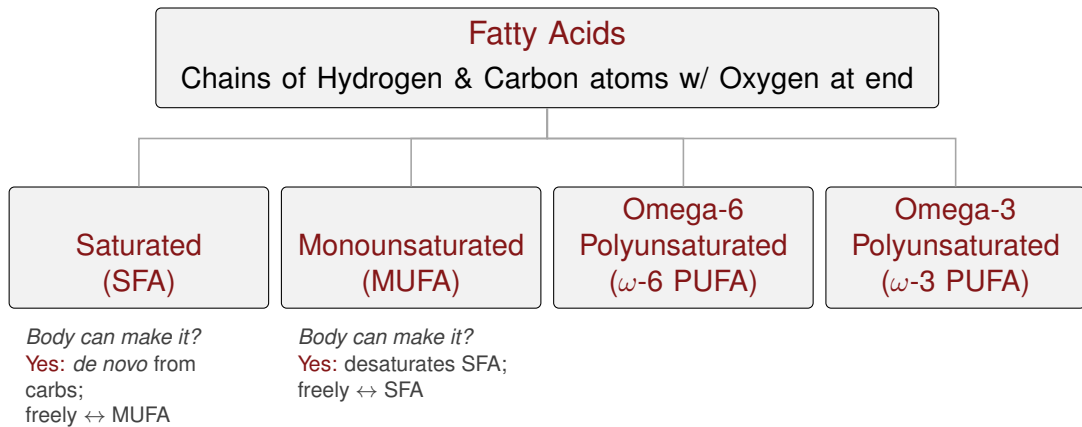
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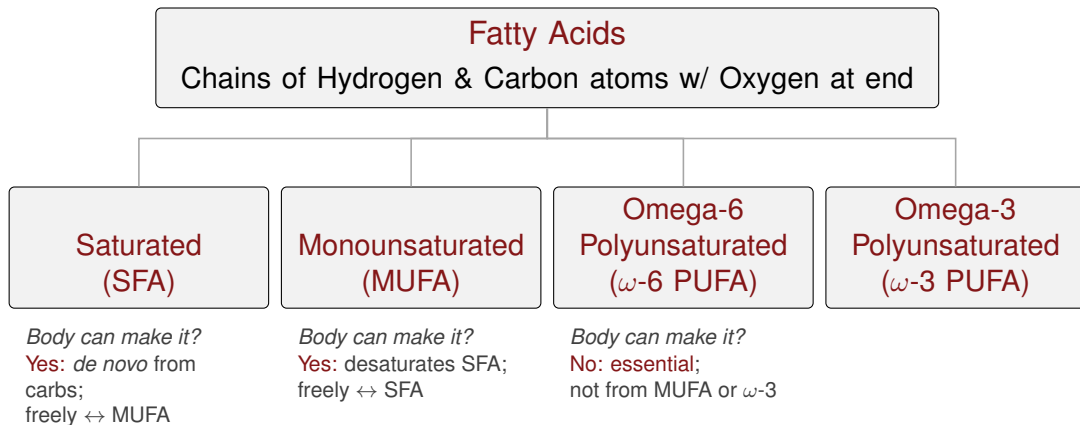
Body can make it?

Yes: *de novo* from
carbs;
freely ↔ MUFA

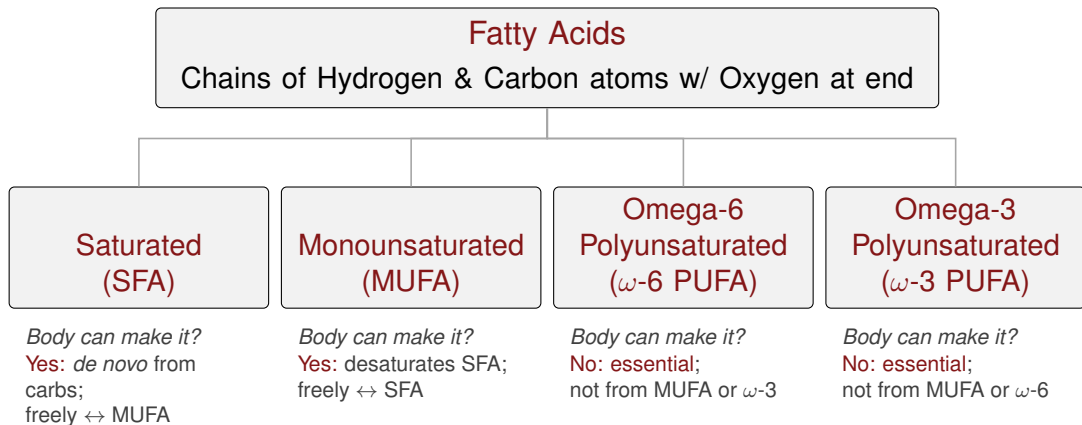
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Fat as an Imperfectly Substitutable Capital Stock

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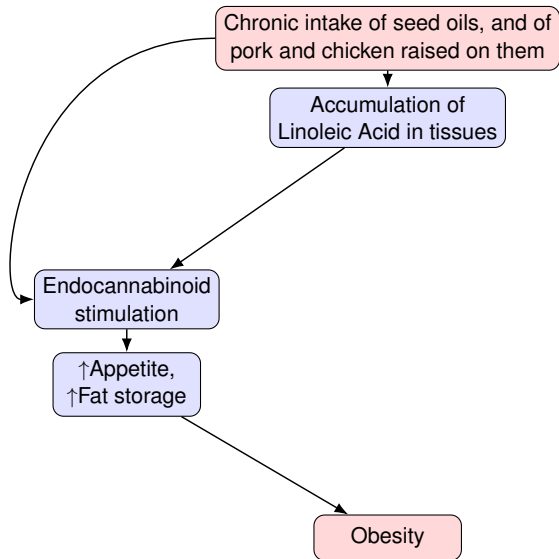
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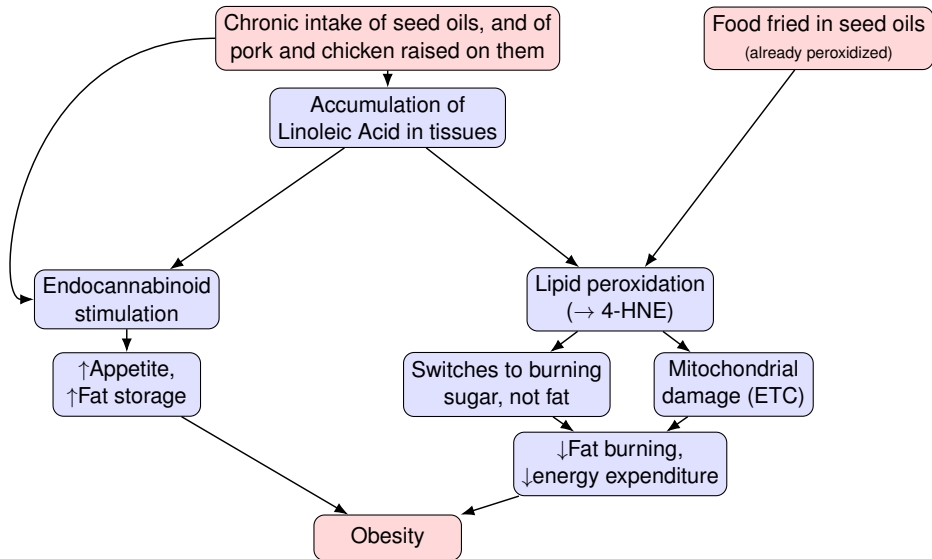
Body fat composition is cumulative dietary investment
(slow accumulation, slow depreciation, imperfect substitution)

No analog for carbohydrates (glycogen is glycogen)

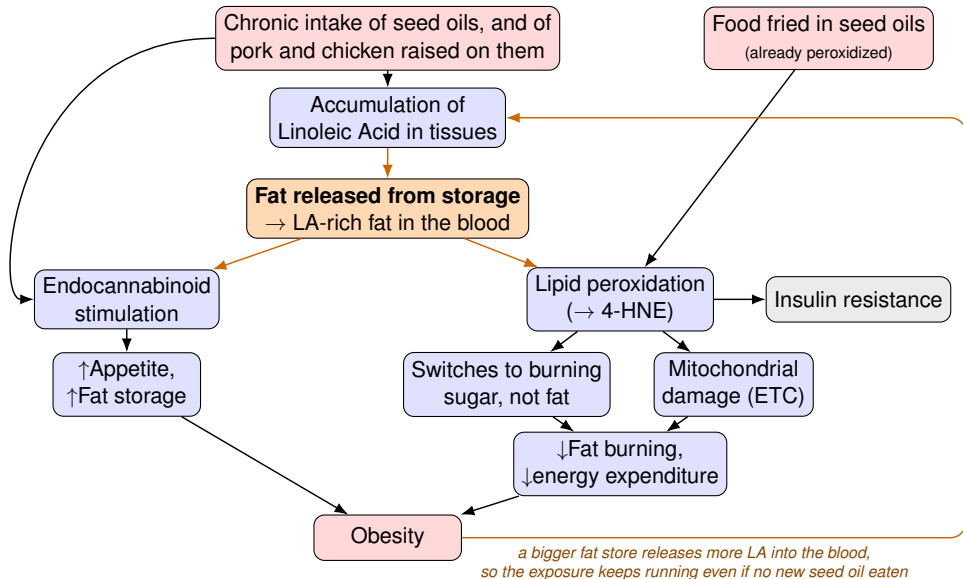
The Biochemistry-Microfounded Causal Model



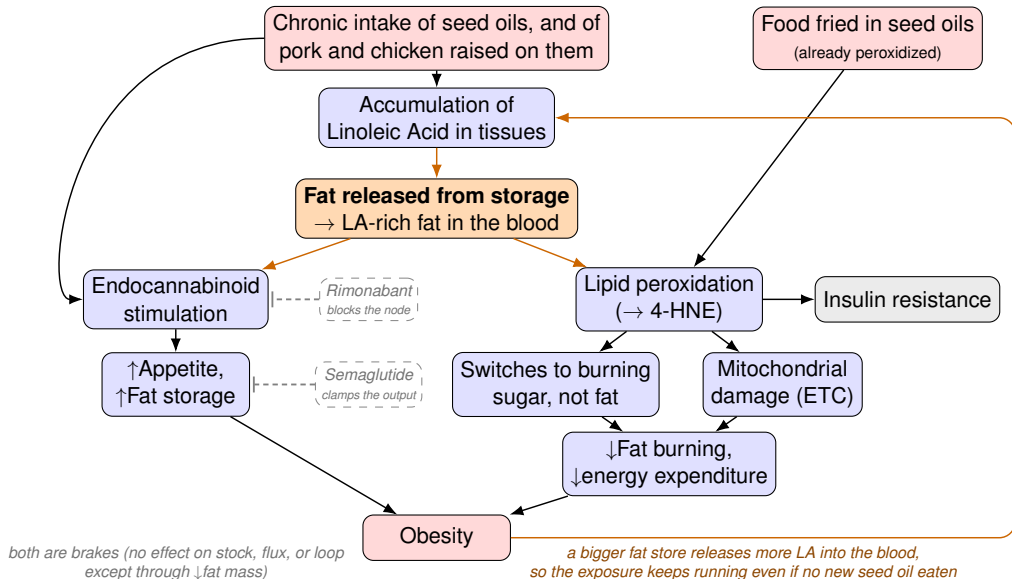
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State-of-the-Art Economic Model of Obesity

Express laws of motion over states W (body weight) and F (fitness)
(Dragone et al., 2026, JHE):

$$\dot{W} = c - \varepsilon u - \delta_w W F, \quad \dot{F} = \frac{u}{F} - \delta_F F$$

c : calorie intake u : exercise time ε : calories burned per unit of exercise

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Problems:

- c is a scalar (no role for dietary composition)
- δ_w is a constant (adaptive thermogenesis; Leibel, Rosenbaum, and Hirsch, 1995)
- εu is linear (TEE constrained; Pontzer and Trexler, 2026; Courtemanche, Pinkston, and Stewart, 2021, EHB)

Where the Law of Motion Comes From

The equation originates in **habit formation literature** rather than physiology:

Economics:

1988	Becker and Murphy	consumption capital; δ is forgetting
1993	Dockner and Feichtinger	how yo-yo dieting can arise
1999	Cawley	renames as “deprec. rate on weight (i.e. metabolism)”
2002	Levy	exogenous scalar; first closed form
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Latest econ innovations are on body comp.; **dietary composition is a first**

Our Generalization: Fat Composition as Choice & State

Add a **choice** variable λ (LA share of **dietary** fat) and a **state** T (LA share of **body** fat):

$$\dot{W} = c - \varepsilon u - \delta_w(T) WF, \quad \delta'_w(T) < 0$$

$$\dot{F} = \frac{u}{F} - \delta_F F$$

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h = half-life of adipose fatty-acid composition, ≈ 2 years (Dayton et al., 1966)

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- $\delta'_w(T) < 0$: an LA-rich fat stock **lowers the metabolic rate** (both mechanisms above)

Pricing dietary fat

The agent now chooses composition λ alongside quantity c :

$$M + w\tau = p_c(\lambda) c + (p_u + w) u + wq, \quad p'_c(\lambda) < 0$$

M : income w : wage τ : time endowment (labor, exerc. u , leisure q) p_u : p. exercising

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- Split c into LA/non-LA fat/non-fat kcal (fat share f , prices $p_L < p_O$):

$$p_c(\lambda) = \lambda f p_L + (1 - \lambda) f p_O + (1 - f) p_N$$
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- LA is **cheapest calorie in food system** (Drewnowski and Popkin, 1997)
 $\Rightarrow$ mechanism for **income gradient** in obesity, and a direct **policy lever**

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4. T is pinned by biology (not a researcher degree of freedom)

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Identifying assumption: these mechanisms generalize to human biology

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- **Slow:** one week of LA feeding moves **nothing**; 14 weeks plus in utero exposure moves everything (Alvheim et al., 2012)

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$p'_c(\lambda) < 0$	LA-rich calories are cheaper	Food-supply prices (Drewnowski and Popkin, 1997; Drewnowski and Darmon, 2005)

In all four δ_w studies the treated mice **did not eat more** than controls $\Rightarrow$ action is on the **expenditure** side, exactly where the scalar model has no room for it

What About the Human Trials That Find Nothing?

The model implies short RCTs are **uninformative by construction**

Trial	Design	Length	Gap closed
Hall et al. (2019)	UPF vs unprocessed, crossover	14 d	1.3%
Leibel et al. (1992)	Metabolic ward, 0–70 en% fat	33 d	3.1%
Courville et al. (2023)	ω -3 supplementation	12 wk	7.7%

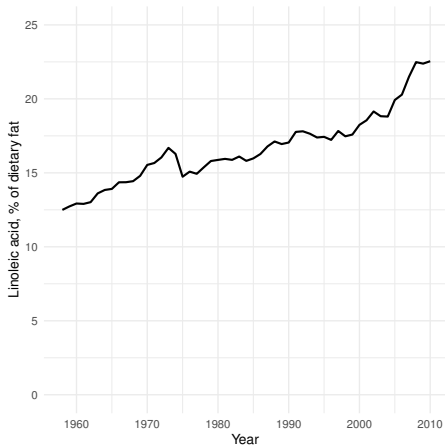
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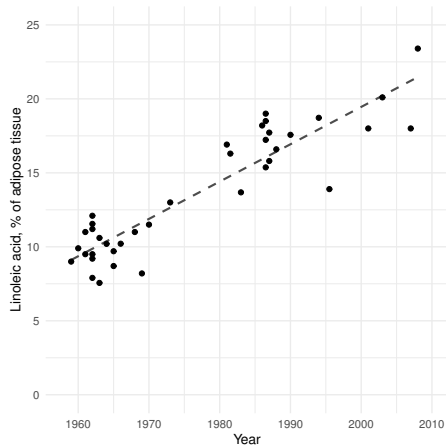
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- Hall et al. (2019) holds T fixed by construction $\Rightarrow$ uninformative in **either** direction
absolute ω -6 **higher** in the arm that ate less (8.1 vs 11.5 g/1,000 kcal)
- Dietary interventions are limited by h but surgical/pharma interventions may not be

Mechanism I: Diet Sets Tissue Composition



LA%, dietary fat



LA%, body fat tissue (Guyenet and Carlson, 2015)

Mechanism II: Tissue Composition Sets δ_w

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- Food intake is same, so mechanism must be expenditure/partitioning, not appetite

Mechanism III: Blocking ECS Node $\Rightarrow$ Fat Loss

The ECS's role in hunger and fine-tuning weight-loss medications

The cautionary tale of the drug **rimonabant**, a drug that blocks the CB1 receptor, is an interesting example of the central role the ECS plays in so many crucial functions. **It was developed as an anti-obesity drug.** The thinking was that the ECS controls hunger. We know this because, among other lines of evidence, cannabis gives you "the munchies," so **if you block the CB1 receptor it should cause weight loss.** Rimonabant did cause weight loss, quite successfully. **But, because the ECS also regulates mood,** it had to be withdrawn from the market on an emergency basis because **people who were taking it were becoming suicidal.** However, we can imagine a case, as we better understand the complexities of the ECS, where **we may be able to create a weight-loss medication that acts on those cannabinoid receptors that affect weight loss, but that doesn't act on those receptors that control mood.**

Human RCT: rimonabant 20 mg, **-4.7 kg** at one year (Christensen et al., 2007)

For comparison: semaglutide 2.4 mg, **-12.7 kg** at 68 weeks (Wilding et al., 2021)

Research continues into **peripherally restricted** CB1 antagonists, which retain the weight loss without acting on the brain (Mazier et al., 2015)

Mechanism IV: Lower ECS Tone $\Rightarrow$ $\uparrow$ Calorie Expenditure

- **Prediction:** lowering endocannabinoid tone should cut fat mass through expenditure, not only intake

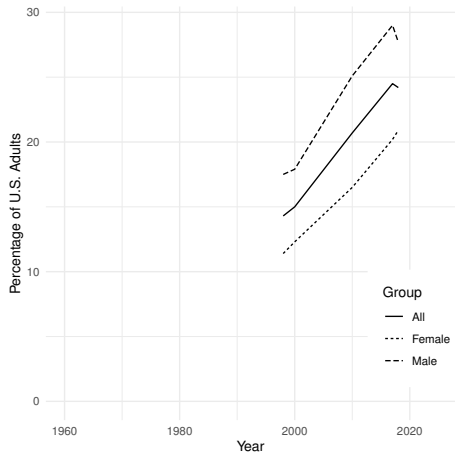
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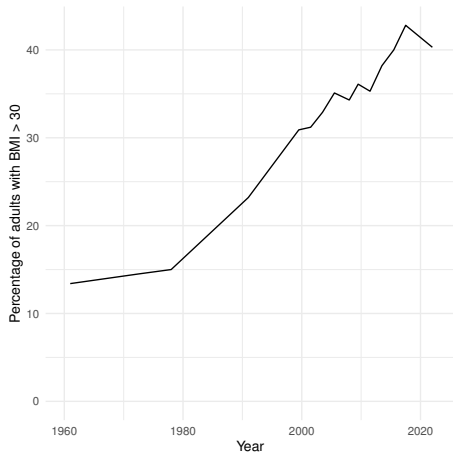
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- **Mice:** gastric bypass turns down gut CB1 and **raises energy expenditure**; cutting the nerve abolishes the effect (Ye et al., 2020)
 - $\rightarrow$ evidence of a **gut-brain axis**

Rival Series I: Physical Activity

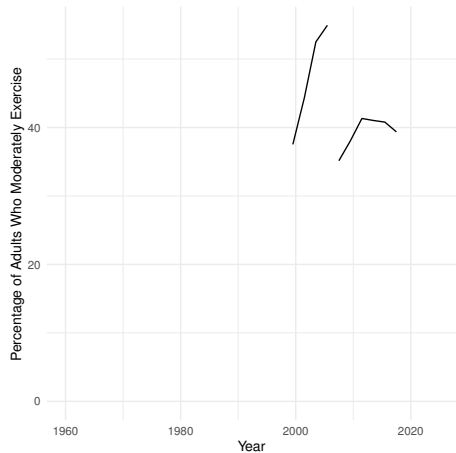


Meeting federal activity guidelines

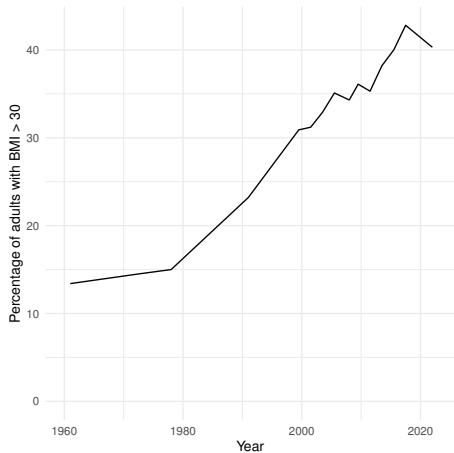


Obesity, 1960–2022

Rival Series I: Physical Activity

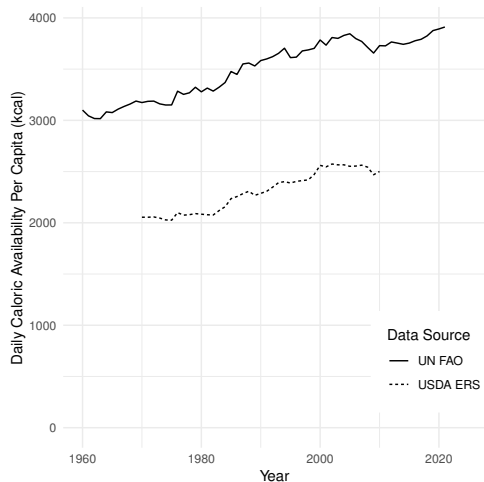


Exercise (NHANES)

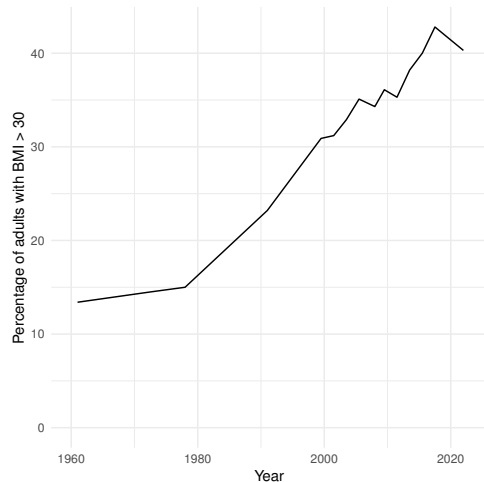


Obesity, 1960–2022

Rival Series II: Total Calories

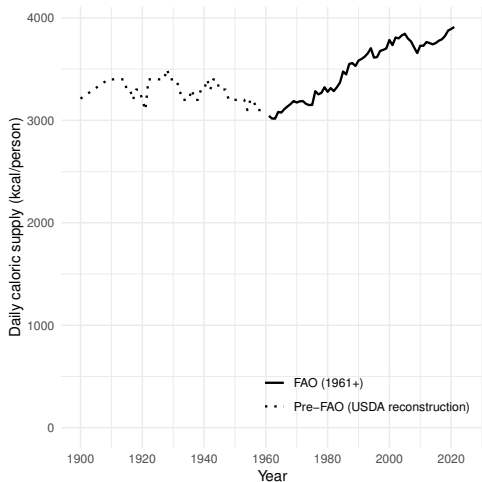


Calories, 1960–2022

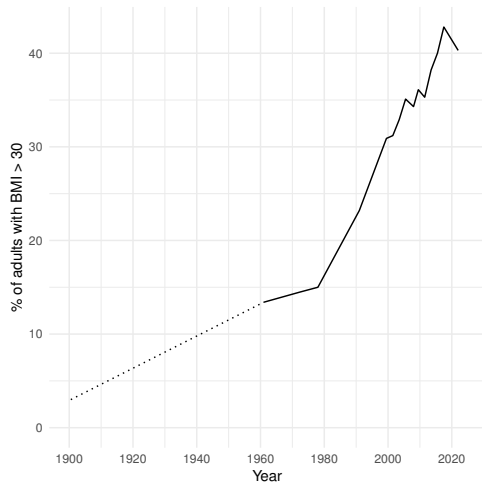


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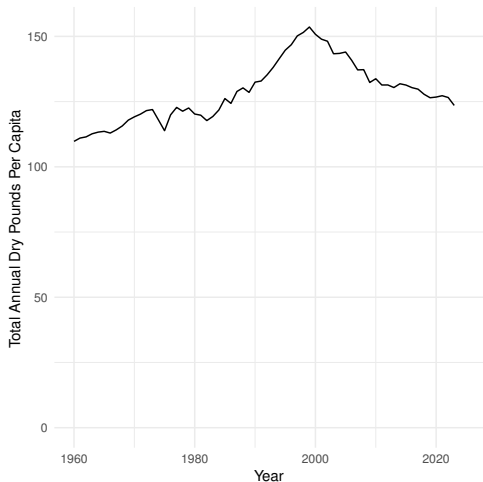


Calories, since 1900

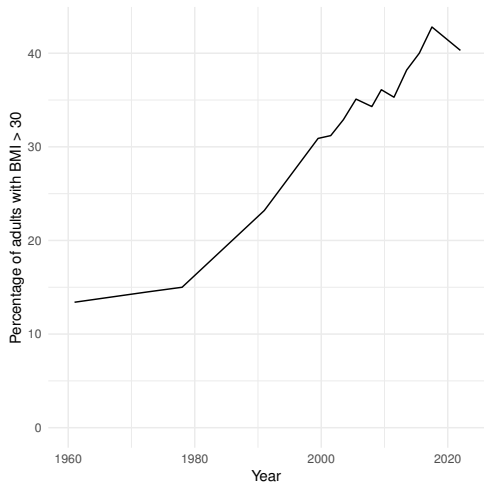


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Rival Series III: Sugar

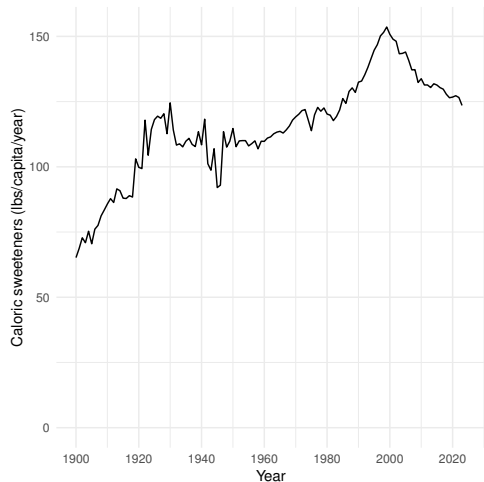


Caloric Sweeteners, 1960–2022

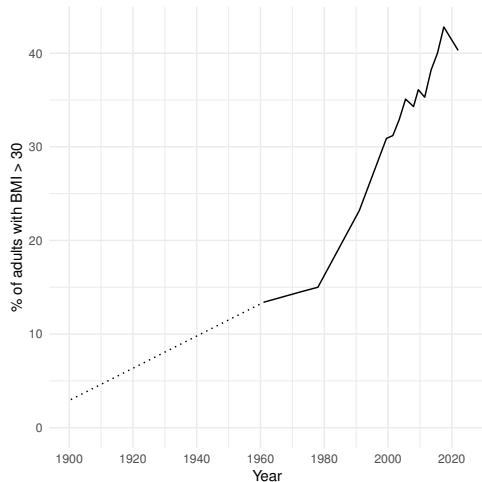


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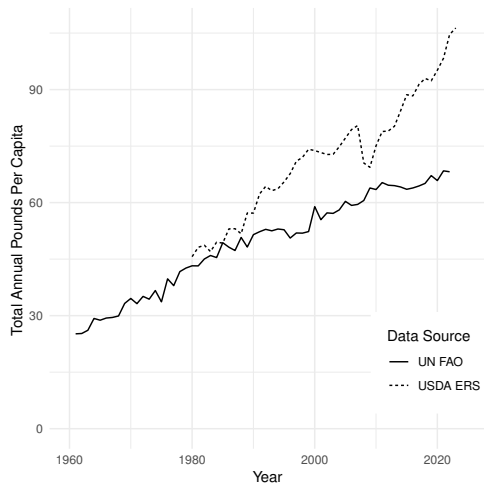


Sweeteners, since 1900

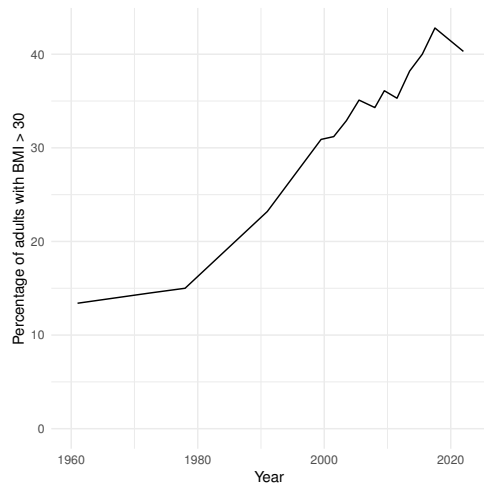


Obesity, since 1900

Exposure (Direct): Seed Oils

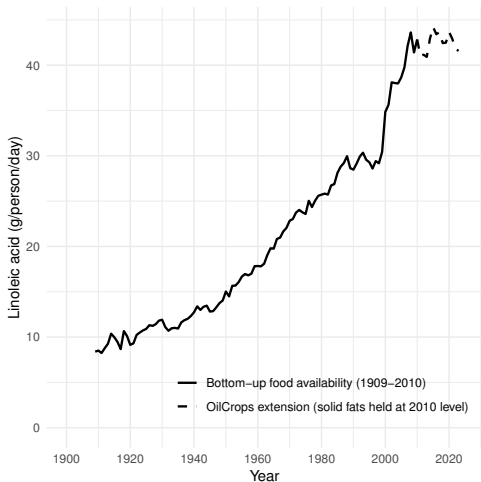


Seed oils, 1960–2022

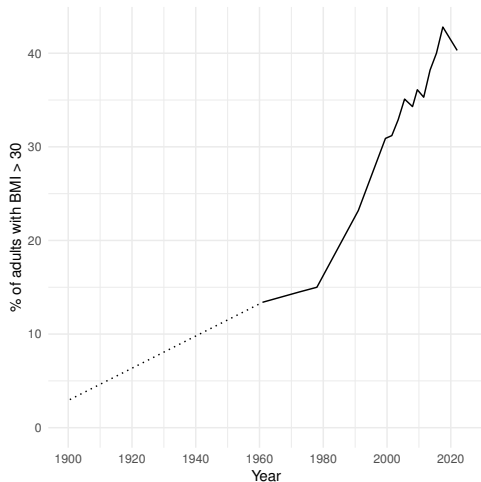


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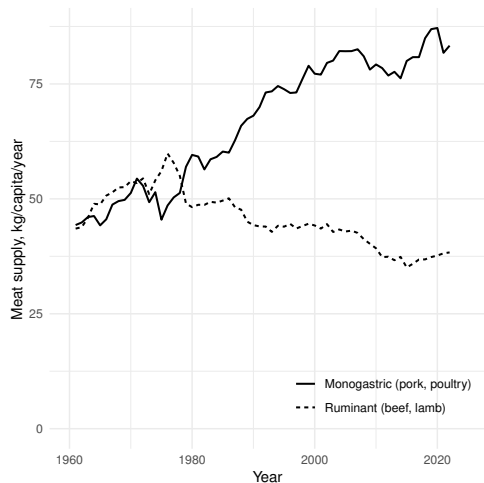


Linoleic acid, since 1900

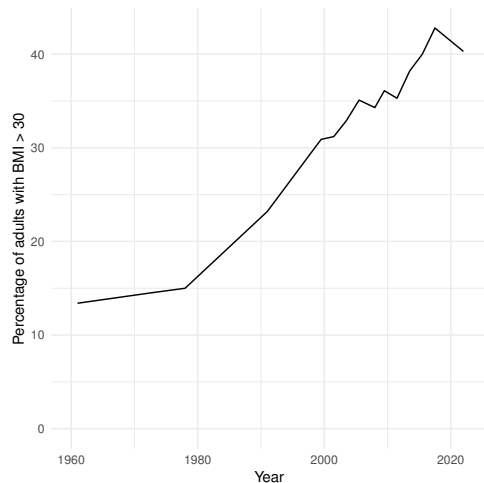


Obesity, since 1900

Exposure (Indirect): Pork & Chicken



Meat supply, 1960–2022



Obesity, 1960–2022

Untargeted Moments: BMI *Distribution*

	NHANES Data		Model prediction	
	1971–75 actual	2017–18 actual	Constant δ (additive)	$\delta(T)$ (multiplicative)
<i>Targeted</i> Median BMI	24.05	28.70	28.70	28.70

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Median BMI	24.05	28.70	28.70	28.70
<i>Not targeted</i>				
sd(log BMI)	0.193	0.246	0.164	0.193
95th percentile	34.70	44.29	39.35	41.41
$p_{95} - p_{50}$	10.65	15.59	10.65	12.71
Skewness of BMI	1.514	1.209	1.514	1.514

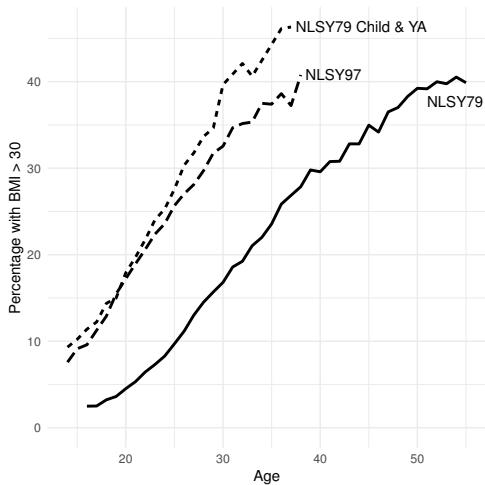
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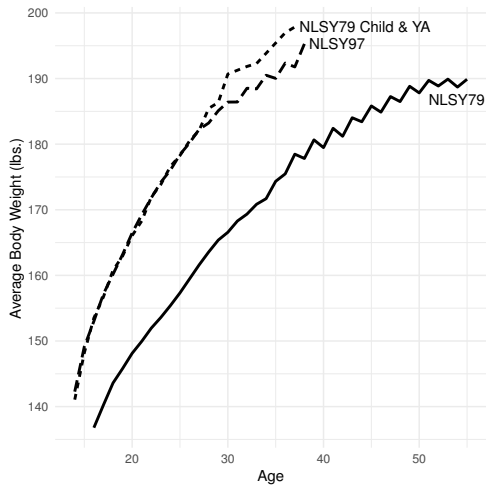
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<i>Share (%) of the 1971–75 to 2017–18 rise explained</i>				
Pr(BMI ≥ 30)	—	—	88.8	95.0
Pr(BMI ≥ 35)	—	—	51.4	69.5
Pr(BMI ≥ 40)	—	—	30.5	54.2

Prediction: Stock Shifts Onset Earlier (& Higher)

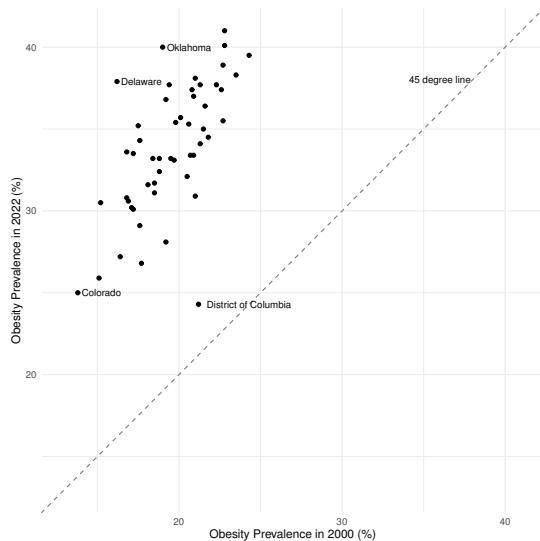


Share obese, by age

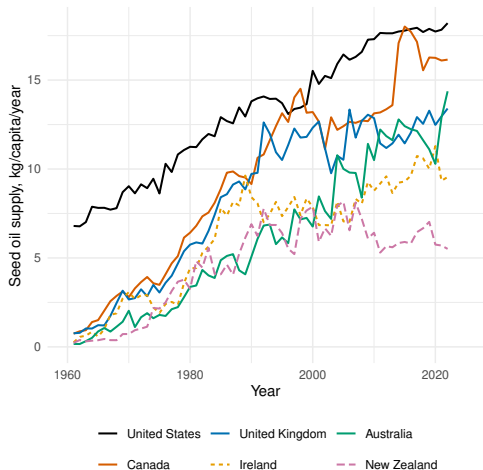


Mean weight (lbs), by age

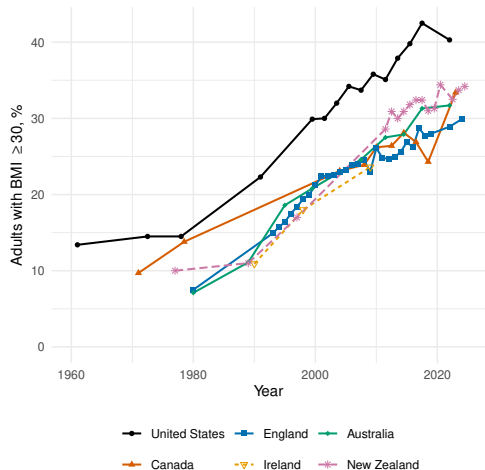
Prediction: National Exposure



Prediction: Same Pattern Where Intake Data Are Reliable



Seed oil supply, 1960–2022



Obesity, measured surveys

Prediction: Movers Converge to Destination Exposure

Prospective survey of >12,000 Japanese men in Japan, Hawaii, California
(Curb and Marcus, 1991)

- Avg BMI nearly equal in HI and CA, much lower in JPN
- Obesity at ages 45–49: 3.2% (JPN) → 11.9% (HI) → 12.9% (CA)
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Caveat: “American lifestyle” bundles many exposures at once

Consistency with health economics literature

Several explanations of obesity implicitly point to seed oils:

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- Fat share of calories: **unrelated** to BMI in 1976–80, **strongly related** by 1999–2006 (Ruhm, 2012)

What the health econ literature is missing

Remaining puzzles that our model fills in:

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- Stock dynamics: BMI $\uparrow$ even after price shock disappears (Strulik, 2014)

Other “Putative” Causes?

McAllister et al. (2009): 22 authors, **ten** contributors beyond diet and exercise, each graded on an evidence hierarchy

1. Infections (Ad-36 & microbiota)
2. Epigenetics
3. Maternal age
4. Reproductive fitness
5. Assortative mating
6. Sleep debt
7. Endocrine disruptors (PBDE, BPA, ...)
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Similar epistemics to us: human RCTs are **infeasible** for slow exposures $\Rightarrow$ animal evidence should carry more weight

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 - Obesogens (McAllister's #7) plausibly **do** vary geographically (Song et al., 2014; Völker et al., 2022; Grandjean et al., 2023)
 - Different limb, so additional explanation

Policy Menu: Target λ or $p_c(\lambda)$

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- Must be paired with greater surveillance in anticipation of food companies’ incentives (Conroy, 1888; Barahona, Otero, and Otero, 2023)

Tradeoffs

Should we treat the ultimate cause, or symptoms?

- **Second best:** pharmaceutical intervention is the incentive-incompatible mechanism of obesity policy, but only works while it is running
 - $\approx 75\%$ of GLP-1 weight loss is regained after stopping (Budini et al., 2026)
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Caveat: policy case overruns the evidence (also true for sugar taxes, package labeling, ...)

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- **Not monocausal**, but composition is absent from economic models of obesity
- Same mechanisms load onto **CVD** and **insulin resistance** $\Rightarrow$ obesity-only externalities are **understated**

Where to next?

- Modeling the political economy dynamics of seed oil / “ultraprocessed food” (and obesity/metabolic syndrome) proliferation
 - economies of scale/scope in raw/intermediate production of seed oils
 - complementarity with food chemistry innovation
 - information asymmetries
 - show how current equilibrium can be disrupted
- Modeling metabolic disease as a macro-style DSGE model
 - use model to simulate how dietary changes result in metabolic disease
 - simulate policies that change food environment
 - estimate “healing” effects of policies (sugar taxes, labeling, seed oil ban, ...)

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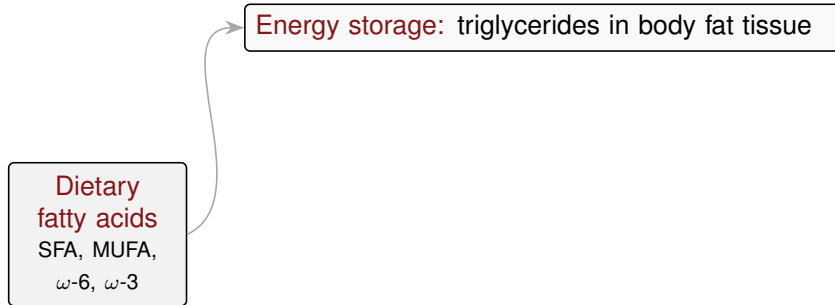
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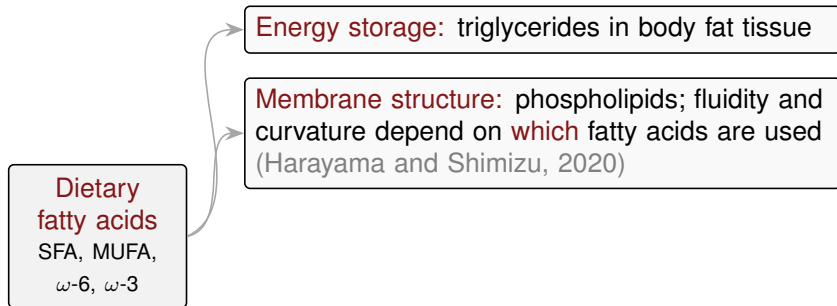
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Backup: Biochemistry Background

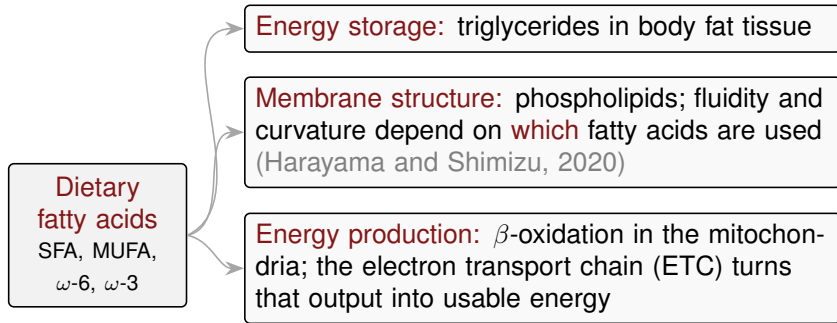
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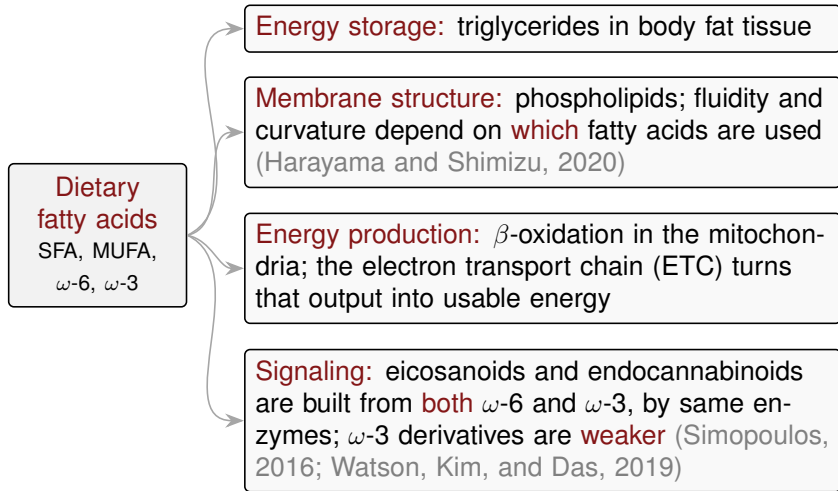
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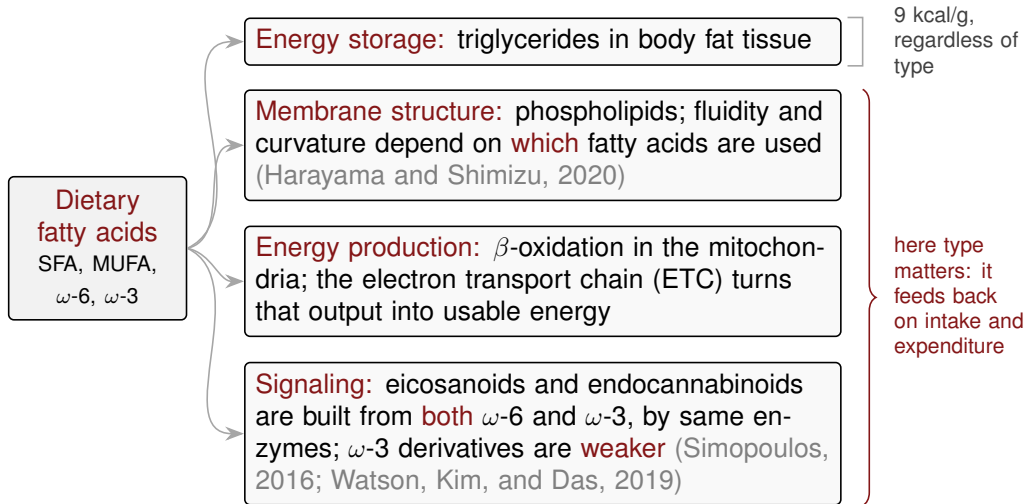
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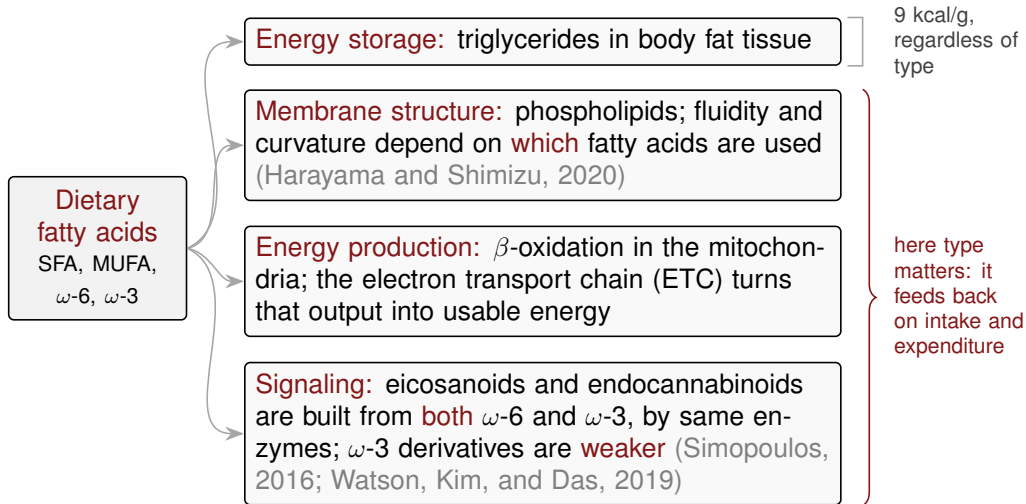
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Some tissues (brain, eyes) need specific fatty acid profiles; body fat is the “residual claimant”

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 - Built on-demand from AA **already stored in cell membranes**, so the input is the tissue **stock**, not today's intake
- CB1 sits in the hypothalamus (brain), gut, liver, and fat
 - Activation raises appetite *and* shifts the body toward storage
 - Result is $\uparrow$ “feed efficiency” (weight gained per calorie eaten)

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- Frying is a **second entry point** where seed oil comes in pre-peroxidized so 4-HNE is directly consumed (Grootveld, Percival, and Grootveld, 2018)

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 - CB1 activation **suppresses** GLP-1 release (Chia et al., 2017; Perez et al., 2024)
- ⇒ GLP-1 sits **downstream** of ω -6 (is **not necessary** for obesity)
- Delete CB1: mice stay **lean**, as predicted (Ravinet Trillou et al., 2004)
 - Delete GLP-1R: deficiency story predicts **fatter**; mice gain **no more** than controls (Scrocchi and Drucker, 1998)