How Medicine Lost Its Soul: The Architecture of Two Manufactured Diseases: Hyperlipidemia & Osteoporosis

How LDL and T-Scores Became Tools of Profit, Not Health

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There are moments in science when the fog suddenly lifts—not because you've made a new discovery, but because you finally see the architecture of a paradigm. Not as truth—but as a deliberate construction.

Since retiring in 2023, I've been studying and speaking about the flaws in the cholesterol-heart hypothesis—the idea that LDL is the root cause of cardiovascular disease. I came to see how deeply broken that model is: built on surrogate markers, driven by industry, and reinforced by decades of fear-based messaging.

Then, by chance, I stumbled into another world—osteoporosis—after encountering a case of an atypical femur fracture in a patient who had been taking the blockbuster drug Prolia. Curious, I began digging.

What I found left me stunned. It was the same blueprint. The same tactics. The same story—just with different characters. Instead of LDL, there was the T-score. Instead of statins, there were bisphosphonates and Prolia. Instead of cholesterol guidelines, there were fracture risk calculators. Different organs, same machinery.

That's when I realized: this isn't just about one or two drugs. It's about a system—a system that turns statistical risk into disease, disease into lifelong treatment, and treatment into a steady profit stream. The osteoporosis story isn't an outlier—it's part of a pattern. And once you see the pattern, you can't unsee it.

I know there are many more examples—others I plan to study, expose, and write about in future essays.

I. The Blueprint: How a Diagnosis Is Manufactured

Step 1: Find a measurable biomarker.

For heart disease, it was LDL.

For bones, it was the T-score from a DEXA scan—a measure of how far your bone density deviates from that of a young adult.

Step 2: Create an arbitrary cutoff.

In 1994, a small, undisclosed WHO committee declared that a T-score of ≤ –2.5 would now define "osteoporosis."

This decision was **not based on fracture data, biological thresholds, or clinical outcomes**—but on statistical deviation alone, drawn from the NHANES III dataset.

The T-score compares a woman's bone density to that of a healthy 30-year-old. But when else in medicine do we define disease by comparing a 70-year-old to her peak biological state at 30?

We don't call age-related muscle loss "sarcopenia" unless it becomes pathological. But with bones, the bar was lowered to make aging itself a disease.

The better predictor should have been the **Z-score**, which compares a patient to their own age group—but it was sidelined. Why? Because using Z-scores would drastically reduce the number of women labeled osteoporotic.

Likewise, the cholesterol cutoff of 200 mg/dL—and later LDL of 130—wasn't based on hard outcomes, but on population averages and risk inflation.

Over time, these thresholds were pushed lower to label more people as "at risk."

The same pattern is now unfolding for **ApoB** and **Lp(a)**—both biomarkers now targeted by billion-dollar drugs in development.

What we're treating isn't disease. We're treating numbers.

Step 3: Use flawed data to justify the cutoff.

The NHANES dataset used to define osteoporosis included **no follow-up** on who actually broke bones. It was a snapshot survey, not a prospective study.

And the **Malmö study**, which fed into the FRAX fracture calculator, wasn't even a clinical study—it was a statistical model based on NHANES-derived fracture estimates, not observed events.

Similarly, Ancel Keys' Seven Countries Study—the origin of the fat/cholesterol myth—was observational, selective, and ignored contradictory evidence.

Step 4: Create a global algorithm.

Enter **FRAX**—a 10-year fracture risk calculator developed under John Kanis at the WHO Collaborating Centre.

It used age, weight, smoking, and—of course—the T-score. But its foundational data was modeled, not observed, and excluded key fracture risk drivers like **nutrition**, **trauma**, or **metabolic health**.

Statins had their counterpart: the ASCVD Risk Calculator.

Plug in age, BP, cholesterol—and you've got a justification for lifelong treatment.

Step 5: Build fear.

Once the diagnosis was set, the messaging followed:

- "Silent killer."
- "Brittle bones."
- "Fracture = nursing home."
- "Cholesterol clogs your arteries."

And the numbers?

Women with osteoporosis (T ≤ –2.5) have a ~2.5% annual fracture risk.

But women with **osteopenia** (T –1 to –2.5) account for the **majority** of fractures—at only ~1.3% risk.

Even those with **normal** bone density fracture at ~0.8%.

That's a **tiny absolute risk difference**—but a **massive psychological impact**.

Same with statins.

They reduce **relative** risk by 20–30%—but the **absolute** risk reduction is often less than **1% over 5 years** in primary prevention.

II. The Real-World Consequences

The result?

A tidal wave of aggressive drug treatment:

- **Bisphosphonates and Prolia** for osteoporosis
  → Can lead to **atypical femur fractures**, **osteonecrosis**, and **rebound bone loss**
- **Statins** for cholesterol
  → Linked to **muscle pain**, **fatigue**, **diabetes risk**, and **cognitive issues**

And all this while **root causes**—like insulin resistance, inactivity, hormonal imbalance, and nutrient depletion—go unaddressed.

III. The Echo Chamber Effect

Studies like CaMos (Canada) and SOF (U.S.) reinforced the osteoporosis narrative by focusing narrowly on T-scores. This singular focus was echoed by major institutions worldwide—including the International Osteoporosis Foundation (IOF), the National Osteoporosis Foundation (NOF) in the U.S., the European Society for Clinical and Economic Aspects of Osteoporosis (ESCEO), and countless national health systems and academic societies—creating a global echo chamber where bone density became the disease, and deviation from a young adult norm became pathology.

Just as in cardiology, any study **challenging** the LDL model was sidelined or underfunded.
A generation of doctors grew up **memorizing thresholds**, **prescribing pills**, and **never questioning** the foundation.

IV. Who Profits?

- **Pharmaceutical companies**, who created drugs for a problem they helped define
- **Imaging manufacturers**, who turned DEXA scans into routine screening tools
- **Medical societies**, funded by industry, endorsing guidelines that expand drug eligibility
- **Continuing Medical Education** (**CME**) programs, sponsored by drug makers, shaping physician beliefs under the guise of "education"
- **Academic researchers**, whose careers and grants often depend on aligning with industry narratives
- **Marketing agencies and media outlets**, who amplify fear and simplify complex science into slogans

This isn't just a conspiracy—it's a business model.

A self-sustaining ecosystem built on invented diagnoses, fear-based messaging, and lifelong treatment.

A pharmaceutical cartel wrapped in the language of science.

And the result?

Healthy women labeled as sick.

Entire populations placed on expensive, risky medications for conditions they may never develop.

V. The Final Verdict

Both the cholesterol and osteoporosis paradigms were built on:

- Arbitrary thresholds masquerading as science
- Surrogate markers treated as clinical endpoints
- Exaggerated relative risks used to fuel fear
- Industry-funded guidelines disguised as medical consensus
- Legitimate risks from drugs minimized or ignored
- Dissenting voices silenced or discredited
- Mistakes reinforced, not corrected—doubling down instead of course-correcting

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Epilogue: Why This Matters

This isn't just a critique of two conditions.

It's a **call to re-examine** all of medicine.

If **T-scores** and **LDL** could be used to build entire medical empires—

What else has been built on sand?

It's time we start asking:

"What is the truth—and where has it been buried?"