

Seed Oils Linked to Early 20th Century Heart Disease Surge

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STORY AT-A-GLANCE

- › My paper, "Seed Oils as a Hypothesized Contributor to Heart Disease: A Narrative Synthesis," explains that heart disease was rare before the 20th century and surged only after industrial seed oils became a dominant part of the food supply, pointing to a long-term dietary driver rather than sudden biological failure
- › Linoleic acid (LA) from seed oils accumulates in your tissues and oxidizes easily, which may create inflammatory damage inside arteries that may build silently for decades before symptoms appear
- › The rise in seed oil consumption preceded the explosion in heart disease by 10 to 20 years, matching the slow timeline of plaque formation inside blood vessels
- › Even if you avoid seed oils at home, LA remains embedded in packaged foods and restaurant meals, resulting in constant exposure that may keep arterial damage ongoing
- › Tracking and reducing LA intake may help shift heart disease from an inevitable outcome of aging into a long-term process you can influence

Heart disease feels like a permanent feature of modern life, but it wasn't always that way. In the late 1800s, coronary heart disease was uncommon, and most people died from infections rather than chronic vascular problems. Today, coronary heart disease sits at the center of cardiovascular mortality, bringing with it chest pain, breathlessness, fatigue, and sudden heart attacks that often appear after years of silent damage.

That contrast alone raises a basic question you deserve an honest answer to: What fundamentally changed? The usual explanations focus on longer lifespans, better diagnostics, or individual behavior. I don't find those answers sufficient.

When I examined long-term mortality data, one pattern stood out: Something changed the internal environment of human arteries long before heart attacks became common. One change stands out because it happened quickly, affected nearly everyone, and reshaped what people ate every single day.

My paper, "Seed Oils as a Hypothesized Contributor to Heart Disease: A Narrative Synthesis," published in the journal *Cureus* on January 21, 2026, explains why the widespread adoption of industrial seed oils deserves closer scrutiny.¹

It synthesizes over 200 references showing that the rapid adoption of LA-rich industrial seed oils in the early 1900s preceded the surge in coronary heart disease deaths by 10 to 20 years — the exact timeframe needed for atherosclerotic plaques to develop — and that LA oxidation generates the same inflammatory aldehydes like 4-HNE now implicated in oxidative damage to the lining of blood vessels.

Coronary heart disease doesn't begin with a heart attack. It begins quietly, with changes inside blood vessels that build year after year. To understand why heart disease became so widespread — and how you can change your own trajectory — you need to see how one dietary shift altered the internal environment of your arteries over time. I break down that evidence step by step in my paper, which you can read in full below.



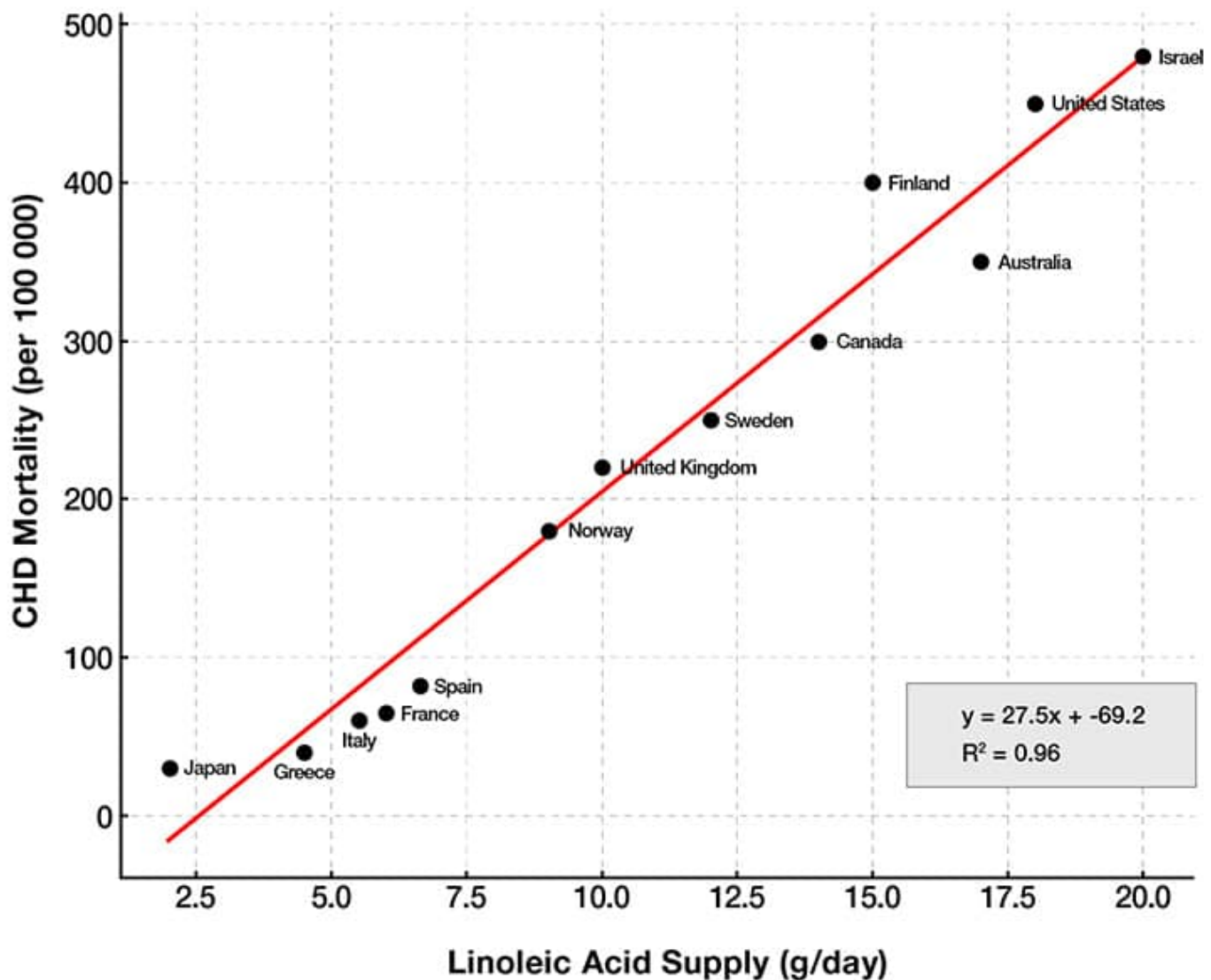
You can also download a simplified version of this paper, rewritten in layman-friendly terms for easier understanding of the science.



How Seed Oils Quietly Reshaped Heart Disease Risk

Here's what the data revealed when I traced the timeline. My paper set out to identify what changed before **heart disease** rates surged in the early 20th century. Instead of focusing on short-term risk markers, I analyzed historical mortality records, U.S. Department of Agriculture (USDA) food supply data, and decades of mechanistic research to align dietary shifts with the slow biology of artery disease.

Cross-National Correlation Between Linoleic Acid Supply and CHD Mortality



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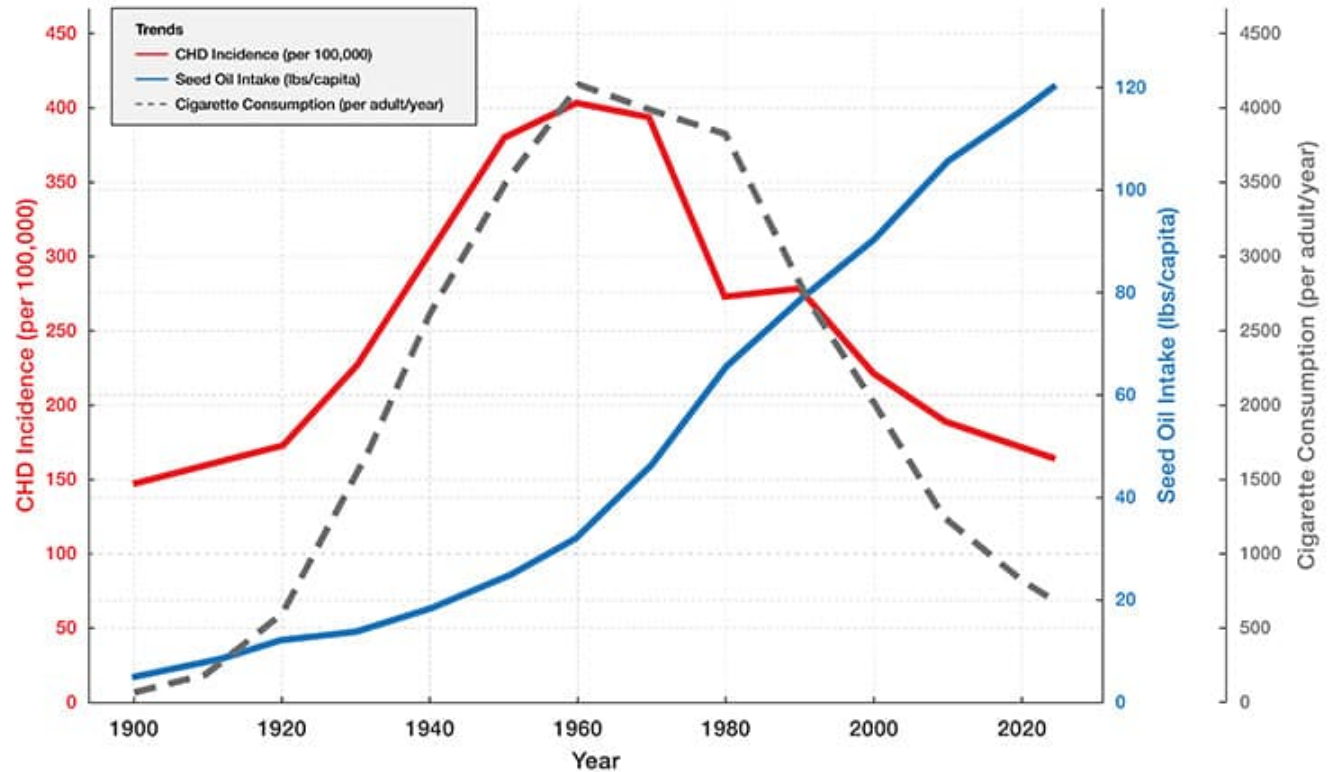
- **A clear dose-response pattern emerged across countries** — As shown in the figure above, countries with higher per-capita **linoleic acid** (LA) availability consistently showed higher rates of coronary heart disease during the mid-20th century. Nations consuming the most LA, including the U.S. and Israel, experienced the highest coronary heart disease mortality, while countries that maintained traditional low-LA diets, such as Japan and Greece, had dramatically lower rates.

This population-level pattern reinforces the sequence seen over time: increased seed oil exposure came first, followed years later by rising heart disease. Although this type of ecological comparison can't prove cause on its own, the uniform gradient across very different countries highlights how sustained LA exposure aligns with long-term arterial risk when an entire food system changes at once.

- **Industrial diets tracked rising heart disease deaths** — As seed oils replaced traditional fats, coronary heart disease mortality climbed from about 137 deaths per 100,000 adults in 1900 to more than 450 per 100,000 by 1968.^{2,3,4} Smoking, longer lifespans, and improved diagnostics were considered, yet none aligned as closely in timing or magnitude with this rise.

As shown in the figure below, U.S. trends from 1900 to 2025 show a parallel rise in coronary heart disease incidence, seed oil intake, and cigarette consumption, with heart disease peaking in the mid-20th century as industrial seed oils became widely adopted. While smoking later declined and heart disease rates fell, seed oil intake continued to climb, highlighting a long-term dietary shift that aligns with changes in cardiovascular risk at the population level.

U.S. Seed Oil Intake, Cigarette Consumption and CHD Incidence (1900-2025)



Sources: USDA-ERS, CDC, Olmsted/Framingham, NHDS/NIS. 2025 is a projected value.

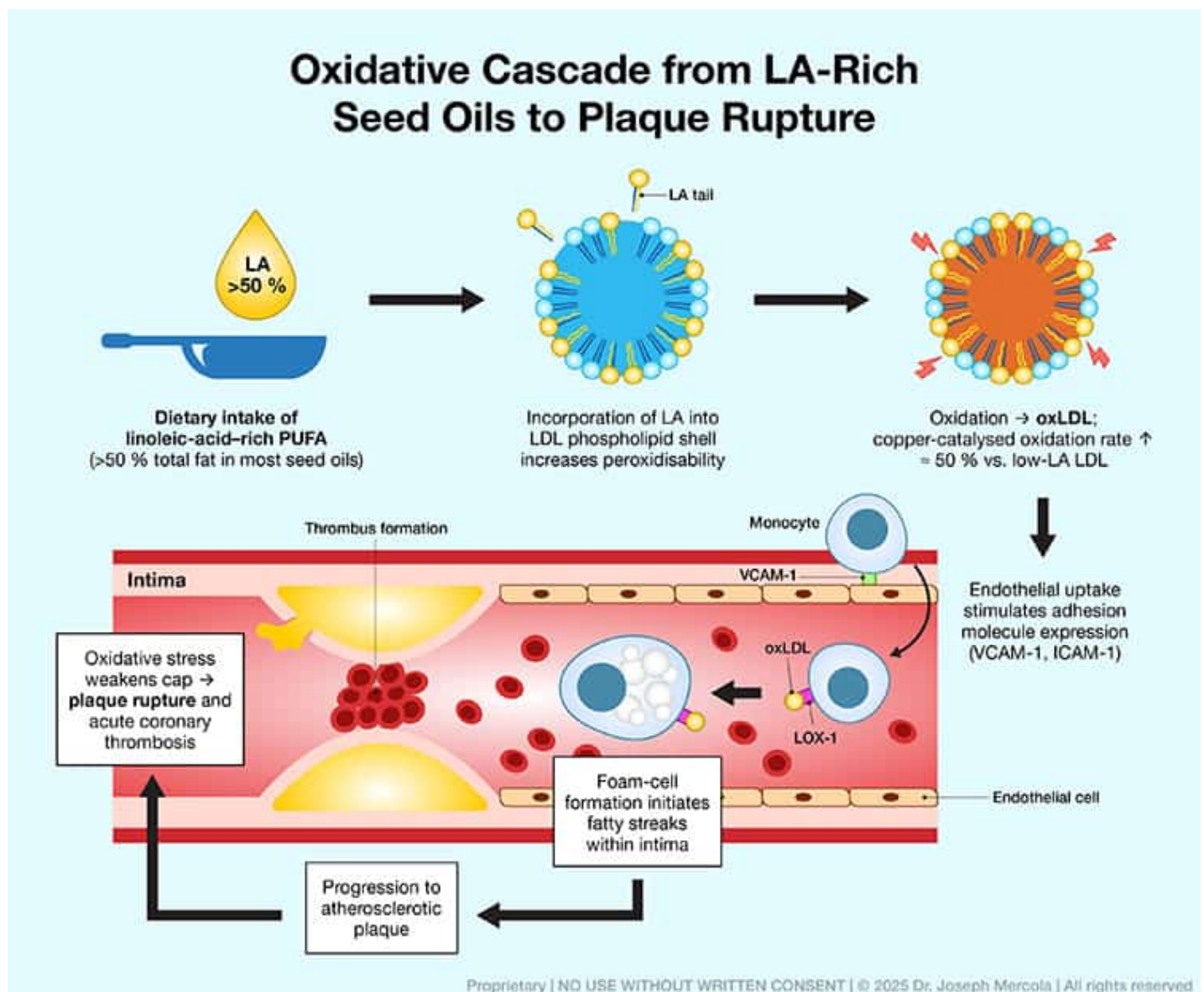
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- **Seed oil exposure accelerated rapidly** — Intake didn't increase gradually. Per-capita seed oil availability doubled within a few decades, rising from roughly 2.7 kilograms (about 6 pounds) to more than 5.4 kilograms (about 12 pounds) per person. Soybean oil drove much of this shift, expanding from near zero in 1909 to more than 11 kilograms (about 24 pounds) per person per year by the late 20th century.
- **Early adopters showed earlier disease** — Urban and industrialized populations that integrated seed oils sooner experienced earlier and higher heart disease rates. Autopsy data from mid-20th-century soldiers even revealed advanced plaque in young adults where it had once been rare. These weren't elderly patients with decades of decline — these were young men in their 20s, already showing the arterial damage of much older people.

Human Tissue Composition Shifted with the Food Supply

But the story doesn't end with food supply data — it shows up in human tissue itself. The research documented a 136% increase in LA stored in body fat during the same period. This means that compared to your great-grandparents, your fat tissue today contains more than twice as much of this unstable, inflammatory fat.

The primary damage wasn't reflected in cholesterol or blood pressure changes. LA oxidizes easily, breaking down into reactive compounds that directly injure blood vessels. These oxidized fragments are chemically "sticky" — they attach to proteins and DNA in ways that trigger immune responses and damage cells. Think of oxidation like rust forming on metal. When LA oxidizes inside your body, it essentially "rusts," creating damaged molecules that irritate and injure your artery walls.



- **Toxic lipid byproducts fueled plaque formation** — As shown in the figure above, fats from LA-rich seed oils become incorporated into circulating LDL particles, where they are far more prone to oxidative damage than more stable fats. Once oxidized, these LDL particles irritate the inner lining of your arteries, switching on inflammatory signals that draw immune cells into the vessel wall.

Those cells absorb the damaged cholesterol and swell into "foam cells," forming the earliest layers of plaque. As oxidized fats continue to accumulate, the plaque grows, the artery wall stiffens, and the protective cap over the plaque weakens, raising the risk of sudden rupture and clot formation over time.

- **Slow plaque growth hid the damage for decades** — Atherosclerosis typically takes 10 to 20 years to become clinically visible. Seed oil intake rose well before heart attacks and sudden cardiac deaths became common, matching the known timeline of arterial injury.
- **Other risk factors amplified but did not initiate disease** — Smoking intensified oxidative injury but did not start the process. Improved diagnostics explained detection, not disease creation. Refined sugar worsened metabolism, yet its rise didn't match the early heart disease inflection.
- **Inflammation kept artery damage switched on** — When seed oil fats break down inside your arteries, they essentially trick your immune cells into treating damaged cholesterol as a threat to engulf — but the cells can't digest it.

Those cells start soaking up damaged cholesterol and turn into bloated "foam cells," which are the first building blocks of artery plaque. At the same time, inflammatory signals stay active and allow injured cells to linger instead of clearing out, letting plaque grow and harden over time.

- **Modern eaters inherit decades of LA exposure** — Even if you don't cook with seed oils, LA is baked into the modern food supply through packaged foods and restaurant meals. That constant exposure supplies the raw material for ongoing

oxidative injury inside your arteries. Once you can see and measure your LA intake, heart disease stops looking like fate and starts looking like what it is: a slow injury you can prevent.

How to Address the Real Problem

If you take one thing from this research, let it be this: Heart disease did not surge because human biology suddenly failed. I look at it as a slow injury driven by a modern fat intake your body wasn't designed to handle. When you lower the oxidative burden on your arteries, you may help support arterial stability and reduce further damage accumulation year after year. This section focuses on removing the root driver first, not masking downstream symptoms.

1. Cap your LA intake in the 2 to 3 gram range per day — This is the foundation.

Excess LA from seed oils loads your tissues with highly unstable fats that break down into inflammatory byproducts. Focus on keeping daily intake between 2 and 3 grams because that aligns far more closely with pre-industrial exposure. (Going much lower than 2 grams a day also isn't recommended, as your mitochondria still requires LA in small amounts.)

To put that in perspective, one tablespoon of soybean oil contains about 7 grams of LA — more than double your daily limit. A single serving of potato chips fried in vegetable oil can contain 3 to 5 grams.

To significantly lower your LA exposure, eliminate soybean, corn, sunflower, safflower, canola, and cottonseed oils entirely. Once these oils are gone, you sharply reduce the raw material that may contribute to arterial inflammation and plaque formation.

2. Track your LA intake so progress becomes visible — Invisible problems stay unsolved. I recommend tracking LA intake directly rather than guessing. To do so, sign up for my Pax health platform. It has a feature called the Seed Oil Sleuth, which

monitors your LA intake to a tenth of a gram. That kind of feedback builds confidence and consistency. You see exactly how daily choices affect long-term risk, which makes the changes stick.

- 3. Stop relying on restaurant food and fried meals** — If you eat out often, this step matters. Restaurants almost universally rely on seed oils because they're cheap and shelf-stable. Fried foods and sautéed dishes expose those oils to high heat, which accelerates oxidation before the food even reaches you. When you cook at home, you regain control over the fats entering your bloodstream, and that may help lower oxidative stress inside your arteries.
- 4. Replace seed oils with stable traditional fats** — I recommend you cook with grass fed butter, ghee, or tallow. These fats resist oxidation and do not fragment into toxic byproducts under heat. When you switch fats, you're not just avoiding harm — you're helping support a calmer biochemical environment inside your blood vessels.
- 5. Avoid high-LA animal foods like chicken and pork** — This step surprises many people. These animals store dietary LA directly in their tissues. That means you absorb it secondhand. Choose grass fed beef or lamb instead, which naturally contain far lower LA levels. This swap lowers your exposure without forcing you to reduce protein intake or calories. For instance, conventional chicken thighs contain about 2 to 3 grams of LA per serving, while grass fed beef contains roughly 0.1 to 0.2 grams.

When you address the cause — unstable industrial fats accumulating in your tissues — you stop fighting your biology and start working with it. Over time, that shift may change how your arteries respond to stress, inflammation, and aging itself.

Because LA accumulates in tissues over years, reducing intake is a long-term strategy. Human trial data on specific timelines for improvement are limited, so individual results are likely to vary, with any benefit to inflammatory markers potentially accumulating gradually over years as tissue LA stores deplete.

FAQs About Seed Oils and Heart Disease

Q: Why weren't seed oils a problem in the past?

A: Seed oils were not widely consumed before the early 1900s. Traditional diets relied on butter, tallow, and lard, which are far more stable fats. The rapid industrial introduction of seed oils dramatically increased LA exposure in a very short period, creating conditions the human body hadn't adapted to over evolutionary time.

For most of human history, LA intake was estimated at 1% to 2% of calories. By the late 20th century, it had risen to 7% to 8% — a change that happened in decades rather than the millennia required for biological adaptation.

Q: What makes LA from seed oils harmful to arteries?

A: LA is highly unstable. When it breaks down, it forms reactive compounds that may damage blood vessel walls, keep inflammation switched on, and may accelerate plaque buildup. Over decades, this ongoing injury is thought to alter the structure and function of arteries in ways that may raise heart disease risk.

Q: If I don't cook with seed oils, am I still exposed?

A: Yes. LA is embedded in the modern food supply. Packaged foods, restaurant meals, sauces, dressings, and fried foods almost always rely on seed oils. Even without using them at home, daily exposure adds up unless intake is intentionally reduced.

Q: How does reducing LA help heart health over time?

A: Lowering LA intake may reduce the raw material that contributes to oxidative damage inside arteries. Over time, some research suggests this may support a calmer internal environment, though evidence that it meaningfully slows plaque accumulation or restores vascular resilience in humans is still preliminary.

Q: What's the most effective first step to reduce risk?

A: The most effective first step is tracking and limiting LA intake to between 2 and 3 grams per day. Once intake becomes visible and measurable, heart disease may feel less inevitable and more like something you can actively influence by reducing chronic exposure rather than chasing symptoms.

This article is for informational purposes only and does not constitute medical advice. Consult a qualified healthcare provider before making changes to your health regimen.

Sources and References

- ¹ [Cureus 18\(1\): e102012](#)
- ^{2, 3} [MMWR Surveill Summ 2018;67\(No. SS-5\):1–11](#)
- ⁴ [Nutr Rev. 2025;83\(7\):e2106-e2112](#)