

Animal Production and Human Health — A Synthesis from Paleoanthropology, Biochemistry, Nutritional Epidemiology, and Pathogen Research

Science Spin-off to the Working Paper ‘Animal, Human, Industry’ v0.2

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Preface to the Spin-off

This paper is a standalone extraction of the natural-scientific-medical part of the larger Working Paper “Animal, Human, Industry” v0.2. It treats Layers 1 through 8 of that paper — paleoanthropology, comparative anatomy, biochemistry, genetics, nutritional epidemiology, the dairy complex, pathogens, and industrial political economy — as well as the v0.2 extension Layer 13 on aquaculture and fisheries.

What is *deliberately* absent: the ethical arguments (Layer 11), the marxist synthesis (Layer 12), the taboo anthropology (Layers 9–10), and the alternative pathways (Layer 14). Whoever seeks these will find them in the Master Paper and in the Marxism Spin-off.

This omission has methodological reasoning. In a specialist audience for public health and nutritional science, ethical and political-economic arguments fall onto different ground than they do among animal-ethics or social-philosophy readers. A *separate* address of these audiences allows more precise methodological concentration in each. The Master Paper remains the locus of the transdisciplinary overall synthesis.

The natural-scientific evidence chain taken on its own is strong enough to bear a standalone statement — even without the ethical-economic extensions. Demonstrating this is the point of the spin-off.

Preliminary Note: Mini-ETHOSLAB Pre-Examination

Following the refined Memory Rule 14 (companion-paper duty, refined 2026-05-19), every standalone deliverable must include the methodological self-examination. For the Science Spin-off:

ME-1 Research subject: Convergence of the natural-scientific and medical evidence on the health effects of industrial animal-product production.

ME-2 Conflicts of interest: No institutional affiliation, no third-party funding, no industry cooperation. The researcher’s own marxist-materialist preformation is declared in the Master Paper; in the present spin-off it is methodologically minimized by the exclusively natural-scientific focus.

ME-3 Harm potential: Medium. Plant-emphasizing dietary recommendations are not universalizable in subsistence, class, and indigeneity contexts — see Layer 12.5b in the Master Paper. The spin-off therefore contains in the closing section an explicit subsistence proviso.

ME-4 Methodological risks: Confirmation bias in source selection; mitigated through explicit source-critique annexes (Master Paper). Industry-funded counter-evidence (PURE, EAT-Lancet

critics) is systematically treated in the Master Paper; the spin-off refers to that treatment rather than reproducing it in shortened form.

ME-5 Hansson-oriented nine-point self-check: Falsifiability, methodological appropriateness, data verifiability, competing hypotheses, effect sizes, replication status, declaration of own position, reproducibility, HARKing avoidance — all nine points are documented in the Master Paper for the underlying layers.

ME-6 Release: The spin-off is released under the conditions that (i) the natural-scientific focus is not read as an implicit claim of absolute universality, (ii) references to the Master Paper for ethical, political-economic, and subsistence-boundary arguments are consistently maintained, (iii) the Companion Paper methodology framework can be consulted when needed.

ME-7 Reference to MYZEL v0.3 instead of own mini knot set: Under refined Memory Rule 14, *standalone theoretical contributions* must include their own mini-MYZEL knot set. This spin-off is methodologically *not a standalone theoretical contribution*, but an *extraction* from the Master corpus for a specific audience. The underlying definitions, claims, and bridges are fully documented in the MYZEL knot set v0.3 (22 definitions, 50 claims, 40 bridges). Relevant for this spin-off are the claims of the layers anthropology, anatomy, genetics, biochemistry, nutrition, dairy, pathogens, industry, and aquaculture (see MYZEL v0.3 convergence analysis). An own mini knot set would only replicate a subset of the corpus and would be methodologically redundant.

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Part I — Natural History and the Body

1. Evolutionary Anthropology: What Was the Human?

The popular narrative “meat made the brain” has shaped the discourse on human dietary history for decades. It traces back to research of the 1960s and 1970s that reconstructed a connection between hominization, tool use, and meat consumption as a causal cascade.

The recent re-analysis of paleoanthropological data has substantially weakened this cascade methodologically. Barr et al. 2022 (*PNAS*, doi:10.1073/pnas.2115540119) show through systematic processing of the East African finds inventory that the temporal correlation between cut-mark traces on bones and brain enlargement is less tight than assumed — the correlation may be an artifact of increased sampling intensity in the younger strata. It remains a *correlation*, not a causally established chain.

Hardy et al. 2015 (*Quarterly Review of Biology*, doi:10.1086/682587) argue in parallel for the central role of starch and root food in hominization — supported by the demonstrable AMY1 gene

copy-number expansion in humans over the past roughly two million years, which supports a plant-emphasizing slant in the Stone Age diet.

What is not tenable are three popular speculations: the Stone Ape hypothesis (McKenna 1992) with psilocybin as driver of hominization — no peer-reviewed support (Rodríguez-Arce & Winkelman 2021, doi:10.3389/fpsyg.2021.729425); the Aquatic Ape hypothesis (Hardy 1960, Morgan); the Raw Primal Diet hypotheses (Vonderplanitz). All three are treated in serious paleoanthropology as methodologically insufficiently supported.

Interim state: The human did not become human primarily through meat consumption. The human became human through tool use, fire, social cooperation, and an expanded food spectrum (which *included* meat without meat being causally dominant).

2. Comparative Anatomy: What Can the Human Digest?

Comparative anatomy provides a tool: the ratio of small to large intestine, the shape of teeth, the pH values of the stomach, and the enzymatic equipment allow inferences about the evolutionarily compatible food spectrum.

Compared to strict carnivores (lion, wolf): the human has a substantially longer intestinal tract relative to body size, lower stomach pH (human ~1.5–3.0 fasting; lion ~1.0–1.5; dog ~1.5), less pronounced canine teeth, no specialized claws. Compared to strict herbivores (cow, horse): the human has no rumen, no horse-dimension cecum fermentation, no pronounced grinding-jaw apparatus.

The human is anatomically an omnivore with a recognizable plant-frugivorous slant. This position is elaborated differentially in several overview works (Milton 1999, *Nutrition* 15(6):488-498; Wrangham & Conklin-Brittain 2003).

Fungi and algae expand the plant spectrum considerably. Mycoprotein (commercially as *Quorn*, fermented from *Fusarium venenatum*) has shown LDL-lowering effects and postprandial insulin improvements in RCTs (Coelho et al. 2021, *Current Developments in Nutrition*; Bottin et al. 2016, *British Journal of Nutrition* 116(2):360–374, doi:10.1017/S0007114516001872). Algal DHA (heterotrophic *Schizochytrium* production) resolves the DHA conversion question without the detour through fish.

DHA conversion: Plant sources provide ALA, which the body converts to DHA with marked sex dependency — typically under 8 percent in men, 9–21 percent in women (Burdge & Calder 2005, doi:10.1051/rnd:2005047). The solution in industrial societies is not necessarily fish but algal DHA (Lane et al. 2014, doi:10.3390/nu6116402).

3. Biochemistry: What Do Animal Products Do in the Body?

High consumption of animal products leads biochemically to several parallel running burden pathways. These are established through independent mechanisms; their effects add up.

TMAO (Trimethylamine-N-oxide): L-carnitine and choline, both abundant in red meat and eggs, are metabolized by gut bacteria to trimethylamine (TMA); the liver oxidizes TMA to TMAO. Plasma TMAO levels correlate in several large cohorts with elevated cardiovascular risk (Wang et al. 2011, *Nature*, doi:10.1038/nature09922; Tang et al. 2013, *NEJM*, doi:10.1056/NEJMoa1109400). Heianza et al. 2017 (*Journal of the American Heart Association*, doi:10.1161/JAHA.116.004947) report in a meta-analysis of 19 studies a 23 percent increase in cardiovascular risk per 10-μmol/L TMAO increase (confidence intervals to be checked in the original study).

Neu5Gc (N-glycolylneuraminic acid): A sialic acid evolutionarily lost in humans but abundant in red meat and dairy products. Human antibody reaction against Neu5Gc is linked to chronic inflammation and elevated cancer risk (Samraj et al. 2015, *PNAS*, doi:10.1073/pnas.1417508112; Padler-Karavani et al. 2008).

IGF-1 (Insulin-like Growth Factor 1): Dairy and meat consumption elevates plasma IGF-1 levels; high IGF-1 is associated with elevated risk of several cancer types (prostate, breast) (Levine et al. 2014, *Cell Metabolism*, doi:10.1016/j.cmet.2014.02.006). The biological pathway runs through mTOR activation.

Heme iron: In contrast to non-heme iron from plant sources, heme iron is *absorbed uncontrolled*. High heme-iron levels are associated with type-2 diabetes, cardiovascular risk, and colorectal cancer (Bao et al. 2012; Fonseca-Nunes et al. 2014).

AGEs (Advanced Glycation End-products): Frying and grilling of animal products generates abundant AGEs; they are associated with accelerated aging, cardiovascular pathologies, and insulin resistance (Uribarri et al. 2010).

Postprandial endotoxemia: Fat-rich animal-product meals briefly elevate plasma LPS levels (lipopolysaccharides from gram-negative gut bacteria), triggering a subclinical inflammatory response (Erridge et al. 2007).

None of these mechanisms is alone “the killer” — they operate in parallel and cumulatively. The nutritional-epidemiological observation of elevated mortality rates with high animal-product consumption is mechanistically plausibilized by this biochemical mechanics, not merely statistically correlated.

4. Genetic Adaptations: What Has Changed Since the Neolithic?

AMY1 (Alpha-Amylase 1): Human genome copy number for AMY1, the salivary enzyme for starch breakdown, is elevated by about a factor of 6 compared to chimpanzees. This expansion is Holocene — it follows the transition to agriculture, not the reverse (Perry et al. 2007, *Nature Genetics*). This supports the Hardy hypothesis of plant-emphasizing starch adaptation.

FADS gene cluster: The efficiency of ALA-to-DHA conversion is genetically variable; certain allele variants (FADS1/2) allow higher conversion rates. These variants are more frequent in populations with long plant-emphasizing tradition (South Asia, the Mediterranean); in Arctic populations they are rarer because DHA was directly available from fish (Buckley et al. 2017).

ApoE: Three main allele variants (E2, E3, E4) modulate cardiovascular risk under animal-fat consumption considerably. ApoE4 carriers are particularly sensitive to saturated animal fats (Minihane 2016).

Lactase persistence: The mutation maintaining lactase activity into adulthood is globally not the norm. Estimated 35 percent of adult humanity is lactase-persistent — primarily in Northern and Central Europe, West Africa, and parts of the Near East (Itan et al. 2010). In East Asia, Southeast Asia, and Sub-Saharan Africa, lactase persistence is rare or absent. The culturally universalized dairy norm of Western dietary recommendations does not reflect global biological reality.

Part II — Science, Industry, and Aquaculture

5. Nutritional Epidemiology and Mendelian Randomization

The nutritional-epidemiological evidence rests on large prospective cohorts and (increasingly) Mendelian randomization studies.

Large cohorts: - EPIC-Oxford (UK, n>65,000): Tong et al. 2019 (*BMJ*, doi:10.1136/bmj.l4897) — lower CHD incidence in vegetarians and vegans, but slightly higher stroke incidence; several companion and follow-up publications. - AHS-2 (USA, n>96,000): Orlich et al. 2013 (*JAMA Internal Medicine*) — lower all-cause mortality in pesco-, lacto-ovo-, and vegan subgroups. - NHANES,

Nurses Health Study, Health Professionals Follow-Up Study: convergent findings under careful adjustment.

Known weaknesses of all cohort studies on nutrition: healthy-user bias (vegetarians are systematically better educated, more athletic, non-smoking), confounding problems, self-report data on diet with substantial measurement error.

Mendelian randomization overcomes part of these limitations. It uses genetic variants as instrumental variables — whoever has higher LDL at birth has it lifelong, independently of lifestyle. The central MR study on LDL and CVD is Ference et al. 2012, *Journal of the American College of Cardiology* 60(25):2631–2639, doi:10.1016/j.jacc.2012.09.017: 9 polymorphisms in 6 genes show convergently a risk-relation of lifelong LDL exposure to coronary events. Order of magnitude of the effect reduction with lifelong LDL lowering: substantially higher than with short-term statin interventions.

The conclusion: LDL is not merely a marker but the *causal* driver of atherosclerosis. Saturated animal fats elevate LDL, established (Mensink WHO 2016 commissioned analysis). Thus the nutritional-policy consequence is mendelically validated — not merely correlatively suspected.

For red meat and type-2 diabetes, the observational evidence is strong (federated meta-analysis of 1.97 million adults, *Lancet Diabetes & Endocrinology* 2024, doi:10.1016/S2213-8587(24)00179-7), while the Mendelian-randomization findings are mixed.

Limitations of MR: pleiotropy, linearity assumption, population stratification. Nonetheless, the LDL MR is one of the most robust causal findings in modern medical epidemiology.

Intervention studies: Ornish et al. 1998 (Lifestyle Heart Trial, *JAMA* 280(23):2001–2007, doi:10.1001/jama.280.23.2001) and Esselstyn 2014 (*Journal of Family Practice*) show in small controlled studies angiographically visible atherosclerosis regression on strictly plant-based diet. Methodologically limited by n, self-selection, compliance — but they show mechanism.

6. The Dairy Complex

The dairy industry in the 20th century substantially shaped the nutritional recommendations. For instance: USDA Food Pyramid 1992, ÖGE pyramid, DGE recommendations.

The recent evidence base:

- Feskanich et al. 2014, *JAMA Pediatrics* 168(1):54–60, doi:10.1001/jamapediatrics.2013.3821 — Nurses Health Study, Health Professionals Follow-Up: no protection against hip fractures by high dairy consumption in adults.
- Michaëlsson et al. 2014, *BMJ*, doi:10.1136/bmj.g6015 — two Swedish cohorts, $n \approx 100,000$: higher milk intake associated with higher all-cause mortality and fracture rate in women (HR 1.93 at >3 glasses milk/day vs. <1; 95% CI 1.80–2.06). Suspected mechanism: D-galactose, a milk-sugar breakdown product, induces oxidative stress in animal models.

The global lactase persistence of about 35 percent makes the universalized dairy norm biologically implausible. In Asia, Africa, and Latin America, adult lactase activity is the exception, not the rule.

The “Milk Makes Tired Men Lively” campaign of the CMA (*Centrale Marketing-Gesellschaft der deutschen Agrarwirtschaft*) ran 56 years and was financed by a legally enforced compulsory levy of farmers — stopped 2009 by the German Federal Constitutional Court as unconstitutional (BVerfG ruling on the *Absatzfondsgesetz* of 3 February 2009; Az. 2 BvL 54/06). The marketing of the dairy industry was a state-supported construct over decades.

7. Pathogens, Pandemics, Antimicrobial Resistance

Industrial animal husbandry is epidemiologically a third-order risk space: concentrated animal density, high genetic uniformity, ongoing antibiotic use, dense human-animal contact zones.

Zoonotic pandemic risks: H5N1, H7N9, H1N1 2009. The H1N1 2009 pandemic origin was popularly traced back to a specific Smithfield subsidiary in Veracruz, Mexico (Granjas Carroll); scientific phylogenetic analysis (Smith et al. 2009, *Nature*, doi:10.1038/nature08182) does not directly support the media narrowing, instead identifying multiple reassortment events in swine populations over several years. What remains structurally: industrial swine husbandry as incubation space.

BSE and vCJD: 1986–2000 British cattle BSE epidemic through animal-meal feeding; 178 documented vCJD cases in the UK (UK National CJD Research and Surveillance Unit; no new cases in the past eight years). The incubation time can exceed five decades (up to 56 years, Collinge et al. 2006).

Antimicrobial resistance (AMR): Over two-thirds of globally produced antibiotics go into animal husbandry. Murray et al. 2022, *Lancet*, doi:10.1016/S0140-6736(21)02724-0 — directly AMR-attributable deaths worldwide for 2019: approximately 1.27 million; additionally approximately 4.95 million deaths with AMR as contributing factor. AMR is one of the central public-health crises of the 21st century — and industrial animal husbandry is one of its main drivers.

8. Industrial Animal Production as a Public-Health Factor

Summarizing, the public-health effects of industrial animal production can be sorted into four dimensions:

(1) Individual mortality and morbidity: Elevated risk of cardiovascular disease, type-2 diabetes, several cancer types — through the biochemical mechanisms documented in Layer 3, epidemiologically confirmed in Layer 5, mendelically established as causal for LDL in Layer 5.

(2) Pandemic risks: Structural risk of zoonotic diseases through industrial animal husbandry. Not every pandemic comes from animal production (COVID-19 is contested), but several historical and current pandemics demonstrably emerged from industrial animal-husbandry complexes.

(3) Antimicrobial resistance: Directly quantifiable public-health burden in order of magnitude one to five million deaths per year — depending on direct attribution vs. association.

(4) Climate effect as public-health mediator: Approximately 14–20% of global greenhouse-gas emissions originate from livestock (FAO 2023, Xu et al. 2021 *Nature Food*); these mediate climate-induced public-health consequences (heat waves, vector spread, food insecurity, migration). This is not the central argument of the spin-off, but it is part of the public-health balance.

JBS, Tönnies, Cargill, Smithfield, Tyson — the largest animal-production corporations have annual turnover in tens of billions USD and corresponding political levers. Joesley Batista, JBS owner, publicly admitted in 2017 under plea-bargain agreement to the bribery of a large number of Brazilian politicians (his own statement: over 1,800 office-holders), including then-President Michel Temer. Ag-gag laws in several U.S. states criminalize filming in animal-husbandry operations — *Animal Legal Defense Fund v. Otter*, 44 F.Supp.3d 1009 (D. Idaho 2014), declared Idaho’s ag-gag law unconstitutional.

The public-health position cannot hide behind the claim that this is “merely politics.” The structural mechanisms undermining public-health recommendations are themselves public-health relevant.

9. (□ Layer 13 Master) Aquaculture and Fisheries as CAFO Extension

When wild fish stocks shrink, aquaculture expands. It has been for decades the fastest-growing segment of animal production. 2022 for the first time: more fish from aquaculture than from wild capture.

Aquaculture reproduces the structural public-health problems of land-based CAFOs underwater:

- **Antibiotic use:** Chilean salmon has held over years the global top value for antibiotic use per ton of produced salmon — orders of magnitude above Norwegian values (Lozano-Muñoz et al. 2018, *Frontiers in Microbiology* 9:1284, doi:10.3389/fmicb.2018.01284). The AMR burden distributes across marine organisms.
- **Pesticide use:** Cypermethrin, emamectin benzoate against salmon lice; retroactive contamination of marine ecosystems and consumed end products.
- **Mangrove destruction:** A considerable share of global mangrove area has been lost in the last 50 years for shrimp farms (Hamilton & Casey 2016, doi:10.1111/geb.12449) — with consequences for carbon sink and hurricane protection.

Wild capture: FAO 2024 assesses about 38% of stocks as overfished, about 50% as maximally sustainably fished, only about 8% as underfished. Bycatch accounts for up to 40% of global catch volume (Davies et al. 2009, *Marine Policy*, doi:10.1016/j.marpol.2009.01.003); the stricter FAO discard estimate is substantially lower at 10.8% (Pérez Roda et al. 2019, FAO Fisheries and Aquaculture Technical Paper 633) — both definitions are methodologically defensible. Global fishery subsidies in order of magnitude 35 billion USD per year (of these about 22 billion capacity-enhancing) structurally maintain stock depletion (Sumaila et al. 2019, *Marine Policy* 109:103695, doi:10.1016/j.marpol.2019.103695).

Estimates of annually killed fish for human consumption vary substantially because fish are traditionally recorded in tons, not individuals. Mood 2010 (self-published report for fishcount.org.uk, not peer-reviewed) estimates for wild capture about 0.8 to 2.3 trillion individuals per year; aquaculture adds another 50 to 170 billion individuals, trend rising (FAO 2024).

Closing Section — Convergence, Limitations, Open Questions

Convergence finding

From nine independent strands (paleoanthropological, anatomical, biochemical, population-genetic, nutritional-epidemiological, dairy-specific, pathogen-related, industrial-political, aquaculture-specific) emerges a convergent finding: high consumption of industrially produced animal products is associated with documented health and public-health risks whose biological mechanisms are *causally* established via Mendelian randomization — not merely correlatively suspected.

This is a strong statement. It is methodologically supported not by a single study but by the convergence of several independent methods. A single layer can be refuted or revised without tilting the overall picture.

Limitations — Subsistence, class, indigeneity

The recommendation to reduce industrial animal products is *not* universalizable onto:

- **Subsistence pastoralism:** Maasai, Mongolians, Tuareg, Sámi, Quechua in landscapes that are not arable. Here ruminants convert grasses and lichens into usable nutrition where direct plant subsistence is not possible.
- **Smallholder mixed farming** in low-income contexts — two to five head of livestock per household using waste products, producing fertilizer, providing risk diversification.
- **Indigenous food sovereignty:** Inupiat whaling, Caribou hunting, Bison restoration — collective self-determination questions beyond Western public-health recommendation logic.

About 600 million people worldwide depend directly on animal husbandry (FAO 2018). About 1.58 billion people cannot financially afford the EAT-Lancet Planetary Health Diet (Hirvonen et al. 2020, doi:10.1016/S2214-109X(19)30447-4). Public-health recommendations that do not incorporate this reality are not universally valid.

The spin-off therefore explicitly maintains: the critique is directed against *industrial* animal production in industrial societies, not against animal husbandry as such. JBS and Tönnies are the addressees, not the Somali camel-herder.

Open questions

- Full source verification for the DOIs marked with `status-zu-verifizieren` in this spin-off.
- Effect-size update for Ference et al. 2012 and the red-meat/diabetes evidence against original studies.
- Updated vCJD case count.
- Systematic LCA comparison study between aquaculture and CAFO swine/cattle.
- Effect-size quantification of the AMR industrial-livestock attribution (Murray 2022 provides order of magnitude, not attribution specificity).

References to the Master Paper

Whoever seeks the following arguments will find them in the Master Paper “Animal, Human, Industry” v0.2:

- Layers 9–10: affect-taboo layers, Pachirat slaughterhouse ethnography, Aghori anthropology
- Layer 11: animal ethics (Singer, Regan, Korsgaard, Nussbaum, Donaldson/Kymlicka, Adams)
- Layer 12: marxist synthesis with metabolic-rift concept and Wadiwel extension
- Layer 14: alternative pathways (precision fermentation, cultivated meat, algae, fungi)
- Source critique Annexes A–E with industry-source treatment and four-asymmetries analysis
- Companion Paper methodology with fourteen-layer-architecture reflection

Footer

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