

From Fryer to Endothelium: Thermo-Oxidation of Repeatedly Heated Palm Oil, Food-Service Exposure, and Cardiometabolic Risk—A Review

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ABSTRACT

Palm oil and palm olein are widely used for deep frying because of their availability, technological performance, and comparatively high oxidative stability. However, repeated exposure to frying temperatures progressively transforms fresh oil into a chemically different mixture containing hydroperoxides, aldehydes, oxidized triacylglycerols, epoxy fatty acids, polymers, and other polar degradation products. This qualitative literature review critically integrates contemporary evidence published mainly during 2020–2026 concerning the thermo-oxidation of palm-based frying oils, real-world food-service exposure, and potential cardiometabolic consequences. The synthesis indicates that palm oil generally withstands thermal oxidation better than many polyunsaturated oils, but this relative stability does not prevent progressive degradation during prolonged or repeated frying. Temperature, cumulative heating time, food moisture, oxygen exposure, fryer configuration, catalytic metals, food residues, antioxidant depletion, and oil-replenishment practices jointly determine degradation. Lipid oxidation products may enter fried foods or cooking fumes and interact with biological systems through oxidative stress, reactive-carbonyl signaling, oxidized-lipoprotein formation, inflammation, impaired nitric-oxide bioavailability, endothelial dysfunction, and metabolic disturbances. Nevertheless, direct longitudinal human evidence specifically attributing cardiovascular outcomes to repeatedly heated palm oil remains limited. The review proposes a “Fryer-to-Endothelium Continuum” linking processing conditions, chemical transformation, exposure, biological responses, and cardiometabolic outcomes, and identifies priorities for exposure biomarkers, standardized frying protocols, food-service monitoring, vendor interventions, and prospective human research.

Keywords: Palm oil; Palm olein; Repeatedly heated cooking oil; Deep frying; Thermo-oxidation; Lipid oxidation products; Aldehydes; Endothelial dysfunction; Cardiometabolic risk; Food-service environment

JEL Classification: I12; I18; L66; Q18; Q53.

1. INTRODUCTION

Deep frying is one of the most widely used thermal food-processing techniques because it rapidly produces foods with desirable color, crispness, aroma, palatability, and shelf characteristics. During conventional deep frying, food is immersed in oil commonly maintained at approximately 150–190°C, where the oil simultaneously functions as a heat-transfer medium and as an ingredient that becomes partially incorporated into the finished product. These conditions expose triglycerides and minor constituents to heat, atmospheric oxygen, water released from food, catalytic metals, food residues, and repeated cooling–reheating cycles. Consequently, frying oil is not chemically static. Hydrolysis, oxidation, polymerization, cyclization, isomerization, and interactions with food progressively alter the original lipid matrix.¹⁻³

Palm oil and especially palm olein occupy an important position among commercial frying media. Their widespread use reflects not only economic availability in palm-producing and importing regions but also functional characteristics such as favorable frying performance, relatively moderate polyunsaturated fatty-acid content, and greater oxidative stability than many linoleic-rich oils. Experimental comparisons have repeatedly shown that palm-based oils may experience slower deterioration than more highly unsaturated alternatives under comparable thermal conditions, although the magnitude of this advantage depends on the exact oil fraction, processing history, antioxidant content, frying temperature, and food system.⁴⁻⁶ Comparative stability therefore needs to be distinguished from absolute resistance to degradation.

This distinction is central to understanding the health relevance of repeatedly heated palm oil. Discussion of palm oil and health has traditionally focused on fatty-acid composition, especially its content of palmitic acid and the broader debate regarding saturated fat and cardiovascular risk. That approach is insufficient when evaluating oil that has been subjected to prolonged or repeated deep frying. Fresh palm oil and oil used repeatedly at 170–190°C are no longer chemically identical exposures. Repeated heating can reduce endogenous antioxidant protection while increasing free fatty acids, peroxide decomposition products, aldehydes, oxidized triglycerides, total polar compounds (TPC), polymers, and other secondary products.⁷⁻⁸ Recent work directly examining repeated heating of palm oil and its fractions similarly demonstrates progressive increases in indicators such as free fatty acids, peroxide value, and p-anisidine value. Thus, the public-health question must extend beyond the fatty-acid profile of the original oil toward the chemical consequences of the frying process itself.

Palm oil illustrates an important paradox. A relatively low content of highly oxidation-prone polyunsaturated fatty acids can confer useful thermal stability, but the oil remains susceptible to deterioration with sufficient thermal load.⁹ For example, [1] found that during prolonged deep frying, palm oil accumulated substantial total polar compounds and aldehydes even though its oxidation profile differed from high-oleic sunflower oil. Palm-specific industrial frying studies likewise demonstrate increases in p-anisidine values and harmful volatile compounds over time.⁶ Repeated-frying studies involving palm oil show increases in physicochemical deterioration indices as the number of cycles rises.⁸ Consequently, the correct interpretation is not that palm oil

is uniquely unstable but that no edible oil should be presumed indefinitely stable under severe repeated thermal exposure.

The issue acquires greater public-health importance when frying practices in real food-service environments are considered. Restaurants, informal street vendors, institutional kitchens, cafeterias, small food enterprises, and households often face economic incentives to maximize oil life. Decisions to replace oil may be based on color, foaming, smell, viscosity, smoking, or subjective experience instead of quantitative measurements. Oil may be topped up with fresh oil rather than completely replaced, stored overnight, reheated the following day, or exposed to residues that accelerate deterioration. Field studies in Uganda, Ethiopia, Ghana, and other settings have documented substantial deterioration of reused oils and considerable variation associated with equipment, fuel source, reuse duration, and food-handling practices.^{1,10,11,12} In Kampala, for example, a large proportion of vendor oils exceeded recommended physicochemical-quality specifications, emphasizing that laboratory discussions of thermal oxidation have a direct counterpart in real food environments.

The health implications of degraded cooking oils are biologically plausible but require careful interpretation. Lipid hydroperoxides formed early during oxidation are unstable and decompose into secondary products that include reactive aldehydes, ketones, alcohols, epoxides, and other oxidized lipids. Among compounds receiving particular attention are malondialdehyde, acrolein, 4-hydroxy-2-nonenal (4-HNE), 4-hydroxy-2-hexenal, and 2,4-alkadienals. These products can interact with proteins, lipids, DNA, and cellular signaling systems and may participate in oxidative stress and inflammation.^{13,14,15} Experimental cardiovascular literature provides additional biological coherence by linking oxidative lipid products, oxidized LDL, reactive aldehydes, inflammatory activation, and endothelial dysfunction with atherogenic pathways.^{16,17,18}

Nevertheless, an important evidentiary distinction is necessary. Demonstrating that repeated heating generates reactive compounds is not equivalent to demonstrating that habitual consumption of palm-oil-fried food causes cardiovascular events in humans. Much of the mechanistic literature uses purified oxidation products, cell cultures, high experimental doses, or animal models. Epidemiological research often evaluates fried-food consumption without characterizing the frying oil, its previous use, the number of heating cycles, or the actual concentrations of oxidation products. Reviews of repeatedly heated oils therefore point toward cardiovascular concern but also reveal substantial uncertainty concerning real-world human dose-response relationships.^{16,19} At the population level, higher fried-food consumption has been associated with coronary artery disease and broader cardiovascular outcomes in a large cohort and a meta-analysis, but those studies did not chemically characterize frying-oil reuse and therefore cannot isolate repeatedly heated palm oil as the causal exposure.^{20,21}

This review addresses that gap through a qualitative, cross-disciplinary synthesis. It asks how repeatedly heated palm oil changes chemically; which frying conditions determine those changes; which compounds and analytical indicators are most informative; how consumers and food-service workers may be exposed; which

biological pathways could connect oxidized frying lipids with endothelial and metabolic dysfunction; how strong the different layers of evidence are; and which practical interventions could reduce exposure.

The article proposes a **Fryer-to-Endothelium Continuum** as an organizing framework. Rather than treating palm oil as an isolated nutrient, the framework conceptualizes risk as a sequence beginning with the oil matrix, proceeding through frying intensity and chemical transformation, extending to dietary and inhalational exposure, and ultimately intersecting with biological susceptibility and cardiometabolic mechanisms. This approach also identifies intervention opportunities before exposure occurs, making the analysis relevant to food science, cardiovascular medicine, occupational health, and public-health governance.

2. LITERATURE REVIEW AND CONCEPTUAL FOUNDATIONS

2.1 Palm Oil as a Frying Medium

The thermal performance of palm oil is strongly influenced by its fatty-acid and triacylglycerol composition. Palm oil contains substantial proportions of palmitic and oleic acids together with smaller quantities of linoleic acid. Palm olein, the more liquid fraction widely used in frying, contains relatively more unsaturated fatty acids than palm stearin while retaining greater oxidative stability than many conventional seed oils rich in linoleic or linolenic acids. The number and position of double bonds influence susceptibility to radical attack; consequently, highly polyunsaturated oils often generate oxidation products more rapidly under severe thermal conditions.^{22,23}

Minor components further modify stability. Tocopherols and tocotrienols can quench lipid radicals and delay propagation, while carotenoids contribute antioxidant activity in less extensively refined palm products. However, thermal exposure progressively consumes these protective constituents. [Loganathan et al. \(2020\)⁷](#) demonstrated changes in carotene and vitamin E retention during heating of red palm olein, emphasizing that the nutritional properties of the starting oil cannot automatically be extrapolated to repeatedly heated oil. Similarly, [Li et al. \(2023a\)²⁴](#) showed that repeated batch frying of palm oils resulted in loss of endogenous vitamin E and that antioxidant supplementation could partly restrain deterioration.

The relative thermal stability of palm olein is nevertheless demonstrable. [Patil, Waghmare and Annapure \(2023\)²⁵](#) found better oxidative performance for palm olein than sunflower oil during repeated frying of Indian battered foods. Industrial-scale work by [Silva et al. \(2024\)⁶](#) likewise confirmed differences in oxidation behavior between palm olein and hydrogenated soybean oil. However, [Teh, Mah and Abd Razak \(2020\)⁴](#) showed that even palm olein deteriorated during prolonged heating once the duration exceeded its relatively stable early phase. These findings support a continuum rather than a dichotomy: palm oil may deteriorate more slowly under particular conditions, but sufficiently intensive heating eventually overwhelms endogenous protection.

2.2 Chemical Reactions During Deep Frying

Deep frying constitutes a complex reaction environment rather than a simple heating process. Food introduces water into the oil, atmospheric oxygen enters at the oil surface, and temperatures are sufficiently high to accelerate radical chemistry. Hydrolysis cleaves ester bonds and produces free fatty acids, monoacylglycerols, and diacylglycerols. Oxidation begins with hydrogen abstraction and formation of lipid radicals, followed by oxygen addition and hydroperoxide formation. Hydroperoxides are themselves unstable at frying temperatures and rapidly decompose into secondary products. Polymerization creates dimers and higher-molecular-weight compounds, while severe heating can promote cyclization and geometrical isomerization.^{3,22,26}

Because hydroperoxides decompose rapidly at high temperatures, peroxide value alone may produce misleading interpretations. An extensively deteriorated frying oil can contain a moderate peroxide concentration simply because primary peroxides have already decomposed into secondary compounds. Consequently, p-anisidine value, TOTOX, TPC, polymerized triglycerides, carbonyl measurements, aldehyde profiles, and increasingly sophisticated lipidomic approaches provide complementary information.^{3,27}

Recent oxidative-lipidomics research demonstrates how chemically diverse this process becomes. [Hu et al. \(2023\)](#)²⁸ identified epoxy-, hydroperoxy-, hydroxy- and oxo-triacylglycerol derivatives during simulated frying, illustrating that non-volatile oxidation products extend far beyond the traditional peroxide–aldehyde model. [Koch, Löwen and Schebb \(2024\)](#) found appreciable concentrations of oxidized fatty acids in fried and highly processed foods, reinforcing the relevance of non-volatile products as a potential dietary exposure.

The food itself changes the reaction environment. Moisture promotes hydrolysis; proteins and carbohydrates contribute reactive intermediates; food residues may catalyze additional reactions; and the porous structure of the food determines oil uptake. [Huang et al. \(2023\)](#)²⁹ showed that amino acids can alter formation patterns of glycerol-core aldehydes, while [Xu et al. \(2020\)](#)³⁰ demonstrated that the amount of these compounds generated under restaurant-style frying varied according to both oil type and fried food. Thus, the toxicological profile of “used oil” cannot be predicted exclusively from the botanical source of the oil.

2.3 Aldehydes and Other Secondary Oxidation Products

Aldehydes are particularly important because many are sufficiently reactive to modify biological macromolecules. Linoleic-acid oxidation can generate 4-HNE and related α,β -unsaturated aldehydes, while oxidation pathways involving different unsaturated fatty acids generate distinct aldehydic fingerprints. [Liao, Zhu and Chen \(2020\)](#) reviewed the occurrence and biological reactivity of food-derived 4-HNE, noting its ability to form adducts with proteins and other molecules. [Scianò et al. \(2025\)](#)¹⁴ similarly emphasized that high-temperature cooking produces numerous aldehydes that may occur both in the residual oil and in cooking fumes. Modern analytical work suggests that aldehyde formation begins rapidly. [Freis and Vemulapalli \(2025\)](#)³¹, using high-field proton NMR, observed new aldehydic signals after short periods of high-temperature frying and

progressive increases with continued heating. Guo et al. (2025)¹⁵ reviewed acrolein, 2,4-decadienal, 4-HNE and related compounds in fried foods and highlighted both analytical and mitigation challenges. Liu et al. (2020)³² also demonstrated that temperature and heating duration substantially determine aldehyde formation.

Glycerol-core aldehydes constitute another emerging group. Unlike freely volatile aldehydes, these compounds remain esterified to glycerol structures and may behave differently during digestion and absorption. Xu et al. (2020)³⁰ documented their accumulation during restaurant deep frying, and Huang et al. (2022)³³ demonstrated temperature- and iron-dependent formation, with ferric ions accelerating production under simulated frying conditions. These findings make fryer materials, trace metals, food residues and processing conditions potentially important exposure modifiers.

2.4 Total Polar Compounds and the Problem of Oil-Quality Measurement

TPC is widely regarded as one of the most useful global indicators of frying-oil deterioration because it captures several classes of compounds that increase as triglycerides undergo oxidation, hydrolysis and polymerization. Chen et al. (2021)³ reviewed the formation, measurement and health significance of polar compounds and showed why TPC is useful for monitoring frying-oil life. Li et al. (2023b)³⁴ further found that TPC could predict the evolution of epoxy fatty acids under fast-food restaurant conditions within the oils they evaluated. Restaurant-scale analysis also shows that the distribution of hydrolytic, oxidized-triacylglycerol and triglyceride-polymer fractions varies with both oil type and fried food, reinforcing the need to interpret aggregate TPC values in context.³⁵

Nevertheless, no single index fully characterizes the chemical complexity of used frying oil. TPC provides an integrated measure but does not identify individual reactive species. Peroxide value may underestimate advanced oxidation; acid value primarily reflects hydrolytic change; p-anisidine value captures certain secondary aldehydes but not the entire oxidation spectrum. This explains the growing interest in rapid multi-parameter sensing, electrochemical approaches, NMR, FTIR, and machine-learning-assisted prediction. Patil et al. (2023a)²⁵, for example, developed electrochemical predictive models for TPC across several frying oils, suggesting a pathway toward affordable point-of-use monitoring.

A practical monitoring framework should therefore distinguish between **operational screening** and **research-grade exposure characterization**. Food vendors require inexpensive, rapid methods that indicate whether oil should be replaced. Research aiming to identify health effects needs substantially more chemical resolution, ideally integrating TPC with aldehyde profiling, oxidized triglycerides, epoxy fatty acids, antioxidant depletion, and polymerized products.

2.5 Process Contaminants Beyond Conventional Lipid Oxidation

Thermal processing of palm oil can also involve contaminants such as 3-monochloropropane-1,2-diol esters (3-MCPDE) and glycidyl esters (GE), whose concentrations are affected by refining history and subsequent

heating. Chu, Noh and Lee (2024)³⁶ examined oxidation products together with 3-MCPDE and GE in different edible and blended oils during French-fry preparation and documented significant changes during frying. Lee et al. (2023)³⁶ similarly evaluated these compounds in diacylglycerol-palm olein blends. A broader review concluded that temperature, heating duration, chloride, moisture and partial acylglycerols can all influence MCPD-ester and GE behavior during frying and baking.³⁷

Importantly, degradation products do not always move monotonically in the same direction during heating; compounds may form and subsequently decompose, migrate into foods, volatilize, or react with other components. Consequently, health interpretation should not assume that every contaminant simply accumulates with every heating cycle. In repeated deep-frying, 3-MCPDE and GE have been shown to follow different trajectories and to partition into fried foods, while periodic-heating studies of palm oil demonstrate that FTIR and NMR fingerprints can be used to model physicochemical deterioration and contaminant formation.^{38,39} Chew et al. (2021)⁴⁰ further demonstrated that upstream treatment of crude palm oil could substantially reduce 3-MCPDE and GE in refined oil and improve subsequent frying performance, illustrating that pre-frying oil quality and refining technology influence the downstream exposure profile.

3. METHOD

3.1 Review Design

This study adopts a **qualitative literature review** design rather than a systematic literature review or meta-analysis. The purpose is interpretive integration across several evidence domains that cannot be meaningfully reduced to a single quantitative endpoint: food chemistry, frying technology, toxicology, cardiovascular biology, nutritional exposure, occupational exposure, food-service practices and public-health intervention.

Accordingly, the review does not claim exhaustive systematic retrieval, does not employ PRISMA flow reporting, and does not calculate pooled effect estimates. Instead, contemporary peer-reviewed literature was purposively identified and organized around predefined conceptual domains within the proposed Fryer-to-Endothelium Continuum. This design is appropriate when the research objective is to understand mechanisms, relationships, evidence gaps, contextual differences and translational implications rather than estimate a single summary effect.

3.2 Literature Identification

Priority was given to peer-reviewed journal articles published from January 2020 through 2026. Searches were structured around combinations of terms related to palm oil, palm olein, deep frying, repeated heating, thermo-oxidation, total polar compounds, aldehydes, glycerol-core aldehydes, oxidized triglycerides, cooking-oil fumes, endothelial dysfunction, oxidized LDL, atherosclerosis, cardiometabolic outcomes, street food, food vendors and frying-oil monitoring.

The conceptual evidence base drew primarily from journals indexed in major academic databases and publisher platforms, including PubMed/MEDLINE, Scopus- and Web of Science-indexed journals, ScienceDirect, Springer, Wiley, Taylor & Francis and other established scholarly repositories. Palm-specific evidence was prioritized whenever available. Evidence derived from other edible oils was included when it clarified general chemical processes, analytical methods, exposure pathways, or biological mechanisms reasonably applicable to oxidized frying lipids.

3.3 Thematic Synthesis

The literature was organized into six principal domains: (1) oil composition and baseline oxidative stability; (2) frying intensity and chemical degradation; (3) degradation markers and hazardous products; (4) transfer into foods and cooking fumes; (5) biological mechanisms related to endothelial and metabolic dysfunction; and (6) food-service practices, monitoring and prevention. This structure allowed chemical, mechanistic, exposure and implementation evidence to be interpreted within a common causal framework while preserving differences in evidentiary directness.

The analysis explicitly distinguished the **directness of evidence**. Palm-specific chemical studies were considered direct evidence for changes occurring in palm-based frying oils. Studies involving other oils were considered supporting evidence for general thermo-oxidation mechanisms. Cell and animal studies were interpreted as mechanistic or toxicological evidence, while observational human studies and human biomarkers were regarded as more directly relevant to population health. This distinction was retained throughout the interpretation to avoid inferring human cardiovascular causality from laboratory findings alone.

4. RESULTS: THEMATIC FINDINGS

4.1 Theme 1: Palm Oil Is Relatively Stable but Becomes a Different Exposure After Repeated Heating

The contemporary literature consistently indicates that fatty-acid composition influences thermal stability. Oils richer in polyunsaturated fatty acids generally provide more double-bond sites susceptible to peroxidation, whereas more saturated and monounsaturated matrices often degrade more slowly during high-temperature use. Palm olein therefore possesses a useful technological advantage in many frying applications.^{22,23,25}

However, the decisive finding is that relative resistance does not prevent progressive deterioration. **Luo et al. (2024)**⁹ observed increasing TPC and aldehydes during prolonged frying with palm oil, with TPC approaching commonly used discard ranges after extensive use. **Zheng et al. (2022)**⁸ found progressive increases in deterioration markers during repeated frying in palm oil. Recent research comparing repeated heating of palm oil, palm olein and palm stearin similarly reported increased free fatty acids, peroxide values and secondary oxidation indices as reuse intensified.

This supports an important conceptual distinction between **identity of the oil** and **history of the oil**. Two samples originally obtained from the same palm olein may become chemically different exposures if one is used

briefly under controlled conditions while another undergoes multiple heating cycles, overnight storage, topping-up, exposure to food residues and prolonged contact with oxygen.

Depletion of protective micronutrients accompanies this transformation. Loganathan et al. (2020) documented losses and changes in carotenoids and vitamin E during heating of red palm olein. Li et al. (2023a) reported losses of endogenous tocopherols and tocotrienols during batch frying of palm oil, while antioxidant intervention partly slowed deterioration. The evolving oxidation profile should therefore be understood partly as a shifting balance between oxidizable substrates, endogenous antioxidant defenses and accumulated degradation products.

4.2 Theme 2: Frying Conditions Can Be as Important as Oil Type

Temperature is a dominant determinant of reaction velocity. Higher frying temperatures accelerate radical formation, hydroperoxide decomposition, aldehyde generation and polymerization. Huang et al. (2022)³³ demonstrated that glycerol-core aldehyde formation increased with thermal severity and could be further accelerated by ferric ions, illustrating the interaction between temperature and catalytic conditions. Liu et al. (2020)³² similarly documented temperature- and time-dependent aldehyde generation in a frying model.

Cumulative exposure time is equally important. Repeated short frying cycles can impose a substantial total oxidative burden, especially when oil is repeatedly cooled and reheated. Intermittent frying differs chemically from continuous frying because cooling cycles allow additional oxygen dissolution while subsequent reheating accelerates reactions. The commonly reported “number of frying cycles” is therefore not a sufficient exposure descriptor unless temperature, cycle duration, downtime, food load and replenishment are also reported.

Food moisture promotes triglyceride hydrolysis, whereas proteins, carbohydrates and minerals influence oxidation chemistry. Food type affected glycerol-core aldehyde generation under restaurant conditions in the study by Xu et al. (2020). Huang et al. (2023)^{29,30} found that amino acids themselves modified formation of glycerol-core aldehydes, demonstrating the importance of oil–food interactions rather than oil chemistry in isolation.

The oil-to-food ratio and replenishment strategy further complicate interpretation. Addition of fresh oil dilutes degradation products and restores antioxidants but does not remove accumulated polymers or residues completely. Continuous commercial fryers in which fresh oil is regularly added may therefore behave differently from small street-food pans in which essentially the same oil is reheated for several sessions.

Fryer materials and geometry influence oxygen transfer, heat distribution and exposure to catalytic metals. Fekadu, Abera and Weldemichael (2024) found measurable differences in oil quality between street vendors using different frying equipment and heat sources. Such real-world variation demonstrates why laboratory frying protocols must be interpreted cautiously when extrapolated to food-service settings.

4.3 Theme 3: The Degradation Mixture Is Chemically Multidimensional

A major thematic finding is that the term “oxidized oil” conceals a chemically heterogeneous mixture. Primary hydroperoxides are only one transient stage. Subsequent products include aldehydes, ketones, alcohols, epoxy derivatives, oxidized triacylglycerols, cyclic structures and polymerized triglycerides.^{3,26,28}

Reactive aldehydes deserve particular attention because of their electrophilic properties. 4-HNE is capable of reacting with cysteine, histidine and lysine residues in proteins and has been linked with oxidative-stress-related disease pathway.^{13,41} Acrolein is another highly reactive compound associated with overheated oils and with experimental vascular injury and atherosclerotic processes.⁴² More recent food-chemistry reviews emphasize that a broad aldehydic spectrum is generated by thermal oxidation and that analytical methods increasingly need to quantify individual species rather than simply total carbonyls.^{14,15}

Frying also generates non-volatile oxidized molecules that remain within the oil and can be incorporated into food. [Hu et al. \(2023\)](#)²⁷ identified multiple oxidized triglyceride derivatives using lipidomic approaches. [Koch, Löwen and Schebb \(2024\)](#)⁴³ detected appreciable dietary quantities of oxylipins in fried and highly processed foods. These findings extend the exposure discussion beyond volatile aldehydes and indicate that the ingested oxidation mixture may contain structurally diverse lipid mediators and their precursors.

Figure 1 summarizes the central conceptual contribution of this review. Rather than attributing health effects directly to palm oil as a commodity, the model positions frying history between the original oil and the eventual biological exposure. This intermediate processing stage determines which degradation products are formed, in what quantities, and through which route consumers or food workers encounter them.

Figure 1. The Fryer-to-Endothelium Continuum

How repeated palm-oil frying can transform a food-processing step into a multi-stage exposure pathway

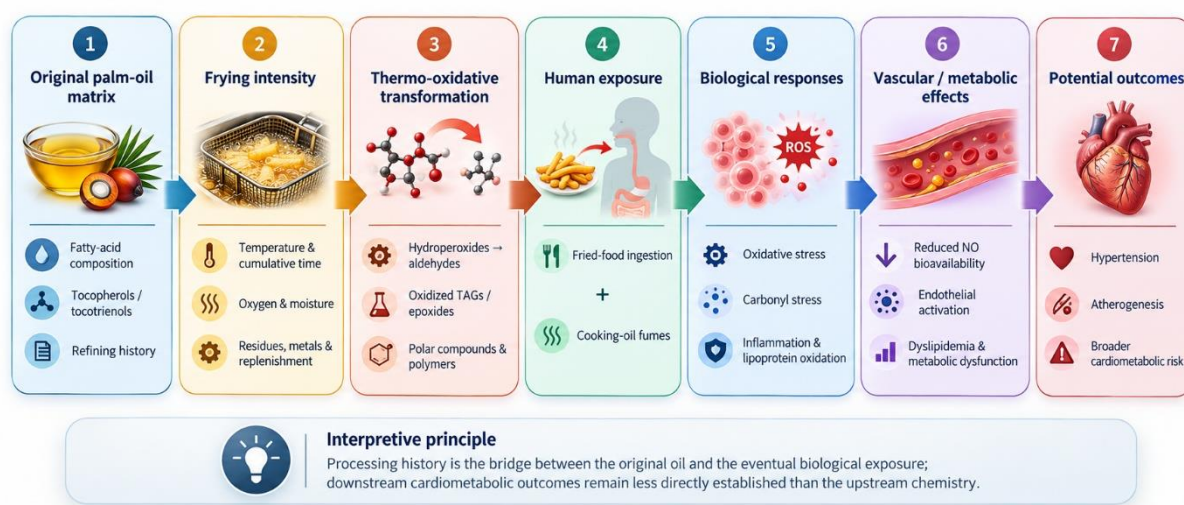


Figure 1: The Fryer-to-Endothelium Continuum

The figure also emphasizes that progression from chemical degradation to clinical cardiovascular disease should not be interpreted as a proven deterministic sequence. Evidence is strongest for the early chemical transformations and for the biological plausibility of several downstream mechanisms; direct human longitudinal evidence for the entire chain remains comparatively weak.

4.4 Theme 4: TPC Is Useful, but a Multi-Marker Strategy Is Scientifically Preferable

TPC has considerable value for commercial monitoring because it increases as oxidation, hydrolysis and polymerization progress. [Chen et al. \(2021\)³](#) identify TPC as an integrated measure with practical importance, while [Li et al. \(2023b\)³⁴](#) demonstrated that it may also correlate with specific emerging hazards such as epoxy fatty acids under restaurant-style frying conditions.

However, reliance on TPC alone may oversimplify exposure. Oils with comparable TPC values can possess different aldehyde, polymer, antioxidant, and oxidized-triglyceride profiles because their fatty-acid compositions and frying histories differ. Accordingly, a research-grade assessment should combine conventional indicators such as acid value, peroxide value, p-anisidine value and TPC with targeted analysis of reactive aldehydes, polymers and oxidized lipid species.

Rapid monitoring technologies may eventually bridge this gap in food-service practice. [Patil et al. \(2023a\)²⁵](#) demonstrated machine-learning-assisted electrochemical prediction of TPC, while [Li et al. \(2020\)⁴⁴](#) evaluated rapid testing of polar compounds under restaurant conditions. Spectroscopic methods, portable dielectric measurements and sensor-based systems could make objective discard decisions more feasible for restaurants and institutional kitchens. The challenge is to achieve sufficient analytical reliability without making monitoring economically inaccessible to small vendors.

[Figure 2](#) illustrates why peroxide value cannot be interpreted in isolation. Primary peroxides can decline when they decompose into secondary products even though the overall oxidative condition of the oil is worsening.

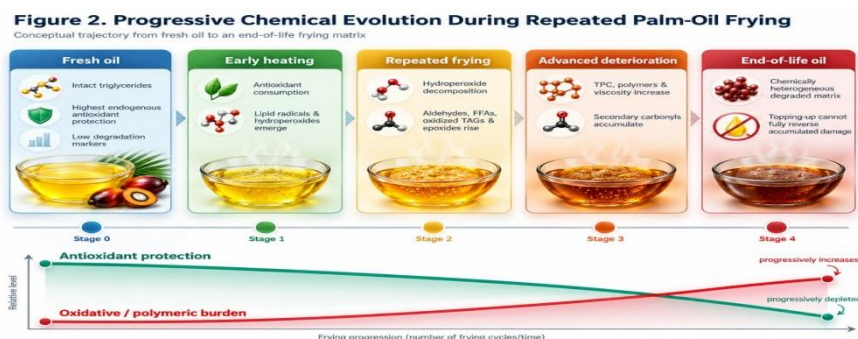


Figure 2: Progressive Chemical Evolution During Repeated Palm-Oil Frying

Accordingly, the transition from an apparently acceptable frying oil to a substantially degraded oil should be evaluated through multiple indicators and the cumulative process history rather than through one measurement or visual observation. Restaurant-scale evidence further shows that the composition of polar degradation fractions depends on both the oil and the food being fried, supporting a multi-marker rather than single-threshold interpretation.³⁸

4.5 Theme 5: Fried Food Is the Principal Dietary Exposure Vehicle

Consumers generally do not drink used frying oil directly; they ingest oxidation products within foods that absorb oil during cooking. Oil uptake depends on food structure, frying duration, temperature, moisture loss and post-frying cooling. Valle et al. (2024)² emphasized the interaction among food structure, frying technique, oil absorption and chemical composition. This means that external oil measurements need to be linked with concentrations actually retained in the edible portion.

Oxidation products may partition differently between oil and food. Some compounds remain in the fryer, others are absorbed into the food, some interact with proteins or starch, and volatile compounds escape into the atmosphere. Wang et al. (2025)⁴⁵, for example, explored interactions between toxic aldehydes and starch-based food components during frying, highlighting the possibility that food matrices influence both retention and transformation of reactive compounds.

Koch, Löwen and Schebb (2024) provide additional evidence that oxidized fatty-acid products occur in actual meals, with particularly elevated concentrations in certain fried and highly processed foods. Therefore, future exposure assessment should measure both the frying medium and the finished food rather than assuming that concentration in the fryer directly equals ingested dose.

4.6 Theme 6: Cooking Fumes Create an Additional Occupational Exposure

High-temperature frying also releases volatile organic compounds, aldehydes and particulate matter. Thus, cooks, street vendors and kitchen workers may experience an exposure pathway not shared to the same extent by consumers. Scianò et al. (2025)¹⁴ note that aldehydes generated from cooking oils occur both within oil waste and in cooking fumes. In a controlled comparison of palm, olive and soybean oils, deep frying produced oil-specific mixtures of ultrafine particles, gaseous aldehydes and particle-bound polycyclic aromatic hydrocarbons, demonstrating that the inhalation profile depends on the frying medium as well as the thermal process.⁴⁶ Experimental research using simulated cooking-oil fumes has also demonstrated oxidative and other biological effects at relatively low exposure levels, although such studies do not reproduce the full complexity of occupational environments (Li et al., 2022b).⁴¹

Ventilation, kitchen volume, fryer temperature, oil type, cooking duration and worker shift length all influence inhalational exposure. Occupational exposure deserves greater emphasis in future palm-oil research because

palm-based frying is prevalent in many commercial and informal food-service sectors. Studies that measure only dietary intake could therefore underestimate the total exposure burden among food workers.

4.7 Theme 7: A Biologically Plausible Pathway Links Oxidized Lipids with Endothelial Dysfunction

The endothelium regulates vascular tone, thrombosis, leukocyte adhesion, barrier function and inflammatory signaling. Nitric oxide generated by endothelial nitric-oxide synthase is fundamental to vasodilation and vascular homeostasis. Oxidative stress can reduce nitric-oxide bioavailability by increasing reactive oxygen species, promoting NO scavenging and altering eNOS signaling. Endothelial dysfunction is therefore both an early indicator and a mechanistic contributor to cardiovascular disease.

Leong (2021) synthesized evidence linking repeatedly heated cooking oils and lipid oxidation products with inflammatory pathways involved in hypertension and atherosclerosis. The proposed mechanism involves oxidative degradation products entering biological systems, increasing oxidative burden and activating inflammatory signaling. Reactive aldehydes such as MDA and 4-HNE are particularly relevant because they can form protein adducts and influence redox-sensitive pathways.

Oxidized lipoproteins constitute another bridge between oxidative exposure and vascular disease. Jiang et al. (2022)¹⁷ described how oxidized LDL contributes to endothelial dysfunction, monocyte recruitment, smooth-muscle-cell responses, platelet activation and plaque progression. Mushenkova et al. (2021)⁴⁷ emphasized macrophage recognition of oxidation-specific epitopes and subsequent inflammatory and foam-cell pathways. Salekeen et al. (2022)¹⁸ similarly placed lipid peroxidation and oxidative imbalance within the broader pathophysiology of atherosclerotic cardiovascular disease.

Oxidized phospholipids add another layer. Koschinsky and Boffa (2022)⁴⁸ reviewed their contribution to the pro-inflammatory properties of lipoprotein(a), while Koutsogianni et al. (2022)⁴⁹ discussed the evolving relationship between oxidized phospholipids, lipoprotein(a) and atherosclerotic risk. Pantazi, Tellis and Tselepis (2022)⁵⁰ further examined oxidized phospholipids and lipoprotein-associated phospholipase A2 within cardiovascular disease biology.

Human association data support the clinical relevance of oxidized LDL as a cardiovascular biomarker, although they do not establish that dietary oxidized lipids are the source. Hong et al. (2023)⁵¹ found an association between oxidized LDL and cardiovascular disease across the literature they synthesized. This distinction is critical: endogenous LDL oxidation can result from many metabolic and inflammatory processes, and circulating oxidized LDL should not be interpreted as a specific biomarker of repeatedly heated palm-oil consumption.

Acrolein provides another mechanistic example. Crowley et al. (2022)⁴² reviewed experimental models linking acrolein exposure with atherosclerotic pathways. Since acrolein can arise from overheated oils as well as numerous environmental sources, it exemplifies both the plausibility and the attribution problem: it is

biologically relevant but insufficiently source-specific to function alone as a biomarker of heated cooking-oil exposure.

4.8 Theme 8: Experimental Evidence Suggests Multi-Organ Effects but Has Translational Limitations

Animal studies frequently report oxidative, inflammatory or metabolic abnormalities following consumption of repeatedly heated oils. Murwani et al. (2024)⁵², for example, found elevations in malondialdehyde, TNF- α and creatinine in rats directly fed used cooking oil relative to some comparison groups. GadAllah, Noaman and Azab (2023)⁵³ reported reproductive toxicity in rats exposed to street-food-related deep-fried palm olein conditions. These findings indicate biological activity of degraded-oil exposures but cannot be translated directly into human cardiovascular risk because animal doses, administration methods and metabolic conditions may differ substantially from normal dietary exposure.

The mode of administration is particularly important. Feeding animals used cooking oil directly may generate substantially higher exposure per unit body mass than eating normal portions of fried food. Species differences in lipid metabolism and detoxification also limit extrapolation. Consequently, experimental models provide evidence of hazard and mechanism rather than precise estimates of real-world human risk.

Figure 3 prevents a common interpretative error in nutritional toxicology by separating stages of the proposed causal pathway according to evidentiary strength. It therefore distinguishes robust upstream chemical evidence from the more limited direct evidence connecting quantitatively characterized exposure with prospective human cardiovascular outcomes.

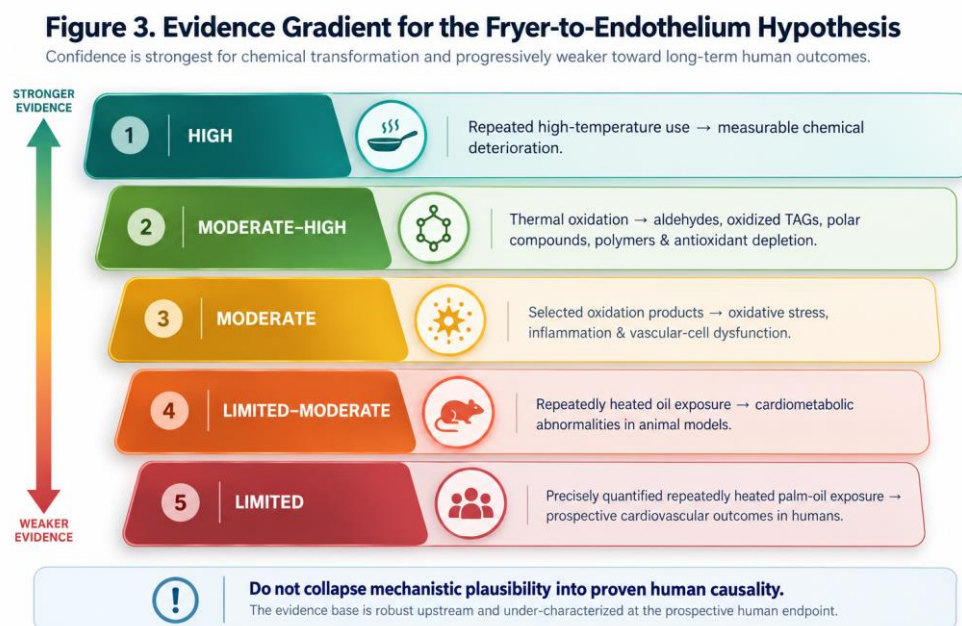


Figure 3: Evidence Gradient for the Fryer-to-Endothelium Hypothesis

The literature strongly demonstrates the formation of degradation products and supports several mechanistic pathways, but the final human disease link remains under-characterized. This distinction should guide both scientific conclusions and public-health communication.

4.9 Theme 9: Real-World Food-Service Practice Is a Major Exposure Determinant

Field evidence shows that oil-management decisions are influenced by economics, equipment and knowledge. Okalany, [Byakika and Mukisa \(2024\)](#) documented poor quality among oils collected from street-food vendors in Kampala and found deterioration after repeated recycling. [Fekadu, Abera and Weldemichael \(2024\)](#)¹¹ showed that frying equipment was associated with differences in used-oil quality. [Aboagye et al. \(2024\)](#)¹ similarly documented deterioration among reused vendor oils in Ghana, while [Mishra et al. \(2023\)](#)¹² examined physicochemical and antioxidant changes in oils used by fried-food vendors in India.

These studies are important because they move the literature away from idealized laboratory fryers. Street-food environments may use pans with large exposed surface areas, uneven heating, charcoal or wood fires, irregular temperature control and repeated overnight storage. Small vendors may face strong financial incentives to reuse oil until sensory deterioration is obvious. In contrast, standardized industrial frying can involve continuous filtration, automatic temperature control, scheduled oil replacement and frequent addition of fresh oil.

Risk assessment therefore needs context. “Three frying cycles” in a laboratory cannot be assumed equivalent to three commercial service periods, because cycle duration, food loading, oxygen exposure and replenishment may be entirely different.

4.10 Theme 10: Mitigation Is Possible at Multiple Points

Preventing oxidation is generally more realistic than attempting to remove all degradation products after they have formed. Temperature control is fundamental because unnecessary overheating accelerates oxidation and polymerization. Removing food residues can reduce localized degradation and catalytic reactions. Appropriate fryer design, filtration, ventilation, replenishment and complete replacement schedules can further reduce exposure.

Antioxidant strategies also demonstrate proof of concept. [Kedir et al. \(2023\)](#)⁵⁴ found that selected plant extracts improved palm-oil stability during frying. [Li et al. \(2023a\)](#)²⁴ demonstrated protection of palm-oil quality and endogenous vitamin E using tea polyphenols and TBHQ. [Li et al. \(2021\)](#)⁵⁵ found that rosemary-based antioxidant strategies could retard deterioration during deep frying, while [Zhang et al. \(2024\)](#)⁵⁶ demonstrated oxidative protection using modified oligomeric proanthocyanidins. [Shen et al. \(2023\)](#)⁵⁷ reviewed strategies involving oil blending and antioxidant stabilization.

These interventions should not be interpreted as permission for unlimited oil reuse. Antioxidants may delay deterioration but cannot indefinitely prevent hydrolysis, polymer formation or accumulation of food-derived

residues. Their most appropriate role is to extend oxidative stability within well-managed frying systems rather than substitute for discard criteria.

Mavlanov et al. (2025)⁵⁸ additionally demonstrated that processing and home-cooking practices can modify trans-fatty-acid profiles, illustrating that thermal management influences multiple nutritional dimensions. Xiang et al. (2024)⁵⁹ similarly emphasized the value of advanced compositional and lipidomic analysis for characterizing oil quality and nutritional change.

5. DISCUSSION AND ANALYSIS

5.1 Reframing the Question: From “Palm Oil” to “Processed Exposure”

The most important conceptual conclusion is that health evaluation based only on the original fatty-acid composition of palm oil is inadequate for repeatedly used frying oil. The oil entering a fryer is a raw material; the oil leaving an extensively used fryer is a processed chemical mixture shaped by time, temperature, oxygen, food and equipment.

This framing resolves an apparent contradiction in the literature. Palm olein can simultaneously be **relatively oxidation-resistant compared with some alternatives** and **capable of substantial deterioration during prolonged use**. The first statement concerns comparative kinetics; the second concerns cumulative exposure. They are not mutually exclusive.

Accordingly, public-health communication should avoid two opposite oversimplifications. The first is that palm oil is automatically dangerous because of its saturated-fat content. The second is that oxidative stability makes palm oil safe regardless of reuse duration. Contemporary evidence supports neither extreme. Instead, stability should be understood as a time-dependent property that interacts with operational conditions.

5.2 Answering RQ1: How Does Repeated Heating Alter Palm Oil?

Repeated heating shifts the chemical profile from intact triglycerides and endogenous antioxidants toward hydrolysis products, oxidized lipids and polymers. Early oxidation generates hydroperoxides, but these compounds rapidly decompose at frying temperatures. Secondary compounds therefore become increasingly important as frying continues.

Palm-specific evidence supports this progression. Teh, Mah and Abd Razak (2020)⁴ showed increasing deterioration during prolonged heating of palm olein. Loganathan et al. (2020) demonstrated losses of antioxidant micronutrients in heated red palm olein. Zheng et al. (2022), Luo et al. (2024), Silva et al. (2024) and Chu, Noh and Lee (2024)^{8,6,9} document changes across free fatty acids, TPC, p-anisidine values, aldehydes, volatile products and processing contaminants under frying conditions. The evidence therefore strongly supports the conclusion that repeatedly heated palm oil is chemically distinguishable from fresh palm oil.

The degree of deterioration nevertheless cannot be represented by the number of cycles alone. A 10-minute cycle at 160°C and an hour of continuous frying at 190°C constitute very different oxidative challenges. Future reporting should therefore include cumulative heating time and thermal profile in addition to cycle count.

5.3 Answering RQ2: Which Operational Factors Matter Most?

Temperature, cumulative time and oxygen availability form the central oxidation triad. Food moisture adds hydrolytic stress, while food type and residues modify the reaction environment. Catalytic metals can accelerate radical formation, as demonstrated by Huang et al. (2022).³³ Fryer design affects surface exposure, temperature uniformity and removal of residues.

Antioxidant depletion represents a dynamic modifier. An oil may perform relatively well early in service but become increasingly vulnerable once endogenous tocopherols and tocotrienols have been consumed. Thus, degradation may accelerate non-linearly rather than progress at a constant rate.

Replenishment creates another complexity. Adding fresh oil can lower measured concentrations of degradation products and restore antioxidants but simultaneously allows aged oil fractions to remain in the system. Studies should document whether oil was completely replaced or merely topped up, because the two practices create different exposure histories.

5.4 Answering RQ3: Which Chemical Indicators Matter Most?

TPC remains valuable for operational monitoring because it captures a broad fraction of altered lipid species and can be assessed using portable devices. Li et al. (2023b)³⁴ demonstrated a relationship between TPC and epoxy fatty acids in their fast-food frying model, supporting its practical relevance.

Nevertheless, health-oriented research requires a broader chemical fingerprint. A future standard panel could include TPC, acid value, p-anisidine value, polymerized triglycerides, selected aldehydes such as acrolein and 4-HNE, glycerol-core aldehydes, oxidized triglyceride classes, tocopherol/tocotrienol depletion and relevant process contaminants. Such a panel would permit comparison across studies and support dose-response analysis. Peroxide value deserves particularly cautious interpretation. Because peroxides are transient at high temperatures, declining PV does not necessarily signify improving oil quality. Measurements should therefore be interpreted alongside secondary oxidation markers.

5.5 Answering RQ4: How Could Repeatedly Heated Oil Affect the Endothelium?

The proposed mechanistic chain begins with reactive lipid oxidation products entering the digestive tract or respiratory system. Some compounds may be absorbed intact; others are metabolized or interact with the gastrointestinal matrix. Exposure may increase systemic carbonyl and oxidative burden, although the extent to which individual dietary compounds survive digestion and reach vascular tissues remains incompletely characterized.

Reactive aldehydes can modify proteins and influence redox-sensitive signaling. 4-HNE is especially relevant because it is both a product of lipid peroxidation and a signaling molecule whose biological effects vary according to concentration.^{13,59} Acrolein has experimentally documented vascular toxicity and can participate in inflammatory and atherosclerotic mechanisms (Crowley et al., 2022).

A second pathway involves amplification of endogenous lipid oxidation. Oxidative and inflammatory stress may promote LDL modification. OxLDL can impair endothelial function, increase adhesion molecules, recruit monocytes and activate macrophage scavenger receptors.^{17,47} Macrophage lipid loading then facilitates foam-cell formation. Oxidized phospholipids on lipoproteins provide additional inflammatory signals, particularly in the biology of lipoprotein(a).^{48,49}

Nitric-oxide impairment links these pathways to vascular function. Excess ROS can reduce NO bioavailability and contribute to vasoconstriction and vascular inflammation. Over time, such disturbances could theoretically contribute to elevated blood pressure, impaired vascular reactivity and atherogenesis. Leong (2021) identifies inflammation-mediated hypertension and atherosclerosis as plausible consequences of lipid-oxidation-product exposure.

However, this chain remains partly inferential for repeatedly heated palm oil specifically. It would be scientifically inappropriate to state that a particular frying-oil aldehyde concentration necessarily produces a quantifiable increase in cardiovascular-event risk. The mechanistic bridge is plausible and supported at multiple biological levels, but the corresponding human exposure-response relationship remains insufficiently defined.

5.6 Answering RQ5: Why Food-Service Environments Deserve Special Attention

Food-service settings can produce higher and more frequent thermal loads than domestic cooking. A household may fry intermittently and discard oil after limited use, whereas a vendor may operate for several hours daily. Conversely, modern industrial fryers may control temperature and filtration more effectively than domestic pans. Exposure therefore cannot be assigned solely according to the label “commercial” or “household.”

Informal street-food sectors are especially relevant because they combine high consumer reach with economic sensitivity. Okalany, Byakika and Mukisa (2024) demonstrated that vendor oils could substantially exceed desirable quality parameters. Fekadu, Abera and Weldemichael (2024) showed equipment-related differences, illustrating that behavior and technology interact. These findings support an intervention approach focused on practical management rather than stigmatization of street foods.

Vendor education should include explanations that dark color alone is an imperfect indicator of degradation and that topping up does not completely reset oil quality. At the same time, recommendations must be economically realistic. Requiring frequent complete disposal without considering oil cost can create poor compliance or unintended incentives.

5.7 Answering RQ6: How Strong Is the Overall Evidence?

The evidence can be arranged into five tiers. These tiers reflect both the directness of the exposure evidence and the degree to which findings can be translated from chemistry and experimental systems to human cardiovascular outcomes.

First, chemical evidence is strong. Repeated high-temperature frying changes edible oils, including palm-based oils. This is consistently demonstrated using physicochemical markers, chromatography, NMR, spectroscopy and lipidomics (Xu et al., 2020; Chen et al., 2021; Luo et al., 2024; Freis and Vemulapalli, 2025).

Second, evidence for generation of biologically reactive products is strong. Aldehydes, oxidized triglycerides, oxylipins and other degradation products are well documented (Hu et al., 2023; Koch, Löwen and Schebb, 2024; Scianò et al., 2025).

Third, mechanistic cardiovascular evidence is strong but not palm-specific. Oxidized LDL, oxidized phospholipids, reactive aldehydes and oxidative stress participate in endothelial and atherosclerotic processes (Jiang et al., 2022; Salekeen et al., 2022; Koschinsky and Boffa, 2022).

Fourth, animal evidence supports biological hazard but is heterogeneous. Direct feeding of degraded oil can produce oxidative and inflammatory changes, but dose and route may differ markedly from ordinary human consumption (Murwani et al., 2024; GadAllah, Noaman and Azab, 2023).

Fifth, direct longitudinal human evidence remains limited. Population studies report associations between frequent fried-food consumption and coronary or broader cardiovascular outcomes, but exposure is generally defined by food frequency rather than by chemically characterized frying-oil history (Honerlaw et al., 2020; Qin et al., 2021). Reviews addressing repeatedly heated vegetable oils likewise identify cardiovascular concerns, yet palm-specific prospective research combining quantitative exposure characterization, biomarkers and clinical endpoints remains insufficient (Kaur, Singh and Misbah, 2026).

This hierarchy constitutes one of the most important conclusions of the review. It allows concern to be expressed without converting mechanistic plausibility into unproven clinical causality.

5.8 Answering RQ7: What Can Be Done Now?

Uncertainty about long-term human risk does not imply that no action is justified. Many recommended practices improve food quality regardless of eventual cardiovascular effect size.

First, unnecessary overheating should be avoided. Maintaining temperature within the lowest range consistent with effective frying reduces reaction rates while preserving food quality.

Second, residues should be regularly removed because burnt particles and food remnants can accelerate deterioration. Routine filtration can reduce prolonged contact between degraded residues and the frying medium, although it does not reverse oxidation products that have already formed.⁵⁷

Third, oil should be protected during storage between service periods, minimizing exposure to air, light and contaminants. Covered, cool and light-protected storage can reduce additional oxidative exposure before the next heating cycle.²²

Fourth, objective monitoring should gradually replace exclusive reliance on sensory judgment. Portable TPC or dielectric devices may offer a pragmatic pathway, particularly where laboratory testing is unrealistic.

Fifth, food-service interventions should combine education with economic assessment. Replacement schedules that are technically ideal but economically impossible are unlikely to achieve sustained adoption.

Sixth, ventilation should be incorporated into occupational-health strategies because frying fumes represent a distinct exposure route. This is particularly relevant because controlled deep-frying experiments show that palm, soybean and olive oils produce distinct mixtures of ultrafine particles, aldehydes and particle-bound polycyclic aromatic hydrocarbons.⁴⁶

Figure 4 emphasizes that risk reduction does not depend on a single technological solution. It can occur throughout the frying system, from procurement and temperature control to objective monitoring and occupational ventilation.

Figure 4. Multi-Level Intervention Framework for Safer Frying

Risk reduction can occur across procurement, operation, monitoring, occupational protection and governance.



Figure 4: Multi-Level Intervention Framework for Safer Frying

The most effective food-policy approach is therefore likely to combine technological, behavioral and regulatory interventions rather than focusing exclusively on the type of vegetable oil. The framework in Figure 4 accordingly combines source control, process control, monitoring, worker protection and governance rather than treating oil choice as the sole determinant of risk.\

5.9 Research Gaps and Future Agenda

5.9.1 Standardizing the Definition of Repeatedly Heated Palm Oil

A major methodological weakness is the absence of a harmonized definition. Future studies should report oil type and fraction, refining status, initial antioxidant profile, temperature, cumulative heating time, number and duration of cycles, cooling interval, food type, oil-to-food ratio, fryer geometry, oil depth, replenishment volume, filtration and storage conditions.

Without such information, “five-times heated oil” cannot be reliably compared across laboratories. A minimum reporting standard would substantially improve reproducibility and permit meaningful comparisons among laboratory, restaurant and street-food studies.

5.9.2 Developing a Palm-Specific Degradation Fingerprint

Future research should combine routine quality indices with targeted lipidomics. Palm-based oils possess fatty-acid and minor-component patterns that may generate different degradation profiles from highly polyunsaturated oils. Identifying reproducible combinations of aldehydes, oxidized TAGs, polymers, tocotrienol depletion and polar compounds could provide a more meaningful exposure metric than cycle number alone.

5.9.3 Human Exposure Biomarkers

The field urgently requires biomarkers that reflect exposure rather than only downstream oxidative stress. MDA, 4-HNE adducts and oxidized LDL are biologically informative but insufficiently source-specific. Future metabolomic work could investigate panels combining dietary oxidation products, specific aldehyde metabolites, oxidized lipid species and detailed dietary data.

Ideally, biomarkers should distinguish recent exposure from long-term habitual exposure and should be validated against chemically characterized meals. Validation should include sensitivity to dose, reproducibility over repeated exposures and discrimination from oxidative stress caused by smoking, air pollution and metabolic disease.

5.9.4 Controlled Human Feeding Studies

Ethically designed feeding studies could compare meals prepared using fresh versus moderately and reproducibly used oil within existing food-safety limits. Outcomes might include postprandial endothelial function, flow-mediated dilation, circulating adhesion molecules, oxidized LDL, lipid peroxidation markers, inflammatory mediators, insulin and triglyceride responses.

Such studies would provide stronger translational evidence than direct ingestion of used oil in animal models. They could also establish whether postprandial changes in oxidative and endothelial markers track the chemically measured degradation products in the meals being tested.

5.9.5 Prospective Observational Studies

Cohort research should move beyond generic questions such as frequency of fried-food consumption. Participants could be characterized according to predominant cooking-oil type, home versus commercial food sources, vendor practices and biomarkers of oxidized-lipid exposure. Repeated dietary assessments and cardiovascular phenotyping would allow more rigorous analysis of long-term relationships.

5.9.6 Food-Vendor Intervention Trials

Cluster-based intervention studies could test affordable strategies such as vendor education, thermometer use, filtration, TPC monitoring, replacement schedules and improved storage. Outcomes should include oil quality, food quality, costs, vendor compliance and sustainability.

The objective is not simply to demonstrate that an intervention works under laboratory supervision but whether it remains feasible in normal business conditions. Implementation outcomes such as cost, adherence, oil-discard frequency and vendor acceptability should therefore be treated as core endpoints rather than secondary observations.

5.9.7 Occupational Studies

Kitchen workers represent a potentially informative population because their exposure can combine inhalational and dietary pathways. Personal air monitoring, urinary aldehyde metabolites, oxidative-stress markers and vascular-function measurements could be integrated with ventilation assessments and frying conditions.

5.9.8 Advanced Rapid Monitoring

The development of electrochemical, spectroscopic and machine-learning systems should continue. [Patil et al. \(2023a\)²⁵](#) demonstrate that rapid prediction of TPC is technically feasible. Future devices could potentially integrate multiple signals and generate simple discard recommendations suitable for small food establishments. However, calibration should account for oil type because dielectric, spectroscopic and chemical responses may vary among palm olein, soybean, sunflower, canola and mixed oils. Oil-specific calibration is especially important if rapid devices are to be used across heterogeneous restaurant and street-food settings.

5.10 Public-Health Interpretation

Public communication regarding repeatedly heated palm oil should be precise. The available evidence does not support reducing the issue to a claim that “palm oil causes cardiovascular disease.” Nor should relative oxidative stability be used to justify indefinite oil reuse.

A more scientifically defensible message is that repeated high-temperature frying progressively degrades all edible oils; the rate and product profile depend on composition and operating conditions; palm oil often demonstrates useful relative stability but still forms degradation products under extended use; and avoiding excessive oil deterioration is a reasonable food-safety objective regardless of whether the exact magnitude of long-term cardiovascular risk has been established. Such wording communicates a preventable processing risk without implying that palm oil alone determines cardiometabolic disease.

This approach also prevents risk communication from becoming entangled unnecessarily with broader political or commodity debates around palm oil. The public-health target is a **processing practice**, not a crop species.

6. CONCLUSION

Repeated heating transforms palm oil and palm olein from their original nutritional matrices into increasingly complex mixtures of hydrolytic, oxidative and polymeric products. Palm-based frying oils often possess greater thermal stability than highly polyunsaturated alternatives, but this advantage is relative rather than absolute. Temperature, cumulative heating duration, oxygen exposure, food moisture, catalytic metals, residues, fryer characteristics, antioxidant depletion and replenishment practices together determine the speed and character of deterioration.

The strongest evidence concerns the chemistry of frying. Contemporary analytical studies demonstrate accumulation of total polar compounds, aldehydes, oxidized triacylglycerols, epoxy products, polymers and other secondary oxidation products during repeated or prolonged frying. Fried foods can retain oxidized lipids, while volatile degradation compounds create an additional exposure pathway through cooking fumes.

A mechanistically coherent biological pathway also exists. Reactive lipid-oxidation products can participate in oxidative and carbonyl stress, inflammatory signaling, lipoprotein oxidation and processes that impair endothelial homeostasis. Oxidized LDL, oxidized phospholipids, macrophage activation, reduced nitric-oxide availability and vascular inflammation provide plausible bridges from oxidative exposure to atherosclerosis and hypertension. Experimental studies of repeatedly heated oils further support biological activity.

However, the scientific evidence is not equally strong across the entire Fryer-to-Endothelium Continuum. Direct prospective human evidence specifically linking quantitatively characterized repeatedly heated palm-oil exposure to cardiovascular events remains limited. Consequently, repeatedly heated palm oil should be treated as a plausible and preventable processing-related exposure rather than as an established singular cause of cardiovascular disease.

Future research should prioritize standardized frying protocols, detailed chemical fingerprints, validated exposure biomarkers, controlled human feeding studies, prospective cohorts, occupational studies and pragmatic food-vendor interventions. From a public-health perspective, temperature control, removal of residues,

protection during storage, objective monitoring of oil deterioration, reasonable replacement schedules, appropriate ventilation and vendor education are immediately actionable measures.

The broader implication is that the health evaluation of edible oils should incorporate **processing history** as well as nutrient composition. The relevant exposure is not simply “palm oil,” but palm oil after a defined sequence of thermal, chemical and operational events. Reframing the problem in this way connects food chemistry with cardiovascular biology and food-service governance and provides a more rigorous basis for both future research and practical risk reduction.

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