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Mini Review

Tocotrienol-Rich Fraction from Palm Oil as a Multifaceted Bioactive Agent in Chronic Disease Prevention: Mechanisms, Evidence, and Clinical Prospects

Loso Judijanto*

IPOSS Jakarta, Indonesia

***Corresponding author:** Loso Judijanto, IPOSS Jakarta, Indonesia.

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Abstract

Tocotrienol-Rich Fraction (TRF) from palm oil represents one of the most promising natural vitamin E–derived bioactives for the prevention and adjunctive management of major chronic non-communicable diseases, including cardiovascular, metabolic, neurodegenerative, and malignant disorders. Compared with tocopherols, palm-derived α-, β-, γ-, and δ-tocotrienols exhibit superior antioxidant, anti-inflammatory, hypolipidemic, neuroprotective, and anticancer properties, largely attributed to their unsaturated isoprenoid side chains and more efficient membrane incorporation. This mini review synthesizes recent evidence (primarily since 2020) on the physicochemical characteristics, bioavailability, and tissue distribution of palm TRF, followed by domain-specific data on cardioprotective, neuroprotective, anticancer, antidiabetic, and immunomodulatory effects from cell, animal, and human studies. Systematic reviews and randomized controlled trials suggest that TRF can improve lipid profiles, attenuate oxidative stress and low-grade inflammation, modulate key signaling pathways such as NF κB and Nrf2, and confer functional benefits on cognitive performance, metabolic parameters, and liver function, with a favorable safety profile at commonly used doses. However, bioavailability limitations, heterogeneity among TRF formulations, and short trial durations limit definitive conclusions about clinical efficacy for hard outcomes. Future research should prioritize standardized TRF preparations, robust dose–response trials in well phenotyped patient populations, and integration with nanodelivery systems to overcome pharmacokinetic barriers. Overall, palm-derived TRF is a biologically plausible, well tolerated, and clinically promising nutraceutical candidate for chronic disease prevention and healthy ageing.

Keywords: Palm oil, Tocotrienol-rich fraction, Vitamin E, Chronic disease, Cardiovascular, Neuroprotection, Metabolic syndrome, Anticancer, Inflammation, Oxidative stress

JEL Classification Codes: I10; I12; I18; Q16; Q57

Introduction

Chronic Non-Communicable Diseases (NCDs)—notably cardiovascular diseases, type 2 diabetes, neurodegenerative disorders, and cancer—remain the leading causes of global morbidity and mortality and are strongly driven by oxidative stress, chronic low-grade inflammation, and metabolic dysregulation. Nutritional strategies and bioactive food components that target these shared mechanisms are increasingly recognized as important adjuncts to pharmacotherapy [1]. Palm oil (*Elaeis guineensis*) is one of the world's most widely produced and consumed vegetable oils and is distinguished by its high content of vitamin E isomers, including both tocopherols and tocotrienols. The Tocotrienol-Rich

Fraction (TRF) derived from palm oil typically contains a mixture of α-, β-, γ-, and δ tocotrienols together with smaller amounts of α tocopherol and constitutes one of the few commercially viable sources of natural tocotrienols. Structurally, tocotrienols share the chromanol ring of tocopherols but possess an unsaturated isoprenoid side chain with three double bonds, conferring greater membrane mobility and enabling more efficient interaction with lipid radicals in polyunsaturated fatty acid–rich bilayers. This structural feature is believed to underpin their stronger antioxidant activity and broader cell-signaling effects compared with tocopherols [2].

Over the last decade, and particularly since 2020, the biomedical literature has documented a rapidly expanding body of preclinical and clinical research on palm TRF in diverse disease domains. Systematic reviews have highlighted potential benefits on lipid profiles, vascular function, neurocognitive outcomes, and metabolic parameters, while scoping reviews emphasize TRF's capacity to modulate oxidative, inflammatory, and apoptotic pathways. At the same time, concerns remain about oral bioavailability, inter individual variability in response, and the lack of large-scale outcome trials [1,3].

This mini review focuses on TRF derived specifically from palm oil and aims to:

a. summarize its physicochemical properties, pharmacokinetics, and bioavailability;

b. synthesize recent evidence on cardioprotective, neuroprotective, anticancer, antidiabetic, and immunomodulatory actions; and

c. critically appraise current clinical trial data and identify translational gaps that must be addressed to realize its full potential as a nutraceutical for chronic disease prevention.

Physicochemical Properties and Bioavailability of Palm TRF

Palm-derived TRF is typically obtained from Refined, Bleached, and Deodorized (RBD) palm oil distillates via multistep fractionation and distillation to concentrate tocotrienols and tocopherols. The resulting mixture commonly contains α -, γ -, and δ-tocotrienol as the predominant species, with total tocotrienol content often exceeding 60–70% of the vitamin E fraction, plus approximately 20–30% α-tocopherol. However, precise ratios vary by manufacturer [4]. At the molecular level, tocotrienols are more lipophilic and exhibit faster membrane recycling than tocopherols, which may contribute to their enhanced chain-breaking antioxidant activity in lipid environments. However, oral TRF shows relatively low and variable bioavailability, largely due to competition with tocopherols for α-tocopherol transfer protein (α-TTP) and shared intestinal transport pathways. A recent systematic review of Randomized Controlled Trials (RCTs) on palm TRF reported that supplementation typically results in measurable but modest increases in plasma tocotrienol concentrations, with inter individual variability influenced by dose, formulation, fed state, and background diet [1].

To address these challenges, several strategies have been explored:

a. Self-emulsifying and nanoemulsified TRF formulations, which improve solubilization in the gastrointestinal tract and enhance Cmax and AUC compared with conventional soft gels [1,3].

b. Lipid-based delivery systems, including Nanostructured Lipid Carriers (NLC) and Solid Lipid Nanoparticles (SLN), that co encapsulate TRF with other lipids to enhance intestinal lymphatic transport and tissue uptake [4].

c. Modulation of co-administered dietary fat and timing relative to meals, which appears to significantly influence absorption [1].

Preclinical data indicate that orally administered palm TRF is distributed to the liver, adipose tissue, brain, and vascular tissues, although brain penetration may be limited and enrichment for specific isomers, such as α-tocotrienol, may occur. Overall, while the pharmacokinetic profile of TRF is compatible with chronic oral supplementation, formulation optimization remains a critical priority to maximize its clinical potential [5].

Cardioprotective and Antihyperlipidemic Mechanisms

Multiple lines of evidence support cardioprotective and antihyperlipidemic roles for palm TRF, with mechanisms spanning cholesterol biosynthesis, lipid peroxidation, and vascular inflammation [1,3]. At the cellular level, tocotrienols can suppress 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, the rate-limiting enzyme in cholesterol synthesis, through post-transcriptional degradation mechanisms distinct from those of statins. This contributes to reductions in total cholesterol and LDL cholesterol observed in several animal models and small human trials. TRF also exerts strong antioxidant effects, reducing LDL oxidation, diminishing Reactive Oxygen Species (ROS), and enhancing endogenous antioxidant enzyme activities such as superoxide dismutase and glutathione peroxidase [6]. In a rat model of high carbohydrate, high fat diet–induced metabolic syndrome, palm TRF supplementation improved plasma lipid profiles, attenuated hypertension, and ameliorated cardiac stiffness and ventricular dysfunction, alongside improvements in liver histology and hepatic enzymes. The authors attributed these benefits to combined hypolipidemic, anti-inflammatory, and antioxidant actions, including reduced inflammatory cell infiltration and collagen deposition in cardiac and hepatic tissues [1,3,7]. Recent narrative and systematic reviews have summarized clinical data suggesting that TRF, often at doses of 200–400 mg/

day, can modestly improve total cholesterol, LDL cholesterol, and triglycerides in individuals with hypercholesterolemia, metabolic syndrome, or high cardiovascular risk. However, results are not entirely consistent across trials. It has been reviewed that palm tocotrienols exhibit antihyperlipidemic and potential cardiovascular therapeutic effects, but it has been highlighted that sample sizes are small and trial durations are generally short [2,7].

Additionally, TRF may influence vascular function by modulating endothelial nitric oxide bioavailability, inhibiting vascular smooth muscle cell proliferation, and downregulating NF κB–mediated inflammatory signaling, although much of this evidence derives from preclinical models. Collectively, current data support a favorable cardiometabolic profile for palm TRF, warranting larger, longer-duration RCTs with hard cardiovascular outcomes [2].

Neuroprotective Effects

The Central Nervous System (CNS) has emerged as a key target for palm TRF, motivated by the vulnerability of neural tissue to oxidative stress, lipid peroxidation, and neuroinflammation. Tocotrienols demonstrate the ability to protect neurons against a variety of insults, including glutamate toxicity, ischemia–reperfusion injury, and Aβ induced oxidative stress in experimental models [1,3]. A 2020 systematic review evaluated preclinical data on the safety and neuroprotective efficacy of palm oil and TRF. All 18 included studies (10 animal, 8 cell-based) reported beneficial effects of TRF or α tocotrienol on cognitive performance, neuronal survival, or neurochemical markers, accompanied by reductions in oxidative stress markers and pro inflammatory cytokines. Importantly, the review found no major safety concerns at doses commonly used in experimental models, supporting the translational potential of TRF for neurological applications [3].

More recently, a scoping review focused on the role of TRF as a neuroprotective agent, highlighting mechanisms such as attenuation of superoxide dismutase overactivity, suppression of TNF α and other pro inflammatory mediators, regulation of neuronal genes and proteins, and preservation of synaptic function. The authors concluded that TRF may support healthy ageing and potentially delay neurodegenerative processes, though human evidence remains limited [5]. Clinical data, while still emerging, are encouraging. Several trials using palm TRF or mixed tocotrienol preparations in populations with white matter lesions or cognitive complaints suggest possible stabilization of white matter lesion volume and improvements in selected cognitive domains, although some of these involve proprietary formulations and relatively small sample sizes. A 2025 RCT using rice-derived tocotrienols demonstrated improvements in memory and sleep quality in adults with subjective memory complaints over 12 weeks, with no serious adverse events, underscoring the safety and neurofunctional potential of tocotrienols as a class [8]. Overall, the weight of preclinical evidence and early clinical signals indicates that palm TRF is a promising neuroprotective nutraceutical, particularly for conditions characterized by oxidative and inflammatory stress in the brain [8].

Anticancer Activity

Palm TRF exhibits multifaceted anticancer properties across several tumor types, including breast, colorectal, and liver cancers. Key mechanisms include modulation of apoptosis, cell-cycle regulation, angiogenesis, and metastatic potential [9]. *In vitro*, tocotrienols have been shown to induce mitochondria-dependent apoptosis via upregulation of pro-apoptotic proteins (e.g., Bax) and downregulation of anti-apoptotic proteins (e.g., Bcl-2), activation of caspase-3, and cleavage of PARP, leading to cancer cell death. Tocotrienols also inhibit NF κB and STAT3 signaling, reducing the expression of genes involved in proliferation, angiogenesis, and invasion. Furthermore, TRF can suppress VEGF expression and microvessel density, supporting an anti-angiogenic role [9-13].

In vivo studies using rodent models have demonstrated that palm TRF can slow tumor growth, reduce tumor multiplicity, and modulate oxidative and inflammatory microenvironments within tumors. These effects often coincide with decreased lipid peroxidation, altered antioxidant enzyme activity, and modulation of cellular redox status, which may sensitize tumor cells to apoptosis [9,13-15]. Human clinical evidence remains preliminary. Small early-phase trials have explored tocotrienol-enriched preparations as adjunctive therapy in breast and ovarian cancer, with some reports of improved response markers and tolerability, but robust, large-scale RCTs are lacking. Reviews emphasize that, while preclinical anticancer data are compelling, substantial methodological heterogeneity and the lack of standardized TRF formulations limit definitive conclusions about clinical efficacy [4,16-18]. The available evidence therefore supports a biologically plausible anticancer role for palm TRF, particularly as an adjuvant nutraceutical within multimodal therapy, pending further well-designed clinical trials [2].

Antidiabetic and Immunomodulatory Roles

The rising prevalence of type 2 diabetes and metabolic syndrome has spurred interest in TRF's effects on glucose homeostasis, insulin sensitivity, and inflammatory pathways that underpin metabolic dysfunction [1,3,7]. Preclinical models show that palm TRF can improve glucose tolerance, enhance insulin sensitivity, and reduce fasting blood glucose and HbA1c in high fat or high carbohydrate diet–induced metabolic derangements. These metabolic benefits are accompanied by reductions in plasma free fatty acids and triglycerides, reductions in hepatic steatosis, and improvements in liver histology, suggesting integrated effects on lipid and glucose metabolism [6]. On the immunological side, TRF modulates both innate and adaptive immune responses, partly by suppressing NF-κB activation and reducing the production of pro-inflammatory cytokines such as TNF-α, IL-6, and IL-1β. Clinical trials and human experimental studies indicate that TRF

supplementation can modify lymphocyte proliferation, CD4+/CD8+ ratios, and B cell counts, reflecting an immunoregulatory effect that may be relevant in chronic inflammatory conditions [1,3].

Recent human work also explores tocotrienol-enriched functional foods. For example, tocotrienol-enriched oat-based supplementation has been reported to improve features of metabolic syndrome, including lipid profiles and selected inflammatory markers, although these data remain limited and require replication. Systematic reviews of RCTs conclude that TRF appears metabolically safe and may confer modest improvements in metabolic risk markers, particularly when combined with lifestyle interventions [1,7].

Taken together, palm TRF exhibits antidiabetic and immunomodulatory properties that align with its broader cardiometabolic and anti-inflammatory profile, reinforcing its potential as a supportive nutraceutical in metabolic disease management [1,3].

Clinical Trial Evidence and Translational Gaps

Several clinical trials and systematic reviews have evaluated TRF supplementation in humans across cardiometabolic, neurological, and general health contexts, generally supporting safety and suggesting beneficial trends, yet revealing important translational gaps [19]. A recent systematic review of randomized controlled trials on the health benefits of palm TRF concluded that supplementation is associated with improvements in various surrogate outcomes, including lipid profiles, oxidative stress markers, inflammatory biomarkers, and functional measures such as cognition and sleep quality, with few serious adverse events reported. Similarly, review emphasizing chronic diseases summarized evidence for TRF in cardiovascular, respiratory, musculoskeletal, and metabolic conditions, underscoring its broad anti-inflammatory and antioxidant capacity [1,17,19-21].

In the neurological domain, early trials using TRF in individuals with white matter lesions and those with subjective memory complaints suggest possible stabilization of brain microstructural damage and improvements in memory and sleep. However, sample sizes are small, and formulations differ across studies. In metabolic syndrome and hyperlipidemia, TRF has been associated with modest reductions in LDL cholesterol and triglycerides, but the results are heterogeneous and sometimes non-significant [7,22-24].

Key translational challenges include:

a. **Bioavailability and Pharmacokinetics:** Variable intestinal absorption, competition with tocopherols, and differences in formulation (pure TRF vs mixed vitamin E products) complicate dose standardization [4,25,26].

b. **Formulation Heterogeneity:** Commercial TRF products differ in isomer composition, α tocopherol content, and co ingredients, making cross trial comparisons difficult [2,27,28].

c. **Trial Design Limitations:** Many studies are small, short term, and focus on intermediate endpoints rather than clinical events [17,19,29].

d. **Population Diversity:** Most trials involve relatively healthy or mildly at-risk individuals; data in high risk, multimorbid, or elderly populations are limited [1,19].

Future research should prioritize well-powered, randomized, double-blind trials with standardized TRF formulations, longer durations, and clinically meaningful endpoints, as well as head-to-head comparisons of different delivery systems to determine optimal dosing strategies [19].

Conclusion and Future Directions

The palm oil–derived tocotrienol-rich fraction is a multifaceted bioactive with compelling preclinical evidence and increasingly supportive clinical evidence for roles in chronic disease prevention and adjunctive management. Its structural distinctiveness from tocopherols confers potent antioxidant, anti-inflammatory, hypolipidemic, neuroprotective, anticancer, antidiabetic, and immunomodulatory properties across diverse experimental models. Human studies and systematic reviews generally confirm a favorable safety profile and show improvements in lipid profiles, oxidative and inflammatory markers, and functional outcomes such as cognition and sleep, although effect sizes are variable and context dependent.

Despite this promise, several critical gaps hinder translation into routine clinical practice. Oral bioavailability remains suboptimal, necessitating innovative delivery systems such as nanoemulsions and lipid nanocarriers tailored for TRF. The absence of standardized, well characterized TRF preparations complicates dose–response assessment and regulatory evaluation. Moreover, existing trials are typically short and underpowered to detect changes in hard clinical outcomes such as myocardial infarction, stroke, or incident dementia. Moving forward, integration of palm TRF into precision nutrition and personalized medicine frameworks—taking into account genetic variability, metabolic phenotype, and co medications—may help identify subgroups most likely to benefit. Collaboration between academia, industry, and regulators will be essential to develop quality assured formulations and robust clinical programs. In the interim, palm TRF can be reasonably regarded as a promising, mechanistically grounded nutraceutical with potential to complement conventional strategies for cardiometabolic health, neuroprotection, and healthy ageing, while recognizing that definitive outcome data are still forthcoming.

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Conflict of Interest

None.

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