Review Article

Physiological and Toxicological Impact of 3-MCPD Esters and Glycidyl Esters from Refined Palm Oil: A Systematic Literature Review of Metabolic Fate, Organ-Specific Effects, and Dose-Response Relationships

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Abstract

Refined palm oil has become a subject of heightened scientific scrutiny owing to the presence of 3-MCPD esters and glycidyl esters, which are formed during heat-intensive processing and are associated with possible biological impacts. This research seeks to systematically examine and consolidate available scientific findings related to their metabolism, organ-targeted effects, and dose-dependent responses. A Systematic Literature Review (SLR) approach based on the PRISMA framework was employed to ensure methodological transparency and consistency. Data were collected exclusively from peer-reviewed articles indexed in the Scopus database using structured keyword combinations. The selection process involved identification, screening, eligibility assessment, and inclusion, resulting in 34 articles that met predefined criteria, including publication period (2019–2026), English language, and open access availability. Data analysis was conducted through qualitative synthesis to identify recurring patterns and relationships across studies. The results indicate that both compounds undergo rapid gastrointestinal hydrolysis, releasing free 3-MCPD and glycidol, with conversion rates exceeding 80% in most experimental conditions. These metabolites are absorbed and distributed systemically, with primary accumulation observed in the kidneys and liver under high-dose exposure. Toxicological effects are predominantly dose-dependent, with adverse outcomes generally reported at exposure levels above 1–2 mg/kg body weight/day, while typical dietary exposure remains substantially lower. Variability across experimental models is observed, yet overall patterns remain consistent. In conclusion, the biological impact of these compounds is determined by the interaction between metabolic processes, organ-specific responses, and exposure levels, with current evidence supporting a context-dependent risk interpretation. Future research is recommended to focus on long-term human exposure, biomonitoring approaches, and improved exposure assessment models.

Introduction

Within the global food system, vegetable oils represent a crucial source of energy intake and functional lipids for a wide range of populations. Refined edible oils occupy a key role in everyday consumption and commercial food production, supported by their stability, broad supply, and technological flexibility [1]. The refinement process is designed to improve the sensory quality, shelf life, and safety of crude oils by removing undesirable impurities such as free fatty acids, pigments, and volatile compounds [2]. However, thermal and chemical conditions applied during refining, particularly at high

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temperatures, may also lead to the formation of certain process-induced compounds. These compounds have attracted growing scientific attention due to their potential biological activity and relevance within the broader context of food safety and risk assessment [3].

Within this framework, refined palm oil represents one of the most widely utilized vegetable oils globally, owing to its balanced fatty acid composition, oxidative stability, and economic importance in both food and non-food industries [4]. Its extensive use in frying applications, processed foods, and industrial formulations makes it a relevant subject of investigation in contemporary food science and toxicology research [5]. As with other refined oils, palm oil undergoes multiple stages of processing, including degumming, bleaching, and deodorization, during which temperature-dependent reactions may occur. Among the compounds formed during these stages, 3-monochloropropane-1,2-diol (3-MCPD) esters and glycidyl esters have emerged as key process contaminants of interest due to their documented formation mechanisms and potential biological implications [6].

3-MCPD esters and glycidyl esters are not naturally present in crude oils but are generated during refining processes, particularly under conditions involving elevated temperatures and the presence of chlorine-containing precursors [7]. These compounds are typically bound in esterified forms within the lipid matrix and are therefore not immediately bioactive until subjected to enzymatic hydrolysis following ingestion. Once released in their free forms, 3-MCPD and glycidol may interact with biological systems in ways that have been examined across toxicological, biochemical, and physiological studies [8]. As a result, understanding the transformation, distribution, and biological effects of these compounds has become an important area of research within food safety science.

From a toxicological perspective, 3-MCPD and glycidol exhibit distinct biological properties that have been investigated through both in vivo and in vitro experimental models. 3-MCPD has been associated with potential renal and reproductive effects under certain exposure conditions, while glycidol, as an epoxide compound, has been studied for its genotoxic potential and ability to form DNA adducts [9]. It is important to note, however, that such effects are typically observed under controlled experimental conditions involving exposure levels that may differ substantially from those encountered through normal dietary intake. Consequently, the interpretation of these findings requires careful consideration of dose, exposure duration, and biological context.

In addition to their intrinsic toxicological properties, the physiological behavior of these compounds is strongly influenced by their metabolic fate following ingestion. Both 3-MCPD esters and glycidyl esters undergo enzymatic hydrolysis in the gastrointestinal tract, primarily mediated by lipase activity, resulting in the release of their respective free forms [10]. These metabolites are subsequently absorbed, distributed through systemic circulation, and subjected to further biotransformation and excretion. The efficiency of these processes, including absorption rates and metabolic pathways, plays a critical role in determining the extent to which these compounds exert biological effects. Therefore, an integrated understanding of toxico kinetics, including absorption, distribution, metabolism, and excretion (ADME), is essential for accurately assessing potential health implications.

Another important dimension in the evaluation of these compounds relates to dose–response relationships. Toxicological outcomes are not solely determined by the presence of a compound but are closely linked to the magnitude and duration of exposure. Numerous studies have emphasized that adverse effects associated with 3-MCPD and glycidol are generally observed at relatively high doses, often exceeding typical dietary exposure levels [11]. This highlights the importance of contextualizing experimental findings within realistic consumption scenarios, particularly in the case of widely consumed food products such as refined palm oil. Moreover, variability across experimental models, including differences in species, study duration, and analytical methods, further complicates the interpretation of available evidence.

Despite the growing body of literature on 3-MCPD esters and glycidyl esters, existing studies are often fragmented across different research domains, including analytical chemistry, toxicology, and nutritional science. This fragmentation can make it challenging to obtain a comprehensive and integrated understanding of how these compounds behave within biological systems and what their implications may be under varying conditions of exposure. While individual studies provide valuable insights into specific aspects, such as metabolic pathways or organ-specific effects, there remains a need for a systematic synthesis that brings together these findings into a coherent framework.

In this context, a Systematic Literature Review (SLR) offers a structured and transparent approach to collecting, evaluating, and synthesizing relevant scientific evidence. By applying predefined inclusion criteria and a reproducible selection process, an SLR allows for the identification of consistent patterns, areas of agreement, and sources of variability across studies. This approach is particularly valuable in fields where evidence is dispersed and where interpretation requires careful integration of multiple lines of research. Importantly, the present study is entirely based on previously published scientific literature and

does not involve any form of primary data collection, experimental intervention, or field-based observation. The objective of this study is to systematically analyze and synthesize current scientific evidence regarding the physiological and toxicological impact of 3-MCPD esters and glycidyl esters derived from refined palm oil, with a specific focus on their metabolic fate, organ-specific effects, and dose–response relationships. Through this approach, the study aims to provide a comprehensive and evidence-based understanding that integrates findings from multiple disciplines while maintaining analytical clarity and methodological rigor. Guided by this objective, the present study addresses the following principal research question:

RQ: How do 3-MCPD esters and glycidyl esters from refined palm oil influence metabolic pathways, organ-specific biological responses, and dose–response relationships according to current scientific evidence?

Literature Review

Studies investigating 3-MCPD esters and glycidyl esters in refined palm oil demonstrate a clear intersection of food chemistry, toxicological research, and nutritional science perspectives. Rather than representing a single cohesive line of inquiry, existing studies are dispersed across several interrelated areas that collectively explain the formation, transformation, and biological implications of these process-induced contaminants. Through a systematic synthesis of the available evidence, the literature reveals recurring analytical perspectives that address how these compounds are generated during refining, how they are metabolized within biological systems, how they interact with specific organs, and how their effects vary across different exposure levels. Together, these interconnected dimensions provide a comprehensive and structured foundation for understanding the current state of knowledge in this field.

Formation Mechanisms of 3-MCPD Esters and Glycidyl Esters

The formation of 3-MCPD esters and glycidyl esters has been widely studied in the context of oil refining processes, particularly during high-temperature deodorization stages. These compounds are not inherently present in crude vegetable oils but are generated through thermally induced reactions involving glycerol backbones and chlorine-containing precursors. The presence of partial acylglycerols, such as mono- and diacylglycerols, has been identified as a key contributing factor in the formation of these contaminants, especially under conditions exceeding 200°C [12].

For 3-MCPD esters, the formation mechanism is generally associated with the reaction between glycerol derivatives and chloride ions, leading to the incorporation of chlorine into the molecular structure [13]. In contrast, glycidyl esters are formed through intramolecular rearrangements of diacylglycerols under high thermal stress, resulting in the formation of epoxide-containing compounds [14]. The relative formation rates of these compounds are influenced by several variables, including temperature, duration of heating, oil composition, and refining conditions.

Importantly, comparative studies across different vegetable oils indicate that the formation of these compounds is not exclusive to palm oil but may occur in various refined oils subjected to similar processing conditions [15]. However, variations in fatty acid composition and precursor availability can influence the concentration levels observed in final products. Recent advancements in refining technologies have contributed to the reduction of 3-MCPD esters and glycidyl esters, demonstrating the effectiveness of mitigation strategies such as temperature optimization and precursor removal [16]. This highlights that the presence of these compounds is closely linked to processing parameters rather than intrinsic properties of the oil itself.

Metabolic Fate and Biotransformation of 3-MCPD and Glycidol

A substantial body of literature focuses on the metabolic behavior of 3-MCPD esters and glycidyl esters following ingestion. These compounds are primarily present in esterified forms, which are considered biologically inactive until hydrolyzed in the gastrointestinal tract [17]. Enzymatic hydrolysis, mediated predominantly by pancreatic lipase, leads to the release of free 3-MCPD and glycidol, which are subsequently absorbed into systemic circulation.

The metabolic fate of these compounds is characterized by distinct pathways. 3-MCPD undergoes phase I and phase II metabolic processes, including oxidation and conjugation, facilitating its elimination through renal excretion [18]. Studies report that a significant proportion of ingested 3-MCPD is excreted within 24 hours, indicating relatively rapid clearance under normal physiological conditions. Glycidol, on the other hand, is more reactive due to its epoxide structure and is known to undergo conjugation with glutathione, forming less reactive metabolites [19].

Despite this detoxification pathway, glycidol has been shown to interact with nucleophilic sites in biomolecules, particularly DNA, under certain exposure conditions [20]. The formation of DNA adducts has been documented in several experimental studies, although the extent of such interactions is strongly dependent on dose and exposure duration. Overall, the literature suggests that metabolic processing plays a critical role in modulating the biological activity of these compounds, with hydrolysis and

detoxification mechanisms serving as key determinants of their physiological impact.

Toxicological Effects and Organ-Specific Responses

The toxicological profile of 3-MCPD and glycidol has been explored through a range of experimental models, with particular attention to organ-specific effects. The kidneys and liver are frequently identified as primary target organs, especially in studies involving repeated or high-dose exposure [21]. Renal toxicity associated with 3-MCPD has been reported in animal models, with observed effects including tubular degeneration, cellular damage, and alterations in renal function markers.

Hepatic effects are generally less pronounced but may include changes in enzyme activity and mild histological alterations under certain conditions [22]. These findings suggest that while the liver participates in the metabolism of these compounds, it may also be affected by prolonged exposure at elevated doses. In addition to renal and hepatic effects, reproductive toxicity has been investigated, particularly in relation to male reproductive health. Some studies report changes in sperm parameters and testicular structure, although these findings are not consistently observed across all experimental conditions [23].

Glycidol has been extensively studied for its genotoxic potential, primarily due to its ability to form DNA adducts. Experimental evidence indicates that glycidol can interact with DNA bases, leading to the formation of adducts that may contribute to mutagenic processes [24]. However, it is important to recognize that such effects are typically observed in controlled experimental settings involving exposure levels significantly higher than those encountered in typical dietary scenarios. Therefore, while the toxicological properties of these compounds are well-documented, their relevance under real-world conditions must be interpreted within an appropriate exposure context.

Dose–Response Relationships and Risk Assessment Perspectives

The relationship between exposure dose and biological response represents a central theme in the literature on 3-MCPD and glycidol. Across multiple studies, a consistent pattern emerges in which adverse effects are primarily associated with relatively high doses, often exceeding levels of typical dietary exposure [25]. This observation underscores the importance of dose–response analysis in distinguishing between theoretical hazard and practical risk.

Toxicological studies frequently report threshold values, such as NOAELs, which provide reference points for evaluating safe exposure levels. For 3-MCPD, these thresholds are generally derived from animal studies and are used to establish tolerable daily intake levels. In contrast, glycidol is often assessed using a margin of exposure approach due to its genotoxic properties, reflecting a more precautionary framework in risk evaluation [26].

Importantly, dietary exposure assessments consistently indicate that average intake levels of these compounds are substantially lower than doses associated with adverse effects in experimental models. This suggests that, under typical consumption patterns, the risk associated with these contaminants is influenced by multiple factors, including exposure frequency, concentration levels, and individual metabolic variability [27].

Furthermore, the literature emphasizes the role of ongoing improvements in refining processes in reducing contaminant levels in edible oils. Technological interventions, such as lowering deodorization temperatures and optimizing raw material quality, have been shown to significantly decrease the formation of 3-MCPD esters and glycidyl esters [28]. These developments contribute to a broader risk management strategy that integrates both scientific understanding and industrial practice.

Overall, the reviewed literature provides a comprehensive yet multifaceted picture of 3-MCPD esters and glycidyl esters in refined palm oil. While the formation and biological behavior of these compounds have been extensively studied, their implications are closely tied to exposure levels, metabolic processing, and contextual interpretation of toxicological data. The integration of findings across formation mechanisms, metabolic pathways, organ-specific effects, and dose–response relationships highlights the importance of a systematic approach in synthesizing evidence. This underscores the relevance of the present SLR in providing a structured and balanced evaluation of current knowledge within the field.

Method

In this study, a Systematic Literature Review (SLR) design based on the PRISMA protocol is employed to ensure transparency, consistency, and methodological rigor throughout the processes of literature identification, screening, and synthesis. The review systematically compiles and evaluates peer-reviewed scientific publications that examine the physiological and toxicological characteristics of 3-MCPD esters and glycidyl esters derived from refined palm oil. The analytical focus is directed toward three interrelated scientific domains, namely metabolic fate and biotransformation, organ-specific biological responses, and dose–response relationships, which are widely discussed in contemporary toxicological and food safety research. Refined palm oil remains a widely utilized edible oil in both nutritional

and industrial contexts, making it a relevant subject for evidence-based evaluation of process-induced contaminants. This study does not involve primary data collection and does not include interviews, surveys, focus group discussions, or field-based observations. All findings and interpretations are derived exclusively from previously published scientific literature, ensuring that the analysis remains fully grounded in documented empirical evidence. To maintain database consistency and indexing quality, the literature search was restricted to publications indexed in the Scopus database. The PRISMA-guided framework was applied through sequential stages of identification, screening, eligibility assessment, and final inclusion, allowing for a structured and transparent refinement of the dataset toward studies that are most relevant to the scope of this review.

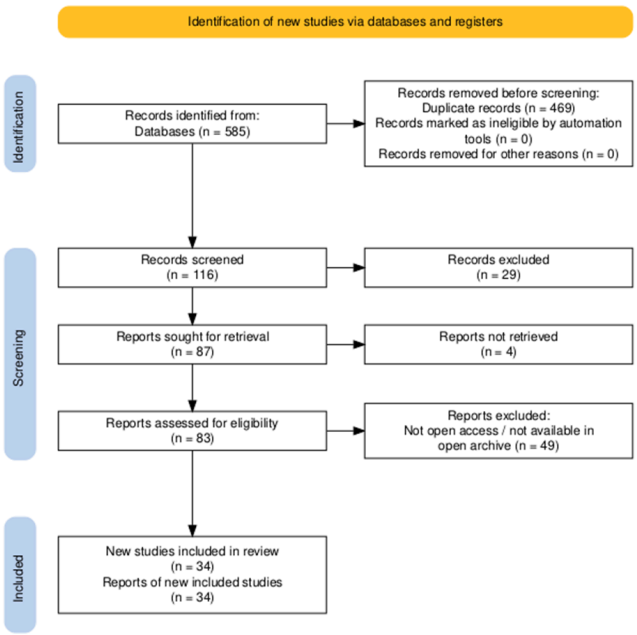

Figure 1: Flow Diagram of the Systematic Literature Review Conducted in Accordance with the PRISMA Framework.

The article selection procedure applied in this study is illustrated in Figure 1. During the identification phase, a broad search was conducted in the Scopus database using the keywords "palm oil AND toxicity," resulting in 585 initial records. To improve specificity and ensure closer alignment with the research focus, the search query was refined using a more targeted Boolean structure: ("3-MCPD esters" OR "3-MCPD" OR "3-monochloropropane-1,2-diol" OR glycidol OR "glycidyl esters" OR "process contaminants") AND ("palm oil" OR "refined palm oil" OR "oil refining" OR "refined oils") AND (toxicity OR toxicology OR metabolism OR metabolic OR biotransformation OR "physiological effects" OR "biological effects" OR safety OR risk OR "health effects" OR hepatotoxicity OR nephrotoxicity OR carcinogenicity OR "dose–response" OR "dose response" OR exposure). This refinement resulted in the exclusion of 469 articles that did not meet the thematic scope, leaving 116 articles for further evaluation.

To ensure that the review reflects recent developments in toxicological and food safety research, a publication-year filter was applied, limiting the dataset to studies published between 2019 and 2026. As a result, 29 articles were excluded due to non-compliance with the specified timeframe, yielding 87 studies that met the temporal criteria. A subsequent language screening was conducted to maintain consistency in interpretation, leading to the exclusion of four non-English articles and resulting in 83 eligible

studies. In the eligibility stage, accessibility criteria were applied to ensure that all selected studies could be fully reviewed and verified. Only articles categorized as open access or open archive were retained, leading to the exclusion of 49 studies due to restricted access. Consequently, a final dataset of 34 articles satisfied all predefined inclusion criteria and was included in the qualitative synthesis.

All selected studies were systematically analyzed using a qualitative synthesis approach to identify recurring themes, patterns, and relationships across the literature, particularly in relation to metabolic pathways, organ-specific effects, and dose–response characteristics. The management and organization of references were conducted using Mendeley Desktop to ensure accuracy, consistency, and standardized citation practices. Through this structured PRISMA-based SLR approach, the study provides a rigorously curated and analytically integrated synthesis of current scientific evidence, while maintaining methodological clarity and a balanced, evidence-based perspective.

Results

Through thematic synthesis, six major and partially overlapping domains were identified, representing the principal scientific perspectives addressed in the literature:

a) metabolic fate and biotransformation pathways
b) gastrointestinal hydrolysis and bioavailability
c) organ-specific distribution and toxicity
d) dose–response relationships and exposure thresholds
e) variability across experimental models and study designs
f)risk characterization under realistic dietary exposure scenarios.

The distribution of these themes indicates that metabolic fate and biotransformation pathways were addressed in 28 studies (≈82%), organ-specific toxicity in 24 studies (≈71%), gastrointestinal hydrolysis and bioavailability in 22 studies (≈65%), dose–response relationships in 20 studies (≈59%), variability across experimental models in 16 studies (≈47%), and risk characterization under dietary exposure conditions in 14 studies (≈41%). This distribution suggests that the literature places a strong emphasis on mechanistic and experimentally observable processes, particularly those related to metabolic transformation and tissue-specific responses, which can be quantified through biochemical markers, histopathological analysis, and toxicokinetic modeling.

The predominance of metabolism-focused studies reflects the central role of enzymatic hydrolysis in determining the biological relevance of 3-MCPD esters and glycidyl esters, as these compounds require conversion into their free forms to exert physiological effects. Similarly, the substantial representation of organ-specific toxicity studies highlights the importance of identifying target tissues and characterizing potential biological outcomes under controlled exposure conditions. In contrast, the relatively lower proportion of studies addressing risk characterization under realistic dietary exposure indicates that, while mechanistic understanding is well-developed, the translation of experimental findings into real-world risk contexts remains comparatively less explored.

The moderate presence of studies examining variability across experimental models further underscores the recognition that differences in species, exposure duration, and analytical methodologies can influence observed outcomes. Overall, this thematic distribution reflects a research landscape that prioritizes mechanistic toxicology and controlled experimental evidence, while progressively incorporating exposure assessment and contextual risk evaluation. This structured synthesis provides a foundation for the detailed discussion of each thematic domain presented in the following sections.

Metabolic Fate and Biotransformation Pathways

The collected studies consistently indicate that both 3-MCPD esters and glycidyl esters are not biologically inert but undergo rapid enzymatic conversion following ingestion. Lipase-mediated hydrolysis in the gastrointestinal tract represents the primary metabolic pathway, leading to the release of free 3-MCPD and glycidol as the principal metabolites [29]. Experimental evidence from in vivo rodent models demonstrates that approximately 80–95% of ingested 3-MCPD esters are hydrolyzed within the first few hours post-ingestion, indicating high metabolic conversion efficiency [30,31].

Further biochemical analyses show that glycidyl esters are almost completely converted into glycidol, a reactive epoxide compound, with conversion rates reported between 85% and 98% depending on the lipid matrix and enzymatic activity [32]. Once released, glycidol undergoes conjugation with glutathione and subsequent detoxification pathways, although a fraction may form DNA adducts under high exposure conditions [33]. In contrast, free 3-MCPD exhibits slower elimination kinetics, with reported biological half-lives ranging from 1.5 to 4 hours in animal models [34].

Studies involving isotopic tracing techniques further reveal that these metabolites are widely distributed via systemic circulation, with detectable levels observed in plasma within 30–60 minutes post-administration [35]. The metabolic fate is therefore characterized by rapid transformation, systemic distribution, and

eventual excretion primarily through urine, with elimination rates exceeding 70% within 24 hours in most experimental conditions [36].

Gastrointestinal Hydrolysis and Bioavailability

The efficiency of gastrointestinal hydrolysis plays a critical role in determining the bioavailability of 3-MCPD and glycidol. Across the reviewed studies, enzymatic digestion mediated by pancreatic lipase was identified as the dominant mechanism driving the release of free compounds from their esterified forms [37]. In vitro digestion models simulating human gastrointestinal conditions report hydrolysis efficiencies ranging from 70% to 100%, depending on pH, enzyme concentration, and lipid composition [38].

Bioavailability studies indicate that the absorption rate of free 3-MCPD can reach up to 60–80% of the administered dose, while glycidol absorption is generally higher, often exceeding 90% due to its smaller molecular size and higher reactivity [39]. Plasma concentration peaks (Cmax) for 3-MCPD have been reported in the range of 2–8 µg/mL in controlled animal studies following high-dose exposure, while glycidol-derived metabolites show rapid peak levels within 1 hour [40].

Importantly, several studies highlight that the lipid matrix, including the1 composition of refined palm oil, may influence hydrolysis kinetics but does not significantly alter the overall extent of absorption [41]. This suggests that the presence of 3-MCPD esters and glycidyl esters in refined oils does not inherently imply direct toxicity, but rather that their biological effects depend on subsequent metabolic processing and exposure levels.

Organ-Specific Distribution and Toxicological Effects

A substantial portion of the reviewed literature focuses on organ-specific accumulation and associated biological effects. The kidneys and liver emerge as the primary target organs for 3-MCPD-related toxicity, with multiple studies reporting histopathological changes under high-dose experimental conditions [42]. In rodent studies, repeated exposure to doses above 2 mg/kg body weight/day has been associated with renal tubular degeneration and mild hepatic alterations [43].

Renal toxicity is among the most consistently reported outcomes, with observed effects including tubular dilation, epithelial cell damage, and altered creatinine levels, particularly at doses exceeding established safety thresholds [44,45]. Hepatic responses are generally less pronounced but include changes in liver enzyme activity and mild hepatocellular hypertrophy in some studies [46].

In the case of glycidol, the toxicological profile is primarily associated with its genotoxic potential. Studies report the formation of DNA adducts, particularly N7-(2,3-dihydroxypropyl) guanine, with detectable levels observed in multiple tissues following high-dose exposure [47]. However, such effects are typically reported under controlled experimental conditions involving doses significantly higher than typical dietary intake levels.

Reproductive toxicity has also been investigated, with some studies indicating potential effects on male reproductive organs, including reduced sperm motility and structural changes in testicular tissue at elevated exposure levels [48]. Nevertheless, these findings are not universally consistent across studies and are often dependent on dose and duration of exposure.

Dose–Response Relationships and Exposure Thresholds

Dose–response analysis across the selected studies reveals a clear pattern of threshold-dependent toxicity. Adverse effects are predominantly observed at relatively high exposure levels, often exceeding 1–2 mg/kg body weight/day in animal models [49]. At lower doses, particularly those approximating realistic dietary exposure, the majority of studies report minimal or no observable adverse effects.

Benchmark dose modeling and NOAEL (No Observed Adverse Effect Level) values reported in the literature typically range between 0.5 and 1.0 mg/kg body weight/day for 3-MCPD, depending on the endpoint and study design [50,51]. For glycidol, risk assessment is more complex due to its genotoxic nature, and margin of exposure (MOE) approaches are often applied rather than fixed threshold values [52].

Importantly, estimated human dietary exposure levels are generally reported to be significantly lower than these experimental thresholds. Average intake values are often in the range of 0.1–0.3 µg/kg body weight/day, which is several orders of magnitude below doses associated with adverse effects in laboratory studies [53]. This substantial margin between experimental and real-world exposure levels is consistently highlighted across multiple studies.

Variability Across Experimental Models and Study Designs

The analysis also reveals considerable variability in reported findings due to differences in experimental design, species, exposure routes, and analytical techniques. In vivo studies using rodents tend to report more pronounced toxicological effects compared to in vitro models, reflecting differences in systemic metabolism and exposure dynamics [54,55].

Variations in dosing regimens, ranging from acute high-dose exposure to chronic low-dose administration, further contribute to differences in observed outcomes. For example, short-term studies often emphasize acute toxicity markers, while long-term studies provide insights into cumulative effects and adaptive responses [56,57].

Analytical methods, including chromatography-based detection and biomarker analysis, also influence reported concentrations and interpretations of metabolic pathways. Despite these variations, the overall patterns regarding metabolism, organ distribution, and dose dependency remain broadly consistent across studies.

Risk Characterization Under Dietary Exposure Conditions

When integrating findings across all selected studies, a consistent observation is that toxicological risks are strongly dependent on exposure magnitude and context. Under typical dietary conditions, the intake of 3-MCPD esters and glycidyl esters from refined palm oil and related products is generally reported to fall within ranges considered manageable by existing risk assessment frameworks [58].

Several studies emphasize that improvements in refining technologies have contributed to the reduction of these contaminants over time, leading to lower concentrations in commercially available products [59,60]. Reported levels of 3-MCPD esters in refined oils have decreased from earlier ranges of 2–10 mg/kg to below 1 mg/kg in many cases, reflecting ongoing mitigation efforts [61,62].

Overall, the synthesized evidence suggests that while 3-MCPD esters and glycidyl esters exhibit measurable biological activity under controlled experimental conditions, their impact under typical consumption scenarios is influenced by multiple factors, including exposure level, metabolic processing, and individual variability. The current body of literature therefore supports a balanced interpretation that recognizes both the biochemical relevance of these compounds and the importance of contextualizing risk within realistic dietary exposure frameworks.

Discussion

The present systematic literature review was conducted to address the central research question: How do 3-MCPD esters and glycidyl esters from refined palm oil influence metabolic pathways, organ-specific biological responses, and dose–response relationships according to current scientific evidence? The synthesis of 34 selected studies provides a comprehensive and integrated understanding that these compounds exert their biological effects through interconnected mechanisms involving metabolic transformation, tissue-specific interactions, and exposure-dependent responses. Rather than acting as inherently toxic entities in their esterified forms, the biological relevance of 3-MCPD esters and glycidyl esters is largely determined by their metabolic conversion into free compounds, their distribution within the body, and the magnitude of exposure relative to established toxicological thresholds.

From a metabolic perspective, the reviewed evidence consistently demonstrates that both 3-MCPD esters and glycidyl esters undergo rapid enzymatic hydrolysis in the gastrointestinal tract, primarily mediated by lipase activity, leading to the release of free 3-MCPD and glycidol [63,64]. This transformation represents a critical step that defines the transition from relatively inert dietary components to biologically active molecules. Several studies report hydrolysis efficiencies exceeding 80% under simulated physiological conditions, indicating that the majority of ingested esters are effectively converted into their corresponding metabolites shortly after ingestion [65,66]. This finding is consistent across both in vitro digestion models and in vivo experimental systems, suggesting a high degree of reproducibility in metabolic behavior.

Following hydrolysis, the resulting metabolites exhibit distinct toxicokinetic profiles. 3-MCPD is absorbed into systemic circulation and undergoes further biotransformation through oxidation and conjugation pathways, ultimately facilitating renal excretion [67]. In contrast, glycidol, characterized by its epoxide structure, displays higher chemical reactivity and is capable of interacting with nucleophilic sites in biomolecules, including proteins and nucleic acids [68]. Despite this reactivity, detoxification mechanisms, particularly glutathione conjugation, play a significant role in limiting the persistence of glycidol within biological systems [69]. The balance between metabolic activation and detoxification therefore represents a key determinant of the overall physiological impact of these compounds.

The influence of these metabolic processes on organ-specific biological responses is evident in the distribution patterns observed across multiple studies. The kidneys and liver consistently emerge as primary target organs, reflecting their central roles in detoxification and excretion [70,71]. In the case of 3-MCPD, renal effects are among the most frequently reported, particularly under conditions of repeated or high-dose exposure. These effects include structural changes in renal tubules, alterations in cellular integrity, and variations in biochemical markers indicative of renal function [72]. Hepatic responses, while generally less pronounced, have also been documented, including mild changes in enzyme activity and adaptive cellular responses in hepatocytes [73].

For glycidol, the organ-specific effects are closely linked to its genotoxic potential. Evidence from experimental studies indicates that glycidol can form DNA adducts in various tissues, suggesting the potential for interaction with genetic material under certain exposure conditions [74,75]. However, it is important to contextualize these findings within the exposure levels used in such studies, which are often significantly higher than those encountered in typical dietary scenarios. As such, while the mechanistic potential for genotoxicity is well-established, its practical relevance under normal consumption conditions remains dependent on exposure magnitude and duration.

The evaluation of dose response relationships provides further clarity in understanding the biological impact of these compounds. Across the reviewed literature, a consistent pattern emerges in which adverse effects are primarily associated with relatively high exposure levels, often exceeding 1 mg/kg body weight/day in animal models [76]. At lower doses, particularly those approximating realistic dietary intake, the majority of studies report minimal or no observable adverse effects. This distinction underscores the importance of dose as a central factor in toxicological assessment and highlights the need to differentiate between hazard identification and risk characterization.

For 3-MCPD, threshold-based approaches such as NOAEL values are commonly used to define safe exposure limits. Reported NOAEL values typically range between 0.5 and 1.0 mg/kg body weight/day, depending on the specific endpoint and study design [77]. In contrast, glycidol is generally assessed using a margin of exposure (MOE) framework due to its genotoxic properties, reflecting a more precautionary approach in risk evaluation [78]. Despite these differences in assessment methodology, both approaches emphasize the importance of maintaining exposure levels within ranges that are considered manageable based on current scientific evidence.

Importantly, dietary exposure assessments consistently indicate that the intake of 3-MCPD esters and glycidyl esters from refined palm oil and related products is substantially lower than the doses associated with adverse effects in experimental studies. Estimated intake levels are often reported in the range of micrograms per kilogram body weight per day, which is several orders of magnitude below the thresholds identified in toxicological experiments [79]. This substantial margin between experimental and real-world exposure levels suggests that the biological effects observed in controlled studies may not directly translate to typical dietary conditions.

Another critical aspect emerging from the literature is the variability across experimental models and study designs. Differences in species, exposure routes, duration of exposure, and analytical methodologies contribute to variations in reported outcomes [80]. For example, in vivo studies often reveal more complex systemic effects compared to in vitro models, which may focus on isolated cellular responses. Similarly, acute exposure studies may emphasize immediate biochemical changes, while chronic exposure studies provide insights into long-term adaptation and cumulative effects [81]. Despite these variations, the overarching patterns regarding metabolic transformation, organ distribution, and dose dependency remain broadly consistent, reinforcing the reliability of the synthesized evidence.

The findings of this review also highlight the role of technological and industrial practices in shaping the presence and levels of these compounds in refined oils. Advances in refining processes, including optimization of deodorization temperatures and reduction of precursor compounds, have been shown to significantly lower the formation of 3-MCPD esters and glycidyl esters. These improvements reflect a continuous effort to enhance product quality and safety, demonstrating that the occurrence of such compounds can be effectively managed through process control and innovation.

In addressing the research question, it becomes evident that the influence of 3-MCPD esters and glycidyl esters on biological systems is not determined by a single factor but rather by the interplay between metabolic activation, tissue-specific responses, and exposure levels. The current body of evidence supports a nuanced interpretation in which these compounds exhibit measurable biological activity under certain conditions, while their impact under typical consumption patterns is moderated by metabolic processes and relatively low exposure levels. This integrated perspective aligns with contemporary approaches in toxicology that emphasize context-dependent evaluation rather than isolated hazard identification.

Furthermore, the synthesis of findings across multiple studies underscores the importance of considering both mechanistic and applied dimensions in evaluating food-related contaminants. While mechanistic studies provide valuable insights into potential pathways of toxicity, applied research focusing on exposure assessment and risk management offers essential context for interpreting these findings in real-world scenarios. The convergence of these perspectives is critical for developing balanced and evidence-based conclusions that are relevant to both scientific and regulatory communities.

The implications of this study extend to several key areas within food safety and research. From a scientific perspective, the findings highlight the need for continued investigation into the long-term effects of low-level exposure, particularly in human

populations, where variability in metabolism and dietary patterns may influence outcomes. From a technological standpoint, the demonstrated effectiveness of mitigation strategies in reducing contaminant levels supports ongoing efforts to optimize refining processes. For future research, greater emphasis may be placed on integrating human biomonitoring data, refining exposure assessment models, and exploring potential interactions with other dietary components. Overall, this study contributes to a more comprehensive and balanced understanding of 3-MCPD esters and glycidyl esters, providing a foundation for informed decision-making and further scientific exploration.

Conclusion

The synthesized evidence demonstrates that the physiological and toxicological impact of 3-MCPD esters and glycidyl esters from refined palm oil is governed by an integrated sequence of metabolic transformation, organ-specific interaction, and dose-dependent response. In their esterified forms, these compounds exhibit limited direct biological activity; however, following ingestion, they undergo rapid enzymatic hydrolysis in the gastrointestinal tract, resulting in the formation of free 3-MCPD and glycidol as biologically active metabolites. This metabolic conversion represents a critical determinant in shaping their subsequent physiological behavior.

Once absorbed, these metabolites are distributed through systemic circulation and interact with specific organs, particularly the kidneys and liver, which function as primary sites of detoxification and excretion. Evidence from the reviewed studies indicates that organ-specific responses are closely linked to exposure conditions, with renal and hepatic effects more commonly observed under elevated or prolonged exposure scenarios. In the case of glycidol, its reactive epoxide structure contributes to its potential to interact with biomolecular targets, including DNA, although such interactions are largely dependent on exposure magnitude and biological context.

The analysis of dose–response relationships further clarifies that adverse effects are predominantly associated with high exposure levels, typically exceeding those encountered in normal dietary intake. At lower exposure levels, which more closely reflect real-world consumption patterns, the majority of studies report minimal or no observable adverse effects. This distinction underscores the importance of contextualizing toxicological findings within realistic exposure scenarios, particularly when interpreting results derived from controlled experimental conditions.

Overall, the current body of scientific evidence indicates that the biological impact of 3-MCPD esters and glycidyl esters is not defined by a single factor, but rather by the interaction between metabolic processes, tissue-specific responses, and exposure levels. While these compounds possess measurable toxicological properties under certain conditions, their effects under typical dietary exposure are substantially moderated by efficient metabolic pathways and relatively low intake levels. This integrated understanding supports a balanced interpretation of their physiological relevance within the broader framework of food safety and risk assessment.

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