UV-Induced Sterol Accumulation in Fungal Cell Membranes as a Model for Understanding Sterol-Mediated Carcinogenic Membrane Remodeling

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Abstract

Sterol-dependent membrane organization provides a mechanistic basis for examining how environmental stress modifies cellular architecture and how related biophysical processes contribute to malignant adaptation. This review critically synthesizes evidence on ultraviolet-induced fungal sterol responses and their relevance to cholesterol-associated membrane remodeling in cancer. Drawing on published experimental and review literature, it examines ergosterol biosynthesis, photochemistry, lipid interactions, membrane domain organization, oncogenic signaling, and methods for characterizing structural alterations. Comparative interpretation emphasizes distinctions between fungal stress physiology and mammalian cancer biology rather than presumed mechanistic equivalence.

The evidence establishes that ultraviolet irradiation can convert ergosterol into vitamin D_2 and other photoproducts, with outcomes influenced by wavelength, exposure conditions, fungal species, and sample preparation. However, persistent ultraviolet-induced ergosterol accumulation within fungal plasma membranes has not been consistently demonstrated. Membrane effects also depend on phospholipid composition, sterol distribution, and bilayer asymmetry. In cancer cells, altered cholesterol metabolism and compartmentalization influence receptor organization, survival signaling, migration, and therapeutic responsiveness, although these processes remain context-dependent. Accordingly, similarities between fungal and malignant membranes concern potentially shared physicochemical principles, not evidence that ultraviolet-exposed fungi reproduce carcinogenesis.

The review identifies insufficient membrane-resolved sterol measurements and limited cross-system validation as principal research gaps. Future studies should combine controlled ultraviolet dosimetry, quantitative lipidomics, photoproduct analysis, fluorescence imaging, and membrane biophysical assays with genetic perturbation and suitable controls. Parallel experiments using fungal, model-bilayer, and mammalian systems are recommended to test causal relationships. Fungal models may therefore inform membrane-targeted cancer research, provided translational claims rest on reproducible mechanistic evidence across distinct biological contexts and experimentally defined conditions rather than structural analogy.

Keywords: Ultraviolet irradiation; Ergosterol metabolism; Fungal cell membranes; Sterol-mediated membrane remodeling; Cholesterol dysregulation; Cancer cell adaptation.

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1. Introduction

1.1. Sterol Homeostasis and Cellular Membrane Architecture

Sterol homeostasis is fundamental to eukaryotic membrane architecture because sterols regulate lipid packing, permeability, mechanical stability, and the spatial organization of membrane proteins. Although fungal and mammalian cells share conserved principles of sterol regulation, their predominant membrane sterols differ: ergosterol characterizes many fungi, whereas cholesterol predominates in animal plasma membranes. These structural distinctions influence sterol interactions with neighboring phospholipids and sphingolipids, making comparative membrane analysis informative without implying functional equivalence between organisms. Sterol concentrations are maintained through coordinated biosynthesis, intracellular trafficking, uptake and redistribution, processes that determine both total sterol abundance and its availability within individual membrane compartments (Koh & Saheki, 2021).

Fungal membranes exhibit heterogeneous lateral organization rather than a uniform lipid bilayer. Their architecture incorporates discrete protein and lipid domains associated with transport, nutrient sensing, polarized growth, and stress adaptation. Ergosterol contributes to ordered membrane environments, but its distribution varies across cellular regions and physiological conditions. Investigations of fungal plasma membrane compartments demonstrate that local sterol organization is closely linked to membrane protein localization and the physical properties supporting cellular function (Athanasopoulos et al., 2019). The membrane compartment of Can1, associated with eisosome scaffolds, exemplifies this relationship: membrane curvature, lipid composition and protein organization collectively influence the stability and responsiveness of specialized surface structures (Zahumensky & Malinsky, 2019).

Sphingolipids provide an additional architectural determinant, participating in tightly packed domains whose molecular composition cannot simply be described as uniformly sterol-enriched. Indeed, certain fungal sphingolipid-rich gel domains are relatively depleted of ergosterol, illustrating that membrane order may emerge through distinct lipid arrangements. Consequently, interpreting apparent sterol accumulation requires spatially resolved measurements alongside assessment of the wider lipid environment, rather than inference from bulk sterol quantities alone (Santos et al., 2020).

Environmental conditions can alter fungal sterol regulation and membrane-associated phenotypes. Research on pathogenic cryptococci demonstrates that nitrogen availability affects ergosterol-related responses and antifungal tolerance, indicating that membrane composition responds to metabolic context (Bosch et al., 2020). Nigerian investigations of azole-resistant Candida albicans further document the detection of ERG11, encoding a central enzyme in ergosterol biosynthesis, among resistant clinical isolates. Such findings establish the biological importance of sterol synthesis pathways, although gene detection alone does not establish increased sterol accumulation or altered membrane architecture (Abdu et al., 2019).

These principles are relevant to studying ultraviolet exposure because radiation could perturb sterol chemistry, cellular redox balance and compensatory lipid regulation. However, accumulation must be distinguished experimentally from sterol oxidation, photoconversion or redistribution. In mammalian oncology, altered cholesterol distribution across plasma membrane domains has been associated with changes in signaling, cell survival and invasive behavior. These observations

provide a mechanistic comparison for investigating whether sterol-dependent architectural responses in fungi illuminate broader principles of pathological membrane remodeling (Maja & Tyteca, 2022). The resulting model should prioritize measurable similarities in lipid organization while recognizing distinct sterol species, cellular regulatory networks, and the absence of demonstrated equivalence between ultraviolet fungal responses and carcinogenesis. This comparison also demands controlled ultraviolet dosimetry, direct lipidomic quantification and imaging capable of distinguishing local membrane enrichment from generalized cellular stress.

1.2. Ultraviolet Radiation and Fungal Membrane Responses

Ultraviolet radiation constitutes an environmental stressor capable of altering fungal physiology through photochemical reactions, oxidative processes and damage to cellular macromolecules. Its biological consequences depend on wavelength, irradiation intensity, exposure duration and fungal species. Within the present review, particular attention concerns the interaction between ultraviolet energy and membrane-associated ergosterol, whose photochemical susceptibility provides a foundation for examining sterol-mediated structural alterations. Experimental investigations demonstrate that ultraviolet exposure generates ergosterol-derived compounds, including previtamin D_2, lumisterol$_2$ and tachysterol$_2$, indicating that fungal sterol profiles can undergo substantial photochemical modification without necessarily demonstrating increased membrane sterol abundance (Kalaras et al., 2012).

The consequences of irradiation extend across distinct fungal systems. Investigations involving Agaricus bisporus established that UV-C exposure converts ergosterol into vitamin D_2 while producing additional photoisomers whose relative concentrations depend on irradiation conditions. These observations indicate that apparent alterations in sterol composition must be interpreted alongside photoproduct formation, particularly when evaluating whether membrane remodeling reflects compensatory biosynthesis, sterol redistribution, or direct photochemical transformation (Sun et al., 2022).

Beyond sterol photochemistry, ultraviolet exposure influences fungal oxidative balance and associated biochemical characteristics. Analyses of irradiated Agaricus bisporus and Pleurotus ostreatus demonstrated increased vitamin D_2 concentrations alongside changes in measured antioxidant activities. Such findings establish that irradiation affects broader fungal biochemical properties, although the measured responses do not independently demonstrate membrane lipid peroxidation or altered bilayer fluidity (Gallotti & Lavelli, 2020).

Fungal resistance to ultraviolet injury also varies according to organismal characteristics and post-irradiation conditions. Experiments involving waterborne Aspergillus niger and Penicillium spores identified differences in UV-C susceptibility and photoreactivation capacity, emphasizing the importance of species-specific stress responses when interpreting radiation-induced cellular alterations (Duque-Sarango et al., 2023).

Membrane integrity represents another experimentally measurable component of fungal stress tolerance. Investigations combining ultraviolet irradiation with chlorine treatment demonstrated changes in fungal spore membrane permeability and intracellular reactive oxygen species, although these combined-treatment effects cannot be attributed exclusively to ultraviolet exposure (Wan et al., 2022).

Environmental investigations from Nigeria further demonstrate the diversity of airborne fungal spores across locations in Lagos, providing ecological context for selecting environmentally relevant fungal species for controlled irradiation experiments (Odebode et al., 2020). Integrating these observations into membrane-focused research requires distinguishing intracellular sterol accumulation from photochemical depletion, altered trafficking, and oxidative injury. Controlled comparisons of irradiated and nonirradiated fungi, incorporating sterol quantification and membrane imaging, could establish whether specific ultraviolet responses reproduce structural features associated with cholesterol-dependent remodeling in malignant cells, without assuming direct equivalence between fungal stress adaptation and carcinogenesis.

1.3. Sterol Dysregulation in Cancer-Associated Membrane Remodeling

Sterol dysregulation represents a component of cancer-associated membrane remodeling, linking metabolic reprogramming with changes in membrane organization and cellular signaling. Unlike fungal membranes, which predominantly contain ergosterol, mammalian plasma membranes depend on cholesterol to regulate lipid packing, permeability, and the spatial distribution of signaling proteins. Malignant transformation can disturb cholesterol biosynthesis, uptake, storage and efflux, thereby modifying both cellular sterol availability and the physical properties of membrane compartments. Cholesterol-enriched lipid rafts may consequently influence receptor clustering, signal transduction and resistance to apoptotic stimuli (Vona, Iessi & Matarrese, 2021).

The relationship between cholesterol abundance and malignant behavior is nevertheless context-dependent. Increased cellular cholesterol does not necessarily imply uniform enrichment across the plasma membrane, because subcellular trafficking and associations with sphingolipids determine where sterols accumulate. Cholesterol–sphingolipid assemblies can provide organized environments for growth-factor receptors and other regulatory proteins. Their dynamic restructuring may modify cellular responses to proliferation signals and therapeutic interventions, making membrane organization an actionable characteristic of cancer cells (Codini, Garcia-Gil & Albi, 2021).

Altered lipid raft organization also influences malignant dissemination. Through effects on adhesion receptors, membrane-associated proteases and cytoskeletal signaling, cholesterol-rich domains can regulate processes relevant to migration, invasion and survival during metastasis. These effects are neither universal nor exclusively dependent on total cholesterol concentration; rather, they reflect the molecular composition, localization and functional interactions of specific membrane domains (Greenlee et al., 2021).

Metabolic regulation reinforces these architectural changes. Transcriptional programs governing lipogenesis and cholesterol metabolism respond to oncogenic signaling, oxygen availability, and nutrient stress. Changes in sterol regulatory pathways can therefore support membrane biogenesis while simultaneously influencing stress adaptation and therapeutic sensitivity. Distinguishing altered cholesterol synthesis from redistribution is particularly important when interpreting tumor-associated lipid phenotypes (Jeong, Lee & Chun, 2021).

Research undertaken in South Africa has emphasized cholesterol accumulation as a contributor to cancer-cell survival and reduced treatment responsiveness, while evaluating cholesterol depletion as a possible therapeutic strategy. Such approaches require consideration of sterol dependence in healthy tissues and the heterogeneity of malignant cells (Gu et al., 2019).

Clinical evidence from Nigeria provides complementary population-level context. The MEND study identified associations between circulating lipid biomarkers and breast cancer, including subtype-dependent patterns, but circulating cholesterol measurements cannot establish membrane sterol enrichment or causation (Gupta et al., 2022). Accordingly, comparisons with ultraviolet-exposed fungal membranes should investigate measurable changes in sterol distribution, lipid order and membrane-associated signaling without assuming that fungal photochemical responses reproduce the mechanisms initiating human malignancy.

1.4. Review Objectives, Scope, and Scientific Significance

This review examines how ultraviolet exposure influences sterol composition and membrane organization in fungi and evaluates the extent to which these responses can inform understanding of sterol-dependent remodeling in cancer cells. Its primary objective is to integrate evidence from fungal physiology, membrane biophysics, sterol metabolism and cancer biology within a comparative framework that distinguishes established mechanisms from experimentally testable hypotheses.

The discussion first considers ergosterol homeostasis and the structural organization of fungal membranes, emphasizing the contributions of sterol distribution, sphingolipid interactions and membrane-associated proteins. It then evaluates ultraviolet-induced photoconversion, oxidative injury and adaptive lipid regulation, with particular attention to whether reported changes represent genuine sterol accumulation, redistribution or depletion. Subsequent analysis examines cholesterol-dependent membrane domains in malignant cells and their relationships with receptor signaling, cellular survival, motility and therapeutic response. These processes provide mechanistic reference points rather than evidence that ultraviolet-exposed fungi cause carcinogenesis.

The review also assesses experimental approaches capable of resolving membrane remodeling, including targeted sterol quantification, lipidomics, fluorescence imaging and measurements of membrane order and permeability. Methodological limitations, differences between fungal and mammalian sterols, and the influence of irradiation conditions are considered when interpreting cross-system comparisons.

Its scientific significance lies in identifying a tractable model for investigating general principles of sterol-mediated membrane adaptation while defining the boundaries of biological inference. By clarifying measurable endpoints and unresolved causal relationships, the review seeks to guide controlled investigations that distinguish photochemical sterol transformations from cellular compensatory responses and establish which membrane features, if any, have meaningful relevance to cancer-associated remodeling under relevant experimental and physiological conditions.

2. UV-Induced Sterol Metabolism in Fungal Membranes

Ultraviolet radiation alters fungal sterol metabolism through photochemical transformation and potentially through cellular responses to irradiation-induced stress. Ergosterol, the principal sterol in many fungal membranes, contributes to membrane organization, permeability, and protein function while serving as a precursor for vitamin D_2 synthesis. Its conjugated double-bond structure permits ultraviolet absorption and subsequent photochemical conversion into previtamin D_2, followed by temperature-dependent isomerization into ergocalciferol. Consequently, ultraviolet exposure can modify fungal sterol composition without necessarily increasing membrane-

associated ergosterol concentrations. Early experimental investigations established that irradiation produces measurable vitamin D_2 from endogenous ergosterol, demonstrating the susceptibility of fungal sterol pools to photochemical transformation (Koyyalamudi et al., 2009).

The extent of sterol conversion depends on irradiation wavelength, intensity, duration, and environmental conditions. Experimental optimization using ultraviolet-B radiation established that controlled exposure can substantially enhance vitamin D_2 formation in Agaricus bisporus powder. These findings indicate that photochemical efficiency is governed by interacting treatment parameters rather than irradiation duration alone (Lee & Ahn, 2016). Differences in ergosterol abundance across mushroom species further influence conversion potential. Comparative analyses of commercially dried mushrooms demonstrated substantial variation in baseline sterol concentrations and ultraviolet-induced vitamin D_2 production, emphasizing species-specific biochemical characteristics (Huang et al., 2017).

Fungal tissue organization introduces additional complexity because sterol availability and radiation penetration differ between exposed surfaces and internal cellular structures. Investigations involving white and brown Agaricus bisporus demonstrated that ultraviolet-C irradiation increased vitamin D_2 concentrations without significantly changing ergosterol abundance immediately after treatment. Subsequent cold storage altered sterol concentrations, indicating that post-irradiation conditions influence measured biochemical outcomes (Guan et al., 2016).

Experimental comparisons using Lentinus edodes and Pleurotus ostreatus identified substantial increases in vitamin D_2 following ultraviolet treatment, accompanied by declining ergosterol concentrations. Importantly, sterol depletion was not fully explained by vitamin formation, suggesting additional photochemical degradation pathways requiring further characterization (Hu et al., 2020).

The physical state of fungal material also influences sterol transformation. Comparisons of dry mushroom powders and ethanol suspensions demonstrated considerably greater vitamin D_2 production in irradiated suspensions, indicating that sterol accessibility, solvent environment and radiation distribution influence photochemical outcomes (Hu et al., 2021).

Processing conditions further complicate interpretation because drying temperature, moisture removal and irradiation exposure interact to determine residual ergosterol and accumulated vitamin D_2. Reviews of mushroom drying technologies emphasize the necessity of distinguishing photoconversion efficiency from changes attributable to processing and storage (Jiang, Zhang & Mujumdar, 2020).

These observations are particularly relevant when investigating ultraviolet-induced sterol accumulation as a proposed mechanism of fungal membrane remodeling. Existing evidence establishes photoconversion more convincingly than compensatory membrane enrichment. Determining whether living fungi increase sterol biosynthesis or redistribute existing sterols following ultraviolet injury therefore requires direct measurements of membrane fractions, biosynthetic activity, and intracellular sterol transport. Such experimental discrimination is essential for evaluating fungal membranes as comparative models of cholesterol-associated architectural remodeling in cancer cells (Leung & Cheung, 2021).

3. Molecular Mechanisms of Sterol-Mediated Membrane Remodeling

Sterol-mediated membrane remodeling encompasses molecular processes through which changes in sterol abundance, distribution, and interactions modify membrane architecture and cellular function. In fungal membranes, ergosterol maintains structural organization, influences permeability and supports membrane-associated proteins. These functions establish a biological basis for investigating whether ultraviolet-induced sterol alterations produce membrane responses resembling selected features of carcinogenic remodeling in mammalian cells (Rodrigues, 2018).

Sterols regulate membrane structure through interactions with phospholipid acyl chains. Their rigid molecular framework can restrict lipid movement, increase molecular packing, and influence bilayer mechanical resistance. Experimental investigations demonstrate that different sterols produce broadly comparable concentration-dependent effects on membrane elasticity, although their magnitudes vary with molecular structure and lipid composition (Henriksen et al., 2006). Molecular dynamics simulations further indicate that ergosterol affects phospholipid ordering, bilayer thickness, and lateral diffusion, providing molecular explanations for changes in membrane organization associated with altered sterol content (Ermakova & Zuev, 2017).

These effects are not uniform across membrane environments. Phospholipid saturation, temperature, and sterol concentration determine the extent of ergosterol-induced ordering. Experimental studies demonstrate different interactions between ergosterol and saturated or unsaturated lipids, indicating that increased sterol abundance cannot universally predict reduced membrane fluidity (Bui et al., 2019). Comparative membrane investigations similarly establish differences between ergosterol and cholesterol in their condensing effects, emphasizing the importance of sterol identity when interpreting similarities between fungal and mammalian membranes (Hung et al., 2016).

Sterol-dependent lateral organization represents another important remodeling mechanism. Differences in sterol–lipid interactions influence the formation, morphology, and stability of membrane domains. Experimental observations demonstrate that ergosterol-containing and cholesterol-containing membranes display distinct domain structures and dynamic behavior, potentially affecting molecular compartmentalization (Galván-Hernández et al., 2020). Related investigations show that both fungal and mammalian sterols can support liquid-ordered phases, although differences in their organization limit direct extrapolation between biological systems (Khmelinskaia et al., 2020).

Membrane remodeling additionally depends on sterol distribution between bilayer leaflets. Studies of yeast plasma membranes indicate that ergosterol is predominantly associated with the cytoplasmic leaflet, demonstrating that fungal sterol organization is spatially asymmetric (Solanko et al., 2018). Cholesterol distribution in mammalian membranes is similarly influenced by lipid interactions and transbilayer transport. Such asymmetry contributes to membrane organization and may influence sterol accessibility, protein interactions, and local mechanical behavior (Steck & Lange, 2018).

Plain black-and-white scientific schematic showing fungal membrane responses to UV exposure, sterol-driven changes in membrane architecture, and cancer-associated membrane remodeling.

Conceptual illustration of ergosterol photoconversion, oxidative membrane injury, sterol redistribution, and their potential relationships with membrane order, permeability, protein localization, and cholesterol-associated signaling. The fungal–cancer comparison represents a mechanistic hypothesis rather than established causal equivalence.

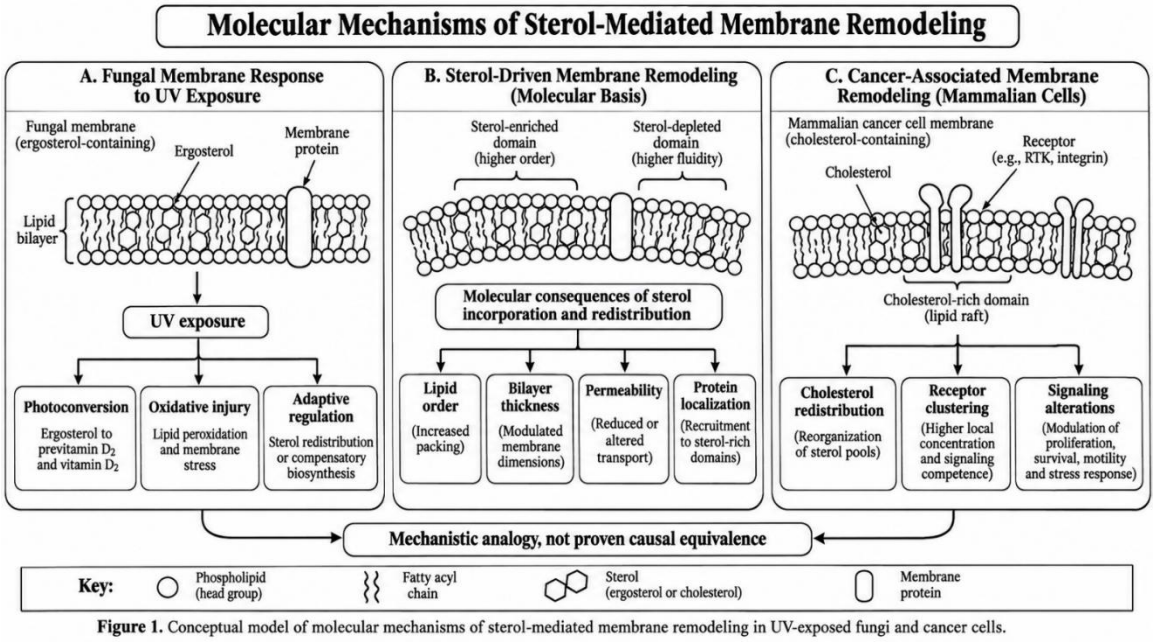


Figure 1. Conceptual model of molecular mechanisms of sterol-mediated membrane remodeling in UV-exposed fungi and cancer cells.

Figure 1. Molecular Mechanisms of Sterol-Mediated Membrane Remodeling

Within ultraviolet-exposed fungi, these molecular relationships suggest several pathways through which membrane architecture could change. Direct ergosterol photoconversion may alter available sterol pools, while oxidative injury could disrupt phospholipid organization and membrane integrity. Compensatory sterol synthesis or redistribution might subsequently restore membrane function, but such responses require experimental confirmation rather than assumption. Distinguishing these mechanisms demands quantitative sterol profiling, membrane-order measurements, and spatial analysis of sterol localization.

The relevance to carcinogenic remodeling arises from shared principles governing lipid organization rather than identical disease mechanisms. In malignant mammalian cells, cholesterol redistribution can influence membrane microdomains and associated signaling processes, whereas fungal ultraviolet responses principally reflect photochemical and cellular stress phenomena. Comparative investigations should therefore establish whether measurable changes in bilayer packing, permeability and protein localization follow similar physicochemical relationships. Integrating controlled irradiation experiments with lipidomics, fluorescence microscopy and membrane biophysical measurements would clarify the extent to which fungal systems provide informative experimental models of sterol-dependent membrane remodeling without attributing carcinogenic properties to fungal adaptation.

4. Fungal Sterol Responses and Carcinogenic Membrane Alterations

Fungal sterol responses provide a comparative framework for examining how changes in membrane lipid composition influence cellular adaptation and pathological remodeling. Ergosterol maintains fungal membrane organization, whereas cholesterol regulates analogous physicochemical properties in mammalian cells. Both sterols contribute to membrane permeability, protein distribution and stress responsiveness, although their regulatory pathways and biological consequences differ. In fungi, ergosterol biosynthesis responds to oxygen availability, metabolic conditions and transcriptional control, illustrating how environmental disturbances can modify membrane sterol homeostasis (Jordá & Puig, 2020).

Sterol redistribution represents an important adaptive mechanism. Investigations of Saccharomyces cerevisiae demonstrate that phospholipid flippases and the membrane protein Sfk1 facilitate ergosterol retention within the plasma membrane. Disruption of these components produces increased membrane permeability and intracellular sterol redistribution, establishing a relationship between lipid asymmetry and membrane integrity (Kishimoto et al., 2021). Such findings suggest that ultraviolet-associated membrane disturbances could alter sterol localization independently of changes in total cellular sterol concentrations.

Fungal membrane remodeling also influences polarized growth and stress tolerance. Experimental studies involving Aspergillus fumigatus demonstrate that cytochrome b5 supports ergosterol biosynthesis and sterol-rich membrane domain accumulation at hyphal tips. Its disruption impairs membrane fluidity and hyphal extension, indicating that sterol organization contributes to cellular morphology and functional adaptation (Zhang et al., 2021).

Oxidative processes introduce another mechanism connecting fungal membrane physiology with pathological lipid remodeling. Ergosterol exhibits antioxidant properties and can protect lipid assemblies against oxidation, suggesting that its concentration and molecular environment influence resistance to oxidative injury (Dupont, 2021). Conversely, experimental ergosterol depletion in Penicillium expansum following bifonazole treatment produces membrane damage, elevated reactive oxygen species, and mitochondrial dysfunction. These observations demonstrate consequences of pharmacologically induced sterol deficiency, rather than establishing identical effects following ultraviolet irradiation (Yang et al., 2022).

Comparable alterations in cholesterol regulation influence malignant cellular behavior. Cholesterol homeostasis contributes to membrane composition, metabolic adaptation, and susceptibility to regulated cell death. Experimental cancer models demonstrate that cholesterol dysregulation can increase resistance to ferroptosis, promoting tumorigenicity and metastatic potential under particular biological conditions (Liu et al., 2021).

Membrane microdomain organization additionally regulates processes associated with cancer progression. Investigations involving leukemia cells show that cholesterol depletion disrupts lipid raft integrity and impairs cellular migration, revealing the functional importance of membrane cholesterol organization (Bobkov et al., 2021). Cholesterol-enriched signaling platforms also facilitate the assembly of inflammatory signaling components, including those associated with interleukin-6 pathways, which can influence oncogenic signaling environments (Mollinedo, 2022).

5. Sterol-Dependent Signaling and Cancer Cell Adaptation

Sterol-dependent signaling constitutes a critical interface between membrane architecture, metabolic regulation, and malignant cellular adaptation. Cholesterol influences cancer progression not merely as a structural membrane constituent but through its capacity to regulate receptor organization, intracellular signaling and metabolic homeostasis. Malignant cells frequently exhibit altered cholesterol synthesis, uptake, efflux and esterification, enabling them to accommodate proliferative demands and environmental pressures. These adaptations are regulated through sterol-responsive transcriptional pathways and oncogenic signaling networks that influence growth, survival and invasive potential (Liu et al., 2023).

Cholesterol-enriched membrane microdomains provide specialized environments for organizing signaling proteins, including growth-factor receptors, integrins and intracellular signaling complexes. Their composition influences receptor interactions and signal propagation through pathways associated with proliferation, apoptosis and metastasis. Nevertheless, lipid raft functions depend on cholesterol distribution and membrane composition rather than total cellular cholesterol alone. Changes in microdomain integrity can therefore either promote or impair malignant signaling, depending on the molecular context (Li et al., 2022).

Interactions between cholesterol metabolism and the phosphatidylinositol 3-kinase–protein kinase B pathway further illustrate this regulatory relationship. Experimental investigations demonstrate that loss of the Fbw7 tumor suppressor alters sterol regulatory element-binding protein activity, promotes cholesterol accumulation, and activates AKT signaling. These findings establish a mechanistic connection between disrupted cholesterol homeostasis and oncogenic survival pathways (Bengoechea-Alonso, Aldaalis & Ericsson, 2022).

Sterol oxidation products also participate in cellular signaling regulation. Investigations involving prostate cancer cells demonstrate that 27-hydroxycholesterol can disrupt cholesterol-rich membrane organization and suppress interleukin-6-mediated JAK–STAT3 signaling. This observation illustrates that sterol metabolites may produce biological effects distinct from those associated with cholesterol enrichment (Dambal et al., 2020).

Therapeutic manipulation of cholesterol metabolism has consequently attracted attention as a strategy for modifying oncogenic signaling. Cholesterol-lowering interventions may influence AKT activation and related survival mechanisms, although their anticancer effectiveness depends on tumor characteristics, treatment exposure and metabolic compensation (Kumar & Mandal, 2021).

Within the tumor microenvironment, interactions between malignant cells, stromal populations and immune components further shape lipid availability and signaling responses. Metabolic reprogramming can support therapeutic resistance and adaptation to nutrient limitation, emphasizing the importance of investigating sterol regulation within its broader cellular context (Kim, Song & Yim, 2023).

6. Experimental Models and Membrane Characterization Techniques

Experimental investigation of ultraviolet-induced sterol remodeling requires integrated methodologies capable of distinguishing photochemical transformations, adaptive sterol regulation,

and changes in fungal membrane architecture. Living fungal cultures, isolated membrane fractions, and artificial lipid bilayers offer complementary experimental systems for evaluating these mechanisms. Quantitative determination of ergosterol is particularly important because changes in total sterol abundance do not necessarily reflect membrane-specific accumulation. Optimized extraction procedures, supported by chromatographic analysis, provide a foundation for measuring ergosterol concentrations across fungal samples while controlling analytical variability (Alekseyeva et al., 2021).

Genetic and biochemical models enable investigation of the functional consequences of altered sterol metabolism. Experiments involving Candida albicans demonstrate that ergosterol availability influences plasma membrane composition and the localization and activity of membrane-associated transport proteins. Combining sterol perturbation with protein localization measurements can therefore clarify relationships between membrane composition and cellular function (Suchodolski et al., 2019).

Fluorescence-based techniques provide additional information concerning membrane organization. Laurdan generalized polarization measurements can characterize changes in lipid packing, while fluorescence anisotropy offers complementary information concerning molecular mobility. These approaches are valuable for evaluating membrane responses following irradiation, provided their measurements are not interpreted as direct evidence of sterol accumulation (Harris, Best & Bell, 2002). Standardized experimental interpretation is equally important because membrane fluidity, lipid order and generalized polarization describe related but distinct physicochemical properties (Jay & Hamilton, 2017).

Table 1. Experimental Models and Analytical Techniques for Investigating UV-Induced Sterol Remodeling

Experimental model or technique	Principal measurements	Relevance to fungal–cancer membrane comparisons
Living fungal cultures	Ergosterol abundance, viability, stress recovery	Identifies adaptive responses following UV exposure
Isolated membrane fractions	Membrane-associated sterol concentration	Distinguishes membrane enrichment from intracellular redistribution
Artificial lipid bilayers	Lipid packing, permeability, membrane order	Evaluates sterol-dependent biophysical properties
HPLC and LC–MS/MS	Ergosterol, vitamin D_2 and sterol photoproducts	Separates accumulation from photoconversion
Fluorescence microscopy and spectroscopy	Membrane order, sterol localization, protein distribution	Characterizes structural and functional membrane changes
Computational imaging and biosensors	Morphological features, biochemical signals	Supports standardized experimental quantification

Source: Author's conceptual synthesis of experimental approaches discussed in the cited literature. The table proposes a combined methodological framework; it does not report results from a single experimental study.

Computational image analysis offers opportunities to improve morphological assessment and experimental reproducibility. Automated feature recognition and template-matching approaches

provide adaptable principles for extracting structural information from biological images, although membrane-specific applications require independent validation against established imaging methods (Ejofodomi et al., 2014). Similarly, artificial intelligence integrated with biosensor technologies may facilitate biological signal classification and high-throughput analysis when appropriate analytical specificity and validation procedures are established (Olorunkosebi et al., 2023).

7. Translational Implications, Research Limitations, and Future Directions

The translational significance of fungal sterol research lies in its capacity to clarify conserved membrane biophysical mechanisms while distinguishing fungal stress adaptation from mammalian oncogenic processes. Fungal systems offer experimentally accessible platforms for investigating sterol metabolism, membrane stability, and responses to environmental disturbances. However, their application to cancer biology requires careful consideration of differences between ergosterol and cholesterol, particularly regarding biosynthetic regulation, membrane distribution, and associated signaling pathways. Studies of fungal sterylglucosides demonstrate that sterol modification influences membrane properties and cellular stress responses, highlighting the importance of examining sterol derivatives alongside unmodified sterols (Pereira de Sá & Del Poeta, 2022).

Ultraviolet irradiation provides an experimentally controllable intervention for examining sterol photochemistry. Research involving Pleurotus florida demonstrates that ultraviolet-B exposure promotes ergosterol conversion into vitamin D_2, with measurable biochemical consequences. Nevertheless, photoconversion should not be interpreted as evidence of increased membrane sterol accumulation. Future investigations must distinguish direct photochemical reactions from compensatory biosynthesis, intracellular redistribution, and oxidative membrane injury (Kaur et al., 2023).

Potential translational applications include identifying membrane characteristics associated with altered sterol availability and evaluating their susceptibility to therapeutic manipulation. Research on membrane-targeting antifungal compounds demonstrates that disrupting fungal membrane organization can influence cellular survival and drug responsiveness. These findings support the use of fungal models for mechanistic screening, although antifungal selectivity cannot establish corresponding anticancer efficacy (Mehta, Saini & Bajaj, 2023).

In cancer research, cholesterol biosynthesis and trafficking constitute potential therapeutic targets because malignant cells may depend on altered sterol regulation. Investigations of sterol synthesis inhibitors identify enzymes such as HMG-CoA reductase and squalene epoxidase as candidates for therapeutic intervention, although biological heterogeneity complicates clinical translation (Feltrin et al., 2020). Complementary evidence indicates that cholesterol metabolism influences tumor survival, immune regulation, and treatment responsiveness, warranting further evaluation of context-dependent therapeutic vulnerabilities (Ediriweera, 2022).

Membrane-specific signaling presents an additional comparative opportunity. Experimental breast cancer research demonstrates that lipid raft-associated protein organization facilitates stable HER2 signaling complexes, illustrating how membrane architecture can influence oncogenic processes (Jeong et al., 2021).

Important limitations include differences between fungal and mammalian membrane composition, uncertainties surrounding ultraviolet-induced ergosterol accumulation, and inadequate validation of cross-species mechanisms. Future research should integrate controlled irradiation protocols, quantitative lipidomics, fluorescence microscopy, and functional membrane assays. Standardized sample preparation, analytical validation and reproducible data interpretation are essential for translating lipid measurements into biologically meaningful evidence (Vvedenskaya et al., 2022). Comparative studies should prioritize matched physicochemical endpoints, independent replication, and mechanistic interventions before advancing therapeutic claims. Such investigations could establish whether fungal membrane responses provide useful experimental analogues for selected features of carcinogenic remodeling without implying direct equivalence between ultraviolet injury and malignant transformation.

Conclusion

This review examined the extent to which ultraviolet-associated changes in fungal sterol metabolism can illuminate the biophysical principles underlying membrane remodeling in malignant cells. Its objectives were addressed by integrating evidence on ergosterol homeostasis, photochemical transformation, lipid organization, experimental characterization, and cholesterol-dependent oncogenic signaling. The synthesis identifies membrane sterol composition, spatial distribution, and interactions with phospholipids and proteins as central determinants of permeability, lipid order, domain organization, and cellular adaptation.

A principal finding is that ultraviolet irradiation can convert fungal ergosterol into vitamin D_2 and other photoproducts, whereas sustained sterol accumulation within fungal plasma membranes remains insufficiently demonstrated. Apparent increases in sterol abundance must therefore be distinguished from redistribution, compensatory synthesis, and changes in extraction or measurement. Studies of fungal membranes also show that sterol effects depend on lipid composition, bilayer asymmetry, and environmental conditions, limiting simple predictions about membrane rigidity or functional outcomes.

Cancer-related evidence reveals that cholesterol metabolism and membrane compartmentalization influence receptor organization, survival signaling, migration, and therapeutic responsiveness. Nevertheless, these associations do not establish that ultraviolet-exposed fungi reproduce carcinogenic mechanisms. The comparative value of fungal models lies instead in experimentally isolating shared physicochemical relationships while recognizing differences in sterol structure, cellular regulation, and pathological context.

Future investigations should employ standardized ultraviolet dosimetry, matched irradiated and control cultures, validated sterol and photoproduct quantification, and spatially resolved membrane imaging. Genetic or pharmacological perturbations should test whether observed structural changes result from sterol synthesis, trafficking, or oxidation. Parallel experiments in defined lipid bilayers and relevant mammalian cell models would strengthen mechanistic comparisons. Independent

replication, dose-response analyses, and transparent reporting are essential before translational claims can be justified. Such evidence could guide precise hypotheses about membrane-targeted therapeutic vulnerabilities. Overall, fungal systems offer a promising but bounded platform for investigating sterol-dependent membrane adaptation, with clinical relevance contingent upon direct causal validation rather than structural analogy alone.

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