ISSN 0026-2617, Microbiology, 2011, Vol. 80, No. 3, pp. 297–306. © Pleiades Publishing, Ltd., 2011.
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REVIEW

The Role of Lipids in the Morphogenetic Processes of Mycelial Fungi

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Received October 12, 2010

Abstract—This review considers the role of different classes of lipids in fungal morphogenesis and, in particular, their capacity to induce dimorphic growth (the ability of fungi to grow in a mycelial or yeastlike form), which is induced by various factors and results in changes in the biosynthetic and energy processes, as well as in structural and morphological changes. The review includes a brief description of the properties of lipids and their functions in the cell and discusses the specific characteristics of lipid metabolism associated with morphogenesis and dimorphism. The differences in lipid compositions between yeastlike cells and mycelium; the role of the structural and regulatory lipids, fatty acids, and their derivatives in morphological transformation of fungi; and the involvement of lipids in signal transduction and host–pathogen interactions are discussed.

Keywords: lipids, fungi, dimorphic growth, morphogenesis, mycelium, yeastlike cells.

DOI: 10.1134/S0026261711030155

INTRODUCTION

Dimorphism—the ability of fungi to switch from mycelial to yeastlike growth—can be induced by numerous biotic and abiotic factors and results in changes in almost all biosynthetic and energy processes, which, in turn, result in structural and morphological changes, adaptive in their nature and aimed at the preservation of viability under varying conditions [1–11].

According to present-day ideas, the formation of yeastlike and hyphal cells of fungi may be described by using the "steady-state" model [12] and the hyphoid model based on the so-called vesicle supply center (VSC) concept [13, 14]. According to the first model, the main components of the cell wall (chitin and glucan) are synthesized at the hyphal apex; there are no covalent bonds between them, which makes the cell wall flexible and easily changeable. Elongation of a hypha takes place only if the cross-linkage of chitin with glucan and chitin crystallization occur. The second model is based on the idea that, at the hyphal apex, there is a special center controlling the flow rate of the vesicles that transport the substrates required for the synthesis of the cell wall. These macrovesicles (Spitzenkörper) appear as dark areas under a light microscope. If the flow rate of microvesicles (chitosomes) is high enough, hyphae elongate and the mycelium develops. F-actin and myosin-5 play a significant role in hyphal elongation [15]. The results of phylogenetic analyses of rRNA and conservative proteins are of special interest. These data demonstrate that the apical growth of fungi is similar to the growth of plant pollen tubes and root fibrils [16].

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There is a strong dependence between lipid synthesis and the key processes involved in the metabolism, growth, and viability of fungi. The role of lipids in the vital functions of fungi—in particular, in morphogenesis—is considered with regard to their involvement in cellular processes as both structural and storage compounds, factors for adaptation under unfavorable conditions, and regulatory compounds [17–24].

Mycelial and yeastlike forms of dimorphic fungi, many of which are pathogenic to plants, animals, and human beings, exhibit differing virulence [25–30]. Therefore, thorough investigation of the biosynthesis and metabolism of lipids (including sterols) and the regulation of these processes have great theoretical value and are of considerable importance for modern medicine, veterinary science, agriculture, and biotechnology.

According to present-day concepts, high concentrations of demethylated sterols, including ergosterol, contribute to the maintenance of the structure and normal functioning of the membranes and correlate with mycelial growth of fungi [31]. The role of sterols in the morphogenetic processes and dimorphic growth of fungi was discussed in our previous review [32], as well as in the work by Martin and Konopka [33] and the review by Alvares et al. [34], discussing the role of membrane sterol–glycolipid complexes ("rafts" or sterol-rich domains) in the arrangement and functioning of cell membranes, in particular, in transport processes, expression of virulence factors, and determination of the cell polarity in clinically significant dimorphic fungi (*Saccharomyces cerevisiae, Criptococcus neoformans, Candida albicans, Aspergillus nidulans,* and *Schizosaccharomyces pombe*). Review by Steinberg [15],

describing a wide range of factors which determine and support polarized (mycelial) growth, contains data on the participation of sterol-rich membrane domains in the formation of hyphae by facilitating apical endocytosis and maintaining the actin cytoskeleton [15].

This review deals with the role of different classes of lipids in fungal morphogenesis and, in particular, in the dimorphism phenomenon.

1. BRIEF DESCRIPTION OF LIPIDS: FUNCTION, BIOSYNTHESIS, REGULATION

Functions of lipids

The chemical structure, physical properties, and functions of fungal lipids are extremely diverse, which is absolutely essential for the cell viability, adaptation to changing environmental conditions, and maintenance of the optimal level of metabolic processes. The main function of lipids is the nonspecific bulk function, which is subdivided into structural/functional and storage parts, and the specific regulatory functions.

Formation of a semitransparent cellular barrier is well known as the primary function of lipid membranes. Moreover, they serve as a matrix for the proteins involved in such important processes as energy production, signal transduction, transport, DNA replication, secretion, regulation of activity of membrane-bound enzymes, etc. The lipid bilayer of cytoplasmic membranes contains phospholipids (PLs), glycolipids (GLs), and sterols.

The morphogenesis of living cells largely depends on the phase state of the membrane lipids, which are responsible for the membrane fluidity (matrix function), stability (mechanical function), and permeability (barrier function). Several phase transitions are known; the transition from the liquid-crystal state to the gel state, when the bilayer structure of the membrane remains intact, while the membrane fluidity changes, has been studied in greatest detail. A membrane can exist in a hexagonal or cubic state; during these phase transitions, the membrane fluidity remains intact, but the bilayer structure changes. Depending on its physiological state, the cell is able to control the arrangement of its membrane by the regulation of the lipid composition of the membrane. For instance, hexagonal and cubic phases appear at high temperatures or low water content [35]. During recent years, phase transitions in both phospholipids and sterols (including cholesterol and ergosterol, which, along with sphingolipids, are the components of the domain structures known as the lipid rafts mentioned above) have attracted intense attention of the researchers [33, 34]. Rafts can be visualized by light microscopy using the polyene antibiotic filipin for specific staining of 3-β-oxysterols. This made it possible to reveal that rafts are located in growing hyphal tips. Inhibition of sphingolipid production in *Aspergillus nidulans* with myriocin resulted in cessation of the hyphal growth and emergence of lateral branching, which indicated that rafts are involved in the polarized growth of fungi [36]. Rafts differ not only in the content of individual lipids and their qualitative composition, but also in size (from 10–100 to 1000 μm). Lipid domains, consisting primarily of sterols (SRDs rafts) are larger in size, actively participate in the morphogenesis of fungi, and are situated specifically in hyphal tips. SRDs rafts were not detected in the budding yeast *Candida albicans*, but were found in the mycelial form of this fungus. Hence, the presence of sterol-containing lipid domains in the membrane may be regarded as one of the mechanisms responsible for morphogenesis [34].

Phospholipids, amphipathic molecules that form the basis of the cell membranes, are precursors of macromolecules [37, 38], molecular chaperones [39]; they participate in protein modification [40], are reservoirs of secondary messengers [41, 42], stabilize the lipid bilayer structure under heat shock conditions [43], and mediate resistance to toxic compounds [44].

It has been reported that, in addition to their trivial functions, lipids are an important component of not only cell membranes, but also chromosomes, the DNA–membrane complex, and nuclear material; they are involved in the regulation of DNA functioning via direct interaction with the DNA structure and due to changes in the activity of the enzymes of nucleic acid metabolism [45–48].

The storage lipids of fungi and yeasts are represented primarily by triacylglycerols (TAGs) and sterol esters (SEs), are essential for the total metabolism of acylglycerols, act as an operating reserve of fatty acids in phospholipid biosynthesis, and are arranged mainly as lipid granules [49–52]. Moreover, there is evidence that small amounts of these lipids can be found in other cellular compartments (microsomes, mitochondria, and nuclei); they are synthesized in situ and have a specific fatty acid composition [53, 54], which indicates that spatially separate TAG pools exist and their functions are not limited to just reserve functions.

The role of lipids in signal transduction is discussed in Section 3 of the review.

Biosynthesis and regulation

Phosphatidylcholine (PC), phosphatidylethanolamine (PEA), phosphatidylserine (PS), and cardiolipin (CL) are the main membrane phospholipids. The pathways of their biosynthesis, which were studied using primarily *S. cerevisiae* as a model, are the same for all fungi and yeasts. Two pathways of PL synthesis exist: the CDP–DAG pathway and the DAG (Kennedy) pathway [55, 56]. The biosynthesis of TAG and phospholipids via the Kennedy pathway progresses in a similar way up to the stage of diacylglycerol (DAG) production [49].

Regulation of these pathways is a complicated process controlled by various genetic and biochemical mechanisms. Inositol plays a key role in the regulation

of the PL synthesis; the inositol-mediated regulation includes gene expression and modulation of the enzymatic activity. Phosphorylation is the main mechanism regulating the activities of transcription factors and of the enzymes of PL biosynthesis by posttranslational modification. Protein kinase A phosphorylates choline kinase, Mg^{2+}-dependent phosphatidate phosphatase, PS synthase, and the Opi1p transcription factor. In addition, the activities of CTP synthase and Opi1p are regulated by protein kinase C [55–59]. The activities of many enzymes involved in PL biosynthesis are also regulated by some phospholipids, sphingolipids, and nucleotides [60].

The enzymes of the PL biosynthesis are located primarily in microsomes, mitochondria, lipid granules, whereas the nuclear and cytoplasmic membranes, vacuoles, and peroxisomes contain only trace amounts of these enzymes [61]. Catabolism of phospholipids is mediated by phospholipases A_1, A_2, C, and D [49, 62].

2. ASPECTS OF LIPID METABOLISM RELATED TO MORPHOGENESIS AND DIMORPHISM

It is well known that chitin synthesis is essential for cell wall formation and cell differentiation in micromycetes. As mentioned above, chitin synthesis is mediated by chitin synthetase, the activity of which depends on the lipid environment. The cell membrane, responsible for polyuronide synthesis, and chitosomes, the membrane organelles directly involved in chitin synthesis, play the key role in this process, as well as in the process of chitin deposition in the cell wall [63, 64]. Nevertheless, it is unclear how these membrane structures affect the formation of the cell wall typical of hyphae and yeastlike cells.

It was demonstrated that membrane-bound chitinolytic enzymes, including endochitinase and N-acetyl-D-glucosaminidase, contribute greatly to the morphological changes during the dimorphic yeasts ⟶ hyphae transformations of the pathogenic zygomycete *Benjaminiella poitrasii* [65]. There is evidence that the phospholipid environment may affect the activity of membrane-bound chitinase in *Mucor mucedo* [66, 67]. Thus, morphogenesis may be influenced by the lipid composition of the membrane. Nevertheless, data on the relations between lipids and morphogenesis are sometimes contradictory.

2.1. Lipids of Yeastlike Cells and Mycelium

Study of the lipid composition of the yeastlike and mycelial forms of *M. genevensis* [68] demonstrated that, in the total lipids of *M. genevensis* hyphae, the levels of sterols and fatty acids were higher than in yeastlike cells, and unsaturated fatty acids were predominant. The fatty acids of yeastlike cells were more saturated. However, when phenylethanol induced yeastlike growth under aerobic conditions, no significant differences were detected in the lipid composition as compared to the mycelium, which prevented unambiguous conclusions regarding the dependence of dimorphism on the lipid composition.

The study of the *M. rouxii* lipids [25, 69] revealed that the lipid content in the fractions of cytoplasmic membranes was higher and more diverse than that in the cell wall. According to the previously published data [68], the concentrations of sterols and fatty acids in the lipids of the *M. rouxii* mycelium developed under aerobic conditions were also higher than in the yeastlike cells grown under anaerobic conditions. The lipids from the fractions of the cytoplasmic membranes of yeastlike cells grown under aerobic conditions in the presence of phenylethanol were similar to the lipids of the mycelium grown without phenyl ethanol. However, the lipid composition of the cell walls of *M. rouxii* cells grown under aerobic conditions in the presence of phenylethanol was similar to that of yeastlike cells grown under anaerobic conditions. The results obtained do not allow us to draw an unambiguous conclusion regarding a direct correlation and demonstrate that the relationships between lipids and morphogenesis are complicated and indirect.

2.2. Inhibition of Lipid Biosynthesis

The use of the antibiotic cerulenin in a number of studies made it possible to clarify the role of lipids in fungal morphogenesis [70–76].

The polyene antibiotic cerulenin inhibits fatty acid synthetase, thereby blocking lipid synthesis. According to some authors, this antibiotic inhibits only fatty acid synthesis [71]; according to others, it inhibits synthesis of both fatty acids and sterols [70, 72, 76], as well as the TAG synthesis by the complete suppression of [^{14}C]-acetate incorporation into sterols and TAG and inhibition of acetate incorporation into fatty acids [73].

The inhibitory effect of cerulenin manifested itself in suppression and complete inhibition of spore germination and growth of yeasts and fungi, *S. cerevisiae* [71, 72], *Botryodiplodia theobromae* [73], and *Epidermophyton floccosum* [76], as well as in the inhibition of spore formation by *S. cerevisiae* [72].

The results obtained by different authors on various models, despite their fundamental similarity, have not shown complete uniformity. According to Ohno et al. [72], the inhibitory effect of cerulenin was neutralized by the introduction of various fatty acids (especially oleic and pentadecanoic acids) into the medium or, to a lesser extent, by sterols. According to Sanadi et al. [76], the inhibitory effect of the antibiotic was completely neutralized by addition of palmitic acid and sterols, and partially neutralized by addition of unsaturated fatty acids. According to Brambl et al. [73], the inhibitory effect was neutralized by the addition of Twin 40 and Twin 60 (derivatives of palmitic and

stearic acids, respectively), but not by free fatty acid or their salts. Interestingly, according to the data obtained by the latter authors, cerulenin had no significant effect on the morphology of fungal mitochondria.

Hence, the results obtained, despite certain peculiarities, demonstrate that lipid synthesis is essential for spore formation. Moreover, it was shown that lipids are important for dimorphic growth, which expands the adaptive capabilities of fungi.

There is evidence in the literature regarding the role of lipids in dimorphic growth of mucor fungi (*M. racemosus*) [75]. After transfer of the anaerobically grown yeastlike fungal cells into aerobic conditions, a transition to mycelial growth was observed, which was accompanied by an increase in both the growth rate and the rate of lipid synthesis. In the presence of sublethal concentrations of cerulenin, the morphological yeasts ⟶ hyphae transformation of fungal cells was not detected after the cells were transferred to aerobic conditions; the cells continued to grow in the form of multipolar budding yeastlike cells. At the same time, lipid synthesis, ornithine decarboxylase activity, and, to a lesser extent, RNA and protein synthesis were inhibited. Not only was fatty acid synthesis inhibited by cerulenin, but also phospholipid synthesis, the rate of which normally increases during morphogenesis. Moreover, the synthesis of phosphatidylcholine and phosphatidylethanolamine was inhibited to a greater degree than the synthesis of cardiolipin, the main phospholipid of mitochondria. The inhibitory effect of the antibiotic was neutralized by addition of Twin 80 (a mixture of fatty acids) into the medium, which suggested that the increase in the rate of fatty acid synthesis and in the rate of phospholipid turnover is necessary for the morphological transition from yeastlike to mycelial growth [75].

As was reported earlier, regulatory lipids, which do not fulfill the structural function in the cell, are involved in morphological transformations. For instance, oxidized derivatives of fatty acids may act as regulators of the morphological transformations of mycelial fungi that are associated with their virulence and is of importance for host–parasite relationships. The study of the phytopathogenic fungus *Ceratocystis ulmi*, which contains oleic, linoleic, and linolenic acids, but not arachidonic acid, demonstrated that the lipoxygenase inhibitors gossypol, nordihydroguaiaretic acid, and propyl gallate induced the dimorphic mycelium ⟶ yeast transformation, whereas the inhibitors of cyclooxygenase and thromboxane synthetase had no such effect [77]. These findings suggested the conclusion that, due to the activity of phytoalexins (plant-derived antimicrobial agents), the above-listed lipids are involved in the regulation of morphogenesis during the dimorphic transformation of fungi.

3. THE ROLE OF LIPIDS IN SIGNAL TRANSDUCTION: LIPIDS ACTING AS SIGNAL MOLECULES

Numerous studies on the role of lipids in signal transduction in cells have been published in which mammalian and plant cells were primarily used as model objects [reviews 62, 78–81]. Nevertheless, despite some differences, the pathways of signal transduction in which lipid molecules are involved are generally the same in the cells of all living organisms.

The transmembrane signal transduction may proceed through ionic channels, kinase receptors, or receptor-activated enzyme inducers that form intracellular secondary messengers. Due to the fact that enzyme inducers are located on the cell surface, the messengers are often formed from membrane components, which are mostly phospholipids. The messengers transmit signals to kinases in various cascades; transcription factors and protein synthesis are then activated, and a physiological response, which can be involved in morphogenetic differentiation and other processes, is initiated. In addition, some physical or chemical signals can directly interact with the lipids of the cell membrane and induce its modification resulting in an alteration of the conformation of the receptor protein and induction of the signal system.

All secondary lipid messengers are formed from the main membrane precursors, phosphatidylcholine (PC) and sphingomyelin (SM). Phosphatidylcholine is hydrolyzed via the receptor-mediated stimulation of the specific phospholipase C with formation of DAG or via phospholipase D stimulation with formation of phosphatidic acid (PA). Then, PA can be converted to lyso-PA under the influence of type A phospholipase or to DAG under the influence of phosphatidate phosphohydrolase. Phosphoinositide-specific phospholipase C acting on PI-4,5-diphosphate is also capable of producing DAG.

The PA, lyso-PA, and DAG produced under the influence of phospholipases are involved in the mediation of many cellular functions [82–84]. DAG is an activator of protein kinase C, whereas PA and lyso-PA have a stimulatory effect on various kinases, activate the Ras-MAPK pathway of signal transduction, and have an effect on the proliferative properties of the cell. Both SM and PC are hydrolyzed by phospholipase C with formation of phosphorylcholine and ceramide, which, in turn, is also a secondary messenger inhibiting phospholipase D activity and thereby participating in the regulation of cell differentiation and other processes [78]. Hence, opposite effects of lipids were observed: DAG stimulated protein kinase C activity (in the presence of Ca^{2+} and phospholipids), whereas the sphingolipids' derivative sphingenine exerted an inhibitory effect.

Various fungi and yeasts, *Ustilago maydis, Candida lipolytica, Aspergillus niger*, etc., exhibited high activities of phospholipases, lipases, and esterases during

the processes of spore formation and germination, as well as during all morphological transformations [24, 85–88]. There is evidence that products of phospholipid decomposition, namely, PA, DAG, and lyso-PA, are messengers of the dimorphic growth in *C. albicans* [89]. It was demonstrated for *S. cerevisiae* that fungal phospholipases play a key role in many pathways of signal transduction and are essential for spore formation [90, 91].

Phospholipase D generates the lipid signals responsible for membrane transport and signal transduction in yeasts and is regulated by phosphoinositides [92]. As mentioned above, the main substrate of phospholipase D is phosphatidylcholine, which is hydrolyzed to phosphatidic acid, which acts as a secondary messenger [93–95] and regulates the production of phosphatidylinositol-4,5-diphosphate [96, 97]. In addition, PA is hydrolyzed to DAG by phosphatidate phosphohydrolase [98, 99]. In a number of studies, the involvement of phospholipase D in dimorphic transformations of the yeastlike form of *C. albicans* to the mycelial form was demonstrated [89, 100].

DAG, another lipid responsible for signal transduction, is additionally produced due to the activity of phospholipase C [41]; it activates protein kinase C [101] and plays an important role in the production and transportation of secretory vesicles from the Golgi complex in the course of *S. cerevisiae* budding [102, 103].

From the published data, it may be concluded that certain lipids, including fatty acid derivatives, along with the above-mentioned functions, are universal regulators in living organisms and exert a multifactor influence on the morphogenesis of fungal plant and mammal pathogens.

For instance, it was demonstrated that plant surface lipids (waxes) are signal molecules in plant–fungus interactions and induce the pathogenic phase of the development of such fungi as *Colletotrichum* sp. [104, 105]. According to Kolattukudi et al. [106], plant surface lipids contain both inducers and inhibitors of spore germination and appressorium (critical for plant infection) formation.

According to other authors, modified fatty acids or structurally related lipids play an important role in the sexual and asexual development of *Neurospora crassa* [107, 108]. For example, linoleic acid and derivatives thereof are involved in the spore formation of *Aspergillus* spp. [109–110] and *Sclerotinia fructicola* [111]. The signal molecules of hydroxylated fatty acids, the so-called psi factors, affected growth, as well as spore formation and aflatoxin production in *A. nidulans* [112, 113], whereas the psi factor representing an oleic acid derivative controlled the ratio between sexual and asexual spores of this species [114]. Moreover, plant fatty acids (hydroperoxylinoleic) are also responsible for the development of *Aspergillus* spp. spores [115].

Mutants of the mycotoxigenic fungi *A. flavus, A. parasiticus*, and *A. nidulans* (pathogens of oil-bearing seeds) deficient in Δ12-desaturase made it possible to reveal the association between the ability to synthesize polyunsaturated fatty acids and fungal development [111]. Mutations in the gene encoding Δ12-desaturase delayed spore germination caused a twofold decrease in the growth rates and inhibited spore formation, as well as made the mutants unable to produce sclerotia. The authors suggest that unsaturated fatty acids act as signals inducing the development of asexual conidiospores, sexual ascospores, and/or sclerotia.

Using the phytopathogenic fungus *Ustilago maydis*, the ability of which to switch from yeastlike to mycelial growth is essential for infection and completion of the life cycle, it was demonstrated that growth in the presence of lipids (corn oil) resulted in the development of a mycelial phenotype very similar to the phenotype that was observed during infection of the host organism [24]. This effect was exerted by the fatty acids produced by fungal extracellular lipases from TAG. The ability of fungi to respond to lipids depends on two signaling pathways, the cAMP/PKA-dependent pathway and the Ras/MAPK pathway, which regulate fusion, mycelial growth, and pathogenesis, whereas lipids may act as one of the signals that induce and maintain the mycelial growth required for invasion and development in the host organism.

The human pathogens *C. albicans* and *Cryptococcus neoformans* are able to produce prostaglandins from both exogenous and endogenous arachidonic acid [21–23]. Prostaglandins, oxygenated derivatives of polyunsaturated fatty acids, increased the viability of *C. albicans* cells and their capacity for pseudohyphal growth. These data indicate that the fatty acid derivatives formed by fungal cyclooxygenases serve as signal molecules and mediators of direct host–pathogen interactions.

CONCLUSIONS

Lipids, structural components of cell membranes, play an important role in the processes of cell metabolism, including membrane transport, as well as in oxidative phosphorylation [116], regulation of the activity of membrane-bound enzymes affecting the cell morphology [60], and the cell wall formation [34, 35]. Moreover, since they form the bulk of storage compounds and are involved in the acyl metabolism, lipids contribute to the maintenance of the metabolic processes and play an important role in signal transduction as the growth conditions change. In recent publications, phenol lipids of different origin and their biological activities in respect to various living organisms, including fungi and yeasts, have received intense scientific attention [116]. The mechanisms of action of resorcinol lipids are diverse and include direct interaction with cell membranes, as well as modulation of the activity of integral membrane proteins and of the

enzymes responsible for free radical formation; their involvement in the inhibition of 3-phosphoglycerate dehydrogenase (the key enzyme of TAG synthesis) and growth inhibition, as well as in their interactions with microbial DNA, was demonstrated [118–120].

Hence, studies of the role of lipids in morphogenetic processes will make it possible to develop new approaches for targeted regulation of growth of both clinically significant and phytopathogenic species and to use the biotechnological potential of fungi more extensively. Fungi are widely used in environmental biotechnologies and various biotechnological processes, are promising producers of pharmacologically active lipid compounds (ergosterol, carotenoids, unsaturated fatty acids, etc.), and are believed to be a good source of alternative biofuel. Investigations of the regulation of the morphogenetic processes and dimorphic growth of fungi are required in order to improve the efficiency of producer strains, and to select optimal growth conditions while retaining high production capacity.

ACKNOWLEDGMENTS

This work was supported by the Russian Foundation for Basic Research, project no 10-04-00659.

REFERENCES

1. Bartnicki-Garcia, S., Symposium on Biochemical Bases of Morphogenesis in Fungi. III. Mould-Yeast Dimorphism of *Mucor*, *Bacteriol. Rev.*, 1963, vol. 27, pp. 293–304.
2. Sypherd, P.S., Borgia, P.T., and Pasnokas, J.L., Biochemistry of Dimorphism in the Fungus *Mucor, Adv. Microbiol. Physiol.*, 1978, vol. 18, pp. 67–104.
3. Leija, A., Ruiz-Herrera, J., and Mora, J., Effect of L-amino acids on *Mucor rouxii* dimorphism, *J. Bacteriol.*, 1986, vol. 168, pp. 843–850.
4. Orlowski, M., *Mucor* Dimorphism, *Microbiol. Rev.*, 1991, vol. 55, no. 2, pp. 234–258.
5. Serrano, O., da Silva, T.L., and Roseiro, J.C., Ethanol-Induced Dimorphism and Lipid Composition Changes in *Mucor fragilis* Ccmi-142, *Lett. Appl. Microbiol.*, 2001, vol. 33, no. 1, pp. 89–93.
6. da Silva, T.L., Pinheiro, H.M., and Roseiro, J.C., Stress-Induced Morphological and Physiological Changes in γ-Linolenic Acid Production by *Mucor fragilis* in Batch and Continuous Cultures, *Enz. Microbial Technol.,* 2003, vol. 40, pp. 1321–1327.
7. Medentsev, A.G., Fain, M.E., Aitkhozhina, N.A., Nikitina, E.T., and Akimenko, V.K., Energy Metabolism in the Fungus *Fusarium bulbigenus* during Transition From Mycelial to Yeastlike Growth, *Biokhimiya*, 1992, vol. 57, no. 3, pp. 389–397.
8. Funtikova, N.S., Mysyakina, I.S., and Poglazova, M.N., Fatty Acid and Lipid Composition of *Mucor lusitanicus* in Relation to Its Dimorphic Growth under Extreme Conditions, *Mikrobiologiya*, 1998, vol. 67, no. 4, pp. 485–491 [*Microbiology* (Engl. Transl.), vol. 67, no. 4, pp. 401–406].
9. Funtikova, N.S., Mysyakina, I.S., and Poglazova, M.N., Morphogenesis and Lipid Composition of *Mucor* Fungi Grown in the Presence of Chloroanilines in Submerged Culture, *Mikrobiologiya*, 1999, vol. 68, no. 4, pp. 467–472 [*Microbiology* (Engl. Transl.), vol. 68, no. 4, pp. 406–411].
10. Mysyakina, I.S. and Funtikova, N.S., Activity of NAD-Dependent Isocitrate Dehydrogenase, Isocitrate Lyase, and Malate Dehydrogenase in *Mucor circinelloides* var. *lusitanicus* INMI under Different Modes of Nitrogen Supply, *Mikrobiologiya*, 2008, vol. 77, no. 4, pp. 453–459 [*Microbiology* (Engl. Transl.), vol. 77, no. 4, pp. 400–406].
11. Mysyakina, I.S. and Funtikova, N.S., Metabolic Characteristics and Lipid Composition of Yeastlike Cells and Mycelium of *Mucor circinelloides* var. *lusitanicus* INMI Grown at a High Glucose Content in the Medium, *Mikrobiologiya*, 2008, vol. 77, no. 4, pp. 460–464 [*Microbiology* (Engl. Transl.), vol. 77, no. 4, pp. 407–411].
12. Wessels, J.G.H., Cells Wall Synthesis in Apical Hyphal Growth, *Rev. Cytol.*, 1986, vol. 104, pp. 37–79.
13. Bartnicki-Garcia, S., Bartnicki, D.D., Gierz, G., Lopez-Franco, R., and Bracker, C.E., Evidence That Spitzenkorper Behavior Determines the Shape of a Fungal Hypha: a Test of Hyphoid Model, *Exp. Mycol.*, 1995, vol. 19, pp. 153–159.
14. Feofilova, E.P., The Fungal Cell Wall: Modern Concepts of Its Composition and Biological Function, *Mikrobiologiya*, 2010, vol. 79, no. 6, pp. 723–733 [*Microbiology* (Engl. Transl.), vol. 79, no. 6, pp. 711–720].
15. Steinberg, G., Hyphal Growth: a Tale of Motors, Lipids and Spitzenkörper, *Eukaryotic Cell*, 2007, vol. 6, no. 3, pp. 351–360.
16. Geitman, A. and Emons, A.M., The Cytoskeleton in Plant and Fungal Cell Tip Growth, *J. Microsc.*, 2000, vol. 198, pp. 218–245.
17. Nozawa, Y. and Kasai, R., Mechanism of Thermal Adaptation of Membrane Lipids in *Tetrahymena pyriformis* NT-1. Possible Evidence for Temperature-Mediated Induction of Palmitoyl-CoA Desaturase, *Biochim. Biophys. Acta*, 1978, vol. 529, no. 1, pp. 54–66.
18. Rao, T.V., Trivedi, A., and Prasad, P., Phospholipid Enrichment of *Saccharomyces cerevisiae* and Its Effect on Polyene Sensitivity, *Can. J. Microbiol.*, 1985a, vol. 31, no. 4, pp. 322–326.
19. Rao, T.V., Das, S., and Prasad, P., Effect of Phospholipid Enrichment on Nystatin Action: Differences in Antibiotic Sensitivity Between *in vivo* and *in vitro* Conditions, *Microbios*, 1985b, vol. 42, nos. 169–170, pp. 145–153.
20. Noverr, M.C., Phare, S.M., Toews, G.B., Coffey, M.J., and Huffnagle, G.B., Pathogenic Yeasts *Cryptococccus neoformans* and *Candida albicans* Produce Immunomodulatory Prostaglandins, *Infect. Immun.*, 2001, vol. 69, pp. 2957–2963.
21. Noverr, M.C., Erb-Downward, J.R., and Huffnagle, G.B., Production of Eicosanoids and Other Oxylipins by Pathogenic Eukaryotic Microbes, *Clin. Microbiol. Rev.,* 2003, vol. 16, no. 3, pp. 517–533.

22. Noverr, M.C. and Huffnagle, G.B., Regulation of *Candida albicans* Morphogenesis by Fatty Acid Metabolites, *Infect. Immun.*, 2004, vol. 72, no. 11, pp. 6206–6210.

23. Klose, J., de Sa, M.M., and Kronstad, J.W., Lipid-Induced Filamentous Growth in *Ustilago maydis, Mol. Microbiol.,* 2004, vol. 52, no. 3, pp. 823–835.

24. Jeennor, S., Laoteng, K., Tanticharoen, M., and Cheevadhanarak, S., Comparative Fatty Acid Profiling of *Mucor rouxii* under Different Stress Conditions, *FEMS Microbiol. Lett.*, 2006, vol. 259, no. 1, pp. 60–66.

25. Kobayashi, S.D. and Cutler, J.E., *Candida albicans* Hyphal Formation and Virulence: Is There a Clearly Defined Role?, *Trends Microbiol.*, 1998, vol. 6, pp. 92–94.

26. Bahn, Y.S., Staab, J., and Sundstrom, P., Increased High-Affinity Phosphodiesterase PDE2 Gene Expression in Germ Tubes Counteracts CAP1-Dependent Synthesis of Cyclic AMP, Limits Hypha Production and Promotes Virulence of *Candida albicans, Mol. Microbiol.*, 2003, vol. 50, no. 2, pp. 391–409.

27. Andrews, D.L., Garcia-Pedrajas, M.D., and Gold, S.E., Fungal Dimorphism Regulated Gene Expression in *Ustilago maydis*: I. Filament Up-Regulated Genes, *Mol. Plant Pathol.*, 2004, vol. 5, pp. 281–293.

28. Ruiz-Herrera, J., Elorza, M.V., Valentin, E., and Sentandreu, R., Molecular Organization of the Cell Wall of *Candida albicans* and Its Relation to Pathogenicity, *FEMS Yeast Res*, 2006, vol. 6, p. 14.

29. Nadal, M., Garcia-Pedrajas, M.D., and Gold, S.E., Dimorphism in Fungal Plant Pathogens, *FEMS Microbiol. Lett.,* 2008, vol. 284, no. 2, pp. 127–134.

30. Klein, B.S. and Tebbets, B., Dimorphism and Virulence in Fungi, *Curr. Opin. Microbiol.*, 2007, vol. 10, no. 4, pp. 314–319.

31. Vanden Bossche, H., Importance and Role of Sterols in Fungal Membranes, in *Biochemistry of Cell Walls and Membranes in Fungi*, Kuhn, P.J., Trinci, A.P.J., Jung, M.J., Goosey, M.W., and Copping, L.G., Eds., Berlin: Springer, 1990, pp. 135–157.

32. Mysyakina, I.S. and Funtikova, N.S., The Role of Sterols in Morphogenetic Processes and Dimorphism in Fungi, *Mikrobiologiya*, 2007, vol. 76, no. 1, pp. 5–18 [*Microbiology* (Engl. Transl.), vol. 76, no. 1, pp. 1–13].

33. Martin, S.W. and Konopka, J.B., Lipid Raft Polarization Contributes to Hyphal Growth in *Candida albicans, Eukariotic Cell*, 2004, vol. 3, no. 3, pp. 675–684.

34. Alvares, F.J., Douglas, L.M., and Konopka, J.B., Sterol-Rich Plasma Membrane Domains in Fungi, *Eukariotic Cell*, 2007, vol. 6, no. 5, pp. 755–763.

35. Antonov, V.F., Lipid Pores, *Sorosovskii Obrazovatel'nyi Zh.*, 1998, vol. 10, pp. 10–17.

36. Cheng, J., Park, T.-S., Fischl, A.S., and Ye, X.S., Cell Cycle Progression and Cell Polarity Require the Sphingolipid Biosynthesis in *Aspergillus nidulans, Mol. Cell Biol.*, 2001, vol. 21, pp. 6198–6209.

37. Becker, G.W. and Lester, R.L., Biosynthesis of Phosphoinositol-Containing Sphingolipids from Phosphatidylinositol by a Membrane Preparation from *Saccharomyces cerevisiae, J. Bacteriol.*, 1980, vol. 142, no. 3, pp. 747–754.

38. Lester, R.L. and Dickson, R.C., Sphingolipids with Inositolphosphate-Containing Head Groups, *Adv. Lipid Res.*, 1993, vol. 26, pp. 253–274.

39. Bogdanov, M., Umeda, M., and Dowhan, W., Phospholipid-Assisted Refolding of an Integral Membrane Protein. Minimum Structural Features for Phosphatidylethanolamine to Act as a Molecular Chaperone, *J. Biol. Chem.*, 1999, vol. 274, pp. 12339–12345.

40. Ichimura, Y., Kirisako, T., Takao, T., Satomi, Y., Shimonishi, Y., Ishihara, N., Mizushima, N., Tanida, I., Kominami, E., Ohsumi, M., Noda, T., and Ohsumi, Y., A Ubiquitin-Like System Mediates Protein Lipidation, *Nature*, 2000, vol. 408, no. 6811, pp. 488–492.

41. Exton, J.H., Signaling through Phosphatidylcholine Breakdown, *J. Biol. Chem.*, 1990, vol. 265, pp. 1–4.

42. Exton, J.H., Phosphatidylcholine Breakdown and Signal Transduction, *Biochim. Biophys. Acta*, 1994, vol. 1212, no. 1, pp. 26–42.

43. Beck, J.B., Mathieu, D., Loudet, C., Buchoux, S., and Dufourc, E.J., Plant Sterols in "Rafts": a Better Way to Regulate Membrane Thermal Shocks, *The FASEB J.,* 2007, vol. 21, pp. 1714–1723.

44. Mishra, P. and Kaur, S., Lipids as Modulators of Ethanol Tolerance in Yeast, *Appl. Microbiol. Biotechnol.*, 1991, vol. 34, pp. 697–702.

45. Struchkov, V.A. and Strazhevskaya, N.B., DNA-Bound Lipids: Composition and Possible Functions, *Biokhimiya*, 1993, vol. 58, no. 8, pp. 1154–1175.

46. Strazhevskaya, N.B., Mulyukin, A.L., Shmyrina, A.S., Kraus, A., Lorents, V., Zhdanov, R.I., and El'-Registan, G.I., Characteristics of *Pseudomonas aurantiaca* DNA Supramolecular Complexes at Various Developmental Stages, *Mikrobiologiya*, 2009, vol. 78, no. 1, pp. 59–67 [*Microbiology* (Engl. Transl.), vol. 78, no. 1, pp. 48–55].

47. Albi, E. and Viola Magni, M.P., The Role of Intracellular Lipids, *Biol. Cell.*, 2004, vol. 96, pp. 657–667.

48. Zhdanov, R.I., Shmyrina, A.S., Zarubina, T.V., Mulyukin, A.L., El-Registan, G.I., Haupt, N., Kraus, A., and Lorenz, W., Nature of DNA-Bound Fatty Acids in *Pseudomonas aurantiaca, FEMS Microbiol. Letts.,* 2006, vol. 265, no. 2, pp. 151–158.

49. Chopra, A. and Khuller, G.K., Lipids of Pathogenic Fungi, *Prog. Lipid Res.*, 1983, vol. 22, pp. 189–220.

50. Sorger, D. and Daum, G., Synthesis of Triacylglycerols by the Acyl-Coenzyme A:Diacylglycerol Acyltransferase Dga1p in Lipid Particles of the Yeast *Saccharomyces cerevisiae, J. Bacteriol.*, 2002, vol. 184, no. 2, pp. 519–524.

51. Daum, G., Wagner, A., Czabany, T., Grillitsch, K., and Athenstaedt, K., Lipid Storage and Mobilization Pathways in Yeast, *Novartis Found. Symp.,* 2007, vol. 286, pp. 142–151.

52. Daum, G., Wagner, A., Czabany, T., and Athenstaedt, K., Dynamics of Neutral Lipid Storage and Mobilization in Yeast, *Biochimie*, 2007, vol. 89, no. 2, pp. 243–248.

53. Davidova, E.G., Belov, A.P., Balashova, L.D., and Zaitsev, S.A., Lipid Composition of Subcellular Structures of the Yeasts Grown under Intense Lipogenesis, *Mikrobiologiya*, 1986, vol. 55, no. 4, pp. 576–581.

54. Zinchenko, G.A., Belov, A.P., and Davidova, E.G., Intracellular Distribution and Composition of Yeast

Triacylglycerols, *Mikrobiologiya*, 1989, vol. 58, no. 6, pp. 934–937.

55. Iwanyshyn, W.M., Han, G.-S., and Carman, G.M., Regulation of Phospholipid Synthesis in *Saccharomyces cerevisiae* by Zinc, *J. Biol. Chem.*, 2004, vol. 279, pp. 21976–21983.
56. Carman, G.M. and Kersting, M.C., Phospholipid Synthesis in Yeast: Regulation by Phosphorylation, *Biochem. Cell Biol.*, 2004, vol. 82, no. 1, pp. 62–70.
57. Jiranek, V., Graves, J.A., and Henry, S.A., Pleiotropic Effects of the *opi1* Regulatory Mutation of Yeast: Its Effects on Growth and on Phospholipid and Inositol Metabolism, *Microbiology (UK)*, 1998, vol. 144, no. 10, pp. 2739–2748.
58. Choi, M.G., Parks, T.S., and Carman, G.M., Phosphorylation of *Saccharomyces cerevisiae* CTP Synthetase at Ser424 by Protein Kinases A and C Regulates Phosphatidylcholine Synthesis by the CDP-Choline Pathway, *J. Biol. Chem.*, 2003, vol. 278, no. 26, pp. 23610–23616.
59. Carman, G.M. and Han, G.S., Regulation of Phospholipid Synthesis in *Saccharomyces cerevisiae* by Zinc Depletion, *Biochim. Biophys. Acta*, 2007, vol. 1771, no. 3, pp. 322–330.
60. Daum, G., Lees, N.D., Bard, M., and Dickson, R., Biochemistry, Cell Biology and Molecular Biology of Lipids of *Saccharomyces cerevisiae, Yeast,* 1998, vol. 14, pp. 1471–1510.
61. Zinser, E., Sperka-Gottlieb, C.D.M., Fasch, E.-V., Kohlwein, S.D., Paltauf, F., and Daum, G., Phospholipid Synthesis and Lipid Composition of Subcellular Membranes in the Unicellular Eukaryote *Saccharomyces cerevisiae, J. Bacteriol.*, 1991, vol. 173, no. 6, pp. 2026–2034.
62. Munnik, T., Irvine, R.F., and Musgrave, A., Phospholipid Signaling in Plants, *Biochim. Biophys. Acta,* 1998, vol. 1389, pp. 222–272.
63. Dow, J.M., Carreon, R.R., and Villa, V.D., Role of Membranes of Mycelial *Mucor rouxii* in Synthesis and Secretion of Cell Wall Matrix Polymers, *J. Bacteriol.*, 1981, vol. 145, no. 1, pp. 272–279.
64. Bartnicki-Garcia, S., Role of Chitosomes in the Synthesis of Fungal Cell Walls, in *Microbiology-1981*, Schlessinger, D., Ed., Washington, DC: Amer. Soc. Microbiol., 1981, pp. 238–241.
65. Ghormade, V.S., Lachke, S.A., and Despande, M.V., Dimorphism in *Benjaminiella poitrasii*: Involvement of Intracellular Endochitinase and N-Acetylglucosaminidase Activities in the Yeast-Mycelium Transition, *Folia Microbiol.*, 2000, vol. 45, no. 3, pp. 231–238.
66. Humphreys, A.M., Phospholipid Requirement of Microsomal Chitinase from *Mucor mucedo, Curr. Microbiol.*, 1984a, vol. 11, pp. 187–190.
67. Humphreys, A.M. and Gooday, D.W., Properties of Chitinase Activity from *Mucor mucedo*: Evidence for Membrane-Bound Zymogenic Form, *J. Gen. Microbiol.*, 1984b, vol. 130, pp. 1359–1366.
68. Gordon, P.A., Stewart, P.R., and Clark-Walker, G.D., Fatty Acid and Sterol Composition of *Mucor genevensis* in Relation to Dimorphism and Anaerobic Growth, *J. Bacteriol.*, 1971, vol. 107, no. 1, pp. 114–120.
69. Safe, S. and Caldwell, J., The Effect of Growth Environment on the Chloroform-Methanol and Alkali-Extractable Cell Wall and Cytoplasmic Lipid Levels of *Mucor rouxii, Can. J. Microbiol.,* 1975, vol. 21, pp. 79–84.
70. Nomura, S., Horiuchi, T., Omura, S., and Hata, T., The Action Mechanism of Cerulenin. I. Effect of Cerulenin on Sterol and Fatty Acid Biosynthesis in Yeast, *J. Biochem.*, 1972, vol. 71, no. 5, pp. 783–796.
71. Greenspan, M.D. and Mackow, R.C., The Effect of Cerulenin on Sterol Biosynthesis in *Saccharomyces cerevisiae, Lipids*, 1977, vol. 12, no. 9, pp. 729–740.
72. Ohno, T., Awaya, J., and Omura, S., Inhibition of Sporulation by Cerulenin and Its Reversion by Exogenous Fatty Acids in *Saccharomyces cerevisiae, Antimicrob. Agents Chemother.*, 1976, vol. 9, no. 1, pp. 42–48.
73. Brambl, R., Wenzler, H., and Josephson, M., Mitochondrial Biogenesis During Fungal Spore Germination: Effects of the Antilipogenic Antibiotic Cerulenin Upon *Botryodiplodia* Spores, *J. Bacteriol.*, 1978, vol. 135, no. 2, pp. 311–317.
74. Daum, G., Gamerith, G., and Paltauf, F., The Effect of Cerulenin and Exogenous Fatty Acids on Triacylglycerol Accumulation in an Inositol-Deficient Yeast, *Saccharomyces carlsbergensis, Biochim. Biophys. Acta*, 1979, vol. 573, no. 2, pp. 413–415.
75. Ito, E., Cihlar, R.L., and Inderlied, C.D., Lipid Synthesis during Morphogenesis in *Mucor racemosus, J. Bacteriol.*, 1982, vol. 152, pp. 880–887.
76. Sanadi, S., Pandey, R., and Khuller, G.K., Reversal of Cerulenin-Induced Inhibition of Phospholipids and Sterol Synthesis by Exogenous Fatty Acids/Sterols in *Epidermophyton floccosum, Biochim. Biophys. Acta*, 1987, vol. 921, no. 2, pp. 341–346.
77. Jensen, E.C., Ogg, C., and Nickerson, K.W., Lipoxygenase Inhibitors Shift the Yeast/Mycelium Dimorphism in *Ceratocystis ulmi, Appl. Environ. Microbiol.*, 1992, vol. 58, no. 8, pp. 2505–2508.
78. Gomez-Müoz, A., Modulation of Cell Signaling by Ceramides, *Biochim. Biophys. Acta*, 1998, vol. 1391, pp. 92–109.
79. Shears, S.B., The Versatility of Inositol Phosphates as Cellular Signals, *Biochim. Biophys. Acta*, 1998, vol. 1386, pp. 49–67.
80. Tarchevskii, I.A. and Chernov, V.M., Molecular Aspects of Phytoimmunity, *Mikol. Fitopatol.*, 2000, vol. 34, no. 3, pp. 1–10.
81. Grechkin, A.N. and Tarchevskii, I.A., The Cellular Signaling Systems and the Genome, *Bioorg. Khim.*, 2000, vol. 26, no. 10, pp. 779–781 [*Russ. J. Bioorg. Chem.,* (Engl. Transl.), vol. 26, no 10, pp. 702–704].
82. Moolenaar, W.H., Lisophosphatidic Acid, a Multifunctional Phospholipid Messenger, *J. Biol. Chem.,* 1995, vol. 270, pp. 12949–12952.
83. Rudge, S.A., Morris, A.J., and Engebrecht, J., Relocalisation of Phospholipase D Activity Mediates Membrane Formation during Meiosis, *J. Cell Biol.*, 1998, vol. 140, no. 1, pp. 81–90.
84. Xie, Z., Fang, M., Rivas, M.P., Faulkner, A.J., Sternweis, P.C., Engebrecht, J.A., and Bancaitis, V.A., Phospholipase D Activity Is Required for Suppression of Yeast Phosphatidilinositol Transfer Protein Defects,

Proc. Natl. Acad. Sci. USA, 1998, vol. 95, pp. 12346–12351.

85. Lloyd, G.I., Morris, E.O., and Smith, J.E., A Study of the Esterases and Their Function in *Candida lipolytica, Aspergillus niger* and a Yeast-Like Fungus, *J. Gen. Microbiol.*, 1970, vol. 63, no. 2, pp. 141–150.
86. Gadd, G.M, Signal Transduction in Fungi, in *The Growing Fungus,* Gow, N.A.R. and Gadd, G.M., Eds., London: Chapman & Hall, 1995, pp. 183–210.
87. Fu, Y., Ibrahim, A.S., Fonzi, W., Zhou, X., Ramos, C.F., and Ghannoum, M.A., Cloning and Characterization of a Gene (LIP1) Which Encodes a Lipase from the Pathogenic Yeast *Candida albicans, Microbiology (UK)*, 1997, vol. 143, no. 2, pp. 331–340.
88. Kumar, C.P., Menon, T., Sundararajan, T., Nalini, S., Thirunarayan, M.A., Rajasekaran, S., and Venkatadesikalu, M., Esterase Activity of *Candida* Species Isolated from Immunocompromised Hosts, *Rev. Iberoam Mycol*, 2006, vol. 23, no. 2, pp. 101–103.
89. McLain, N. and Dolan, J.W., Phospholipase D Activity Is Required for Dimorphic Transition in *Candida albicans, Microbiology (UK)*, 1997, vol. 143, pp. 3521–3526.
90. Ella, K.M., Dolan, J.W., and Meier, K.E., Characterization of a Regulated Form of Phospholipase D in the Yeast *Saccharomyces cerevisiae, Biochem. J.*, 1995, vol. 307, pp. 799–805.
91. Ella, K.M., Dolan, J.W., Qi, C., and Meier, K.E., Characterization of *Saccharomyces cerevisiae* Deficient in Expression of Phospholipase D, *Biochem. J.*, 1996, vol. 314, pp. 15–19.
92. Sciorra, V.A., Rudge, S.A., Jiyao, Wang., and McLaughlin, S., Dual Role for Phosphoinositides in Regulation of Yeast and Mammalian Phospholipase D Enzymes, *J. Cell Biol.,* 2002, vol. 159, no. 6, p. 1039.
93. Amsterdam, A., Dantes, A., and Liscovitch, M., Role of Phospholipase D and Phosphatidic Acid in Mediating Gonadotropin-Releasing Hormone-Induced Inhibition of Preantral Granulose Cell Differentiation, *Endocrinology*, 1994, vol. 135, pp. 1205–1211.
94. Moritz, A., De Graan, P.N.E., Gipsen, W.H., and Wirtz, K.W.A., Phosphatidic Acid Is a Specific Activator of Phosphatidilinositol-4-Phosphate Kinase, *J. Biol. Chem.*, 1992, vol. 267, pp. 7207–7210.
95. Ha, K.-S. and Exton, J.H., Activation of Actin Polymerization by Phosphatidic Acid Derived from Phosphatidylcholine in IIC9 Fibroblasts, *J. Cell Biol.*, 1993, vol. 123, pp. 1789–1796.
96. Zhang, Q., Griffith, J.M., and Grant, B.R., Role of Phosphatidic Acid During Differentiation of *Phytophthora palmivora* Zoospores, *J. Gen. Microbiol.*, 1992, vol. 138, pp. 451–459.
97. Jenkins, G.H., Fisette, P.L., and Anderson, R.A., Type I Phosphatidilinositol-4-Phosphate 5-Kinase Isoforms Are Specifically Stimulated by Phosphatidic Acid, *J. Biol. Chem.*, 1994, vol. 269, pp. 11547–11554.
98. Morlock, K.R., McLaughlin, J.J., Lin, Y.-P., and Carman, G.M., Phosphatidate Phosphatase from *Saccharomyces cerevisiae*: Isolation of 45- and 104-kDa Forms of the Enzyme That Are Differentially Regulated by Inositol, *J. Biol. Chem.*, 1991, vol. 266, pp. 3586–3593.
99. Wu, W.-I., Lin, Y.-P., Wang, E., Merrill, A.H., and Carman, G.M., Regulation of Phosphatidate Phosphatase Activity from the Yeast *Saccharomyces cerevisiae* by Sphingoid Bases, *J. Biol. Chem.,* 1993, vol. 268, pp. 13830–13837.
100. Hube, B., Hess, D., Baker, C.A., Schller, M., Schafer, W., and Dolan, J.W., The Role and Relevance of Phospholipase D1 during Growth and Dimorphism of *Candida albicans, Microbiology (UK)*, 2001, vol. 47, no. 4, pp. 879–889.
101. Luo, B., Prescott, S.M., and Topham, M.K., Association of Diacylglycerol Kinase Zeta with Protein Kinase Cα: Spatial Regulation of Diacylglycerol Signaling, *J. Cell Biol.*, 2003, vol. 160, no. 6, p. 929.
102. Gaits, F. and Fourcade, O., Le Balle, F., Gueguen, G., Gaige, B., Gassama-Diagne, A., Fauvel, J., Salles, J.P., Mauco, G., Simon, M.F., and Chap, H., Lysophosphatidic Acid as a Phospholipid Mediator: Pathways of Synthesis, *FEBS Lett.*, 1997, vol. 410, no. 1, pp. 54–58.
103. Kearns, B.G., McGee, T.P., Meyinger, P., Gedvilaite, A., Phillips, S.E., Kagiwada, S., and Bankatis, V.A., Essential Role for Diacylglycerol in Protein Transport from the Yeast Golgi Complex, *Nature*, 1997, vol. 387, no. 6628, pp. 101–105.
104. Macko, V., Inhibitors and Stimulants of Spore Germination and Infection Structure Formation in Fungi, in *The Fungal Spore. Morphogenetic Controls*, Turian, G. and Holh, H.R., Eds., New York: Academic, 1981, pp. 565–584.
105. Podila, G.K., Rogers, L.M., and Kolattukudy, P.E., Chemical Signals from Avocado Surface Wax Trigger Germination and Appressorium Formation in *Colletotrichum gloeosporioides, Plant Physiol.*, 1993, vol. 103, pp. 267–272.
106. Kolattukudy, P.E., Rogers, L.M., Li, D., Hwang, C.S., and Flaishman, M.A., Surface Signaling in Pathogenesis, *Proc. Natl. Acad. Sci. USA,* 1995, vol. 92, pp. 4080–4087.
107. Nukina, M., Sassa, T., Ikeda, M., Takahasi, K., and Toyota, S., Linolenic Acid Enhances Perithecial Production in *Neurospora crassa, Agric. Biol. Chem.,* 1981, vol. 45, pp. 2371–2373.
108. Goodrich-Tanrikulu, M., Howe, K., Staford, A., and Nelson, M.A., Changes in Fatty Acid Composition of *Neurospora crassa* Accompany Sexual Development and Ascospore Germination, *Microbiology (UK)*, 1998, vol. 144, pp. 1713–1720.
109. Rai, J.N., Tewari, J.P., and Sinha, A.K., Effect of Environmental Conditions on Sclerotia and Cleistothecial Production in *Aspergillus*, *Mycopathol. Mycol. Appl*, 1967, vol. 31, pp. 209–224.
110. Calvo, A.M., Hinze, L.L., Gardner, H.W., and Keller, N.P., Sporogenic Effect of Polyunsaturated Fatty Acids on Development of *Aspergillus* spp., *Appl. Environ. Microbiol.,* 1999, vol. 65, pp. 3668–3673.
111. Wilson, R.A., Calvo, A.M., Chang, P.K., and Keller, N.P., Characterization of the *Aspergillus parasiticus* Δ12-Desaturase Gene: a Role for Lipid Metabolism in the *Aspergillus*-Seed Interaction, *Microbiology (UK)*, 2004, vol. 150, no. 9, pp. 2881–2888.

112. Katayama, M. and Marumo, S., R(2)-Glycerol Monolinoleate, a Minor Sporogenic Substance of *Sclerotinia fructicola, Agric. Biol. Chem.*, 1978, vol. 42, pp. 1431–1433.

113. Champe, S.P., Rao, P., and Chang, A., An Endogenous Inducer of Sexual Development in *Aspergillus nidulans, J. Gen. Microbiol.*, 1987, vol. 133, pp. 1383–1387.

114. Mazur, P., Nakanishi, K., El-Zayat, A.A.E., and Champe, S.P., Structure and Synthesis of Sporogenic Psi Factors from *Aspergillus nidulans, J. Chem. Soc., Chem. Commun.*, 1991, vol. 20, pp. 1486–1487.

115. Calvo, A.M., Gardner, H.W., and Keller, N.P., Genetic Connection between Fatty Acid Metabolism and Sporulation in *Aspergillus nidulans, J. Biol. Chem.*, 2001, vol. 276, pp. 25766–25774.

116. Kerwin, J.L, Fatty Acid and Fungal Development: Structure-Activity Relationships, in *Ecology and Metabolism of Plant Lipids. Amer. Chem. Soc. Symposium no. 325*, Fuller, L. and Nes, W.D., Eds., 1987, ch. 20, pp. 329–342.

117. Stasiuk, M. and Kozubek, A., Biological Activity of Phenolic Lipids, *Cell. Mol. Life Sci.*, 2010, vol. 67, pp. 841–860.

118. Konanykhina, I.A., Shanenko, E.F., Loiko, N.G., Nikolaev, Yu.A., and El'-Registan, G.I., Regulatory Effect of Microbial Alkyloxybenzenes of Different Structure on the Stress Response of Yeast, *Prikl. Biokhim. Mikrobiol.*, 2008, vol. 44, no. 5 [*Appl. Biochem. Microbiol.* (Engl. Transl.), vol. 44, no. 5, pp. 518–522].

119. Feresin, G.E., Tapia, A., Sortino, M., Zacchino, S., de Arias, A.R., Inchausti, A., Yaluff, G., Rodriguez, J., Theoduloz, C., and Schmeda-Hirschmann, G., Bioactive Alkyl Phenols and Embelin from *Oxalis erythtrorhiza, J. Ethnopharmacol.*, 2003, vol. 88, pp. 241–247.

120. Murata, M., Irie, J., and Homma, S., Inhibition of Lipid Synthesis of Bacteria, Yeast and Animal Cells by Anacardic Acids, Glycerol-3-Phosphate Dehydrogenase Inhibitors from *Ginkgo*, *Lebensm. Wiss. Technol.*, 1997, vol. 30, pp. 458–463.