Glycerophosphocholine-dependent Growth Requires Gde1p (YPL110c) and Git1p in Saccharomyces cerevisiae*

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Edward Fisher1, Claudia Almaguer5, Roman Holic1, Peter Gricic5, and Jana Patton-Vogt†‡*

From the 4Department of Biological Sciences, Duquesne University, Pittsburgh, Pennsylvania 15282 and the 5Institute of Animal Biochemistry and Genetics, Slovak Academy of Sciences, 900 28 Ivanka pri Dunaji, Slovak Republic

The yeast Saccharomyces cerevisiae synthesizes and degrades the major glycerophospholipids via pathways that are very similar to those employed by higher eukaryotes (1, 2). One pathway of phospholipid degradation in yeast as well as higher eukaryotes is decylation, which results in the formation of water-soluble glycerophosphodiesterases. S. cerevisiae cells growing in medium containing nonlimiting amounts of P1 and inositol deacylate phosphatidylinositol via the action of phospholipases of the B type, Plb3p and Plb1p, to produce glycerophosphoinositol (GroPIns)2 (3–5). Much of the GroPIns produced is excreted into the medium, and external GroPIns can be transported into the yeast cell in times of nutritional stress (phosphate or inositol limitation) via the per- mease encoded by GIT1 (see Fig. 1) (6, 7). Git1p, a member of the major facilitator superfamily of transport proteins (8), was originally isolated based upon its ability to confer growth to an inositol auxotroph supplied with GroPIns as its inositol source (6). Subsequent studies have shown that although inositol limitation up-regulates Git1p transport activity, phosphate limitation does so to a much greater extent and GroPIns can be used as the sole source of phosphate for the cell (7). The enzyme(s) required for GroPIns catabolism remain uncharacterized.

Phosphatidylcholine (PC) decylation also occurs in S. cerevisiae, resulting in the formation of intracellular and extracellular glycerophosphocholine (GroPCho) (Fig. 1). In general, S. cerevisiae produces more internal than external GroPCho (9). However, external GroPCho production increases as the pH of the medium is raised above 5 (11). Similarly, internal GroPCho production increases upon increased flux through the CDP-choline pathway for PC synthesis as a consequence of temperature elevation or choline supplementation (9). S. cerevisiae Net1p, a phospholipase B and homolog of human neuropathy target esterase, is responsible for the production of intracellular GroPCho via PC deacylation (10). Plb1p (11) is thought to be primarily responsible for the formation of extracellular GroPCho (Fig. 1).

As is true for S. cerevisiae, various mammalian cells respond to an increase in PC synthesis by increasing PC deacylation (12–14), suggesting a conserved role for this degradative pathway in maintaining PC homeostasis. The build-up of GroPCho in the cell has been associated with a number of disease processes, including cancer (15) and Alzheimer disease (16). In addition, GroPCho has been implicated in diverse cellular functions such as maintenance of renal osmolarity (17), inhibition of lysophospholipase activity (18), and inhibition of phosphatidylinositol transfer protein alpha (19). Clearly, cell physiology impacts, and is impacted by, GroPCho levels. In turn, the level of GroPCho in the cell is a function of both its formation via PC deacylation and its degradation via glycerophosphodiesterases. Glycerophosphodiesterase encoding genes and glycerophosphodiesterase activities acting upon GroPCho have been reported for several cell types, including Escherichia coli (20), Haemophilus influenzae (21), carrot cell wall (22), kidney (23), and brain (24). GroPIns-specific glycerophosphodiesterase activities have been observed in various rat tissues (25, 26), and a gene encoding a glycerophosphoinositol glycerophosphodiesterase (GDE1/MIR16) has been cloned from rat (27).

The use of S. cerevisiae as a powerful model for studying phospholipid metabolism is well established (1, 2). An understudied aspect of this metabolism is that of the glycerophosphodiesterases, such as GroPCho, produced through phospholipid deacylation. Thus, these studies were undertaken to further our knowledge of glycerophosphodiester metabolism in this important model organism. We demonstrate that the protein encoded by YPL110c (here named GDE1) affects glycerophosphocholine levels in the cell, most probably by acting as a glycerophosphodiester phosphodiesterase. Furthermore, we report that GroPCho is transported intact into the cell in a manner dependent upon the GroPIns permease, Git1p.

**MATERIALS AND METHODS**

**Strains and Media**—Strains were maintained on YES medium (1% yeast extract, 2% Bactopeptone, 2% glucose). The base medium for experiments was chemically defined synthetic media (4) lacking inositol (I−). Where indicated, I− medium was supplemented with 75 μM inositol to make I+ medium. For experiments involving the overexpression of GDE1 from the GAL1 promoter (see Fig. 4), the strains were grown in...
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I— medium lacking dextrose but containing 2% raffinose and 2% galactose. For experiments involving the addition of exogenous GPls or GPlCho (Sigma; catalog number G4007), the base medium was altered by substituting 1g of KCl for 1g of KH2PO4/liter and adding KH2PO4 to a final concentration of 0.2 m M (low Pi) or 10 m M (high Pi) (7). Strains obtained from Research Genetics (JPV125 and JPV126) were checked by PCR to confirm the expected gene deletion. Strain JPV131 was isolated following tetrad dissection of diploid JPV126. To make the gde1Δ ypl206cΔ (JPV431) double mutant, the KanMX marker of strain JPV125 was exchanged with HIS3 using the marker swap plasmid M4754 (28). Plasmid M4754 was digested with NotI, and the released fragment containing HIS3 flanked by regions of the KanMX gene was used to transform JPV125. His- colonies were checked by PCR to verify integration at the correct location, and the resulting strain was named JPV433. Strain JPV431 was isolated following tetrad dissection of the diploid formed by crossing JPV433 with JPV131 (TABLE ONE).

Construction of pGAL-GST-GDE1 Allele—Plasmid pFA6a-kanMX6-PGAL-GST (29) was used as template to amplify a module for insertion into the genome at the 5’ end of GDE1. The 5’ ends of the forward and reverse primers bore 40 nucleotides homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target genes: forward primer, 5’-TAA TGG CGA CTT CCA GTT GCC AGG TGG AGC AAT GTT ACA GAA TTC GAG CTC GTT GGC AAA GGT TTT TCC GAA CTT CAT ACC GGG AAC CAC ATC CGG GTA-3’, and reverse primer, 5’-CTC TGG AAT CGC ATG ATT GGC AAA GGT TTT TCC GAA CTT CAT ACC GGG AAC CAC ATC CGG GTA-3’. The PCR product was transformed (30) into JPV203, and G418-resistant colonies were selected (31). To verify integration at the correct location, genomic DNA was isolated and used as template in PCR using forward primer, 5’-TGG CGA CTT CCA GTT GCC AGG TGG AGC AAT GTT ACA GAA TTC GAG CTC GTT GGC AAA GGT TTT TCC GAA CTT CAT ACC GGG AAC CAC ATC CGG GTA-3’, and reverse primer, 5’-GAC AAT GTT ACA GAA TTC GAG CTC GTT TAA AC-3’, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequences, followed by 20 nucleotides homologous to the plasmid. The underlined sequences are homologous to the target gene sequenc...
Production of Radiolabeled GroPls and GroPCho—Tritium-labeled GroPls (\(^{3}H\text{GroPls}\)) and GroPCho (\(^{3}H\text{GroPCho}\)) were produced through the decylation of phosphatidyl-myo-\([\text{2-}^{3}H]\)inositol and phosphatidylcholine-methyl-\([\text{3}H]\), respectively (American Radiolabeled Chemicals), as described (37).

RESULTS

Gde1p (YPL110cp) and YPL206c Contain Glycerophosphodiester Phosphodiesterase (GPDE) Motifs—A thorough understanding of the importance of phospholipid decylation to cellular physiology requires knowledge about all aspects of the metabolism, including the fate of the remaining counts being \([\text{14}C]\)choline.

Exogenous \([\text{14}C]\)choline-GroPCho Accumulates in the Cytosolic Fraction of gde1Δ Mutant in a GT11-dependent Manner—Although the \([\text{14}C]\)choline labeling studies clearly indicated a role for GDE1 in affecting GroPCho levels, it could be argued that Gde1p acts by negatively regulating PC deacylation, thereby affecting GroPCho levels. To test that possibility, we undertook experiments to (i) determine whether exogenous \([\text{3}H]\)choline-GroPCho can enter the yeast cell, and, if so, (ii) analyze the \([\text{3}H]\)choline-GroPCho metabolites found in wild type and gde1Δ strains upon exogenous \([\text{3}H]\)choline-GroPCho labeling. Using this approach, we would eliminate the intermediate labeling of PC. Because GroPls transport is up-regulated in low phosphate conditions (7) and microarray studies (45) have shown GDE1(YPL110c) expression

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**TABLE ONE**

| Yeast strains     | Relevant genotype | Genotype                             | Source                |
|-------------------|-------------------|--------------------------------------|-----------------------|
| JPV125            | gde1Δ             | his3Δ1, leu2Δ0, met15Δ0, ura3Δ0, YPL110c::KanMX, MATa | Research Genetics     |
| JPV126            | YPL206cΔ/YPL206c  | his3Δ1/his3Δ, leu2Δ0/leu2Δ0, met15Δ0/MET15, lys2Δ0/LYS2, ura3Δ0/ura3Δ0, YPL206c::KanMX/YPL206c, MATa/a | Research Genetics     |
| JPV131            | YPL296cΔ          | his3Δ1, leu2Δ0, ura3Δ0, YPL296c::KanMX, MATa | This study            |
| JPV203            | wild type         | his3Δ1, leu2Δ0, met15Δ0, ura3Δ0, MATa | Research Genetics     |
| JPV431            | gde1Δ YPL206cΔ    | his3Δ1, leu2Δ0, met15Δ0, ura3Δ0, YPL110c::HIS3, YPL206c::KanMX, MATa | This study            |
| JPV433 gde1Δ      | his3Δ1, leu2Δ0, met15Δ0, ura3Δ0, YPL110c::HIS3, MATa | This study            |
| JPV436 pGAL-GST-YPL110c | his3Δ1, leu2Δ0, met15Δ0, ura3Δ0, YPL110c::KanMX::pGAL-GST-YPL110c, MATa | This study            |
to increase in low phosphate conditions, we reasoned that growing the cells in low Pi medium might maximize GroPCho uptake and utilization. When a wild type strain was grown in low Pi medium supplemented with \(10^{-9}/H_9262\)M \([3H]\)choline-GroPCho, a small amount GroPCho was found intact in the trichloroacetic acid-soluble fraction of the cell (Fig. 5A). In addition, label was found in the membrane fraction, suggesting that choline was liberated from GroPCho via a glycerophosphodiesterase and subsequently incorporated into PC. In support of that reasoning, we found \([3H]\)choline in the medium fraction of the wild type strain (Fig. 5B). In contrast, growth of the \(gde1^\Delta\) mutant in the presence of \([3H]\)choline-GroPCho resulted in a much different labeling pattern. As compared with wild type, the \(gde1^\Delta\) mutant exhibited an...
increased accumulation of \([3H]\)choline-GroPCho in the trichloroacetic acid extract (Fig. 5C), less counts associated with the membrane fraction (Fig. 5A), and little or no \([3H]\)choline in the medium or trichloroacetic acid extract (Fig. 5, B and C). Importantly, little or no \([3H]\)choline-GroPCho incorporation (Fig. 5A) occurred in a strain bearing a deletion in the gene encoding the glycerophosphoinositol (GroPIns) transport protein, \(GIT1\) (Fig. 1), suggesting that GroPCho, in addition to GroPlns, is a substrate for Git1p.

**GDE1 and GIT1 Are Required for Utilization of GroPCho as Phosphate Source**— \(S. cerevisiae\) was shown previously to be able to utilize GroPlns as its sole source of phosphate (7). We found that wild type \(S. cerevisiae\) could also use GroPCho as sole phosphate source but that deletion of \(GDE1\) abrogated that ability (Fig. 6). As expected from the labeling results with \([3H]\)choline-GroPCho (Fig. 5), the \(GIT1\) gene was also required for growth on GroPCho (Fig. 6). In contrast, the sole choline transporter of \(S. cerevisiae\), encoded by the \(HNMI\) gene (46), was not required.

**Deletion of GDE1 and/or YPL206c Does Not Alter Catabolism of Exogenous GroPlns**—The role of the two potential glycerophosphodiesterase encoding genes in GroPlns metabolism was also investigated. The majority of the GroPlns produced by \(S. cerevisiae\) is excreted into the medium (3). Furthermore, \(S. cerevisiae\) transports GroPlns into the cell where it is catabolized, presumably by a glycerophosphodiesterase, and its inositol portion is used in the synthesis of phosphatidylinositol (4). Thus, we analyzed the ability of the mutants to incorporate \([3H]\)inositol derived from exogenously supplied \([3H]\)GroPCho into phosphatidylinositol. No differences in labeling patterns were observed between wild type, \(gde1\Delta\), \(YPL206c\Delta\), and \(gde1\Delta\) \(YPL206c\Delta\) strains (Fig. 7A). Finally, strains bearing deletions in \(GDE1\) and/or \(YPL206c\) grew similarly to wild type when GroPlns was supplied as sole phosphate source (Fig. 7B).

**Steady State Phospholipid Composition**—The phospholipid composition of wild type and \(gde1\Delta\) mutant strains was compared for cultures grown in \(1^{-}\) and \(1^{+}\) synthetic media. No significant difference between the mutant and wild type strain was observed.

**DISCUSSION**

The studies reported here represent a substantial advancement in our understanding of the GroPCho metabolism in \(S. cerevisiae\). We report the identification of a novel gene product, Gde1p, involved in GroPCho turnover. In addition, we report that GroPCho is taken up by yeast cells, that its transport across the plasma membrane is dependent upon the Git1p permease, and that GroPCho, like GroPlns (7), can be used as the sole source of phosphate for the cell. Also implicit from the described studies is that GroPCho acts as a source of choline for use in the synthesis of PC.

Several lines of evidence indicate that \(GDE1\) encodes a glycerophosphodiesterase responsible for the hydrolysis of GroPCho in the cell. To begin with, upon uniform labeling of cells with \([14C]\)choline, the trichloroacetic acid-soluble portion of a \(gde1\Delta\) mutant contains more GroPCho as compared with wild type (Fig. 3), whereas strains overexpressing \(GDE1\) contain less GroPCho (Fig. 4). Because exogenous \([14C]\)choline must first be incorporated into PC before a deacylation event liberates GroPCho into the trichloroacetic acid extract, it could be argued that Gde1p acts to negatively regulate PC deacylation, instead of acting to hydrolyze GroPCho. Arguing against that interpretation of the data is that a similar build-up of internal GroPCho is seen when a \(gde1\Delta\) mutant is exogenously labeled with \([3H]\)choline-GroPCho (Fig. 5). Furthermore, the product of glycerophosphodiesterase activity, \([3H]\)choline, was detected in the medium of a wild type strain labeled with \([3H]\)choline-GroPCho, whereas little or no \([3H]\)choline was detected in the medium or trichloroacetic acid extract of a \(gde1\Delta\) mutant (Fig. 5). Thus, the enzymatic reaction liberating choline from GroPCho that occurs in a wild type strain is greatly decreased in a \(gde1\Delta\) mutant. The final piece of evidence indicating a role for Gde1p in GroPCho hydrolysis is the finding that a wild type strain, but not a \(gde1\Delta\) mutant, can utilize GroPCho as the sole phosphate source (Fig. 6).

The absence of any label derived from \([3H]\)choline-GroPCho within the cells of a \(gde1\Delta\) mutant indicates two important facts: (i) Git1p is required for GroPCho transport and (ii) label enters the cells as an intact GroPCho molecule. If GroPCho were degraded extracellularly, \([3H]\)choline would require its transporter, Hnm1p, to enter the cells, not Git1p. Indeed, the increased intracellular GroPCho observed in a \(gde1\Delta\) mutant strongly argues for the role of Gde1p as an intracellular GroPCho glycerophosphodiesterase.

**FIGURE 5.** \([3H]\)Choline-GroPCho supplied in the growth medium accumulates in the cytosolic fraction of \(gde1\Delta\) mutant. A, strains were grown to \(A_{600} = 1-1.5\) (late logarithmic phase) in low \(P_{1}\), \(1^{+}\) medium. The distribution of counts between the trichloroacetic acid (TCA)-soluble and membrane fractions of the cell was determined for each strain. The values represent the means ± S.E. of three independent experiments. B, the identity of the counts in the trichloroacetic acid-soluble and medium fractions was determined by ion exchange column chromatography. *, not determined. wt, wild type.
The ability of yeast to transport GroPCho through Git1p is an exciting, if unexpected, finding. Our previous work has shown Git1p to be a GroPIns transporter with little affinity for GroPCho (6). Indeed, short term transport assays indicate that the rate of GroPCho transport is roughly ten times less than that of GroPIns and that cells grow less robustly when 100 μM GroPCho is supplied in the medium as compared with 100 μM GroPIns (data not shown). Our previous work has demonstrated that GIT1 expression is up-regulated by phosphate limitation and that Git1p is required for the utilization of GroPIns as the phosphate source for the cell. We now report that Git1p is required for GroPCho uptake and for utilization of GroPCho as a phosphate source. These findings broaden the substrate range for Git1p and strengthen the suggestion that one role for Git1p is to provide a mechanism for surviving low phosphate stress by scavenging glycerophosphodiesters. Because S. cerevisiae secretes phospholipases B (5), it is easy to envision a situation in which phospholipases are dispatched to hydrolyze phospholipids, releasing glycerophosphodiester, which can then be taken up into the cell.

In addition to being used as a phosphate source, our studies also indicate that GroPCho acts as a precursor for PC synthesis. Upon labeling with [14C]choline, the build-up of GroPCho seen in a GDE1 mutant occurs at the expense of incorporation of label into PC (Fig. 3), suggesting that the pool of choline found in GroPCho is normally recycled back into PC biosynthesis. Similarly, overexpression of GDE1 (Fig. 4) results not only in less GroPCho accumulation but greater incorporation of choline label into PC, suggesting accelerated recycling of the GroPCho-derived choline pool into PC. Finally, upon labeling with [3H]choline-GroPCho, choline label is clearly found associated with the membrane fraction of the cell.

Strong homologs of Git1p are found in many fungal and plant species, including Candida albicans, Neurospora crassa, and Arabidopsis thaliana (www.ncbi.nlm.nih.gov/BLAST/). Although clear sequence
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homologs have not been noted in mammalian cells, a definitive determination of the existence of Gitlp-like proteins in mammals awaits a better understanding of the elements responsible for conferring glycerophosphodiester substrate specificity to the transport protein. Interestingly, evidence for GroPIns-4-P transport activity in Swiss 3T3 cells has been reported (47).

Our labeling data suggest that GroPCho is hydrolyzed by Gde1p to produce choline and glycerophosphate (Fig. 1). The subsequent metabolism employed by S. cerevisiae to grow on glycerophosphate in the absence of inorganic phosphate (Fig. 6) is not known but will be the subject of future research. The finding that the gde1Δ mutant incorporates some label from [3H]choline-GroPCho into the membrane fraction (Fig. 5) suggests that another gene product may exist that is capable of releasing free [3H]choline for subsequent synthesis of PC. However, that hypothetical gene product is not robust enough to allow for detection of [3H]choline in the medium or trichloroacetic acid extract or to support growth of a gde1Δ mutant on GroPCho. The in vivo hydrolyzing ability of Gde1p appears to be specific for GroPCho, because we could not detect any alterations in GroPIns metabolism in a gde1Δ mutant (Fig. 7A), and gde1Δ could utilize GroPIns as sole phosphate source as well as wild type (Fig. 7B). Deletion of the other S. cerevisiae open reading frame containing a GPDE motif, YPL206c, has no effect upon GroPIns or GroPCho metabolism based upon the labeling and growth experiments described here. The nature of the metabolite(s) hydrolyzed by YPL206cp is under study.

We can envision four potential reasons for our inability to detect in vitro glycerophosphodiesterase activity: (i) The GDE1-GST fusion protein may not be active. Arguing against this interpretation of the data is that a strain (JPV436) bearing the fusion construct clearly contains less label from [3H]choline-GroPCho into the membrane fraction upon cell disruption by bead disruption (data not shown). Future studies on Gde1p will include [3H]choline upon the transporter required for GroPCho uptake, as evidenced by the finding that a gitΔ mutant labeled with [3H]choline has wild type GroPCho levels, but not the increased levels seen in a gde1Δ mutant (data not shown). However, the predicted domain structure of Gde1p suggests potential protein-protein interactions. Ankyrin repeats (38) are located in the middle of Gde1p, roughly localized between residues 300 and 600. Ankyrin repeats have been found in organisms ranging from viruses to humans, and their role in mediating protein-protein interactions is well documented (42). The SPX (38) domain located at the N terminus of Gde1p also has the potential to mediate a protein-protein interaction. The SPX domain of yeast Syg1p binds to the G-protein β-subunit and inhibits transduction of the mating pheromone signal (43). Interestingly, a number of proteins involved in phosphate sensing and metabolism also contain N-terminal SPX domains (38). Our finding that Gde1p is required for the utilization of GroPCho as phosphate source, together with microarray studies reporting increased GDE1 (YPL110c) expression in low phosphate conditions (45), would seem to be in keeping with the observation that Gde1p has an SPX domain. Gde1p was predicted by a global analysis of green fluorescent protein-tagged proteins (49) to be cytoplasmic. We found the GST-tagged version of the protein in the membrane fraction upon cell disruption by detergents and in both the membrane and soluble fractions upon glass bead disruption (data not shown). Future studies on Gde1p will include analyses of its cellular localization, its potential binding partners, and its role in phosphate metabolism.

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Edward Fisher, Claudia Almaguer, Roman Holic, Peter Griac and Jana Patton-Vogt

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