Carbon Source Regulation of PIS1 Gene Expression in Saccharomyces cerevisiae Involves the MCM1 Gene and the Two-component Regulatory Gene, SLN1*

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The Saccharomyces cerevisiae PIS1 gene encodes phosphatidylinositol synthase. The amount of phosphatidylinositol synthase is not affected by the presence of inositol and choline in the growth medium. This is unusual because the amounts and/or activities of other phospholipid biosynthetic enzymes are affected by these precursors, and the promoter of the PIS1 gene contains a sequence resembling the regulatory element that coordinates the inositol-mediating regulation (UASINO). We found that transcription of the PIS1 gene was insensitive to inositol and choline and did not require the putative UASINO regulatory sequence or the cognate regulatory genes (INO2 and OPI1).

The PIS1 promoter includes sequences (MCEs) that bind the Mcm1 protein. Because the Mcm1 protein interacts with both the Sln1 and the Gal11 regulatory proteins, we examined the effect of mutant alleles of the MCM1 and SLN1 genes and carbon source on expression of the PIS1 gene. We found that expression of the PIS1 gene was reduced when cells were grown in a medium containing glycerol and increased when grown in a medium containing galactose relative to cells grown in a glucose medium. The glycerol-mediated repression of PIS1 gene expression required both the MCM1 gene and the MCEs, whereas the SLN1 gene was required for full galactose-mediated induction of a PIS1-lacZ reporter gene.

Thus, PIS1 gene expression is unique among the phospholipid biosynthetic structural genes because it is uncoupled from the inositol response and regulated in response to the carbon source. This is the first example in yeast of a complete circuit linking a stimulus (carbon source) to gene regulation (PIS1) using a two-component regulator (SLN1).

How cells respond to environmental changes has been the subject of intense investigation. In procaryotes, the two-component regulatory system is a well defined and recurring mechanism. The two-component regulatory systems include a sensor and a response regulator that modulate gene expression in reaction to changes in the environment (1). Even though two-component systems have been studied in procaryotes for many years, they have only recently been identified in Saccharomyces cerevisiae (2–5). An example of this is the yeast SLN1 gene product that includes both a sensor and a response regulator (3). The Sln1p is known to modulate the activity of the Mcm1 transcriptional regulatory protein (6, 7). Two questions that need to be addressed are which environmental cues are sensed by this two-component regulatory system, and which genes are targeted for a response. Here, we report that regulation of PIS1 gene expression by carbon sources involves the SLN1 and MCM1 genes.

The major phospholipids in yeast membranes, phosphatidyl-inositol (PI) and phosphatidylcholine, are synthesized by two branches of the phospholipid biosynthetic pathway that diverge from a common lipid precursor, CDP-diacylglycerol (8–10). PI can be synthesized from glucose 6-phosphate, which is converted to inositol by the soluble enzyme, inositol-1-phosphate synthase (IPS) (Fig. 1A). Inositol and CDP-diacylglycerol are subsequently converted to PI by the membrane-associated enzyme PI synthase (PIS) (11–13). The yeast PIS1 gene (Fig. 1A) (11, 14, 15) was cloned by its ability to complement a pis mutant that is conditionally auxotrophic for inositol (11). This pis mutant strain requires high concentrations of inositol (greater than 100 μM) for growth because of a lowered affinity of the PIS enzyme for myo-inositol (16). Disruption of the genomic PIS1 locus is lethal, establishing that it encodes an essential function (14).

In large part, our knowledge of the regulation of phospholipid biosynthetic gene expression has evolved from studies carried out on the INO1 gene that encodes IPS (Fig. 1A). For example, IPS activity, IPS subunit amount, and INO1 transcript levels are all reduced when yeast cells are grown in the presence of inositol and choline (17–19). Conversely, in the absence of inositol and choline, the INO1 gene is maximally expressed (19). This pattern of regulation has also been documented for other phospholipid biosynthetic genes (CHO1, CHO2, OPI3, and CKI) that encode enzymes of the phosphatidylcholine branch of the pathway (20–23). The response to inositol is coordinated by a common cis-acting promoter sequence (5′CATGTGAAAT3′), designated the UASINO element (also called the ICRE) (24–28), that serves as a binding site for the Ino2p-Ino4p heterodimeric activator complex (29–31). In addition to the activator complex, regulation of phospholipid biosynthesis requires a negative acting gene, OPI1 (32). Strains that carry opl mutant alleles constitutively overexpress the INO1 and CHO1 genes (19, 20).

The PIS enzyme is unique among the phospholipid biosynthetic enzymes because its activity is not regulated in response to inositol and choline (13). In addition, immunoblot studies

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1 The abbreviations used are: PI, phosphatidylinositol; PIS, PI synthase; IPS, inositol-1-phosphate synthase; bp, base pair(s).
show that the amount of the 34,000-Da subunit of PIS is unaffected by inositol (13). This lack of effect on PIS activity and subunit amount appears to be due to a lack of regulation at the transcriptional level (23). These observations are surprising because the PIS1 promoter does contain a potential UAS INO element that matches the consensus UAS INO element in 8 of 10 base pairs (see Fig. 1B).

In this study, we found that expression of the PIS1-lacZ fusion gene was unresponsive to inositol in a wild type strain and insensitive to ino2 and opi1 mutations. Instead, expression of the PIS1 and PIS1-lacZ genes was regulated in response to different carbon sources. This regulation required the MCM1 and SLN1 genes and two Mcm1p-binding sites (MCE) present in the PIS1 promoter.

**MATERIALS AND METHODS**

**Strains, Media, and Growth Conditions**—The yeast strains used in this study are listed in Table I. Yeast cultures were grown at 30 °C in synthetic media (33) containing 2% glucose (v/v) and either containing (I°C) or lacking (I°C) 75 μM inositol and 1 mM choline. Where appropriate, galactose or glycerol (2% v/v) was substituted for glucose.

**β-Galactosidase Assays**—β-Galactosidase assays were performed as described previously (24). Units of β-galactosidase activity were defined as (A_{420}/min/mg of total protein) × 1000. Protein concentration in each extract was determined using a Bio-Rad protein assay kit.

**Plasmid Constructions**—Promoter fusions to the lacZ reporter gene were created by inserting restriction fragments from the PIS1 promoter into YEp357R (34). Each construct used an EcoRI site to fuse the four amino-terminal amino acids from PIS1 (Fig. 1B) in-frame with the lacZ gene. Plasmid pMA109 was constructed using a 960-bp EcoRI-EcoRI restriction fragment from pPI514 (14). Similarly, pMA107 contained a 629-bp HindIII-EcoRI restriction fragment and pMA108 contained a 134-bp XhoI-EcoRI fragment (Fig. 1B).

Plasmid pMA101 was generated for the purpose of synthesizing a PIS1 cRNA probe to be used in Northern and slot blot hybridizations. This plasmid was constructed by subcloning a 996-bp EcoRI fragment from pPI514, containing the entire PIS1 coding sequence (14), into pGEM1.

**RNA Analysis**—RNA was isolated from yeast using a glass bead disruption and hot phenol extraction (35). RNA probes (cRNA) for Northern and slot blot hybridizations were synthesized using the Gemini II core system (Promega) from plasmids linearized with a restriction enzyme and transcribed with an RNA polymerase as follows (plasmid/restriction enzyme/RNA polymerase): pMA109/SalI/T7 (PIS1) and...
**RESULTS**

**The UAS$_{INO}$ Element in the PIS1 Promoter Is Inactive**—The presence of a UAS$_{INO}$-like element in the PIS1 promoter (Fig. 1B) suggested that PIS1 transcription might be regulated by the cognate regulatory genes, INO2 and OPI1 (8–10). To examine this possibility, PIS1-lacZ expression was examined using a fusion of 926 bp of the PIS1 promoter to lacZ (pMA109). The wild type (BRS1001) and opl1 (BRS1021) mutant strains were grown in I$_1$C$_1$ (lacking inositol and choline) and I$_2$C$_2$ (lacking both inositol and choline) media (containing 75 μM inositol, respectively). The amount of the PIS1-lacZ transcript was assayed by quantitative slot blot hybridization and normalized to the ACT1 transcript (pMA109). The wild type strain (BRS1001) and strains containing ino2 (8–10) did not express any particular levels of the PIS1-lacZ transcript regardless of carbon source. However, in the mutant strain (BRS1021), the PIS1-lacZ transcript was induced 42-fold when cells were grown in the presence of inositol and choline (Fig. 2B). These experiments suggest that the putative UAS$_{INO}$-like element present in PIS1 promoter does not function in the same capacity as the UAS$_{INO}$ elements present in the promoters of the other phospholipid biosynthetic genes (8–10).

**Expression of the PIS1 Gene Is Regulated by Carbon Source**—We examined if PIS1 gene expression was affected by growth in media containing different carbon sources. This line of inquiry was suggested by the observation that the PIS1 promoter binds Mcm1p (38) and because Mcm1p is a target of the Gal11 regulatory protein that is involved in carbon source regulation of gene expression (39). Expression of the PIS1 gene was quantitated by slot blot hybridization of total cellular RNA (normalized using an ACT1-specific probe) from a wild type strain (BRS1001). RNA was purified from this strain grown in complete synthetic media containing 75 μM inositol and either 2% glucose, 2% galactose, or 2% glycerol. The amount of the PIS1 transcript increased 51% when cells were grown in galactose and reduced 53% when grown in glycerol relative to growth in a glucose medium (Fig. 3).

**MCM1 Regulates PIS1 Gene Expression**—Since the MCEs in the PIS1 promoter were known to bind Mcm1p (38), this raised the possibility that MCM1 may regulate PIS1 expression in response to the carbon source. To address this, we tested the effect of a temperature-sensitive mcm1 mutant allele on PIS1 gene expression. A strain harboring an mcm1$^{150}$ mutant allele (C2-110L) (6) and an isogenic wild type strain (C2-2μM) were grown at 30°C in media containing either 2% glucose, 2% galactose, or 2% glycerol. Upon reaching midlog phase, half of each culture was maintained at 30°C (permissive), while the other half was shifted to 37°C (restrictive). The cultures were allowed to grow for an additional 4 h prior to isolating RNA. Expression of the PIS1 transcript was assayed by quantitative slot blot hybridization and normalized to the ACT1 transcript. In the C2-2μM wild type strain, PIS1 transcript levels were similar to those described for the BRS1001 wild type strain (compare Figs. 3 and 4A). That is, growth in galactose yielded a slight increase in expression, whereas growth in glycerol yielded a significant decrease in expression. This pattern of expression was conserved in the C2-2μM strain.

**PIS1 Regulation**

![Diagram](https://example.com/diagram.png)

**Fig. 2. The PIS1 gene is not a member of the inositol-responsive regulon.** A, the INO2 and the OPI1 regulatory genes are not required for regulation of PIS1 gene expression. PIS1-lacZ gene expression was assayed in a wild type strain (BRS1001) and strains containing ino2Δ (BRS2001) and opl1 (BRS1021) mutant alleles. B, deletion analysis of the PIS1 promoter and schematic representation of PIS1 promoter fusions to the lacZ reporter gene. The positions of the potential UAS$_{INO}$ element (open box), MCEs (hash-marked box), and TATA box (black box). Plasmids pMA109, pMA107, and pMA108 contained 960, 629, and 134 bp, respectively, of the PIS1 promoter fused to lacZ. Units of β-galactosidase were equal to 1000 × optical density at 420 nm/min/mg of total protein. Each value represents the mean of three independent trials, and standard deviations were less than 15% in all cases. Abbreviations: I$^–$ or I$^+$, absence or presence of 75 μM inositol, respectively; C$^–$ or C$^+$, absence or presence of 1 mM choline, respectively.
(mcm1ts) grown at 30 °C (Fig. 4B). However, the shift to the restrictive temperature had a significant impact on PIS1 transcription in the mcm1ts strain (C2-110L) when grown in glycerol. Under these conditions, there was a 110% increase in PIS1 transcription relative to cells grown at 30 °C (Fig. 4B). It should be noted that there was substantially greater standard deviation observed with the mcm1ts mutant strain (C2-100L) relative to the wild type strain (C2-2 μM). This increase in standard deviation was due to differences between colonies. That is, the pattern of expression in the three growth conditions (Fig. 4B) was identical for each colony.

SLN1 Regulates PIS1 Gene Expression—SLN1 is a two-component regulator (2–4) that modulates Mcm1p activity (7). To determine if expression of the PIS1 gene is a downstream target in the SLN1 regulatory pathway, we examined the effect of an sln1 mutant allele on native PIS1 transcription. Expression of the PIS1 gene was quantitated from wild type (JF819) and sln1 mutant (JF1359) strains grown in media containing inositol and either 2% glucose, 2% galactose, or 2% glycerol (Fig. 5). Transcription of the PIS1 gene was reduced 34% in an sln1 mutant strain (JF1359) relative to the wild type strain (JF819) when cells were grown in the glucose medium (Fig. 5). There was also a modest increase in the level of PIS1 expression in the sln1 mutant strain when cells were grown in the galactose medium, but there was no effect when grown in the glycerol medium (Fig. 5).

We also examined the effect of the sln1 mutant allele on expression of the PIS1-lacZ fusions (Fig. 6). The full-length promoter fusion (pMA109), in the wild type strain (JF819), was expressed at high amounts in the glucose and galactose media (99 and 235 units, respectively) and reduced to 44 units of activity in the glycerol medium. In the sln1 mutant strain (JF1359), expression from the full-length promoter fusion (pMA109) was decreased 44% when grown in the glucose medium. This result is consistent with the effect of the sln1 mutant allele on expression of the native PIS1 gene (Fig. 5). Analysis of the pMA107 construct, which lacks 331 bp relative to pMA109, yielded an interesting result. There was a 57% decrease in expression in the sln1 mutant strain (JF1350) relative to the wild type strain when cells were grown in the galactose medium (Fig. 6A). This suggests that the SLN1 gene product is a positive regulator of PIS1 gene expression in response to galactose. Deletion of the two MCEs (pMA108) resulted in much higher levels of expression of the PIS1-lacZ fusion in all three carbon sources (Fig. 6C). There was an approximately 5-fold increase in expression when pMA108 transformants were grown in either glucose or galactose media relative to pMA109 transformants. However, the sln1 mutation had even more dramatic effect on lacZ expression from pMA108 (28-fold) when cells were grown in glycerol medium (Fig. 6C). These results suggest that the region that contains the MCEs (HindIII to XhoI) must include a general repressor element as well as a glycerol-specific repressor element.

DISCUSSION

We report an analysis of the regulation of PIS1 gene expression. The initial experiments examined the function of a potential UASINO element in the PIS1 promoter. The data showed that the UASINO element present in the PIS1 promoter was not functional in its native context (Fig. 2). Expression of the PIS1-lacZ reporter gene (Fig. 2) was unresponsive to inositol and choline supplementation and unaffected by mutations in the INO2 and OPI1 regulatory genes (Fig. 2). It was necessary to

**Fig. 3.** Analysis of PIS1 expression in response to carbon source. Transcription of the PIS1 gene was quantitated by slot blot hybridization with a PIS1-specific cRNA probe and normalized for loading variations by hybridization with an ACT1-specific cRNA probe. Wild type cells (BRS1001) were grown in complete synthetic media containing either 2% glucose, 2% galactose, or 2% glycerol. Each value represents the mean of a minimum of three independent trials.

**Fig. 4.** Effect of an mcm1ts mutant allele on PIS1 gene expression. Transcription of the PIS1 gene was quantitated by slot blot hybridization with a PIS1-specific cRNA probe and normalized for loading variations by hybridization with an ACT1-specific cRNA probe. Wild type cells (C2-2μM, A) and mcm1ts mutant cells (C2-110L, B) were grown in complete synthetic media containing either 2% glucose (striped bars), 2% galactose (solid bars), or 2% glycerol (stippled bars) at either 30 or 37 °C. Each value represents the mean of three independent trials.

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**Fig. 5.**
examine if PIS1 expression was affected by the ino2 and opi1 mutants because of recent reports of UAS elements that utilize some of these regulatory genes to bring about constitutive gene expression (40–42). Thus, PIS1 is the only phospholipid biosynthetic gene whose expression is not co-regulated with that of the other genes in the pathway (8–10).

The presence of a nonfunctional UASINO element in the PIS1 promoter is curious. However, the lack of regulation of PIS1 gene expression in response to inositol is logical since the product of the PIS1 gene utilizes inositol as a substrate (13). The absence of UASINO function may be explained by the two base deviations relative to the consensus sequence (5’ CATA- TGAAGT 3’ compared with 5’ CATGTGAAAT 3’). But it is equally conceivable that the PIS1 version of the UASINO element may be functional in some other physiological context. For example, it is noteworthy that removal of the UASINO element increased expression of the PIS1-lacZ reporter gene by a factor of 2 when grown in the presence of inositol and choline (Fig. 2B). Coincidently, expression of the PIS1-lacZ gene was also increased 3.6-fold when an ino2Δ mutant strain was grown in the presence of inositol and choline (Fig. 2A). Thus, the PIS1 UASINO element may function in a different capacity than the consensus UASINO element.

The most novel finding was that PIS1 gene expression was responsive to the carbon source. The PIS1 gene is the first phospholipid biosynthetic gene shown to be subject to regulation in response to different carbon sources. The particular pattern of regulation is also unusual. For most yeast genes subject to carbon source-mediated regulation, glucose is usually the repressing condition, and galactose is the inducing condition (43). However, in the case of the PIS1 gene, glycerol is the repressing condition (Fig. 3). The only other systems that have been described where gene expression is lowered by growing cells in a nonfermentable carbon source (i.e. glycerol, ethanol, or acetate) are the ribosomal protein genes (44) and genes encoding glycolytic enzymes (45–48). However, the mechanism for regulation of glycolytic gene and ribosomal protein gene expressions is likely to be different from the mechanism controlling PIS1 gene expression. It has been shown that glycolytic gene expression and ribosomal protein gene expression are controlled by a UAS element that dictates glucose inducibility. The mechanism regulating PIS1 expression is likely to involve a repressor-mediated mechanism since removal of promoter sequences (such as the MCEs) yielded increased expression in the presence of glycerol rather than reduced expression in the presence of glucose (Fig. 6). Regulation of PIS1 expression by carbon sources may coordinate PI biosynthesis with macromolecular synthesis (e.g. ribosome assembly). This has been previously suggested by the fact that yeast cells unable to synthesize PI die (inositol-less death) unless macromolecular synthesis is halted (49).

The data show that the MCM1 gene and MCEs are required for the glycerol-mediated repression of PIS1 gene expression. Expression of the native PIS1 gene was increased in the mcm1Δ mutant strain grown in glycerol (Fig. 4B). The amount of PIS1 gene expression in glycerol-grown cells was slightly greater than the amount of expression in either glucose- or galactose-grown cells. This pattern of expression was also observed with a PIS1-lacZ fusion lacking the MCEs (pMA107 in Fig. 6C). The ability of the MCM1 gene product to function as a repressor has been described previously. It is known that the MCM1p functions to repress an a-specific gene (STE2) in a a cells in cooperation with the a2 repressor (6). The promoter of the STE2 gene is known to include an MCE that binds Mcm1p (50).

In addition to the MCM1-mediated glycerol repression, there must also be a general repressor present between the HindIII and XhoI sites. This is indicated by the fact that the amount of ß-galactosidase activity in the pMA108 transformants is 5-fold greater under all carbon sources compared with the amount of

**Fig. 5. Native PIS1 gene expression in an sln1 mutant strain.** Transcription of the PIS1 gene was quantitated by slot blot hybridization with a PIS1-specific cRNA probe and normalized for loading variations by hybridization with an ACT1-specific cRNA probe. A wild type strain (JF819) (striped bars) and an sln1 mutant strain (JF1359) (solid bar) were grown in media containing either 2% glucose, 2% galactose, or 2% glycerol. Each value represents the mean of a minimum of three independent trials.

**Fig. 6. Effect of an sln1 mutation on expression of a PIS1-lacZ fusion.** Effect of an sln1 mutation on lacZ expression from pMA109 (A), pMA107 (B), and pMA108 (C). An sln1 mutant strain (JF1359) (solid bars) was transformed with each of the three plasmids (shown in Fig. 2B) and assayed for ß-galactosidase activity. Activity in the sln1 mutant strain was compared with that of a wild type strain (JF819) (striped bars). Cells were grown in media containing inositol and either 2% glucose, 2% galactose, or 2% glycerol. Each value represents the mean of a minimum of three independent trials. Units of ß-galactosidase are equal to 1000 × optical density at 420 nm/min/mg of total protein.
activity in pMA107 transformants (Fig. 6B).

The carbon source regulation was also affected in an sln1 mutant strain (Fig. 6B). The data suggest that Sln1p may be a positive regulator of PIS1 expression in response to galactose. It is curious that there was no effect of the sln1 mutation on galactose-mediated expression from pMA109 (Fig. 6A) or the native PIS1 gene (Fig. 5). This suggests that there may exist regulatory elements upstream of the HindIII site that interact with Sln1p. It is possible that Sln1p galactose-mediated regulation of native PIS1 transcription occurs only under specific growth conditions. The data also showed a slight decrease in expression of the native PIS1 gene when sln1 mutant cells were grown in glucose media (Fig. 5). However, this effect was observed for all of the promoter constructs in response to almost all carbon sources (Fig. 6). Thus the glucose-dependent effect of the sln1 mutation on PIS1 expression may be an indirect consequence of the mutation.

The observation that SLN1 affected expression from the PIS1 promoter in pMA108 is nevertheless significant given that the SLN1 gene encodes a regulatory protein that resembles bacterial two-component regulators (3) and because Sln1p is a regulator of Mcm1p activity (7). Two-component regulators have been identified as a major mechanism by which bacterial cells can transduce an extracellular stimulus into a genetic response (1). However, there were no examples of a yeast two-component system with a recognized external stimulus and a target gene. Thus, the yeast PIS1 gene provides the first such example.

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