PLC1 encodes a calcium-dependent phospholipase C that hydrolyzes phosphatidylinositol 4,5-biphosphateto generate the signaling molecules diacylglyceroland inositol 1,4,5-triphosphate. IP3 is released from the plasma membrane and is the precursor of all other inositol phosphates, an important class of signaling molecules that regulate many cellular processes. Plc1p activity is involved in nutrient sensing, filamentous growth, nuclear export of mRNA, actin organization, protein synthesis, kinetochore function, transcriptional regulation, vacuolar fusion, and PKA-mediated stress response. Little is known about the regulation of PLC1, but consensus binding sites for the transcription factors Hsf1p and Rap1p have been identified upstream of the PLC1 gene. plc1 null strains have a growth defect, the severity of which is dependent on both the genetic background and the nutrient content of the media. In some strain backgrounds, the null mutant is inviable, while in other strains cell growth rate is merely reduced and inviability is seen only at higher temperatures. Other plc1 null phenotypes include extreme sensitivity to hyperosmotic stress; increased resistance to calcium inhibition; defects in sporulation; failure to form pseudohyphae under filamentous growth-inducing conditions; increased aberrant chromosome segregation; and, when shifted to higher temperatures arrests as large, multibudded cells. Phospholipase C is highly conserved from yeast to humans; homologs include S. pombe plc1 and D. discoideum pipA. Several isoforms of phospholipase C exist in mammals, and S. cerevisiae Plc1p is most similar in structure to mammalian PLC-delta.