SWE1encodes a protein kinase involved in regulating the G2/M transition. Swe1p inhibits the kinase activity of the main cell-cycle cyclin-dependent kinase Cdc28p through phosphorylation of a conserved tyrosine residue, Y19. Y19 phosphorylation is reversed by the phosphatase Mih1p, which is homologous to CDC25 in other organisms. Swe1p-mediated inhibition of Cdc28p is important for delaying mitosis until all appropriate conditions are met, and appears to regulate Clb-Cdc28p complexes to different degrees depending on which B-type cyclin is involved. Swe1p is also important for delaying meiosis when the pachytene checkpoint is triggered. In addition to checkpoint functions, a Swe1p-mediated G2 delay is employed during filamentous growth to promote bud elongation and invasive growth. Swe1p may also be required for reentry into the cell cycle after a G1 arrest caused by defects in ribosome biogenesis or protein synthesis. Swe1p expression is cell-cycle regulated, with accumulation beginning in S phase. As the cell cycle progresses, Swe1p undergoes a complex series of sequential phosphorylations by a variety of kinases, including Cdc5p, Cla4p, and Clb-Cdc28p, which result in hyperphosphorylation and subsequent ubiquitin-mediated degradation. Swe1p abundance also increases transiently in response to ethanol stress. swe1 null mutants display altered cell size and cell cycle kinetics, and are hypersensitive to ethanol. Overexpression of Swe1p leads to a G2 arrest and in some strain backgrounds null mutants enter mitosis prematurely. Homozygous diploid swe1 null mutants are able to complete multiple rounds of pre-meiotic DNA replication within a single cell cycle, which results in some cells displaying more than 4 spores in a single ascus. In pre-mitotic cells, Swe1p localizes to the nucleus and also to the daughter-side of the mother-bud neck where it may be marked for degradation. Swe1p homologs have been identified in several organisms, including Schizosaccharomyces pombe, Xenopusand humans, where they are required to govern entry into mitosis and to delay cell cycle progression in response to DNA damage.