DPM1 encodes the gene for dolichyl phosphate mannosesynthasewhich adds a mannose moiety to dolichyl phosphate on the cytosolic side of the endoplasmic reticulum; then the sugar moiety flips into the lumen of the ER, where Dol-P-Man serves as a source of mannose for three types of protein modifications: N-linked glycosylation, O-linked glycosylation, and synthesis of glycosyl phosphatidylinositolanchors. A similar reaction is catalyzed by Alg5p, which adds glucose to Dol-P, but Alg5p participates only in N-linked glycosylation. Deletion of DPM1 is lethal, but conditional mutants accumulate lipid-linked oligosaccharideswith five mannose residueswhich are added to LLO's on the exterior of the ER by several proteins, including Alg1p, Alg2p, and Alg7p. Mutants lacking Dpm1p also fail to initiate O-mannosylation of proteins, and they accumulate unmannosylated phosphatidylinositol which is not incorporated into proteins. Similar phenotypes are also observed in mutants lacking Sec53p or Sec59p which catalyze reactions upstream of Dol-P-Man synthesis. Human Dpm1p also has Dol-P-Man synthase activity, but lacks lacks a C-terminal hydrophobic domain that allows localization to the ER membrane. Fully active ER-bound human Dol-P-Man synthase is a complex of three proteins: Dpm1p, Dpm2p, and Dpm3p. Mutations in DPM1 are associated with the congenital disorder of glycosylation CDG-Ie.