URA2 encodes a bifunctional protein, comprised of an N-terminal carbamoyl phosphate synthetase domainand a C-terminal aspartate transcarbamoylasedomain, that catalyzes the first two steps of de novo synthesis of pyrimidine ribonucleotides. The Ura2p CPSase domain converts glutamine to carbamoyl-phosphate, then this intermediate is sequestered and channeled to the ATCase active site where it is synthesized into carbamoyl-aspartate. Linking these two domains is a region with similarity to dihydroorotase, the enzyme that catalyzes the third reaction of the pyrimidine biosynthesis pathway. In S. cerevisiae this DHOase-like domain is non-functional, but in the mammalian homolog of Ura2p, CAD, this domain is enzymatically active. Because Ura2p is integral in regulating the de novo synthesis of pyrimidine nucleotides and this process important in cancer cell metabolism, the regulation and function of CAD has been studied in the context of improving chemotheraputic strategies.The final product of this pathway, UTP, represses URA2 transcription and also inhibits Ura2p enzymatic activities. UTP-mediated repression of URA2 transcription has been shown to have less of an effect on pathway regulation than feedback inhibition of Ura2p. During protein regulation, UTP binds to a site in the CPSase domain, directly inhibiting CPSase activity. In contrast, the ATCase domain contains no UTP binding site and repression of ATCase is dependent upon the presence of the CPSase domain, suggesting that the mechanism of regulation is different for the two domains.