



Aspartate Transcarbamylase (ATCase) “EC 2.1.3.2” is an important enzyme for catalyzing the first step of the biosynthesis of pyrimidine nucleotides [1]. It catalyzes the transfer of the carbamoyl group of carbamoyl phosphate as a donor to the amino acid aspartate as an acceptor to form carbamoyl aspartate [2]. There is an evidence that the mammalian enzyme is a member of a multifunctional complex called CAD that is responsible for the catalysis of the initial steps of this synthetic process [1,3]. In fact, the bacterial ATCase from *E. coli* is allosterically inhibited by the nucleotides cytidine triphosphate, uridine triphosphate and deoxy-cytidine triphosphate [1,4]. This allosteric behavior is missed in the mammalian ATCase as reported in the investigation of this enzyme from rat liver [5], whereas it obeys the conventional kinetic behavior of Michaelis and Menten assumption [6]. However, it was reported that the mammalian enzyme is inhibited by uridine monophosphate and 5-bromouridine [7], a phenobarbital derivative [6] and some quinazolinone compounds [8]. Recently, it was found that both the bacterial and mammalian enzymes are *in vitro* and *in vivo* inhibited by the phenobarbital analogues thymidine, phenobarbital and thiobarbituric acid [9]. This inhibition is valuable for the regulation of pyrimidine nucleotide biosynthesis. It will be promising for the control of the growth of cancer cells since some ATCase inhibitors are considered as anti-proliferative compounds [10,11].

References

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