On the conditions for which the Atm protein can switch off the DNA damage signal in a p53 model

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Abstract : In this paper, we consider a recently developed p53 model which simulates interactions between the proteins p53, Mdm2, Atm and DNA damage signal. We apply analytic methods to answer this question : how can the Atm protein contribute positively in DNA healing process? Indeed, we determine regions of parameters into which the Atm protein can switch off damage signals, or conversely, it can lead DNA to a permanent damage.

Keywords : DNA healing; Mdm2 protein; Atm protein

1 Introduction

Today, it is probably well known that one of the key players in cancer development is p53, a tumor suppressor. The p53 protein is a transcription factor encoded by a gene whose disruption is associated with approximately 50 to 55 percent of human cancers. The p53 protein acts as a checkpoint in the cell cycle, either preventing or initiating programmed cell death (apoptosis) [1]. Regarding to the recent experimental observations, biologists consider three major functions for the p53 protein, as, it arrests the cell cycle, thereby giving the cell time to correct any DNA damage, activates transcription of gene indirectly responsible of DNA repair and can be cause of apoptosis [2,3]. We know that p53 regulates itself through its interaction with an intermediate protein which is called Mdm2, [4]. A recent elementary model which is motivated biologically, formulates this interaction as below

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