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Review Article [FullText] [PDF]

TP53 codon 72 Gene Polymorphism Paradox in Associated with Various Carcinoma Incidences, Invasiveness and Chemotherapy Responses

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Running title: *TP53* codon 72 Gene and Chemotherapy Response

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TP53 is the most common mutated gene in human cancers. Approximately half of all human malignancies exhibit *TP53* mutations. The *TP53* codon 72 polymorphism is a single-nucleotide polymorphism (SNP) in exon 4, resulting in the expression of either arginine (CGC) or proline (CCC) residues. In this article, we review literatures published in MEDLINE, and attempt to describe how these two polymorphic variants of TP53 are functionally distinct, and how they influence cancer vulnerability and response to chemotherapy. The Arg72 variant has been shown to be more likely to induce apoptosis than the Pro72 variant, due to its ability to localize itself to mitochondria and trigger the release of cytochrome c into the cytosol. However, but the influence of the *PT53* codon 72 polymorphism on the risk of developing various cancers, and their progression remains inconclusive because there has been no sustained evidence supporting a crucial role for the codon 72 variant in cancer therapy till now. We hypothesize that TP53 might not only be involved in cell cycle control and the apoptosis induction response to DNA damage, but may also modulate individual cancer risk, and that this may correlate with the biofunctions of the two codon 72 variants. Additionally, latent factors might function synergistically with codon 72 variants to confer susceptibility to cancer development, progression, prognosis, and therapeutic responsiveness. Further etiological investigations are essential to reveal the association of and interaction between genetic and environmental factors in relation to carcinogenesis.