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# A Medical Briefing on the p53 Tumour Suppressor Gene

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**ABSTRACT**

The p53 tumour suppressor gene is a nucleic acid sequence with a known function. In biology and medicine its role has been to reduce tumorigenesis and / or proliferation. However, with the application of substances, tissue growth inhibition is a possibility. In cancer of the breast, etc., it has been observed the p53 function can be affected, thus affecting bodily functions, since p53 is responsible for regulating cell cycle events. In this social commentary, clarity on the p53 tumour suppressor gene would be provided.

**KEYWORDS:** Metastasis, Homeostasis, P53, Growth Phases, Cisplatin, Calcium, Fractions, Locomotion

The p53 tumour suppressor gene is a sequence of nucleic acids that play an important role in growth and metastasis in human tissues (Singh, 2019). This sequence of nucleic acids is vital in human life, as rubisco is to C3 and C4 plants (Singh, 2019). In mammals, p53 determines the ultimate lifespan of a cell. This means that in order to make tissues, which eventually go to make organs, a regulated sequence of nucleic acids is necessary in order to prevent unwanted necrosis from happening. This implies that the p53 gene has medical implications if it is not regulated during mitosis, and, thus also meiosis, in cells (Shaw, 1996). However, although tissue stasis is controlled by the p53 suppressor gene, in predicting cells, its role is less prominent. This doesn't mean that p53 isn't active in the latter situation, instead, this means that p53 is active in promoting cell death events in other tissues (Giono and Manfredi, 2006). This has dire medical consequences in that p53 nucleic acid sequences I its natural state doesn't necessarily promote cell cycle events. This is because a dysfunction in p53 tumour suppressor genes could be a result of point mutations. It's possible for this to be done if a person is taking drugs or medication prescribed for treating a medical condition (Singh, 2019). This is true in patients who have life-threatening ailments like cancer, tuberculosis and HIV (Singh, 2019). In these patients this is the case because of the immune-compromised state of the host, but more so because of serum-circulating drug concentrations (Singh, 2019). This means the p53 gene mutations can in fact promote cell death in healthy tissues. This is a major medicinal problem. However, although medical interventions are in place, the role of the p53 tumour suppressor gene will remain unaltered (Agarwal *et al.*, 1995; Chen, no year supplied). In breast cancer, for example, the cells are in a constant state of growth in untreated patients. This is the case because, here, the p53 tumour suppressor gene promotes rapid growth of cancer through the 4 stages (Wang *et al*, no date supplied). In all human cells, the p53 tumour suppressor gene is central in cell cycle events. This is a discovered fact because without p53 it has been found that human systems would be dysfunctional. Tis gene enables human cells to pass through growth phase 1 and 2, separated by respiration events. This means that p53 plays an important role in preventing tumour formation in human cells (Gordon et al., no year supplied; Shaw, 1996). In laryngeal cancer cells it has been found that p53 functioning depends largely on the introduction of substances. In the case of plant compounds / fractions, it has been found that a mixture of cell cycle events occurs. This has been attributed to solvents that induce cell proliferation and cell death at different intensities (Singh, 2019). Furthermore, in the case of signal

transduction, it has been found that p53 tumour suppressor is, in fact, affected by cisplatin, a drug compound that affects the entry of calcium into and out of the cell. However, although the role of the p53 tumour suppressor gene shouldn't be complicated, as it's a known fact, its role is complicated by the ideas that its role in cell cycle events is to bring tissue homeostasis (Agarwal *et al*., 1995). However, its role is complicated because it affects the process of mitotic catastrophe. In mitotic catastrophe, p53 genes affect mitotic fibres involved in cell proliferation. This triggers events that instead of suppressing tumour growth, tumour persists and result in tumorigenesis, or carcinogenesis (Shaw, 1996). A remarkable feature of the p53 tumour suppressor gene, though, is to be able to regulate cell bodily functions, inducing those pertaining to movement and locomotion in mammals (Singh, 2019).

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Rishan Singh is a biologist based in the province of KwaZulu-Natal in Durban, South Africa. He has published widely in the plant sciences. During the early phases of his career, his research focussed on the physiology of plants. He has had several career transitions as a biologist. At the University of KwaZulu-Natal and the Durban University of Technology, he has had some teaching responsibility in addition to laboratory exposure with students. He has enjoyed scholarships from the South African National Research Foundation and a private institution in South Africa. His science contributions have varied over the years, and have encompassed many disciplines. He has also published English literature.