

Charge Transport of Cancer-Related Genes

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We report on theoretical studies [1] of point mutations effects on charge transfer properties in various DNA sequences related to genes from cancer databases, in particular the tumour-suppressor gene p53. On the basis of effective single-strand or double strand tight-binding models which simulate hole propagation along the DNA, we perform statistical analysis of charge transmission modulations associated with all possible point mutations. We find that in contrast to non-cancerous mutations, mutation hotspots tend to result in significantly weaker changes of transmission properties. This suggests that charge transport could play a significant role for DNA-repairing deficiency yielding carcinogenesis. The data for p53 is corroborated by another 34 cancer-related genes, including 20 of the most important tumour suppressors, with known genotype and phenotype analyzed. There are strikingly many properties of charge transport for these cancer-related genes which behave similar to the p53 gene. These new results strongly suggest that the aforementioned scenario of early pathogenesis related to charge transport can be applied to a wide range of disease-related genes.

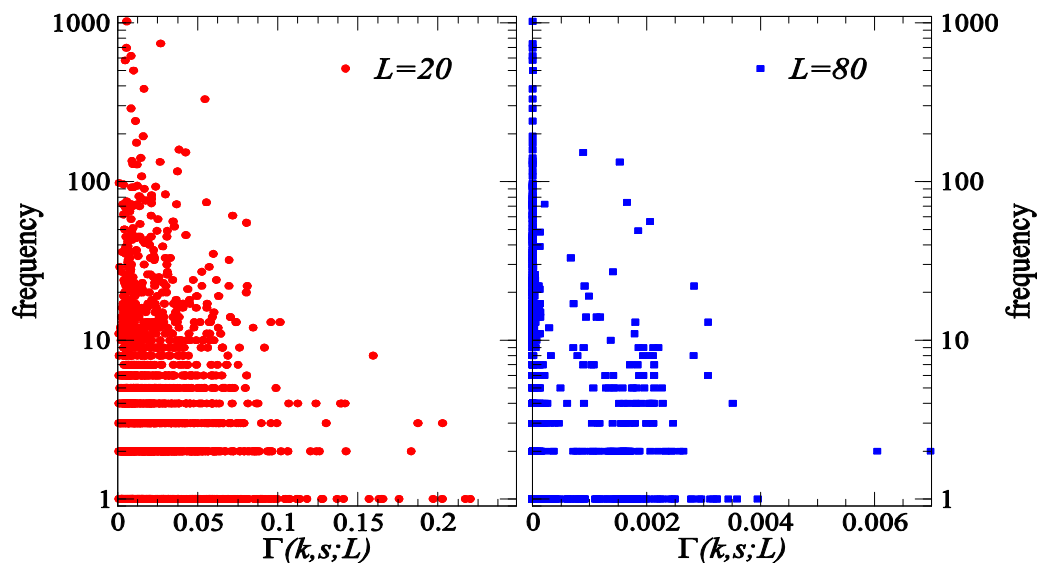


Figure 1 Scatter plots of the frequency of mutation versus the mean charge transport change for the p53 gene.

[1] "Point Mutations Effects on Charge Transport Properties of the Tumor-Suppressor Gene p53", C.-T. Shih, S. Roche, R. A. Römer, Phys. Rev. Lett. 100, [018105-4](#) (2008)