

Mechanisms that could increase cancer vulnerability in COVID-19 mRNA vaccine recipients

Abstract

The BNT162b2 and mRNA-1273 vaccines have been rolled out globally in high numbers. Even minimal risk groups have been fully vaccinated in many countries. We observe certain links that show there are ways cancer can have an easier path to establishing itself as a result of COVID-19 mRNA vaccination.

Dysregulation by spike protein

Proteins p53 and BRCA1 are well-known tumor suppressor proteins that regulate downstream genes in response to numerous cellular stresses and are frequently mutated in human cancer ["p53"][\[Silver, Daniel P., and David M. Livingston\]](#). It is observed to be inhibited by SARS-CoV-2 [\[Stingi, Aureliano, and Lu.\]](#). The S2 (Spike protein S2) subunit of Sars-CoV-2 has known interactions with p53, but also with BRCA-1/2 proteins [\[Singh, Nishant, and Anuradha Bharara Singh.\]](#). That could also mean that the proteins generated by the mRNA vaccines would also interact with the p53 gene, and inhibit this tumor suppressor. The SARS-CoV spike protein is also known to cause apoptosis in Vero E6 cells. Cleavage of the S protein into fragments was suggested in previous studies, which includes a form that resembles the S2 protein in this study, suggesting the spike proteins are enough to degrade p53 [\[Chow, K. Y., et al.\]](#)[\[Barhoumi, Tlili, et al.\]](#). Even though vaccines help prevent SARS-CoV-2 spread and infection individuals with solely vaccination-gained immunity might suffer from opsonization on initial infection, eventually resulting in high viral load and spike overload [\[Bahnan, Wael, et al.\]](#)[\[Acharya, Charlotte B., et al.\]](#). It is important to note that certain results suggest that recombinant spike-based SARS-CoV-2 immunogen glycosylation reproducibly recapitulates signatures of viral glycosylation. [\[Allen, Joel D., et al.\]](#). Which could potentially be cancer-enhancing [\[Kremsreiter, Stefanie Maria, et al.\]](#), even though this does not seem like a very likely scenario, it would probably increase the chances of p53 being inhibited [\[May, and May\]](#).

Code issues in mRNA

Several natural mechanisms of termination suppression exist, including ribosomal frameshifting. The process of protein synthesis termination, although effective, is not 100% efficient [\[Dabrowski, Maciej, et al.\]](#). Several +1 frameshift sites have also been recognized in eukaryotic mRNA. For example, the expression of mammalian antizyme 1 (AZ1) requires a +1 frameshift [\[Moon, et al.\]](#). Codon misreading tRNAs are known to promote tumor growth in mice [\[Santos, Mafalda, et al.\]](#), and it's known that many human cancers can be caused by illogical frameshifts [\[Lee, Hui-Ling Rose, & Joseph Dougherty.\]](#)[\[X., Xia.\]](#). In BNT162b2 and mRNA-1273 vaccines there is a relatively high chance of such occurrences due to poor choices of stop codons and shoddy optimization [\[Mordstein, Christine, et al.\]](#).

Discussion

Tumor suppressors are important in preventing cancer from occurring. Mass vaccination on low risk groups requires more data on this matter before determining the safety of the current mRNA vaccines.

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