

A new paper shows spike toxicity in Zebrafish

A study on spike run-off into our environment



Jessica Rose
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This is something I have been concerned about from day 1. Every time I paddle out I wonder how much potential exposure to certain bacteria and viruses I am getting. It doesn't concern me too much since I have a rule about not paddling out on certain days.¹

|| If it's brown, I'm not down. Jessica Rose



Figure 1: <https://www.instagram.com/ioshbernard/>

Figure 1. https://www.instagram.com/josibonhara_

Us surfers really have to be careful about our surf spots being polluted by human sewage and run-off especially after rains.² Drainpipes following heavy rains are a definitive no-go zone. It is maintained that one should wait ~72 hours following heavy rains to paddle out in areas where effluent is known to be present and problematic. We surfers really are a tough bunch, but we are far more likely to get sick out there than even swimmers.³

This article entitled “[Toxicity of spike fragments SARS-CoV-2 S protein for zebrafish: A tool to study its hazardous for human health?](#)” was published in March, 2022 in the journal *Science of the Total Environment*. The authors use a model for studying the effects of spike on humans ‘using Zebrafish as a tool to assess the harmful effects of SARS-CoV-2 in the aquatic environment’. They found that the spike protein from SARS-CoV-2 is highly toxic to Zebrafish.

The reason every single person on Earth needs to be concerned about this (and should have been years ago) is because of conserved homologies between humans and other species. For example, genetic homology between zebrafish and humans is highly conserved whereby [~70% of human genes](#) can be found in our stripy little friends.⁴

Our respective ACE-2 receptors are 72% sequence similar.



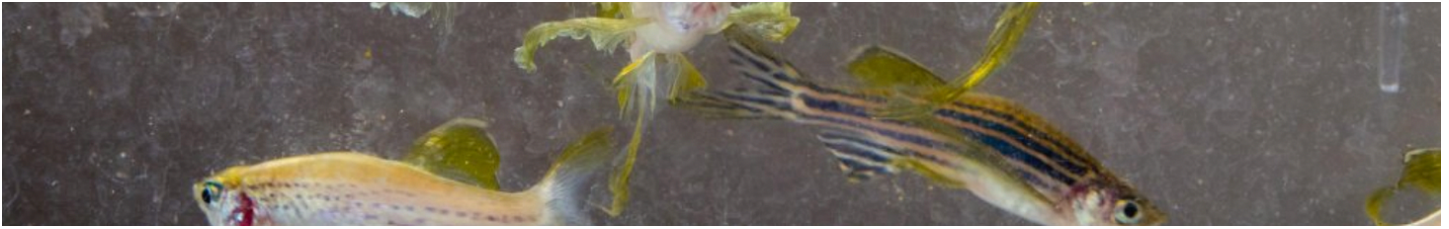


Figure 2: Zebrafish in a tank. <https://irp.nih.gov/blog/post/2016/08/why-use-zebrafish-to-study-human-diseases>.

Please read this article. In the very first line of the abstract, a point of dire concern is addressed. I am particularly concerned about their findings since the claim of the COVID-19 injectable product manufacturers has always been that their modified mRNA spike templates yield the spike from the original Wuhan strain (that would be the one with the furin cleavage, the superantigen and the amyloidogenic sites), and also since the modus operandi of this new technology is to hi-jack the human host cell machinery to translate the modified mRNA coding templates into copious amounts of this spike protein.

Despite the significant increase in the generation of SARS-CoV-2 contaminated domestic and hospital wastewater, little is known about the ecotoxicological effects of the virus or its structural components in freshwater vertebrates.

I don't want to downplay the fact that SARS-CoV-2 is also problematic from this point of view. Because of the furin-cleavage site, the spike protein once bound to ACE-2, is cleaved and this yields 3 free S1 subunits per spike trimer, able to impose whatever subsequent damage they may. For someone with a high viral replication rate and high viral load, this will necessarily imply more free spike.

We demonstrated, for the first time, that zebrafish injected with fragment 16 to 165 (rSpike) [aka 'their spike bit'], which corresponds to the N-terminal portion of the protein, presented mortalities and adverse effects on liver, kidney, ovary and brain tissues.

Oh dear. This confirms two things for me.

1. The spike can inflict severe damage on other beings in our ecosystem.
2. The spike protein on its own, no virus required, can bind ACE-2 to impose its damages via intracellular signaling cascades. In fact, "77% of the human ACE2

damages via intracellular signaling cascades. In fact, 77% of the human ACE2 residues of the interface are similar in zebrafish ACE2 sequence". (Reference #1)

They found that the difference in survival rate between their spike bit-injected fish and control fish, was statistically-significant.

The relative risk of death in the period studied between the groups was significant (chi square = 79.70; $p < 0.0001$).

The authors also found evidence of (temporary?) deleterious effects of their spike bit on the ovaries and testes of the fish.

With respect to the reproductive tissue, female zebrafish injected with rSpike displayed severe damage in the ovary (follicular atresia, cellular infiltration, and disorganized extracellular matrix) after 7 days of protein inoculation.

They found evidence that their spike bit induced inflammatory processes in the brain.

In our study, the rSpike was responsible for generating an inflammatory process in the brain (Fig. 4e), characterized by an intense influx of mononuclear cells, but no histopathological lesions, these inflammatory infiltrate findings were confirmed by immunohistochemical analysis.

They report evidence of thromboses associated with damage to the olfactory epithelium/deep medullary veins leading to severe encephalitis and demyelinating lesions.

The application of spike in zebrafish's olfactory epithelium causes thrombosis of the deep medullary veins (Cavalcanti et al., 2020).⁵ Damage to the structure and function of this system can lead to severe encephalitis, toxic encephalopathy, and, after viral infections, severe acute demyelinating lesions (Wright et al., 2008).⁶

The authors also raise the point that I have been screaming about for almost two years and that is the real possibility of the spike-ACE-2 binding event and the resultant intracellular cascade (cell signaling) of the cell and all the immunological mediator activation that would accompany this to induce a complete biological disaster. I have been looking at this from the point of the view of the Renin Angiotensin Aldosterone System (RAAS), but the

systemic implications for spike-ACE-2 interaction/intracellular signaling cascade/activation events are astronomical. Why wouldn't they be?

It was hypothesized that cell signaling elicited by the spike protein fragments that occur in cells would predispose injected individuals to develop complications that are seen in severe and fatal COVID-19 conditions. If this hypothesis is correct, then the strategies to treat COVID-19 should include, in addition to agents that inhibit the viral replication, therapeutics that inhibit the viral protein fragment-mediated cell signaling.

I am very glad this got published in *Science of the Total Environment* (Impact Factor: 10.753) but this should be in *Nature* (Impact Factor: 42.778), and at the same time...

...this modified spike protein shouldn't be in NATURE.

- 1 <https://surftweeters.com/surf-for-humanity/surfers-against-sewage-goes-no-holds-barred-to-protect-our-waves/>
- 2 <https://thewaldenwordcom.wordpress.com/2019/03/05/rain-drops-runoff-surfing-on-sewage-in-the-drop-spot/>
- 3 Dickinson G, Lim KY, Jiang SC. Quantitative microbial risk assessment of pathogenic vibrios in marine recreational waters of southern california. *Appl Environ Microbiol.* 2013 Jan;79(1):294-302. doi: 10.1128/AEM.02674-12. Epub 2012 Oct 26. PMID: 23104412; PMCID: PMC3536113.
- 4 Howe K, *et al.* The zebrafish reference genome sequence and its relationship to the human genome. *Nature.* 2013 Apr 25;496(7446):498-503. doi: 10.1038/nature12111. Epub 2013 Apr 17. Erratum in: *Nature.* 2014 Jan 9;505(7482):248. PMID: 23594743; PMCID: PMC3703927.
- 5 D.D. Cavalcanti, E. Raz, M. Shapiro, S. Dehkharghani, S. Yaghi, K. Lillemoe, P.K. Nelson. Cerebral venous thrombosis associated with COVID-19. *Am. J. Neuroradiol.*, 41 (8) (2020), pp. 1370-1376, 10.3174/ajnr.A6644.
- 6 E.J. Wright, B.J. Brew, S.L. Wesselingh. Pathogenesis and diagnosis of viral infections of the nervous system. *Neurol. Clin.*, 26 (3) (2008), pp. 617-633, 10.1016/j.ncl.2008.03.006.