# The Serpent's Bite

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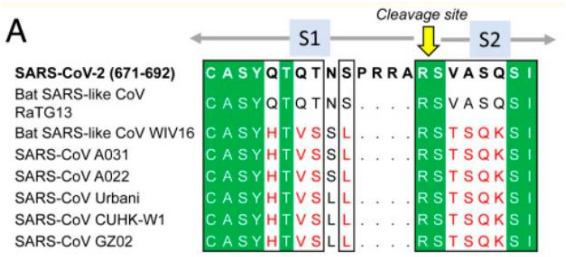
Source : https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7568239/

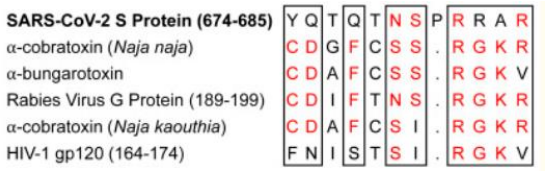
**THE PRRAR INSERT**

Firstly, you can see that the PRRAR is unique to Sars Cov 2 compared to the other corona viruses, and therefore is likely to be the result of Gain of Function research.




**COMPARISON TO OTHER SPECIES**

Secondly, we can compare this inserted sequence to its natural occurrence in other species and in other viruses, to gain an idea of its function.




**ACETYLCHOLINE RECEPTOR BINDING**

These sequences all bind to the Acetylcholine receptor

a) **HIV1 gp120**
This paper shows that HIV-1 gp120 binds to the acetylcholine receptor
https://febs.onlinelibrary.wiley.com/doi/epdf/10.1016/0014-5793%2892%2981380-5

b) **RABIES**
This paper shows that RABIES binds to the acetylcholine receptor
https://pubmed.ncbi.nlm.nih.gov/2361061/

c) **BUNGARO TOXIN**
This paper shows that BUNGARO TOXIN binds the acetylcholine receptor
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1303215/pdf/0943.pdf

d) **COBRA TOXIN**
This paper shows that COBRA TOXIN binds to the acetylcholine receptor
https://proteopedia.org/wiki/index.php/Acetylcholine_Receptor_and_its_Reaction_to_Cobra_Venom

**e) C19 SPIKE**

This paper shows that C19 SPIKE binds to the acetylcholine receptor
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7386492/pdf/nihpp-2020.07.16.206680.pdf

Curiously, the word "Furin" derives from the word "Furia" which is synonymous with the word "Rabia" in latin. All three words mean "Violence". https://es.thefreedictionary.com/Furia.

It would appear that HIV, Rabies, Bungaro, Cobra and the Spike protein are all *"bites of the Serpent"*

> *"Further Examination of the Motif near **PRRA** Reveals Close Structural Similarity to the SEB Superantigen as well as Sequence Similarities to Neurotoxins and a Viral SAg. The insertion PRRA together with seven sequentially preceding residues and succeeding R685 (conserved among β-CoVs) form a motif, Y674QTQTNSPRRAR685, **homologous to those of neurotoxins from Ophiophagus (cobra) and Bungarus genera, as well as the neurotoxin-like regions from three RABV strains (20) (Fig. 2D). We further noticed that the same segment bears close similarity to the HIV-1 glycoprotein gp120** SAg motif F164 to V174."*

HIV attacks T cells. COVID also produces lymphocytopenia.

**PRRA Sequence and Bacterial Toxins – Staphylococci and Streptococci**

> *"This close sequence similarity to both bacterial and viral SAgs, in support of the potential superantigenic character of the stretch Y674 to R685 of SARS-CoV-2 S, directed us to further analyze its local sequence and structure.*
>
> *Our analysis led to an interesting sequence similarity between the fragment T678 to Q690 of SARS-CoV-2 S and the SEB superantigenic peptide T150NKKKATVQELD161. This dodecapeptide sequence shows strong conservation among a broad range of staphylococcal and streptococcal SAgs.*
>
> *What is even more interesting is that SARS-Cov-2 motif showed a palindromic behavior with respect to this superantigenic SEB sequence, in the sense that a broader stretch, from E661 to R685, could be aligned to the SAg peptide in the reverse direction as well."*

A

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SEB SAg:        (150)TN-KKKATVQELD(161)
SARS-CoV-2:     (678)TNSPRRARSVASQ(690)
SARS-CoV:       (664)SLL----RSTSQK(672)

Inv SEB SAg:  (161)DLE----------QVTA-KKKNT(150)
SARS-CoV-2:   (661)ECDIPIGAGICASYQTQ-TNSPRRAR(685)
SARS-CoV:     (647)ECDIPIGAGICASYHTVSLL-----R(667)
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> *"This brings to our attention the versatility and high propensity of the SARS-CoV-2 S TCRVβbinding site residues to potentially elicit an SAg-like response. "*

**The Sequence Similarity is Missing in Sars 1**

> *"We note that the sequentially aligned segment of SARS1 (S664 to K672) bears minimal similarity to the SEB SAg "*

**Secondary Structures are Also Similar**

> *"Significantly, the structure of the SARS-CoV-2 S SAg-like segment and that of SEB peptide also exhibit a remarkable similarity (Fig. 3 B and C): A salt bridge (E159–K152 in SEB and E661–R685 in SARS-CoV-2 S) stabilizes both structural motifs; the relative orientations of three positively charged residues (K152, K153,*

*and K154 in SEB and R682, R683, and R685 in SARS-CoV-2 S) are maintained; and an asparagine (N151 in SEB, N679 in SARS-CoV-2) completes this motif. All three features are absent in SARS1 S (Fig. 3D).*

*A β-hairpin that apparently serves as a scaffold is conserved in all three spikes, and we observe a pair of cysteines that may potentially form a disulfide bond in SARS-Cov-2 and SARS1 spikes (C662−C671 and C648−C657, respectively).*

*This analysis overall indicates that the segment T678NSPRRAR685 may potentially form a putatively superantigenic core, consistently aligned against various bacterial or viral SAgs (Figs. 2C and 3 A–C) with or without the participation of the adjoining amino acids.*

*However, combined broader sequence and structure analysis in Fig. 3 A (Right) and B and C, reveals an even more compelling feature: This putative SAg core is structurally consolidated by spatial proximity to a conserved acidic segment, E661CD663, which forms a highly stable salt bridge with the polybasic segment PRRAR of SARS-CoV-2 S, much in the same way as the salt bridge observed in SEB (but not in SARS1 S), complemented by an asparagine shared between SARS-CoV-2 S and SEB (but not SARS1 S), and the SAg character may be conferred by this type of structural scaffolding."*

**Binding to T Cell Receptors – CD28**

*"We note that the SEB superantigen peptide Y150NKKKATVQELD161* ***has been reported to bind CD28*** *(21), a TCR that provides costimulatory signals required for T cell activation and survival. CD28 and TCRV domains share the same (immunoglobulin, Ig) fold (Fig. 3E), and the binding mechanism shown in Fig. 1B could adapt, with minor rearrangements, to interactions with other Ig-fold molecules including neutralizing antibodies. Because of the homologous superantigenic segment of SEB binding CD28, we also tested the potential binding of SARS-CoV-2 spike residues E661 to R685 onto CD28. Our simulations indicated that the same segment can equally bind to CD28, further supporting the strong propensity of the fragment to stimulate T cell activation."*

Binding to T cells causes dysregulation of the T cells

https://www.nature.com/articles/s41423-020-0424-9.pdf

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7136698/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3172200/pdf/pbio.1001149.pdf

HIV attacks T cells. Covid characterised by Lymphocytopenia.

HIV sequences found in Covid - https://www.biorxiv.org/content/10.1101/2020.01.22.914952v2.full.pdf

**Was the PRRA sequence added by design?**

Did the Chinese publish a paper stating that they had added HIV Gp120 to Corona?

Did Montagnier show that HIV Gp120 was in the Spike sequence?