

**Supplementary Table 1:**

Thirty two studies that use the name “ORF3b”. The first set includes papers that use the name “ORF3b” to refer to the 22 codon ORF (recommended name ORF3b), the second set includes papers for which the referent could not be determined, and the third set includes papers that refer to the 57 codon ORF (recommended name ORF3d) or to both, conflating them. In each case empirical studies are listed first. We report the way in which we determined which entity was referred to and brief notes on the nature of the study or of the conflation between the ORFs. Often the actual ORF being referred to was not made entirely clear in the paper itself and we inferred it from references it cited, so it is possible that in some cases that was not the ORF the study authors intended.

Authors and journal	Study	URL	Citation	Study type	Recommended name for presumed referent of “ORF3b”	Notes
<b>Studies that use “ORF3b” to refer to the 22-codon ORF</b>						
Wu et al. <i>Nature</i>	A new coronavirus associated with human respiratory disease in China	<a href="https://doi.org/10.1038/s41586-020-2008-3">https://doi.org/10.1038/s41586-020-2008-3</a>	[1]	Genome report	ORF3b (from their Table S6)	Their Fig. 1 and Table S6 refer to the whole region homologous to SARS-CoV ORF3b without noting the early stop codons present in the SARS-CoV-2 sequence.
Konno et al. <i>Cell Reports</i>	SARS-CoV-2 ORF3b is a potent interferon antagonist whose activity is increased by a naturally occurring elongation variant	<a href="https://doi.org/10.1016/j.celrep.2020.108185">https://doi.org/10.1016/j.celrep.2020.108185</a>	[2]	Empirical	ORF3b (named as 22 amino acid protein)	Report interferon antagonist activity
Lokugamage et al. <i>Journal of Virology</i>	Type I interferon susceptibility distinguishes SARS-CoV-2 from SARS-CoV	<a href="https://doi.org/10.1128/jvi.01410-20">https://doi.org/10.1128/jvi.01410-20</a>	[3]	Empirical	ORF3b (refers to an alignment with SARS-CoV ORF3b)	No gene-specific experiments for ORF3b. Refer to a 24 amino acid protein (should be 22 unless referring to second ORF in 3b-region)
Xia et al.	Evasion of Type I Interferon by SARS-CoV-2	<a href="https://doi.org/10.1016/j.celrep.2020.108234">https://doi.org/10.1016/j.celrep.2020.108234</a>	[4]	Empirical	ORF3b (from primer sequences in their Table S1)	No inhibition of IFN-β production by ORF3b reported.

Zhang et al. (BioRxiv preprint)	A Systemic and Molecular Study of Subcellular Localization of 2 SARS-CoV-2 Proteins	<a href="https://doi.org/10.1101/2020.08.02.233023">https://doi.org/10.1101/2020.08.02.233023</a>	[5]	Empirical	ORF3b (from primer sequences in their Table 1)	Report cytoplasmic location of ORF3b after expression from construct
Sa Ribero et al. <i>PLOS Pathogens</i>	Interplay between SARS-CoV-2 and the type I interferon response	<a href="https://doi.org/10.1371/journal.ppat.1008737">https://doi.org/10.1371/journal.ppat.1008737</a>	[6]	Review	ORF3b (cites Konno et al.)	Their Fig. 1 shows the ORF at the wrong end of the SARS-CoV ORF3b homologous region

### Studies that use “ORF3b” with unclear referent

Lei et al. <i>Allergy</i>	Antibody dynamics to SARS-CoV-2 in asymptomatic COVID-19 infections	<a href="https://doi.org/10.1111/all.14622">https://doi.org/10.1111/all.14622</a>	[7]	Empirical	Referent unclear - no sequence details	Test for antibody responses
Nabeel-Shah et al. (BioRxiv preprint)	SARS-CoV-2 Nucleocapsid protein attenuates stress granule formation and alters gene expression via direct interaction with host mRNAs	<a href="https://doi.org/10.1101/2020.10.23.342113">https://doi.org/10.1101/2020.10.23.342113</a>	[8]	Empirical	Referent unclear - no sequence details	Report protein-protein interactions (see their TableS1)
Yuen et al. <i>Emerging Microbes and Infections</i>	SARS-CoV-2 nsp13, nsp14, nsp15 and orf6 function as potent interferon antagonists	<a href="https://doi.org/10.1080/22221751.2020.1780953">https://doi.org/10.1080/22221751.2020.1780953</a>	[9]	Empirical	Referent unclear - shown in 3b location (Fig. 1A) but no sequence info.	Referent is ORF3d according to later paper Lam et al. from same authors. Implied functional inference based on SARS-CoV ORF3b. Only weak interferon antagonist activity reported.
Sun (BioRxiv preprint)	The discovery of gene mutations making SARS-CoV-2 well adapted for humans: host-genome similarity analysis of 2594 genomes from China, the USA and Europe	<a href="https://doi.org/10.1101/2020.09.03.280727">https://doi.org/10.1101/2020.09.03.280727</a>	[10]	Computational	Referent unclear - no sequence details	Report sequence features

Studies that use “ORF3b” to refer to the 57-codon ORF or to both, conflating the two						
Chan et al. <i>Emerging Microbes &amp; Infections</i>	Genomic characterization of the 2019 novel human-pathogenic coronavirus isolated from a patient with atypical pneumonia after visiting Wuhan	<a href="https://doi.org/10.1080/22221751.2020.1719902">https://doi.org/10.1080/22221751.2020.1719902</a>	[11]	Genome report	ORF3d (sequence shown in their Fig. 4)	Homology with ORF3b of SARS-CoV is implied by presenting an alignment with SARS-CoV-2 ORF3d in context of discussing the functions of SARS-CoV ORF3b. Many papers subsequently cite and make functional inferences on this basis.
Kim et al. G3	A Flexible Genome-Scale Resource of SARS-CoV-2 Coding Sequence Clones	<a href="https://doi.org/10.1534/g3.120.401554">https://doi.org/10.1534/g3.120.401554</a>	[12]	Laboratory resource - sequence clone collection	ORF3d (58 amino acid protein)	This resource is widely used. Refers to a 58 amino acid protein (i.e. 57 + stop). Claims “Induce inflammatory response and inhibit the expression of IFN $\beta$ ” (presumably based on assumption of homology to SARS-CoV ORF3b)
Banerjee et al. <i>Cell</i>	SARS-CoV-2 Disrupts Splicing, Translation, and Protein Trafficking to Suppress Host Defenses	<a href="https://doi.org/10.1016/j.cell.2020.10.004">https://doi.org/10.1016/j.cell.2020.10.004</a>	[13]	Empirical	ORF3d (construct sourced from Kim et al.)	Report binding to host mRNAs
Gordon et al. <i>Nature</i>	A SARS-CoV-2 protein interaction map reveals targets for drug repurposing	<a href="https://doi.org/10.1038/s41586-020-2286-9">https://doi.org/10.1038/s41586-020-2286-9</a>	[14]	Empirical	ORF3d (location shown in their Supp. Fig. 1c)	Report protein-protein interactions. Make functional inference based on SARS-CoV ORF3b
Hachim et al. <i>Nature Immunology</i>	ORF8 and ORF3b antibodies are accurate serological markers of early and late SARS-CoV-2 infection	<a href="https://doi.org/10.1038/s41590-020-0773-7">https://doi.org/10.1038/s41590-020-0773-7</a>	[15]	Empirical	ORF3d (primers in their Supp. Table 5)	Report antibody response. Make functional inference based on SARS-CoV ORF3b
Hayn et al. (BioRxiv preprint)	Imperfect innate immune antagonism renders SARS-CoV-2 vulnerable towards IFN- $\gamma$ and - $\lambda$	<a href="https://doi.org/10.1101/2020.10.15.340612">https://doi.org/10.1101/2020.10.15.340612</a>	[16]	Empirical	ORF3d (ORF construct from Gordon et al.)	ORF3b and ORF3c both mentioned. Intended ORFs unclear apart from the reference to the ORF3c construct's provenance, as “ORF3b” is shown embedded within “ORF3c” in their Fig1A.
Lam et al. <i>Emerging Microbes &amp;</i>	Loss of orf3b in the circulating SARS-CoV-2 strains	<a href="https://doi.org/10.1080/22221751.2020.1852892">https://doi.org/10.1080/22221751.2020.1852892</a>	[17]	Empirical	ORF3d. Refers to a 57 a.a. protein which is	Report interferon antagonism for full length ORF3d which is lost in a widely circulating truncated variant.

Infections					distinguished from the 22a.a. ORF.	
Laurent et al. (BioRxiv preprint)	Global BioID-based SARS-CoV-2 proteins proximal interactome unveils novel ties between viral polypeptides and host factors involved in multiple COVID19-associated mechanisms	<a href="https://doi.org/10.1101/2020.08.28.272955">https://doi.org/10.1101/2020.08.28.272955</a>	[18]	Empirical	ORF3d (sequence in their Supp. Table 3)	Report that ORF3d “is a Golgi-associated protein interacting with ESCRT-0”. However make functional inference based on SARS-CoV ORF3b
Samavarchi-Tehrani et al. (BioRxiv preprint)	A SARS-CoV-2 - host proximity interactome	<a href="https://doi.org/10.1101/2020.09.03.282103">https://doi.org/10.1101/2020.09.03.282103</a>	[19]	Empirical	ORF3d (sequence in their Supp. Table 1)	Report localization to the Golgi and a relationship to LAMTOR1 (as with St-Germain et al.)
St-Germain et al. (BioRxiv preprint)	A SARS-CoV-2 BioID-based virus-host membrane protein interactome and virus peptide compendium: new proteomics resources for COVID-19 research	<a href="https://doi.org/10.1101/2020.08.28.269175">https://doi.org/10.1101/2020.08.28.269175</a>	[20]	Empirical	ORF3d (57 a.a. protein in their Supp. Table 1)	Host protein proximity interactome study. Report involvement in mTOR signalling and possible role in disrupting antiviral immune function.
Michel et al. <i>Virology Journal</i>	Characterization of accessory genes in coronavirus genomes	<a href="https://doi.org/10.1186/s12985-020-01402-1">https://doi.org/10.1186/s12985-020-01402-1</a>	[21]	Computational	ORF3d (they distinguish it from SARS-CoV ORF3b)	Predict ORF3d is not functional based on lack of enrichment of ‘X motif’ sequence elements. Also study ORF3b (not ORF3d) in other genomes and predict it is not functional. Use the same name (“ORF3b”) for both, following previous papers.
Pasquier et al. (BioRxiv preprint)	Computational search of hybrid human/SARS-CoV-2 dsRNA reveals unique viral sequences that diverge from other coronavirus strains	<a href="https://doi.org/10.1101/2020.04.08.031856">https://doi.org/10.1101/2020.04.08.031856</a>	[22]	Computational	ORF3d (cites Chan et al.)	No gene-specific results for ORF3d

Sadegh et al. <i>Nature Communications</i>	Exploring the SARS-CoV-2 virus-host-drug interactome for drug repurposing	<a href="https://doi.org/10.1038/s41467-020-17189-2">https://doi.org/10.1038/s41467-020-17189-2</a>	[23]	Computational	ORF3d (use data of Gordon et al.)	Claim immune-related, apparently based on SARS-CoV ORF3b. Explore relationships between interacting host proteins and potential drugs
Celik et al. <i>Placenta</i>	Factors preventing materno-fetal transmission of SARS-CoV-2	<a href="https://doi.org/10.1016/j.placenta.2020.05.012">https://doi.org/10.1016/j.placenta.2020.05.012</a>	[24]	Review	ORF3d (cite Chan et al.)	Functional inference based on SARS-CoV ORF3b
Garofalo et al. <i>Vaccines</i>	Prospects of Replication-Deficient Adenovirus Based Vaccine Development against SARS-CoV-2	<a href="https://doi.org/10.3390/vaccines8020293">https://doi.org/10.3390/vaccines8020293</a>	[25]	Review	ORF3d (note no homology to SARS CoV ORF3b)	ORF3d only briefly mentioned
Helmy et al. <i>Journal of Clinical Medicine</i>	The COVID-19 Pandemic: A Comprehensive Review of Taxonomy, Genetics, Epidemiology, Diagnosis, Treatment, and Control	<a href="https://doi.org/10.3390/jcm9041225">https://doi.org/10.3390/jcm9041225</a>	[26]	Review	ORF3b and ORF3d - both entities referred to and not distinguished	Conflate ORF3b and ORF3d, citing papers relating to each as if referring to the same entity
Taefehshokr et al. <i>Frontiers in Immunology</i>	Covid-19: Perspectives on Innate Immune Evasion	<a href="https://doi.org/10.3389/fimmu.2020.580641">https://doi.org/10.3389/fimmu.2020.580641</a>	[27]	Review	ORF3b and ORF3d - both entities referred to and not distinguished	Conflate ORF3b and ORF3d, citing papers relating to each as if referring to the same entity
Wu et al. <i>Frontiers in Microbiology</i>	Severe Acute Respiratory Syndrome Coronavirus 2: From Gene Structure to Pathogenic Mechanisms and Potential Therapy	<a href="https://doi.org/10.3389/fmicb.2020.01576">https://doi.org/10.3389/fmicb.2020.01576</a>	[28]	Review	ORF3b and ORF3d - both entities referred to and not distinguished	Discussion of potential function of ORF3d based on assumption of homology with SARS-CoV ORF3b
Yang et al. <i>Virology Journal</i>	SARS-CoV-2: characteristics and current advances in research	<a href="https://doi.org/10.1186/s12985-020-01369-z">https://doi.org/10.1186/s12985-020-01369-z</a>	[29]	Review	ORF3d (cite Chan et al.)	Implied functional inference based on SARS-CoV ORF3b. Claim 67 amino acids (should be 57).
Yi et al. <i>International</i>	COVID-19: what has been learned and to be	<a href="https://doi.org/10.7150/ijbs.45134">https://doi.org/10.7150/ijbs.45134</a>	[30]	Review	ORF3b and ORF3d - both	Conflate ORF3b and ORF3d, referring to both as if to the same entity

<i>Journal of Biological Sciences</i>	learned about the novel coronavirus disease				entities referred to and not distinguished	
Yoshimoto <i>The Protein Journal</i>	The Proteins of Severe Acute Respiratory Syndrome Coronavirus-2 (SARS CoV-2 or n-COV19), the Cause of COVID-19	<a href="https://link.springer.com/article/10.1007/s10930-020-09901-4">https://link.springer.com/article/10.1007/s10930-020-09901-4</a>	[31]	Review	ORF3d (cite Chan et al. and Gordon et al.)	Claim homology to SARS-CoV ORF3b
Zinzula <i>Biochemical and Biophysical Research Communications</i>	Lost in deletion: The enigmatic ORF8 protein of SARS-CoV-2	<a href="https://doi.org/10.1016/j.bbrc.2020.10.045">https://doi.org/10.1016/j.bbrc.2020.10.045</a>	[32]	Review	ORF3b and ORF3d - both entities referred to and not distinguished	Conflate ORF3b and ORF3d, referring to both as if to the same entity

## References

1. Wu F, Zhao S, Yu B, et al (2020) A new coronavirus associated with human respiratory disease in China. *Nature* 579:265–269
2. Konno Y, Kimura I, Uriu K, et al (2020) SARS-CoV-2 ORF3b Is a Potent Interferon Antagonist Whose Activity Is Increased by a Naturally Occurring Elongation Variant. *Cell Rep* 108185
3. Lokugamage KG, Hage A, de Vries M, et al (2020) Type I Interferon Susceptibility Distinguishes SARS-CoV-2 from SARS-CoV. *J Virol* 94.: <https://doi.org/10.1128/JVI.01410-20>
4. Xia H, Cao Z, Xie X, et al (2020) Evasion of Type I Interferon by SARS-CoV-2. *Cell Rep* 33:108234
5. Zhang J, Cruz-cosme R, Zhuang M-W, et al (2020) A Systemic and Molecular Study of Subcellular Localization of SARS-CoV-2 Proteins. *Cold Spring Harbor Laboratory* 2020.08.02.233023
6. Sa Ribero M, Jouvenet N, Dreux M, Nisole S (2020) Interplay between SARS-CoV-2 and the type I interferon response. *PLoS Pathog* 16:e1008737
7. Lei Q, Li Y, Hou H, et al (2020) Antibody dynamics to SARS-CoV-2 in asymptomatic COVID-19 infections. *Allergy* 26:1193
8. Nabeel-Shah S, Lee H, Ahmed N, Marcon E (2020) SARS-CoV-2 Nucleocapsid protein attenuates stress granule formation and alters gene expression via direct interaction with host mRNAs. *bioRxiv*

9. Yuen C-K, Lam J-Y, Wong W-M, et al (2020) SARS-CoV-2 nsp13, nsp14, nsp15 and orf6 function as potent interferon antagonists. *Emerg Microbes Infect* 9:1418–1428
10. Sun W (2020) The discovery of gene mutations making SARS-CoV-2 well adapted for humans: host-genome similarity analysis of 2594 genomes from China, the USA and Europe. *Cold Spring Harbor Laboratory* 2020.09.03.280727
11. Chan JF-W, Kok K-H, Zhu Z, et al (2020) Genomic characterization of the 2019 novel human-pathogenic coronavirus isolated from a patient with atypical pneumonia after visiting Wuhan. *Emerging Microbes & Infections* 9:221–236
12. Kim D-K, Knapp JJ, Kuang D, et al (2020) A Flexible Genome-Scale Resource of SARS-CoV-2 Coding Sequence Clones
13. Banerjee AK, Blanco MR, Bruce EA, et al (2020) SARS-CoV-2 Disrupts Splicing, Translation, and Protein Trafficking to Suppress Host Defenses. *Cell*. <https://doi.org/10.1016/j.cell.2020.10.004>
14. Gordon DE, Jang GM, Bouhaddou M, et al (2020) A SARS-CoV-2 protein interaction map reveals targets for drug repurposing. *Nature*. <https://doi.org/10.1038/s41586-020-2286-9>
15. Hachim A, Kavian N, Cohen CA, et al (2020) ORF8 and ORF3b antibodies are accurate serological markers of early and late SARS-CoV-2 infection. *Nat Immunol* 21:1293–1301
16. Hayn M, Hirschenberger M, Koepke L, et al (2020) Imperfect innate immune antagonism renders SARS-CoV-2 vulnerable towards IFN- $\gamma$  and - $\lambda$ . *Cold Spring Harbor Laboratory* 2020.10.15.340612
17. Lam J-Y, Yuen C-K, Ip JD, et al (2020) Loss of orf3b in the circulating SARS-CoV-2 strains. *Emerg Microbes Infect* 1–678
18. Laurent EMN, Sofianatos Y, Komarova A, Gimeno JP (2020) Global BioID-based SARS-CoV-2 proteins proximal interactome unveils novel ties between viral polypeptides and host factors involved in multiple COVID19 .... *bioRxiv*
19. Samavarchi-Tehrani P, Abdouni H, Knight JDR, et al A SARS-CoV-2 – host proximity interactome
20. St-Germain JR, Astori A, Samavarchi-Tehrani P, et al (2020) A SARS-CoV-2 BioID-based virus-host membrane protein interactome and virus peptide compendium: new proteomics resources for COVID-19 research. *Cold Spring Harbor Laboratory* 2020.08.28.269175
21. Michel CJ, Mayer C, Poch O, Thompson JD (2020) Characterization of accessory genes in coronavirus genomes. *Virol J* 17:131
22. Pasquier C, Robichon A (2020) Computational search of hybrid human/ SARS-CoV-2 dsRNA reveals unique viral sequences that diverge from other coronavirus strains. *Cold Spring Harbor Laboratory* 2020.04.08.031856

23. Sadegh S, Matschinske J, Blumenthal DB, et al (2020) Exploring the SARS-CoV-2 virus-host-drug interactome for drug repurposing. *Nature Communications* 11
24. Celik O, Saglam A, Baysal B, et al (2020) Factors preventing materno-fetal transmission of SARS-CoV-2. *Placenta* 97:1–5
25. Garofalo M, Staniszewska M, Salmaso S, et al (2020) Prospects of Replication-Deficient Adenovirus Based Vaccine Development against SARS-CoV-2. *Vaccines (Basel)* 8.: <https://doi.org/10.3390/vaccines8020293>
26. Helmy YA, Fawzy M, Elaswad A, et al (2020) The COVID-19 Pandemic: A Comprehensive Review of Taxonomy, Genetics, Epidemiology, Diagnosis, Treatment, and Control. *J Clin Med Res* 9.: <https://doi.org/10.3390/jcm9041225>
27. Taefehshokr N, Taefehshokr S, Hemmat N, Heit B (2020) Covid-19: Perspectives on Innate Immune Evasion. *Front Immunol* 11:580641
28. Wu J, Yuan X, Wang B, et al (2020) Severe Acute Respiratory Syndrome Coronavirus 2: From Gene Structure to Pathogenic Mechanisms and Potential Therapy. *Front Microbiol* 11:1576
29. Yang Y, Xiao Z, Ye K, et al (2020) SARS-CoV-2: characteristics and current advances in research. *Virol J* 17:117
30. Yi Y, Lagniton PNP, Ye S, Li E (2020) COVID-19: what has been learned and to be learned about the novel coronavirus disease. *International journal of*
31. Yoshimoto FK (2020) The Proteins of Severe Acute Respiratory Syndrome Coronavirus-2 (SARS CoV-2 or n-COV19), the Cause of COVID-19. *Protein J* 39:198–216
32. Zinzula L (2020) Lost in deletion: The enigmatic ORF8 protein of SARS-CoV-2. *Biochem Biophys Res Commun.* <https://doi.org/10.1016/j.bbrc.2020.10.045>