

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 |
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| Studies that use “ORF3b” to refer to the 22-codon ORF | | | | | | |
| Wu et al. <i>Nature</i> | A new coronavirus associated with human respiratory disease in China | https://doi.org/10.1038/s41586-020-2008-3 | [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 | https://doi.org/10.1016/j.celrep.2020.108185 | [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 | https://doi.org/10.1128/jvi.01410-20 | [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 | https://doi.org/10.1016/j.celrep.2020.108234 | [4] | Empirical | ORF3b (from primer sequences in their Table S1) | No inhibition of IFN- β production by ORF3b reported. |

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| Zhang et al. (BioRxiv preprint) | A Systemic and Molecular Study of Subcellular Localization of 2 SARS-CoV-2 Proteins | https://doi.org/10.1101/2020.08.02.233023 | [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 | https://doi.org/10.1371/journal.ppat.1008737 | [6] | Review | <i>ORF3b</i> (cites Konno et al.) | Their Fig. 1 shows the ORF at the wrong end of the SARS-CoV ORF3b homologous region |
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| Studies that use “ORF3b” with unclear referent | | | | | | |
| Lei et al. <i>Allergy</i> | Antibody dynamics to SARS-CoV-2 in asymptomatic COVID-19 infections | https://doi.org/10.1111/all.14622 | [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 | https://doi.org/10.1101/2020.10.23.342113 | [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 | https://doi.org/10.1080/22221751.2020.1780953 | [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 | https://doi.org/10.1101/2020.09.03.280727 | [10] | Computational | Referent unclear - no sequence details | Report sequence features |

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| Studies that use “ORF3b” to refer to the 57-codon ORF or to both, conflating the two | | | | | | |
| Chan et al. <i>Emerging Microbes & Infections</i> | Genomic characterization of the 2019 novel human-pathogenic coronavirus isolated from a patient with atypical pneumonia after visiting Wuhan | https://doi.org/10.1080/22221751.2020.1719902 | [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 | https://doi.org/10.1534/g3.120.401554 | [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 β ” (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 | https://doi.org/10.1016/j.cell.2020.10.004 | [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 | https://doi.org/10.1038/s41586-020-2286-9 | [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 | https://doi.org/10.1038/s41590-020-0773-7 | [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- γ and - λ | https://doi.org/10.1101/2020.10.15.340612 | [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 & Infections</i> | Loss of orf3b in the circulating SARS-CoV-2 strains | https://doi.org/10.1080/22221751.2020.1852892 | [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. |

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| <i>Infections</i> | | | | | 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 | https://doi.org/10.1101/2020.08.28.272955 | [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 | https://doi.org/10.1101/2020.09.03.282103 | [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 | https://doi.org/10.1101/2020.08.28.269175 | [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 | https://doi.org/10.1186/s12985-020-01402-1 | [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 | https://doi.org/10.1101/2020.04.08.031856 | [22] | Computational | ORF3d (cites Chan et al.) | No gene-specific results for ORF3d |

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| Sadegh et al. <i>Nature Communications</i> | Exploring the SARS-CoV-2 virus-host-drug interactome for drug repurposing | https://doi.org/10.1038/s41467-020-17189-2 | [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 | https://doi.org/10.1016/j.placenta.2020.05.012 | [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 | https://doi.org/10.3390/vaccines8020293 | [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 | https://doi.org/10.3390/jcm9041225 | [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 | https://doi.org/10.3389/fimmu.2020.580641 | [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 | https://doi.org/10.3389/fmicb.2020.01576 | [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 | https://doi.org/10.1186/s12985-020-01369-z | [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 | https://doi.org/10.7150/ijbs.45134 | [30] | Review | ORF3b and ORF3d - both | Conflate ORF3b and ORF3d, referring to both as if to the same entity |

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| <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 | https://link.springer.com/article/10.1007/s10930-020-09901-4 | [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 | https://doi.org/10.1016/j.bbrc.2020.10.045 | [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 |

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