Whole Genome Sequences Analyses of Indonesian Isolates SARS-CoV-2 Variants and their Clinical Manifestations

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Abstract

Background: SARS-CoV-2 virus is the cause of the global pandemic since the end of the year 2019. Since then, the virus has had mutations that cause different types of variants with various effects on those infected. This has complicated human intervention for prevention. Indonesia is one of the countries which was heavily affected by the pandemic specifically from May to August 2021, and it is a country that has recorded many distinct isolates.

Methods: GISAID database was used to obtain the Indonesian isolates while NCBI BLAST was utilized for comparison of all variants, and MAFFT version 7 for multi comparison.

Results: There were 9,488 isolates in Indonesia as of November 2021 where most include the Delta variant. Most of the isolates have mutations common to the ones from other countries. Although there are some atypical ones such as the mutation V1264L in the Delta variant that was suspected play a role in worsening the pandemic.

Conclusions: The Delta variant had the most mutations in the Spike protein, when compared to the Alpha and Beta variants, giving its important roles in infectivity and vigorous entry into cells, explaining why in the period of May to November 2021 in Indonesia, there was a rocket of cases for the Delta variant unlike the other variants

Background

Whole-genome sequencing (WGS) can give us more understanding of the biology and epidemiology of the SARS-CoV-2 virus, it is a very significant method of analysis. The infamous COVID-19 virus has a +ssRNA genome that is linear and it is from a family called coronaviruses (CoVs) which was named after its spikes on the surfaces that look like crowns. Coronaviruses can spread respiratory diseases in mammals and birds, and were discovered in humans in the mid-1960s. There are seven common human coronaviruses (HCoVs) which are divided again into either alpha or beta coronaviruses. Though, in the focus of this COVID-19 pandemic, the three main greatly pathogenic HCoVs manifested in 2002, 2012, and 2019, which include respectively the SARS-CoV, MERS-Cov, and the SARS-CoV-2 viruses (1).

From the various lineages evident by summer 2020, it was revealed that the SARS-CoV-2 has gone and is going through genetic mutations (2). Thus, characterization of the virus strains from different places is needed, Indonesia included. Based on the GISAID SARS-CoV-2 genome database, the lineage that is predominant in Indonesia is the B.1 lineage which varies from the Wuhan isolate (2), more specifically the B.1.466.2 variants (3).

Suitable actions can be taken when the virus is understood deeply. Following its different variants and pinpointing them can help us in preventing the pandemic from worsening in Indonesia. With the whole genome sequence of Indonesian isolates analyzed, including its wild type, differences and similarities can be examined. Between each isolate, there will be mutations in the sequences that form various

variants and types of the virus, which can affect people differently. The whole genome sequence will contain the nucleotide sequences for the structural proteins of the virus as well. These structural proteins include the spike, envelope, membrane, and nucleocapsid proteins, which are labelled S, E, M, and N respectively.

Specific to Indonesia, the determination of isolates, the study of their genomics, mutations, and types can bring about a much clearer distinction or observation on similarities. Any changes of the virus's genome, that translates to the virus proteins for example, can cause different clinical manifestations. The distinction between the Indonesian isolates, or the similarities observed, can help people know and plan better for prevention in the country, such as in the process of vaccine design or development.

Methods

Whole genome sequences (WGS) of SARS-CoV-2 viruses of Indonesia isolates were obtained from the GISAID and NCBI database. The randomly chosen isolates were selected and analyzed. The Wuhan SARS-CoV-2 genome NC_045512.2 from the NCBI database was used as references, as well as the Indonesian genome EPI_ISL_435282 from the GISAID database. Genome sequences of each variant found in Indonesia were obtained from the GISAID database. From the GISAID database, FASTA files were acquired. These genome sequences were compared to each other, as well as the Wuhan and Indonesian genome references, with Nucleotide BLAST and MAFFT version 7. The bioinformatics tools were used to compare the genome sequences for two alignments and multiple alignments, respectively.

Results

SARS-CoV-2 Variants Found in Indonesia

As of November 2021, there were around 9,488 SARS-CoV-2 viruses obtained in the GISAID database with the number of variants as shown in Table 1. From the collected isolates, there are four variants identified, namely: the Delta, Alpha, and Beta variants. The other variants, such as the Lambda, Omicron and Mu variants of the virus have not been found in Indonesia at the time the data was retrieved.

Table 1 Percentage of SARS-CoV-2 Variants from Indonesian Isolates
Deposited in GISAID Database as of November 2021

Database	Variants	Number of Viruses Collected	Percentage (%)
	Delta	5,512	58.1
	Alpha	78	0.822
	Beta	22	0.232
	Gamma	0	0
	Lambda	0	0
	Mu	0	0
GISAID	Wild Type	3,876	40.9

Figure 1 shows that the highest amount of Delta variant of the SARS-CoV-2 virus collected in Indonesia was in the time period from May 1 until August 1, 2021. The second highest is that also of the Delta variant from August 1 until November 1, 2021. It can be seen that the Delta variant is the variant with the highest number of infections when compared to the other three, and Table 1 supports this as well with the Delta variant being 55.7% out of all viruses collected and submitted into the GISAID database.

Discussion

Comparison of Wuhan Isolate (Reference) with Indonesian Wild-Type

By using NCBI BLAST, the first Wuhan SARS-CoV-2 Wild-Type Isolate (NC_045512.2) from NCBI was compared with an Indonesian Wild-Type Isolate (ESP_ISL_435282) from GISAID and it shows 99% query cover having slight differences on the N gene. The only differences include, from the Indonesian isolate nucleotide range of 29866 to 29920, two substitutions of nucleotides are present, the first from adenine (A) to guanine (G) and the second from guanine (G) to cytosine (C). Due to these, there were two substitutions of amino acids, the first from lysine (K) to arginine (R) and the second from aspartic acid (D) to histidine (H). At the beginning and last of the Wuhan isolate nucleotide range of 29292 to 29346, an aspartic acid exists, which is not present in the Indonesian isolate. The GISAID database indicates that the isolate ESP_ISL_435282 has an amino acid substitution on the non-structural protein 2 (NSP 2) from isoleucine (I) to valine (V), NSP2 I281V, and non-structural protein 12 (NSP12) from alanine (A) to valine (V) NSP12 A399V. NSP2 is claimed to be the key place of viral pathogenicity which could explain why the SARS-CoV-2 virus is more contagious than other SARS (4).

Comparison and Analysis of Wuhan Isolate (Reference) with Indonesian Delta Variant

The Wuhan isolate was then blasted with a Delta variant sample obtained from the GISAID database from Indonesia with high non-coding sequences. Pairwise similarities are shown with the line graph in Figure 2 and the empty spaces indicate the differences in genome or nucleotide sequences. The query

cover was 96%. It can be seen that the Wuhan isolate has more differences when compared to the Indonesian Delta variant sample rather than the Indonesian isolate genome that was chosen. These differences include substitutions and also deletions of nucleotides, and some do not cause any amino acid changes, but some do. There are patterns of substitutions as well that can be observed from Table 2 (Tables not shown in manuscript are in the supplementary file). The most frequent substitutions in the ORF1ab gene include substitutions from nucleotide C to G with seven occurrences out of twelve, and amino acid proline (P) substitutions which occurred five times out of twelve mutations.

In addition, the Indonesian virus isolate EPI_ISL_2537488.2 has an amino acid substitution from S26L on the ORF3a protein due to a C to T nucleotide substitution.

Table 2 shows that there is a well-known mutation on the S gene that codes for the spike protein of the Delta variant, which is the D614G amino acid substitution. D614G substitution increases infectivity and stability of virions, which results in a more vigorous entry of the virus into lung epithelial cells and causes viral replication enhancement (5). Other important substitutions in the spike protein from EPI_ISL_2537488.2 include L452R, P681R, T478K (6). P681R mutation causes increase in infectivity because of the slight increase in proteolytic processing (7). L452R mutation could also possibly reduce the binding ability of REGN10933 and P2B-2F6 antibodies to the variant strains (7) which causes increase in infectivity and decrease in the neutralizing activity of RBD-specific mAbs (8). L452R and T478K mutations were shown to affect ACE2 binding, showing increased stabilization of the RBD-ACE2 complex (6). P681R mutation, on the furin cleavage site, was also proven to aid transmissibility due to higher rate of membrane fusion and internalization. This was because of an increase in basicity of the poly-basic stretch, and the higher rate of S1-S2 cleavage (6). Aside from those common mutations found in the S gene, other mutations found from the EPI_ISL_2537488.2 include T19R, D950N, and V1264L.

It can be observed from the mutations of the ORF1ab gene in Table 2 that there are many C to thymine (T) nucleotide substitutions and the next leading one is A to G. These high frequencies of mutation happening on the ORF1ab gene is explained because its codes for non-structural proteins (NSP1 to NSP16) that has been studied to have the most missense mutation (9), as can be observed in Table 2, which explains why the ORF1ab gene has the most substitution mutations. The prominent C to T and A to G mutations can result in mutations of amino acids that affect replication of the virus' RNA (1). Bakhshandeh et al. states that C to T and A to G nucleotide substitutions in some positions of the ORF1ab gene are common in isolated genomes of Europe which causes severe infection with higher intensity than other regions (10). Mutations due to nucleotide and amino acid substitution can cause changes in the structure and function of proteins. Mutations in these genes could be assumed to help in inhibiting the immune response of the person with the virus, causing increased fatality. The virus is known to activate pathogenic Th1 to secrete proinflammatory cytokines (11).

Next, two different Delta variants isolated in Indonesia are blasted. The previous blast was from May 2021, and the next two were EPI_ISL_6262561 and EPI_ISL_6827388, from August and November 2021 with a 3-month period respectively. There are signature Spike mutations in the S gene typical for the Delta

and Delta Plus variant. From the BLAST results, the EPI_ISL_2537488.2 and EPI_ISL_6262561 isolates were shown to have an atypical V1264L mutation in the S gene, and in the EPI_ISL_6827388 isolate there are S704L and P1263L mutations.

Table 4 Comparison of Signature Delta and Delta Plus Variant Spike Mutations with Chosen Indonesian Isolates

No.	Indonesian Isolate Delta Variant Accession ID from GISAID	Signature Delta and Delta Plus Variant (*) Spike Mutations (Kannan et al. 2021)	Present Mutations	Other Mutations
1	EPI_ISL_2537488.2 (May 2021)	T19R, (V70F*), T95I, G142D, E156-, F157-, R158G, (A222V*), (W258L*), (K417N*), L452R, T478K, E484Q, D614G, P681R, and D950N	T19R, L452R, T478K, D614G, P681R, D950N	V1264L
2	EPI_ISL_6262561 (August 2021)		T19R, L452R, T478K, D614G, P681R, D950N	V1264L
3	EPI_ISL_6827388 (November 2021)		L452R, T478K, D614G, P681R, and D950N	S704L, P1263L

In 2020, the mutation S704L was geographically found in North America (13). P1263L typically in the USA, Scotland, and Europe (14), and V1264L in North America. Aside from the geographical location these mutations are mostly found in, not much about the effect of these mutations has been covered in previous research. Although EPI_ISL_6827388 in the GISAID database was said to have an amino acid substitution A222V, which would indicate a Delta Plus variant, from the BLAST results the mutation was not shown. The presence of an A222V mutation represents increased virality (15). P1263L is a mutation that is not included in the SARS-CoV-2 structure and is at the near end tail of the Spike protein (16). As seen from Table 4, there is an uncommon substitution of V1264L in two of the Indonesian isolates. This mutation was suspected to drive the pandemic in Indonesia (17). Research by (17) states that this mutation may improve the performance of the spike protein because it aids in optimizing the function of the protein's cytoplasmic tail, and helps in expanding the variant's evolutionary cage. Another major spike mutation from the BLAST results that was not present in the Indonesian isolates was the E484Q. The signature mutations mainly work in increasing the affinity of ACE2 which increases the transmissibility, pathogenicity, and risk of immune escape (18). E484Q in particular with L452R demonstrates a decrease in sensitivity to vaccine-elicited neutralizing antibodies (19). The absence of E484Q mutation may

indicate that vaccination could be more effective in Indonesia to fight the Delta variant. The mutation D950N has also been stated to improve the dynamics of the spike protein (20).

Table 5 Comparison of Signature Delta Variant Glycoprotein Mutations with Chosen Indonesian Isolates

No.	Indonesian Isolate Delta Variant Accession ID from GISAID	Recorded Delta Glycoprotein Mutations (No Variant Specified) (Jakhmola et al. 2021)	Signature Delta Glycoprotein Mutations (Ghosh et al. 2021)	Present Mutations
1	EPI_ISL_2537488.2 (May 2021)	D3G, V10A, V70F, H125Y, P123S, D209Y, T175M, K15R	D3G, I82T	-
2	EPI_ISL_6262561 (August 2021)			I82T
3	EPI_ISL_6827388 (November 2021)			I82T

Recorded mutations in the M gene of any other SARS-CoV-2 virus are shown in Table 5. The mutation V10A generates an N-myristylation site and P132S generates a casein kinase II phosphorylation site (22). M proteins interact with the other proteins and changes in PKC phosphorylation sites may change the virus' endocytosis and dynamic ruffling (23). This feature is shown from respiratory syncytial virus (RSV) which has this characteristic, and this helps evasion into the phagolysosome pathway (24). Table 5 shows that the isolates obtained from Indonesia only has the common M gene mutation for the Delta variant which is I82T mutation. I82T mutation in the M gene was proposed to improve biological fitness and changing uptake of glucose during viral replication (18).

Table 6 Comparison of Signature Delta Variant Nucleocapsid Mutations with Chosen Indonesian Isolates

No.	Indonesian Isolate Delta Variant Accession ID from GISAID	Recorded Delta Nucleocapsid Phosphoprotein Mutations in India (No Variant Specified) (Azad 2021)	Signature Delta Nucleocapsid Phosphoprotein Mutations (Syed et al. 2021)	Present Mutations
1	EPI_ISL_2537488.2 (May 2021)	P6T, P13L, S33I, R92S, G120R, L139F, A152S, A156S, R191L, S194L, S202N, R203K, G204R, M234I, G236C, P302S, P344S, D348Y, T362I, T393I	R203M, D377Y	R203M, G215C, D377Y
2	EPI_ISL_6262561 (August 2021)			D63G, R203M, G215C, D377Y
3	EPI_ISL_6827388 (November 2021)			D63G, R203M, G215C, D377Y

able Table 6 show that there is no similarity between normally found nucleocapsid phosphoprotein mutations in SARS-CoV-2 in India and the chosen Delta ones from Indonesia when compared, but has the proven highly prevalent missense mutation of R203M and D377Y, specifically for the Delta variant (21). Mutations in the N gene of the SARS-CoV-2 virus, specifically the R203M mutation in the Delta variant, has been shown to improve the mRNA packaging and replication which creates higher levels to 1000-fold of viral RNA in patients (26). The presence of this mutation could explain the rocketing of the number of SARS-CoV-2 cases in the period of May to November based on Figure 1 in Indonesia. The mutations D63G, R203M, G215C, D377Y are in the N protein, and due to the high immunogenicity and conservation of the N gene, N proteins with these mutations are available and used as a diagnostic tool (27).

Table 7 Comparison of Signature Delta Variant ORF7a Mutations with Chosen Indonesian Isolates

No.	Indonesian Isolate Delta Variant Accession ID from GISAID	Signature NS7a/ORF7a Mutations (Gupta et al. 2021)	Present Mutations
1	EPI_ISL_2537488.2 (May 2021)	V82A, T120I	In ORF7a: V82A In ORF7b: T40I
2	EPI_ISL_6262561 (August 2021)		In ORF7a: V82A, T120I In ORF7b, T40I
3	EPI_ISL_6827388 (November 2021)		In ORF7b, T40I

Mutations in the ORF7a protein can cause reduced immunity because it has been found to bind to human monocytes which would reduce antigen-presenting ability and can result in high expression of pro-inflammatory cytokines (29).

Table 8 Comparison of Signature Delta Variant ORF3a Mutations with Chosen Indonesian Isolates

No.	Indonesian Isolate Delta Variant Accession ID from GISAID	Signature NS3/ORF3a Mutations (Gupta et al. 2021)	Present Mutations
1	EPI_ISL_2537488.2 (May 2021)	S26L	S26L
2	EPI_ISL_6262561 (August 2021)		S26L
3	EPI_ISL_6827388 (November 2021)		S26L

The S26L mutation is common in ORF3a protein and was analyzed to have effects on the stability of the protein (30).

Table 9 Comparison of Signature Delta Variant ORF1ab Mutations with Chosen Indonesian Isolates

No.	Indonesian Isolate Delta Variant Accession ID from GISAID	Signature ORF1ab Mutations (Gupta et al. 2021)	Present Mutations	Other Mutations
1	EPI_ISL_2537488.2 (May 2021)	**Sub-lineage I**: A1306S, P2046L, P2287S, T3255I, T3446A, G5063S, P5401L, and A6319V **Sub-lineage II**: P309L, A3209V, V3718A, G5063S, and P5401L **Sub-lineage III**: A3209V, V3718A, T3750I, G5063S, and P5401L	**Sub-lineage I**: A1306S, P2046L, P2287S, T3255I, T3446A, G5063S, P5401L, A6319V **Common in B.1.617.2 lineage**: P4715L	P380L, T1496I, V2930L
2	EPI_ISL_6262561 (August 2021)	**Sub-lineage IV**: P309L, D2980N, and F3138S **Common in B.1.617.2 lineage**: P4715L	**Sub-lineage I**: A1306S, P2046L, T3255I, A6319V **Common in B.1.617.2 lineage**: P4715L	P380L, P1921Q, T3646A, I3738T
3	EPI_ISL_6827388 (November 2021)		**Sub-lineage I**: A1306S, P2046L, T3255I, A6319V, P2287S **Sub-lineage II and III**: G5063S, P5401L **Common in B.1.617.2 lineage**: P4715L	A11V, T1822I, S2114F, V2930L, T3646A

Table 9 shows all three of the isolates contain P4715L (NSP 12) mutation common in the B.1.617.2 lineage in the ORF1ab polyproteins of the SARS-CoV-2 virus, and contain many mutations from some sub-lineages of the Delta variant based on the research by (28). P4715L mutation is one of the four significant mutations which act as either a replicase or helicase, and play an important role in viral RNA synthesis (31). The three isolates contain other mutations that are not present and similar mutations as well. These other mutations could play an important role in improved virality due to the ORF1ab playing an essential role in the synthesis of the virus' RNA. P5041L mutation plays a role as helicase and other mutated amino acids could be assumed with roles, such as immune evasion, innate immunity interaction and inflammasome interaction (32).

It can be observed as well from the analyses with BLAST that the mutations in the E gene and other protein genes for the three Delta variant samples from Indonesian isolates are not commonly present.

The three Delta variants were aligned with MAFFT version 7 (https://mafft.cbrc.jp/alignment/server/index.html) and the phylogenetic tree was observed using the PhyloIO_, a sub menu of the online software as done in the to view the grouping of the isolates more clearly. It can be seen that the outgroup is only the original Wuhan isolate with accession ID NC_045512_2 from NCBI, while all the other three Indonesian isolates from GISAID are similar and grouped in one clade albeit the scale bars showing some sequence divergence. Thus, the Delta variant isolates from Indonesia have varying sequence divergence when compared to the original Wuhan wild type isolate and to each other.

Comparison and Analysis of Indonesian Alpha Variants

Table 10 shows that there is a mutation on the S gene that codes for the spike protein of the Alpha variant, which is N(Asparagine) to Y(Tyrosine) at position 501 (N501Y) which makes binding to ACE2 receptor stronger (33). The other substitutions are D614G, P681H, and T1238I. The mutation at position 614 for substitution of aspartic acid to glycine (D614G) has been explained before. The proline to histidine mutation at position 681 (P681H) was proven to not significantly affect viral entry or cell to cell spread (34). There is also a threonine to isoleucine substitution (T1238I), a substitution of a polar to non-polar amino acid at position 1238, this causes the forming of an extra helix because of the polarity change (35).

It can be seen from Table 11 that the S gene that codes for D614G substitution that affect the increases of infectivity and stability of virions, which results in a more vigorous entry of the virus into lung epithelial cells and causes viral replication enhancement (9) (23). Then there are other mutations R(Arginine) to M(Methionine), G(Glycine) to C(Cysteine), and D (Aspartic Acid) to Y(Tyrosine).

The Spike protein has been suspected to have more effects than its common signature. It was reported by a paper that the Spike protein could change its shape to make it prone to bind to more cells, and some other papers show that it itself can harm endothelial cells and can disturb the blood-brain barrier (36). Due to the Spike protein having a major role in the SARS-CoV-2 disease, for the Alpha and Beta Indonesian variants, the mutations in the S gene will be focused on to be analyzed.

Based on the analysis of Table 10 and 11, it can be concluded that some mutations in the Alpha variant are present as shown in Table 12.

Table 12 Comparison of Signature Alpha Variant Spike Mutations with Chosen Indonesian Isolates

Indonesian Isolate Alpha Variant Accession ID from GISAID	Signature Alpha Variant Mutations in Spike Protein (La Rosa et al. 2021)	Present Mutations
EPI_ISL_3138832	H69del (94%), V70del (95%), Y144del (94%), E484K (0.2%), S494P (0.3%), N501Y (97%), A570D (98%)	H69del, V70I, N501Y, A570D

Table 12 indicates that the Indonesian isolate contains the common H69del, N501Y, and A570D mutation, and instead of V70del, it has a nucleotide substitution of V70I. These mutations as mentioned are known to improve binding affinity to the ACE2 receptor and allow antibody escape. All the present mutations in the S gene for the Indonesian Alpha variant combined have possibilities to increase transmission and infection severity (38).

Comparison and Analysis of Indonesian Beta Variants

Based on the analysis of Table 13 and 14, it can be concluded that some mutations in the Beta variant are present as shown in Table 15.

Table 15 Comparison of Signature Beta Variant Spike Mutations with Chosen Indonesian Isolates

Indonesian Isolate Beta Variant Accession ID from GISAID	Signature Beta Variant Mutations in Spike Protein (La Rosa et al. 2021) (Mohammadi et al. 2021)	Present Mutations
EPI_ISL_2500469	L18F, D80A (87%), D215G (85%), L242- (78%), L243- (77%), R246I, P384L (1.2%), K417N (88%), E484K (87%), N501Y (87%), N501Y (87%), D614G, A701V, LAL 242-244 del	K417N, E484K, N501Y, D614G, A701V

The mutations from Table 15 combined can increase transmission and reinfection rates (38). The mutations present in the Indonesian isolates are all included in the Spike mutations of interest for the Beta variant based on (39) it was proven that these mutations have evidence that they affect transmissibility, immunity, and severity. It has been suggested that the mutations K417N and E484K may defeat the polyclonal antibody response, where E484K may facilitate escape from some antibodies (40). The mutations N501Y and D614G have been discussed before.

Clinical Manifestations

In the context of vaccines, the Delta variant with its many mutations that has been covered has a significant reduction for neutralization efficacy of sera from vaccines, similar to that of Beta variant while the Alpha variant only has modest reduction (38). All these could explain how the Delta variant spread

rapidly and fatally in Indonesia from May to August 2021, and why it still remains as the highest number of variant infecting residents from August to November 2021.

Table 16 Clinical Manifestations of Specific Variant Mutations from SARS-CoV-2 found in Indonesia

No	Variant	Protein	Mutation	Clinical Manifestation
1	Delta	S gene	V1264L	Helps in optimizing the function of the spike protein's cytoplasmic tail and expands the variant's evolutionary cage which increases performance of the virus.
2	Delta	S gene	L452R	Improves infectivity by reducing neutralizing activity of antibodies
3	Delta	S gene	T478K	Affects ACE2 binding
4	Delta	S gene	D614G	Improves infectivity and stability of virions causing more vigorous entry to host cells
5	Delta	S gene	P681R	Improves infectivity and transmissibility
6	Delta	S gene	D950N	Contribution in regulating dynamics of the spike protein
7	Delta	M gene	I82T	Increases uptake of glucose in viral replication and improves biological life of the virus
8	Delta	N gene	R203M	Increases packaging of mRNA and replication
9	Delta	ORF3a gene	S26L	Affects stability of protein
10	Alpha	S gene	H69del, N501Y, A570D, V70I	Increases transmission and infection severity, efficiency in virus entry, and binding affinity to ACE2 receptor for antibody escape
11	Beta	S gene	N501Y	Improves viral entry to cells
12	Beta	S gene	E484K	Aid in escape from antibodies

Conclusion

Most of the Indonesian isolates have mutations typical of that of variants found in the world, although there are some atypical ones, such as the mutation V1264L in the Delta variant that was suspected to worsen the pandemic in Indonesia. The Delta variant had the most mutations in the Spike protein, which have been found to play essential roles in infectivity, vigorous entry into host cells and more suspected major impacts, when compared to the Alpha and Beta variants. This could explain why in the period of May to November 2021 in Indonesia, there was a rocket of cases for the Delta variant unlike the other variants. As of November 2021, the number of SARS-CoV-2 cases in Indonesia is led by the Delta variant, followed by the Wild Type of the SARS-CoV-2 virus in Indonesia.

Declarations

Supplementary Information

The supplementary materials are available at https://docs.google.com/document/d/1gLd9mo0vXpJJKJOunL5L0Gy9J1QMVBr5xwdQOFlQjRg/edit?usp=sharing

Ethics approval and consent to participate

Not applicable

Consent for publication

Not applicable

Availability of data and materials

The datasets generated and/or analysed during the current study are available in the GISAID database, https://www.gisaid.org/, and NCBI database, National Center for Biotechnology Information (nih.gov)

Competing interests

We declare that there are no competing interests

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Authors' contributions

KAA designed the study. ELL, TDFH analysed the data and wrote the manuscript. All authors read and approved the final version of the manuscript

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Tables

Tables 2, 3, 10, 11, 13, and 14 are available in the supplementary files section.

Figures

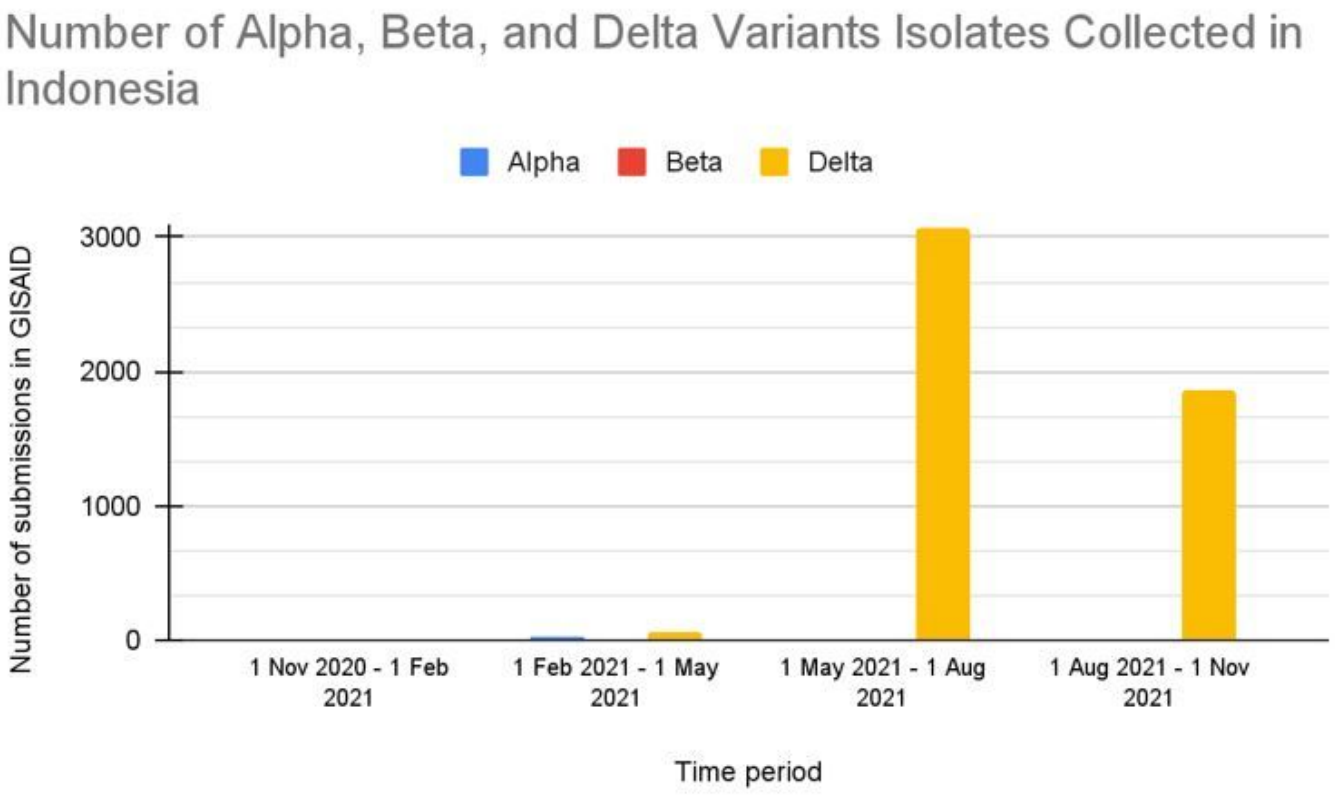

Figure 1

Number of Variants in Indonesia submitted into GISAID with when sample was collected

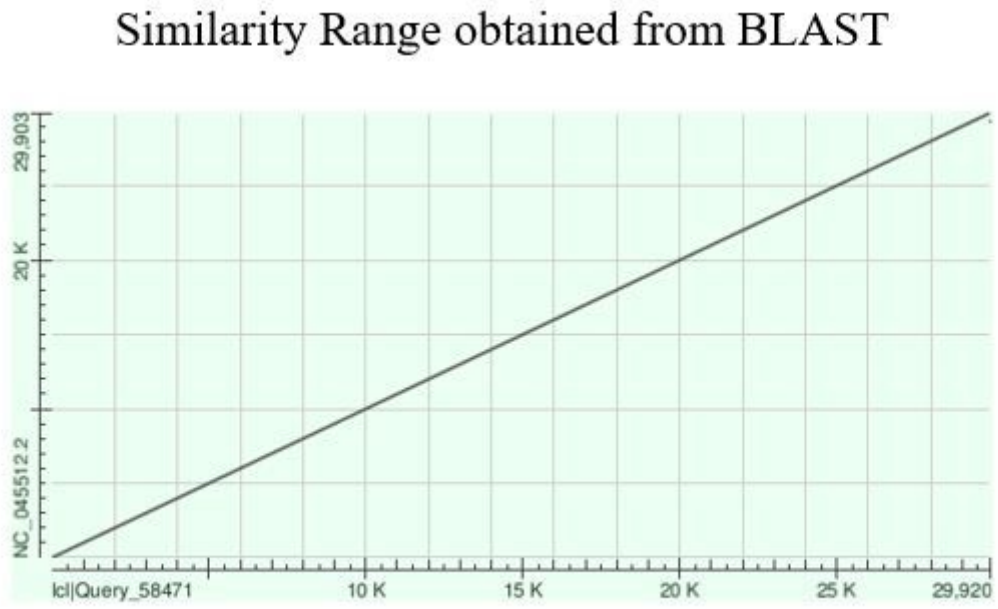

Figure 2

NC_045512.2 from NCBI vs Indonesian Wild-Type ESP_ISL_435282 from GISAID Nucleotide

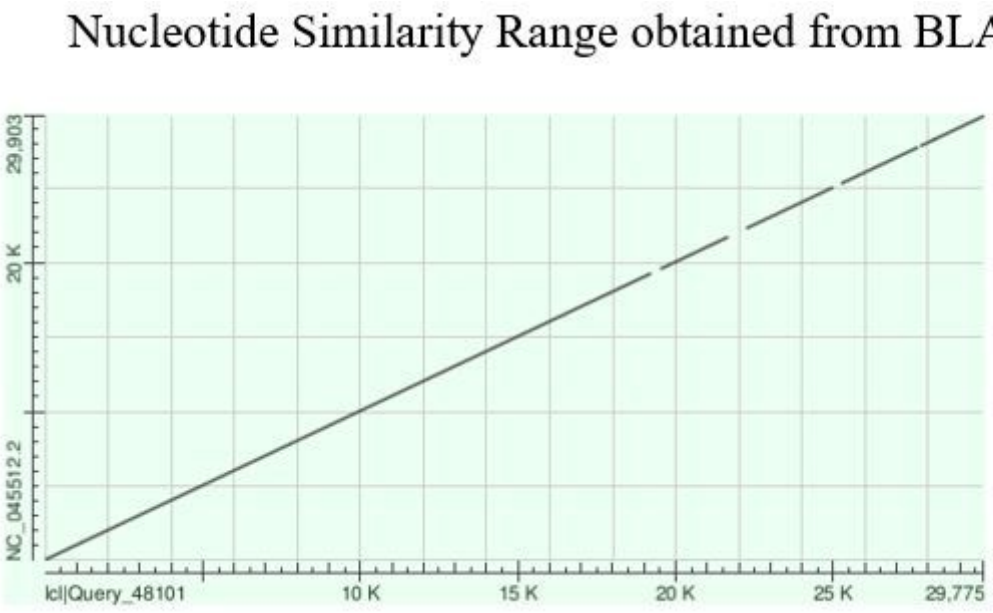

Figure 3

NC_045512.2 from NCBI vs Indonesian Delta EPI_ISL_2537488.2 from GISAID

Figure 4

Phylogenetic Tree of Chosen Delta Variant Indonesia SARS-CoV-2 Isolates with the

NC_045512.2 as reference

Supplementary Files

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- SupplementaryFile.pdf