Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Gene Reports 21 (2020) 100925

Contents lists available at ScienceDirect

Gene Reports

journal homepage: www.elsevier.com/locate/genrep

Genetic variations among SARS-CoV-2 strains isolated in China

Eltayib H. Ahmed-Abakur [a,b], Tarig M.S. Alnour [a,b,*]

[a] University of Tabuk, Faculty of Applied Medial Sciences, Department of Medical Laboratory Sciences, Tabuk, Saudi Arabia
[b] AlZaiem AlAzhari University, Faculty of Medical Laboratory Sciences, Department of Microbiology and Immunology, Khartoum North, Sudan

ARTICLE INFO

Keywords:
COVID-19, coronavirus
Pandemic
Viral genomics, blast
SARS-CoV-2

ABSTRACT

The rapid spread of COVID-19, which has led to a global pandemic, has placed public health systems under severe pressure. Identifying variations in SARS-CoV-2 strains from different regions is a key factor for understanding the pathogenic mechanisms, aid in diagnosis, prevention and therapy of this disease. The present study is an analytical descriptive study aimed to determine genetic variations among SARS-CoV-2 strains isolated in China. Sixty six complete genome sequences of the virus were retrieved from NCBI, the sequence of original Wuhan strain accession number NC 045512 was used as the reference sequence. Each genome sequence was blasted against the original Wuhan strain; the analysis was done using NCBI Nucleo-blast. The collected sequences showed 10 different variants. One hundred and thirty four mutations were identified among the variants of SARS-CoV-2 in this study; most of them 52.2% (70/134) were missense point mutation, majority of the mutations 65.7% (88/134) occurred in the open reading frame a/b (ORFab), few mutations occurred in the structural viral genome, each of spike (S) gene and nucleocapsid (N) gene showed 4 mutations; 2 silent point mutations and 2 missense point mutations occurred in each gene whereas membrane (M) gene showed silent point mutation and no mutation observed in the envelope E gene. The remarkable observation in this study showed by Yunnan variant accession number MT226610 which exhibited high incidence of mutations, it displayed 28 different point mutations; only 3(10.7%) of them were silent mutations while the rest were missense mutations. Our analysis showed several mutations including spike S gene and membrane M gene which may be responsible for a change in the structures of target proteins.

1. Introduction

COVID-19 is one of the most contagious pandemics faced the world, initially it was first reported in December 2019 in Wuhan, China (Abduljalil and Abduljalil, 2020; Deng et al., 2020; Lam, 2020), this pandemic disease had spread to 215 countries and territories around the world, more than 18 million person had infected to date (August 02, 2020), with 689,164 reported deaths, and the cases have increased as high as 5 times in less than a month (worldomater, n.d.). The case numbers under-estimate the real number of infections due to mild or asymptomatic cases and inability to test all population (Deng et al., 2020).

Coronavirus disease (COVID-19) is a respiratory illness primarily affects the respiratory system causing flu-like illness with symptoms such as cough, fever, and in severe cases, difficulty in breathing (Abduljalil and Abduljalil, 2020; Khailany et al., 2020; Naqvi et al., 2020). Because of its marked similarity in terms of biological nature and clinical symptoms with the causative agent of severe acute respiratory syndrome (SARS), the novel coronavirus was termed SARS-CoV-2 by the International Committee on Taxonomy of Viruses (Abduljalil and Abduljalil, 2020; Lokmana et al., 2020). SARS-CoV-2 has been identified as enveloped, positive-sense un-segmented single-stranded RNA viruses that belong to the genus Betacoronavirus, Coronaviridae family of Nidovirales order (Lokmana et al., 2020; Raza et al., 2020; Uddin et al., 2020). They have been classified into four genera that include α-, β-, γ-, and δ- coronaviruses (Raza et al., 2020). Among them, α- and β- CoVs infect mammals, γ- coronaviruses infect avian species, and δ-coronaviruses infect both mammals and avian (Khailany et al., 2020; Naqvi et al., 2020; Lokmana et al., 2020). The genome size of SARS-CoV-2 approximately 29.9 kb encoding 27 proteins from 14 ORFs including 15 nonstructural, 8 accessory, and 4 major structural proteins and lacking the haemagglutinin-esterase gene (Abduljalil and Abduljalil, 2020; Khailany et al., 2020; Lokmana et al., 2020). The longest ORF (ORF1 a/b) is located at the 5′ terminus encodes for nonstructural proteins, the 3′ terminus of the genome encodes for structural proteins including surface (S), envelope (E), membrane (M), and nucleocapsid (N) proteins with

* Corresponding author at: University of Tabuk, Faculty of Applied Medial Sciences, Department of Medical Laboratory Sciences, Saudi Arabia.
E-mail addresses: eosman@ut.edu.sa (E.H. Ahmed-Abakur), telnour@ut.edu.sa (T.M.S. Alnour).

https://doi.org/10.1016/j.genrep.2020.100925
Received 30 August 2020; Received in revised form 7 October 2020; Accepted 14 October 2020
Available online 17 October 2020

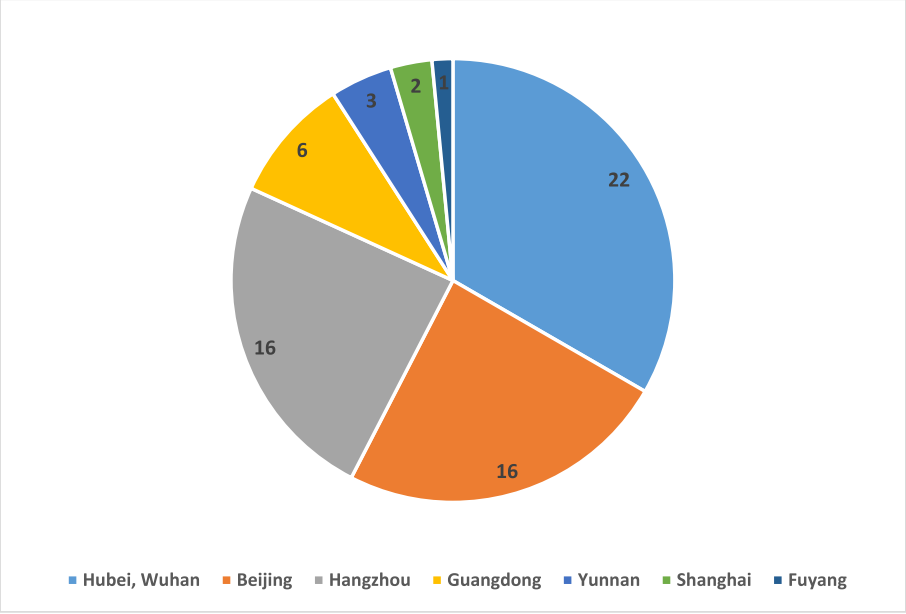

Fig. 1. Distribution of genome sequences of SARS-CoV-2 strains among different regions (province/city) in china.

other accessory proteins (Abduljalil and Abduljalil, 2020; Khailany et al., 2020; Naqvi et al., 2020; Lokmana et al., 2020; Uddin et al., 2020). Based on the phylogenetic studies, the SARS-CoV-2 is categorized in the same lineage that includes SARS coronavirus (SARS-CoV) which causes Severe Acute Respiratory Syndrome (SARS) and MERS-CoV which causes Middle East respiratory syndrome (Lokmana et al., 2020; Raza et al., 2020). The SARS-CoV-2 genome shared about 79–82% sequence identity with MERS-CoV and SARS-CoV (Naqvi et al., 2020; Raza et al., 2020; Uddin et al., 2020). Moreover, the SARS-CoV-2 genome has great sequence similarity (89–96.3%) with two bat coronaviruses; bat-SLCoVZC45 and bat-SL-CoVZXC21 (Abduljalil and Abduljalil, 2020; Uddin et al., 2020).

The World Health Organization (WHO) declared SARS-CoV- 2 as a public health emergency of international concern on 30 January 2020 and as a controllable pandemic on 11 March 2020 (World health organization, n.d.). No effective treatment is available for this disease and much is still unknown about it (Abduljalil and Abduljalil, 2020; From the American Association of Neurological Surgeons (AANS) et al., 2018). However, genomic epidemiology of emerging viruses has proven to be a useful tool for outbreak investigation and for tracking virus evolution (Deng et al., 2020). Therefore, the present study aimed to determine the genetic variations in SARS-CoV-2 strains isolated in China.

2. Method and materials

This study is an analytical descriptive study, aimed to determine the genetic variations associated with the SARS-CoV-2 among different strains isolated in China. The study covered 6 Chinese regions (City/province), 67 genome sequences of the virus were retrieved from NCBI Virus Variation Resource repository (https://www.ncbi.nlm.nih.gov/genbank/sars-cov-2-seqs/) and these strains have been studied according to the time of isolation and the origin. One strain was excluded due to incomplete genomic sequences. The NCBI sequence of SARS-CoV-2 accession number NC045512 which represent original Wuhan strain (submitted on December 2019), was used as the reference sequence. Each genome sequence was blasted against the original Wuhan strain. The analysis was done using NCBI Nucleo-blast and variations of the nucleotides and proteins were reported.

3. Results

Complete genome sequences of the virus from 6 different regions in China (Fig. 1) showed 10 different variants (Table 1). The 1st variant which considered the origin of disease was Wuhan strain which submitted to the gene bank on the mid of December 2019 (accession number NC045512). On the same month similar strains were identified in Beijing (accession numbers MT291827 & MT291830) and in Shanghai (accession number MN908947). The Wuhan strain (variant 1) was continuing to infect human until the end of January 2020.

The sequence identity matrix showed high homology among the viral strains, out of 66 genome sequences of the SARS-CoV-2, 32 (48.5%) were identical (Table 2a) and showed complete genetic similarity to the sequence of the 1st isolate of SARS-CoV-2 (accession number NC045512). Twenty two strains 22 (33.3%) showed 99.99% identity and 10 (15.2%) strains displayed 99.98% identity, the rest 2(3.03%) exhibited less than 99.98% identity to the Wuhan strain NC045512 (Tables 2b, 2c).

One hundred and thirty four mutation were identified among the variants of SARS-CoV-2 in this study; most of them 52.2% (70/134) were missense point mutations, the silent point mutation account for 29.1% (39/134) while non-encoding mutations were 18.7% (25/134), majority of the mutations 65.7% (88/134) occurred in the open reading frame 1 a/b (ORF1 a/b) which covers most of the viral genome (21,555/29,904 nucleotides), followed by 18.7% (25/134) in 3′UTR terminal loop. All the mutations occurred in the ORF3a, ORF8 and ORF10 were missense point mutations (Fig. 2). Very few mutations observed in structural viral genome; each of spike (S) gene and nucleocapsid (N) gene showed 4 mutations; 1.5% (2/134) silent point mutations and 1.5% (2/134) missense point mutations occurred in each gene, while membrane (M) gene showed only one silent point mutation and no mutation observed in the envelope (E) gene (Fig. 2).

The main observation in this study showed in Yunnan variant (accession number MT226610) which exhibited high incidence of mutations, it displayed 28 different point mutations; only 3(10.7%) of them were silent while the rest were missense mutations. Most of these mutations 26 (89.3%) occurred in the ORF1ab; one mutation occurred in the S gene (T21784A) and one in ORF8. Another missense point in spike gene mutation was C21711T observed in the Fuyang variant accession number MT281577 (Table 2b).

Table 1
Showed the date of collection, city and main mutations of variants of SARS-CoV-2 which specified in this study.

Red color pointed the mutation.
(V) denoted for variant.
(S) Silent mutation.
(M) missense mutation.
*Insignificant mutation in the 5′ end.
**Mutation in the 3′ terminal loop with no reflection of amino acid changes.

Table 2a
showed the completely identical sequences of SARS CoV-2 variants compared to Wuhan variant NC045512.

Number	City	Accession #	Collection date	% of identity to Wuhan
1	Wuhan	NC_045512	December 2019	
2	Beijing	MT093631	8-1-2020	100%
3	Beijing	MT039874	22-1-2020	100%
4	Wuhan	MN988669	2-1-2020	100%
5	Wuhan	MN988668	2-1-2020	100%
6	Shanghai	MN908947	12-2019	100%
7	Hangzhou	MT039873	20-1-2020	100%
8	Wuhan	MT019533	1-1-2020	100%
9	Wuhan	MT019532	30-12-2019	100%
10	Wuhan	MT019531	30-12-2019	100%
11	Beijing	MT291830	30-12-2019	100%
12	Beijing	MT291829	30-12-2019	100%
13	Beijing	MT291828	30-12-2019	100%
14	Beijing	MT291827	30-12-2019	100%
15	Wuhan	MN996530	30-12-2019	100%
16	Wuhan	MN996528	30-12-2019	100%
17	Hangzhou	MT253710	21-1-2020	100%
18	Hangzhou	MT253709	21-1-2020	100%
19	Hangzhou	MT253708	21-1-2020	100%
20	Hangzhou	MT253707	25-1-2020	100%
21	Hangzhou	MT253706	22-1-2020	100%
22	Hangzhou	MT253705	22-1-2020	100%
23	Hangzhou	MT253704	25-1-2020	100%
24	Hangzhou	MT253703	25-1-2020	100%
25	Hangzhou	MT253702	21-1-2020	100%
26	Hangzhou	MT253701	21-1-2020	100%
27	Hangzhou	MT253700	25-1-2020	100%
28	Hangzhou	MT253699	24-1-2020	100%
29	Hangzhou	MT253698	23-1-2020	100%
30	Hangzhou	MT253697	23-1-2020	100%
31	Hangzhou	MT253696	23-1-2020	100%
32	Wuhan	LR757996	1-1-2020	100%

Variants 2–6 (Table 1) sequenced during December 2019 were detected in all Chinese regions except Hangzhou, these variants characterized by missense point mutation in the Open Reading Frame 1 A and B (ORF1 a/b). Variant 2 (accession number LR757996) was observed later in Beijing, Guangdong and Yunnan. This variant showed C8782T silent point mutation in ORF1 a/b and T28144C missense mutation in the ORF8, upon spread in various regions extra mutations were observed in Beijing (accession numbers MT291826, MT291831-34, MT135043); Guangdong (accession numbers MN938384, MN975262 and MT123292); and in Yunnan (accession numbers MT049951 and MT226610), C29095T is silent mutation of the N gene observed in the Guandong variants (accession numbers MN938384 and MN975262). Silent point mutation T21644A in S gene was observed in Yunnan variants (accession numberMT049951) (Table 2c).

Variant 7 (accession number MT259230), sequenced in January 2020 was detected in Wuhan, it characterized by C20694T missense point mutation in the ORF1a/b and G29868C in the 3′ terminal loop among several other mutations. Guangdong variants 8 (accession numbers MT123291 and MT123293) is seems to share C17373T silent point mutation with the variant 7, both were isolated on January 2020 but Guangdong variant is earlier. An important missense point mutation (C29302T) of the N gene was observed in the Guangdong variant 9 (accession number MT123290) which isolated in February 2020. Yunnan variant 10 (accession number MT396241) showed 2 different mutation in the ORF1 a/b site (Table 1).

4. Discussion

Identifying variations in strains from different regions is a key factor for understanding the pathogenic mechanisms of this disease (From the

Table 2b
Showed the mutations in SARS CoV-2 Yunnan variant MT226610 compared to Wuhan variant NC045512.

Number	City	Accession #	Collection date	% of identity to Wuhan	Mutations locations	Target gene	Protein
33	Yunnan	MT226610	20-1-2020	99.91%	G4288T	ORF1ab	E1341D
					A4307C	ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab	K1348Q
					A7479G	ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab	N2405S
					C8782T	ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab ORF1ab	Silent M
					G11083T	ORF1ab	L3606F
					G11207C	S gene	A3648P
					T11233G	ORF8	Silent M
					G12041C		D3926H
					G12160C		E3965D
					G12202C		K3979N
					G12208T		K3981N
					G12355C		Q4030H
					G12378A		R4038K
					G12464T		A4067S
					G12467T		A4068S
					G12491T		D4076Y
					G12514C		Silent M
					G12572T		D4103Y
					G12578T		D4105Y
					G12582T		S4106I
					G12600A		S4112N
					G12660C		R4132T
					G12685C		Q4140H
					G12773T		A4170S
					G12793T		K4176N
					G20980C		D6906H
					T21784A		N6997K
					T28144C		L9117S

Table 2c
Showed the mutations and degrees of sequences identity of SARS CoV-2 variants isolated in china compared to Wuhan variant NC045512.

Number	City	Accession #	Collection date	% of identity to Wuhan	Mutations locations	Target gene	Protein
34	Yunnan Province	MT396241	6-3-2020	99.99%	G1433A	ORF1ab	E390K
					G15910T	ORF1ab	D5216Y
35	Shenzhen, Guangdong	MN975262	11-1-2020	99.98%	C9561T	ORF1ab	S3099L
					T15667C	ORF1ab	Silent
					T28144C	ORF8	L9117S
					C29095T	N gene	Silent
36	Fuyang, Anhui	MT281577	10-3-2020	99.98%	T721C	ORF1ab	Silent V544L
					G1895T	ORF1ab	L3606F
					G11083T	ORF1ab	S6973L
					C21711T	S gene	G8450V
					G26144T	ORF3a	–
					A29695G	3′UTR	
37	Beijing	MT291835	27-1-2020	99.99%	T7077C	ORF1ab	Silent Silent
					C15861G	ORF1ab	
38	Beijing	MT291834	28-1-2020	99.98%	G2867A	ORF1ab	Silent Silent
					T4402C	ORF1ab	L1599F
					G5062T	ORF1ab	Silent
					C8782T	ORF1ab	L9117S
					T28144C	ORF8	
39	Beijing	MT291833	28-1-2020	99.99%	T4402C	ORF1ab	Silent
40	Beijing	MT291832	25-1-2020	99.99%	G5062T	ORF1ab	L1599F
41	Beijing	MT135044	28-1-2020	99.99%	C8782T	ORF1ab	Silent
42	Beijing	MT135042	28-1-2020	99.99%	T28144C	ORF8	L9117S
43	Beijing	MT135041	28-1-2020	99.99%			
44	Beijing	MT291831	24-1-2020	99.98%	C8782T	ORF1ab	Silent
					G11937A	ORF1ab	C3891Y
					C16293T	ORF1ab	Silent
					T28144C	ORF8	L9117S
45	Beijing	MT135043	28-1-2020	99.98%	T4402C	ORF1ab	Silent
					G5062T	ORF1ab	L1599F
					C8782T	ORF1ab	Silent
					T28144C	ORF8	L9117S
					A29301T	N gene	D9503V
46	Guangdong, Guangzhou	MT123290	5-2-2020	99.99%	A63T	?	–
					C15324T	ORF1ab	Silent
					C29302T	N gene	P9504S
47	Guangdong, Guangzhou	MT123293	29-1-2020	99.98%	G654A	ORF1ab	G130E
					G6819T	ORF1ab	S2185I
					T6996C	ORF1ab	I2244T
					C17373T	ORF1ab	Silent
					G29527A	3′UTR	–
48	Guangdong, Guangzhou	MT123292	27-1-2020	99.99%	C8782T	ORF1ab	Silent
					T12534C	ORF1ab	T4090I
					T13072C	ORF1ab	Silent
					T28144C	ORF8	L9117S
49	Guangdong, Guangzhou	MT123291	29-1-2020	99.99%	G6819T	ORF1ab	S2185I
					C17373T	ORF1ab	Silent
					C19611T	ORF1ab	T6449I
					G29527A	3′UTR	–
50	Wuhan	MT259231	25-1-2020	99.99%	C20692T	ORF1ab	P6810S
51	Wuhan	MT259229	26-1-2020	99.99%	G29868C	3′UTR	
52	Wuhan	MT259230	25-1-2020	99.99%	C17373T	ORF1ab	Silent
					G29868C	3′UTR	
53	Wuhan	MT259228	26-1-2020	99.98%	C20692T	ORF1ab	P6810S
					T29546G	3′UTR	
					T29846A	3′UTR	
					T29847G	3′UTR	
					G29861A	3′UTR	
					G29868C	3′UTR	
54	Wuhan	MT259227	26-1-2020	99.97%	T1623C	ORF1ab	I453T
					T3299C	ORF1ab	Silent
					C20692T	ORF1ab	P6810S
					C25158T	S gene	Silent
					A29843G	3′UTR	
					T29844G	3′UTR	
					T29846G	3′UTR	
					T29847G	3′UTR	
					G29868C	3′UTR	
55	Wuhan	MT259226	10-1-2020	99.98%	T565C	ORF1ab	
					C17825T	ORF1ab	T5853I
					T27384C	ORF6	Silent
					G29573A	ORF10	V9594I
					G29861A	3′UTR	
					G29868C	3′UTR	

(*continued on next page*)

Table 2c (*continued*)

Number	City	Accession #	Collection date	% of identity to Wuhan	Mutations locations	Target gene	Protein
56	Wuhan	MT019530	30-12-2019	99.98%	T104A T111C T112G C119G T120C G124A	5′ end leading sequence mutation	–
57	Wuhan	MT019529	23-12-2019	99.99%	A3778G A8388G T8987A	ORF1ab ORF1ab ORF1ab	Silent N2708S F2908I
58	Shanghai	MT121215	2-2-2020	99.99%	C6026T C12473T	ORF1ab ORF1ab	P1921S Silent
59	Guangdong	MN938384	10-1-2020	99.99%	C8782T T28144C C29095T	ORF1ab ORF8 N gene	Silent L9117S Silent
60	Beijing	MT291826	30-12-2019	99.99%	T4946C C8782T T28144C	ORF1ab ORF1ab ORF8	S1561P Silent L9117S
61	Wuhan	MN996531	30-12-2019	99.99%	A8001C C9534T	ORF1ab ORF1ab	D2579A T3090I
62	Wuhan	MN996529	30-12-2019	99.99%	G7016A A21137G	ORF1ab ORF1ab	G2251S K6958R
63	Wuhan	MN996527	30-12-2020	99.99%	G21318A A24325G	ORF1ab S gene	D7018N Silent
64	Yunnan	MT049951	17-1-2020	99.98%	C75A C8782T G11083C T21644A T28144C	ORF1ab ORF1ab ORF1ab M gene ORF8	– Silent L3606F Silent L9117S
65	Wuhan	LR757998	16-12-2019	99.99%	C6968A T11764A	ORF1ab ORF1ab	Silent Silent
66	Wuhan	LR757996	26-12-2019	99.99%	C8782T T28144C	ORF1ab ORF8	Silent L9117S

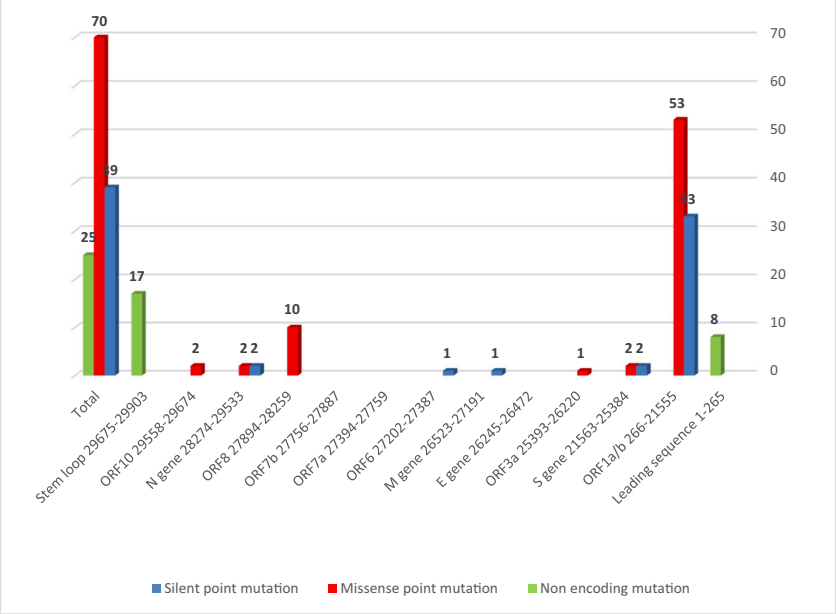

Fig. 2. Types and distribution of mutations in SARS-CoV-2 variants.

American Association of Neurological Surgeons (AANS) et al., 2018). The present study exhibited 10 different variants; this finding was in alignment with several reports which showed several variants of SARS-CoV-2 (Abduljalil and Abduljalil, 2020; Uddin et al., 2020; Yu et al., 2020). Forster et al., carried out study using a phylogenetic network analysis approach on 160 full length genomes and reported that the virus seems to be evolving into three central variants distinguished by amino acid changes (Forster et al., 2020). Based on phylogenetic analyses Abduljalil and Abduljalil concluded that SARS-CoV-2 genomes sequenced showed genomic variations among strains from different regions and countries through different period (Abduljalil and Abduljalil, 2020). This diversity may be owing to the nature of virus itself where the RNA viruses tend to harbor error-prone RNA dependent RNA polymerases which makes occurrence of mutations and recombination events rather frequently (Uddin et al., 2020). Kupferschmidt stated that SARS-CoV-2 like other coronaviruses appears to accumulate, on average, one or two mutations per month (Kupferschmidt, 2020).

Our study showed that the majority of the mutations 65.7% (88/134)

occurred in the open reading frame 1 a/b (ORF1 a/b), similar result concerning the frequency of mutations in ORF1 a/b was reported by Khailany et al. (2020)) ORF1 a/b covers most of the viral genome and encoded for nonstructural proteins (Abduljalil and Abduljalil, 2020; Lokmana et al., 2020) that collectively involved in virus replication and possibly in immune evasion (Abduljalil and Abduljalil, 2020). Hurst et al. (2013) reported that there was a basic connection between the nsp3 association and the inception of coronavirus infection whereas Wan et al. mentioned that the area of ORF1a/b is the most important factor among coronaviruses (Wan et al., 2020).

Spike protein (S) of SARS coronavirus (SARS-CoV) play a major role in SARS-CoV-2 pathogenesis, it attaches the virus to its cellular receptor, angiotensin-converting enzyme 2 (ACE2) (World health organization, n. d.; Wan et al., 2020). Our study showed limited mutations in the structural genes, each of spike (S) gene and nucleocapsid (N) gene revealed 4 mutations; 2 silent point mutations and 2 missense point mutations, while membrane (M) gene showed only one silent point mutation and no mutation observed in the envelope (E) gene, this findings are in alignment with other study conducted by Lokmana et al. who analyzed 320 whole-genome sequences and 320 spike protein sequences of SARS-CoV-2 and reported just one deletion in the spike (S) protein (Lokmana et al., 2020) and with Wang et al. who studied genetic variations among 95 full length genomic sequences of SARAS-CoV-2 strains and they stated that SARS-COV-2 is relatively conserved, especially in the E region (Wang et al., 2020). The viral spike protein is thought to have a crucial role in drug and vaccine development as reported previously in managing the viruses like SARS-CoV and MERS-CoV (Tian et al., 2020), therefore mutation in this gene might affect the severity and spreads of the SARS-CoV-2 as well suppresses the efforts of developing vaccine.

In this study the Yunnan variant accession number MT226610 exhibited high incidence of mutations; it displayed 28 different point mutations including one mutation in the S gene (T21784A), another missense point in spike gene mutation was C21711T observed in the Fuyang variant accession number MT281577. The T21784A point mutation can leads to replaced asparagine by leucin whereas the MT281577 point mutation leads to substitute serine with leucin in the position 6973 of the amino acid sequence, this altering might affect the viral protein functions. However, hyper mutations may change the virus proteins, thus affecting the virus behavior and potency; this may lead to different waves of disease and complicated the efforts of producing vaccines.

Several common gene mutations were observed among the SARS-CoV-2 sequence in China. These mutations follow standard roles and common among different countries. The most counted mutations in our study were T4402C, G5062T, C8782T, C17373T, C20692T, T28144C, C29095T, and G29868C. The T4402C mutation which leads to silent mutation in the ORF1 a/b gene segment was noted in the strain isolated from Beijing (variant # 2) and always associated with C8782T, G5062T and T28144C mutations, similar T4402C and G5062T point mutation were observed in two strains isolated in South Korean (Yang et al., 2020), C8782T was the predominant mutation reported in SARS-CoV-2 gene mutation around the world (Yang et al., 2020; Mercatelli and Giorgi, 2020), this mutation is always coexisting with the missense point mutation of the ORF8 gene segment T28144C (Mercatelli and Giorgi, 2020). The C17373T silent mutation was observed in Wuhan and tends to spread to Guangdong. Same mutation had noticed in Singapore and USA (Li et al., 2020). The C20692T was restricted to Wuhan and was coexisting with the G29868C gene mutation of the 3′ terminal loop. This mutation was also noticed by Sharp and Dange (2020). The C29095T mutation of the N gene was also reported in the USA (Yang et al., 2020).

5. Conclusion

Study the sequencing of coronavirus at different points/regions can tell how the virus is adapting and can indicate the disease epidemiology, pathogenesis and may help in developing treatment and prevention. Our analysis showed several mutations including spike S gene and membrane M gene which may be responsible for a change in the structures of target proteins.

Declaration of competing interest

This research is NOT funded fully or partially from any governmental or non-governmental agencies.

References

Abduljalil, J.M., Abduljalil, B.M., 2020. Epidemiology, genome, and clinical features of the pandemic SARS-CoV-2: a recent view. New Microbes New Infect. 35, 100672. Published 2020 Mar 31. https://doi.org/10.1016/j.nmni.2020.100672.

Deng, X., et al., 2020. Genomic surveillance reveals multiple introductions of SARS-CoV-2 into northern California. Science. https://doi.org/10.1126/science.abb9263.

Forster, P., Forster, L., Renfrew, C., Forster, M., 2020. Phylogenetic network analysis of SARS-CoV-2 genomes. Proc. Natl. Acad. Sci. U. S. A. 117, 9241–9243.

From the American Association of Neurological Surgeons (AANS), American Society of Neuroradiology (ASNR), Cardiovascular and Interventional Radiology Society of Europe (CIRSE), Canadian Interventional Radiology Association (CIRA), Congress of Neurological Surgeons (CNS), European Society of Minimally Invasive Neurological Therapy (ESMINT), European Society of Neuroradiology (ESNR), European Stroke Organization (ESO), Society for Cardiovascular Angiography and Interventions (SCAI), Society of Interventional Radiology (SIR), Society of NeuroInterventional Surgery (SNIS), World Stroke Organization (WSO), Sacks, D., Baxter, B., et al., 2018. Multisociety consensus quality improvement revised consensus statement for endovascular therapy of acute ischemic stroke. Int. J. Stroke 13 (6), 612–632. https://doi.org/10.1177/1747493018778713.

Hurst, K.R., Koetzner, C.A., Masters, P.S., 2013. Characterization of a critical interaction between the coronavirus nucleocapsid protein and nonstructural protein 3 of the viral replicase-transcriptase complex. J. Virol. 87 (16), 9159–9172.

Khailany, R.A., Safdar, M., Ozaslan, M., 2020. Genomic characterization of a novel SARS-CoV-2. Gene Rep. 19, 100682.

Kupferschmidt, K., 2020. Genome analyses help track coronavirus' moves. Science 367 (6483), 1176e1177.

Lam, T.T., 2020. Tracking the genomic footprints of SARSCoV- 2 transmission. Trends Genet. 36 (8).

Li, J., Li, Z., Cui, X., Wu, C., 2020. Bayesian phylodynamic inference on the temporal evolution and global transmission of SARS-CoV-2. J. Inf. Secur. 81 (2), 318–356. Aug.

Lokmana, S.M., Rasheduzzaman, Salauddin, A., Barua, R., Tanzina, A.Y., Rumi, M.H., Hossain, I., Siddiki, Z., Mannan, A., Hasan, M., 2020. Exploring the genomic and proteomic variations of SARS-CoV-2 spike glycoprotein: a computational biology approach. Infect. Genet. Evol. 84, 104389.

Mercatelli, D., Giorgi, F., 2020. Geographic and genomic distribution of SARS-CoV-2 mutations. Front. Microbiol. 11, 1800. https://doi.org/10.3389/fmicb.2020.01800.

Naqvi, A.A.T., Fatima, K., Mohammad, T., Fatima, U., Singh, I.K., Singh, A., Atif, S.M., Hariprasad, G., Hasan, G.M., Hassan, I., 2020. Insights into SARS-CoV-2 genome, structure, evolution, pathogenesis and therapies: structural genomics approach. Biochim. Biophys. Acta (BBA) - Mol. Basis Dis. 1866, 165878.

Raza, H., Wahid, B., Rubib, G., Gulzar, A., 2020. Molecular epidemiology of SARS-CoV-2 in Faisalabad, Pakistan: a real world clinical experience. Infect. Genet. Evol. 84, 104374.

Sharp, K., Dange, S., 2020 May. Comparative analysis of SARS-COV2 with special emphasis of the genome sequences. J. Pure Appl. Microbiol. 14 (suppl1), 1053–1064. https://doi.org/10.22207/JPAM.14.SPL1.44.

Tian, X., Li, C., Huang, A., et al., 2020. Potent binding of 2019 novel coronavirus spike protein by a SARS coronavirus-specific human monoclonal antibody. Emerg. Microbes Infect. 9 (1), 382–385.

Uddin, M., Mustafa, F., Rizvi, T.A., Loney, T., Al Suwaidi, H., Al-Marzouqi, A.H.H., Eldin, A.K., Alsabeeha, N., Adrian, T.E., Stefanini, C., Nowotny, N., Alsheikh-Ali, A., Senok, A.C., 2020. SARS-CoV-2/COVID-19: viral genomics, epidemiology, vaccines, and therapeutic interventions. Viruses 12, 526. https://doi.org/10.3390/v12050526.

Wan, Y., Shang, J., Graham, R., Baric, R.S., Li, F., 2020. Receptor recognition by the novel coronavirus from Wuhan: an analysis based on decade-long structural studies of SARS coronavirus. J. Virol. 9, 4. https://doi.org/10.1128/JVI.00127-20 (e00127-20).

Wang, C., Liu, Z., Chen, Z., Huang, X., Xu, M., He, T., Zhang, Z., 2020. The establishment of reference sequence for SARS-CoV-2 and variation analysis. J. Med. Virol. https://doi.org/10.1002/jmv.25762.

World health organization. https://www.who.int/news-room/detail/29-06-2020-covidtimeline. (Accessed 30 July 2020).

worldomater. https://www.worldometers.info/coronavirus/. (Accessed 30 July 2020).

Yang, X., Dong, N., Chan, E., Chen, S., 2020. Genetic cluster analysis of SARS-CoV-2 and the identification of those responsible for the major outbreaks in various countries. Emerg. Microbes Infect. 9, 1287–1299.

Yu, W., Tang, G., Zhang, L., Corlett, R., 2020. Decoding the evolution and transmissions of the novel pneumonia coronavirus (SARSCoV-2/HCoV-19) using whole genomic data. Zool. Res. 41 (3), 247e257.