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CORRECTION
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Corrigendum: MSH3 Homology and Potential Recombination Link to SARS-CoV-2 Furin Cleavage Site

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In the original article, there was an error. The words 'SARS.Cov2' and 'furing' should be 'SARS-CoV-2' and 'furin'.

A correction has been made to **Abstract**:

"Among numerous point mutation differences between the SARS-CoV-2 and the bat RaTG13 coronavirus, only the 12-nucleotide furin cleavage site (FCS) exceeds 3 nucleotides. A BLAST search revealed that a 19 nucleotide portion of the SARS-CoV-2 genome encompassing the furin cleavage site is a 100% complementary match to a codon-optimized proprietary sequence that is the reverse complement of the human mutS homolog (MSH3). The reverse complement sequence present in SARS-CoV-2 may occur randomly but other possibilities must be considered. Recombination in an intermediate host is an unlikely explanation. Single stranded RNA viruses such as SARS-CoV-2 utilize negative strand RNA templates in infected cells, which might lead through copy choice recombination with a negative sense SARS-CoV-2 RNA to the integration of the MSH3 negative strand, including the FCS, into the viral genome. In any case, the presence of the 19-nucleotide long RNA sequence including the FCS with 100% identity to the reverse complement of the MSH3 mRNA is highly unusual and requires further investigations".

In the original article, there was an error. The words "infectivity of SARS Co-V-2 in 2019" should be" infectivity of SARS-CoV in 2009" and the reference (8) should be (5).

A correction has been made to **Introduction**:

"Based on a recent publication describing insertion variants of SARS-CoV-2 (1) we would like to bring the attention to our recent findings related to the sequence of the furin cleavage site (FCS) in SARS-CoV-2 Spike (S) protein. The SARS-CoV-2 causing the COVID-19 pandemic (2) has 82.3% amino acid identity to bat coronavirus SL-CoVZC45, 77.2% amino acid identity to SARS-CoV, and 96.2% genome sequence identity to bat coronavirus RaTG13. While numerous point mutation

differences exist between SARS-CoV-2 and RaTG13, only one insertion and dissimilarity exceeding 3 nucleotides (nt): a 12-nucleotide insertion coding for four amino acids (aa 681-684, PRRA) in the SARS-CoV-2 S protein has been discovered. This polybasic FCS differentiates SARS-CoV-2 from other b-lineage betacoronaviruses or any other sarbecovirus (3). An FCS addition enhanced the infectivity of SARS-CoV in 2009 (4). The absence of this FCS results in attenuated SARS-CoV-2 variants useful for animal vaccination, accentuating its relevance to human infection (5). This FCS is vital for human and ferret transmission (6), expands viral tropism to human cells (7), and is requisite for severe disease in two animal models of SARS-CoV-2 (5)".

In the original article, there was an error. The reference (9) should be (9, 10) and the word "eukaryotic" should be "mammalian".

A correction has been made to SARS-CoV-2 Spike Protein and MSH3, paragraph four: "The proprietary sequence SEQ ID11652, read in the forward direction, encodes a 100% amino acid match to the human mut S homolog 3 (MSH3) (9, 10). MSH3 is a DNA mismatch repair protein (part of the MutS beta complex) (11). SEQ ID11652 is transcribed to a MSH3 mRNA that appears to be codon optimized for humans (12). We did not find the 19-nucleotide sequence CTCCTCGGCGGGCACGTAG in any mammalian or viral genomes except SARS-CoV-2 with 100% coverage and identity in the BLAST database (Supplementary Tables 1–3)".

In the original article, there was an error. The references (15, 16) should be (14).

A correction has been made to Discussion, paragraph two:

"Overexpression of MSH3 is known to interfere with mismatch repair (MSH2 sequestration from the MutS alpha complex comprising MSH2 and MSH6 results in MSH6 degradation and MutS alpha depletion) (13), which holds virologic importance. Induction of DNA mismatch repair deficiency results inpermissiveness of influenza A virus (IAV) infection of human respiratory cells and increased pathogenicity (14). Mismatch repair deficiency may extend shedding of SARS-CoV-2 (14)".

In the original article, there was an error. The word "eukaryotic" should be "mammalian".

A correction has been made to **Discussion,** paragraph three:

"The absence of CTCCTCGGCGGGCACGTAG from any mammalian or viral genome in the BLAST database makes recombination in an intermediate host an unlikely explanation for its presence in SARS-CoV-2. A human-codon-optimized mRNA encoding a protein 100% homologous to human MSH3 could, during the course of viral research, inadvertently or intentionally induce mismatch repair deficiency in a human cell line, which would increase susceptibility to SARS-like viral infection. Infection of SEQ ID11652-MSH3-transduced human cells by a SARS-like virus could enable copy choice recombination (15). Replication of SARS-CoV-2 and other single stranded RNA viruses with an RNA genome of positive polarity is initiated by the synthesis of negative strand RNA in the cytoplasm of infected cells (17) (**Figure 1**). The negative strand RNA is a template for synthesis of positive stranded RNA utilized for translation of non-structural proteins, the replication and transcription complex, or new virion capsids. Coronaviruses generate double stranded RNA at an early stage of infection through genomic replication and mRNA transcription (18)".

In the original article, there was an error. The word "discontinued" should be "discontinuous".

A correction has been made to **Discussion,** paragraph four:

"Acquisition of the reverse complement FCS sequence from an overexpressed positive sense MSH3 mRNA could occur through copy choice recombination with a negative sense SARS-CoV-2 RNA intermediate (15), involving jumping from one template to another (19) (**Figure 1**). The homology between SARS-CoV-2 and other known coronaviruses is discontinuous and most SARS-CoV-2 sequences derive from a relatively recent common ancestor with bat RaTG13. Moreover, similarity plots (SimPlots) have identified sudden changes in sequence identity between SARS-CoV-2 and RaTG13, signaling potential recombination events, which could explain the capability of SARS-CoV-2 binding to ACE2 through its RBD, which is not the case for the RaTG13 RBD (15)".

In the original article, there was an error. The reference "(4) Walls AC, Park YJ, Tortorici MA, Wall A, McGuire AT, Veesler D. Structure, Function, and Antigenicity of the SARS-CoV-2 Spike Glycoprotein. Cell. (2020) 181:281–92.e286. doi: 10.1016/j.cell.2020. 02.058" should be "(4) Belouzard S, Chu V, Whittaker G. Activation of the SARS Coronavirus Spike Protein *via* Sequential Proteolytic Cleavage at 2 different Sites. *Proc Natl Acad Sci USA*. 2009; 106: 5871-6".

A correction has been made to **References**:

"(4) Belouzard S, Chu V, Whittaker G. Activation of the SARS Coronavirus Spike Protein *via* Sequential Proteolytic Cleavage at 2 different Sites. *Proc Natl Acad Sci USA*. 2009; 106: 5871-6".

The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

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Conflict of Interest: KL was employed by PanTherapeutics.

The remaining authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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