



FARGEN

Spyr korona eftir, hvør tú ert?

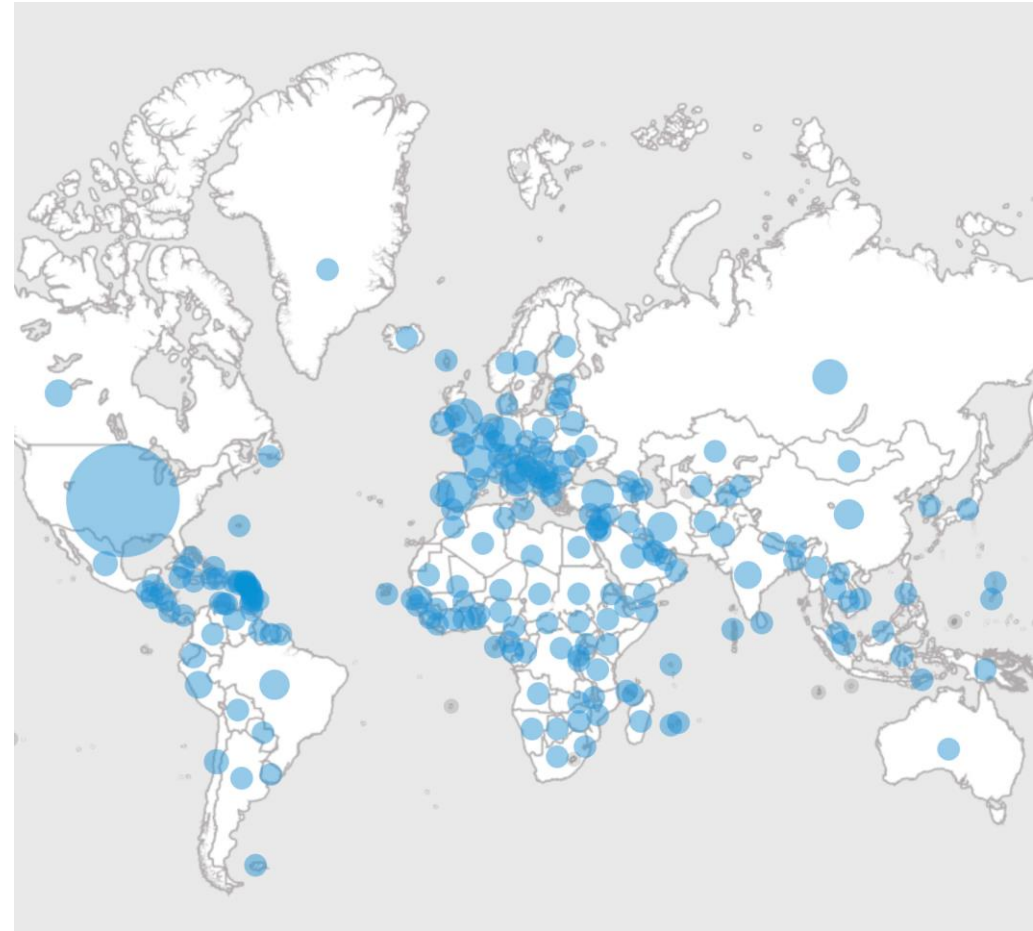
Hevur tín ílegusamanseting týdning fyri
sjúkugongdina hjá COVID-19?

Noomi Oddmarsdóttir Gregersen, PhD

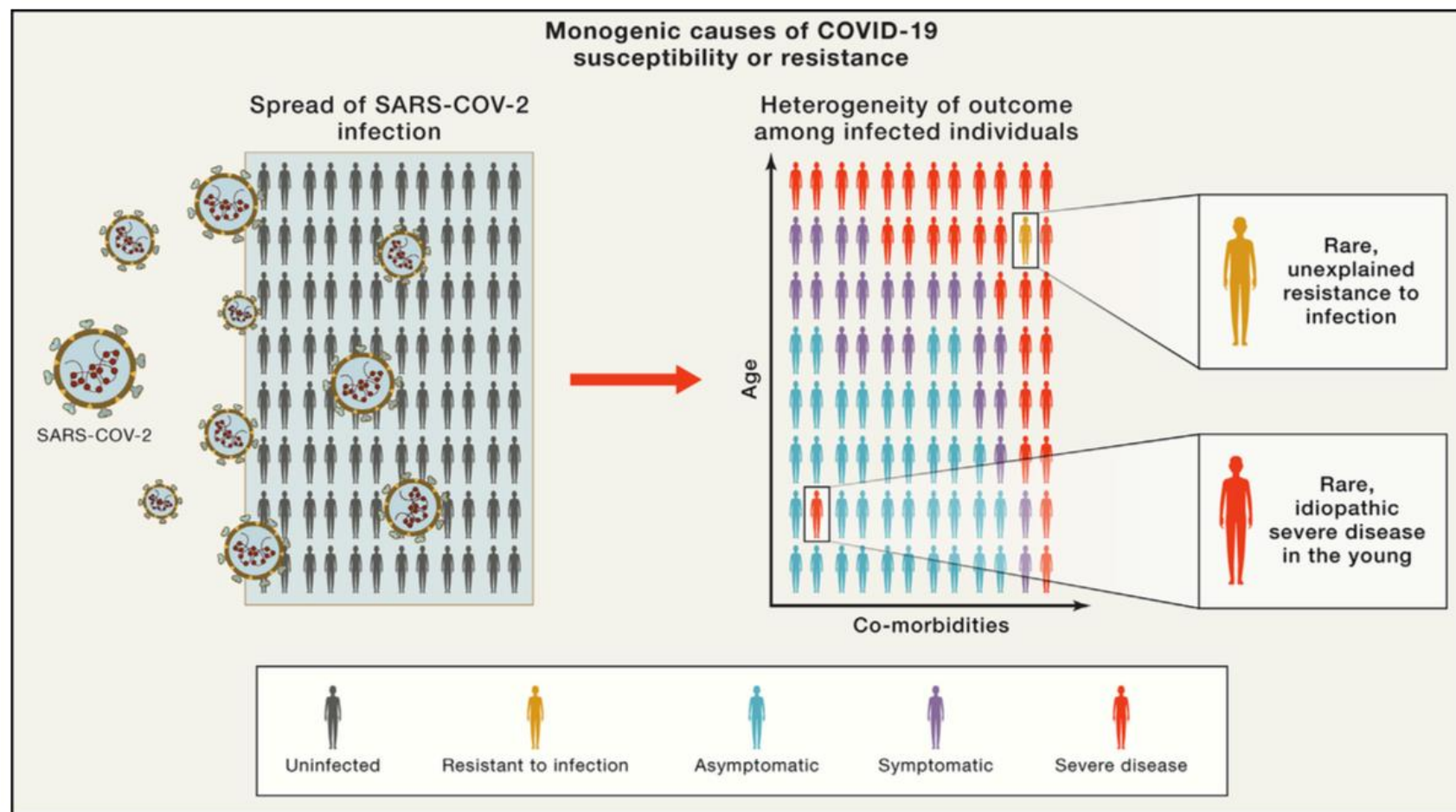
Vísindavøka 2020

COVID-19 tann 6. november 2020

- 219 tjóðir ávirkaðar...higartil
 - 47 milliónir smittað
 - 2 millónir deyð
- Ymiskt, hvussu korona ávirkar
 - Alt frá ongi sjúkueyðkenni til lívshættislig sjúkueyðkenni
- Kann ílegusamansetingin hava týdning?



Hví tosa um ílegusamanseting?



COVID-19 Host Genetics Initiative

THE NEW ENGLAND JOURNAL OF MEDICINE

ORIGINAL ARTICLE

Genomewide Association Study of Severe Covid-19 with Respiratory Failure

frontiers in immunology

BACKG: There are seven cases of Covid-19 with severe respiratory failure. We did a meta-analysis of the literature. We found that the risk of severe respiratory failure is increased in individuals with certain genetic variants. This article was published on June 17, 2020, at NEJM.org.

RESULTS: We found that the risk of severe respiratory failure is increased in individuals with certain genetic variants. This article was published on June 17, 2020, at NEJM.org.

CONCLUSIONS: We found that the risk of severe respiratory failure is increased in individuals with certain genetic variants. This article was published on June 17, 2020, at NEJM.org.

OPEN ACCESS

Editorial by Anne Placi, Institut National de la Santé et de la Recherche Médicale (INSERM), France

Reviewed by Sergio Rosenthal, National Institute of Health (NIH), United States; Carlos Rodriguez Gallego, University Hospital of Gran Canaria Dr. Negrín, Spain

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MINI REVIEW

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POLICY

The COVID-19 Host Genetics Initiative, a global effort to define the role of host genetic factors in susceptibility to the SARS-CoV-2 virus pandemic

The COVID-19 Host Genetics Initiative^{1,2}

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Introduction

The COVID-19 pandemic is a global crisis causing severe disruptions across the economy and health system. Insights into how to better understand and treat COVID-19 are desperately needed.

Early studies have focused on the clinical characteristics [1–3], epidemiology [1, 4, 5], and genomic characterization [6–8] of SARS-CoV-2 infection. These studies have also highlighted the value and importance of transparent data sharing across countries, which have enabled the life tracking of the disease widespread worldwide [9, 10]. The role of host genetics in impacting susceptibility and severity of COVID-19 has been less studied. Previous work has supported the role of human leukocyte antigen (HLA) in susceptibility [11] and severity [12] for several viral infections. Moreover, a synonymous variant in the IFN-induced transmembrane protein-3 gene has been reported to cause severe clinical outcomes in patients infected with H7N9 and H1N1 influenza viruses [13, 14], although results did not reach established P value thresholds ($P < 5 \times 10^{-8}$). In addition, candidate variant studies have suggested host factors that are critical for severe disease in other coronavirus infections, such as infections due to the related SARS-CoV [15].

Given the importance and urgency of exploring the role of the host genome in conjunction with COVID-19 clinical and genomic variability, and the recognition that this can only be achieved with the combined effort of the scientific community, we launched the “COVID-19 Host Genetics Initiative”. This initiative brings together the human

Summary

The SARS-CoV-2 pandemic raises many scientific and clinical questions. These include how host genetic factors and pathogenesis. New work is emerging related to SARS-CoV-2: previous work has been concerned with different aspects. We reviewed the literature on host genetic factors related to coronavirus studies. We identified 1,832 articles of potential relevance. Seventy-five involved human host genetic studies of specific genes or loci aside from one meta-analysis, all were candidate-driven studies, typical research subjects and loci. Three additional case reports were described. Multiple significant loci were to susceptibility (seven of which identified protective alleles) and 16 related to outcomes (three of which). The types of cases and controls used varied considerably: four studies used traditional registry studies, 10 involved both human and non-human host genetic factors related to coronavirus, (genetic) host genetic factors related to coronavirus, and 194 involved study of non-genetic host factors involving immunopathogenesis. Previous human studies have been limited by issues that may be numbers of eligible participants and limited availability of advanced genomic methods; however, in addition, we outline key genes and loci from animal and human host genetic studies that may be COVID-19. We also discuss how previous studies may direct current lines of inquiry.

Introduction

In late December 2019, a reported across several (WHO) declared the current pandemic in immunology (HCoV) 229E, NL63, OC-43, HKU-1, and NL63. The 2019/20 outbreak of coronavirus disease (COVID-19) has threatened to have the capability to cause a global pandemic. The 2019/20 outbreak of coronavirus disease (COVID-19) has threatened to have the capability to cause a global pandemic. The 2019/20 outbreak of coronavirus disease (COVID-19) has threatened to have the capability to cause a global pandemic.

Keywords: SARS-coronavirus immunodeficiency, whole exome sequencing, COVID-19

Editorial by Anne Placi, Institut National de la Santé et de la Recherche Médicale (INSERM), France

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Human Genomics

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Commentary

A Global Effort to Define the Human Genetics of Protective Immunity to SARS-CoV-2 Infection

Jean-Laurent Casanova^{1,2,3,4,5,6,*}, Helen C. Su⁷, and the COVID Human Genetic Effort^{1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18,19,20,21,22,23,24,25,26,27,28,29,30,31,32,33,34,35,36,37,38,39,40,41,42,43,44,45,46,47,48,49,50,51,52,53,54,55,56,57,58,59,60,61,62,63,64,65,66,67,68,69,70,71,72,73,74,75,76,77,78,79,80,81,82,83,84,85,86,87,88,89,90,91,92,93,94,95,96,97,98,99,100}

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Abstract

The COVID-19 pandemic has strengthened the interest in the biological mechanisms interplay between infectious agents and the human host. The spectrum of phenotypic COVID-2 infection, ranging from the absence of symptoms to severe systemic complications, what extent the variable response to coronavirus (CoV) is influenced by the variable host genetics. To explore the current knowledge about this question, we designed a systematic review of the scientific literature published from Jan. 2003 to June 2020, to include studies on the caused by SARS-CoV-1, MERS-CoV and SARS-CoV-2 (namely SARS, MERS and COVID-19) and human genetic variants were tested as predictors of clinical phenotypes. An ad hoc protocol for the rapid review process was designed according to the PRISMA at the PROSPERO database (ID: CRD42020180860). 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Keywords: COVID-19, Coronavirus, Genomic biomarker, Human host, Genetic susceptibility, Genotype, Polymorphism

Introduction

The COVID-19 pandemic has strengthened the interest in the biological mechanisms interplay between infectious agents and the human host. The spectrum of phenotypic COVID-2 infection, ranging from the absence of symptoms to severe systemic complications, what extent the variable response to coronavirus (CoV) is influenced by the variable host genetics. To explore the current knowledge about this question, we designed a systematic review of the scientific literature published from Jan. 2003 to June 2020, to include studies on the caused by SARS-CoV-1, MERS-CoV and SARS-CoV-2 (namely SARS, MERS and COVID-19) and human genetic variants were tested as predictors of clinical phenotypes. An ad hoc protocol for the rapid review process was designed according to the PRISMA at the PROSPERO database (ID: CRD42020180860). The systematic workflow provided abstracted (28 on SARS, 1 on MERS, 3 on COVID-19) reporting data on 26 discovery of investigated candidate genes (2 as associated with COVID-19), the top-ranked genes ACE2, CLEC4E, IL10, IL1, IL6, IL12A, IL12B, IL12C, IL12D, IL12E, IL12F, IL12G, IL12H, IL12I, IL12J, IL12K, IL12L, IL12M, IL12N, IL12O, IL12P, IL12Q, IL12R, IL12S, IL12T, IL12U, IL12V, IL12W, IL12X, IL12Y, IL12Z, IL12AA, IL12AB, IL12AC, IL12AD, IL12AE, IL12AF, IL12AG, IL12AH, IL12AI, IL12AJ, IL12AK, IL12AL, IL12AM, IL12AN, IL12AO, IL12AP, IL12AQ, IL12AR, IL12AS, IL12AT, IL12AU, IL12AV, IL12AW, IL12AX, IL12AY, IL12AZ, IL12BA, IL12BB, IL12BC, IL12BD, IL12BE, IL12BF, IL12BG, IL12BH, IL12BI, IL12BJ, IL12BK, IL12BL, IL12BM, IL12BN, IL12BO, IL12BP, IL12BQ, IL12BR, IL12BS, IL12BT, IL12BU, IL12BV, IL12BW, IL12BX, IL12BY, IL12BZ, IL12CA, IL12CB, IL12CC, IL12CD, IL12CE, IL12CF, IL12CG, IL12CH, IL12CI, IL12CJ, IL12CK, IL12CL, IL12CM, IL12CN, IL12CO, IL12CP, IL12CQ, IL12CR, IL12CS, IL12CT, IL12CU, 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Keywords: COVID-19, Coronavirus, Genomic biomarker, Human host, Genetic susceptibility, Genotype, Polymorphism

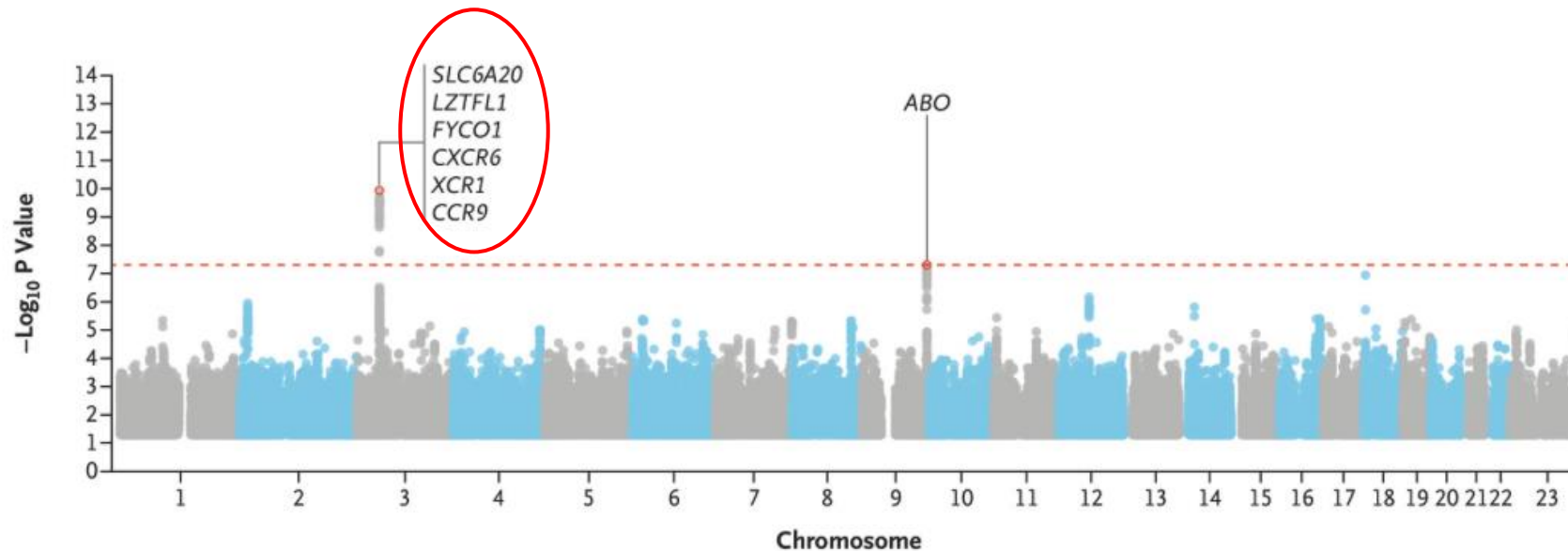
Introduction

The COVID-19 pandemic has strengthened the interest in the biological mechanisms interplay between infectious agents and the human host. The spectrum of phenotypic COVID-2 infection, ranging from the absence of symptoms to severe systemic complications, what extent the variable response to coronavirus (CoV) is



ORIGINAL ARTICLE

Genomewide Association Study of Severe Covid-19 with Respiratory Failure

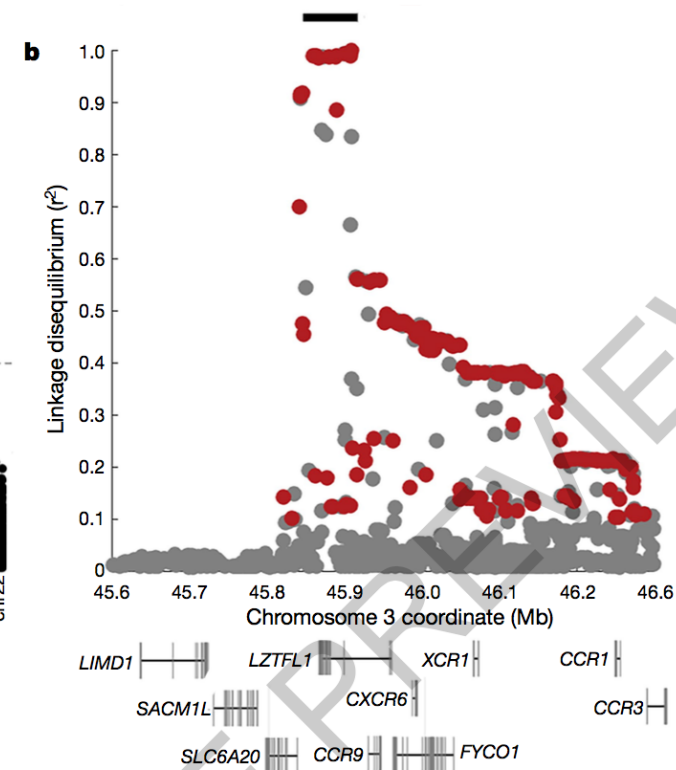
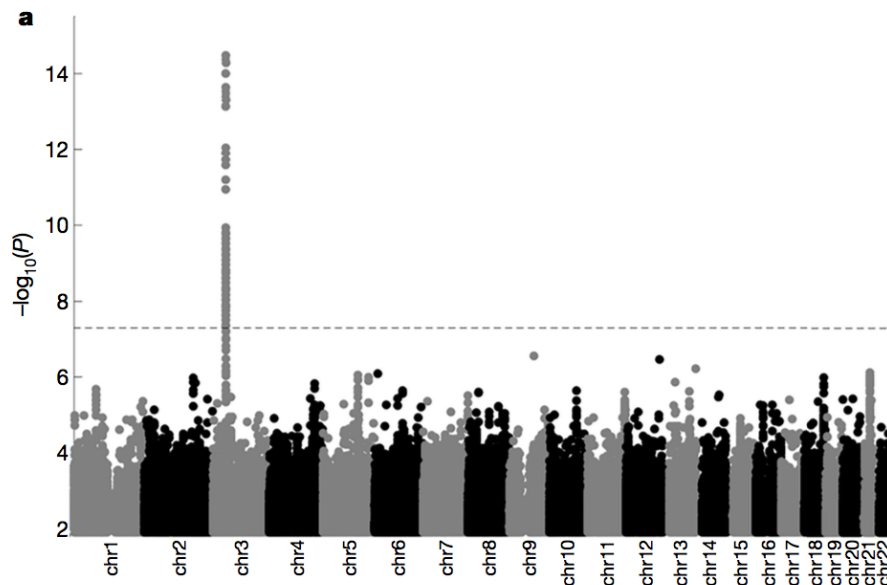
The Severe Covid-19 GWAS Group*



The major genetic risk factor for severe COVID-19 is inherited from Neanderthals

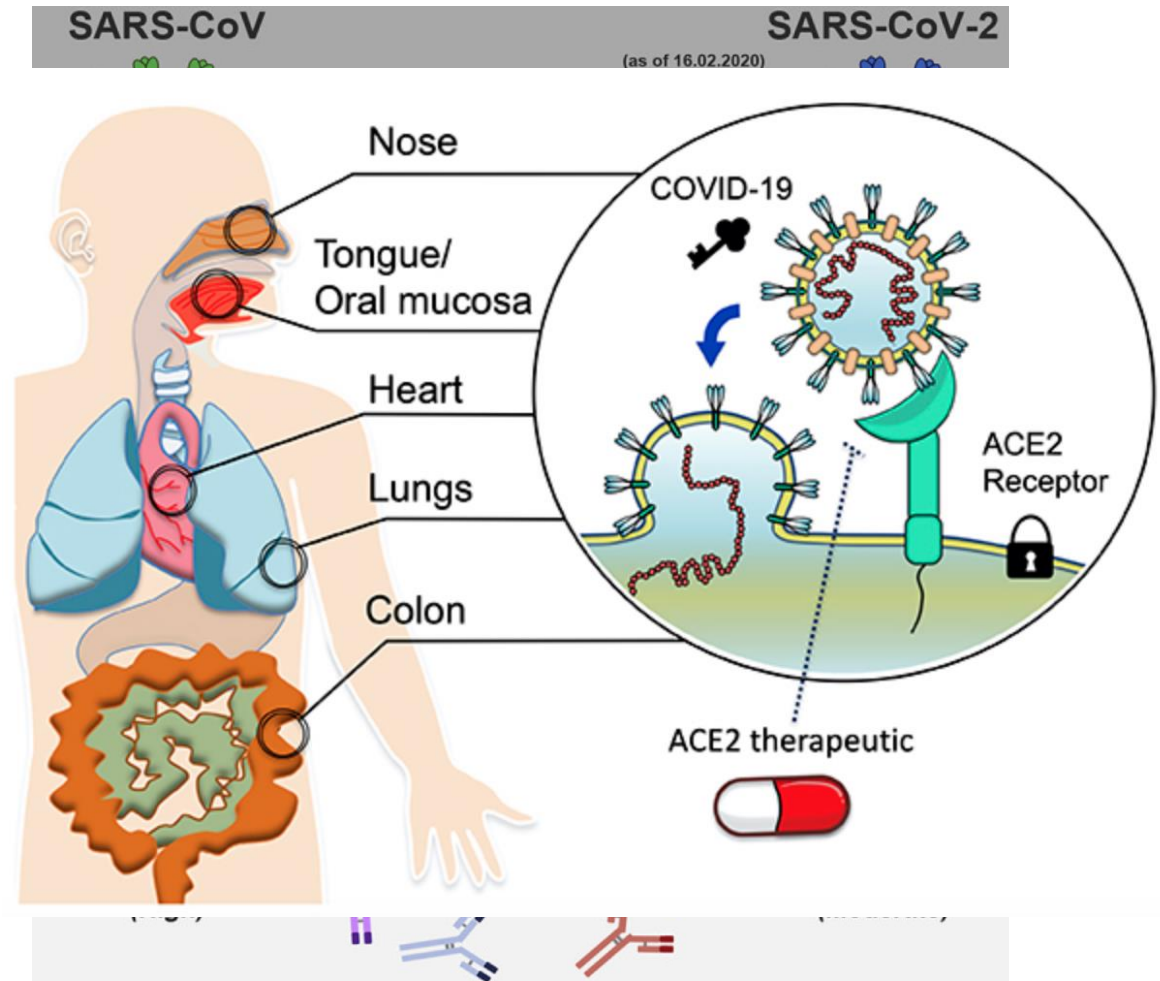
Hugo Zeberg  & Svante Pääbo 

Nature (2020) | [Cite this article](#)



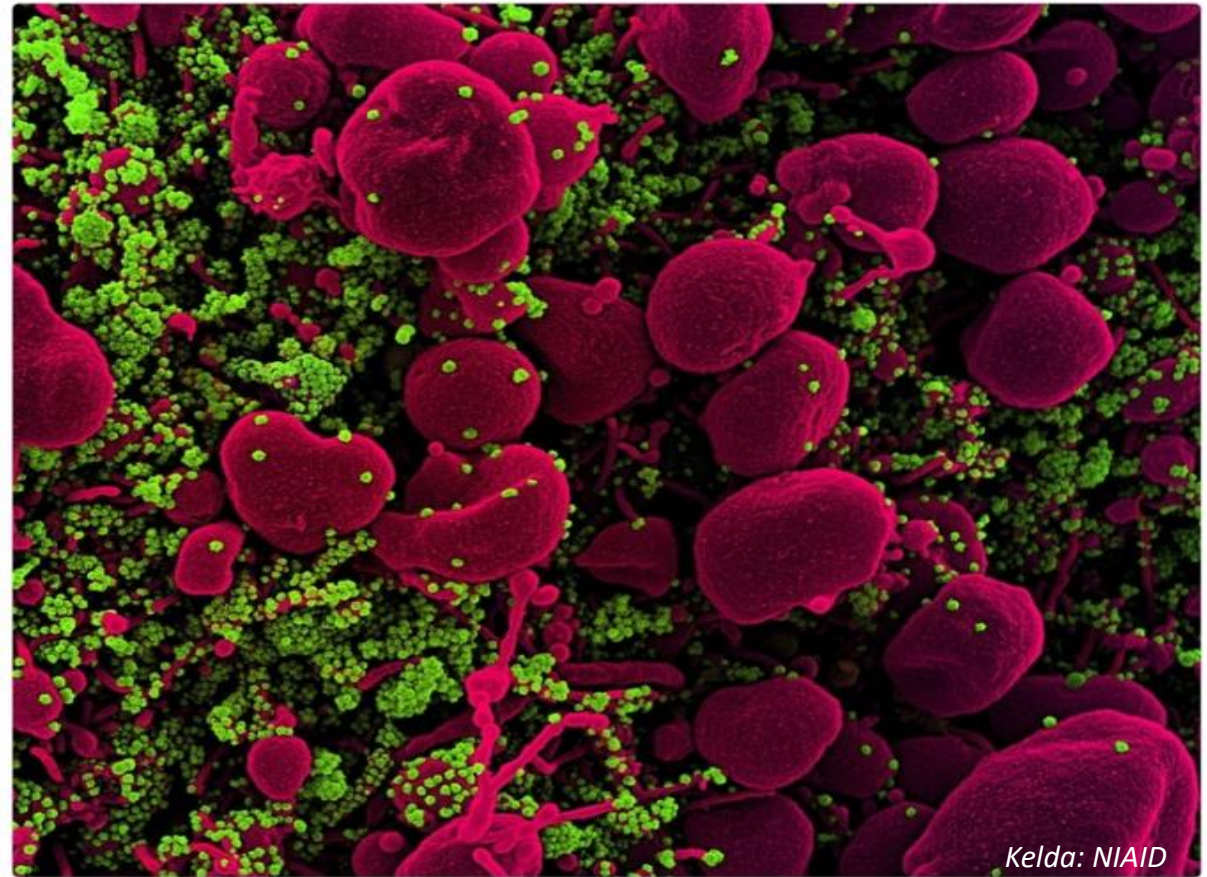
Hvat vita vit longu nú um COVID-19?

- Líkskapur millum SARS-CoV og COVID-19 (Lu et al. 2020)
- ACE2 er hurðin inn í kyknuna
- Reseptorur í yvirflatanum á ymsum kyknum
- Tal av reseptorum kann hava ávirkan á sjúkugongdina
- ACE2 er eitt møguligt target fyri eina vaccinu



Hvat vita vit longu nú um COVID-19?

- Blóðflokkur hevur ávirkan
 - Blóðflokkur A:
 - Øktur vandi fyri smittu
 - Øktur vandi fyri andaleiðstrupulleikum
 - Blóðflokkur O:
 - Minni vandi fyri smittu
 - Minni vandi fyri andaleiðstrupulleikum



Hvat gera vit í Føroyum?

- Kanna um ávísar ílegur hava samband við ymisk sjúkueyðkenni
- Kanna um ávísar ílegur kunnu verja ein ella økja um vandan fyri smittuni

Álvarslig sjúkutekin

- Miðalaldurin er 60 ár
- 14% menn og 86% kvinnur
- 43% feilaðonkið
- 57% hava høgt blóðtrýst ella astma
- 86% taka heilivág dagliga
- Ongin roykir

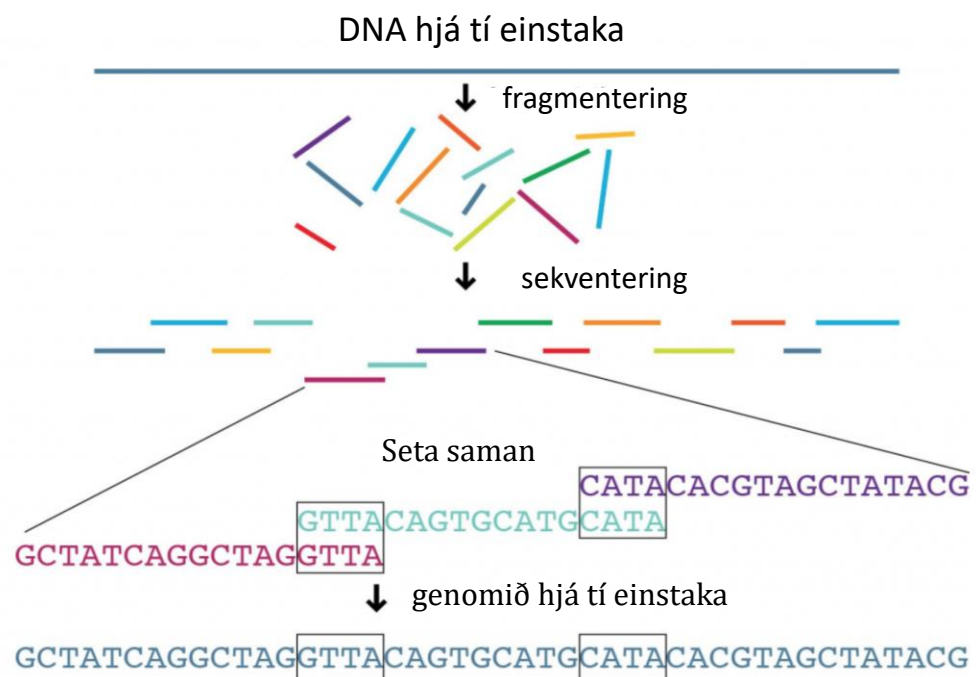
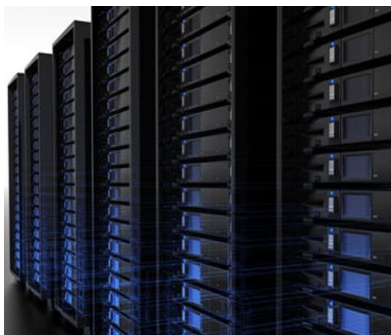
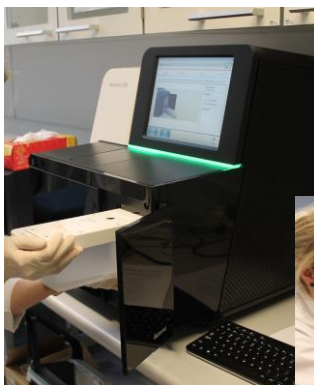
Ongi sjúkutekin

- Miðalaldurin er 43 ár
- 36% kvinnur og 64% menn
- 64% feilaðonkið
- 21% hava høgt blóðtrýst
- 29% taka heilivág dagliga
- 36% roykja dagliga

Blóðroynd til DNA

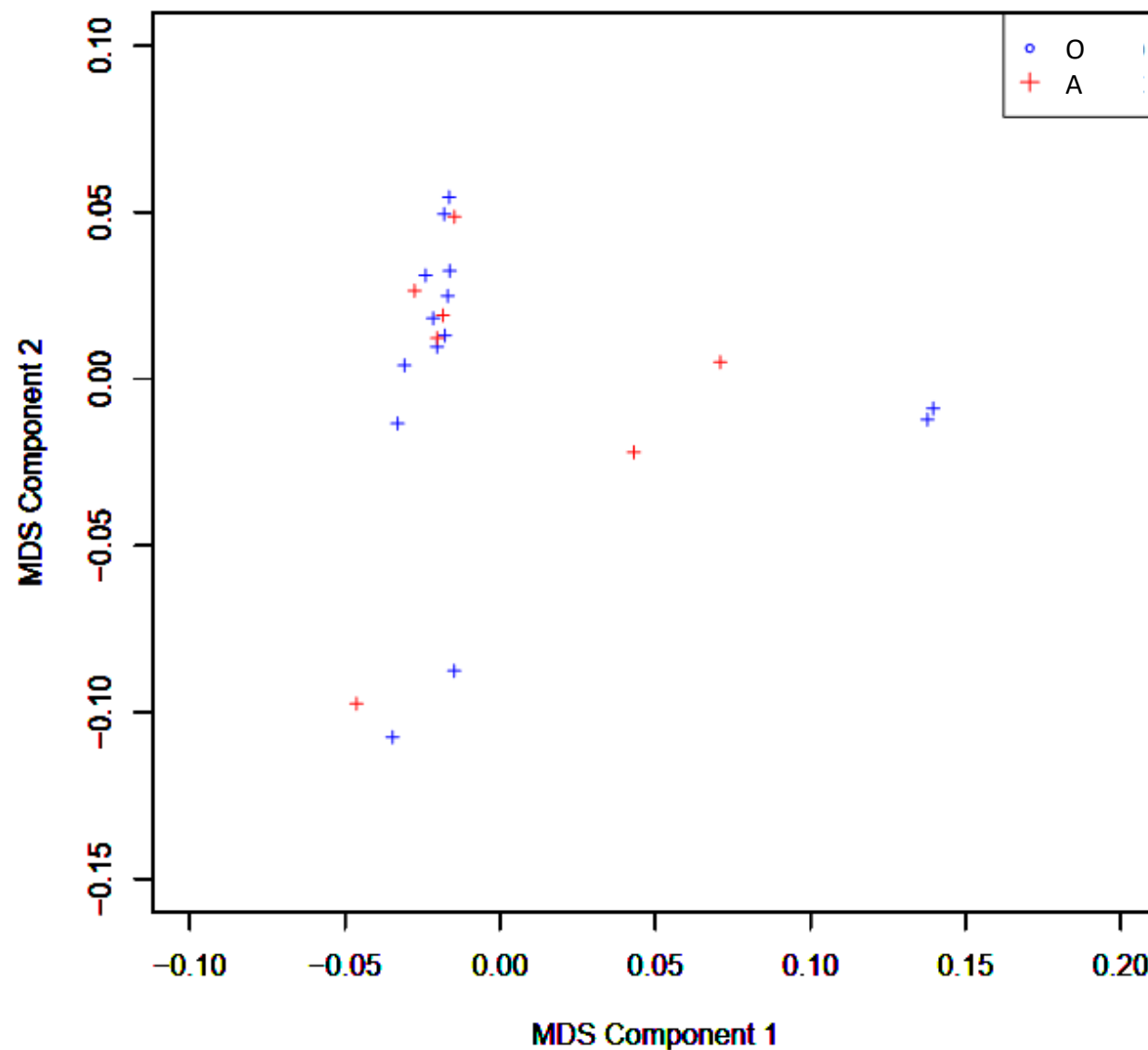


Ílegulesing



Føroyar í mun til Europa

- Føroyingar bólka seg fyri seg sjálvar
- Kann hava týðning fyri sjúkugongd og viðgerð
- COVID-19 sjúklingarnir bólka seg væl saman



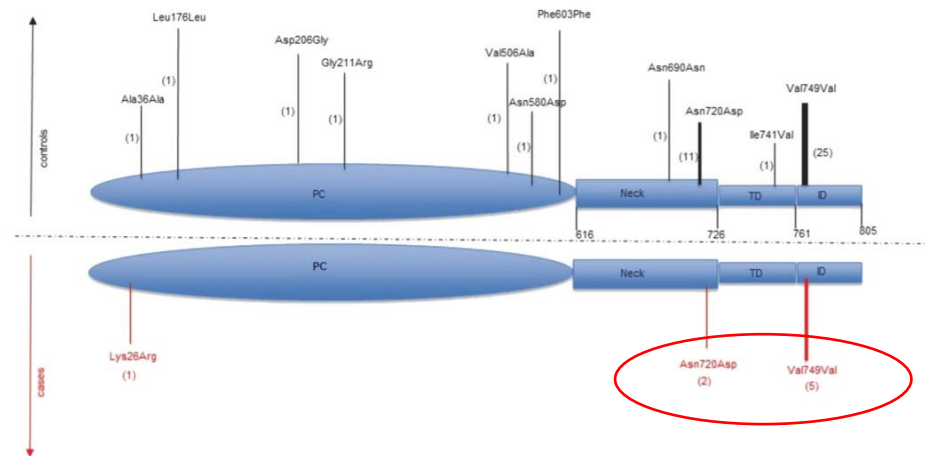


ACE2 í føroyska COVID-19-bólkinum

- Serliga trýggjar ílegubroytingar hava týðning fyri, hvussu hart COVID-19 rakar
- Tvær av teimum eru í føroysku royndunum
- Ílegubroytingarnar eru bert funnar hjá teimum við álvarsligum sjúkuteknum
- Kanning av fleiri einstaklingum fer at staðfesta, um ACE2 hevur ávirkan á sjúkugongdina ella/og smittuvandan

ACE2 gene variants may underlie interindividual variability and susceptibility to COVID-19 in the Italian population

Elisa Benetti¹ · Rossella Tita² · Ottavia Spiga³ · Andrea Cioffi⁴ · Giovanni Birollo⁵ · Alessandro Bruselles⁶ · Gabriella Doddato⁷ · Annarita Giliberti⁷ · Caterina Marconi⁸ · Francesco Musacchia⁹ · Tommaso Pippucci¹⁰ · Annalaura Torella¹¹ · Alfonso Trezza³ · Floriana Valentino⁷ · Margherita Baldassarri⁷ · Alfredo Brusco^{5,12} · Rosanna Asselta^{13,14} · Mirella Bruttini^{2,7} · Simone Furini¹ · Marco Seri^{8,10} · Vincenzo Nigro^{9,11} · Giuseppe Matullo^{5,12} · Marco Tartaglia⁴ · Francesca Mari^{2,7} · GEN-COVID Multicenter Study · Alessandra Renieri^{2,7} · Anna Maria Pinto²



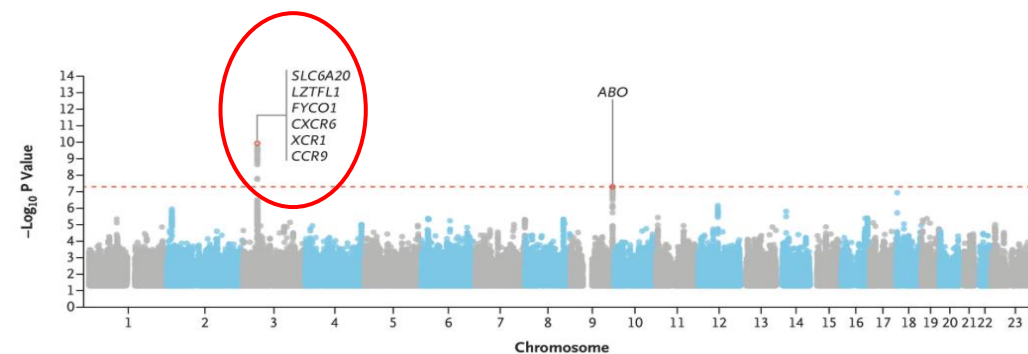
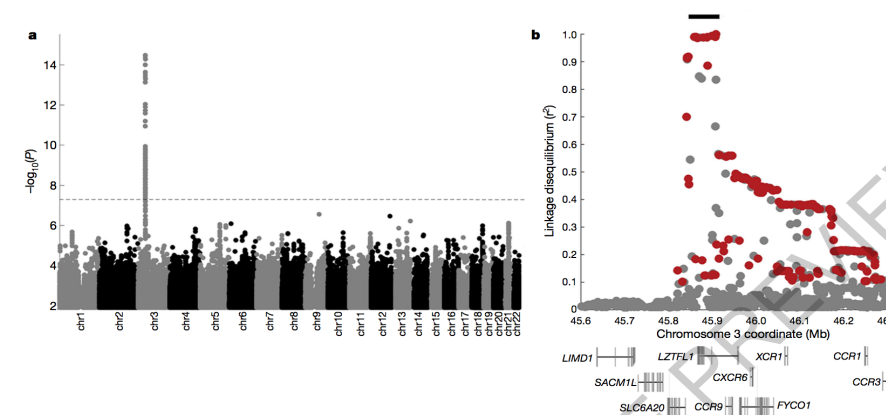
Kromosom 3 í føroyska COVID-19-bólkinum

- Kanna “vandamiklar” ílegubroytingar á kromosom 3
- Hypotesa: Tey, sum eru verri fyri, hava fleiri “vandamiklar” ílegubroytingar

Miðaltal av “vandamiklum” ílegubroytingum á kromosom 3:

	Álvarslig sjúkutekin	Ongi sjúkutekin
Ongi broyting	16	15
"Vandamikil" broyting	3	3
Partvís vandamikil	3	4
Samla	22	22

- Í miðal hava tey, sum vóru verri fyri, IKKI fleiri “vandamiklar” ílegubroytingar



ABO blóðflokkar í føroyska COVID-19-bólkinum

- Genetiskan markør fyri blóðflokk O (rs8176719)

- “Frameshift” ílegubroyting

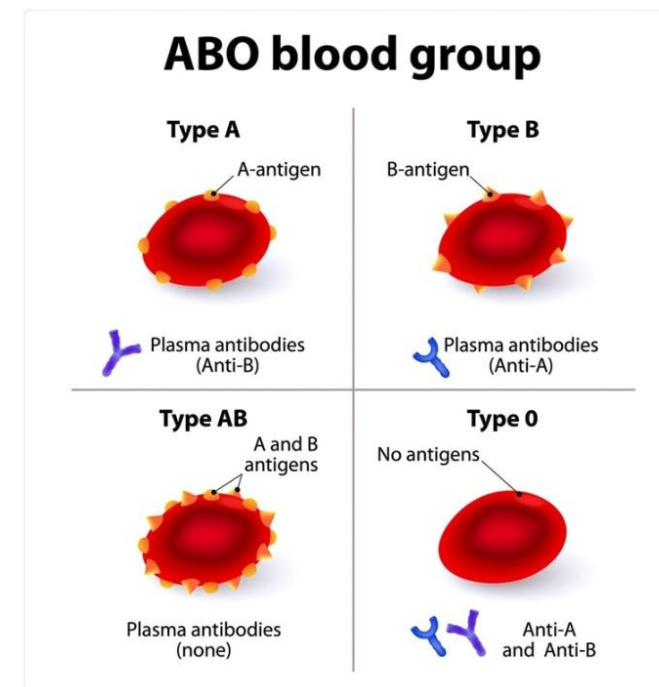
- Minst til, at blóðflokkur A:

- Øktan vanda fyri smittu
- Øktan vanda fyri andaleiðstrupuleikum

- Úrslit:

Blóðflokkur í COVID-19 bólkinum (n=21)

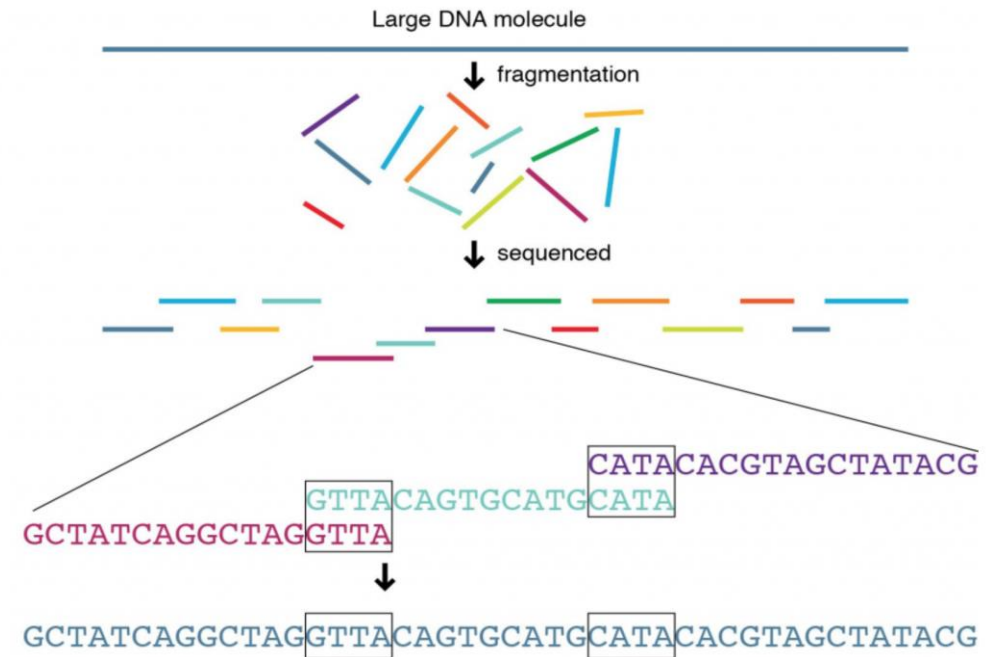
	Álvarlig sjúkuteikin	Ongi sjúkuteikin
O	5 (71%)	8 (57%)
A ella B	2 (29%)	6 (43%)



- Vit kunnu ikki staðfesta, at blóðflokkur hevur ávirkan á smittuvandan ella sjúkugongdina

Næsta stig

- Ílegulesa øll tey smittaðu, sum vilja luttaka
- Kanna ílegur saman við øðrum faktorum
 - Aldur, kyn, sjúku, andevni, royking osv.
- Vit fara nokk ikki at finna eina ílegu, sum greiðir alt
- Men íleguupplýsingar fara at hjálpa til við viðgerð
- Íleguupplýsingar fara hjálpa til við váðameting



Takk til

- Leivur N. Lydersen, Ílegusavnið, FarGen
- Ólavur Mortensen, Ílegusavnið, FarGen
- Elisabet Thomsen, Ílegusavnið, FarGen
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