

Transmission of SARS-CoV-2 variants in Switzerland

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Summary

- **The proportion of SARS-CoV-2 variants (501Y, B.1.1.7) among confirmed cases rapidly increases in Switzerland.**
- **The increase in the proportion of SARS-CoV-2 variants (501Y, B.1.1.7) in Geneva is around two weeks ahead of the rest of Switzerland.**
- **We estimate an increase in transmissibility of around 50% for the new SARS-CoV-2 variants (501Y, B.1.1.7).**
- **The findings underline the importance of efficient control measures in order to prevent an increase in SARS-CoV-2 incidence in Switzerland.**

Results

Three recently detected variants (B.1.1.7, B.1.351, P.1) of SARS-CoV-2 have been detected in various countries worldwide including Switzerland. We aim at tracking the spread of these variants in the cantons of Geneva, Bern, and Zurich, and in Switzerland overall. As a comparison, we also provide an analysis of the spread of B.1.1.7 in Denmark. Finally, we estimate the current proportion of the variants for a given region and their increased transmissibility.

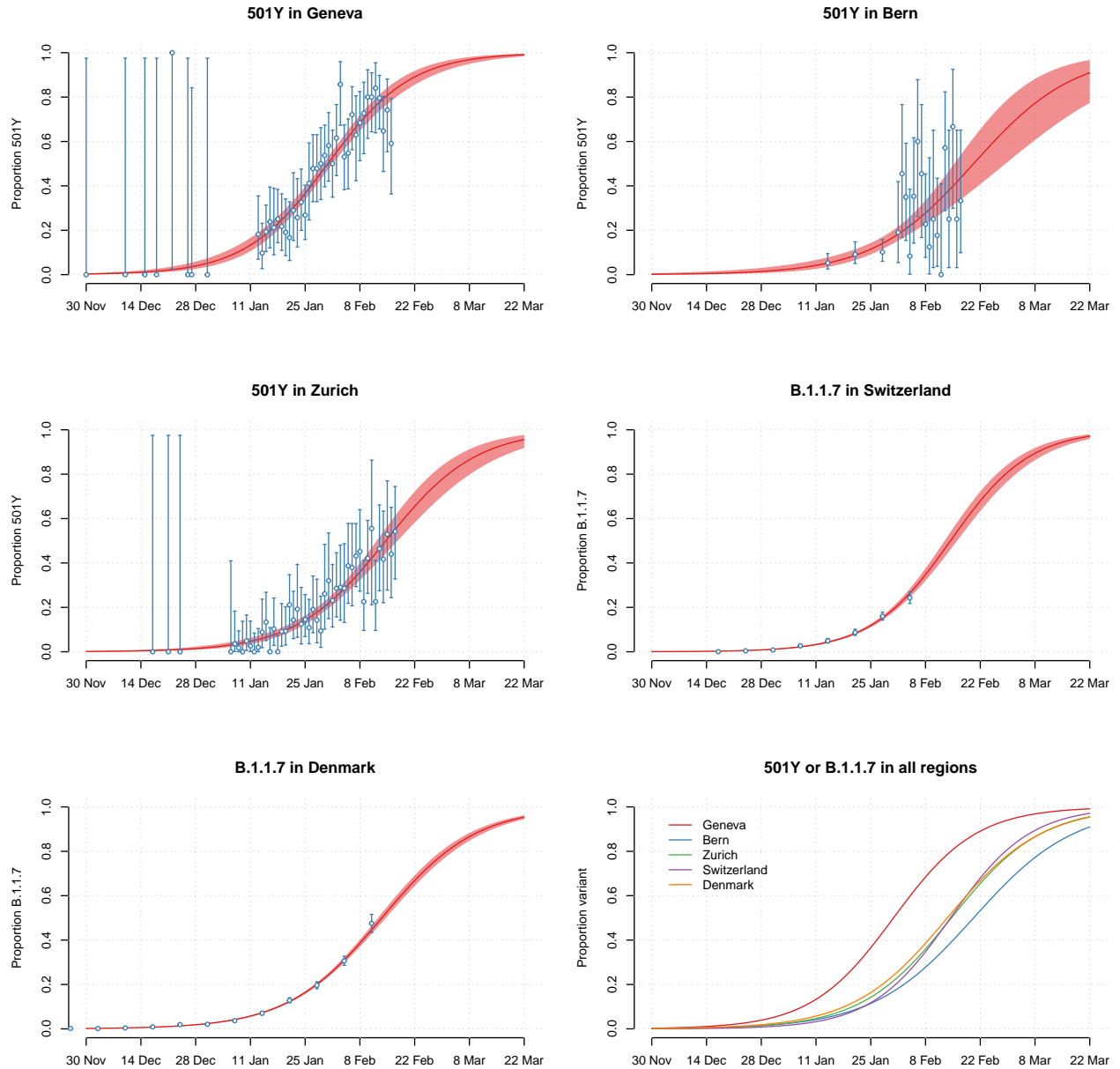


Figure 1. Increase in the proportion of SARS-CoV-2 variants among positive samples in Switzerland and Denmark. Note that the projected trajectories for Zurich and Switzerland are overlapping. Error bars and shaded areas correspond to 95% confidence intervals of the data (blue) and model (red), respectively.

The proportion of the variants rapidly increases in all regions (Figure 1). Fitting a logistic growth model to the data allows to quantify and project the increase in the proportion of the new variants (see Methods) (Figure 2, Table 1). Note that due to the delay from infection to sample collection, the shown increase and the estimated proportions of the new variants reflect the epidemic situation from around one week ago.

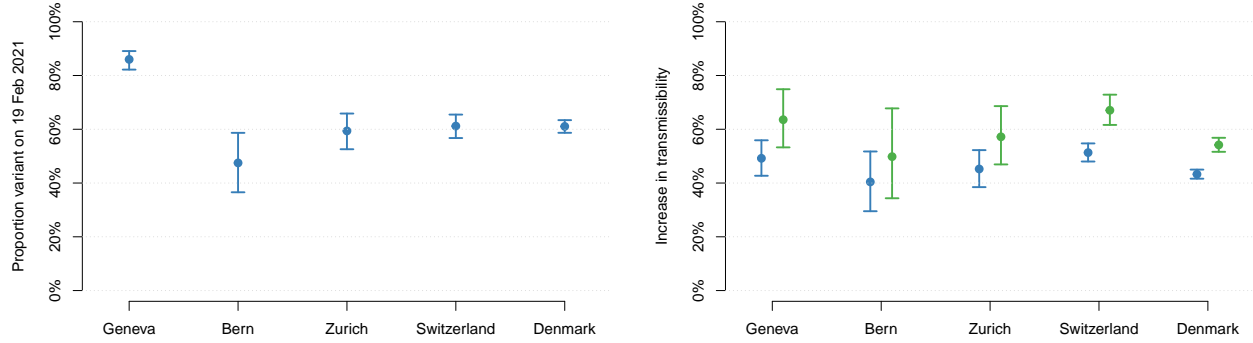


Figure 2. Estimated proportion of the variant (left) and increase in transmissibility (right). The increase in transmissibility is estimated assuming the effective reproduction number of the wild-type $R_w \approx 1$ and an exponentially (blue) and delta (green) distributed generation time of 5.2 days (Ganyani et al.). Error bars correspond to 95% confidence intervals.

Table 1. Estimated proportion of the variant and increase in transmissibility. The increase in transmissibility is estimated assuming the effective reproduction number of the wild-type $R_w \approx 1$ and an exponentially (blue) and delta (green) distributed generation time of 5.2 days (Ganyani et al.).

Region	Variant	Proportion at 19 Feb 2021	Increase in transmissibility
Geneva	501Y	86% (82%-89%)	49% (43%-56%) - 64% (53%-75%)
Bern	501Y	47% (37%-59%)	40% (30%-52%) - 50% (34%-68%)
Zurich	501Y	59% (53%-66%)	45% (38%-52%) - 57% (47%-69%)
Switzerland	B.1.1.7	61% (57%-65%)	51% (48%-55%) - 67% (62%-73%)
Denmark	B.1.1.7	61% (59%-63%)	43% (42%-45%) - 54% (52%-57%)

The estimates for the increase in transmissibility are in good agreement with earlier findings for B.1.1.7 in the UK (Volz et al., Davies et al., Leung et al.). The estimate for B.1.1.7 in Denmark appears to be somewhat lower than the estimates for Switzerland. This could be due to the following reasons. First, the effective reproduction number Re was relatively low in Denmark at the end of December 2020 and in early January 2021 (see here), which would result in a slower increase in the proportion of the new variant and consequently a biased estimated of the increase in transmissibility. Second, the increase of new variants could at least partly be driven by immune escape and Denmark has arguably a lower overall infection attack rate than Switzerland. Third, data from Switzerland could be biased towards higher coverage of new variants due to current outbreak investigations. In contrast, Denmark sequences a considerable proportion of all confirmed SARS-CoV-2 cases and their sample might thus be more representative.

Taken together, these findings underline the importance of efficient control measures in order to prevent an increase in SARS-CoV-2 incidence in Switzerland.

Methods

Data

We use data sets from the following five regions for our analysis (download here):

- **Geneva, Switzerland:** We use samples that were sent to the Geneva University Hospitals for primary diagnosis of SARS-CoV-2. All positives were re-screened for 501Y using RT-PCR (mostly B.1.1.7). To cover the period of November and December 2020, we use sequence data from randomly chosen samples from Geneva that were submitted to GISAID by the Swiss Viollier Sequencing Consortium from ETH Zurich.
- **Bern, Switzerland:** We use samples from SARS-CoV-2-positive cases that were re-screened for 501Y using RT-PCR at the Institute for Infectious Diseases, University of Bern.

- **Zurich, Switzerland:** We use samples from SARS-CoV-2-positive cases from the University Hospital Zurich and test centers at Limmattal Hospital in Schlieren (ZH) and Spital Männedorf that were re-screened for 501Y using RT-PCR at the Institute of Medical Virology, University of Zurich. In addition, we use SARS-CoV-2-positive samples from Kantonsspital Winterthur and its walk-in test center that were re-screened for 501Y using RT-PCR.
- **Switzerland:** We use data on the frequency of B.1.1.7 among randomly chosen SARS-CoV-2 samples that underwent genetic characterization and are provided by the Swiss Viollier Sequencing Consortium from ETH Zurich and the Swiss National COVID-19 Science Task Force.
- **Denmark:** We use data on the frequency of B.1.1.7 among sequenced genomes from the Danish Covid-19 Genome Consortium.

Model

Competitive growth between variant and non-variant ('wild-type') strains of SARS-CoV-2 can be described by the following two ordinary differential equations:

$$\frac{dW}{dt} = \beta SW - \gamma W, \quad (1)$$

$$\frac{dV}{dt} = \beta(1 + \tau)SV - \gamma V, \quad (2)$$

where W and V are individuals infected with wild-type and variant, respectively, and S the population of susceptibles. For the variant, the transmission rate β is increased by the factor $1 + \tau$ and both strains are cleared at rate γ . For $\tau > 0$, one can easily show that the proportion of the new variant among all infections increases according to logistic growth:

$$p(t) = \frac{V(t)}{W(t) + V(t)} = \frac{1}{1 + \kappa e^{-\rho t}}, \quad (3)$$

where $\kappa = W(0)/V(0)$ and $\rho = \beta\tau S$, i.e., the difference in the net growth rates between the variant and the wild-type. ρ can be directly estimated by fitting a logistic growth model (binomial regression) to the proportion of the new variant. The increased transmissibility of the variant is then given by $\tau = \rho/(\beta S)$. Since the effective reproduction number of the wild-type is $R_w = \beta S/\gamma$ and the generation time is $D = 1/\gamma$, we obtain $\tau = \rho D/R_w$. For a situation where $R_w \approx 1$, we can simplify to $\tau \approx \rho D$. This derivation assumes an exponentially distributed generation time. For a delta distributed generation time, one finds that $\tau \approx e^{\rho D} - 1$.

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