# Dyke wardens or Drivers?

# Why children may play an attenuating role in the spread of SARS-CoV-2

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Abstract

The role of children in the current SARS-CoV-2 pandemic remains unclear. It has been inferred from other viral epidemics that children may play a driving role in the transmission chain. However, the epidemiological record has challenged this assumption as children only infrequently have been identifed as active infectors. At the same time the SARS-CoV-2 pandemic seems to proceed at a surprisingly slow pace in populations with a high proportion of children. In this review we explore the role of children from a multipronged theoretical framework based on the current epidemiological as well as basic scientific record. In particular, we follow up on suggestions that children may have a moderating rather than aggravating impact on the SARS-CoV-2 epidemic and ask if this may in part be based on age dependent effects.

Keywords: SARS-CoV-2, epidemic, transmission, children, viral load, school closure, COVID 19

# Introduction

One of the many unresolved questions in the current SARS-CoV-2 pandemic revolves around children: what role do they play in the transmission chain? The scientific evidence base is controversial.(1) On the one hand SARS-CoV-2 positive children may carry equal(2)(3) or even higher(4) viral loads in their upper airways compared to SARS-CoV-2 positive adults suggesting similar infective potential. On the other hand the epidemiological record is markedly thin when it comes to active transmission by children. Nearly seven months into the pandemic and with the worldwide case load exceeding 16 million relatively few documentations of active transmission by children have accrued.(5) Thus, the concern viewing children in general as potential drivers of the epidemic has yet to be substantiated with real world data.

At the same time there is emerging evidence from analyses of school outbreaks, contact tracing and seroprevalence studies that children may have to be viewed as a disparate entity in terms of their epidemiological role, with younger children being less frequently identified as both infectors and infectees than older children or youths.(6)(7)(8)(9)

Recently, another unresolved issue in the SARS-CoV-2 pandemic has given rise to reassessing the role of children in the epidemic spread as the dissemination of COVID-19 has followed a different trajectory than originally predicted. In many developing countries the epidemic seems to proceed at a slower pace and to be clinically less severe than in the initial hotbeds in China, Europe and the US. For some, this has again brought the children into focus as a possible explanation for the unexpected dynamic.(10)

Here, we systematically explore the role of children from a multipronged framework based on the current epidemiological as well as basic scientific record, asking: Could children be dampening rather than driving the SARS-CoV-2 epidemic?

To explore this hypothesis we reassess the known facts about transmission of SARS-CoV-2 and discuss if some of these factors may result in a moderating impact of children, both on the speed of SARS-CoV-2 transmission and the clinical severity of the disease in those infected.

**Exhibit 1: SARS-CoV-2 transmits more easily from symptomatic subjects**

It is uncontested that contact with a symptomatic, i.e. clinically affected individual carries the highest risk of becoming infected with SARS-CoV-2.(11) Given the lesser likelihood of children to present with clinical disease, the risk for a given person to pick up SARS-CoV-2 from a child should be considerably smaller compared to becoming infected from an adult (assuming, of course, that a person interacts equally with children and with adults).(12)(13)(14) It may be argued that the relatively low prevalence of COVID-19 in children may be an early wave phenomenon with children catching up as the population spread of SARS-CoV-2 progresses. However, as of now, the lag in prevalence of symptomatic presentations - i.e. recognized COVID-19 disease - between children and adults persists.(15)

*Exhibit 1 therefore lends credibility to the assumption that transmission events are less likely to be caused by children.*

**Exhibit 2: SARS-CoV-2 transmits less easily from truly asymptomatic subjects**

There is substantial debate about the prevalence and infectious potential of truly asymptomatic subjects, i.e. subjects that do not develop symptoms throughout the course of their infection. Estimates for adults in high quality studies(16)(17)(18)(19) and two meta-analyses(20)(21) are reported around 15 percent, with a range from 4 to 33 percent. Less data are available on children. Several studies suggest that truly asymptomatic courses are age

dependent, with a higher prevalence in younger adults(16)(22) and especially children.(23)(24)(21) In well documented contact studies both in households and community settings truly asymptomatic cases seem to be about half as common in children than in adults.(25)(26)

To which extent asymptomatic subjects are able to infect others is still under investigation. While it has been shown that truly asymptomatic individuals may carry viral loads comparable to those symptomatically infected,(27)(28)(29) there is an emerging consensus that transmission rates in truly asymptomatic cases are considerably lower than in pre-symptomatic or symptomatic cases,(30)(20)(21) possibly owing to the fact that viral clearance is severity dependent and faster in asymptomatic than in symptomatic individuals.(31) A recent analysis of individual level data suggests that truly asymptomatic transmission may account for only a very small fraction of transmissions, in the bracket of 3,4 to 6,6 percent, depending on assumptions.(32) Therefore, with children presumably being about twice as likely to experience truly asymptomatic courses than adults, they may well play a lesser role also in regard to asymptomatic SARS-CoV-2 transmission than adults.

*Exhibit 2 therefore also lends credibility to the assumption that transmission events are less likely to be caused by children.*

**Exhibit 3: SARS-CoV-2 transmits less easily from mildly affected subjects**

There is agreement in the literature that children who fall ill with COVID-19 are many times more likely to be only *mildly clinically affected* compared to the average sick adult. At the same time it has been shown that mildly affected individuals are less contagious at any given point in their disease course.(33) Further, mildly affected patients present with shorter infectious periods.(33) Not surprisingly, individuals with mild clinical courses of COVID-19 have been linked to reduced secondary attack rates.(34) Further, secondary cases may harbor

significantly lower viral loads than index-cases - which is relevant in respect to children as they are usually among the secondarily infected.(35) Therefore, if the formula "severe COVID-19 equals more powerful and prolonged viral spread" holds true, children in general should be rather benign players in the transmission chain as they rarely present with severe clinical courses.

*Exhibit 3 therefore also lends credibility to the assumption that transmission events are less likely to be caused by children.*

**Exhibit 4: SARS-CoV-2 may transmit less easily to children**

The potential to transmit SARS-CoV-2 clearly depends on the likelihood to become infected, to start with. To which extent the likelihood of becoming infected with SARS-CoV-2 differs between children and adults is a matter of ongoing controversy. While some studies suggest equal infectibility of children and adults,(12)(36)(37) other studies indicate a lesser likelihood of children to get infected, (29)(38)(39)(40)(24)(41)(42)(43)(44)(45) with some evidence for this being valid for the younger age group only(46)(47)(48) - a finding recently again contradicted by evidence from a large field study in a summer camp that has documented equal susceptibility between 6 to 10 year olds and older children.(49) The findings of possibly reduced susceptibility of children have been interpreted as epidemiological artifacts, reasoning that children might just not have had the same opportunities as adults to get infected during widespread preschool and school closures(2) or that children may more frequently go undetected because of their often asymptomatic presentation.(50) However, this argument conflicts with stringent household monitoring studies of index cases and their contacts which are assumed to be unbiased models for measuring differential susceptibility of children versus adults.(51) Indeed, those carefully conducted studies attribute a lower risk of infection to children living in the same

household.(52)(29) Clearly, this controversy remains to be finally settled, especially with regards to possible age-specific differences.

*Exhibit 4 - if substantiated - may also support the assumption that transmission events are less likely to emanate from children. However, this correlation clearly is contentious.*

**Exhibit 5: SARS-CoV-2 may transmit less likely from children for biophysical and immunological reasons**

Multiple lines of reasoning from the fields of immunology and biophysics may explain why children, even if infected with SARS-CoV-2, may not pass on their viral loads *as effectively as adults.*

- In SARS-CoV-2 infections, the maximum amount of viral material transmitted does not necessarily correlate with upper airway viral load. It also depends on the duration of viral shedding, which has been shown to be age dependent for SARS-CoV-2, with shorter durations seen in children.(31) The degreee of viral transmission also depends - to a large extent - on whether the lungs are also infected, leading to high viral sputum loads.(53)(54) Also, in the case of lung involvement, viral spreading occurs during a longer period of time than with only upper airway involvement.(53)(55) Considering that pneumonia seems to be much less likely in paediatric COVID-19 than in adult COVID-19,(56) children should be less likely to be among the subjects typically transmitting high viral loads.
- Children may not be part of this highly spreading sub-cohort for another, partially related, reason: transmission of SARS-CoV-2 is a matter of droplet and aerosol dissemination physics. As soon as coughing sets in, an infected person reaches a much larger number of people than if he or she does not cough. Currently available data indicate that children infected with SARS-CoV-2 are substantially less likely to

present with a cough than adults.(57) Moreover, if children do cough, the droplets may be less likely to reach adult height.

- The biophysical "disadvantage" of children may be most relevant for the transmission by aerosols, highly relevant to the spread of SARS-CoV-2 and highly variant between subjects.(58) Here, infected children may be at a considerable disadvantage both by virtue of their milder clinical presentation,(59) small stature, their small lung volumes and their inability to project their air stream with force.(60) With regards to the physiologic prerequisites of explosive articulation and generation of strong air streams children simply do not live up to adult standards - as inefficiently as they can blow out candles at their birthday party, they are physically less able to spread their viruses – except for close contact with their peers or parents.
- The latter circumstance, i.e. the propensity of children to stay within a framework of close contacts, may itself be highly relevant to the transmission question. The more regular the contacts between infectees and infectors, the more likely a possible infection will occur during the incubation period, i.e. during a period of lower viral load rather than during symptomatic stages, where viral loads are typically higher. This may explain why SARS-CoV-2 infections run a milder course if they are acquired in the household from family members(35) and why the severity and case fatality rate of COVID-19 may have decreased so drastically in recent months with transmission incidents shifting from community spread to household spread.(61)(62) As children may be the family members with the most constant and intimate contacts in a given household it may be reasonable to postulate that, if children were to transmit, the severity of the disease transmitted should be in a more favorable clinical range.
- Children may be weaker transmitters for immunological reasons. First, upper respiratory infections run rampant especially in the preschool age group, with a high

prevalence of adenoviral, metapneumoviral, coronaviral or respiratory synthitial viral infections, all of which may compete for replicative resources.(63) Also, children have a much higher innate immunological capacity to mount a strong defense even to as of yet unknown pathogens, possibly enabling the child host to contain replication and/or suppress inflammation at the level of the mucus membranes.(64) This may in part be reflected by a faster mucosal humoral immune response and IgA production which in turn might reduce transmissibility of SARS-CoV-2 in children.(65)(66)

*Exhibit 5 therefore again lends credibility to the assumption that transmission events are less likely to be caused by children.*

**Testing the exhibits**

The presented lines of evidence converge to the overall suggestion that children may be less likely to transmit SARS-CoV-2 than adults. If factually confirmed by future research the five exhibits presented would translate into a lesser ability of children to sustain the infection chain. This in return may retroact on the population spread of SARS-CoV-2 and affect both speed and clinical severity of the epidemic wave in a given country. After all, modeling SARS-CoV-2 transmission in a population with a high percentage of non-transmitters would result in a completely different epidemic curve than modeling transmission within intact, fully susceptible transmission networks.

According to these assumptions, children should then

- show low rates of child-to-other transmission
- be less likely to be index cases in household spreads
- be less likely to be index cases in school spreads
- be less likely to induce super spreading events
- be part of less severe epidemics in countries where children abound.

These five premises are indeed supported by the current epidemiological record:

- Reports of confirmed child-to-other transmission remain rare.(14)(45)(9)(67)(68)(69)(37)(22)(70) While this could reflect a lag between the "child epidemic" and the "adult epidemic"(2) , the record on child-to-other-transmission remains remarkably spotty as of now. However, more observations from high incidence settings (i.e.in the United States) or from countries that have reverted to full school operations are required to settle this question.
- Studies of household or community contacts show that children only rarely act as index cases,(71)(72)(25)(73)(74)(43)(50) with some evidence that this may be different for children 10 years and older.(6) If and how this pattern changes with the reopening of schools is being investigated.(45) This question again requires continuing observation efforts as the current low incidence situation may not offer sufficient opportunities for children to attain their infective potential.
- Several studies have investigated the role of children in suspected "school outbreaks" so far. They have usually identified adults as origins for the outbreak and have found no or very few cases of transmission by children within school settings,(26)(70)(69)(7)(45)(75) with notable exceptions in a French and in an Israeli high school, where secondary attacks in the school setting have happened to a larger extent(8)(76) (for a review, see (77). A comparative analysis of seroprevalence data in children between Finland and Sweden, countries that have applied very different school policies during the pandemic, has found no measurable impact of closing or not closing schools on the number of cases in school-aged children.(78)
- Many of the above arguments align to make children less likely to play a role as super spreaders; after all, super spreading events are more likely to be fueled by aerosol transmission and more likely to be induced by highly contagious individuals. Yet,

super spreading from children cannot be excluded as children do roam, tend to gather in groups and are unable to keep their distance. However, if one considers that high levels of contagiousness are tied to lower airway involvement, severity of disease, presence of cough and the ability to load a high number of aerosols children may well play a minor role with regards to super spreading. Indeed, to our knowledge, no child so far has been implicated as the index case of a super spreading event.

- The case fatality rate of COVID-19 in a given country seems to be inversely related to the level of development, with countries like Jordan, the West Bank and Gaza Strip and many countries in Africa and the Indian subcontinent reporting comparatively few fatalities and a surprisingly slow pace of the epidemic spread of SARS-CoV-2 despite crowded living conditions hardly amenable to social distance measures.(79) Five months into the epidemic India´s fatality rate is being reported at 23 per million inhabitants, withe some indication of seroprevalence rates up to 23 percent in some hotspots like Delhi(80) - compared to a fatality rate of 800 per million in Belgium and 700 per million in Great Britain.(81) Similar proportions are being reported from many African and other middle east developing countries like Jordan or Palestine.(82)(83)(84) While it could be argued that this current situation in developing countries arises from a relatively low proportion of old people, an additional explanation may also be plausible considering the theoretical arguments above: the high proportion of children who may play an attenuating role in the spread of COVID-19 in developing countries (proportion of children 0-14 years for Africa: 41%; for Jordan: 35%; for India: 28%; for Italy, Sweden, Belgium: around 13-14 %).

The assumption of a lesser role of children in the current COVID-19 epidemic has been critically commented, mainly with reference to three observations:

First, children have been shown to carry somewhat similar viral loads on their upper airways than adults,(2)(3) (with other studies reporting lower(35) or higher(4) viral loads for children). However, direct and indirect evidence indicates that viral load measurements may not be a good measure of overall infectivity. For one the virus material harvested from mucus membranes has been shown to be only a poor predictor of the presence of infectious virus.(85)(31) For instance, no study so far has detected replicable virus in a COVID-19 patient beyond day 9 of illness in spite of the persistence of high viral loads on the mucus membranes.(31) Moreover, comparisons of viral loads between age groups may be notoriously difficult to interpret without adjustment for the time points of sampling possibly explaining some of the contrasting results.(35) Also, as delineated above, the degree of transmission of the local flora depends on immunological and biophysical processes which may or may not be present or effective in a given subject.

Second, children are known to play a driving role in other epidemics of respiratory viruses, notably influenza.(86) However, there are notable differences in children´s role in the COVID-19 pandemic as compared to the influenza pandemics.(87)(88) In the case of influenza, children usually carry a higher viral load on their upper airways than adults, which they also shed readily and for a longer time period.(89)(90) In addition, unlike SARS-CoV-2 infections, children very often become ill during an influenza infection and then quickly start coughing and secreting mucus or sputum. Not surprisingly, in influenza, children account for more than half of the seeding into family households.(72) In contrast to influenza, children have assumed a totally different role in the spread of two other pandemic corona diseases, namely SARS and MERS. In both pandemics, children did not become ill to any significant extent, nor did they apparently contribute significantly to the transmission chain.(91)(92) In SARS, for example, only one single case of transmission from a pediatric patient has been reported.(93)

Third, a reverse causation argument for a potentially driving role of children has been derived from the putative large effects of school closures in the SARS-CoV-2 epidemic. As of yet however, the data on the efficacy of school closures as a non-pharmacological intervention in the SARS-CoV-2 pandemic are widely conflicting. While some studies considered school closures as the largest attributable effect amongst all non-pharmacological interventions(94)(95) or attributed large mitigation effects from retrospective modelling between US states(96), other studies have reported intermediate,(97) or only small to zero effects.(98)(99)(100)(24) This controversial situation may arise from the quandary that many of the non-pharmacological measures were introduced during a very short time frame and are overlaid by population composition and population behavior which are notoriously difficult to disentangle even in the most sophisticated statistical models of spreading.(101) Also it has to be kept in mind that closures of schools without transmission mitigation has to be considered a different intervention than closure of schools adjusted to the pandemic challenges. The same caution may be indicated for the retrospective modeling of the epidemiological effects of school re-openings, where results vary between no effect on the epidemic spread and possible contribution to transmission rebound, with some evidence for age-dependent effects.(102)(103)(104)

## Summary

Transmission of SARS-CoV-2 has been shown to be more likely in individuals who are sick with COVID-19, who run severe courses of the disease, who are presymptomatic and who present with cough. All of these features are cumulatively many times more common in adults than in children. Conversely, subjects have been shown to be poor spreaders of SARS-CoV-2 if they run mild cases or remain truly asymptomatic - features that are more typical for children than for adults. The epidemiological record gathered so far is in line with the

assumption of inferior epidemic potential of children, an assessment recently also made by Goldstein et al.(105) Rather than being "drivers" of the epidemic children may therefore well be "dyke wardens", i.e. play an *attenuating role* in the population spread of SARS-CoV-2. Their dampening role may have both epidemiological and clinical implications:

- Epidemiologically, children may prove to play a benign role, both by virtue of their inability to generate super spreading events and by virtue of their lesser ability to infect others. This would render the child population somewhat of an epidemiological dead end and the single child a non-functional link in the transmission chain – in stark contrast to the known role of children in the spread of other viral diseases such as influenza.
- Clinically, children could as well rather be dyke wardens than drivers: Worse clinical outcomes in infected individuals have been linked to the inhalation of high viral doses and thus certain characteristics of the infector, especially severe disease and the presence of cough - all of which are typical for adult COVID-19 but not for paediatric COVID-19. In other words, it may well be a better option for a given person to acquire SARS-CoV-2 from a child rather than from an adult.

As a caveat and occasion for further discussion there is evidence that the role of children may in part hinge on what is being considered a "child". There are clear indications both in the immunological and in the epidemiological record that younger children may differ in their biological responses to SARS-CoV-2 from older children or youths.(106) Transmissions in school settings for instance have so far been next to absent in studies of primary schools but have been clearly documented in two high schools.(8)(76) Likewise, transmission by pediatric index cases in households may be age dependent.(6) The infection rates as well as the morbidity rates also clearly follow an inversely age related pattern across the first two decades.(107) Therefore, future research may well identify a demarcation line

beyond which children may play a different game with respect to the COVID-19 epidemic. The question where and how to draw this line may be most relevant to public policy decisions including school policies.(108)

## Conclusion

The role of children in the SARS-CoV-2 epidemic remains disputed. Some scientists have alerted the public towards a potentially driving role of children in the SARS-CoV-2 epidemic, partially based on laboratory findings of equal viral loads in children and in adults.(2)(1)(109) Starting from these concerns, we have reassessed the available literature on transmission dynamics in real life. According to this review the role of children may be a favourable, if complex one. On the one hand there are suggestions that children may play an attenuating role both with respect to epidemiological and clinical dynamics. On the other hand some of these effects may be age dependent, with younger children more likely candidates for a lesser role in transmission.

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