

5.5(n)

From: clerk@hamilton.ca
To: [Kolar, Loren](#)
Subject: Fw: ATTN: Board of Health RE: Mandatory Masks
Date: July 9, 2020 8:54:47 AM
Attachments: [image.png](#)
[image.png](#)
[image.png](#)
[covid-19-daily-epi-summary-report.pdf](#)
[Rancourt-Masks-dont-work-review-science-re-COVID19-policy.pdf](#)

From: Amy Newton [REDACTED]
Sent: Wednesday, July 8, 2020 10:06 PM
To: clerk@hamilton.ca
Subject: ATTN: Board of Health RE: Mandatory Masks

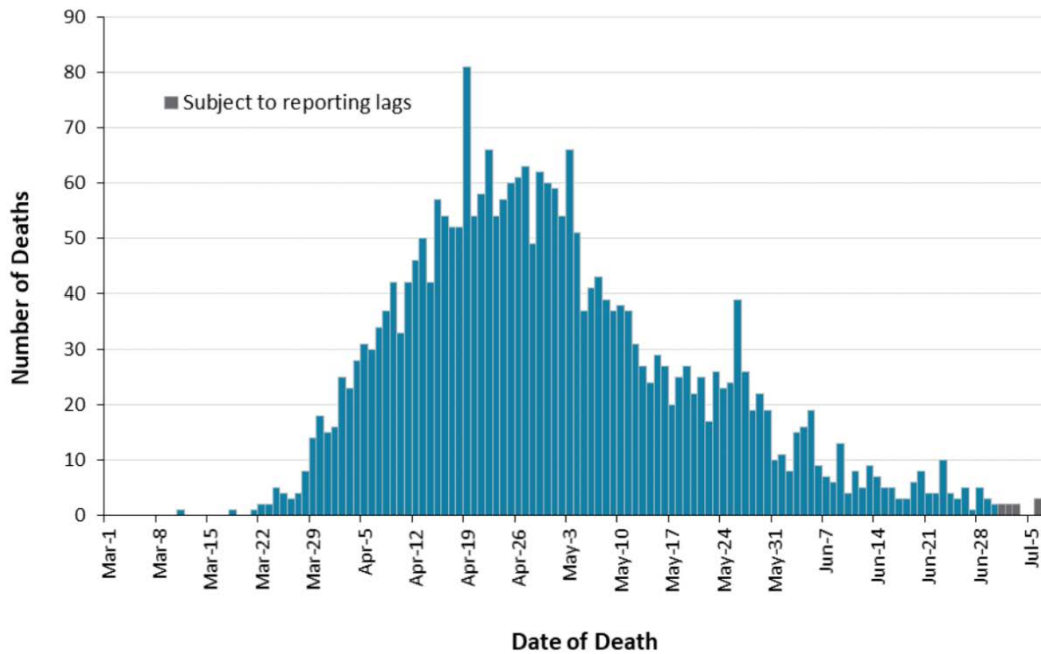
Dear Chair and Members of Board of Health,

I am a resident of Glanbrook and would like to express to you concern about the proposal to adopt mandatory mask requirements in Hamilton. I will make this point form out of respect for everyone's time.

1. The current data in Hamilton and Ontario don't appear to support mandating masks at this time. According to the attached report today from Ontario Public Health, it would seem that cases and severity (deaths) have gone down without this mandate. The original goal was to flatten the curve and that has been accomplished at this time. I have attached the full report but embedded this image here as well:

Severity

Figure 4. Confirmed deaths among COVID-19 cases by date of death: Ontario, March 1, 2020 to July 7, 2020



Note: Cases without a death date are not included in the figure.

Data Source: iPHIS plus

2. Our hospitals are both reporting the great news this week that they currently have zero covid positive cases. This has been accomplished without mask mandates.

HHS <https://www.hamiltonhealthsciences.ca/covid19>

COVID-19

Number of patients with confirmed COVID-19 we
are currently caring for in our hospital:
Zero

Current Overall Occupancy:
82%

St. Joe's <https://www.stjoes.ca/coronavirus>

▼ COVID-19 Cases at St. Joe's

Updated July 8th at 2:38 p.m.

Number of COVID-19 patients St. Joseph's Healthcare Hamilton is currently caring for in hospital	0
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[Click here](#) for more information on the status of cases in Hamilton.

3. I am concerned that mandating masks will further polarize our city and discriminate against those who cannot wear masks due to valid health concerns. Our community already has a high uptake in mask usage and those who cannot or chose not to wear masks don't seem to be getting in the way of our great work of flattening the curve. So why now?

4. I came across this interesting "work in progress" report by Denis Rancourt of Ottawa (attached: Masks Don't Work). I know the title is a bit off-putting but I do think the information contained within is worth consideration.

Finally, as a quick personal story, my mom works at a grocery store and found that after her 5-8 hour shifts of wearing a mask, she had headaches, pale skin, dark circles around her eyes and generally feeling unwell. As a normally healthy person, she was alarmed by this and has

decided that it is not good for her overall health to be wearing a face covering for long periods of time. **My concern is that we are trying to prevent a respiratory illness and mandating face coverings in the absence of good safety studies may be counter-productive to that goal.**

I do appreciate your time and consideration.

Respectfully,

Amy Newton

A solid black rectangular redaction box covering the signature area.

Daily Epidemiologic Summary

COVID-19 in Ontario: January 15, 2020 to July 7, 2020

This report includes the most current information available from iPHIS and other local case management systems (iPHIS plus) as of **July 7, 2020**.

Please visit the interactive [Ontario COVID-19 Data Tool](#) to explore recent COVID-19 data by public health unit, age group, sex, and trends over time.

A weekly summary report is available with additional information to complement the daily report.

This **daily** report provides an epidemiologic summary of recent COVID-19 activity in Ontario. The change in cases is determined by taking the cumulative difference between the current day and the previous day.

Highlights

- There are a total of 36,178 confirmed cases of COVID-19 in Ontario reported to date.
- Compared to the previous day, this represents:
 - An increase of 118 confirmed cases (percent change of +5.4%)
 - An increase of 9 deaths (percent change of +350.0%)
 - An increase of 202 resolved cases (percent change of +14.1%)

In this document, the term 'change in cases' refers to cases publicly reported by the province for a given day. Data corrections or updates can result in case records being removed and or updated from past reports and may result in subset totals for updated case counts (i.e., age group, gender) differing from the overall updated case counts.

The term public health unit reported date in this document refers to the date local public health units were first notified of the case.

Case Characteristics

Table 1a. Summary of recent cases of COVID-19: Ontario

	Change in cases July 6	Change in cases July 7	Percentage change July 7 compared to July 6	Cumulative case count as of July 7
Number of cases	112	118	+5.4%	36,178
Number of deaths	2	9	+350.0%	2,700
Number resolved	177	202	+14.1%	31,805

Note: The number of cases publicly reported by the province each day may not align with case counts reported to public health on a given day; public health unit reported date refers to the date local public health was first notified of the case.

Data Source: iPHIS plus

Table 1b. Summary of recent cases of COVID-19 by age group and gender: Ontario

	Change in cases July 6	Change in cases July 7	Cumulative case count as of July 7
Gender: Male	66	49	16,624
Gender: Female	48	67	19,272
Ages: 19 and under	14	13	1,800
Ages: 20-39	44	34	10,618
Ages: 40-59	33	35	10,995
Ages: 60-79	14	15	6,810
Ages: 80 and over	7	21	5,946

Note: Not all cases have a reported age or gender reported. Data corrections or updates can result in case records being removed and or updated from past reports and may result in subset totals (i.e., age group, gender) differing from past publicly reported case counts.

Data Source: iPHIS plus

Table 2. Summary of recent cases of COVID-19 in long-term care homes: Ontario

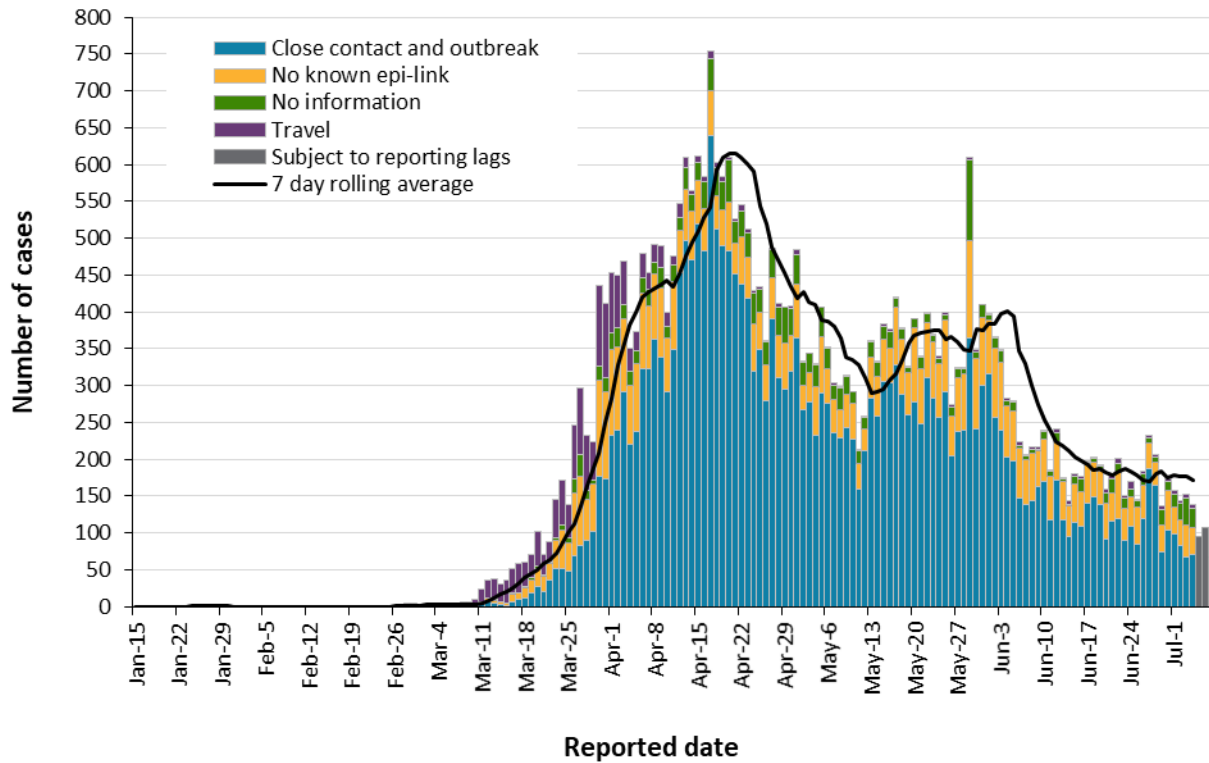
Long-term care home cases	Change in cases July 6	Change in cases July 7	Cumulative case count as of July 7
Residents	2	5	5,521
Health care workers	1	16	2,343
Deaths among residents	0	5	1,722
Deaths among health care workers	0	0	7

Note: Information for how long-term care home residents and health care workers are identified is available in the technical notes. The change in cases in these categories may represent existing case records that have been updated.

Data Source: iPHIS plus

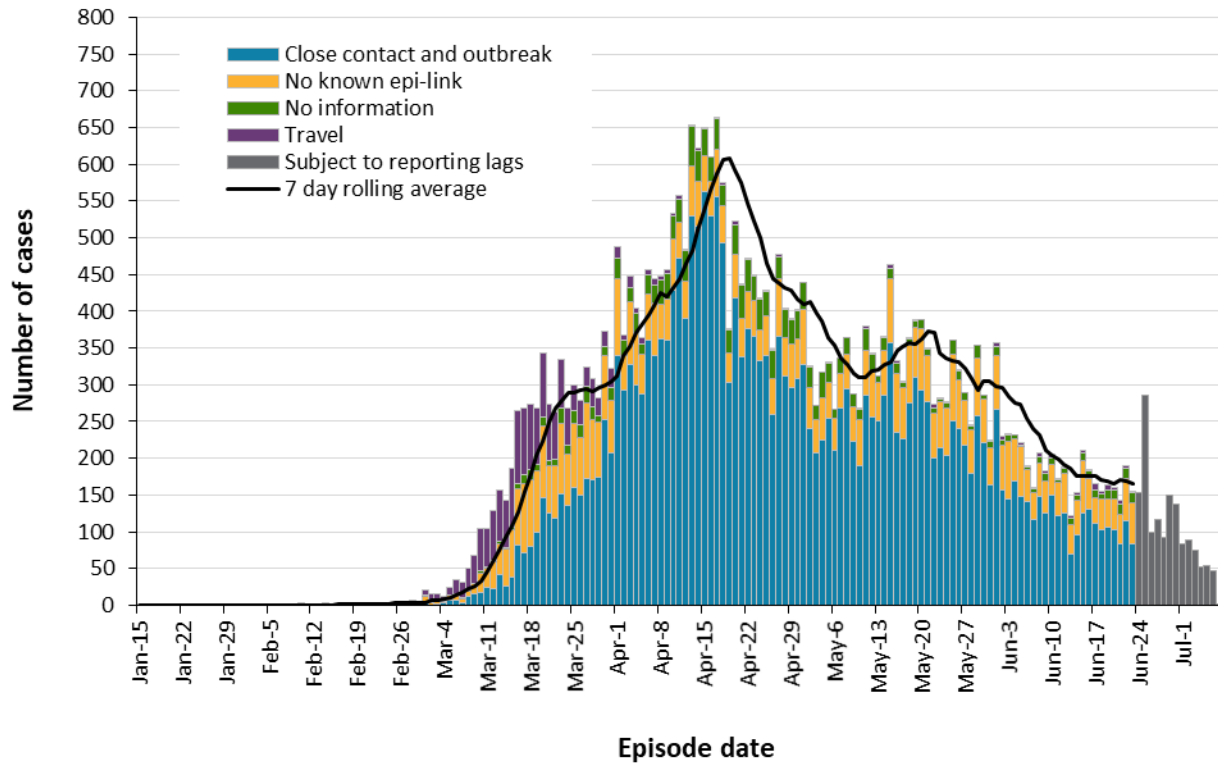
Time

Figure 1. Confirmed cases of COVID-19 by likely acquisition and public health unit reported date: Ontario, January 15, 2020 to July 7, 2020



Data Source: iPHIS plus

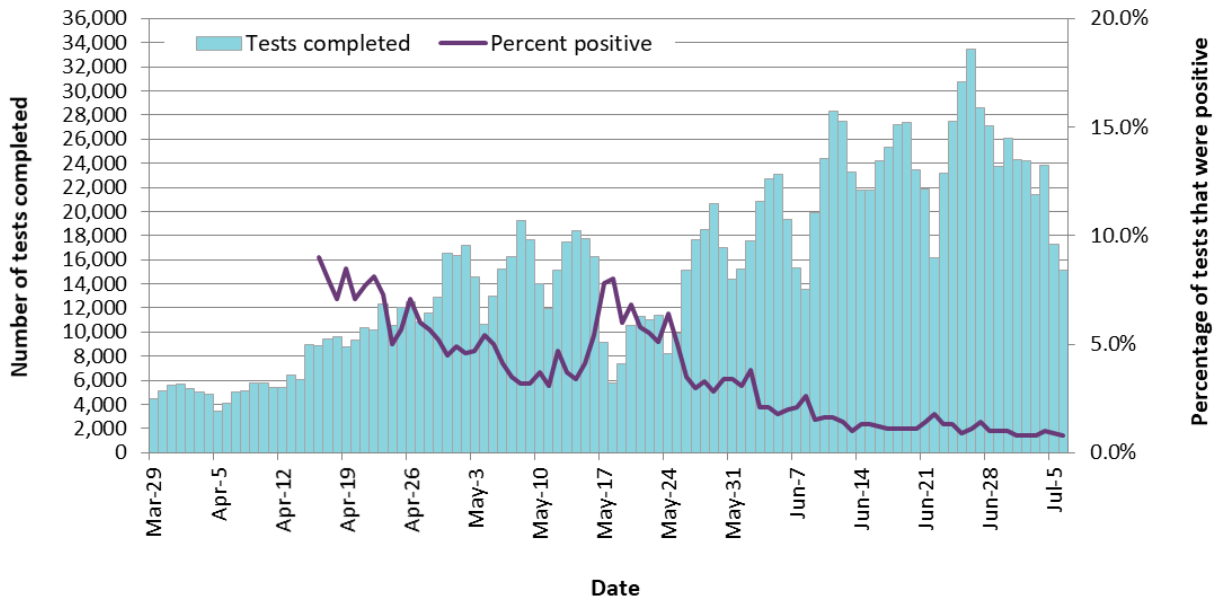
Figure 2. Confirmed cases of COVID-19 by likely acquisition and approximation of symptom onset date: Ontario, January 15, 2020 to July 7, 2020



Note: Not all cases may have an episode date and those without one are not included in the figure. Episode date is defined and available in the technical notes.

Data Source: iPHIS plus

Figure 3. Number of COVID-19 tests completed and percent positivity: Ontario, March 29, 2020 to July 6, 2020

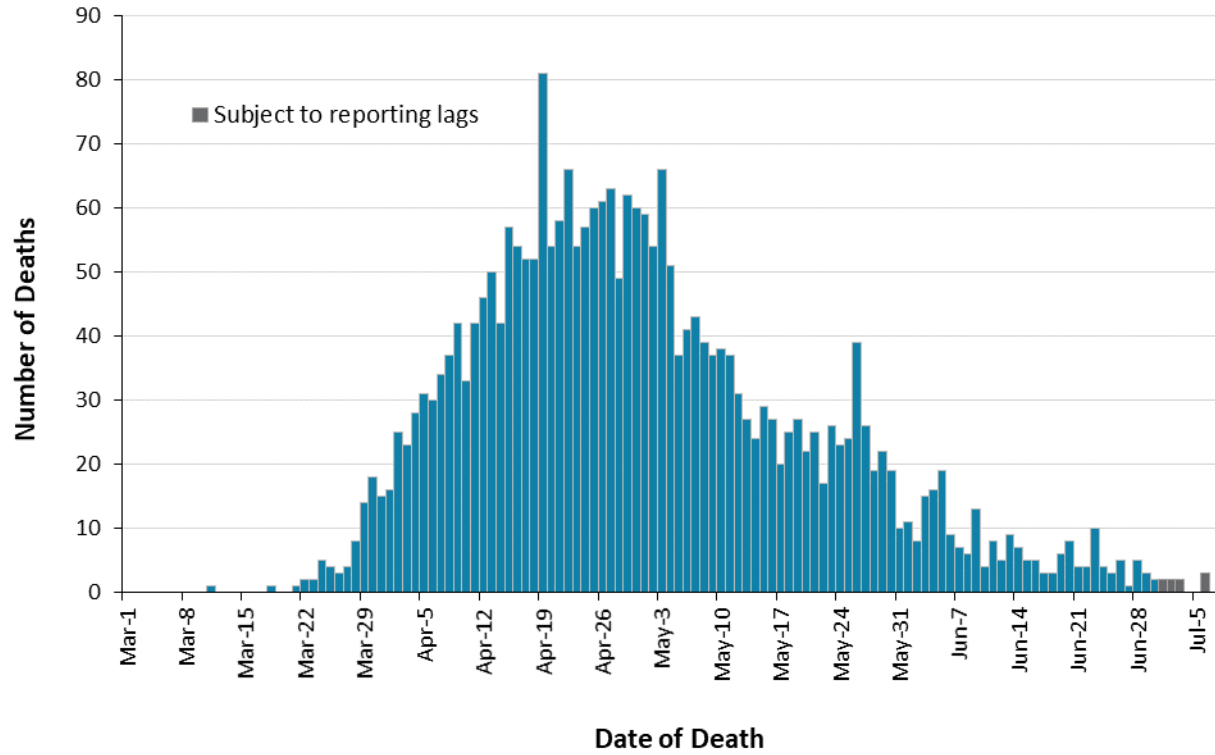


Note: The number of tests performed does not reflect the number of specimens or persons tested. More than one test may be performed per specimen or per person. As such, the percentage of tests that were positive does not necessarily translate to the number of specimens or persons testing positive.

Data Source: The Provincial COVID-19 Diagnostics Network, data reported by member microbiology laboratories.

Severity

Figure 4. Confirmed deaths among COVID-19 cases by date of death: Ontario, March 1, 2020 to July 7, 2020



Note: Cases without a death date are not included in the figure.

Data Source: iPHIS plus

Table 3. Confirmed cases of COVID-19 by severity: Ontario

	Cumulative case count as of July 7	Percentage of all cases
Cumulative deaths reported (please note there may be a reporting delay for deaths)	2,700	7.5%
Deaths reported in ages: 19 and under	1	0.1%
Deaths reported in ages: 20-39	11	0.1%
Deaths reported in ages: 40-59	109	1.0%
Deaths reported in ages: 60-79	718	10.5%
Deaths reported in ages: 80 and over	1,861	31.3%
Ever in ICU	965	2.7%
Ever hospitalized	4,452	12.3%

Data Source: iPHIS plus

Geography

Table 4. Summary of recent cases of COVID-19 by public health unit and region: Ontario

Public Health Unit Name	Change in cases July 6	Change in cases July 7	Cumulative case count	Cumulative rate per 100,000 population
Northwestern Health Unit	0	2	41	46.8
Thunder Bay District Health Unit	0	0	93	62.0
TOTAL NORTH WEST	0	2	134	56.4
Algoma Public Health	0	0	24	21.0
North Bay Parry Sound District Health Unit	0	0	35	27.0
Porcupine Health Unit	0	0	67	80.3
Public Health Sudbury & Districts	0	0	67	33.7
Timiskaming Health Unit	0	0	18	55.1
TOTAL NORTH EAST	0	0	211	37.7
Ottawa Public Health	0	6	2,123	201.3
Eastern Ontario Health Unit	0	0	166	79.5
Hastings Prince Edward Public Health	0	0	44	26.1
Kingston, Frontenac and Lennox & Addington Public Health	0	0	104	48.9
Leeds, Grenville & Lanark District Health Unit	0	0	354	204.4
Renfrew County and District Health Unit	0	0	29	26.7
TOTAL EASTERN	0	6	2,820	146.4

Public Health Unit Name	Change in cases July 6	Change in cases July 7	Cumulative case count	Cumulative rate per 100,000 population
Durham Region Health Department	6	3	1,724	242.0
Haliburton, Kawartha, Pine Ridge District Health Unit	0	1	201	106.4
Peel Public Health	39	27	6,027	375.3
Peterborough Public Health	0	0	95	64.2
Simcoe Muskoka District Health Unit	0	2	607	101.2
York Region Public Health	10	13	3,082	251.4
TOTAL CENTRAL EAST	55	46	11,736	261.9
Toronto Public Health	30	50	13,511	433.0
TOTAL TORONTO	30	50	13,511	433.0
Chatham-Kent Public Health	1	0	162	152.4
Grey Bruce Health Unit	0	0	107	63.0
Huron Perth Public Health	-1	0	59	42.2
Lambton Public Health	0	0	286	218.4
Middlesex-London Health Unit	0	1	631	124.3
Southwestern Public Health	0	1	85	40.2
Windsor-Essex County Health Unit	9	4	1,675	394.3
TOTAL SOUTH WEST	9	6	3,005	177.7
Brant County Health Unit	0	0	133	85.7
City of Hamilton Public Health Services	3	-1	847	143.0

Public Health Unit Name	Change in cases July 6	Change in cases July 7	Cumulative case count	Cumulative rate per 100,000 population
Haldimand-Norfolk Health Unit	0	1	431	377.8
Halton Region Public Health	9	1	781	126.2
Niagara Region Public Health	2	3	763	161.5
Region of Waterloo Public Health and Emergency Services	3	3	1,313	224.7
Wellington-Dufferin-Guelph Public Health	1	1	493	158.1
TOTAL CENTRAL WEST	18	8	4,761	167.1
TOTAL ONTARIO	112	118	36,178	243.4

Note: Health units with data corrections or updates could result in records being removed from totals resulting in negative counts.

Data Source: iPHIS plus

Outbreaks

Table 5. Summary of recent confirmed COVID-19 outbreaks reported in long-term care homes, retirement homes and hospitals by status: Ontario

Institution type	Change in outbreaks July 6	Change in outbreaks July 7	Number of ongoing outbreaks	Cumulative number of outbreaks reported
Long-term care homes	3	-1	44	370
Retirement homes	0	1	15	154
Hospitals	0	0	6	94

Note: Ongoing outbreaks includes all outbreaks that are 'Open' in iPHIS without a 'Declared Over Date' recorded.

Data Source: iPHIS

Technical Notes

Data Sources

- The data for this report were based on:
 - Information extracted from the Ontario Ministry of Health (Ministry) integrated Public Health Information System (iPHIS) database, as of **July 7, 2020 at 4 p.m.**
 - Information successfully uploaded to the Ministry from Local Systems: Toronto Public Health (Coronavirus Rapid Entry System) CORES, The Ottawa Public Health COVID-19 Ottawa Database (The COD) and Middlesex-London COVID-19 Case and Contact Management Tool (CCMtool) as of **July 7, 2020 at 2 p.m.**
- iPHIS and iPHIS plus (which includes iPHIS, CORES, The COD and COVID-19 CCMtool) are dynamic disease reporting systems, which allow ongoing updates to data previously entered. As a result, data extracted from iPHIS and the Local Systems represent a snapshot at the time of extraction and may differ from previous or subsequent reports.
- Ontario population projection data for 2020 were sourced from Ministry, IntelliHEALTH Ontario. Data were extracted on November 26, 2019.
- COVID-19 test data were based on information from The Provincial COVID-19 Diagnostics Network, reported by member microbiology laboratories.

Data Caveats:

- The data only represent cases reported to public health units and recorded in iPHIS plus. As a result, all counts will be subject to varying degrees of underreporting due to a variety of factors, such as disease awareness and medical care seeking behaviours, which may depend on severity of illness, clinical practice, changes in laboratory testing, and reporting behaviours.
- Lags in iPHIS plus data entry due to weekend staffing may result in lower case counts than would otherwise be recorded.
- Only cases meeting the confirmed case classification as listed in the MOH [COVID-19 case definition](#) are included in the report counts from iPHIS plus.
- The number of tests performed does not reflect the number of specimens or persons tested. More than one test may be performed per specimen or per person. As such, the percentage of tests that were positive does not necessarily translate to the number of specimens or persons testing positive.
- Reported date is the date the case was reported to the public health unit.
- Case episode date is based on an estimate of the best date of disease onset. This date is calculated based on either the date of symptom onset, specimen collection/test date, or the date reported to the public health unit.

- Resolved cases are determined only for COVID-19 cases that have not died. Cases that have died are considered fatal and not resolved. The following cases are classified as resolved:
 - Cases that are reported as ‘recovered’ in iPHIS
 - Cases that are not hospitalized and are 14 days past their episode date
 - Cases that are currently hospitalized (no hospital end date entered) and have a status of ‘closed’ in iPHIS (indicating public health unit follow-up is complete) and are 14 days past their symptom onset date or specimen collection date
- Hospitalization includes all cases for which a hospital admission date was reported at the time of data extraction. It includes cases that have been discharged from hospital as well as cases that are currently hospitalized. Emergency room visits are not included in the number of reported hospitalizations.
- ICU admission includes all cases for which an ICU admission date was reported at the time of data extraction. It is a subset of the count of hospitalized cases. It includes cases that have been treated or that are currently being treated in an ICU.
- Orientation of case counts by geography is based on the diagnosing health unit (DHU). DHU refers to the case's public health unit of residence at the time of illness onset and not necessarily the location of exposure. Cases for which the DHU was reported as MOH (to signify a case that is not a resident of Ontario) have been excluded from the analyses.
- Likely source of acquisition is determined by examining the exposure and risk factor fields from iPHIS and local systems to determine whether a case travelled, was associated with an outbreak, was a contact of a case, had no known epidemiological link (sporadic community transmission) or was reported to have an unknown source/no information was reported. Some cases may have no information reported if the case is untraceable, was lost to follow-up or referred to FNIHB. Cases with multiple exposures or risk factors were assigned to a single likely acquisition source group which was determined hierarchically in the following order:
 - For cases with an episode date *on or after* April 1, 2020: Outbreak-associated > close contact of a confirmed case > travel > no known epidemiological link > information missing or unknown
 - For cases with an episode date *before* April 1, 2020: Travel > outbreak-associated > close contact of a confirmed case > no known epidemiological link > information missing or unknown
- Deaths are determined by using the outcome field in iPHIS plus. Any case marked ‘Fatal’ is included in the deaths data. Deaths are included whether or not COVID-19 was determined to be a contributing or underlying cause of death as indicated in the iPHIS field Type of Death.
 - The date of death is determined using the outcome date field for cases marked as ‘Fatal’ in the outcome field.

- iPHIS cases for which the Disposition Status was reported as ENTERED IN ERROR, DOES NOT MEET DEFINITION, DUPLICATE-DO NOT USE, or any variation on these values have been excluded.
- Ongoing outbreaks are those that are reported in iPHIS as 'Open' without a 'Declared Over Date' recorded.
- 'Long-term care home residents' includes cases that reported 'Yes' to the risk factor 'Resident of nursing home or other chronic care facility' and reported to be part of an outbreak assigned as a long-term care home (via the Outbreak number or case comments field); or were reported to be part of an outbreak assigned as a long-term care home (via the outbreak number or case comments field) with an age over 70 years and did not report 'No' to the risk factor 'Resident of nursing home or other chronic care facility'. Excludes cases that reported 'Yes' to both risk factors: 'Resident of nursing home or other chronic care facility' and 'health care worker'.
- The 'health care workers' variable includes cases that reported 'Yes' to any of the occupation of health care worker, doctor, nurse, dentist, dental hygienist, midwife, other medical technicians, personal support worker, respiratory therapist, first responder.
- 'Health care workers associated with long-term care outbreaks' includes 'health care workers' reported to be part of an outbreak assigned as a long-term care home (via the outbreak number or case comments field). Excludes cases that reported 'Yes' to risk factors 'Resident of nursing home or other chronic care facility' and 'Yes' to the calculated 'health care workers' variable.
- Percent change is calculated by taking the difference between the current day and previous day, divided by the previous day count.

Disclaimer

This document was developed by Public Health Ontario (PHO). PHO provides scientific and technical advice to Ontario's government, public health organizations and health care providers. PHO's work is guided by the current best available evidence at the time of publication.

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For Further Information

For more information, email cd@oahpp.ca.

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Masks Don't Work: A review of science relevant to COVID-19 social policy

Technical Report · April 2020

DOI: 10.13140/RG.2.2.14320.40967/1

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Masks Don't Work

A review of science relevant to COVID-19 social policy

Denis G. Rancourt, PhD
Researcher, Ontario Civil Liberties Association (ocla.ca)

Working report, published at Research Gate
(https://www.researchgate.net/profile/D_Rancourt)

April 2020

Summary / Abstract

Masks and respirators do not work.

There have been extensive randomized controlled trial (RCT) studies, and meta-analysis reviews of RCT studies, which all show that masks and respirators do not work to prevent respiratory influenza-like illnesses, or respiratory illnesses believed to be transmitted by droplets and aerosol particles.

Furthermore, the relevant known physics and biology, which I review, are such that masks and respirators should not work. It would be a paradox if masks and respirators worked, given what we know about viral respiratory diseases: The main transmission path is long-residence-time aerosol particles ($< 2.5 \mu\text{m}$), which are too fine to be blocked, and the minimum-infective-dose is smaller than one aerosol particle.

The present paper about masks illustrates the degree to which governments, the mainstream media, and institutional propagandists can decide to operate in a science vacuum, or select only incomplete science that serves their interests. Such recklessness is also certainly the case with the current global lockdown of over 1 billion people, an unprecedented experiment in medical and political history.

Review of the Medical Literature

Here are key anchor points to the extensive scientific literature that establishes that wearing surgical masks and respirators (e.g., “N95”) does not reduce the risk of contracting a verified illness:

Jacobs, J. L. et al. (2009) “Use of surgical face masks to reduce the incidence of the common cold among health care workers in Japan: A randomized controlled trial”, *American Journal of Infection Control*, Volume 37, Issue 5, 417 - 419.

<https://www.ncbi.nlm.nih.gov/pubmed/19216002>

N95-masked health-care workers (HCW) were significantly more likely to experience headaches. Face mask use in HCW was not demonstrated to provide benefit in terms of cold symptoms or getting colds.

Cowling, B. et al. (2010) “Face masks to prevent transmission of influenza virus: A systematic review”, *Epidemiology and Infection*, 138(4), 449-456.

doi:10.1017/S0950268809991658

<https://www.cambridge.org/core/journals/epidemiology-and-infection/article/face-masks-to-prevent-transmission-of-influenza-virus-a-systematic-review/64D368496EBDE0AFCC6639CCC9D8BC05>

None of the studies reviewed showed a benefit from wearing a mask, in either HCW or community members in households (H). See summary Tables 1 and 2 therein.

bin-Reza et al. (2012) “The use of masks and respirators to prevent transmission of influenza: a systematic review of the scientific evidence”, *Influenza and Other Respiratory Viruses* 6(4), 257–267.

<https://onlinelibrary.wiley.com/doi/epdf/10.1111/j.1750-2659.2011.00307.x>

“There were 17 eligible studies. ... None of the studies established a conclusive relationship between mask/respirator use and protection against influenza infection.”

Smith, J.D. et al. (2016) “Effectiveness of N95 respirators versus surgical masks in protecting health care workers from acute respiratory infection: a systematic review and meta-analysis”, *CMAJ* Mar 2016, cmaj.150835; DOI: 10.1503/cmaj.150835

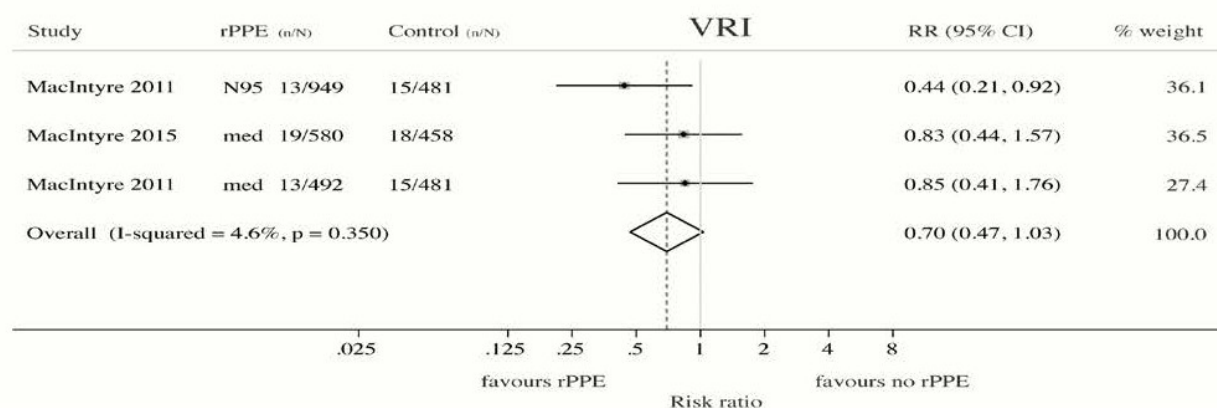
<https://www.cmaj.ca/content/188/8/567>

“We identified 6 clinical studies ... In the meta-analysis of the clinical studies, we found no significant difference between N95 respirators and surgical masks in associated risk of (a) laboratory-confirmed respiratory infection, (b) influenza-like illness, or (c) reported work-place absenteeism.”

Offeddu, V. et al. (2017) “Effectiveness of Masks and Respirators Against Respiratory Infections in Healthcare Workers: A Systematic Review and Meta-Analysis”, *Clinical Infectious Diseases*, Volume 65, Issue 11, 1 December 2017, Pages 1934–1942, <https://doi.org/10.1093/cid/cix681>

<https://academic.oup.com/cid/article/65/11/1934/4068747>

“Self-reported assessment of clinical outcomes was prone to bias. Evidence of a protective effect of masks or respirators against verified respiratory infection (VRI) was not statistically significant”; as per Fig. 2c therein:



Radonovich, L.J. et al. (2019) “N95 Respirators vs Medical Masks for Preventing Influenza Among Health Care Personnel: A Randomized Clinical Trial”, *JAMA*. 2019; 322(9): 824–833. doi:10.1001/jama.2019.11645

<https://jamanetwork.com/journals/jama/fullarticle/2749214>

“Among 2862 randomized participants, 2371 completed the study and accounted for 5180 HCW-seasons. ... Among outpatient health care personnel, N95 respirators vs medical masks as worn by participants in this trial resulted in no significant difference in the incidence of laboratory-confirmed influenza.”

Long, Y. et al. (2020) “Effectiveness of N95 respirators versus surgical masks against influenza: A systematic review and meta-analysis”, *J Evid Based Med*. 2020; 1- 9. <https://doi.org/10.1111/jebm.12381>

<https://onlinelibrary.wiley.com/doi/epdf/10.1111/jebm.12381>

“A total of six RCTs involving 9 171 participants were included. There were no statistically significant differences in preventing laboratory-confirmed influenza, laboratory-confirmed respiratory viral infections, laboratory-confirmed respiratory infection and influenza-like illness using N95 respirators and surgical masks. Meta-analysis indicated a protective effect of N95 respirators against laboratory-confirmed bacterial colonization (RR = 0.58, 95% CI 0.43-0.78). The

use of N95 respirators compared with surgical masks is not associated with a lower risk of laboratory-confirmed influenza.”

Conclusion Regarding that Masks Do Not Work

No RCT study with verified outcome shows a benefit for HCW or community members in households to wearing a mask or respirator. There is no such study. There are no exceptions.

Likewise, no study exists that shows a benefit from a broad policy to wear masks in public (more on this below).

Furthermore, if there were any benefit to wearing a mask, because of the blocking power against droplets and aerosol particles, then there should be more benefit from wearing a respirator (N95) compared to a surgical mask, yet several large meta-analyses, and all the RCT, prove that there is no such relative benefit.

Masks and respirators do not work.

Precautionary Principle Turned on Its Head with Masks

In light of the medical research, therefore, it is difficult to understand why public-health authorities are not consistently adamant about this established scientific result, since the distributed psychological, economic and environmental harm from a broad recommendation to wear masks is significant, not to mention the unknown potential harm from concentration and distribution of pathogens on and from used masks. In this case, public authorities would be turning the precautionary principle on its head (see below).

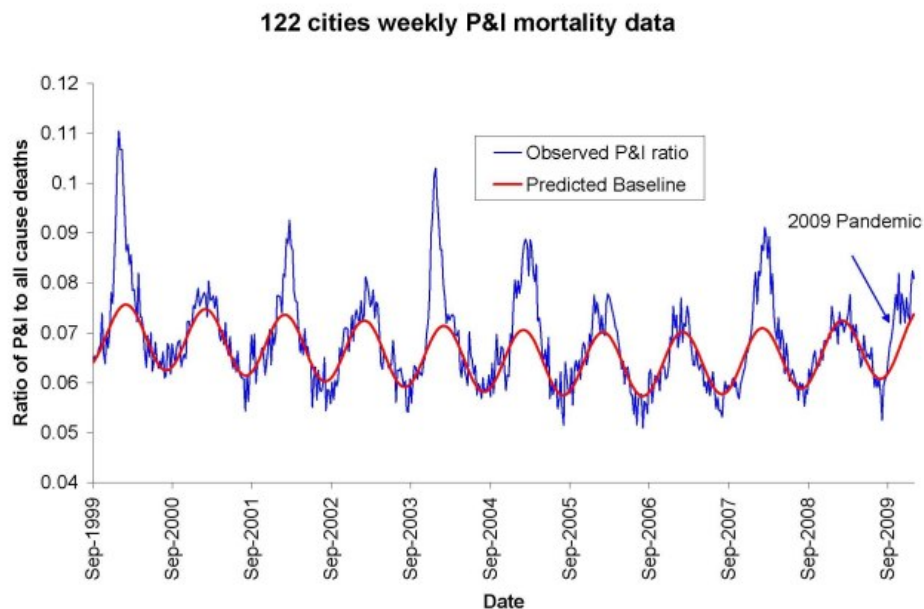
Physics and Biology of Viral Respiratory Disease and of Why Masks Do Not Work

In order to understand why masks cannot possibly work, we must review established knowledge about viral respiratory diseases, the mechanism of seasonal variation of excess deaths from pneumonia and influenza, the aerosol mechanism of infectious disease transmission, the physics and chemistry of aerosols, and the mechanism of the so-called minimum-infective-dose.

In addition to pandemics that can occur anytime, in the temperate latitudes there is an extra burden of respiratory-disease mortality that is seasonal, and that is caused by viruses. For

example, see the review of influenza by Paules and Subbarao (2017). This has been known for a long time, and the seasonal pattern is exceedingly regular.

For example, see Figure 1 of Viboud (2010), which has “Weekly time series of the ratio of deaths from pneumonia and influenza to all deaths, based on the 122 cities surveillance in the US (blue line). The red line represents the expected baseline ratio in the absence of influenza activity,” here:



The seasonality of the phenomenon was largely not understood until a decade ago. Until recently, it was debated whether the pattern arose primarily because of seasonal change in virulence of the pathogens, or because of seasonal change in susceptibility of the host (such as from dry air causing tissue irritation, or diminished daylight causing vitamin deficiency or hormonal stress). For example, see Dowell (2001).

In a landmark study, Shaman et al. (2010) showed that the seasonal pattern of extra respiratory-disease mortality can be explained quantitatively on the sole basis of absolute humidity, and its direct controlling impact on transmission of airborne pathogens.

Lowen et al. (2007) demonstrated the phenomenon of humidity-dependent airborne-virus virulence in actual disease transmission between guinea pigs, and discussed potential underlying mechanisms for the measured controlling effect of humidity.

The underlying mechanism is that the pathogen-laden aerosol particles or droplets are neutralized within a half-life that monotonically and significantly decreases with increasing ambient humidity. This is based on the seminal work of Harper (1961). Harper experimentally showed that viral-pathogen-carrying droplets were inactivated within shorter and shorter times, as ambient humidity was increased.

Harper argued that the viruses themselves were made inoperative by the humidity (“viable decay”), however, he admitted that the effect could be from humidity-enhanced physical removal or sedimentation of the droplets (“physical loss”): “Aerosol viabilities reported in this paper are based on the ratio of virus titre to radioactive count in suspension and cloud samples, and can be criticized on the ground that test and tracer materials were not physically identical.”

The latter (“physical loss”) seems more plausible to me, since humidity would have a universal physical effect of causing particle / droplet growth and sedimentation, and all tested viral pathogens have essentially the same humidity-driven “decay”. Furthermore, it is difficult to understand how a virion (of all virus types) in a droplet would be molecularly or structurally attacked or damaged by an increase in ambient humidity. A “virion” is the complete, infective form of a virus outside a host cell, with a core of RNA or DNA and a capsid. The actual mechanism of such humidity-driven intra-droplet “viable decay” of a virion has not been explained or studied.

In any case, the explanation and model of Shaman et al. (2010) is not dependant on the particular mechanism of the humidity-driven decay of virions in aerosol / droplets. Shaman’s quantitatively demonstrated model of seasonal regional viral epidemiology is valid for either mechanism (or combination of mechanisms), whether “viable decay” or “physical loss”.

The breakthrough achieved by Shaman et al. is not merely some academic point. Rather, it has profound health-policy implications, which have been entirely ignored or overlooked in the current coronavirus pandemic.

In particular, Shaman’s work necessarily implies that, rather than being a fixed number (dependent solely on the spatial-temporal structure of social interactions in a completely susceptible population, and on the viral strain), the epidemic’s **basic reproduction number** (R_0) is highly or predominantly dependent on ambient absolute humidity.

For a definition of R_0 , see HealthKnowledge-UK (2020): R_0 is “the average number of secondary infections produced by a typical case of an infection in a population where everyone is susceptible.” The average R_0 for influenza is said to be 1.28 (1.19–1.37); see the comprehensive review by Biggerstaff et al. (2014).

In fact, Shaman et al. showed that R_0 must be understood to seasonally vary between humid-summer values of just larger than “1” and dry-winter values typically as large as “4” (for example, see their Table 2). In other words, the seasonal infectious viral respiratory diseases that plague temperate latitudes every year go from being intrinsically mildly contagious to

virulently contagious, due simply to the bio-physical mode of transmission controlled by atmospheric humidity, irrespective of any other consideration.

Therefore, all the epidemiological mathematical modelling of the benefits of mediating policies (such as social distancing), which assumes humidity-independent R_0 values, has a large likelihood of being of little value, on this basis alone. For studies about modelling and regarding mediation effects on the effective reproduction number, see Coburn (2009) and Tracht (2010).

To put it simply, the “second wave” of an epidemic is not a consequence of human sin regarding mask wearing and hand shaking. Rather, the “second wave” is an inescapable consequence of an air-dryness-driven many-fold increase in disease contagiousness, in a population that has not yet attained immunity.

If my view of the mechanism is correct (i.e., “physical loss”), then Shaman’s work further necessarily implies that the dryness-driven high transmissibility (large R_0) arises from small aerosol particles fluidly suspended in the air; as opposed to large droplets that are quickly gravitationally removed from the air.

Such small aerosol particles fluidly suspended in air, of biological origin, are of every variety and are everywhere, including down to virion-sizes (Despres, 2012). It is not entirely unlikely that viruses can thereby be physically transported over inter-continental distances (e.g., Hammond, 1989).

More to the point, indoor airborne virus concentrations have been shown to exist (in day-care facilities, health centres, and onboard airplanes) primarily as aerosol particles of diameters smaller than $2.5 \mu\text{m}$, such as in the work of Yang et al. (2011):

“Half of the 16 samples were positive, and their total virus concentrations ranged from 5800 to 37 000 genome copies m^{-3} . On average, 64 per cent of the viral genome copies were associated with fine particles smaller than $2.5 \mu\text{m}$, which can remain suspended for hours. Modelling of virus concentrations indoors suggested a source strength of $1.6 \pm 1.2 \times 10^5$ genome copies $\text{m}^{-3} \text{air h}^{-1}$ and a deposition flux onto surfaces of 13 ± 7 genome copies $\text{m}^{-2} \text{h}^{-1}$ by Brownian motion. Over 1 hour, the inhalation dose was estimated to be 30 ± 18 median tissue culture infectious dose (TCID_{50}), adequate to induce infection. These results provide quantitative support for the idea that the aerosol route could be an important mode of influenza transmission.”

Such small particles ($< 2.5 \mu\text{m}$) are part of air fluidity, are not subject to gravitational sedimentation, and would not be stopped by long-range inertial impact. This means that the slightest (even momentary) facial misfit of a mask or respirator renders the design filtration norm of the mask or respirator entirely irrelevant. In any case, the filtration material itself of

N95 (average pore size $\sim 0.3\text{--}0.5\ \mu\text{m}$) does not block virion penetration, not to mention surgical masks. For example, see Balazy et al. (2006).

Mask stoppage efficiency and host inhalation are only half of the equation, however, because the minimal infective dose (MID) must also be considered. For example, if a large number of pathogen-laden particles must be delivered to the lung within a certain time for the illness to take hold, then partial blocking by any mask or cloth can be enough to make a significant difference.

On the other hand, if the MID is amply surpassed by the virions carried in a single aerosol particle able to evade mask-capture, then the mask is of no practical utility, which is the case.

Yezli and Otter (2011), in their review of the MID, point out relevant features:

- most respiratory viruses are as infective in humans as in tissue culture having optimal laboratory susceptibility
- it is believed that a single virion can be enough to induce illness in the host
- the 50%-probability MID (“TCID₅₀”) has variably been found to be in the range 100–1000 virions
- there are typically $10^3\text{--}10^7$ virions per aerolized influenza droplet with diameter $1\ \mu\text{m} - 10\ \mu\text{m}$
- the 50%-probability MID easily fits into a single (one) aerolized droplet

For further background:

- A classic description of dose-response assessment is provided by Haas (1993).
- Zwart et al. (2009) provided the first laboratory proof, in a virus-insect system, that the action of a single virion can be sufficient to cause disease.
- Baccam et al. (2006) calculated from empirical data that, with influenza A in humans, “we estimate that after a delay of ~ 6 h, infected cells begin producing influenza virus and continue to do so for ~ 5 h. The average lifetime of infected cells is ~ 11 h, and the half-life of free infectious virus is ~ 3 h. We calculated the [in-body] basic reproductive number, R_0 , which indicated that a single infected cell could produce ~ 22 new productive infections.”
- Brooke et al. (2013) showed that, contrary to prior modeling assumptions, although not all influenza-A-infected cells in the human body produce infectious progeny (virions), nonetheless, 90% of infected cell are significantly impacted, rather than simply surviving unharmed.

All of this to say that: if anything gets through (and it always does, irrespective of the mask), then you are going to be infected. Masks cannot possibly work. It is not surprising, therefore, that no bias-free study has ever found a benefit from wearing a mask or respirator in this application.

Therefore, the studies that show partial stopping power of masks, or that show that masks can capture many large droplets produced by a sneezing or coughing mask-wearer, in light of the above-described features of the problem, are irrelevant. For example, such studies as these: Leung (2020), Davies (2013), Lai (2012), and Sande (2008).

Why There Can Never Be an Empirical Test of a Nation-Wide Mask-Wearing Policy

As mentioned above, no study exists that shows a benefit from a broad policy to wear masks in public. There is good reason for this. It would be impossible to obtain unambiguous and bias-free results:

- Any benefit from mask-wearing would have to be a small effect, since undetected in controlled experiments, which would be swamped by the larger effects, notably the large effect from changing atmospheric humidity.
- Mask compliance and mask adjustment habits would be unknown.
- Mask-wearing is associated (correlated) with several other health behaviours; see Wada (2012).
- The results would not be transferable, because of differing cultural habits.
- Compliance is achieved by fear, and individuals can habituate to fear-based propaganda, and can have disparate basic responses.
- Monitoring and compliance measurement are near-impossible, and subject to large errors.
- Self-reporting (such as in surveys) is notoriously biased, because individuals have the self-interested belief that their efforts are useful.
- Progression of the epidemic is not verified with reliable tests on large population samples, and generally relies on non-representative hospital visits or admissions.
- Several different pathogens (viruses and strains of viruses) causing respiratory illness generally act together, in the same population and/or in individuals, and are not resolved, while having different epidemiological characteristics.

Unknown Aspects of Mask Wearing

Many potential harms may arise from broad public policies to wear masks, and the following unanswered questions arise:

- Do used and loaded masks become sources of enhanced transmission, for the wearer and others?

- Do masks become collectors and retainers of pathogens that the mask wearer would otherwise avoid when breathing without a mask?
- Are large droplets captured by a mask atomized or aerolized into breathable components? Can virions escape an evaporating droplet stuck to a mask fiber?
- What are the dangers of bacterial growth on a used and loaded mask?
- How do pathogen-laden droplets interact with environmental dust and aerosols captured on the mask?
- What are long-term health effects on HCW, such as headaches, arising from impeded breathing?
- Are there negative social consequences to a masked society?
- Are there negative psychological consequences to wearing a mask, as a fear-based behavioural modification?
- What are the environmental consequences of mask manufacturing and disposal?
- Do the masks shed fibres or substances that are harmful when inhaled?

Conclusion

By making mask-wearing recommendations and policies for the general public, or by expressly condoning the practice, governments have both ignored the scientific evidence and done the opposite of following the precautionary principle.

In an absence of knowledge, governments should not make policies that have a hypothetical potential to cause harm. The government has an onus barrier before it instigates a broad social-engineering intervention, or allows corporations to exploit fear-based sentiments.

Furthermore, individuals should know that there is no known benefit arising from wearing a mask in a viral respiratory illness epidemic, and that scientific studies have shown that any benefit must be residually small, compared to other and determinative factors.

Otherwise, what is the point of publicly funded science?

The present paper about masks illustrates the degree to which governments, the mainstream media, and institutional propagandists can decide to operate in a science vacuum, or select only incomplete science that serves their interests. Such recklessness is also certainly the case with the current global lockdown of over 1 billion people, an unprecedented experiment in medical and political history.

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