VAPING, SARS-CoV-2 AND COVID-19
TECHNICAL INFORMATION FOR VAPERS
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Roberto Sussman* and Carmen Escrig **

* Institute for Nuclear Sciences, National University of México UNAM. Physics PhD. Director of Pro-Vapeo México AC
** Universidad Autónoma de Madrid, Spain. Biology PhD specialized in Virology. Coordinator of the Medical Platform for Tobacco Harm Reduction in Spain.

Summary

WHY THIS DOCUMENT? The spread of the SARS-CoV-2 pandemic provides fertile ground for spreading misinformation on vaping. Vapers must be equipped with solid information and data to counterargue.
ON SMOKING. The relation between smoking and the progression to severe conditions of COVID-19 is still uncertain, though identified vulnerability conditions for this progression (cardiovascular and respiratory disease, diabetes) in mostly senior patients are strongly correlated with long term harms from smoking. However, currently observed data from Chinese and USA studies shows unexpectedly low rates of smokers among COVID-19 patients (even in serious condition). Much more research is needed on this issue.
ON VAPING. There is no compelling evidence that vaping (intrinsically) increases the risk of infection or progression to severe condition of COVID-19. When evaluating risks on vapers it is necessary to consider that most are ex-smokers or still smokers. Vapers with a long previous smoking history could exhibit conditions seen in vulnerable patients. However, this would not be an effect of vaping but of previous smoking. Since completely switching from smoking to vaping improves cardiovascular and respiratory conditions, smokers who switch to vaping are expected to have a better prognosis if infected by SARSCoV-2, but more research is needed on this issue.
ON PROPYLENE GLYCOL AS A PROTECTIVE AGENT. Because of its hygroscopic nature propylene glycol (PG) vapor (but not PG droplets) can act as environmental disinfectant wiping out pathogens under specific physical conditions. However, there is no evidence on whether this effect will work on SARS-CoV-2 and in the context of vaping.
ON ENVIRONMENTAL VAPOR. There are no reported and verified cases of contagion. Exhaled vapor from an infected vaper is a negligible contagion factor: it can spread very few virus carrying droplets, as much as blowing or mouth breathing, slightly more than sedentary breathing and far less than coughing or sneezing. However, exhaled vapor can only spread the virus when vaping, thus infected vapers (or any infected person) will spread much more viruses over time from their continuous regular breathing.
RECOMMENDATIONS. Vapers who no longer smoke should not go back to smoking. The precautions to prevent contagion from virus carried by e-cigarette vapor are the same "social distancing" and hygiene measures recommended to all the population including non-vapers: avoid physical contact and proximity to others. For vapers specifically: vape with low powered devices when accompanied, avoid vaping in public indoor spaces and in outdoor spaces vape at least 2 meters away from others.

The misinformation pandemic

Unfortunately, the spread of the SARS-CoV-2 pandemic follows the years long ongoing pandemic of serious misinformation on vaping. One of the main spearheads of this misinformation is undoubtedly Professor Stanton Glantz from the University of California at San Francisco. In his professional blog [1] Professor Glantz squarely puts vaping and smoking on equal footing as serious risk factors for progression to COVID-19. Specifically, Glantz justifies this assessment by stating that:

> *The recent excellent summary of the evidence on the pulmonary effects of e-cigarettes reported multiple ways that e-cigarettes impair lungs' ability to fight off infections.*

A statement followed by listing a litany of adverse effects of vaping on respiratory infections, all taken from studies examined in the review by Gotts et al [2] (the "excellent summary"). While the popular journal Scientific American recognizes that *Vapers' risk of viral infections has not been studied much* [3], it has cited Glantz and has also recycled some of the results reported by Gotts et al.

The review by Gotts et al, which Glantz and Scientific American take as source, is extremely superficial, biased and selective, it cited uncritically only studies reporting adverse effects, all of which are either acute effects without clinical relevance or cross sectional studies based on small samples of vapers in which the huge confounding effect of previous smoking history was not properly handled (see a critique of such studies in a much more balanced and extensive review of respiratory effects of vaping by Polosa et al [4]). Moreover, Gotts et al (and Glantz quoting them) interpret the results in a very selective manner. A representative example of their modus operandi is furnished by their assessment of the results obtained by one of the revised studies by Saudt et al [5]. From Glantz's exact quote of Gotts et al we have

> *Healthy non-smokers were exposed to e-cigarette aerosol, and bronchoalveolar lavage was obtained to study alveolar macrophages. The expression of more than 60 genes was altered in e-cigarette users' alveolar macrophages two hours after just 20 puffs, including genes involved in inflammation.*

Curiously, Gotts et al and Glantz omit mentioning that the effects examined in [5] were acute and that the same study reports that "*No significant changes in clinical parameters were observed*". Gotts et al and Glantz quoting them also omit mentioning evidence pointing in the opposite direction: as reported by several studies reviewed by Polosa et al [4] the usage of e-cigarettes actually reduces the presence of pathogens and respiratory infections. A significant decrease of respiratory infections in e-cigarette users has also been reported in a large scope randomized controlled trial researching smoking cessation [6], a result based on a 12 months long clinical observation on a large sample of subjects. This result (and similar results in other randomized trials reviewed in [7]) are real life observational results that are more relevant to assess the possible immune response of vapers in the context of COVID-19 than the adverse acute effects in idealized lab studies reported uncritically by Gotts et al in [2] and recycled by Glantz and Scientific American.

Professor Glantz is perhaps the most vocal spearhead, but he is far from being the only academic in the vast USA sourced anti-vaping activism, which is now presenting the relation of vaping and the SARS-CoV-2 pandemic through the grossly biased assessments from reviews like that of Gotts et al, conflating carelessly the risks of vaping and smoking and ignoring all contrary or critical evidence. It is very unfortunate that mainstream academia, politicians and the media in the USA are predominantly fed by this constant flow of misinformation, as can be seen in statements by the Major of New York City, Bill de Blasio [8], and by various media outlets [9].

As expected, the misinformation described above is further spreading. Notwithstanding the lack of hard evidence, Bloomberg's news agency [10] features prominently an email received from Michael Felberbaum, an spokesman for the Food and Drug Administration (FDA), explicitly stating that "*e-cigarettes can damage lung cells*", adding that people who vape (as those who smoke) "*may have increased risk for serious complications from COVID-19*". It also cites Dr Nora Volkow, the director of the National Institute of Drug Addiction (NIDA), stating that coronavirus could be an all especially serious threat to those who vape and smoke tobacco or marijuana. This media piece is providing global reach to the same type of misleading statements expressed by Glantz, all of which lack any bearing on the available data. As a reaction to this misinformation, a latter signed by 13 international scientific authorities has been sent to the FDA expressing deep concern on this issue [11]:

> *It is likely that many older adult vapers will have underlying conditions that increase their vulnerability and likelihood of severe or fatal COVID-19 symptoms. This is because many are former or current smokers and will have accumulated damage to their cardiovascular and respiratory systems through many years of smoking. Many will be vaping with the express purpose of reducing their smoking-related risks and/or relieving their symptoms. It is therefore particularly important that great care is taken with advice to this group. On what basis is FDA confident that it is right to discourage people with underlying smoking-related conditions from vaping at this time, given the likely alternative for many is a return to smoking? Where is the evidence-based reasoning that advising adult smokers against vaping is appropriate for the protection of public health at any time, but especially during this COVID-19 crisis? We know of no relevant and informative evidence on vaping and COVD-19 and the evidence on smoking and COVID-19 is inconclusive and contradictory.*

COVID-19 and smokers

A good reference reviewing the available evidence on the relation between smoking, vaping and COVID19 has been the article written by Farsalinos, Barbouni and Niaura (its first version was dated March 23 [12], see also the professional blog entry of Farsalinos [13]). The authors revised the data from five studies available at the time on patients infected by SARS-CoV-2, concluding that the relation between smoking cigarettes and the severity of COVID-19 in infected Chinese patients was uncertain, as smokers were underrepresented among the patients (to put this in context: 52.1% of Chinese men smoke whereas only 2.7% of women do).

In his blog entry Farsalinos examined in more detail the data from the study with the largest sample [14] at the time: 1096 patients, of whom only 12.5% were current smokers (1.9% ex-smokers), which (as in the other studies) is a much lesser proportion than that found among the population bearing in mind that 58.1% of the sample were men and practically 100% older than 15 years (to be representative of the population we would expect the proportion of smokers in the sample to be 29%). Of the 1096 patients:

- 926 were reported without severe affectation (11.8% smokers)
- 173 were reported with severe affectation (11.8% smokers)
- 67 were reported in critical situation with intensive care, mechanical ventilation or dead (25.8% smokers)

These numbers indicate a higher proportion of smokers among those with severe outcomes, but still lower than in the general Chinese population given the high smoking prevalence among Chinese men. Evidently, smoking contributes to identified vulnerability conditions, such as cardiovascular ailments, diabetes or chronic lung disease, but nevertheless smokers were underrepresented.

However, more information and other studies have been emerging gradually (see for example [15]) and in fact, the data is apparently leading to surprising outcomes. In its most recent update (dated March 29) of Farsalinos, Barbouni and Niaura [16] the authors conclude that results *"do not support the argument that current smoking is a risk factor for hospitalization for COVID-19 and might even suggest a protective role"*. This unexpected result might be related to the *"downregulation of ACE2 expression that has been previously known to be induced by smoking"*.

ACE2 is the receptor that SARS-CoV-2 recognise in the cell surface acting as a key and lock mechanism to enter the cell and infect it. So, a lower presence of ACE2 ("the lock") in the cell surfaces could, at first sight, possibly suppose an advantage. However, studies show that ACE2 is also involved in protective mechanisms of the lung. It may protect against severe lung injury induced by respiratory virus infection in experimental mouse models and in paediatric patients. ACE2 also protects against severe acute lung injury that can be triggered by sepsis, acid aspiration, SARS, and lethal avian influenza A H5N1 virus infection [17]. Therefore, the low expression of ACE2 could on the one hand protect from infection by having fewer viral receptors ("locks") present, but on the other hand be detrimental by decreasing the protective capacity of the lung.

Further paradoxical results as those found in China have also emerged in recent data from the Centers for Disease Control and Prevention (CDC) in USA [18]. Among 7,074 COVID-19 patients whose medical data was fully complete, only 96 (1.3%) were smokers and only 165 (2.3%) were ex-smokers. These percentages were far lower than the values that would be expected given the current and former smoking prevalence in the US: 13.8% and 22% respectively (2018).

As can be seen from this summary, the relation between smoking and COVID-19 is still uncertain and all statements on this issue are premature. More data and further research is still necessary to establish with confidence how current and former smokers can be affected by the development of COVID-19. In fact, as stated in the letter signed by experts sent to the FDA [10]:

"Other confounding factors need to be considered and the accuracy of the recorded smoking status needs to be determined before making any firm conclusions. As a result, the generalized advice on quitting smoking as a measure to improve health risk remains valid, but no recommendation can be currently made concerning the effects of smoking on the risk of hospitalization for COVID-19".

The effect of COVID-19 on vapers

Contrary to statements by misinformation sources that we have revised, there is simply no compelling evidence suggesting that vaping has the capacity to affect negatively the immune body response in order to produce the development and progression of the diseases caused by SARS-CoV-2 on e-cigarette users.

To better understand the possibility of a progression of infection leading to COVID-19 in vapers it is necessary to bear in mind that the overwhelming majority are smokers or ex-smokers, some of them dragging long histories of previous smoking (as stated in [11]). This smoking history is an important factor that could easily render as vulnerable a vaper who (say) smoked 20 or 30 years, even if he/she has been (typically) 2-3 years vaping without smoking.

Such vaper, theoretically, would be more susceptible to the complicated etiology of COVID-19. However, and in the absence of more evidence, this would not be an intrinsic effect of vaping, but of smoking, and thus it does not justify casting vaping as a risk factor on equal footing as smoking (as inferred from misleading statements by Glantz [1] and Bloomberg News [10] that have been recycled by the media in all countries).

In fact, bearing in mind that smokers improve their biomarkers and their respiratory and cardiovascular conditions when they switch completely to vaping, it is highly plausible (as Farsalinos argues [13]) that they would have a better prognosis under possible progression of COVID-19 if they no longer smoke, even if they have smoked before. This effect would be even more pronounced if it turns out that smoking is a determinant factor in the evolution to severe complications from COVID-19 but, as we have seen this is still far from proven.

E-liquids and SARS-CoV-2

It is also important to stress that there cannot be contagion of SARS-CoV-2 virus through e-liquids containing the virus. The survival ability of SARS coronavirus in environments seems to be relatively strong, but studies show thermal instability at 56°C [19]. Pathogens have been detected in e-liquids, however contagion of SARS-CoV-2 virus or any other pathogen is impossible from this route, as no pathogen can survive at temperatures of 180-220 °C involved in the aerosol generation from the e-liquid (they stop functioning as the macromolecules making them up fragment due to temperature).

Propylene glycol as a disinfectant

There has been mention in social networks that vaping might be protective in comparison with smoking on infection risks from COVID-19 [20], pointing out to experiments conducted in the 1940's in which propylene glycol (PG) vapor was used as environmental disinfectant that removes pathogens in hospitals, military barracks and other places. The experimental procedure was as follows [21,22]: pathogens (bacteria) were delivered in aqueous droplets from aerosolized cultures into the test chamber (the control being a chamber with pathogens without the PG aerosol). PG aerosol or PG vapor is then continuously supplied into the test chamber with a ventilator evenly dispersing it.
Tests for various ranges of ambient temperatures and relative humidity levels were conducted with various procedures to collect the bacteria. As the PG droplets in the aerosol rapidly evaporate, they release PG vapor at concentrations between 0.05 and 0.66 ppm or 200 to 3000 μg/m^3 (micrograms per cubic meter). The cleansing effect was most efficient at lower temperatures (in the range 15-37 degrees C) and under intermediate relative humidity levels (between 27% and 91%, peaking at around 42%), though the cleansing effect was still possible (though slower) at low relative humidity (10%) with sufficiently high PG vapor concentration.

The physical property explaining this effect [23] is the hygroscopic nature of PG vapor (not the aerosol droplets). As the PG droplets evaporate below air saturation, they release PG vapor molecules dispersing at high velocities and (because hygroscopicity) these molecules condense (are rapidly accreted) into the aqueous droplets containing the pathogens. The latter are eliminated by numerous fast collisions with the accreted PG molecules once the latter accumulate to form 70-80% of the droplets mass. This effect is no longer effective in both extremes of humidity: at 0% relative humidity the droplets evaporate very fast and at close to 100% relative humidity they condense, leading to a steady state which limits the available PG vapor (see [23] for details).

It is difficult to relate these highly controlled and idealized experiments to the erratic and highly variable conditions in vaping. For starters, pure PG (as aerosol or as vapor) in these experiments was supplied continuously and spread evenly, whereas in vaping the aerosol is a mixture of PG and other compounds (glycerol, VG, nicotine, with residual concentrations of mostly aldehydes), it is supplied into the surrounding air (when inhaled or exhaled) intermittently during puffs and spreads unevenly. Second, PG concentrations in vaping are very variable, rapidly changing with time and position. While PG concentrations in the experiments might match those of inhaled vapor, this disinfectant effect is unlikely to occur inside the respiratory tracts in which relative humidity is close to 100%. The exhaled environmental vaping aerosol might approach better the experimental conditions: PG/VG droplets evaporates rapidly, thus releasing PG vapor molecules, while relative humidity levels of 40-70% are not unrealistic, but PG vapor concentrations might be too low (chamber studies measure about 200 μg/m^3 (micrograms per cubic meter) [24,25] the lower limit concentrations in the experiments in [21,22]).

Moreover, given the observed reduction of respiratory infections in users of e-cigarettes [4,6,7], it is possible to speculate that at least in some occasions environmental conditions allowing for this effect could have occurred when vaping. The air cleansing experiments conducted in the 1940's only involved bacteria and the influenza virus, there is no way without experimental evidence to infer if this could happen with SARSCoV-2 and in the conditions of environmental e-cigarette aerosol.

Many viruses (and there is ample variation on this) cannot survive long time outside the protective envelope of a humid medium (the saliva droplets) or outside their host cell in the body tissues. However, it is not known if this is the case also with SARS-CoV-2**.**

Exhaled vapor as a possible path to spread SARS-Co-V2

A worrying theoretically possible path of infection of the SARS-Co-V2 virus is by breathing environmental aerosol (i.e. "vapor") exhaled by vapers.

Can this exhaled vapor spread SARS-CoV-2? As stated by Rosanna O'Connor, director of the Tobacco Alcohol and Drugs of Public Health England [26], currently there is no evidence of contagion through vapor exhaled by users of e-cigarettes. Professor Neil Benowitz of the University of California at San Francisco [27] declared that:

> *It is my understanding that exhaled e-cigarette vapor consists of very small particles of water, propylene glycol and glycerine and flavor chemicals, not droplets of saliva. The vaping aerosol evaporates very quickly, while particles that are emitted when coughing or sneezing are large particles that persist in the air for a relatively long period of time. Thus, I would not think that vapers present any risk of spreading COVID-19, unless they are coughing when they exhale the vapor.*

As a contrast to these declarations, the Scottish microbiologist Tom McLean, chief scientific advisor of the Nanotera Group, has claimed [28] that exhaled vapor can spread the virus, even comparing exposure to exhaled vapor as "being spit in your face". As we show below, McLean's statements are completely mistaken, while Professor Benowitz is right (though his description of contagion through aerosol droplets is oversimplified).

Airborne contagion of SARS-CoV-2 might occur through virus exposure associated to two types of airborne bioaerosols [29]: (1) large saliva droplets transmission from the exhaled breath of an infected person laying sufficiently close [30] and (2) larger distance transmission of buoyant smaller droplets or droplets nuclei (5-10 micron solid residues containing the pathogens that result when the droplets evaporate) when the infected person sneezes or coughs [31,32]. The exhaled vapor from an infected e-cigarette user is a nonbiological aerosol potentially carrying pathogens, as it is bound to carry into the environment buoyant droplets produced by atomization of secretions in the respiratory system of the vaper [30]. The rate of transmission of the virus strongly depends on the air flow dynamics of the different aerosols.

Normal sedentary breath is a nearly laminar flow in the upper respiratory tracts [29], giving rise to a low velocity turbulent flow when exhaled by the nose or mouth [33]. It spreads very few saliva droplets (roughly 1 droplet per cubic centimeter in healthy subjects) whose size peaks at 1 micron but grow from hygroscopic coagulation, with droplets smaller than 5 microns rapidly evaporating. The droplets are transported short distances within the personal breathing zone [30,33].

As a contrast, sneezing is a very rapid multiphase explosive turbulent flow whose dynamics is quite elaborate [31,32]: it can spread up to millions of droplets typically falling to the ground at 2 meters but smaller droplets or droplets nuclei remaining buoyant for long times and possibly travelling by diffusion and up to 6-8 meters away (even upwards by buoyant convection). Coughing is also an explosive turbulent flow that can spread thousands of droplets.

The exhaled vapor is a diluted aerosol made almost exclusively of very light and rapidly moving "particles" (propylene glycol and glycerol PG/VG droplets) suspended in a gaseous medium of nearly the same chemical composition. Since most inhaled aerosol is absorbed and exhalation favors hyperfine droplets, their mean diameters are in the 100-300 nm range [34,35] (one nanometer nm is 1 billionth of a meter) and they evaporate very rapidly (20 seconds per puff). The gas is supersaturated and the whole aerosol (gas and droplets) disperses completely in less than 2-3 minutes with some droplets impacting walls or falling to the ground.

Chamber experiments reveal that the exhaled cloud does not transport the droplets for large distances: at 1.5 meters from the exhalation source they are barely detectable, with their particle number density almost indistinguishable from background values for all particle sizes (submicron, $PM_{2.5}$ and PM_{10}). For low powered devices this distance is likely to be less than 1 meter.

The flow associated with exhaled vapor (or smoke) is comparable to that of gentle air blowing or mouth breathing, characterized by slightly larger velocities than breathing through the nose, but much slower than coughing or sneezing [30,33,36]. An infected vaper will exhale as much saliva droplets as these low velocity expiration mechanisms, but their numbers are far fewer than the rapidly evaporating PG/VG droplets in the e-cigarette aerosol. As a consequence, the saliva droplets have a negligible effect on the flow dynamics even if they experience hygroscopic growth and smaller ones evaporate with their nuclei staying buoyant for much longer times. Since the cloud of PG/VG droplets of exhaled vapor are barely detectable at 1.5 meters (less for low powered devices), it is extremely unlikely that the few pathogen containing saliva droplets dragged by the exhaled flow of an infected vaper would be transported as far as these distances even if they remain buoyant. As a contrast, sneezing and coughing are able to transport large numbers of airborne pathogens for large distances.

However, the frequency of vaping must be considered besides the difference between the dynamics of the involved aerosols. An infected vaper will spread into the environment a small number of virus (a few droplets per exhalation [30]) **and only** when he/she vapes (typically 200 times per day), whereas normal breathing by any person (whether a vaper or not) implies a flow of a similar amount of virus but delivered constantly. Coughing and sneezing are also intermittent virus flows, but they spread a large number of virus only in each expiration event. In fact, breath is a far more serious contagion factor than these explosive intermittent events: two hours of normal breathing spreads more droplets than coughing 100 times [36]. Paradoxically, an infected vaper will spread much more virus from his/her regular breathing than from his/her vaping.

Evidently, Rosanna O'Connor and Professor Benowitz are right: exhaled vapor as a risk of SARS-CoV-2 contagion does not require more strict protective measures than those contemplated for non-vapers.

The associated flow dynamics suggests that keeping the same 1.5 to 2 meters "social separation" distance recommended for non-vapers should prevent any contagion from an infected vaper. Aerosol dynamics fully corroborates that microbiologist Tom McLean is mistaken: contagion risk from exhaled vapor is not even remotely comparable to the contagion risk from sneezing or coughing.

Contagion of COVID-19 on surfaces

One of the mechanisms of contagion of viruses is physical contact with surfaces where they lie and then to touch the mouth, nose or eyes. It is known that viruses can survive on surfaces and that typically they lie inside thin liquid films that form when the saliva droplets impact the surfaces when transported by sneezing, coughing or sneezing [29]. This type contagion is thus theoretically possible from saliva droplets containing SARS-CoV-2 dragged by exhaled vapor and impacting the surface, but the risk should be comparable to that from droplets impacting a sufficiently close surface from normal breath.

How long can the virus they survive? It depends on the virus: it was reported that SARS-CoV-2 remains stable, viable and functional for several hours and (in some materials) up to 3 days [37], but this comes from extremely idealized laboratory experiments that bear no relation with the realistic deposition of a virus on a surface: the researchers inoculate the virus in a host liquid protective solution on the surface and afterwards verify its viability. In the case of that SARS-CoV-2 it is not known how much time the virus can survive on surfaces under realistic conditions, saliva, exposure to real-life humidity, temperature and solar radiation conditions in different environments and if they can survive without their protective envelope.

RECOMMENDATIONS TO VAPERS

On the basis of the information provided, we recommend

- If you vape do not revert to smoking (if you are a dual user try to become an exclusive vaper). Regardless of the relationship between smoking and the development of COVID-19, smoking remains a leading cause of illness and death.
- If you enjoy vaping and do not smoke, to quit vaping must be a personal choice not an obligation
- Be discreet and do not call unwanted attention (bear in mind that these are difficult times and that a lot of non-vapers have been exposed to a lot of misinformation).
- Avoid big clouds in public at all costs (even outdoors).
- Use low powered devices whenever possible and when others are around. The risk of spreading the virus with discrete vaping in low powered devices is roughly equivalent to the risk of spreading it through normal sedentary breathing.
- Avoid vaping in enclosed public spaces and try to keep at least 2 meters distance from others when vaping outdoors.
- Maintain extreme hygiene measures with the devices and disinfect them regularly. We keep them on hand for a long time and put them in our mouths dozens or even hundreds of times a day.

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