Conseil
Supérieur de la Santé

SARS-COV-2 ET L'UTILISATION DE TUNNELS D'OZONE POUR « DÉSINFECTER » LES CHARRIOTS ET LES CLIENTS

AVRIL 2020
CSS N° 9593

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Service public Fédéral de la Santé publique, de la Sécurité de la Chaîne alimentaire et de l'Environnement

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Conseil Supérieur de la Santé. SARS-CoV-2 et l'utilisation de tunnels d'ozone pour « désinfecter » les charriots et les clients. Bruxelles: CSS; 2020. Avis n° 9593.

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Conseil
Supérieur de la Santé

AVIS DU CONSEIL SUPERIEUR DE LA SANTE N° 9593

SARS-CoV-2 et l'utilisation de tunnels d'ozone pour « désinfecter » les charriots et les clients

Version validée par le Bureau du Collège
29 avril 2020[1]

I INTRODUCTION ET QUESTION

Ce 23 avril, le Conseil Supérieur de la Santé (CSS) a reçu une demande urgente de réponse à la question parlementaire portant la référence 55005338C formulée par Madame Sophie Rohonyi (DéFI) et adressée à Madame la ministre Maggie De Block.

La question est formulée comme suit :

> Beaucoup d'employeurs sont à l'affût de solutions visant à reprendre leur activité commerciale tout en assurant la protection du personnel et des clients. Il faut cependant rester prudents, et ne pas donner à la population l'illusion d'une protection efficace.
>
> Une grande surface a ainsi installé un tunnel d'ozone. Il s'agit d'une sorte de sas dans lequel les clients et leurs caddies passent 9 secondes dans une brume d'ozone censée tuer virus et bactéries. Les petites gouttes projetées seraient composées d'eau et d'ozone. Aucun produit chimique ne serait ajouté. L'investissement serait de l'ordre de 10.000 euros.
>
> Madame la ministre, j'aimerais connaître votre avis sur ces tunnels car, s'ils sont efficaces, ils pourraient être recommandés dans le cadre du déconfinement, par exemple à l'entrée des gares, des grandes surfaces, des entreprises etc. A contrario, s'ils sont inefficaces, il me semble utile de le faire savoir par une mise en garde, pour ne pas entretenir une illusion de protection au point de négliger les règles d'hygiène et de distanciation sociale.
>
> Ma question est dès lors simple :
> Cette piste a-t-elle été étudiée d'un point de vue scientifique ? Si oui, quelles conclusions en avez-vous tiré ?

[1] Le Conseil se réserve le droit de pouvoir apporter, à tout moment, des corrections typographiques mineures à ce document. Par contre, les corrections de sens sont d'office reprises dans un erratum et donnent lieu à une nouvelle version de l'avis.

Étant donné l'extrême urgence de la demande, celle-ci a été directement transmise à une sélection d'experts issus des domaines de l'infectiologie, de la toxicologie et de l'écologie humaine. L'avis a été validé par le Bureau du Collège du CSS.

Liste des abréviations

CSS	Conseil supérieur de la Santé
MDRO	Bactéries multi-résistantes aux antibiotiques
SARS	*Severe Acute Respiratory Syndrome*

Mots clés et MeSH *descriptor terms*[2]

Mesh terms*

Keywords	**Sleutelwoorden**	**Mots clés**	**Schlüsselwörter**
SARS-CoV-2	SARS-CoV-2	SARS-CoV-2	SARS-CoV-2
Ozone tunnel	ozontunnel	Tunnel d'ozone	Ozontunnel
Disinfection	ontsmetting	Désinfection	Desinfektion
Respiratory tract	luchtwegen	Voies respiratoires	Atemwege

MeSH (Medical Subject Headings) is de thesaurus van de NLM (National Library of Medicine) met gecontroleerde trefwoorden die worden gebruikt voor het indexeren van artikelen voor PubMed http://www.ncbi.nlm.nih.gov/mesh.

II AVIS

Avis

En l'absence de preuves scientifiques évidentes et avérées de l'efficacité du système évoqué dans cette question parlementaire pour « désinfecter » les clients et le personnel des supermarchés (ou les chariots) et au vu du risque, même à des doses très faibles, de manifestations de toxicité (notamment pulmonaire) pour les personnes les plus fragiles, **le CSS émet un avis fortement défavorable à la mise en circulation et la généralisation de cette technique en Belgique**. **Une interdiction devrait même être envisagée par les autorités. Une communication claire à la population et aux secteurs qui désirent tester cette technique (probablement pour des raisons marketing) doit être rapidement assurée afin d'éviter des investissements inutiles.**

Argumentation

L'ozone (à l'état liquide ou gazeux) est un puissant oxydant qui est déjà utilisé dans de nombreuses applications pour la désinfection des locaux, de l'eau, etc. Ses propriétés oxydantes peuvent détruire les microorganismes dont les coronavirus comme le SARS (*Severe Acute Respiratory Syndrome*) (peu de données sur le SARS-CoV-2) mais peuvent également interagir avec les tissus humains.

Une utilisation de l'ozone gazeux pendant une durée (au moins 10 minutes) et une concentration élevée (10 à 20 ppm) est incompatible avec une présence humaine. Elle créerait des lésions (irritations) sur les muqueuses des yeux et des poumons par exemple. L'ozone gazeux lié à la pollution est également un facteur aggravant des pathologies pulmonaires et

[2] Le Conseil tient à préciser que les termes MeSH et mots-clés sont utilisés à des fins de référencement et de définition aisés du scope de l'avis. Pour de plus amples informations, voir le chapitre « méthodologie ».

de l'asthme. L'ozone, tel qu'il est fréquemment présent dans l'air ambiant, a très probablement des effets pathogènes importants sur la santé humaine, à savoir des maladies respiratoires chroniques, des cancers du poumon et des maladies cardiovasculaires. Bien que les études épidémiologiques n'aient pas toutes démontré les effets de l'ozone sur la santé, les preuves sont convaincantes. Cependant, l'ozone présente également des caractéristiques mutagènes et délétères pour l'ADN évidentes. Les résultats des expériences sur des cellules in vitro et sur des animaux ne concordent pas toujours, sans doute en raison de l'extrême instabilité de l'ozone, mais il est clair que l'ozone a des propriétés mutagènes et donc potentiellement cancérigènes.

Une utilisation à des seuils permis par les autorités sanitaires pour l'exposition de la population à l'ozone est probablement inefficace pour tuer les microorganismes. Ceci n'est pas le cas avec les désinfectants, détergents classiques qui sont eux efficaces sur le SARS-CoV-2 et d'usage plus courant.

L'avis 9277 du CSS, qui traite des recommandations en matière de prévention, maîtrise et prise en charge des patients porteurs de bactéries multi-résistantes aux antibiotiques (MDRO) dans les institutions de soins (CSS 2019), mentionne, dans le chapitre dédié aux méthodes alternatives pour le nettoyage et la désinfection des surfaces sous les « systèmes de décontamination automatisés », que "Des systèmes automatisés de désinfection des locaux ont été mis au point pour la décontamination des objets et des surfaces. Ces systèmes utilisent différents biocides, tels que le peroxyde d'hydrogène (H_2O_2), l'acide peracétique, l'ozone, ainsi que la vapeur d'eau ou les UV. **Cette technologie « *no touch* » présente l'avantage qu'elle est moins dépendante de son utilisateur pour obtenir une désinfection appropriée en termes d'homogénéité de l'ensemble de la surface et un temps de contact optimal du produit spécifique appliqué** (Anderson *et al.*, 2011, Matlow *et al.*, 2012). Bien que ces systèmes permettent d'obtenir une décontamination optimisée, **ils ne peuvent pas se substituer au nettoyage quotidien**. Les souillures organiques doivent être éliminées avant de pouvoir appliquer les désinfectants. En outre, ces systèmes ne peuvent généralement pas être utilisés avant la sortie d'hospitalisation du patient parce que ces produits sont trop toxiques ou comportent un risque au plan de la de sécurité (Dancer, 2014). Les principales différences entre les systèmes utilisant des UV-C et ceux à base de peroxyde d'hydrogène sont que les UV-C ne peuvent pas éliminer une biocharge sur une surface qui ne se trouve pas dans le prolongement direct du spectre d'émission des UV. La vapeur de peroxyde d'hydrogène et d'acide peracétique (Mana T. *et al*., 2017) permet une meilleure élimination des spores bactériennes. Jusqu'à présent, ces systèmes ont principalement démontré leur efficacité pour la désinfection des surfaces sans recherche spécifique quant à leur impact sur les agents pathogènes aéroportés (Dancer, 2014 ; Mana T. *et al.*, 2017). »

Considérant la très grande réactivité de l'ozone avec toutes les surfaces, il est peu probable que cette technique ait un impact majeur sur la pollution environnementale (mis à part la consommation énergétique liée au générateur). Ceci n'est plus vrai si l'on combine l'ozone à d'autres désinfectants comme le chlore par exemple.

La toxicité de l'ozone gazeux nécessite des mesures de sécurité plus strictes comme sa détection dans l'atmosphère ambiant par un ozonomètre ; celui-ci est en mesure d'arrêter la production du générateur dès que le seuil de 0,3 ppm dans l'air est dépassé. Une utilisation en extérieur et via de l'ozone dissous dans l'eau est probablement moins dangereuse que dans un espace clos sous forme gazeuse.

La consommation d'énergie due à la production d'ozone par décharge électrique est un autre facteur limitant à prendre en compte en cas d'extension large de cette technique.

Les propriétés oxydantes de l'ozone sont bien connues sur une multitude de microorganismes. Le SARS-CoV-2 étant relativement sensible à certains facteurs physiques et chimiques, il est probable que l'ozone ait un effet sur le SARS-CoV-2. Néanmoins, seules 2 publications sont ressorties à ce sujet (non liées à l'utilisation de tunnels d'ozone) dans une recherche PubMed. La conclusion est qu'il faudrait des études cliniques complémentaires pour confirmer l'efficacité et l'intérêt de l'utilisation de l'ozone dans le cadre de la prise en charge médicale des personnes infectées par le SARS-CoV-2.

L'utilisation de l'ozone chez l'homme comme traitement médical est controversée, avec des partisans et des opposants absolus. Plus récemment, l'un des principaux défenseurs (Robert Jay Rowen) a publié un article sur l'utilisation de l'ozone pour la lutte contre le SARS-CoV-2 (doi : 10.4103/2045-9912.273962). Bien que son plaidoyer (et celui d'autres personnes) soit principalement basé sur la ressemblance entre le SRAS et le SARS-CoV-2 et sur le fait que le virus du SRAS peut être détruit in vitro par l'ozone, ces affirmations ne reposent pas sur des informations médicales solides. Les effets positifs présumés sur les patients atteints d'Ebola ont été décrits par Robert Rowan. Néanmoins, aucune référence n'est trouvée dans la littérature médicale classique. Les opposants sont très clairs : l'ozone est un oxydant trop puissant pour une application humaine. En tant que telle, l'ozonothérapie est généralement considérée comme une "thérapie alternative" dans la pratique médicale et devrait donc être évitée ou très strictement contrôlée avec des dangers bien réels pour la santé.

L'une des méthodes de traitement à l'ozone consiste en l'administration intraveineuse d'une solution iso-osmotique saturée d'ozone qui a un effet toxique sur de nombreux organes. Cependant, et bien qu'il faille éviter l'administration d'ozone par voie intraveineuse, cette méthode est moins dangereuse que l'administration d'eau de Javel par voie intraveineuse, telle qu'elle est évoquée aux États-Unis dans la presse. Il faut être clair : cette information n'est pas un plaidoyer en faveur du traitement à l'ozone dans la lutte contre le SARS-CoV-2 pour la population générale.

En ce qui concerne l'utilité des tunnels désinfectants (ozone et autres) dans le cadre d'une épidémie virale respiratoire, on peut lire ceci :

- " *But their use to stop the spread of Covid-19 as yet do not have the scientific evidence of effectiveness and definitely should not be used for the general public in a community or workplace setting*" - Prof Datuk Dr Lokman Hakim Sulaiman, Pro-Vice Chancellor (Research), Professor of Public Health, International Medical University.
- " *The technology was employed in Spain and Italy, but well after the coronavirus spread through the population* ".
- Elle a été employée également au Mexique.

Elle ne semble pas avoir été utilisée de manière importante dans d'autres pays touchés par l'épidémie au Covid-19

Considérant la manière dont la technique est utilisée dans d'autres pays (passage de quelques secondes des personnes et des chariots sous le tunnel), nous supposons que si les mains ne sont pas propres avant, si la barre du chariot n'est pas propre avant et que les personnes passent dans le tunnel en poussant leur chariot, le virus est moins accessible à l'ozone sous les mains et sur la barre du chariot. Dès lors, le risque subsiste dans le magasin si les autres mesures ne sont pas strictement respectées. Si celles-ci sont effectivement respectées, le tunnel d'ozone apparaît plus comme une mesure marketing qu'un réel outil efficace en termes de santé publique.

En cette période de pénurie et de tension financière généralisée, l'argent dépensé pour cette technique serait dès lors, plus utile et efficace pour mettre en œuvre les techniques qui ont démontré de manière claire et scientifiquement fondée leur utilité.

Conclusions et recommandations

Dans le cadre cette question parlementaire sur l'utilité, l'efficacité et l'innocuité pour la population et l'environnement des tunnels d'ozone pour la lutte contre le SARS-CoV-2, le Conseil Supérieur de la Santé conclut et recommande la position suivante :

En l'absence de preuves scientifiques évidentes et avérées de l'efficacité du système évoqué dans cette question parlementaire pour « désinfecter » les clients et le personnel des supermarchés (ou les chariots) et, au vu du risque, même à des doses très faibles, de manifestations de toxicité (notamment pulmonaire) pour les personnes les plus fragiles,

le CSS émet un avis fortement défavorable à la mise en circulation et la généralisation de cette technique en Belgique. **Une interdiction devrait même être envisagée par les autorité. Une communication claire à la population et aux secteurs qui désirent tester cette technique (probablement pour des raisons marketing) doit être rapidement assurée afin d'éviter des investissements inutiles.**

L'installation de tunnels à ozone à l'entrée des supermarchés dans lesquels les chariots, avec les clients ou le personnel, seraient exposés à l'ozone pendant 9 secondes à des concentrations utiles et efficaces (10 à 20 ppm) pour neutraliser le SARS-CoV-2 présent, **n'est pas une option soutenable du point de vue de la protection de la santé humaine**.

L'installation de tunnels à ozone avec un passage rapide et des concentrations faibles d'ozone « compatibles » avec la protection de la santé humaine **sera quant à elle inefficace pour neutraliser le SARS-CoV-2**.

De plus, même avec ces temps de passage courts et des faibles doses, les risques d'irritation des muqueuses (notamment pulmonaires) sont réels surtout pour les personnes les plus fragilisées, allergiques (période actuelle en Belgique) ou asthmatiques. Les propriétés cancérigènes de l'ozone pour l'homme plaident également en faveur d'une interdiction de ces techniques appliquées à l'être humain et à grande échelle.

De plus,

Avec les règles d'application dans le pays et les règles d'hygiène des mains et des surfaces renforcées, **il n'y a pas d'évidence scientifique claire indiquant que le fait d'aller faire ses courses soit une voie de contamination et de transmission importante du virus SARS-CoV-2.**

Cette technique ne peut pas, par définition, éliminer le portage du virus dans le corps (nez, bouche, poumon) et donc sa propagation par voie aérienne par le client ou le personnel asymptomatique, à l'intérieur du lieu soit disant « protégé pendant 60 minutes » grâce au tunnel d'ozone. **Les personnes infectieuses le seront toujours après le passage du tunnel**.

Les tunnels d'ozone procurent donc un faux sentiment de sécurité et pourraient réduire l'attention sur les règles qui sont-elles efficaces, simples, peu coûteuses, peu énergivores et surtout non toxiques.

Les recommandations classiques de confinement en cas de symptômes, de distanciation physique, de concentration maximale de personnes dans un espace clos, d'hygiène des mains (avant de rentrer dans un magasin et pendant ses courses), de minimisation de la propagation lors de la toux, du port du masque, etc. sont efficaces et probablement suffisantes pour limiter le risque à un niveau acceptable au vu de la situation belge actuelle.

Le renforcement de l'hygiène des surfaces (des chariots de courses quand ils sont imposés par exemple) et dans tout le magasin sont efficaces et prioritaires.

En cas d'utilisation de techniques à **l'ozone gazeux** pour la désinfection de locaux vides (médicaux, industriels, transports publics, etc.), les concentrations et durées d'exposition sont beaucoup plus importantes et **incompatibles, pour être efficaces, avec une exposition humaine directe en même temps**. Ces techniques permettent d'obtenir une décontamination optimisée mais, elles ne peuvent pas se substituer au nettoyage quotidien des surfaces. **Les souillures organiques doivent être éliminées avant de pouvoir appliquer les désinfectants de type ozone gazeux ou autres**.

La période actuelle de sensibilité importante de la population non immunisée à l'infection au SARS-CoV-2 et l'augmentation des allergies et de l'asthme en cette période également ne nous semblent pas compatibles avec une exposition renforcée à un agent chimique irritant pour les muqueuses et les voies respiratoires.

III COMPOSITION DU GROUPE DE TRAVAIL

La composition du Bureau et du Collège ainsi que la liste des experts nommés par arrêté royal se trouvent sur le site Internet du CSS (page : Qui sommes-nous).

Tous les experts ont participé ***à titre personnel*** au groupe de travail. Leurs déclarations générales d'intérêts ainsi que celles des membres du Bureau et du Collège sont consultables sur le site Internet du CSS (page : conflits d'intérêts).

Les experts suivants ont participé à l'élaboration et à l'approbation de l'avis. Le secrétariat scientifique a été assuré par Jean-Jacques DUBOIS, Roland HUBNER, Fabrice PETERS et Marleen VAN DEN BRANDE.

FRAEYMAN Norbert	Toxicologie et toxicologie de l'environnement	UGent
HENS Luc	Ecologie humaine	VITO
VAN LAETHEM Yves	Infectiologie	UMC Sint-Pieter
VAN LAREBEKE Nicolas	Toxicologie	VUB/UGent

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https://www.ncbi.nlm.nih.gov/pubmed/32228825

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V ANNEXES

1. Extraits des références scientifiques

https://www.epa.gov/indoor-air-quality-iaq/ozone-generators-are-sold-air-cleaners

1 **How is Ozone Harmful?**

2 **The same chemical properties that allow high concentrations of ozone to react with organic material outside the body give it the ability to react with similar organic material that makes up the body, and potentially cause harmful health consequences. When inhaled, ozone can damage the lungs. Relatively low amounts can cause chest pain, coughing, shortness of breath and throat irritation. Ozone may also worsen chronic respiratory diseases such as asthma and compromise the ability of the body to fight respiratory infections. People vary widely in their susceptibility to ozone. Healthy people, as well as those with respiratory difficulty, can experience breathing problems when exposed to ozone. Exercise during exposure to ozone causes a greater amount of ozone to be inhaled, and increases the risk of harmful respiratory effects. Recovery from the harmful effects can occur following short-term exposure to low levels of ozone, but health effects may become more damaging and recovery less certain at higher levels or from longer exposures (US EPA, 1996a, 1996b).**

3 **Are Ozone Generators Effective in Controlling Indoor Air Pollution?**

4 **Available scientific evidence shows that at concentrations that do not exceed public health standards, ozone has little potential to remove indoor air contaminants.**

https://www.who.int/emergencies/diseases/novel-coronavirus-2019/advice-for-public/myth-busters

Can spraying alcohol or chlorine all over your body kill the new coronavirus?

No. Spraying alcohol or chlorine all over your body **will not kill viruses that have already entered your body**. Spraying such substances can be harmful to clothes or mucous membranes (i.e. eyes, mouth). Be aware **that both alcohol and chlorine can be useful to disinfect surfaces, but they need to be used under appropriate recommendations.**

https://www.ozonetech.com/sites/default/files2/pdf/Ozone_disinfection_of_SARS_Contaminated_Areas.pdf

https://www.ozomax.com/applications/disinfection.php

https://www.ncbi.nlm.nih.gov/pubmed/32303365

Abstract

Pneumonia caused by coronavirus, which originated in Wuhan, China, in late 2019, has been spread around the world already becoming a pandemic. Unfortunately, there is not yet a specific vaccine or effective antiviral drug for treating COVID-19. Many of these patients deteriorate rapidly and require intubation and are mechanically ventilated, which is causing the collapse of the health system in many countries due to lack of ventilators and intensive care beds. In this document we review two simple adjuvant therapies to administer, without side effects, and low cost that could be useful for the treatment of acute severe coronavirus infection associated with acute respiratory syndrome (SARS-CoV-2). VitaminC, a potent antioxidant, has emerged as a relevant therapy due to its potential benefits when administered intravenous. The potential effect of vitaminC in reducing

inflammation in the lungs could play a key role in lung injury caused by coronavirus infection. Another potential effective therapy is ozone: it has been extensively studied and used for many years and its effectiveness has been demonstrated so far in multiples studies. Nevertheless, our goal is not to make an exhaustive review of these therapies but spread the beneficial effects themselves. Obviously clinical trials are necessaries, but due to the potential benefit of these two therapies we highly recommended to add to the therapeutic arsenal.

https://www.ncbi.nlm.nih.gov/pubmed/32228825

Abstract

SARS-CoV-2, also referred to as CoV-19, is an RNA virus which can cause severe acute respiratory diseases (COVID-19), with serious infection of the lower respiratory tract followed by bronchitis, pneumonia and fibrosis. The severity of the disease depends on the efficiency of the immune system which, if it is weak, cannot stem the infection and its symptoms. The new CoV-19 spreads in the population at a rate of 0.8-3% more than normal flu and mostly affects men, since immune genes are more expressed on the X chromosome. If CoV-19 would spread with a higher incidence rate (over 10%), and affect the people who live in closed communities such as islands, it would cause many more deaths. Moreover, people from the poorest classes are most at risk because of lack of health care and should be given more assistance by the competent authorities. To avoid the aggravation of CoV-19 infection, and the collapse of the health system, individuals should remain at home in quarantine for a period of approximately one month in order to limit viral transmission. In the case of a pandemic, the severe shortage of respirators and protective clothing, due to the enormous demand and insufficient production, could lead the CoV-19 to kill a large number of individuals. At present, there is no drug capable of treating CoV-19 flu, the only therapeutic remedies are those aimed at the side effects caused by the virus, such as inflammation and pulmonary fibrosis, recognized as the first causes of death. One of the COVID-19 treatments involves inhaling a mixture of gaseous hydrogen and oxygen, obtaining better results than with oxygen alone. It was also noted that individuals vaccinated for viral and/or bacterial infectious diseases were less likely to become infected. In addition, germicidal UV radiation "breaks down" the oxygen O2 which then aggregate into O3 (ozone) molecules creating the ozone layer, capable of inhibiting viral replication and improving lung respiration. All these precautions should be taken into consideration to lower the risk of infection by CoV-19. New anti-viral therapies with new drugs should also be taken into consideration. For example, microbes are known to bind TLR, inducing IL-1, a pleiotropic cytokine, highly inflammatory, mediator of fever and fibrosis. Therefore, drugs that suppress IL-1 or IL-1R, also used for the treatment of rheumatoid arthritis are to be taken into consideration to treat COVID-19. We strongly believe that all these devices described above can lead to greater survival and. therefore, reduction in mortality in patients infected with CoV-19.

There are more than 17 scientific studies that show Ozone **gas** is able to destroy the SARS **coronavirus**.

https://www.josam.org/josam/article/view/35

The ozone concentrations necessary for effective virucidal inactivation remain sparsely reported, but have been estimated for this application, based upon extrapolations of the available scientific data. **The required ozone concentrations of 10 to 20 ppm are easily achieved and maintained for the necessary period of at least 10 minutes by continuous or intermittent operation of the ozone generator**. There are important safety and health factors to consider when using ozone, but otherwise we conclude that it can be used widely, on a large scale, as an improvised disinfectant, specifically for inactivating viruses.

http://www.china.org.cn/opinion/2020-02/26/content_75747237_4.htm
Ozone **[RH: gas]**, **though highly effective for sterilization and disinfection, will cause discomfort, or irritate mucous membranes, when it reaches a certain concentration level. Therefore, it is mainly used in unmanned environment.**

https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0035108
"Exposure to oxidant air pollution is associated with **increased respiratory morbidities and susceptibility to infections**."

https://clinmedjournals.org/articles/jide/journal-of-infectious-diseases-and-epidemiology-jide-6-113.php?jid=jide

https://eu.courier-journal.com/story/news/politics/2020/04/24/coronavirus-ozone-therapy-falsely-touted-covid-19-treatment/3021728001/ <= law enforcement !

Le pouvoir mutagène de l'ozone

N. Li, H. Yang, Z. Fang, P. Y. Wang, J. Han, L. Tian, J. Yan, Z. G. Xi, and X. H. Liu. [Effects of acute ozone exposure on genotoxicity of lung cells in rats]. *Zhongguo Ying.Yong.Sheng Li Xue.Za Zhi.* 35 (2):97-100, 2019.

OBJECTIVE: To clarify the genotoxicity induced by acute exposure of ozone with different concentrations on pulmonary cells in rats. METHODS: Thirty-six Wistar rats were randomly divided into control group (filtered air exposure) and ozone exposure group (0.12 ppm, 0.5 ppm, 1.0 ppm, 2.0 ppm, 4.0 ppm) with 6 in each group. After rats were exposed to different concentrations of ozone for 4 h, lung tissues were taken and single cells were isolated. Then, 8-hydroxydeoxyguanosine (8-OHdG) was quantitatively detected by enzyme-linked immunosorbent assay. Comet assay, micronucleus test and DNA- protein cross-linking assay were used to analyze DNA and chromosome damages. RESULTS: Compared with the control group, the content of 8-OHdG in lung tissue was increased significantly from the ozone exposure concentration of 0.12 ppm, reaching the highest value at 0.5 ppm. With the increase of ozone exposure concentration, the tail rate of comets was increased gradually, and there was a significant dose-effect relationship. The cross-linking rate of DNA- protein was increased first and then was decreased with a maximum value at 2.0 ppm group. Although the micronucleus rate of lung cells showed an upward trend, there was no significant difference compared with the control group. CONCLUSION: Acute exposure of ozone at low concentrations (0.12 ppm) could lead to DNA damage in the pulmonary cells of rats, while no significant chromosome damage was found even in the group with ozone concentration reached to 4 ppm

S. A. Jorge, C. F. Menck, H. Sies, M. R. Osborne, D. H. Phillips, A. Sarasin, and A. Stary. Mutagenic fingerprint of ozone in human cells. *DNA Repair (Amst)* 1 (5):369-378, 2002.

Ozone is an important factor in urban pollution and represents a major concern for human health. The chemical reactivity of ozone toward biological targets and particularly its genotoxicity supports a possible link between exposure and cancer risk, but no molecular data exist on its mutagenic potential in human cells. Using a shuttle vector, we showed that ozone is indeed a potent mutagen and we characterized the mutation spectrum it produced in human cells. Almost all mutations are base substitutions, essentially located at G:Cs (75%), typical of reactive oxygen species (ROS), but occurring in a specific pattern, i.e. a similar extent of GC:TA (28%), GC:CG (23%) and GC:AT (23%). The targeted distribution of mutations and identification of hotspot sequences define the first molecular fingerprint of mutations induced by ozone in human cells. Possible applications derived from our results with respect to ozone genotoxicity should help determining quantifiable biomarkers of ozone exposure in human health, especially for carcinogenesis

S. az-Llera, Y. Gonzalez-Hernandez, E. A. Prieto-Gonzalez, and A. Azoy. Genotoxic effect of ozone in human peripheral blood leukocytes. *Mutat.Res.* 517 (1-2):13-20, 2002.

The genotoxic effect of ozone was studied in human leukocytes in vitro, using the single cell gel electrophoresis (SCGE) assay. Cell treatment for 1 h at 37 degrees C with 0.9-5.3 mM O(3) resulted in a dose-dependent increase of DNA damage, comparable to that induced by 4-40 mM of H(2)O(2), used as a positive control. This effect of ozone was reversed by post-treatment incubation of the cells for 45-90 min at 37 degrees C, and prevented by pre-incubation of the cells with catalase (20 microg/ml). These results demonstrate that O(3) induces DNA-damage in primary human leukocytes. The damage is rapidly repaired, and probably mediated by the formation of H(2)O(2)

C. Borek, A. Ong, and M. Zaider. Ozone activates transforming genes in vitro and acts as a synergistic co-carcinogen with gamma-rays only if delivered after radiation. *Carcinogenesis* 10 (8):1549-1551, 1989.

An earlier study indicated that ozone (O3), a major pollutant in our atmosphere, acts as a carcinogen as well as a synergistic co-carcinogen with radiation in cultured hamster embryo cells and in mouse C3H10T1/2 cells. In this investigation we further characterize the oncogenic action of ozone, alone or in combination with radiation, on C3H10T1/2 cells with particular emphasis on transformation produced by different temporal patterns of dose delivery of these two agents and low dose effects. We report that ozone-induced transformation involves the activation of dominant transforming genes, thereby indicating that DNA is a target in ozone induced carcinogenesis. We also report that ozone (5 p.p.m. for 5 min) acts as a synergistic co-carcinogen only if delivered after radiation (4 Gy or gamma-rays); when cells are exposed to ozone prior to radiation no enhanced rates of transformation are observed. Our findings also show that ozone at a low dose of 1 p.p.m. (for 5 min) does not act as a carcinogen but does interact as a co-carcinogen with ionizing radiation. The data indicate that the dose and sequence in which ozone and radiation are delivered have important implications for the putative carcinogenic effects of these two agents, a factor that heretofore has not been recognized.

Les effets de l’ozone sur la santé humaine

I. Manisalidis, E. Stavropoulou, A. Stavropoulos, and E. Bezirtzoglou. Environmental and Health Impacts of Air Pollution: A Review. *Front Public Health* 8:14, 2020.

One of our era's greatest scourges is air pollution, on account not only of its impact on climate change but also its impact on public and individual health due to increasing morbidity and mortality. There are many pollutants that are major factors in disease in humans. Among them, Particulate Matter (PM), particles of variable but very small diameter, penetrate the respiratory system via inhalation, causing respiratory and cardiovascular diseases, reproductive and central nervous system dysfunctions, and cancer. **Despite the fact that ozone in the stratosphere plays a protective role against ultraviolet irradiation, it is harmful when in high concentration at ground level, also affecting the respiratory and cardiovascular system**. Furthermore, nitrogen oxide, sulfur dioxide, Volatile Organic Compounds (VOCs), dioxins, and polycyclic aromatic hydrocarbons (PAHs) are all considered air pollutants that are harmful to humans. Carbon monoxide can even provoke direct poisoning when breathed in at high levels. Heavy metals such as lead, when absorbed into the human body, can lead to direct poisoning or chronic intoxication, depending on exposure. Diseases occurring from the aforementioned substances include principally respiratory problems such as Chronic Obstructive Pulmonary Disease (COPD), asthma, bronchiolitis, and also lung cancer, cardiovascular events, central nervous system dysfunctions, and cutaneous diseases. Last but not least, climate change resulting from environmental pollution affects the geographical distribution of many infectious diseases, as do natural disasters. The only way to tackle this problem is through public awareness coupled with a multidisciplinary approach by scientific experts; national and international organizations must address the emergence of this threat and propose sustainable solutions

N. Wang, K. Mengersen, S. Tong, M. Kimlin, M. Zhou, L. Wang, P. Yin, Z. Xu, J. Cheng, Y. Zhang, and W. Hu. Short-term association between ambient air pollution and lung cancer mortality. *Environ.Res.* 179 (Pt A):108748, 2019.

RATIONALE: Long-term exposure to air pollution has been associated with increased lung cancer incidence and mortality. However, the short-term association between air pollution and lung cancer mortality (LCM) remains largely unknown. METHODS: We collected daily data on particulate matter with diameter <2.5mum (PM2.5), particulate matter with diameter<10mum (PM10), sulfur dioxide (SO2), and ozone (O3), and LCM in three of the biggest cities in China, i.e. Beijing, Chongqing, and Guangzhou, from 2013 to 2015. We first estimated city-specific relationships between air pollutants and LCM using time-series generalized linear models, adjusting for potential confounders. A classification and regression tree (CART) model was used to stratify LCM risk based on combinations of air pollutants and meteorological factors in each city. Then we pooled the city-specific associations using random-effects meta-analysis. Meta regression was used to explore if city-specific characteristics modified the air pollution-LCM association. Finally, we stratified the analyses by season, age, and sex. RESULTS: Over the entire period, the current-day concentrations of PM2.5 and PM10 in Chongqing and PM2.5, PM10, and SO2 in Guangzhou were positively associated with LCM (Excess risk ranged from 0.72% (95% CI 0.27%-1.17%) to 6.06% (95% CI 0.76%-11.64%) with each 10mug/m(3) increment in different pollutants), but the association between current-day air pollution and LCM in Beijing was not significant (P>0.05). When considering the environmental and weather factors simultaneously, current-day PM2.5, relative humidity, and PM10 were the most important factors associated with LCM in Beijing, Chongqing, and Guangzhou, respectively. LCM risk related with daily PM2.5, PM10, and SO2 significantly increased with the increasing annual mean temperature and humidity of the city, w**hile LCM risk related with daily O3 significantly increased with the increases of latitude, annual mean O3 concentration, and socioeconomic level**. After stratification, the current-day PM2.5, PM10, and O3 during the warm season in Beijing and PM2.5, PM10, and SO2 during the cool season in Chongqing and Guangzhou were positively associated with LCM (Excess risk ranged from 0.93% (95% CI 0.42%-1.45%) to 7.16% (95% CI 0.64%-14.09%) with each 10mug/m(3) increment in different pollutants). Male and the elderly lung cancer patients were more sensitive to the short-term effect of air pollution. CONCLUSIONS: Lung cancer patients should enhance protection measures against air pollution. More attentions should be paid for the high PM2.5, PM10, and O3 during the warm season in Beijing, and high PM2.5, PM10, and SO2 during the cool season in Chongqing and Guangzhou

L. Bai, S. Shin, R. T. Burnett, J. C. Kwong, P. Hystad, Donkelaar A. van, M. S. Goldberg, E. Lavigne, S. Weichenthal, R. V. Martin, R. Copes, A. Kopp, and H. Chen. Exposure to ambient air pollution and the incidence of lung cancer and breast cancer in the Ontario Population Health and Environment Cohort. *Int.J.Cancer* 146 (9):2450-2459, 2020.

Lung and female breast cancers are highly prevalent worldwide. Although the association between exposure to ambient fine particulate matter (PM2.5) and lung cancer has been recognized, there is less evidence for associations with other common air pollutants such as nitrogen dioxide (NO2) and ozone (O3). Even less is known about potential associations between these pollutants and breast cancer. We conducted a population-based cohort study to investigate the associations of chronic exposure to PM2.5 , NO2 , O3 and redox-weighted average of NO2 and O3 (Ox) with incident lung and breast cancer, using the Ontario Population Health and Environment Cohort (ONPHEC), which includes all long-term residents aged 35-85 years who lived in Ontario, Canada, 2001-2015. Incident lung and breast cancers were ascertained using the Ontario Cancer Registry. Annual estimates of exposures were assigned to the residential postal codes of subjects for each year during follow-up. We used Cox proportional-hazards models adjusting for personal- and neighborhood-level covariates. Our cohorts for lung and breast cancer analyses included ~4.9 million individuals and ~2.5 million women, respectively. During follow-up, 100,146 incident cases of lung cancer and 91,146 incident cases of breast cancer were diagnosed. The fully adjusted analyses showed positive associations of lung cancer incidence with PM2.5 (hazard ratio [HR] = 1.02 [95% CI: 1.01-1.05] per 5.3 mug/m(3)) and NO2 (HR = 1.05 [95% CI: 1.03-1.07] per 14 ppb). **No associations with lung cancer were observed for O3 or Ox** . Relationships between PM2.5 and NO2 with lung cancer exhibited a sublinear shape. We did not find compelling evidence linking air pollution to breast cancer

F. Kazemiparkouhi, K. D. Eum, B. Wang, J. Manjourides, and H. H. Suh. Long-term ozone exposures and cause-specific mortality in a US Medicare cohort. *J.Expo.Sci.Environ.Epidemiol.*, 2019.

We examined the association of long-term, daily 1-h maximum O3 (ozone) exposures on cause-specific mortality for 22.2 million US Medicare beneficiaries between 2000-2008. We modeled the association between O3 and mortality using age-gender-race stratified log-linear regression models, adjusted for state of residence. We examined confounding by (1) adjusting for PM2.5 (particles with aerodynamic diameters <2.5 mum) and NO2 (nitrogen dioxide) exposures, temperature, and neighborhood-level characteristics and behaviors, and (2) decomposing O3 into its temporal and spatio-temporal components and comparing estimated risk ratios. We also examined sensitivity of our results to alternate exposure measures based on warm-season 8-h daily maximum and 24-h average exposures. We found increased risks from long-term O3 exposures to be strongest and most consistent for mortality from respiratory disease (1.030, 95% CI: 1.027, 1.034) (including COPD (chronic obstructive pulmonary disease)), CHF (congestive heart failure), and lung cancer (1.015, 95% CI: 1.010, 1.020), with no evidence of confounding by PM2.5, NO2, and temperature and with results similar across O3 exposure measures. While significant, associations between long-term O3 exposures and CVD (cardiovascular)-related mortality (1.005, 95% CI: 1.003, 1.007) were confounded by PM2.5 and varied with the exposure measure, with associations no longer significantly positive when warm-season 8-h maximum or 24-h average O3 was used to assess exposures. **In this large study, we provide strong evidence that O3 exposure is associated with mortality from respiratory-related causes and for the first-time, lung cancer, but raise questions regarding O3-related impacts on CVD mortality.** Our findings demonstrate the need to further identify potential confounders

S. Faridi, M. Shamsipour, M. Krzyzanowski, N. Kunzli, H. Amini, F. Azimi, M. Malkawi, F. Momeniha, A. Gholampour, M. S. Hassanvand, and K. Naddafi. Long-term trends and health impact of PM2.5 and O3 in Tehran, Iran, 2006-2015. *Environ.Int.* 114:37-49, 2018.

The main objectives of this study were (1) investigation of the temporal variations of ambient fine particulate matter (PM2.5) and ground level ozone (O3) concentrations in Tehran megacity, the capital and most populous city in Iran, over a 10-year period from 2006 to 2015, and (2) estimation of their long-term health effects including all-cause and cause-specific mortality. For the first goal, the data of PM2.5 and O3 concentrations, measured at 21 regulatory monitoring network stations in Tehran, were obtained and the temporal trends were investigated. The health impact assessment of PM2.5 and O3 was performed using the World Health Organization (WHO) AirQ+ software updated in 2016 by WHO European Centre for Environment and Health. Local baseline incidences in Tehran level were used to better reveal the health effects associated with PM2.5 and O3. Our study showed that over 2006-2015, annual mean concentrations of PM2.5 and O3 varied from 24.7 to 38.8mugm(-3) and 35.4 to 76.0mugm(-3), respectively, and were significantly declining in the recent 6years (2010-2015) for PM2.5 and 8years (2008-2015) for O3. However, Tehran citizens were exposed to concentrations of annual PM2.5 exceeding the WHO air quality guideline (WHO AQG) (10mugm(-3)), U.S. EPA and Iranian standard levels (12mugm(-3)) during entire study period. We estimated that long-term exposure to ambient PM2.5 contributed to between 24.5% and 36.2% of mortality from cerebrovascular disease (stroke), 19.8% and 24.1% from ischemic heart disease (IHD), 13.6% and 19.2% from lung cancer (LC), 10.7% and 15.3% from chronic obstructive pulmonary disease (COPD), 15.0% and 25.2% from acute lower respiratory infection (ALRI), and 7.6% and 11.3% from all-cause annual mortality in the time period. We further estimated that deaths from IHD accounted for most of mortality attributable to long-term exposure to PM2.5. The years of life lost (YLL) attributable to PM2.5 was estimated to vary from 67,970 to 106,706 during the study period. **In addition, long-term exposure to O3 was estimated to be responsible for 0.9% to 2.3% of mortality from respiratory diseases.** Overall, long-term exposure to ambient PM2.5 and O3 contributed substantially to mortality in Tehran megacity. Air pollution is a modifiable risk factor. Appropriate sustainable control policies are recommended to protect public health

S. Cakmak, C. Hebbern, L. Pinault, E. Lavigne, J. Vanos, D. L. Crouse, and M. Tjepkema. Associations between long-term PM2.5 and ozone exposure and mortality in the Canadian Census Health and Environment Cohort (CANCHEC), by spatial synoptic classification zone. *Environ.Int.* 111:200-211, 2018.

Studies suggest that long-term chronic exposure to fine particulate matter air pollution can increase lung cancer mortality. We analyzed the association between long term PM2.5 and ozone exposure and mortality due to lung cancer, ischemic heart disease, and chronic obstructive pulmonary disease, accounting for geographic location, socioeconomic status, and residential mobility. Subjects in the 1991 Canadian Census Health and Environment Cohort (CanCHEC) were followed for 20years, and assigned to regions across Canada based on spatial synoptic classification weather types. Hazard ratios (HR) for mortality, were related to PM2.5 and ozone using Cox proportional hazards survival models, adjusting for socioeconomic characteristics and individual confounders. An increase of 10mug/m(3) in long term PM2.5 exposure resulted in an HR for lung cancer mortality of 1.26 (95% CI 1.04, 1.53); the inclusion in the model of SSC zone as a stratum increased the risk estimate to HR 1.29 (95% CI 1.06, 1.57). After adjusting for ozone, HRs increased to 1.49 (95% CI 1.23, 1.88), and HR 1.54 (95% CI 1.27, 1.87), with and without zone as a model stratum. HRs for ischemic heart disease fell from 1.25 (95% CI 1.21, 1.29) for exposure to PM2.5, to 1.13 (95% CI 1.08, 1.19) when PM2.5 was adjusted for ozone. For COPD, the 95% confidence limits included 1.0 when climate zone was included in the model. HRs for all causes of death showed spatial differences when compared to zone 3, the most populated climate zone**. Exposure to PM2.5 was related to an increased risk of mortality from lung cancer, and both ozone and PM2.5 exposure were related to risk of mortality from ischemic heart disease, and the risk varied spatially by climate zone**

A. J. Cohen, M. Brauer, R. Burnett, H. R. Anderson, J. Frostad, K. Estep, K. Balakrishnan, B. Brunekreef, L. Dandona, R. Dandona, V. Feigin, G. Freedman, B. Hubbell, A. Jobling, H. Kan, L. Knibbs, Y. Liu, R. Martin, L. Morawska, C. A. Pope, III, H. Shin, K. Straif, G. Shaddick, M. Thomas, Dingenen R. van, Donkelaar A. van, T. Vos, C. J. L. Murray, and M. H. Forouzanfar. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. *Lancet* 389 (10082):1907-1918, 2017.

BACKGROUND: Exposure to ambient air pollution increases morbidity and mortality, and is a leading contributor to global disease burden. We explored spatial and temporal trends in mortality and burden of disease attributable to ambient air pollution from 1990 to 2015 at global, regional, and country levels. METHODS: We estimated global population-weighted mean concentrations of particle mass with aerodynamic diameter less than 2.5 mum (PM2.5) and ozone at an approximate 11 km x 11 km resolution with satellite-based estimates, chemical transport models, and ground-level measurements. Using integrated exposure-response functions for each cause of death, we estimated the relative risk of mortality from ischaemic heart disease, cerebrovascular disease, chronic obstructive pulmonary disease, lung cancer, and lower respiratory infections from epidemiological studies using non-linear exposure-response functions spanning the global range of exposure. FINDINGS: Ambient PM2.5 was the fifth-ranking mortality risk factor in 2015. Exposure to PM2.5 caused 4.2 million (95% uncertainty interval [UI] 3.7 million to 4.8 million) deaths and 103.1 million (90.8 million 115.1 million) disability-adjusted life-years (DALYs) in 2015, representing 7.6% of total global deaths and 4.2% of global DALYs, 59% of these in east and south Asia. Deaths attributable to ambient PM2.5 increased from 3.5 million (95% UI 3.0 million to 4.0 million) in 1990 to 4.2 million (3.7 million to 4.8 million) in 2015. **Exposure to ozone caused an additional 254 000 (95% UI 97 000-422 000) deaths and a loss of 4.1 million (1.6 million to 6.8 million) DALYs from chronic obstructive pulmonary disease in 2015**. INTERPRETATION: Ambient air pollution contributed substantially to the global burden of disease in 2015, which increased over the past 25 years, due to population ageing, changes in non-communicable disease rates, and increasing air pollution in low-income and middle-income countries. Modest reductions in burden will occur in the most polluted countries unless PM2.5 values are decreased substantially, but there is potential for substantial health benefits from exposure reduction. FUNDING: Bill & Melinda Gates Foundation and Health Effects Institute

R. W. Atkinson, B. K. Butland, C. Dimitroulopoulou, M. R. Heal, J. R. Stedman, N. Carslaw, D. Jarvis, C. Heaviside, S. Vardoulakis, H. Walton, and H. R. Anderson. Long-term exposure to ambient ozone and mortality: a quantitative systematic review and meta-analysis of evidence from cohort studies. *BMJ Open.* 6 (2):e009493, 2016.

OBJECTIVES: While there is good evidence for associations between short-term exposure to ozone and a range of adverse health outcomes, the evidence from narrative reviews for long-term exposure

is suggestive of associations with respiratory mortality only. We conducted a systematic, quantitative evaluation of the evidence from cohort studies, reporting associations between long-term exposure to ozone and mortality. METHODS: Cohort studies published in peer-reviewed journals indexed in EMBASE and MEDLINE to September 2015 and PubMed to October 2015 and cited in reviews/key publications were identified via search strings using terms relating to study design, pollutant and health outcome. Study details and estimate information were extracted and used to calculate standardised effect estimates expressed as HRs per 10 ppb increment in long-term ozone concentrations. RESULTS: 14 publications from 8 cohorts presented results for ozone and all-cause and cause-specific mortality. **We found no evidence of associations between long-term annual O3 concentrations and the risk of death from all causes, cardiovascular or respiratory diseases, or lung cancer. 4 cohorts assessed ozone concentrations measured during the warm season. Summary HRs for cardiovascular and respiratory causes of death derived from 3 cohorts were 1.01 (95% CI 1.00 to 1.02) and 1.03 (95% CI 1.01 to 1.05) per 10 ppb, respectively.** CONCLUSIONS: Our quantitative review revealed a paucity of independent studies regarding the associations between long-term exposure to ozone and mortality. The potential impact of climate change and increasing anthropogenic emissions of ozone precursors on ozone levels worldwide suggests further studies of the long-term effects of exposure to high ozone levels are warranted

S. C. Anenberg, L. W. Horowitz, D. Q. Tong, and J. J. West. An estimate of the global burden of anthropogenic ozone and fine particulate matter on premature human mortality using atmospheric modeling. *Environ.Health Perspect.* 118 (9):1189-1195, 2010.

BACKGROUND: Ground-level concentrations of ozone (O3) and fine particulate matter [< or = 2.5 microm in aerodynamic diameter (PM2.5)] have increased since preindustrial times in urban and rural regions and are associated with cardiovascular and respiratory mortality. OBJECTIVES: We estimated the global burden of mortality due to O3 and PM2.5 from anthropogenic emissions using global atmospheric chemical transport model simulations of preindustrial and present-day (2000) concentrations to derive exposure estimates. METHODS: Attributable mortalities were estimated using health impact functions based on long-term relative risk estimates for O3 and PM2.5 from the epidemiology literature. Using simulated concentrations rather than previous methods based on measurements allows the inclusion of rural areas where measurements are often unavailable and avoids making assumptions for background air pollution. RESULTS: **Anthropogenic O3 was associated with an estimated 0.7 +/- 0.3 million respiratory mortalities (6.3 +/- 3.0 million years of life lost) annually**. Anthropogenic PM2.5 was associated with 3.5 +/- 0.9 million cardiopulmonary and 220,000 +/- 80,000 lung cancer mortalities (30 +/- 7.6 million years of life lost) annually. Mortality estimates were reduced approximately 30% when we assumed low-concentration thresholds of 33.3 ppb for O3 and 5.8 microg/m3 for PM2.5. These estimates were sensitive to concentration thresholds and concentration-mortality relationships, often by > 50%. CONCLUSIONS: Anthropogenic O3 and PM2.5 contribute substantially to global premature mortality. PM2.5 mortality estimates are about 50% higher than previous measurement-based estimates based on common assumptions, mainly because of methodologic differences. Specifically, we included rural populations, suggesting higher estimates; however, the coarse resolution of the global atmospheric model may underestimate urban PM(2.5) exposures

2. Quelques références issues de la presse

https://www.thebulletin.be/disinfection-tunnels-and-safe-cabins-introduced-supermarkets (Belgium)

https://www.dailymail.co.uk/news/article-8185503/Mexico-outdoor-food-market-installs-ozone-based-sanitation-tunnels-protect-shoppers-COVID-19.html (Mexico)

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- ➔ Prof Datuk Dr Lokman Hakim Sulaiman, Pro-Vice Chancellor (Research), Professor of Public Health, International Medical University – Opinion – https://www.amdi.usm.my/index.php/ibsc-lokman

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