SMELL, TASTE AND COVID-19:
TESTING IS ESSENTIAL

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Abstract. During the Covid-19 pandemic it became clear that smell and taste (chemosensory) disturbance is very common in the early stages of disease. This article addresses: 1) why Covid-19 specifically targets the modalities of smell and possibly taste and what is the mechanism 2) what is the frequency of smell and taste loss and, 3) what is the overall prognosis. It is suggested that mouth breathers may be at particular risk of Covid-19. Symptom-based questionnaires are likely to underestimate the prevalence of chemosensory impairment by as much as 50%. The prevalence of smell loss is so high that a person who has normal olfaction on formal testing is unlikely to be infected significantly with Cov-2. Furthermore, someone without symptoms who has an abnormal smell test could still be infected and liable to spread the disease. Brief, low cost, olfactory tests are available that would permit a high throughput in field stations and airports. A normal result might obviate the need for a nasopharyngeal swab for the Cov-2 virus.

On March 20th 2020, Claire Hopkins, President of the British Rhinological Society and Nirmal Kumar, President of ENT UK circulated a letter to fellow members that drew attention to the heightened incidence of isolated anosmia in their clinics. In normal circumstances they would see around one case of post-viral anosmia per month whereas in the recent past this had increased to 4 per week and remarkably all were under 40 years old. They questioned whether new onset anosmia in relatively young people might be an early warning of Covid-19 infection and emphasised the presence of similar observations from China, South Korea and Iran.

It is unusual for impairment of smell sense to be such a prominent symptom of upper respiratory viral infection. The majority of the latter are recognised in ear nose and throat (ENT) units particularly as a symptom from middle-aged or elderly in the aftermath of upper respiratory infection (URT). Most individuals who experience URT such as the common cold, accept a degree of smell impairment that results typically from a blocked nose, but what is remarkable about Covid-19 is that its occurrence is often early, of acute onset, severe and only occasionally associated with a blocked nose. These thoughts have been given preliminary support from a study of 10 patients with proven Covid-19 infection compared to 10 people with an acute cold and 10 healthy controls. Using the extended version of Sniffin’ Sticks there were significant differences: those with Covid-19 infection scored lower than the acute cold group, with identification scores affected more than threshold or discrimination.
Several questions arise: why should Covid-19 specifically target chemosensation (i.e. smell and taste) and what is the mechanism? What is the frequency of smell and taste loss and what is the overall prognosis?

1. **Why should Covid-19 specifically target the sense of smell and possibly taste?**

Preliminary information about the likely mode of nasal invasion is just emerging. The virus, SARS-CoV-2, (shortened here to Cov-2) that causes the illness, Covid-19, infects cells through interactions between its spike protein and the ACE2 protein on target cells (Figure 1).

**Figure 1.** Molecular structure of the SARS-Cov-2 virus to show how the virus can attach to a pneumocyte (alveolar cell) that lines the alveoli. Reproduced with permission from David Baker, Blizard Institute, Queen Mary, University of London.

This interaction requires cleavage of the spike protein, likely by the cell surface protease, serine (TMPRSS2) and other proteases such as cathepsin B and L. It has been demonstrated that non-olfactory epithelial cells from the human upper airway express high levels of ACE2 and serine proteases as shown in Figure 2, a finding that implies they could act as a viral reservoir.

**Figure 2.** Simplified model for CoV-2-induced anosmia/hyposmia in COVID-19 based on results obtained from patients and animal models. Article from Open Access journal reproduced with permission from Bilinska and Butowt.

According to Brann and colleagues olfactory epithelial sustentacular cells, horizontal basal cells and Bowman’s gland cells express the receptors required for entry of CoV-2 but there is no ACE2 expression in mature olfactory receptor neurones. In essence they propose that the anosmia of Covid-19 relates to primary infection of non-neuronal cell types and by implication, that smell loss is a consequence of local inflammation in and around the nasal neuro-epithelium. This concept has received preliminary confirmation from MRI-based studies that reveal congestion in the olfactory cleft – the area that houses olfactory neurones. Although these findings are plausible it is possible, as the authors suggest, that other non-ACE2 dependent receptors may facilitate cellular entry of CoV-2. These observations are preliminary and it is still possible that CoV-2 may involve the olfactory...
Indeed MRI-based studies have shown oedema of the olfactory bulb\(^9\) (Figure 3) as well as more central changes, namely in the gyrus rectus\(^{10}\) and by CT/PET, in the orbitofrontal cortex\(^{11}\).

**Figure 3.** Transient olfactory bulb oedema as shown in coronal 3D MRI T2-weighted imaging (1.5T) during anosmia (day 7; C) compared to recovery (day 24; D). Olfactory bulb (ob; pink) displays transient volume and signal increase, olfactory cleft oedema (OC; brown), and focal left ethmoid (eth; green) sinusitis (*), and normal cranial fossa (grey line) and orbit (orb; yellow). Reproduced from Figure 1 C and D with permission from Laurendon et al\(^9\).

There is provisional evidence that ACE2 receptors are present in the tongue (Figure 4) particularly taste buds and to lesser degree in the lingual epithelium\(^{12,13}\). Cov-2 can be isolated from saliva\(^{14}\) thus there is a plausible mechanism for such infection to involved taste bud receptors. Less is known about ACE2 expression in the major taste nerves, namely the chorda tympani and glossopharyngeal nerves.

**Figure 4.** Bulk RNA-seq analysis of public datasets. Bar plot of ACE2 expression in normal tissues from FANTOM5 CAGE dataset, coloured by organs. Reproduced from Figure 1b with permission under Open Access from: Xu et al\(^{14}\).

2. **What is the frequency of chemosensory loss?**

There have been numerous estimates worldwide, but with a few exceptions detailed below, most have been based on questionnaire surveys without objective measurement and several have not contained a control group. Samples have been varied: some are based predominantly on out-patients others reliant on in-patients with testing at varying stages of illness. The largest investigation\(^{15,16}\) employed a smartphone-based app to retrieve symptomatic data on over 2 million people in UK and USA and found that in those reporting chemosensory impairment, 65% had a subsequent positive PCR for Covid-19. When this was combined with fever, cough, fatigue and loss of appetite the correlation with PCR for Cov-2 was very high. A large meta-analysis totalling 38,198 subjects\(^{17}\) documented an overall prevalence of smell impairment in Caucasians of 49% and 16.7% in Asians. Taste symptoms occurred in 51% Caucasians and 18% Asians. Other studies show wide
estimates of prevalence – up to 70% with an approximately equal rate for smell or taste. Sometimes isolated impairment of smell or taste is documented as a presenting symptom. A study from San Diego based on ambulatory individuals with influenza-like symptoms, noticed that subjective report of smell impairment was associated with a 10-fold lower risk of hospital admission for Covid-19. This finding is discussed further below.

Surveys that have relied on patient reports are susceptible to multiple confounders including recall bias and a tendency to over-representation of female respondents. Even more importantly, less than 40% of individuals are actually aware of a proven olfactory defect. For subjective awareness, the defect needs to be bilateral and of at least moderate severity. Furthermore, smell loss and taste loss are very frequently confused. Most people who complain of impaired taste have reduced olfaction whereas it is unusual for someone with primary taste loss to complain of smell impairment. The mechanism of this phenomenon has not been satisfactorily explained.

Patient reports of olfactory impairment are therefore intrinsically unreliable and will tend to underestimate the true picture due to lack of awareness and confusion with taste. Furthermore, if taste is really affected in Covid-19 any such deficiency would inflate estimates of smell impairment where based on subjective reports. According to PubMed, at the time of writing there have been 14 articles worldwide where various objective olfactory measurements have been made (Table 1). Case numbers range by centre from 14-345 individuals. In nearly all instances a confirmatory polymerase chain reaction (PCR) test for Cov-2 has been undertaken. In only 9 cases was there a control group and where present the PCR test is not stated in 7 of these. Matching by age and gender was performed in just 4 instances. Subjective awareness of olfactory loss was indicated in 12 studies with a prevalence ranging from 28%-86% (mean 54%). Some authors have used non-standard olfactory measurement e.g. modified ethyl-alcohol threshold test or an in-house identification test of 10 odours neither containing details of control data. In one article, patients quarantined at home were instructed on how to make up their own smell and taste ingredients, despite the existence of readily available standardised commercial test kits for home use. The 4-odour Pocket Smell Identification Test used by one group or the 3-odour Quick Smell Identification Test (Q-SIT) employed by others are more appropriate for rapid screening in the clinic, rather than large research projects. For example, a score of 3/3 correct answers on the Q-SIT is likely to indicate normal olfaction but as emphasised by the authors, a value of 2/3 could represent either hyposmia or a normal result because of wide variance. Nonetheless, a score of 3/3 would help exclude
anosmia where a low-cost, high-volume survey is required. The data from Iran\textsuperscript{31,32} have been criticised unfairly because many of the 40 odours used in the identification kit were allegedly unfamiliar to Iranians\textsuperscript{33}. However, the test used was in fact specifically modified to account for cultural differences\textsuperscript{34}.

It is important to be aware of the time of olfactory assessment in calculating the prevalence of Cov-2 related smell impairment, whether based on questionnaires or psychophysical tests. Clearly the closer to acute symptom onset, the more chance of an abnormal result. In four instances this information is not supplied. Where the time of testing is supplied, this ranges from 4-37 days.

Taking into account the above reservations, there are just four more robust studies that have used standardised smell tests, have a control group, time of examining 14 days or less and adequate patient numbers, arbitrarily set at 45 or more \textsuperscript{28,31,32,35}. With this reservation, it may be inferred a) that subjective awareness of smell impairment is highly variable i.e. 28%-49% b) olfactory impairment on objective testing is present in 84%-98%. c) in general, hospitalised patients who are assessed within 14 days of symptom onset have more abnormal smell tests (71%-98%). The picture for outpatients is less clear.

Compared to subjective patient reports, smell measurement will therefore uncover a further 40% - 50% of proven Cov-2 infected people, indicating that the olfactory defect is near universal. In practical terms this means that an abnormal smell test may be present in someone with no symptoms and yet be capable of spreading the virus. Conversely, a normal standardised smell test such as the Sniffin’ Sticks or UPSIT should help exclude the presence of Covid-19 and would be valuable for mass population screening.

A less clear picture is available for the sense of taste. Only 6 studies report taste measurement (Table 1) and details of a control group are not given in four of these. Just three centres\textsuperscript{27,34,36} implemented a standardised measurement (taste strips) and documented a normal result in a total of 40 patients from two centres\textsuperscript{27,36} with an abnormal value from one unit\textsuperscript{34} (5/72; 7%). The other three\textsuperscript{25,26,37} used in-house tests and observed abnormalities ranging from 27%-49%. No reliable conclusion can be drawn from these limited observations.

Table 1. Summary of articles where objective chemosensory testing was undertaken. CCCRC= Connecticut Chemosensory Clinical Research Center orthonasal olfaction test. PCR=polymerase chain reaction. ID=identification. Q-SIT= Quick Smell Identification Test. UPSIT=University of Pennsylvania Smell Identification Test. SST = Sniffin’ Sticks test). TOT= time of testing. The Taste Strips Test uses four tastants at four different concentrations.
3. **Prognosis.**

Some subjective patient reports describe recovery of olfaction in 2-6 weeks. This finding is exemplified by one article with serial longitudinal objective assessment\textsuperscript{32}. Return to normal was shown in nearly two thirds (61%) within 8 weeks (Figure 5). At that point, 35% still had varying degrees of impairment although complete smell loss affected just 4%. Distortion of perceived smells (cacosmia) and smell hallucinations (phantosmia) are recognised in the established and disease recovery phase\textsuperscript{40}.

The olfactory neuroepithelium has considerable capacity for regeneration, provided the stem cell layer is not damaged\textsuperscript{41}. This process is unlikely to account for the rapid subjective recovery that in some instances appears complete in as little as 2 weeks (Figure 5). Such swift improvement is more in keeping with resolution of inflammation/oedema surrounding the nasal neuro-epithelium as shown on by MRI (Figure 3). There are insufficient reports relating to the prevalence and recovery of taste impairment.

**Figure 5.** Proportion of patients with varying degrees of recovery according to COVID-19 symptom onset. All initial (n = 100) and follow-up (n = 82) scores are combined. Reproduced from Figure 4 with permission under Open Access from Moein et al\textsuperscript{32}.

**Potential risk for mouth breathers.** There are multiple causes of mouth breathing. It relates usually to nasal obstruction from a displaced septum, congestion, polyps and a variety of developmental abnormalities of the nasal cavity including Down’s syndrome. In some it is just a bad habit. Most snorers breathe through the mouth and there is evidence that people with obstructive sleep apnoea are mouth breathers\textsuperscript{42}. In the San Diego study of olfaction and Covid-19\textsuperscript{19} it was speculated that milder cases of COVID-19 may present with severe anosmia and higher self-reporting, compared to the undetected or slight hyposmia associated with moderate to severe COVID-19 cases. If correct, this dichotomy may relate in part, to an individual’s pattern of inspiration. Thus, habitual nose-breathers would direct airborne virus into the nasal passages where there are multiple immune-based defence functions that serve as a primary mucosal immune barrier e.g. the nasopharynx-associated lymphoid tissue\textsuperscript{43} (NALT) known collectively as Waldeyer’s ring. A mouth-breather would therefore bypass the nasal component i.e. the adenoid and tubal tonsils and have to rely on the laryngeal and lingual tonsils. In theory, those who have had tonsillectomy or adenoidectomy might be more susceptible to subsequent viral infection although the consensus view is against this.
contention. A further defence mechanism favouring nose breathers relates to increased synthesis of sino-nasal nitric oxide (NO) which is an integral and highly conserved part of the host immune response. It acts as a first-line of defence against micro-organisms and upregulates ciliary motility. At low concentration, NO acts as a signalling molecule that promotes growth and activity of immune cells. At high concentrations it binds DNA, proteins and lipids, thereby inhibiting or killing target pathogens. In support of this in the clinical setting, 6 human volunteers were infected with human rhinovirus (HRV-16), a non-enveloped RNA virus. Elevated nitric oxide synthase mRNA was detected in nasal turbinate scrapings from infected individuals and increased levels of exhaled NO from the nose and lower airways. Others are exploring the possible benefits of inhaled nitric oxide in acute respiratory distress syndrome.

Discussion.

Smell impairment occurs in Covid-19 probably by involvement of ACE2-expressing cells (particularly the sustentacular cells) in the nasal olfactory area rather than the olfactory neurones per se thus resulting in a local inflammatory response in the nasal cleft, thereby impairing olfactory transduction. Involvement of the olfactory bulb and its central connections may occur in more advanced cases. The value of subjective reports is severely limited by low sensitivity to an established smell defect and confusion with taste impairment. Objective testing suggests that there is smell loss in nearly all patients suffering from Covid-19. In theory, a mouth breather would be more at risk of lung infection (and severe Covid-19) than a nose breather. Partial or complete smell recovery takes place in around two thirds subjects over a period of 2-6 weeks. Hence, anosmia constitutes an important warning symptom and sign of infection by Cov-2 and has been highlighted in the UK public domain since June 2020.

Olfactory testing elevates the detection rate of a defect by about 50% i.e. from around 30-40% according to symptoms, to more than 90% where based on measurement. The importance of smell testing as opposed to smell questioning cannot be emphasized more strongly. The prevalence of smell symptoms and signs is so high that a person who has normal olfaction on testing by procedures listed below, is unlikely to be infected with Cov-2 or if so, their viral load would be low and unlikely to result in transmission to others. Where resources are limited it is suggested that a rapid screening test of olfaction could be used in field stations or airports as a substitute for or complementary to nasal and throat swabs. False positives i.e. anosmia may result from other rhinotropic viruses but these patients would require formal viral testing in any event. The risk of a false negative result is low with estimates ranging from 2%-28%. If one excludes the small
study of 41 patients that implemented a 4 odour test\cite{27} then the false negative rate drops to 2%-
16%).

The position regarding taste impairment is less clear. Subjective complaint of dysgeusia is frequent
but in most instances represents confusion with olfactory loss. Objective evaluation of taste
impairment is complex and reviewed elsewhere\cite{49}.

There are clearly some weaknesses in this analysis. Although 14 studies address Covid-19 and
hyposmia, patient numbers are relatively small and the tests employed are varied, sometimes
unorthodox. Some groups have no control group and do not state clearly the time of testing – or if
so, the range in days is wide. Other investigations have been published without peer review or
possibly submitted (and published) in haste, given the urgency of the current pandemic. Ethnic
differences may account for some of the wide variation in results\cite{17}. Despite this, the conclusions
based primarily on the 4 most thorough investigations, show a consistent relationship between smell
impairment and Covid-19 and support the main messages in this paper.

The Way Forward.

The following are suggested for future Covid-19 chemosensory research

1. Smell measurement should be undertaken by centres with a proven track record of
   chemosensory research using internationally recognised tests
2. Large numbers of cases at varying stages of disease and healthy controls should be collected
   in the community and hospital setting. The number required should be determined from
   power calculations based on the number of test odours used.
3. Any of the following identification procedures would be suitable for large scale studies in
   walk-in centres or airports:
   - Sniffin’ Sticks, 12 odour version. This may be re-used multiple times within its
     shelf life. Given the potential risks of transmission it would be best administered
     by a trained operator rather than the subjects themselves
   - B-SIT (Brief Smell Identification Test). This is a ‘scratch and sniff’ procedure for
     single use only and comprises 12 odours. It is suitable for on-site or home
     completion
   - NHANES-8. (US National Health and Nutrition Examination Survey). This is a low
     cost 8 odour version similar in principle to the B-SIT. It is under evaluation for
     future field use.

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The above procedures could be undertaken in Covid-19 walk-in centres or airports as an inexpensive screening procedure. An initial large-scale trial would be required to assess the sensitivity of the chosen test. Based on current data, a normal result would likely avoid the need for nasal/throat swabs whereas an abnormal result would require formal virological analysis.

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Footnote: Q-SIT (Quick Smell Identification Test), B-SIT (Brief Smell Identification Test), UPSIT (University of Pennsylvania Smell Identification Test) and the Pocket Smell Test are trademarks of Sensonics International, Haddon Heights, New Jersey, USA.

Sniffin’ Sticks and Taste Strips are trademarks of Burghart Messtechnik GmbH, Wedel, Germany.

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Ethical approval. Not required

Contributions. This article is the sole work of the author
| Reference and Country of Test | Type of tests | CASES | CONTROLS | CASES. Number aware of smell / or taste impairment | CASES | Smell test results | CONTROLS | Smell test results | CASES: Taste test results |
|-------------------------------|---------------|-------|----------|---------------------------------|-------|-------------------|----------|-------------------|----------------------------|
| **Altin**<sup>17</sup> Turkey. | 16 odour SST ID test In house taste ID of sucrose, salt, vinegar and coffee. | 81 in-patients. 40 female. Mean age: 54y. All PCR positive. TOT not stated. | 40 age/gender matched healthy controls. 19 female (47%). Mean age: 55y. Source not stated. All PCR negative. TOT not stated. | 50/81 (62%) | Median score 6/16Percent abnormal not stated | Median score 10/16Percent abnormal not stated | 22/81 (27%) abnormal |
| **Becksberger**<sup>18</sup> Germany | 12 odour SST ID test Taste Strips Test | 14 in-patients for smell tests. Taste test in 10. Mean age 46y. 13 female. Cov-2 status not stated. TOT 4-23 days from symptom onset | None | 26/63 (41%)complained of loss of smell or taste | 10/14 (71%) abnormalNot helped by nasal decongestant | None | All 10 patients were normal |
| **Calvo-Henriquez**<sup>19</sup> Spain | Modified ethyl alcohol threshold test | 129 in- or out-patients. Mean age 55y. 67 (52%) female. Severe cases excluded. All PCR positive. TOT not given | 146 healthy hospital staffMean age 55y. 76 female. (52%). PCR: not stated. TOT not given | Not stated | Abnormal thresholdNot supplied directly | Not done |
| **Chung**<sup>20</sup> Hong Kong | UPSIT and Butanol threshold test (BTT) | 18 mildly infected in-patients. Mean age 28y. 11 female (61%). All PCR positive. Median TOT: 14 days. | 18 students or healthcare workers. Mean age 31y. 13 (72%) female. PCR not stated. TOT not given | 12/18 (67%) | Abnormal BTT in 6/18 (33%). All 6 had abnormal UPSIT. | Not given | Not done |
| **Hintschich**<sup>21</sup> Germany | Pocket Smell Test (4 odours) Taste Strips Test Both self-administered | 41 patients under home quarantine. All PCR positive. Median age 37y. 28 (68%) female. TOT: 3 days after positive PCR. Median of 13 days after first symptoms | 30 patients. Source: not stated. Median age 33y. 22 (73%) female. All negative for IgG antibodies. TOT not stated. | 25 (61%) for smell. 18 (44%) for taste | 22 (54%) abnormalWhere there was subjective loss of smell, abnormal in 18 (72%) | Not stated | Not significantly different from controls |
| **Hornuss**<sup>24</sup> Germany | 12 odour SST ID test | 45 in-patients. 20 female (44%). Median age 56y. All PCR positive. Mean duration of symptoms / time of testing: 10 days. | 45 asymptomatic in-patients or health-care workers. Median age 54y. Gender not stated. PCR: not done. TOT not stated | Smell: 22/45 (49%) | 38/45 (84%) abnormal | 12/45 (27%) abnormal | Not done |
| **Le Bon**<sup>25</sup> Belgium | Extended SST (threshold, discrimination and identification to 16 odours). Taste Strips Test | 72 outpatients. 49 (66%) female. Mean age 38.9y. 35 PCR positive. 47 IgG antibody positive. TOT: mean of 37 days after symptom onset | None | Smell: 100% as self selected | 27/72 (38%) abnormalMain effect on threshold scores. 45 normal (62%). | None | 5/72 (7%) abnormal. |
| Lechien (Belgium) | SST ID test | 46 out-patients with ‘initial sudden olfactory anosmia’. Mean age: 40.6y. 46 female (59%) PCR positive in 42/46 when tested in <12 days from symptom onset. None | Total: 36. Source not stated. Mean age: 37.2y. 19 female (53%). PCR: not stated. TOT not stated | Smell: 35/41 (86%) had subjective loss as reported from earlier study. | None | Not done |
| Lima (Brazil) | QSIT. 3 odour ID test | 57 Out-patients. 31 females (54%). Mean age 41.4y. All PCR positive. All but two had mild disease. Mean symptom duration: 4 days. | Smell: 34/57 (60%). Total: 20/23 (87%) abnormal in those with subjective smell loss. 11/34 (32%) abnormal in those without subjective smell loss. | 4/36 (11%) abnormal | Not done |
| Moein (Iran) | UPSIT. Revised Persian language version | Total: 60. All in-patients. 20 female (33%). Mean age: 46y. All PCR positive. TOT: <14 days of symptom onset. 60 healthy sex & age-matched controls from prior study. PCR: not stated. | Total: 51. 11 female (37%). Mean age 45.4y. PCR: not stated. | Not stated | Not done |
| Moein (Iran) | UPSIT. Revised Persian language version | Total: 100 initial inpatients. Mean age 45y. 33 females (33%). TOT: near end of acute disease phase. After symptom onset 82 retested at 1-4 weeks. 51 retested at 6-8 weeks. All PCR positive. 51 healthy age- & sex-matched to 52 COVID patients from prior study. 19 female (37%). Mean age 45.4y. PCR: not stated. | Smell: 28/100 (28%). Total: 96/100 (96%) abnormal on initial testing. Mean UPSIT score 22/40. | Not stated | Not done |
| Tsivgoulis (Greece) | Q-SIT. 3 odour ID test | Total: 22 in-patients. Mean age 55y. 10 female (45%). TOT: mean of 12 days after hospital admission. All PCR positive. 22 age- & sex-matched controls taken from movement disorders clinic. PCR: not stated. TOT not stated. | Not stated | 17/22 (77%) abnormal | Not done |
| Vaira (Sardinia) | CCCRC. In-house ID of 10 odours and butanol threshold. In-house taste identification for: salt, sugar, lemon & coffee solutions | Total: 72 health personnel. 25 in-patients. Others out-patients. 45 female (62%). Mean age 49y. TOT: mean 19 days from symptom onset. All PCR positive. | Smell and/or taste symptoms in 53/72 (74%). 60/72 (83%) abnormal for composite olfactory score (threshold and discrimination) | None | Abnormal: 35/72 (49%) |
| Vaira (Italian multicentre) | For quarantined patients: home self-administered and prepared odor discrimination test to 6 odour classes. Also used home self-administered and prepared solutions to 4 tastants. For in-patients: CCCRC | Total 345 patients. 161 in quarantine (self evaluated at home). 184 in-patients. 199 (58%) female. Mean age 49y. TOT: mean 15 days from symptom onset. All PCR positive. None | Smell and/or taste symptoms in 256 (74%) Overall percentages not supplied. From sequential graphs: around 70% abnormal for olfaction. 45% overall abnormal on taste test. | None | Abnormal in 190 cases (49%) |
Figure 1. Molecular structure of the SARS-Cov-2 virus to show how the virus can attach to a pneumocyte (alveolar cell) that lines the alveoli. Reproduced with permission from David Baker, Blizard Institute, Queen Mary, University of London.

106x62mm (96 x 96 DPI)
Figure 2. Simplified model for CoV-2-induced anosmia/hyposmia in COVID-19 based on results obtained from patients and animal models. Article from Open Access journal reproduced with permission from Bilinska and Butowt6

119x87mm (96 x 96 DPI)
Figure 3. Transient olfactory bulb oedema as shown in coronal 3D MRI T2-weighted imaging (1.5T) during anosmia (day 7; C) compared to recovery (day 24; D). Olfactory bulb (ob; pink) displays transient volume and signal increase, olfactory cleft oedema (OC; brown), and focal left ethmoid (eth; green) sinusitis (*), and normal cranial fossa (grey line) and orbit (orb; yellow). Reproduced from Figure 1 C and D with permission from Laurendon et al9.
Figure 4. Bulk RNA-seq analysis of public datasets. Bar plot of ACE2 expression in normal tissues from FANTOM5 CAGE dataset, coloured by organs. Reproduced from Figure 1b with permission under Open Access from: Xu et al14.
Figure 5. Proportion of patients with varying degrees of recovery according to COVID-19 symptom onset. All initial (n = 100) and follow-up (n = 82) scores are combined. Reproduced from Figure 4 with permission under Open Access from Moein et al32.