Covid – Where Now?

Where is the UK now, as at March 2024, in regard to Covid?

**Data Presentation Changes**

The coronavirus dashboard was decommissioned on Thursday, 14 December 2023. As at 11 December 2023 the government coronavirus.data.gov.uk website stated: "the decommissioning process for the coronavirus (Covid – 19) dashboard has started with the final data update expected to take place on Thursday, 14 December 2023.

Following that and as at 17 December 2023 the government website at https://coronavirus.data.gov.uk/details/whats-new informed the public that the final date update of the Coronavirus (COVID-19) dashboard took place on Thursday 14th December 2023 and that from Thursday 21st December the public could use the UKHSA data dashboard https://ukhsa-dashboard.data.gov.uk/ for data on respiratory viruses including COVID-19.

So, now the United Kingdom health security agency (UKHSA) is the place to go to for finding out Covid data.

However, as at 16 December 2024 a notice on https://ukhsa-dashboard.data.gov.uk/ stated that as the UKHSA dashboard is still undergoing statistical review the public should not use the dashboard data and instead for reporting and analytical purposes should use the COVID-19 dashboard (https://coronavirus.data.gov.uk/) and the weekly surveillance report https://www.gov.uk/government/statistics/national-flu-and-covid-19-surveillance-reports-2023-to-2024-season

As at March 2024 the UKHSA dashboard states:

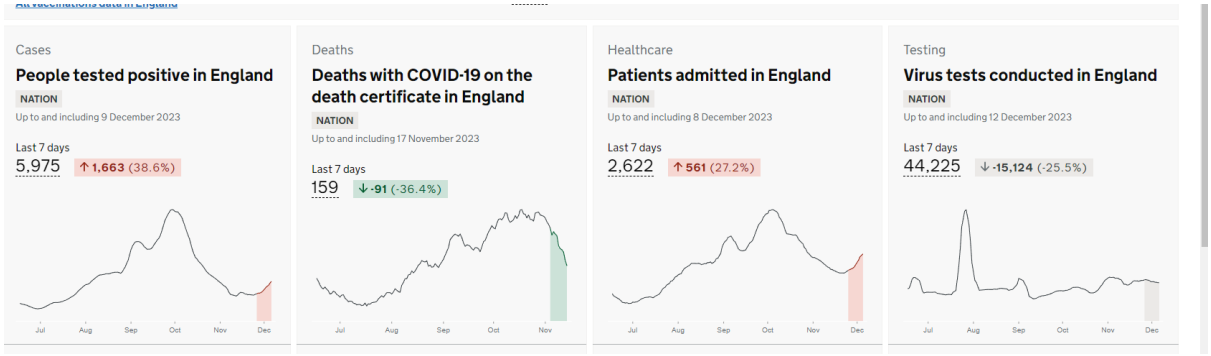
"*The UKHSA data dashboard shows public health data across England. It builds on the success and is an iteration of the COVID-19 in the UK dashboard.*

*Initially, the dashboard presents data on respiratory viruses. In the future, it will grow to present a wider range of data on public health topics in line with the remit of the UKHSA. Find out more about the UKHSA data dashboard.*

*From next month, the NHS is changing how often it publishes healthcare data. The data will be available monthly instead of weekly. From 4 April, updates to healthcare metrics will be paused. From May, we will start updating healthcare data monthly*."

For historical comparison note that data as at 16 December 2023 showed the following:

(1) Up to and including 9 December 2023 – 5,975 people tested positive in the last 7 days which was an increase of 1,663 (38.6%) - England
(2) Deaths with COVID-19 on the death certificate in England up to and including 17 November 2023 = 159 in the last 7 days (down by 91 (-36.4%)
(3) Patients admitted in England up to and including 8 December 2023 in the last 7 days was 2,622 (up 561 (27.2%))
(4) The number of virus tests conducted in England up to and including 12 December 2023 in the last 7 days was 44,225 (down 15,124 (-25.5%)

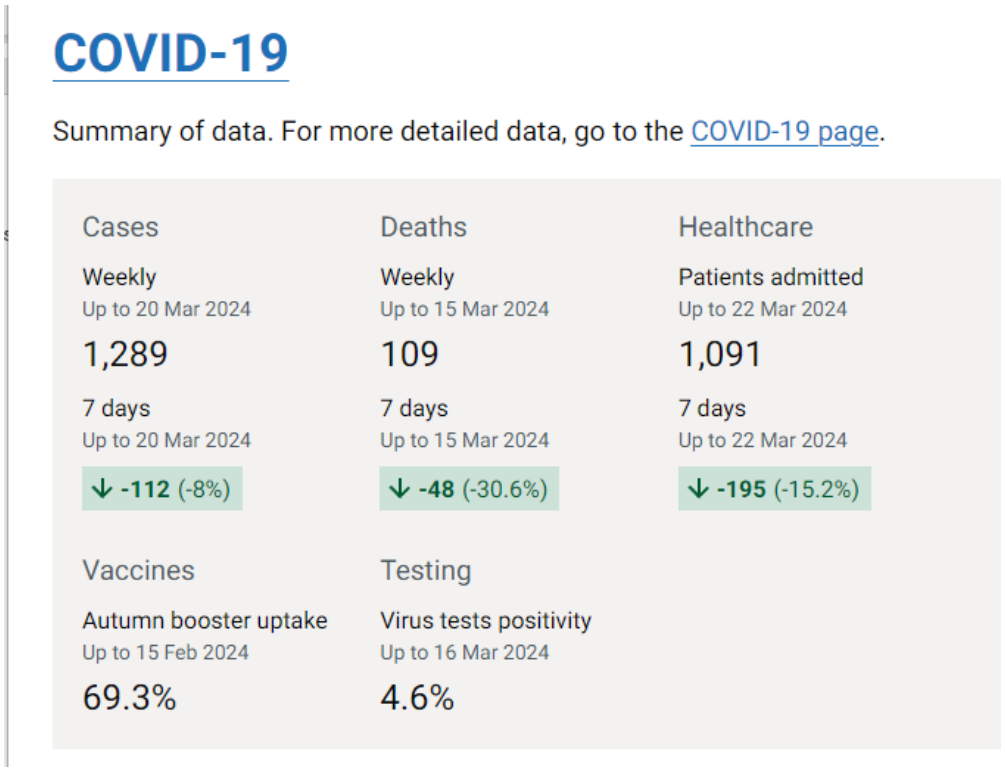



The reasons for the move of data in December 2023 and no longer relying on separate website solely for COVID-19 data is explained in a statement on the coronavirus.data.gov.uk at https://coronavirus.data.gov.uk/details/whats-new/record/2ab6c2eb-4a5d-4659-8925-7910263bda41 states that:

"***We looked at the relevance, suitability and availability of all metrics on the COVID-19 dashboard. As we are now 'Living with COVID', we have selected the most useful metrics to show on the UKHSA data dashboard***."

Previous data showed a summary of data shows up two including 2 December 2023 there were 4146 cases in the last 7 days in England, 245 weekly deaths in England (up to and including 10 November 2023), 2064 patients in England over the last seven days (up to and including 1 December 2023) admitted to hospital, as at 5 December 2023 39,017 virus tests conducted in England.

As at 28 March 2024 details at the UKHSA website (https://ukhsa-dashboard.data.gov.uk/) show the following data:



**UK Variant at September 2023**

As at September 2023 the variant BA.2.86 was noted in the SARS-CoV-2 variant surveillance and assessment: technical briefing 55 which can be found at:

https://www.gov.uk/government/publications/investigation-of-sars-cov-2-variants-technical-briefings/sars-cov-2-variant-surveillance-and-assessment-technical-briefing-55

so, as at said 19 September 2023 the BA.2.86 variant is noted as having an apparently slighter higher human ACE two binding affinity than ex-BBC variants tested.

The role of ACE-2 receptors has, during the Covid crisis, bin considered by various scientists. For example in the National Centre for biotechnology information paper "Understanding the role of ACE-2 receptor in pathogenesis of COVID-19 disease: a potential approach for therapeutic intervention Pharmacol Rep. 2021; 73(6): 1539–1550. Published online 2021 Jun 27. doi: 10.1007/s43440-021-00303-6 (Authors Shirbhate Panel, Patel, Kamal Jawaid, Gorain Kesharwani and Rajak) which can be found at:
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8236094/#:~:text=Here%2C%20the%20ACE2%20receptors%20serve,tract%20%5B26%2C%2037%5D

it is described as follows. Angiotensin-converting enzyme (ACE) and its similar enzyme ACE 2 is an enzyme that is found extensively within the human body and is: "particularly overexpressed on intestinal epithelial cells of the gut, endothelial and smooth cells of the blood vessels, heart… Long… Brain testes and on tubular epithelial cells of kidney. It contains 805 amino acids. It is a type I transmembrane protein. Importantly, as this paper says,: "studies reported that ACE 2 receptors serve as an entrance for the access of coronaviruses… Into human cells".

This facilitation is essentially because unfortunately the ace receptors have a molecular build that is almost a lock shape for the key shape spikes that are found on coronavirus.

In another paper found on the National library of medicine titled: "**COVID-19: angiotensin-converting enzyme 2 (ACE2) expression and tissue susceptibility to SARS-CoV-2 infection by S Beyerstedt, Casoro and Rangel:**

**COVID-19 pandemic is caused by the novel coronavirus SARS-CoV-2. Angiotensin-converting enzyme 2 (ACE2) is not only an enzyme but also a functional receptor on cell surfaces through which SARS-CoV-2 enters the host cells and is highly expressed in the heart, kidneys, and lungs and shed into the plasma. ACE2 is a key regulator of the renin–angiotensin–aldosterone system (RAAS). SARS-CoV-2 causes ACE/ACE2 balance disruption and RAAS activation, which leads ultimately to COVID-19 progression, especially in patients with comorbidities, such as hypertension, diabetes mellitus, and cardiovascular disease. Therefore, ACE2 expression may have paradoxical effects, aiding SARS-CoV-2 pathogenicity, yet conversely limiting viral infection. This article reviews the existing literature and knowledge of ACE2 in COVID-19 setting and focuses on its pathophysiologic involvement in disease progression, clinical outcomes, and therapeutic potential.**

**A Nature article which can be found at https://www.nature.com/articles/s41586-024-07029-4 and published on 21 February 2024 notes the following in its abstract (note references 1-14 removed but which can be found at the website address above):**

**DOI**

**https://doi.org/10.1038/s41586-024-07029-4**

"Persistent SARS-CoV-2 infections may act as viral reservoirs that could seed future outbreaks, give rise to highly divergent lineages and contribute to cases with post-acute COVID-19 sequelae (long COVID). However, the population prevalence of persistent infections, their viral load kinetics and evolutionary dynamics over the course of infections remain largely unknown. Here, using viral sequence data collected as part of a national infection survey, we identified 381 individuals with SARS-CoV-2 RNA at high titre persisting for at least 30 days, of which 54 had viral RNA persisting at least 60 days. We refer to these as 'persistent infections' as available evidence suggests that they represent ongoing viral replication, although the persistence of non-replicating RNA cannot be ruled out in all. Individuals with persistent infection had more than 50% higher odds of self-reporting long COVID than individuals with non-persistent infection. We estimate that 0.1–0.5% of infections may become persistent with typically rebounding high viral loads and last for at least 60 days. In some individuals, we identified many viral amino acid substitutions, indicating periods of strong positive selection, whereas others had no consensus change in the sequences for prolonged periods, consistent with weak selection. Substitutions included mutations that are lineage defining for SARS-CoV-2 variants, at target sites for monoclonal antibodies and/or are commonly found in immunocompromised people. This work has profound implications for understanding and characterizing SARS-CoV-2 infection, epidemiology and evolution."