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COVID-19 in Europe: the Italian lesson

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Severe acute respiratory syndrome coronavirus 2 is rapidly spreading worldwide,[1] and WHO declared the coronavirus disease 2019 (COVID-19) outbreak a pandemic on March 11, 2020.[2]

The outbreak has hit Europe; as of March 20, 2020, Italy has the second-largest number of confirmed cases, after China. As elegantly presented by Andrea Remuzzi and Giuseppe Remuzzi,[3] a rapid surge of cases is posing a serious threat to the Italian national health system because of the limited capacity of intensive care unit departments. The Italian Government introduced progressive mitigation measurements on March 9 and March 11, 2020, to drastically limit social interactions and prevent virus diffusion.[4,5] Projections in Remuzzi and Remuzzi's exponential model,[2] according to data trends before March 8, predicted more than 30 000 cases by March 15, 2020. Real data from the Center for Systems Science and Engineering at Johns Hopkins University suggest a slight deviation from those predictions, with a recorded number of 24 747 cases by March 15, 2020, suggesting that measures introduced by March 11, 2020, began reducing the number of new cases within 3–4 days.

All other European countries appear to be in a similar situation, with just a short time-lag of a couple of weeks (figure). We urge all countries to acknowledge the Italian lesson and to immediately adopt very restrictive measures to limit viral diffusion, ensure appropriate health-system response, and reduce mortality, which appears to be higher than previously estimated, with a crude case-fatality rate of almost 4%.[6]

We declare no competing interests.

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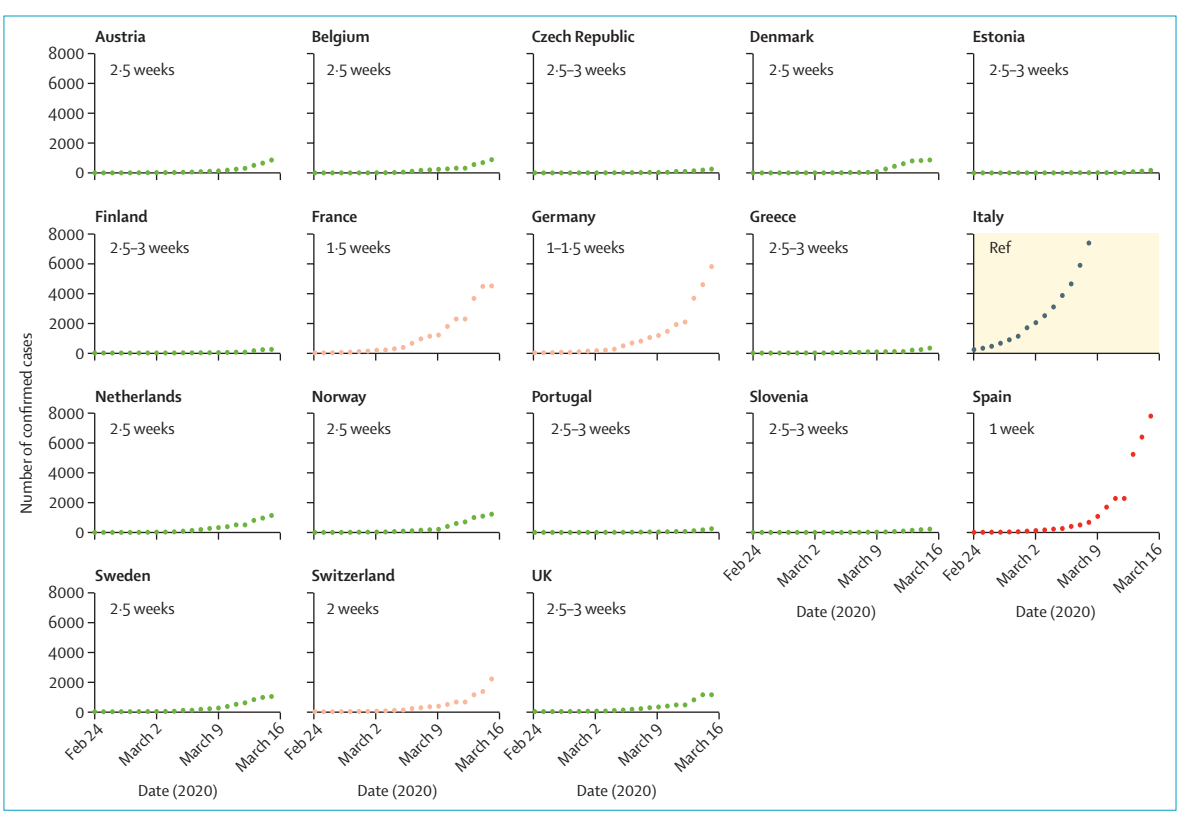

***Figure*: Epidemic curves for European countries, with estimated lag time from Italy's situation, as of March 15, 2020**
Green dots are for countries with more than 2 weeks of lag time from Italy; orange is for countries with 1–2 weeks of lag time; and red is for countries with 1 week or less of lag time. The Italian data curve is cut at 8000 cases to convey easier interpretability of lag times. Source: Center for Systems Science and Engineering, Johns Hopkins University.

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Immunosuppression for hyperinflammation in COVID-19: a double-edged sword?

Mehta and colleagues[1] postulate that hyperinflammation in coronavirus disease 2019 (COVID-19) could be a driver of severity that is amenable to therapeutic targeting since retrospective data have shown that systemic inflammation is associated with adverse outcome. However, correlation does not equal causation, and it is equally plausible that increased virus burden (secondary to failure of the immune response to control infection) drives inflammation and consequent severity (as shown for other viruses[2]) rather than augmented inflammation being an inappropriate host response that requires correction.

The authors hypothesise that approaches such as corticosteroids or Janus kinase (JAK) inhibitors could be considered if hyperinflammation is present.[1] Broad immunosuppression in patients with overwhelming viral illness might be inadvisable. Beneficial anti-inflammatory effects should be weighed up against the potentially detrimental effects of inhibiting antiviral immunity, thereby delaying virus clearance and perpetuating illness. Accordingly, findings from multiple studies in humans and animals indicate that corticosteroid immunosuppression (both inhaled and systemic) impairs induction of anti-viral type-I interferon responses to a range of respiratory viruses,[3,4] effects that are likely to also occur in the context of COVID-19. Selective therapies with JAK inhibitors could be expected to have similar effects. JAK-STAT signalling is a major component of the type-I interferon pathway.[3] Tofacitinib has been shown to inhibit interferon-α production in vitro.[5] Suppression of interferon or other mediators (eg, interleukin 6) could also promote secondary bacterial infection and further complicate the disease course.[3]

The decision to pharmacologically immunosuppress a critically unwell patient with COVID-19 remains a difficult one. Possible beneficial effects of reducing inflammation should be carefully weighed up against the potential for deleterious impairment of anti-microbial immunity.

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Authoritarianism and the threat of infectious diseases


Chris McGrath/Staff/Getty Images

Punitive social policy, encompassing the dismantling of the welfare state with the expansion of the penal state and its associated institutions, as nicely stated by Elias Nosrati and Michael Marmot in their Perspective,[1] might indeed be considered an upstream social determinant of health. Nosrati and Marmot's analysis relates to the findings described by Navarro and colleagues,[2] linking political ideology with policies aimed at reducing social inequalities such as welfare state and labour market policies.

The increasingly punitive policy environment in North America, Europe, and some South American countries (eg, Brazil and Argentina) is probably related to the spread of an authoritarian ideology that has xenophobia at its core.

However, a trait of those repulsion speeches that is often missed is how immigrants or strangers are referred to as parasites or contagious agents. Recent examples include the xenophobic and, in some cases violent, acts committed against Asiatic citizens in Western countries since the coronavirus disease 2019 outbreak began,[3] or Donald Trump's statement that "tremendous" infectious disease is pouring across the Mexican border into the USA.[4] Constant upheavals by the Trump and Orbán[5] administrations about migrants, some of Brexit's collateral effects,[6] and statements about refugees by the Italian politician Matteo Salvini[7] and the Spanish far-right Vox party[8] can be understood in this context.

It is not that foreigners cannot transmit threatening infectious

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