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COVID - 19 – Pandemic or Endemic

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"With multiple species of CoVs circulating in the wild amongst different animal species that may constantly interact with one another, it is likely not a matter of if, but when, the next recombinant CoV will emerge and cause another outbreak in the human population." – 2016

The authors, of this paper titled: Epidemiology, Genetic Recombination, and Pathogenesis of Coronaviruses, would have been surprised to see this prophetic comment transpire into reality so quickly. In 2020 we are amidst a pandemic caused by a novel pathogen unlike other Influenza driven pandemics of the 19th and 20th century. The pandemic has been colloquially referred to as a 'black swan' but it is not so, because it was highly probable and adequately predicted as imminent if not immanent in our highly interconnected society by various epidemiological and infectious disease experts. The authors based their comment on their scientific understanding of Coronaviruses (CoV):

The large genome in CoV allows for extra plasticity in genome modification by mutations and recombinations thereby increasing the probability for intraspecies variability, interspecies 'host jump', and novel CoVs with a high genetic diversity, with unpredictable changes in virulence during human infections to emerge under the right conditions.

Epidemiologists principally classify non-sporadic disease prevalence into endemic (translated from Greek root as: in the people), epidemic (upon the people) and pandemic (all the people). Unbeknownst to the semantic or historical origin of these labels, pathogens continue to replicate, mutate and infect new species across time and geographies. The huge impact of such pandemics when calculated in terms of human morbidity, mortality and longevity is the focus of this article. SARS-CoV-2 (Severe Acute Respiratory Syndrome – Coronavirus-2) is a novel human pathogen and the resultant pandemic has few precedents to guide us into predicting the outcomes. We will attempt to delineate the differences and similarities between this Coronavirus pandemic and past Influenza pandemics to learn about the ebb and flow of such illnesses and what can be the range of potential outcomes for insurers. **The interplay of viral origin, genomic differences, transmission cycle, clinical picture, medical management, vaccine development and public policy response will determine the if COVID-19 pandemic will ebb into epidemic waves and then disappear or will flow low and deep to become endemic with seasons and cycles.**

SARS-CoV-2 is a novel Beta-Coronavirus that is believed to have jumped from animal hosts to humans at the end of 2019 in China. The resultant illness named COVID-19 (Coronavirus disease

2019) is a generally mild flu like illness with symptoms as simple as anosmia (loss of sense of smell) but can also present as a life-threatening respiratory arrest and hence the nomenclature of SARS in the name of the virus. Infection with SARS-CoV-2 can also be completely asymptomatic or be associated with malaise, fever, cough and other flu like symptoms. Generally, the viral shedding starts within 48 hours of infection and may continue up to 4-5 days after resolution of symptoms making it a rapidly propagating outbreak. Asymptomatic carriers have been found to be a major contributor to the spread of the virus and have been linked to multiple super-spreader events where one index case is responsible for transmitting the virus to many subsequent cases. The high transmissibility of SARS-CoV-2 to infect subsequent super-spreaders who further propagate the disease through local and international travel established COVID-19 as a pandemic instead of remaining a local infection cluster.

Human infection with coronaviruses has been reported and researched since the 1960s and SARS-CoV-2 is the 7^{th} virus of the family to infect humans. Four of the coronaviruses (OC43, HKU1, 229E, NL63) cause mild common cold like illness and are responsible for about 15% of common colds in any given season in temperate climates. Three of the coronaviruses (SARS-CoV-1, MERS-CoV and now SARS-CoV-2) cause a potentially life-threatening illness in humans and have distinct identified animal reservoirs from where they 'jump' to cause human infections. SARS was a novel coronavirus whose outbreak in 2002 and 2003 killed 774 people, largely travelers and hospital personnel. It was most contagious when someone was severely ill, making quarantines very effective because by the time someone was most contagious, they were usually already confined to a hospital bed. MERS, which has killed at least 845 people since 2012, is likewise most often transmitted from terribly ill patients. While SARS-CoV-1 and MERS-CoV are not particularly adapted to humans, SARS-CoV-2 has spread to humans via a spillover event or an interspecies host jump from bats/civets and seems to be highly adapted to humans and has high pathogenicity, very high transmissibility and varied virulence. With 1.6 million cases and 600,000 deaths across 213 countries in less than 6 months, SARS-CoV-2 has caused a true pandemic with established excess mortality in many regions of the world. The scale of the COVID-19 pandemic leads it to be compared to the 1918 flu pandemic and posits it to be a 1 in 200 years event.

The 1918 epidemic has been adequately studied and genealogy of the virus established via recent studies from specimens preserved in the Arctic and Alaska. The 1918 pandemic was caused by the Influenza A virus, H1N1 subtype, which along with the 1968 pandemic, H3N2 subtype, is the most predominant virus in circulation till date. The current quadrivalent Flu vaccine offers protection against the H1N1, H3N2, Influenza B/Victoria and Influenza B/Yamagata strains. The current H1N1 and H3N2 are not the exact same strains that caused the pandemic because the Influenza virus undergoes frequent mutations, but they are the same subtypes. The H/N subtypes are further divided into strains depending on the genetic mutations that keep happening as the virus replicates within the cells. The 1918 pandemic was a result of an interspecies jump of novel avian flu virus into new human hosts while the 1957 and 1968 pandemics were the result of reassortment of circulating 1918 virus and circulating avian flu strains. This difference partially explains the extremely severe illness caused by a novel virus (1918 flu) in naïve population and

how historical cross-immunity protects against “reassortment” (1957 and 1968 flu) genetic mutations. Cyclicity of Influenza pandemics is highly controversial marked with conflicting evidence, fragmented theories and incomplete proofs. **Predicting the cycles of pandemics is fraught with a very high degree of uncertainty**. What is certain is that pandemics can emerge either via genetic reassortment or **emergence of novel human pathogens which was the case in the 1918 flu pandemic and seems true for SARS-CoV-2**. The novel antigens of H1N1 influenza virus lacked the shared antigenic history and hence elicited a strong immune response in naïve individuals leading to a “cytokine storm” which led to multisystem disease and a severe respiratory illness with quick progression of disease leading to deaths within 2 days of infection.

These findings are strikingly familiar to the current reports and medical literature of severe outcomes of SARS-CoV-2 infections. The differences are significant too: SARS-CoV-2 leads to a milder illness in most people, no super-imposed/secondary bacterial pneumonia due to better hygiene and sanitation, vastly improved medical management, higher probability of an effective vaccine and strong public health response by the governments across the world. The public health policy has been adopted on various levels by society with wide ranging social compliance, probably dependent on collectivist versus autonomous social structures. The speed of transmission has been higher in current times of air-travel as compared to the 1918 pandemic when most transcontinental spread was by maritime route. The advancement of contact tracing and technology based rapid identification of index and community cases has resulted in being able to use quarantines judiciously minimising the adverse socio-economic impact associated with curtailing civil participation. The globally synchronised efforts to rapidly develop an effective vaccine are indeed driving a confidence in the general public that we will probably have a vaccine as soon as 2021. The guidance and adherence to the policy suggestions of epidemiological models has helped prevent overwhelming the healthcare systems and truly achieved “flattening of the curve”. The rapid genetic sequencing of the virus and globally syndicated sharing of such data has helped identify new strains/clades of SARS-CoV-2 and advance diagnostic and therapeutic research. The treatment for a complex clinical case is guided by evidence-based medicine akin to other viral illnesses and the sufficiency of artificial ventilatory support has indeed saved a lot of lives. The real time sharing of medical literature using electronic media has accelerated the pace of correct management of rare and life-threatening complications of SARS-CoV-2 like pediatric multisystem inflammatory syndrome or stress related cardiomyopathy in adults.

There are a lot of areas that still need to be studied specifically the higher incidence of severe illness in the elderly and those with pre-existing conditions. Vulnerable populations such as prisoners, the homeless and refugees who do not have access to proper healthcare or live in proximity to each other are at higher risk of infection and might become long term carriers and SARS-CoV-2 could become endemic in these populations. There is a slew of second order consequences of COVID-19 wherein people are postponing elective surgeries, avoiding emergency medical care for critical illnesses like angina and myocardial infarction or even stroke leading to worse outcomes like sudden cardiac death. The CDC reported that **in the 10 weeks following declaration of the COVID-19 national emergency, emergency department visits**

declined 23% for heart attack, 20% for stroke, and 10% for hyperglycemic crisis. The reduced cancer screening and delayed detection of breast and colon cancer itself is predicted to cause 10,000 additional deaths in the US over the next decade. Substantial increases in the number of avoidable cancer deaths are to be expected as a result of diagnostic delays due to the COVID-19 pandemic in the UK as well. Polio, measles and other diseases will increase as COVID-19 forces the suspension of vaccination campaigns and the developing and poor African countries will be especially affected. Frequent handwashing, use of masks and social distancing are leading to the reduced spread of routine infections that cause cold and other illnesses and we might possibly see a reduced winter 2020-21 mortality when COVID-19 and the Influenza season merge (syndemic). Some experts argue that the syndemic of Influenza and COVID-19 may overwhelm the healthcare system and cause unprecedented 'flu' mortality.

From the perspective of the insurance and pensions industry the disease has not impacted all socio-economic classes equally and there is a wide variance by region, gender and socio-economic factors. Various data sources show that the current level of excess deaths due to COVID-19 is about 3-7%, dependent on how severely the country was affected by the pandemic. The excess mortality is derived from the general population as a whole and **hence caution needs to be adopted for assessing the impact on insurance portfolios – both protection and longevity.** COVID-19 fatality rates are generally proportional to the overall mortality risk (increasing with age, comorbidities and male gender) and lower and different than the fatality rates for the Spanish flu which seems to suggest that the net impact of COVID-19 to the insured portfolio will be less than the impact to the general population due to the effect of socioeconomic status and prudent insurance risk selection. However, the insurance industry must remain cautious as the medium- and longer-term impact of the measures and restrictions implemented to combat this initial wave of SARS-CoV-2 remain completely unknown and one could justifiably predict mortality effects in either direction. Further-more the pandemic and post-pandemic transmission dynamics of SARS-CoV-2 itself will depend on factors including the degree of seasonal variation in transmission, the duration of immunity, and the degree of cross-immunity between SARS-CoV-2 and other coronaviruses, as well as the intensity and timing of control measures. Based on the current scientific knowledge published in various trustworthy journals it is plausible that SARS-CoV-2 pandemic will continue in near foreseeable future and might turn endemic with seasonal variation in prevalence till a significant percentage of population develops immunity with either vaccines or infections. On the brighter side, it might be possible that social distancing, hand hygiene, and masks at a high degree of compliance (e.g. Hong Kong) may lead to reduction in overall impact of SARS-CoV-2 and other viral infections as well.

In summary this is neither the first nor the last pandemic that we confront, and what we are trying to achieve with any projection is to solve a jigsaw puzzle which not only does not have a reference image but also does not have all its pieces.

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