managing these patients at this time. In addition, we only sampled once from the eye of each patient, which can decrease the prevalence owing to false-negatives. Regardless, these preliminary results are shared in an effort to inform ophthalmologists and others around the world regarding ocular symptoms with COVID-19.

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Invited Commentary
Humans, Viruses, and the Eye—An Early Report From the COVID-19 Front Line
Alfred Sommer, MD, MHS

Viruses have always been part of the human experience, most often in a delicate balance, otherwise the virus and its human host would become extinct. But this relationship is not particularly stable. Like all living substances, viruses mutate, sometimes in ways that cause explosive, severe disease. Viral outbreaks have killed off virtually entire populations, as happened when measles was first introduced into the previously naive population of the Faroe Islands, or when Europeans brought smallpox to North America. Human-kind, as a whole, has survived evolving viral threats by evolving with them; prior exposure to related viruses produces varying degrees of resistance (immunity) to new strains. Humans with less resistance die, often despite modern medical intervention, while others remain entirely asymptomatic. Medical interventions (principally vaccines) and stringent public health measures have often altered the outcome, but not necessarily in predictable ways.

The last “great” pandemic was the 1918 influenza pandemic that killed an estimated 20 to 50 million people worldwide. Its spread was enhanced by troop movements during World War I. The young, perhaps because of their more vigorous immune response, were disproportionately affected, dying from exuberant pulmonary exudation.1

As I compose this article, in mid-March 2020, the World Health Organization has just proclaimed the latest outbreak, termed novel coronavirus 19 (COVID-19), a global pandemic, the president of the United Sates has declared a national emergency, and all of Italy is locked down to contain its spread. Its future course and duration remain unknown. Estimates of case fatality range from 1% to 5% (10-50 times the mortality rate of seasonal influenza, although the real rate will only be known once we conduct serosurveys to determine the frequency of asymptomatic infections) and the elderly are at the greatest risk of severe disease and death. Modern air travel facilitated global dissemination within a very few months of its origins in Hubei province, China. This issue of JAMA Ophthalmology brings us necessarily preliminary but valuable insights from the front line.2 Wu et al2 examined the conjunctiva of 38 patients hospitalized in Hubei province, China, with presumed COVID-19. Conjunctivitis was present in 12 (32%) and it was most evident and severe in the sickest patients. The virus was...
detectable on swabs of the conjunctiva of 2 of 11 patients tested (18%). These 2 also yielded positive nasopharyngeal swab results (100%), whereas only 28 of the 38 patients (74%) did. No other ocular parameters were investigated.

The primary importance of this finding is epidemiologic: it confirms other reports that the virus can invade the conjunctiva, which might, in turn, serve as a source of its spread. Containing viral spread is the primary means by which we protect people and populations from newly emerging infections. Seasonal influenza is minimized (although rarely contained) by a global system that tracks emerging new strains and uses these to develop better-matched vaccines (of variable efficacy).3 The 2003 SARS outbreak, caused by another coronavirus, was a bullet we dodged. Highly lethal, far more so than COVID-19, it was not nearly as communicable. This explains why those at highest risk of infection were hospital workers, who were in close contact with infected patients, and why vigorous containment procedures were able to mitigate what might otherwise have become a disastrous pandemic.

China, where the virus first transferred from its natural animal reservoir (generally thought to be bats4) to humans, experienced an explosive epidemic that strict quarantine measures largely contained (with more than 80,000 clinical cases and 3,180 deaths5). But the rest of the world did little to anticipate and contain its introduction and local spread until it became a pandemic. Effective containment requires an understanding of a virus’s mode of transmission and rapid and vigorous use of appropriate interventions designed to stop it. Unfortunately, this is a lesson we keep forgetting.

Biomedical science has provided us increasingly effective antimicrobial defenses from antibiotics to vaccines. But microbes continuously evolve; our defenses must evolve with them. The power of vaccines and antimicrobials repeatedly elicit a false sense of security: in 1967, then-US Surgeon General William H. Stewart allegedly assured Congress, “It’s time to close the book on infectious disease.”6 We keep making the same mistake: underestimating the threat of new infectious agents and failing to implement public health interventions as rapidly and vigorously as required.

Ironically, the earliest cries of alarm about COVID-19 were made by Dr Li Wenliang, a Chinese ophthalmologist caring for patients in Wuhan. He earned the ire of the Chinese government for alerting the public and calling for action. He died at age 34 years from the disease.7 We do not know whether he became infected from contact with patients’ eyes.

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