for part of the time up to 9 months of age. After the booster, the levels are similar, so herd immunity is likely to be similar. Therefore, the authors conclude that the 6 week, 14 week, and 9 month schedule is superior, but that the 6 week, 10 week, and 9 month schedule might be used where programmatic issues dictate, as the herd effects would be similar. The 6 week, 10 week, and 9 month schedule might leave infants more susceptible to some serotypes, but this would soon be covered by herd immunity. If countries were to introduce this schedule with poor coverage and therefore limited herd immunity, this could lead to vaccine failures in children yet to receive a booster dose.

The study rationale was based on the perception that caregivers would not accept the administration of multiple injections at a single visit.[10] A systematic review found high caregiver acceptance of multiple injections in a single visit, even when concerns were expressed about the number of vaccine injections children received.[11] Countries need to balance concerns such as this against the superior immunogenicity seen when the two primary doses of PCV are separated by 2 months rather than 1 month.

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Socioeconomic disparities and infection: it's complicated

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The associations between low socioeconomic status and poor health have long been appreciated and are supported by a vast body of evidence.[1] Although this overall relationship remains robust, in *The Lancet Infectious Diseases*, Alessandro Pini and colleagues[2] show that the association between markers of low socioeconomic status and incidence of infectious diseases in Sweden, a high-income country, is not at all straightforward. Pini and colleagues[2] used 10 years of Swedish national notification data to study the association between 29 infectious diseases and socioeconomic characteristics. They used a matched case-control design and analysed the data of more than 1 million people aged 18–65 years.

Pini and colleagues[2] grouped their findings into three patterns. The first pattern supports the overall premise of the social determinants of disease—people with markers of lower socioeconomic status had higher disease incidence. This association was seen for invasive bacterial infections, hepatitis B, hepatitis C, tuberculosis, and antimicrobial-resistant infections. These infections are the ones that could be affected by public health campaigns to address social determinants of disease. The second pattern was the opposite—ie, markers of lower socioeconomic status were associated with lower disease incidence. Diseases in this group included food-borne and water-borne diseases and dengue. The authors postulate that this pattern could be partly

attributable to different risk behaviours of people with higher socioeconomic status, including international travel and consuming restaurant-cooked or higher-risk foods (eg, rare beef and shellfish). A systematic review of food-borne disease in high-income countries also noted an association between higher socioeconomic status and higher incidence of salmonellosis and campylobacteriosis.[3] The third pattern, which was perplexing, was that a group of infections showed inconsistent associations between markers of socioeconomic disadvantage and disease incidence.

This study has several strengths, most notably the use of 10 years of population-level data involving a laudable 170 000 cases notified with 29 infectious diseases and 850 000 controls, an interesting topic highly worthy of examination, and the ability to match individual-level socioeconomic data for cases with infectious diseases and controls. This avoids potential misclassification bias associated with using area-based markers of socioeconomic deprivation.

One weakness of this study is the age restriction of 18–65 years, resulting in exclusion of one in three notified cases. The authors excluded paediatric, adolescent, and older populations because not all the selected socioeconomic status indicators (eg, employment) were relevant to, or available for, these age groups. That this study was unable to cover the entire population is a shame, given that children and older people have the highest incidence of many of the diseases included.

Another omission was analysis of disease incidence among Indigenous population (eg, the Sami population) compared with non-Indigenous populations. In 2017, we published an analysis of sociodemographic risk factors for infectious diseases in Australia and showed a substantially increased risk among Aboriginal and Torres Strait Islander people compared with the non-Indigenous population.[4] In the USA from 2007 to 2011, notification incidence was higher among American Indians and Alaska Natives than in people of white ethnicity for 14 of 26 notifiable diseases analysed.[5] Although the majority of the literature shows poorer health and social outcomes for Indigenous populations, a recent review reported that life expectancy at birth did not differ between Indigenous and non-Indigenous populations in Sweden, which contrasts with a gap in life expectancy at birth of more than 5 years for Indigenous populations in Australia, Cameroon, Canada, Greenland, Kenya, New Zealand, and Panama.[6] It would be interesting to know if the more equitable health outcomes for Indigenous people in Sweden extend to notifiable infectious diseases. Another question that was not answered in Pini and colleagues'[2] analysis is that of temporal trends: are inequities in infectious-disease notification incidence increasing (as has been noted in New Zealand) or decreasing (as we noted in Australia) over time?[4,7]

Despite these limitations, this important work highlights diseases for which inequities exist even in a high-income country with a universal health-care system, and which should be a focus of public health intervention. The approach used was feasible because of the ability to link individual-level data from a range of datasets via the personal identity number assigned to all residents staying at least 1 year in Sweden,[8] thereby providing an inexpensive and reproducible approach. The findings suggest that education, employment, and household income may affect health outcomes via different pathways. The results provide baseline data against which the impact of public health action to address inequity in infectious diseases can be measured.

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