Tobacco smoke exposure in early life: the first African cohort studies

TOBACCO SMOKE (TS) is a leading cause of mortality, responsible for around 6.1 million deaths and for morbidity that causes at least 143.5 million disability-adjusted life years worldwide per year. Maternal smoking is an important risk factor for adverse maternal and foetal outcomes, and environmental TS exposure pre- and post-birth is increasingly recognised to cause childhood illness and developmental delay. In this issue of the Journal, the article by Vanker et al. is one of the very few collaborations to report on maternal smoking patterns in a low-to-middle-income country. They go further, and have enough reliable data to relate maternal smoking to infant birth outcomes. This setting is all the more relevant as it is becoming a new battleground for tobacco companies who are shifting their emphasis due to taxation and legislation in high-income countries.

The authors are to be congratulated in several ways; although we are not sure of the refusal rate, they provide unique cross-sectional data comparing two well-described local populations of mixed race and predominantly black African communities, suggesting clear differences in smoking prevalence between the two. They are unique in comparing the sensitivity of self-reported versus objective (validated) smoking status using urinary cotinine in both populations. Although relatively expensive compared to exhaled carbon monoxide (especially on this scale), urinary cotinine is a better validation method here as it will not be affected by burning of domestic fossil fuels and, of course, can be collected from newborns without much cooperation! The authors provide rational explanations for the discrepancies in the sensitivity of self-reported versus validated smoking status between the populations, and place them within a live socio-cultural context. The study methodology is strengthened by the fact that all births were recruited from a single hospital so all dyads have identical obstetric services, and, moreover, the ethical element was maintained, as all smokers were offered quit counselling. A member of the study team was present at all 792 births to collect questionnaires and maternal/newborn urine directly. The team maintained a remarkable 91% follow-up rate to repeat the questionnaires and urine testing at 6–10 weeks post-birth; this in itself is no mean feat in any health system, let alone following young women going through chaotic changes in their lives.

One important finding was that high levels of urinary cotinine were more common in infants than their mothers, suggesting high rates of smoking in other household contacts. Environmental TS will be combined in many houses with high levels of pollution from domestic fuels which are increasingly associated with adverse respiratory outcomes, including later COPD. Finally, there was enough complete data to properly correlate maternal smoking status with an adverse birth outcome, which showed the predictable association with decreased infant birth-weight for age Z-score. The study was large enough with enough follow-up data to allow multi-regression to reduce the effects of confounders.

It is vital that this well-described cohort be followed through early years to adulthood, hopefully with longitudinal lung function and clinical outcomes. I hope the Drakenstein Child Health Study will continue to reveal rich data and contribute to our understanding of public health for low- and middle-income countries as much as landmark cohorts such as the Lung Health Study or Framingham have done for high-income countries. It can supply valuable ammunition for those governments that need it in their battlegrounds.

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References