higher CD4 thresholds remains unclear but enthusiasm for that approach is likely to wane.[5,6] One recent large randomised clinical study of starting and stopping at CD4 cell counts above 350/μL revealed only slightly increased risk of minor HIV-related complications in the interruption arm (Staccato; table).[7]

The implications for the individual management of antiretroviral therapy level are less clear. Because of the Trivacan and SMART studies, the most recent US Department of Health and Human Services guidelines state that treatment interruptions should be avoided in clinical practice and should only be done in a closely monitored clinical trial.[8] However, this is not practical because many patients in clinical practice have significant side-effects, pill fatigue, or treatment failure. Presumably any study that specifically focuses on those with strong reasons to stop therapy might show a benefit (or at least a lack of harm) in going on and off therapy.

The fundamental challenge raised by Trivacan and SMART is how to translate data from broad heterogeneous study populations to an individual. For patients who are doing well on a stable regimen, the Trivacan and SMART data clearly indicate that uncontrolled HIV replication is more harmful than modern treatment regimens, and that well-tolerated drugs should be continued indefinitely. This finding is not surprising and was widely accepted even before these studies were done (at least it pertains to regions where treatment is widely available). At what point the harm associated with treatment in an individual outweighs the harm associated with uncontrolled HIV replication requires a careful understanding of treatment-related side-effects, HIV pathogenesis, and the consequences of interrupting therapy. The Trivacan and SMART studies provide a quantitative risk assessment of the consequences of stopping therapy and are therefore important studies for all treating clinicians. The task at hand now is to ensure that all patients—including those in resource-constrained regions, such as West Africa—have access to well-trained health-care providers who can readily adopt emerging data into any decision regarding when (if ever) to stop antiretroviral therapy.

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Delayed cord clamping increases infants' iron stores

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It is startling to see how a seemingly insubstantial change in practice might affect long-term results for infants. When a medical text recommended immediate cord clamping in 1913, science was in its infancy and expert opinion guided practice. Today, we have the benefit of scientific evidence to advise our actions. The article in today's *Lancet* by Camila Chaparro and colleagues[1] provides additional weight to the growing evidence that our haste to clamp the umbilical cord and pass the baby off is ill-advised. The mounting evidence that delayed cord clamping benefits both term and preterm infants continues to build.[2,3]

Chaparro and colleagues' international interdisciplinary study with 358 randomised infants shows that waiting 2 min before clamping the umbilical cord provided the infants with more body iron at 6 months of age without causing any harm at birth. The rationale for the study—the link between iron deficiency in infancy and neurodevelopmental delays—is well founded. The number of infants in the study was ambitious and the protocol for the 6-month follow-up was meticulously planned. The exclusion criteria were carefully selected to rule out women with conditions that negatively affect neonates.

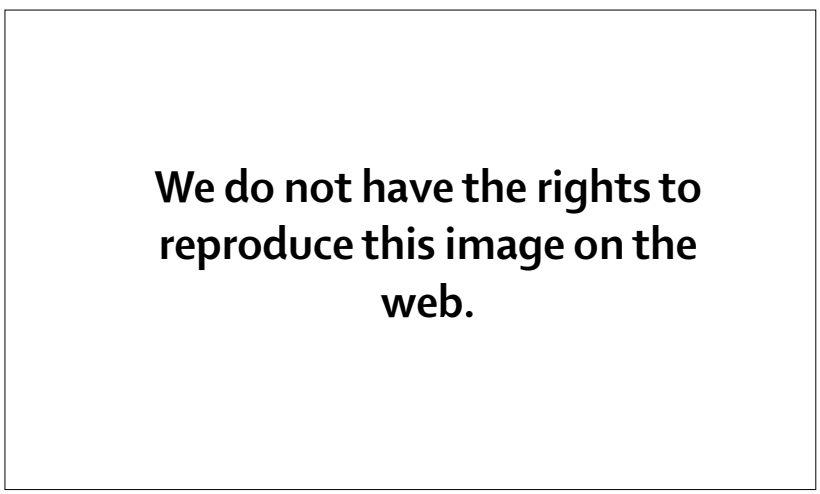
Most importantly, the team used multiple methods for identifying the main outcome variable of iron status at 6 months of age. There were no differences between the groups of infants on the concerns about harm: neonatal jaundice or polycythaemia. The protocol required that obstetricians and midwives wait up to 2 min to clamp and cut the umbilical cord. Studies in which cord clamping was delayed for 3 min or longer also failed to show harm with the assigned delay in cord clamping.[2]

Infants with delayed cord clamping in Dewey and colleagues' study were held level with the mother's body during the 2-min wait. Lowering the infant can speed the transfusion.[4] In our randomised trial of a brief delay (30–45 s) in cord clamping at preterm births, we lowered the infant below the introitus or incision to hasten transfusion.[5] However, if one places the infant on the mother's abdomen briefly before cutting the cord, the infant might receive less placental transfusion.[6]

The results of Chaparro and colleagues' study suggest that a modification might be indicated in the active management of the third-stage programme for developing countries, in which anaemia in infancy is often endemic. Active management of the third stage prevents maternal haemorrhage after birth. As defined initially and researched, the process involved three steps: (1) a drug is used to contract the uterus; (2) the cord is clamped immediately; and (3) traction is applied to the cord to speed delivery of the placenta. The most important part of active management is getting the uterus to contract to avoid uterine atony. There is a fear that giving the drug to contract the uterus (uterotonic) with the infant still attached to the umbilical cord would cause "overtransfusion." Yao and co-workers showed that giving such a drug and delaying cord clamping did not result in overtransfusion because the blood volumes of the infants in this study never went above 90 mL/kg, even with a 5–6-min wait to clamp.[7] Two of the large studies on active management of the third stage reported that infants who had immediate cord clamping weighed substantially less than the infants who had delayed cord clamping.[8,9] We believe that more research on the use of uterotonic drugs while leaving the cord intact is essential.

Chaparro and colleagues' study increases concern about the ethics of routine cord-blood collection without adequate informed consent and the advertisement of cord blood as "medical waste." It is very plausible that those magical stem cells found in cord blood have multiple protective and preventive roles to play in an infant's developing body.[10] We support the American Academy of Pediatrics' statement which does not recommend cord-blood collection and storage unless there is a known family need.[11] However, that valuable cord blood should not be medical waste—let the infants have what they need first.

This excellent study by Chaparro and colleagues, the largest ever done on delayed cord clamping with long-term follow-up, adds important evidence in favour of delayed cord clamping at the births of term infants.



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Racism, socioeconomic deprivation, and health in New Zealand

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Inequalities in health are deplored in modern democratic nations and equal opportunities are extolled in principle, if not in practice. Through the caste system, slavery, colonisation, aristocracy, apartheid, and Nazism, inequalities were institutionalised. The historical legacy of racism and economic inequality of opportunity casts its shadow even now, and the study by Ricci Harris and colleagues[1] in today's *Lancet* explores the idea.

Inequalities in health status, disease occurrence, and mortality are shaped by accumulated wealth, material circumstances, environmental quality, nutrition, a wide range of personal behaviours, genetic inheritance, and health services. Within multiethnic societies, European-origin White populations (henceforth, "White" as per Bhopal's glossary[2]) are characterised by being richer, more powerful, and enjoying better material circumstances, environmental quality, and health services than non-White ethnic-minority populations. Ethnic-health inequalities in such societies are inevitable.

Racism is the belief that some racial, ethnic, religious, and cultural groups are better than others. This belief, combined with power, leads to actions favouring the supposedly superior groups. The power to enact such beliefs is, currently, mostly in the hands of White populations. Some like to believe racism is unimportant in modern, industrialised, multiethnic societies, others believe that racism is at the heart of ethnic and racial disparities in health and health care.[3] We need data to progress our understanding.

Equity is the core ethical principle underpinning equality of health care. It is based on fairness and justice. Most research studies present data from the White population as the standard against which to compare minority groups. Not every inequality shown is an inequity. Ethnic variations in smoking, for example, are not necessarily inequitable but such variations in access to smoking cessation services would be. Differences in life expectancy in different ethnic groups are usually inequitable, because they mainly result from other social injustices. Epidemiological data are essential to the identification of inequities, and monitoring effectiveness of interventions to redress them.

Harris and colleagues' study is of special interest, not only because empirical research on racism and health is rare outside the USA, but because it concerns the Māori population, an Indigenous ethnic minority. The term indigenous is usually used to mean a population belonging naturally to a place in the sense of long-term ancestral origins—eg, Aborigine (it might also mean the majority population—eg, in the UK—as an alternative to the word White).[2]

Indigenous populations across the world have poor health, and many were decimated and demoralised in the colonial era. Compared with other Indigenous populations, including Native Americans and Australian Aborigines, the Māori population largely escaped such a fate. By contrast with other colonised Indigenous and migrant ethnic minorities, Māoris are perceived to be highly politically and socially organised, empowered, and in control. This perception, at least in part, is related to the negotiated Treaty of Waitangi of 1840 that enshrined Māori rights, and that still plays a major role in governing relations between Māoris and other New Zealanders.[4] Nonetheless, Māori health is comparatively poor.

Bramley and colleagues[5] compared the health inequalities in New Zealand and the USA, with the European (effectively White) and White population, respectively, in each of these countries as the point of comparison. They used a wide range of health and health-care indicators. The inequalities were massive—eg, life expectancy in Māori men was 8·9 years less than in European men in New Zealand, larger than the 7·4 year difference between American Indian or Alaskan native and White men in the USA. In virtually every indicator, inequalities were considerable, and greater in New Zealand than in US comparisons. The life-expectancy deficit in Australian Aboriginal and Torres Strait Islanders is closer to 20 years.[6] By contrast, mortality differences between