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See Online for appendix

***Panel:* Research in context**

Continuous efforts have been made in improving child mortality estimation since the publication of GBD 2010.[18] In this study, significant improvements have been made on several fronts. First, we employed a mixed effects model to adjust non-sampling data biases with source-type specific fixed effects across all countries and source-specific random effects within country. We selected one specific data source in each country as the reference source, calculated the difference in the summed fixed and random effects between other sources and the reference source, and subtracted this difference from each non-reference source to adjust for data bias. In the case that multiple sources were selected as the reference, we took the average value of the selected sources. More than 300 all-cause mortality experts from around the world contributed to the selection of the reference data sources. Second, we used a non-linear mixed effects model to more accurately capture the functional form between child mortality rate and other factors including HIV/AIDS. This has significant implications for the estimation of child mortality in the most recent time period in which data are sparse and covariates have a more pronounced effect on final estimates. Third, we improved our mortality estimation strategy for neonatal deaths. The new strategy we employed accounted for the fact that few children die from HIV in the neonatal age group, and helps improve our estimated age distribution of deaths in children under 5.

robust, and do not overlap in only eight of 188 cases. Continued improvements in methods and data availability, especially for recent years, make the assessment of trends comparatively unstable. The correlation between UNICEF annual rates of change from 1990 to 2007, published in 2009, and in 2013, is 0·79. The correlation between this study and Rajaratnam and colleagues[13] is 0·82. Improvements in methods and data are to be encouraged, but these perhaps surprisingly modest correlations mean that the public health community should be cautious in over-interpreting trends.

This analysis has many limitations. First, we attempted to explicitly model the non-sampling error that affects different surveys in each country (panel). This approach avoids estimation of false trends due to compositional bias in the data available for a given year but depends on the validity of the estimates of non-sampling error. Unfortunately, external validation of this process is not possible except in countries with complete vital registration systems, but most of these countries do not collect summary or complete birth history data. Second, the trend for the most recent years is a short-term estimate for many countries. Our estimates might be too high or too low in these cases and the Gaussian process regression appropriately generates widening uncertainty intervals for them. However, time lags between data collection and inclusion in our synthesis are shortening for many countries. For example, we included results from the sample registration system in India to 2012, and also data for China through to 2013. Third, in our analysis of the factors contributing to under-5 mortality change in each region, we included country random effects and fixed effects on year interacted with region. We might have underestimated the contribution of local policy and health-system organisation if these changes are associated over time within a region. Fourth, although we systematically searched and identified sources of data for under-5 mortality, we probably did not identify all data sources. The large set of collaborators from 100 countries who participated in GBD 2013 has helped to identify new sources and assess the quality of existing data, but this information base can be expanded in the future. Fifth, we used the Shapley decomposition method to parse out the contribution of different factors to changes in under-5 deaths. This method, although computationally intensive, is intuitive. Although other methods have been proposed to decompose effects of different factors on indicators of interest, Shapley value decomposition, to our knowledge, is most suitable in our application.[86,87]

The vigorous debate on setting development goals for the post-2015 era is predicated on the belief that global goal setting and quantitative monitoring can catalyse change. The acceleration of decreases in under-5 mortality beyond that expected on the basis of income, education, and the secular trend, especially in some sub-Saharan African countries, coincides with the MDG era and increased investments in these countries in health and social development programmes by various donors. As the end of the MDG era approaches, the global public health community might better serve the needs of countries by focusing on the accelerated decreases after 2000 reported here, rather than on which countries will achieve the arbitrary but seemingly useful targets set by the MDGs. Galvanising political commitment to ensure life-saving technologies are implemented will be crucial. The essential health intelligence that comes from large global monitoring efforts such as the GBD study will better focus attention on countries where progress has been disappointing. The consequences of not doing so—more than 3 million preventable child deaths in 2030—would be a scathing indictment of the failure of the donor, research, and international development community to collectively build on the impressive reductions in child mortality that we have come to expect.

Contributors

CJLM, ADL, and HW conceived of the study and provided overall guidance. HW, CAL, MMC, CEL, AES, HA, MI, and LS analysed child mortality data sources. CJLM, ADL, HW, CAL, and MMC reviewed each cycle of estimation in detail. HW, CAL, MMC, MDM, CEL, AES, BP, CJLM, and ADL prepared the first draft. HW, ADL, CJLM, CAL, MMC, MDM, CEL, and AES finalised the draft based on comments from other authors and reviewer feedback. All other authors reviewed results, provided guidance on the selection of key data sources, and reviewed the paper.

Declaration of interests
The authors alone are responsible for the views expressed in this article and they do not necessarily represent the views, decisions, or policies of the institutions with which they are affiliated. BDG works for AMP, which receives grant specific support from Crusell, GlaxoSmithKline, Merck, Novartis, Pfizer, and Sanofi Pasteur. JAS has received research grants from Takeda and Savient and consultant fees from Savient, Takeda, Regeneron and Allergan. He is a member of the executive of OMERACT, an organisation that develops outcome measures in rheumatology and receives arms-length funding from 36 companies; a member of the American College of Rheumatology's Guidelines Subcommittee of the Quality of Care Committee; and a member of the Veterans Affairs Rheumatology Field Advisory Committee. GAM is required to include the following statement: The views expressed in this article are those of the authors and do not necessarily represent the views of the National Heart, Lung, and Blood Institute, National Institutes of Health, Department of Health and Human Services, or any other government entity. We declare that we have no further competing interests.

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Trends in perinatal mortality and its risk factors in Japan

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Background

The perinatal mortality rate (PMR) has decreased rapidly in Japan since the 1950s. Perinatal death consists of fetal death (stillbirths after 22 weeks of gestational age), or early neonatal mortality (ENM), which occurs within 7 days after birth, and reducing the PMR requires action on both stillbirths and ENM. This study aimed to: 1) provide the most up-to-date estimate of the trend in perinatal mortality, and 2) identify its risk factors.

Figure 1. Trend in perinatal mortality rate by sex.

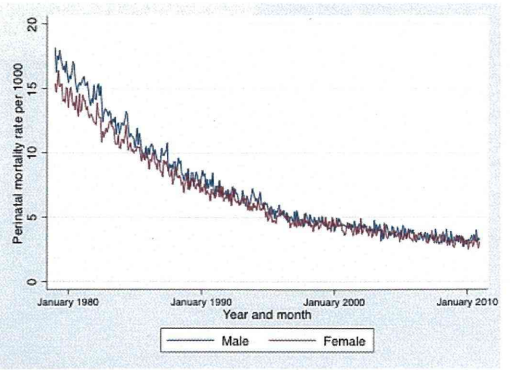

Table 1. ARIMA time series analysis of perinatal mortality rate by sex.

	Risk ratio	95% CI	P value
Male			
Rate ratio (Annual)	0.949	0.936 – 0.961	<0.001
AR (1)	-0.128	-0.571 – 0.316	0.573
Female			
Rate ratio (Annual)	0.950	0.940 – 0.960	<0.001
AR (1)	-0.036	-0.557 – 0.486	0.894

Methods

We used a full dataset of singleton mortality records from the Japan national vital registration system for the period 1979 - 2010. We conducted an ARIMA time series analysis of the annual PMR, by sex, from 1979 to 2010. Risk factors for perinatal mortality were analyzed using multi-level Poisson regression with a random effect for prefecture.

Findings

Between 1979 and 2010 there were 40,833,957 pregnancies, and 355,193 perinatal deaths. We found an annual decrease in PMR of 5% (95%CI: 4 – 7%) for both sexes, adjusting for serial dependence (Table 1). Key perinatal mortality risk factors are shown in Table 2.

Table 2. Multilevel regression model of key risk factors for perinatal mortality

	Risk ratio	95% CI	P value
Birth weight			
Normal (2500 – 4000g)	1.00		N/A
High (>4000g)	2.51	2.20 – 2.85	<0.01
Low (2000 – 2499g)	4.39	4.19 – 4.60	<0.01
Very low (1500 – 1999g)	5.72	5.39 – 6.08	<0.01
Extremely low (<1500g)	4.16	3.94 – 4.41	<0.01
Maternal age			
25-29	1.00		N/A
15-19	0.94	0.88 – 1.01	0.08
20-24	1.07	1.03 – 1.11	<0.01
30-34	1.09	1.06 – 1.12	<0.01
35-39	1.39	1.34 – 1.44	<0.01
40-49	1.90	1.79 – 2.01	<0.01
50-63	1.55	0.39 – 6.18	0.54
Gestational age			
Term (37 – 41 weeks)	1.00		N/A
Premature (<37 weeks)	2.68	2.56 – 2.81	<0.01
Post mature (>41 weeks)	4.25	3.81 – 4.73	<0.01
Household occupation			
Large company	1.00		N/A
Farmer	1.36	1.28 – 1.45	<0.01
Self-employed	1.30	1.24 – 1.35	<0.01
Small company	0.89	0.87 – 0.92	<0.01
Casual/other	1.24	1.20 – 1.29	<0.01
Unemployed or unknown	1.64	1.56 – 1.72	<0.01

Interpretation

We identified a constant annual percentage decline in PMR. Postmature neonates were at higher risk of death, as were the infants of older mothers. To continue to reduce the PMR, further targeting of risk factors is needed.

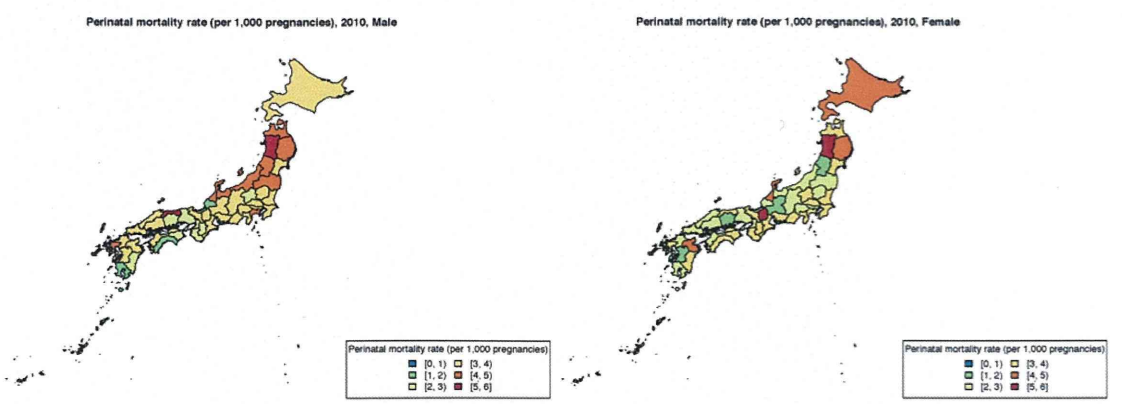

Figure 2. Subnational mapping of perinatal mortality rate (left: male, right: female)

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