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Rejoinder

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We would like to thank Drs. Ruppert, Carroll, Cook and Stuart, for their insightful and constructive comments. Their discussion has provided new insights into our proposed approach. We are in agreement with all their points. Below is a summary of our comments in response to their suggestions.

Drs. Ruppert and Carroll correctly pointed out that our measurement error analysis, aimed at predicting the birth weight $W_i(0), W_i(1)$ for the babies that had their weights measured after the 72 hours, relies on parameter estimation outside the Gibbs Sampling. We agree that our approach might underestimate the uncertainty. However with 800 observations, we believe that the linear regression model for the pairs of points $(W_{t_i}(z), t_i)$ is estimated well. The authors proposed an elegant alternative, a heteroskedastic measurement error model, which is consistent with a full Bayesian analysis. We applaud the authors for such an ingenious idea. Visual inspection of the cross-sectional data (Figure 2) indicates that heteroskedasticity might not be a major issue. However, their approach is still challenged by the lack of longitudinal data on the birth weight, and as in our formulation, it must rely on informative prior assumptions or additional data sources.

The authors introduced a regression model for the birth weight that is consistent with our approach and that facilitates the elicitation of the prior value for ρ. Thank you! In fact, if there is no interaction between infants and treatment, then $W_i(0) = W_i(1)$ and therefore $\rho = 1$. Therefore we agree with the authors that ρ may be even higher than the correlation between successive children with the same mother or even identical twins. At the other end, we think that it is unlikely for ρ to be negative. It is plausible to assume that the between infants heterogeneity ($\sigma^2_{w,1}$) will be larger (and not smaller) than the between infants heterogeneity on how they respond to the treatment. Regardless, the authors have provided a nice alternative way of thinking about this problem. We also agree that we could have used an informative prior on the non-identified nuisance parameters such as (ρ, ψ). We have just preferred to show the sensitivity of the results to alternative choices of ρ and ψ. Again, we agree with the authors that the use of penalized splines could have been a valid alternative.

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We agree with Drs. Ruppert and Carroll that it is important to separate the inferences that depend on the counterfactuals and therefore that depend on ρ and ψ from those that do not. Figures 4 and 5 show the marginal posterior distribution of the percentile-specific treatment effects on birth weight (Δ_p^W) and on mortality (Δ_p^Y) under several model specifications. The parameter Δ_p^W is defined as $Q_1(p) - Q_0(p)$ where $Q_1(p)$ and $Q_0(p)$ are the quantile functions of the marginal distributions of $W_i(0)$ and $W_{i'}(1)$ respectively and therefore does not depend on ρ. The parameter Δ_p^Y defines the difference in the probability of death between treated and non-treated infants who are at the same percentiles of their respective birth weight distribution. Thus Δ_p^Y is not a causal parameter, because these differences correspond to two different subpopulations of babies.

The results in Figure 6 are the only ones that show posterior distributions on causal parameters which therefore depend upon comparisons of counterfactuals for the same baby. The most important result here is the one indicating that for the stratum of the low birth weight and a large causal effect of the treatment on the birth weight $W(0) < 2500$ & $W(1) - W(0) > 50$), there is a beneficial effect of the treatment on survival. We believe this result is real under the unverifiable assumption that $0 < \rho < 1$. Under the model (2) proposed by the discussants, $0 < \rho < 1$ if we are willing to assume that: 1) infants respond differently to the treatment ($\sigma^2_{w,2} > 0$); and 2) the between infants heterogeneity will be larger (and not smaller) than the between infants heterogeneity on how they respond to the treatment ($\sigma^2_{w,2} < \sigma^2_{w,1}$), as is plausible. Finally, we agree with the discussants that the results on Figure 6 are robust to the choice of (ψ, ρ) probably because the identified part of their regression model (2) ($\boldsymbol{x}_i^T \boldsymbol{\beta}_x + z^* \boldsymbol{x}_i^T \boldsymbol{\beta}_{zx}$) dominates the subject-specific component ($w_{i,1} + z^* w_{i,2}$).

We would like to thank Drs. Cook and Stuart for providing a clear and insightful description of post-stratification and on the interpretation of causal and population average parameters. In their discussion they wrote "it strikes us as odd, however, that the principal strata are defined by the absolute value of the causal effect of the supplementation on birth weight". We agree and in fact in the revised version of the paper we have considered the difference $W_i(1) - W_i(0)$. They also comment "For clinical practice, it would also be interesting to know what proportion of the population falls into each of the four principal strata.". We also agree and we have added this result in Figure 7 of the paper. We are glad to see that our methods can be extended and applied to other areas. We found the potential applications and extensions of our approach illuminating.