The Overall Effects of the Ebola Outbreak on Birthweight

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Abstract

This paper examines the relationship between the overall effects of the Ebola outbreak on birthweight, using 4,185 women living in Sierra Leone. To identify causal effects, I assume that the respondents didn't plan their pregnancies around the crisis. I find that the crisis lowered birthweight by 0.042 (SE = 0.017) kilograms at 5% significance. The average birthweight in the control group is 3.223 (SD = 0.522) kilograms. This amounts to a 1.300% change in birthweight from the Ebola outbreak. In addition, I tested if urban or rural areas would have differential effects. With the interaction term I find that birthweight decreased by 0.034 (SE = 0.019) kilograms at 10% significance. This paper demonstrates that the Ebola outbreak is suggestive of effects on fetal health.

Keywords: Sierra Leone, West Africa, *Zaire ebolavirus*, fetal origins hypothesis, epidemic, panic, difference-in-difference

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I. Introduction

The importance of development during the fetal period is well established. Many aspects of development may affect the child years down the road. Development of the brain, metabolism, and cardiovascular system are very sensitive during the fetal stages. Birthweight is a common measure used as a simple proxy for the health of the fetus (Almond & Currie 2011).

The Ebola crisis was a major event for the residents of West Africa. The disease spread rapidly and killed quickly, and its rise caused worldwide panic. Sierra Leone was one of the main centers of the crisis. There were multiple lockdowns, many districts put under quarantine, and the health system was completely overloaded.

This paper examines the effects of the Ebola crisis on the birthweight of the child exposed in utero. I use data from the Multiple Indicator Cluster Survey (MICS) in Sierra Leone published by the United Nations International Children's Emergency Fund (UNICEF) in 2017, specifically from the birth history (bh) and women (wm) datasets. My level of observation are individual children, whose mothers are between 15–49 years of age. To identify causal effects, I use an ordinary least squares linear regression with controls.

I find that the stress from Ebola causes birthweight to go down by 0.042 kilograms, with a standard error of 0.017. This result was found at 5% significance. The mean and standard deviation of the birthweight of the control group is 3.223 and 0.522, respectively. Therefore, the treatment causes a 1.300% change in birthweight at 5% significance. The results are suggestive of a causal link between the general effects of the Ebola crisis and the health of the fetus.

I run a separate equation with a differential effect. I test for an interaction between my treatment and a dummy variable denoting whether the mother lives in an urban area or not (urban is 1, rural is 0). I hypothesize that mothers in urban and rural areas may have different levels of exposure to the Ebola crisis due to different population densities.

With my new equation, I estimate that the Ebola crisis lowers birthweight by 0.034 (SE = 0.019) kilograms at 10% significance after adding controls. This is a 1.053% drop in from the control mean. The difference between the urban treatment and rural treatment effects is 0.029 (SE = 0.037) kilograms, found with no significance, a 0.898% difference from the control group. Adding the interaction term yields weaker results than before.

I have run a list of observational variables to see if my treatment and control are balanced. Even though I get multiple significant differences, they are of very small magnitude. Furthermore, adding controls to my main regression does not change the result or p-value, showing that any differences between treatment and control are within the margin of rounding.

I have listed similar work which measures the effects of maternal stress and maternal depression on children in utero. Others are literature reviews seek to measure the effects of Ebola on infected mothers and on infected babies.

II. Background and Literature Review

Background of the Crisis

An Ebola (*Zaire ebolavirus*) epidemic occurred in Sierra Leone during 2014 to 2015, alongside neighboring countries Guinea and Liberia. The epidemic started in Guinea and quickly spread around West Africa. The disease is thought to have originated when a child in a bat-hunting family contracted the disease. Transmission is believed to be by handling bushmeat such as bats and monkeys, which are important sources of protein in West Africa.

The disease is very severe and has a very high culling rate, at around 70% for those in West Africa during the crisis (57–59% if hospitalized). The disease kills within 6 to 16 days of contraction. The virus spreads through contact with bodily fluids from people or animals. Spread can also occur through items recently contaminated with bodily fluids.

One aspect of Sierra Leone that is alleged to have aided the disease are the very involved funeral processes. Funeral practices involve rubbing the body in oil, dressing them in fine clothes, then having those at the funeral hug and kiss the body. Even though the body is recently deceased, Ebola viruses remain active in high concentrations.

Sierra Leone's outbreak began in late May, when 14 people returned from a funeral of a traditional healer who had been trying to cure other Ebola victims in Guinea. The first person reported to be infected was a tribal healer. She had treated an infected person and died on May 26. She was buried traditionally, and contact with her body during the funeral process further spread the disease. The disease then overwhelmed the local hospital and killed 12 nurses who worked there.

On June 12, Sierra Leone declared a state of emergency in the Kailahun District, where it announced that schools, cinemas, and nightlife places would be closed. The district border Guinea and Liberia were also closed, but many locals were able to cross using unofficial routes. Summer rainfall and flooding interfered with the fight against Ebola.

By July 11, the first case reported in the capital of Sierra Leone, Freetown. On July 29, Sheik Umar Khan, Sierra Leone's only hemorrhagic fever expert, died after contracting the disease at his clinic. On July 30, Sierra Leone declared a state of emergency and deployed troops to quarantine hot spots. Awareness campaigns in Freetown were delivered through radio and loudspeakers. A law was passed which criminalized hiding someone with the disease.

During September, the disease spread exponentially. People were dumping their dead in the streets and body cleanup teams were having a hard time keeping up. As many as 20–30 bodies needed to be buried each day. WHO estimated on September 21 that Sierra Leone's capacity to treat Ebola cases fell short by 532 beds.

Sierra Leone imposed a three-day lockdown on its population on September 19–21. During this time 28,500 volunteers went door to door providing information about the disease and establishing Ebola surveillance teams. On September 24 three more districts were put under isolation. At this point 2 million people were in areas of restricted travel.

By October 2, 5 people per hour were contracting the disease. The estimated number of infected had been doubling every 20 days. Sierra Leone hospitals were running out of supplies. Grave diggers were making the graves too shallow, so as a result wildlife had dug up and ate the corpses. In addition, some bodies were left out in the street for days as no one was coming to collect them. It was reported that piles of corpses were building up south of Freetown. Other diseases such as malaria, pneumonia, and diarrhea were not being treated properly because the health system was under too much pressure.

In October and November, the United Kingdom had sent a hospital ship and planned to build three laboratories in Sierra Leone. By November 4, it was reported that thousands were violating quarantine in search of food. The Disaster Emergency Committee found that the aggressive quarantines and food shortages were making the situation worse.

In late November and December, the disease began to slow down. In December 5, it was reported that 100 people were getting infected daily. Traditional burials were criminalized.

On January 9, the Ebola caseload was reduced to 10,000. On January 10, Sierra Leone had declared its first Ebola free district. Throughout 2015 and January 2016 Ebola cases continued to dwindle until the country was declared Ebola free.

Throughout February and March, the number of cases once again started to rise. In March Sierra Leone once again went under lockdown for three days.

For the rest of the year, smaller cases of Ebola continued to pop up, and small-scale panics ensued. Only by March of 2016 was Sierra Leone finally declared Ebola free. Ultimately the disease had 14,061 confirmed cases in Sierra Leone and took 3,955 lives.

Literature Review

Almond & Currie (2011) reviews the details and developments on David J. Barker's fetal origin hypothesis. Barker hypothesized that slight changes to the fetal environment can cause major changes in health for adults down the line. The health effects can remain latent for many years, resulting in diseases

such as cardiovascular disease or type II diabetes appearing mid-life. They also provide details on how economists got involved in providing evidence for the hypothesis.

A related paper by Rodney & Mulligan (2014) measures the stress levels of pregnant women in the Democratic Republic of Congo, then measures various indicators of health for their babies. The study took place during a civil war in the country. Rape was being used as a tool of war, and much stress results in women in the area from actual rape, the knowledge that they could be raped at any time, and the social ostracism that follows. They find that the effects of both stress "due to war" and "due to mundane events" has very large reductions on birthweight at 1% significance. They also report that stress "due to material deprivation" causes large reductions at 5% significance.

Bebell, Oduyebo, & Riley (2017) have reviewed the existing literature on the effects of Ebola on pregnant women. They report that various studies find that pregnancy is associated with severe illness and death among Ebola patients, with numbers ranging from 74–100% mortality. However, some case studies report that these results are overstated, and that the true mortality may be around 5 of 13 women (39%).

Suri, et al. (2014) reviewed the prospective literature on the long-term effects on neurobehavioral effects on children if they were exposed in utero to untreated maternal depression. They find that the exposed face short term effects of increased distress, less-than-optimal orientation and motor activity, and disrupted sleep. Longer-term effects include disruptive social behavior, depression, and changes in the period of sensitivity for language discrimination.

Another literature review by Nelson, et al. (2016) find that all reported babies that have contracted Ebola have died. If contracted in utero, then the babies die within 19 days of birth. Not much else is known on the effects of the disease on babies; the only known symptom that the babies face is fever.

III. Data

To examine the effect of Ebola exposure on children in utero, I use the MICS of Sierra Leone. This was collected in year 2017 by Statistics Sierra Leone with technical support by UNICEF. The data is cross sectional, taken in a randomized fashion. The sample includes urban and rural areas in all four regions of the country (Eastern, Northern, Southern Provinces and the Western Area) and all 14 districts of the country. The sample was stratified by urban and rural areas. Within each stratum, a specified number of census enumeration areas were selected with probability proportional to size. After a household listing was carried out within the selected enumeration areas, a sample of 26 households was drawn in each sample enumeration area. All enumeration areas were visited during the fieldwork period (April–May 2017). Response rates are 99.9% for both children's questionnaires and 99.3% for the women questionnaire.

The main sample are the birth history and women (age 15–49) datasets merged. The birth history includes information from two questionnaires, one for children between ages under 5, and the other for children of ages between 5–17. The total sample size is 47,082. These datasets collect information about the health of individual children and women. Any observations which reported missing values for the treatment, control, or the outcome have been excluded, totaling 42,897 exclusions. My main unit of observation is the individual child. After all changes, I have 4,185 observations in the data.

Table 1, Column 1 presents baseline summary statistics. There I test for a significant difference between the treatment and control. The variables I test are: ethnicity, age of mother, highest grade achieved, an urban/rural indicator, if the woman has ever been married or lived with a man, her age when she first had sex, and the gender of the baby. To test for differences among ethnicity, I created two dummy variables, one for the Mende ethnicity and the other for the Temne. These are the most common ethnicities in the region, with Mendes representing 45.69% of the sample and Temnes representing 23.18%. The "highest grade" variable records the last grade the woman attended, from grades 1 to 13. The few women who went to higher education or vocational school (90/1,130) were marked as going to

grade 14. This variable has 5 missing observations for my sample, for which I have imputed the mean. The urban/rural variable is a dummy which records 1 if she lives in an urban area and 0 if not. 27.98% of the sample lives in an urban region. “Ever married or lived with a man” is also a dummy with 1 for yes and 0 for no. 85.97% of women have been married or lived with a man. The baby’s sex is a dummy which is recorded as 1 if he is a boy and 0 if she is a girl. 50.68% of the sample is male. The average mother’s age of the sample is 29.358. The average age of first sex is 15.983. There were 511 missing observations for “age of first sex” so I have imputed the mean onto those observations.

It is well known that the birthweight of a child can be used as a basic signal of their health. The birthweight of the fetus can be affected by a variety of shocks during the fetal period. It is not a particularly comprehensive or sensitive measure: some studies have found latent effects in children with normal birthweight, while others found no latent effects in children born with low birthweight. However, there has been no convergence on an alternative, superior metric to birthweight. A good measure of latent effects should be sensitive to all periods of pregnancy, be easy to measure, and be widely available. To meet these ends, no better metric has been suggested to date (Almond & Currie 2011). Therefore, I will use birthweight as my outcome variable.

IV. Empirical Approach

Identification Strategy

I will use the Ebola outbreak of 2014–15 as a source of exogenous variation. The outbreak was unexpected and sudden. I am relying on this fact for my identification assumption: that women didn’t time their pregnancies to avoid the outbreak. I am identifying the effects on birthweight by separating the treatment cohorts by year. The children born in 2011–2012 form the control group and the ones born in June 2014–2015 form the treatment. My treatment variable begins at June because the Ebola outbreak starts in May 2014. I will not use children born after 2015 because there were still traces of the disease around the country until 2016.

The evidence from Table 1 shows that there are significant differences among my treatment and control. The mother's age, her highest grade achieved, her urban/rural status, and if she has ever been married or lived with a man all have differences of 1% significance. The treatment group is 1.601 years younger, achieved 0.862 levels more of education, is 0.047 percentage points more likely to live in an urban area, and is 0.058 percentage points less likely to be married or live with a man. Both ethnicity variables contain differences at 10% significance. The treatment group is 0.025 percentage points more likely to be Mende and 0.023 percentage points less likely to be Temne. The higher education level and the higher urban status for the treatment group may artificially increase the results, dampening my magnitude of the effect on outcome. As for the other variables, it is unclear which way they could sway my results.

To be a valid counterfactual, the treatment and control group need to be balanced across observables and unobservables, such that any difference between the two arise because of the treatment. I cannot do anything about the unobservables, but I can try to control some of the observables. My imbalances in my Table 1 put my identification assumption at risk, though the small magnitude of difference for all of my variables is an indicator that they may not be an issue.

Econometric Specification

To estimate the causal effect of Ebola on birthweight, I will use an ordinary least squares linear regression. My input variable is a dummy variable of the treatment as described. My output is the birthweight. My first equation measures the effect of Ebola on birthweight with no controls. The second equation adds in my Table 1 variables as controls.

Here is my first estimate:

(1) $Birthweight_i = \beta_0 + \beta_1 Ebola_i + \varepsilon_i$

where $Birthweight$ is the birthweight of baby i and $Ebola$ is my treatment variable. β_1 is the treatment effect, which is expected to be negative.

To counteract bias from my Table 1 variables, I will use this equation:

(2) $Birthweight_i = \beta_0 + \beta_1 Ebola_i + \beta_2 Mende_i + \beta_3 Temne_i + \beta_4 Age_i + \beta_5 HighestGrade_i + \beta_6 Urban_i + \beta_7 MarriedOrLivedWithMan_i + \beta_8 FirstSex_i + \beta_9 Male_i + \varepsilon_i$

where the variables are in order as described in Table 1, for baby i.

Lastly, to estimate the differential effect of the stress from the Ebola crisis to birthweight by urban/rural membership, I use:

(3) $Birthweight_i = \beta_0 + \beta_1 Ebola_i + \gamma(Ebola \times Urban)_i + \beta_6 Urban_i + \varepsilon_i$

To the extent that higher density of people in urban regions will cause an increase in the panic and exposure to the Ebola crisis. My differential term γ tests if my hypothesis is true, and like last time β_1 dictates my treatment effect. I add in my Table 1 variables as controls once again:

(4) $Birthweight_i = \beta_0 + \beta_1 Ebola_i + \gamma(Ebola \times Urban)_i + \beta_2 Mende_i + \beta_3 Temne_i + \beta_4 Age_i + \beta_5 HighestGrade_i + \beta_6 Urban_i + \beta_7 MarriedOrLivedWithMan_i + \beta_8 FirstSex_i + \beta_9 Male_i + \varepsilon_i$

V. Results

Table 2 presents the results of the estimates from Equation 1. I find that women who are pregnant during the Ebola crisis have babies that weight 0.042 (SE = 0.017) less than the control group at 5% significance. My Equation 2 reports the same result. The mean and standard deviation of the control is 3.223 (SE = 0.522) kilograms, therefore, the effect I estimate is 1.300%. The differences between my Equation 1 and Equation 2 are within the rounding margins, revealing that my confounding variables have very little influence on my final results.

$HighestGrade$, $MarriedOrLivedWithMan$, and $Male$ are all correlated with the outcome at 1% significance. $MarriedOrLivedWithMan$ and $Male$ add 0.095 and 0.056 to the outcome if true,

respectively. Age also has small correlation with the outcome at 10% significance. $Mende$ contributes 0.037 kilogram, $Temne$ contributes 0.042, and $Urban$ contributes 0.038, all at 10% significance.

Table 3 presents the results of the estimates of Equation 3. This equation also tests for a difference-in-difference estimate $Ebola \times Urban$. I find that women who are pregnant during the Ebola crisis have babies that weight 0.036 (SE = 0.019) less than the control group at 10% significance. The difference between the urban treatment effect and the rural treatment effect is 0.029 (SE = 0.037) at no significance. Babies in urban areas weight 0.065 kilograms more, found at 10% significance.

Equation 4 reports a that the treatment decreased birthweight by 0.034 (SE = 0.019) kilograms at 10%. This is a 1.053% drop from the control mean. The difference-in-difference reported a drop of 0.029 (SE = 0.037) kilograms, found with no significance. This is a 0.898% drop from the control. $MarriedOrLivedWithMan$ and $Male$ had the largest effects in Equation 4, with birthweight gains of 0.095 and 0.056 respectively. Both have p-values under the 1% level. $Urban$ also influenced Equation 4, reporting an increase of 0.053 at 10% significance. $Mende$ and $Temne$ raise birthweight by 0.037 and 0.042 respectively, at 10% significance. Age and $HighestGrade$ also had small effects in Equation 4.

VI. Validity and Potential Threats

The Ebola outbreak was more than just a pandemic, it caused worldwide political panic as to try and contain and fix the problem. The government and international organizations such as the World Health Organization became heavily involved in trying to contain the disease. Many districts were isolated, many people were put under quarantine, and the country went into a total lockdown for three days. The women exposed to the Ebola crisis most likely faced significant mental hardship. Their elevated stress and depression levels may have influenced the weight of their child. Rodney & Mulligan (2014) and Suri, et al. (2014) concur with this point, in that they show that maternal stress or depression do have significant effects on the child's birthweight.

Guinea and Liberia have had similar issues tackling the Ebola. The populations of all three countries had similar cultures which contributed to the spread of the disease. Attending a traditional burial and contact with bushmeat were among the top risk factors for contracting the disease. I would say that the results of this study can be considered as externally valid if applied to these areas.

Ebola, once contracted, has a very high mortality rate, at ≈70% in West Africa during the crisis, and 57–59% if hospitalized. As stated before, pregnant women have it worse, with an average of 86% mortality across studies (Bebell, et al. 2017). It takes only 6–16 days to kill the patient. This high culling rate means that infected children are never born and the effects on their health are never recorded. In addition, Nelson, et al. (2016) reports that Ebola has a 100% mortality rates on neonates. These facts are a glaring internal validity issue, as anyone in the treatment group who actually got infected has a very high chance of not appearing in the data. This will artificially decrease the magnitude of the results.

The index case for this particular epidemic was a one-year-old boy in Guinea, who died in December of 2013. Everyone in my control sample was born at least a year before, so it is unlikely that the group was contaminated.

GDP per capita is strictly higher in the treatment period than in the control, so with higher living standards average birthweight may have rose between the two periods. This will also artificially decrease the magnitude of the results.

Is there a chance that the government organization Sierra Leone Statistics tampering with the study? It is doubtful, given that there really isn't any incentive to do so, and the fact that the study had oversight by UNICEF most likely means that it was carried out responsibly.

The fact that UNICEF oversaw the study also means that there most likely wasn't any unstated measurement shortcomings. All questionnaires used have had a compliance rate of above 99%.

VII. Conclusion

I have found that the overall effects of Ebola on pregnant women cause a 1.300% drop in birthweight. These results are significant at the 5% level. Adding in an urban/rural interaction term generates a new treatment estimate of a 1.053% drop in birthweight. The difference between the urban and rural treatment effects was not significant. These results are suggestive of a causal link between the total effects of the Ebola epidemic and the birthweight of children. What remains to be seen is the mechanism which causes these effects. It the virus itself infecting the fetus? Is it the stress from the environment? Is it due to maternal depression?

These results are important as they highlight that Ebola may have intergenerational effects. As stated in the fetal origins hypotheses, issues during development of the fetus can cause latent effects in children, causing problems in adulthood. These results suggest that the Ebola crisis may have effects on children in the long run.

Table 1: Summary Statistics and Baseline Balance

	All	Treatment	Control	Difference
	(1)	(2)	(3)	(4)
Ethnicity: Mende	0.457	0.469	0.443	0.025*
	(0.498)	(0.499)	(0.497)	
Ethnicity: Temne	0.232	0.221	0.244	-0.023*
	(0.422)	(0.415)	(0.429)	
Age of Mother	29.358	28.605	30.206	-1.601***
	(6.671)	(6.914)	(6.280)	
Highest grade	2.913	3.318	2.457	0.861***
	(4.276)	(4.456)	(4.017)	
Lives in Urban Area	0.280	0.302	0.255	0.047***
	(0.449)	(0.459)	(0.436)	
Ever Married or Lived with a Man	0.860	0.833	0.890	-0.058***
	(0.347)	(0.373)	(0.313)	
Age at First Sex	15.983	15.995	15.970	0.025
	(2.080)	(2.079)	(2.081)	
Is the Newborn Male	0.507	0.509	0.505	0.004
	(0.500)	(0.500)	(0.500)	
N	4,185	2,216	1,969	

Notes: Columns 1–3 present the averages and standard deviations. Column 4 presents the difference between Column 2 and Column 3.

A dummy variable denoting if the woman had health insurance was omitted because almost no one has health insurance in Sierra Leone (2.05% of the sample). There are 14+ ethnicities in Sierra Leone, the ones presented are the largest ones (Mende at 45.69% of the sample, Temne at 23.18%). There are no 0 values for "Age at First Sex". "Highest Grade" denotes the last grade the woman had finished, within grades 1–13, and 14 being recorded for the few (2.15% of the sample) who went to higher education or vocational school.

* denotes significance at 10%, ** significance at 5%, and *** significance at 1%.

Table 2: Effect of Ebola Crisis on Birthweight

	Birthweight	
	(1)	(2)
Ebola	-0.042**	-0.042**
	[0.017]	[0.017]
Ethnicity: Mende		0.037*
		[0.019]
Ethnicity: Temne		0.042*
		[0.023]
Age of Mother		0.002*
		[0.001]
Highest Grade		0.009***
		[0.002]
Lives in Urban Area		0.038*
		[0.020]
Ever Married or Lived with a Man		0.095***
		[0.024]
Age of First Sex		-0.004
		[0.004]
Is the Newborn Male		0.056***
		[0.016]
Constant	3.223***	3.045***
	[0.012]	[0.078]
Observations	4,185	4,185
R-squared	0.002	0.014
Mean of Birthweight in the Control Group	3.223	3.223
Standard Deviation of Birthweight in the Control Group	0.522	0.522

Notes: Standard errors in brackets. This table presents ordinary least squares linear regressions for the treatment's effect on birthweight. The construction of the treatment is as described in earlier in this paper.

* denotes significance at 10%, ** significance at 5%, and *** significance at 1%

Table 3: Differential Effect of Urban/Rural Household

	Birthweight	
	(1)	(2)
Ebola	-0.036*	-0.034*
	[0.019]	[0.019]
Ebola × Urban	-0.029	-0.029
	[0.037]	[0.037]
Ethnicity: Mende		0.037*
		[0.019]
Ethnicity: Temne		0.042*
		[0.023]
Age of Mother		0.002*
		[0.001]
Highest Grade		0.009***
		[0.002]
Lives in Urban Area	0.065**	0.053*
	[0.028]	[0.028]
Ever Married or Lived with a Man		0.095***
		[0.024]
Age at First Sex		-0.004
		[0.004]
Is the Newborn Male		0.056***
		[0.016]
Constant	3.206***	3.041***
	[0.014]	[0.078]
Observations	4,185	4,185
R-squared	0.003	0.014
Mean of Birthweight in the Control Group	3.223	3.223
Standard Deviation of Birthweight in the Control Group	0.522	0.522

Notes: Standard errors in brackets. This table presents ordinary least squares linear regressions for the treatment's effect on birthweight. Ebola × Urban measures the difference-in-difference between the treatment effect and the urban/rural status. The construction of the treatment is as described in earlier in this paper.

* denotes significance at 10%, ** significance at 5%, and *** significance at 1%

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