Prenatal Exposure to a Public Health Crisis and Birth Weight: Exploiting a Natural Experiment in Hong Kong

Ming Zhao

ABSTRACT

This article examines the effect of prenatal maternal exposure to a public health crisis on birth weight. Exploiting severe acute respiratory syndrome (SARS) as an exogenous source of stress and using Hong Kong Panel Study of Social Dynamics (HKPSSD), I surprisingly find that maternal exposure to stress results in a significant increase in birth weight. When treating SARS as a randomized experiment, the results highlight the importance of exposure to the third trimester with other trimester(s); when regarding SARS as a quasi-experiment, based on the propensity score technique, the results underline the role of the third trimester itself. Although there is inconsistency, both experiments agree on the significance of the third trimester and the direction of the treatment. The mechanism driving this positive effect can be mothers' more attention on the third trimester of gestation in the first place and an increase in their health-enchancing behaviors as a result of exposure to the health crisis during the stage. Moreover, this effect serves as a population parameter derived from bootstraping.

INTRODUCTION

"The womb may be more important than the home," Barker conjectured in the beginning of the 1990s (p.1111). Recent research highlights the importance of the interaction between genes and environment before birth for birth outcomes as well as outcomes later in life. As a growing body of literature on fetal origins suggests, *in utero* conditions have started to pose effects to individuals, this study examines the first effect of prenatal maternal stress: birth weight, particularly, the extent to which exposure to stress during pregnancy impacts birth weight and how maternal stress affects the outcome. As a public health crisis, the epidemic of severe acute respiratory syndrome (SARS) that struck Hong Kong in 2003 is exploited as a natural experiment and regarded as a source of maternal stress. Earthquakes, terrorist attacks, wars, and ethnic discrimination, as stressors, have been used as natural experiments to explore the effects of maternal stress (e.g., Torche 2011; Eskenazi et al. 2015; Lauderdale 2006; Novak et al. 2017). However, as far as I know, an epidemic outbreak as an exogenous source of prenatal maternal stress has never been studied on birth outcomes.

To explore the relationship between maternal stress and birth weight in Hong Kong, I situate my empirical work in the periods around SARS drawing on a city-wide representative survey. I first present the overview of SARS and maternal stress, and then review previous studies on this topic with a focus on findings derived from the research design of a natural experiment. In the analysis part, I treat SARS as a randomized experiment and a quasi-experiment separately, where OLS, propensity score matching, and bootstrapping are employed in the process.

BACKGROUND

SARS as a Public Health Crisis

In March 2003, the World Health Organization (WHO) issued a global alert about a deadly new infectious disease and named it severe acute respiratory syndrome (SARS). By 7 August 2003, SARS had spread to 32 countries with a cumulative total of 8,422 cases and 919 deaths. Hong Kong was one of the most severely affected areas. SARS struck Hong Kong in March 2003. From 11 March to 11 July, a total of 1,755 cases were identified and 298 people died of the disease. The death rate was about 17%, which is much higher than the global, 11%.

SARS in Hong Kong experienced three phases. The first took place in March. This was an explosive outbreak in a hospital, affecting a large number of hospital staff and medical students. The second was an outbreak in a community as a result of the spread of infection from the hospital to the community, and it reached its peak in early April. The third began in early May with continuing occurrence of the disease in eight hospitals and more than 170 housing estates throughout the city (Lee 2003). SARS constituted a crisis for nearly every Hong Kong residents, not only because it is novel, unknown, but also uncontrollable (Gan, Liu, and Zhang 2004). As a public health crisis, SARS has three hallmarks: 1) “the situation is exigent,” 2) “the anticipated or potential harm would be calamitous,” and 3) “the harm cannot be avoided through ordinary procedures,” noted by Haffajee et al. (2014).

Maternal Stress as Mechanisms

Stress has various definitions in terms of types and causes. Following Kugelmass and Lynch (2014), the stress is usually a process that begins with exposure to a stressor and ends with distress. The stressor can be any conditions or experiences that potentially challenge individuals’ adaptive capacities, such as divorce, unemployment, and war. Maternal stress has connected to the release of stress hormones in the mother, including corticotrophin-releasing hormone,

adrenocorticotrophic hormone, and cortisol. These hormones help prepare the preganant women to cope with the stressor. However, they are able to pass from the mother to fetus via the placenta and produce a long-term reprogramming and dysregulation of the hypothalamic-pituitary-adrenal axis, the central stress response system. As a result, exposure to high levels of stress hormones can pose negative consequences to birth weight directly. Additionally, a normal full-term pregnancy is divided into three trimesters; it has found that birth weight can be determined even in the first stage (Smith et al. 1998).

SARS as Exogenous Sources of Maternal Stress

SARS as a public health crisis is corelated to stress. Given SARS-associated coronavirus transmission requires close contact with an infected person, including coughing, sneezing, and chatting, the commonsense health measures seem unnecessary, because people display predicted health anxiety when noticing illness related information and such response is distributed in the general population (Hadjistavropoulos, Craig, and Hadjistavropoulos 1998; Thompson et al. 2017). Moreover, like other health crises such as H1N1 flu in 2009 and Ebola in 2014 (McDonnell, Nelson, and Schunk 2012; Thompson et al. 2017), SARS-related media exposure is highly likely to be associated with psychological distress. That is, threats to public health often exceed the impact on physical health and reach to cognitive responses.

LITERATURE REVIEW

Among studies on relationships between stress during pregnancy and birth weight (Buffa et al. 2018; Torche 2011; Eskenazi et al. 2007; Lauderdale 2006; Novak et al. 2017; Davis and Sandman 2011; Baibazarova et al. 2012; Mclean et al. 2018), measures of maternal stress vary widely. They are generally categorized into perceived stress, mental disorders, life events,

cortisol assessments, and natural disasters or terrorist attacks. It has been hypothesized that prenatal maternal stress predicts low birth weight. However, the results are not consistent across studies. The reasons for the inconsistency may attribute to the variations in stress measures and societal settings. Furthermore, most of the studies on prenatal maternal stress present threats to internal validity because stress due to different stressors may not have been independent of women's own characteristics. In other words, stress is not randomly assigned and characteristics of a woman that increase her risk of experiencing stress are likely to be heritable and genetically influence her child, so it is difficult to disentangle genetic effects from effects of prenatal conditions and postnatal environments (Cao-Lei et al. 2016; Torche 2011). Consequently, the approach to use exposure to an independent random stressor, such as natural or human-made disasters, can reduce such threats to validity.

Disasters used as natural experiments in previous studies on the relationships between prenatal maternal stress and birth outcomes include the 1994 Northridge Earthquake in California (Glynn et al. 2001), the September 11 attacks in 2001 (Eskenazi et al. 2007), the 2004 Madrid train bombings (Marco, Moris, and Fabrizio 2020), the 2005 Chile earthquake (Torche 2011), the Mexican Drug War since 2006 (Torche and Villarreal 2014; Brown 2018), the 2008 immigration raid in Iowa (Novak et al. 2017) as well as the ethnic discrimination after September 11th (Lauderdale 2006).

Specifically, the study by Glynn et al. (2001) using the California earthquake suggested that stress experienced in early pregnancy is associated with reduced gestational length. Later, Eskenazi et al. (2007) reported an increase in newborns weighing 2kg though a decrease in 2-2.5kg in the week of September 11th in NYC; they also reported infants who had experienced the disaster in the first or second trimester of gestation have increased odds of 1.5kg. In a recent

study by Marco, Moris, and Fabrizio (2020), they found prenatal maternal stress has an effect on low birth weight for children exposed to the bombing during the second trimester. Exploiting the Chile earthquake, Torche (2011) found that maternal stress measured by a major earthquake in early pregnancy produces significant decline in birth weight. Using the Drug War in Mexico, Torche and Villarreal (2014) observed a positive effect of exposure to homicides in the first trimester; however, the effect observed by Brown (2018) is negative. Moreover, Lauderdale (2006) and Novak et al. (2017) showed that infants of Arabic-named and Latina women who experienced the unexpected immigration raid and ethnic discrimination, respectively, in the first trimester of gestation have higher risks of low birth weight than other groups.

Among these studies, almost all show that maternal stress during pregnancy has a negative effect on birth weight although they fail to agree on the significance of different trimesters. Nevertheless, Torche and Villarreal surprisingly found the effect positive. According to the researchers, the reason for the unexpected relationship is an increase in mothers' health-enhancing behaviors as a result of exposure to violence in the first trimester of gestation.

METHODS

Data

The data this study relies on are the Hong Kong Panel Study of Social Dynamics (HKPSSD). HKPSSD is a city-wide representative household panel survey launched in 2011 with following waves in 2013, 2015, and 2017. The baseline survey interviewed 3,214 households including 7,218 adults aged above 15 and 958 children. The three waves of follow-up surveys covered 2,165 households (with 4,270 adults and 623 children), 2,404 households (with 5160 adults and 477 children), and 2,000 households (with 3,407 adults and 404 children).

Additional refreshment samples of 1,007 households (including 1,960 adults and 145 children), 505 households (including 892 adults and 90 children), and 840 households (including 1,102 adults and 119 children) were added in 2014, 2015, and 2020, respectively. Taken together, the surveys interviewed 16,486 adults and 1, 889 children in total.

Variables

The dependent variable in this study is birth weight measure in kilogram, focusing on children who were born and conceived in Hong Kong during SARS from March to July 2003, specifically newborns in March 2003 (conceived in July 2002) and to March 2004 (conceived in July 2003). Birth weight in this period is compared with that of infants born one year earlier from February 2002 throughout February 2003. Four time points are distinguished to examine the effect of SARS across gestational ages, including born before SARS (T0=1) and experiencing SARS during the first (T1=1), second (T2=1), and third (T3=1) trimester. Because the epidemic took five-month long, many of births were impacted more than one trimester and some (infants born in September 2003) even carried all the three. Covariates include children's gender, mothers' years of schooling and age at delivery as well as monthly household income per capita.

Statistical Models

SARS is exploited as a natural experiment because treatment and control groups are determined by factors out of researchers' control. Firstly, I treat SARS as a randomized experiment because each subject has an equal chance of experiencing the epidemic outbreak. However, randomization does not guarantee comparison among groups, according to Sekhon and Titunik (2012), so I then regard SARS as a quasi-experiment. In the randomized experiment, the outcome Y_i is modeled by:

$$Y_i = \alpha + \beta D_i + \theta X_i + \varepsilon_i \quad (1)$$

where D_i is the treatment refering to the three trimesters and X_i is a vector of covariates. The coefficient β for D_i measures the average treatment effect of the time points. The quasi-experiment assumes that the treatment and control groups are uncomparable, so propensity score matching is adopted. The matching process is defined as:

$$(Y_{i1}, Y_{i0}) \perp D_i \mid X_i$$

$$P_i = \Pr(D_i = 1 \mid X_i)$$

$$(Y_{i1}, Y_{i0}) \perp (D_i \mid P_i)$$

where P_i is the conditional probability of the treatment estimated by logistic regression given covariates. Using equation (1) again in this experiment, β captures the average treatment effect after matching. Finally, I exploit the bootstrap sampling method for both randomized and quasi-experiments to make inference about the population parameter.

RESULTS

Table 1 presents descriptive statistics across treatment and control groups. Panel A is divided into who were born before SARS and who were affected by SARS during gestation. That is, in the randomized experiment, the sample size of the treatment and control is 74 and 72, respectively. In the treatment group, three groups are identified by trimester. As Panel B shows, the sample size is 42 in the first, 43 in the second, and 39 in the third. The total number of the subjects in the subgroups seems greater than that of the whole treatment group because most children experienced more than one trimester. Panel C displays control groups in the quasi-

experiment. They are comparable to the treatment groups of the three trimesters using propensity score matching on covariates, in which the statistics of male, mother's schooling, mother's age at birth, and monthly household income per capita are balanced between Panel B and Panel C.

[Table 1 about here]

I first consider SARS as a randomized experiment. Table 2 displays the estimated treatment effects on birth weight relying on the randomized experiment. For Model 1 and Model 2 in Table 2, the coefficients of the first and third trimesters are significantly positive regardless of including covariates or not. The Model 3 and Model 4 have additional two variables: whether experiencing both first and second trimesters and whether experiencing both second and third trimesters during SARS. The results suggest that the effects of the added variables are significantly positive, while the net effect of the second trimester becomes negative. Furthermore, I add whether experiencing all three trimesters in Model 5 and Model 6. It is the only significant variable aside from mother's age at birth and it is positive as well.

Table 3 also displays the treatment effects for randomized experiment, but it divides the analytical samples into three groups by trimester and each of them with the control group as the subsamples. For the models in the first two columns, the first trimester is the treatment instead of all three. Holding covariates constant, among those who experienced the first trimester during SARS, the third trimester is significantly positive, compared to children born before SARS. The models in the second columns use the second trimester as the treatment. Specifically, among those who experienced the second trimester, all three trimesters are significant, but the effect of the second is negative in contrast to the positive effect of the first and third. Compared the third trimester with the control, the first trimester has a significant positive effect. Table 2 and Table 3

present consistent results that experiencing more than one trimester yields positive effects on birth weight.

[Table 2 about here]

[Table 3 about here]

In contrast to Table 3, Table 4 shows the estimated treatment effects in the quasi-experiment. However, after using balanced samples for each treatment, the results in the two tables are very similar. The first and third trimesters have positive effects separately and collectively. The second trimester seems to pose negative impact on birth weight but the first and third trimesters weigh against the importance of the second trimester.

[Table 4 about here]

Adopting bootstraps with 100 replications, standard errors that represent the population are obtained in the models for both ramdonized and quasi- experiments, as shown in Table 5. The results of the randomized experiment estimated by bootstraping display that the first and second trimesters become insignificant, in contrast to Table 3, when comparing the second trimester to the control. Thus, the results indicate that experiencing the third with another trimester or trimesters has positive effects on birth weight. Looking at the quasi-experiment, the results are different. Compared Model 6 with Model 3, the first trimester changes to insignificant while the third becomes significant when the third trimester serves as the treatment. In this way, as long as exposure to SARS during the third trimester, birth weight is increased.

[Table 5 about here]

In summary, exploiting SARS as a randomized experiment and as a quasi-experiment separately, combined with the bootstrap method, yields slightly different results. The former finds the importance of exposure to the third trimester with other trimester(s) while the latter emphasizes the role of the third trrimester itself. However, both experiments agree on the significance of the third trimester and the direction of the treatment effect on birth weight.

DISCUSSION AND CONCLUSION

By using SARS as an exogenous source of stress, the influence of maternal stress on birth weight is examined. Maternal stress as the treatment is measured with three stages during the pregnancy. Because SARS continued for five months, multiple trimesters can be experienced during the crisis. When considering SARS as a randomized experiment, the treatment and control groups are assumed to be comparable. In this condition, by applying boostrapts, I find that experiencing the third and another trimester(s) has a positive effect on birth weight. When regarding SARS as a quasi-experiment, propensity score matching on covariates is used to balance between the treated and the untreated. By applying bootstraps again, I find that provided exposure to the third trimester during SARS, there is an increase in birth weight. The findings by the two experiments are not completely consistent, but both two highlight the positive effect of the third trimester.

In contrast to previous studies that exploit disasters as natural experiments, the positive effect of stress on birth weight is unexpected. Among all the studies I review, most of them found that exposure to prenatal maternal stress negatively impacts birth weight, but only one found it positive, which is by Torche and Villarreal (2014) with a focus on exposure to violence during the Mexican Drug War. In terms of the timing of the stress effect in the studies, they all

emphasized the significance of early and middle stages of gestation and only one reported that the third trimester is negatively associated with birth weight. Therefore, the findings in this study provide an altrenative perspective to explain the relationship between matenal stress and birth weight.

Torche and Villarreal (2014) suggested, the mechanism driving the positive effect of the first trimester on birth weight is mothers' growing health-enhancing behaviors, particulary the use of prenatal care, as a result of exposure to homicides. Similarry to violence could further health-enhancing behaviors, health anxiety has the function to change behavioral responses; when illness related information noticed, individuals tend to display not only cogitive negativity but also behavioral avoidance (Hadjistavropoulos, Craig, and Hadjistavropoulos 1998). This process involves coping, which is made up coginitive appraisal and coping behavior (Folkman and Lazarus 1985; Folkmanet al.1986). Coping can be further divided into problem-focused coping and emotion-focused coping. During the SARS, pregnant women are very likely to use both coping methods to make adjustments to avoid or reduce harm for the unborn child. As a result, health-enhancing behaviors to prevent the potential risk is also applicable for pregnant women in Hong Kong during the public health crisis. The behaviors may include but not limited to appropriate weight gain and increasing nutrient intakes.

One of the potential limitations of the study may be the uncertainty of measure for maternal stress because there is no population reports of distress and anxiety during or following SARS. Another foreseeable limitation is generalization. It will be difficult to apply the findings outside the context of Hong Kong because variation in settings does exist, for example, pregnancy culture and health care systems. In spite of these limitations, the findings contribute to an emerging body of evidence showing that maternal stress in the late pregnancy affects birth

weight, and it is the first time to use an epidemic outbreak as a source of stress to study birth outcomes.

APPENDIX

Table 1. Descriptive Statistics across Groups by Treatment

	Panel A		Panel B			Panel C		
	Control group	Treatment group	Treatment Groups of			Comparable Control Groups for		
			1st	2nd	3rd	1st	2nd	3rd
1st trimester (%)	-	56.76	100.00	30.23	8.97	-	-	-
2nd trimester (%)	-	58.11	30.95	100.00	30.77	-	-	-
3rd trimester (%)	-	52.7	8.33	27.91	100.00	-	-	-
Male (%)	54.17	60.81	71.43	62.79	51.28	69.05	65.12	58.97
Mother's schooling	11.528	10.581	10.429	10.535	10.692	10.190	10.163	10.103
	(3.113)	(2.862)	(2.724)	(3.119)	(2.930)	(2.578)	(2.554)	(2.817)
Mother's age at birth	30.694	30.905	30.190	31.512	31.692	30.310	29.953	30.974
	(6.058)	(6.284)	(6.500)	(7.042)	(6.424)	(6.326)	(6.550)	(6.667)
Monthly household income /capita	9895.652	6947.109	6469.312	6191.924	7367.308	7577.863	6226.149	7359.921
	(8825.696)	(5681.063)	(7577.863)	(4716.716)	(6156.705)	(7294.811)	(4020.356)	(6534.696)
Birth weight	3.008	3.132	3.179	3.114	3.244	3.038	2.993	2.903
	(0.620)	(0.720)	(0.871)	(0.878)	(0.857)	(0.609)	(0.591)	(0.623)
N	72	74	42	43	39	42	43	39

Table 2. Randomized Experiment: OLS Estimates of the Effect of Materenal Stress on Birth Weight

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Born before SARS (T0=1)						
First trimester (T1=1)	0.315**	0.315**	0.110	0.103	0.120	0.129
	(0.149)	(0.149)	(0.178)	(0.179)	(0.157)	(0.158)
Second trimester (T2=1)	-0.241	-0.260	-1.162***	-1.113***	-0.221	-0.238
	(0.162)	(0.162)	(0.334)	(0.329)	-0.157	(0.157)
Third trimester (T3=1)	0.387**	0.410***	0.192	0.242	0.181	0.215
	(0.150)	(0.148)	(0.183)	(0.180)	(0.160)	(0.158)
T1 + T2			0.959***	0.923***		
			(0.340)	(0.335)		
T2 + T3			0.921***	0.824**		
			(0.338)	(0.334)		
T1 + T2 + T3					0.940***	0.873***
					(0.299)	(0.295)
Child is male		-0.092		-0.092		-0.091
		(0.113)		(0.111)		(0.110)
Mother's schooling		0.018		0.014		0.014
		(0.020)		(0.019)		(0.019)
ln (Household income /capita)		-0.019		-0.025		-0.024
		(0.047)		(0.046)		(0.046)
Mother's age at birth		-0.181**		-0.153**		-0.152**
		(0.075)		(0.074)		(0.074)
(Mother's age at birth)$_2$		0.003**		0.002+		0.002+
		(0.001)		(0.001)		(0.001)
Constant	2.948***	6.052***	3.008***	5.767***	3.008***	5.758***
	(0.075)	(1.085)	(0.076)	(1.064)	(0.076)	(1.060)
N	146	146	146	146	146	146
R-squared	0.054	0.132	0.117	0.185	0.116	0.184

Standard errors in parentheses

*** p<0.01, ** p<0.05, + p<0.10

Table 3. Randomized Experiment: OLS Estimates of the Effect of Materenal Stress on Birth Weight by Trimester

	T1 vs. Control		T2 vs. Control		T3 vs. Control	
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Born before SARS (T0=1)						
First trimester (T1=1)	0.170	0.111		1.019***		1.012***
	(0.140)	(0.191)		(0.307)		(0.303)
Second trimester (T2=1)		-0.170	0.106	-1.100***		-0.248
		(0.230)	(0.140)	(0.354)		(0.241)
Third trimester (T3=1)		1.063***		1.064***	0.235+	0.232
		(0.296)		(0.301)	(0.142)	(0.191)
Child is male		-0.119		-0.071		-0.087
		(0.137)		(0.136)		(0.131)
Mother's schooling		0.024		0.015		-0.010
		(0.023)		(0.023)		(0.025)
ln (Household income/capita)		-0.010		-0.012		0.057
		(0.054)		(0.051)		(0.069)
Mother's age at birth		-0.169**		-0.162+		-0.187**
		(0.083)		(0.082)		(0.090)
(Mother's age at birth)$_2$		0.002+		0.002+		0.003+
		(0.001)		(0.001)		(0.001)
Constant	3.008***	5.894***	3.008***	5.773***	3.008***	5.814***
	(0.085)	(1.231)	(0.086)	(1.180)	(0.084)	(1.257)
N	114	114	115	115	111	111
R-squared	0.013	0.210	0.005	0.188	0.025	0.196

Standard errors in parentheses

*** p<0.01, ** p<0.05, + p<0.10

Table 4. Quasi-Experiment: OLS Estimates of the Effect of Materenal Stress on Birth Weight by Trimester

	T1 vs. Control		T2 vs. Control		T3 vs. Control	
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Born before SARS (T0=1)						
First trimester (T1=1)	0.140	0.053		0.983***		0.983***
	(0.164)	(0.191)		(0.311)		(0.312)
Second trimester (T2=1)		-0.197	0.121	-1.054***		-0.276
		(0.224)	(0.161)	(0.364)		(0.248)
Third trimester (T3=1)		1.032***		1.059***	0.341**	0.382+
		(0.287)		(0.304)	(0.170)	(0.212)
Child is male		-0.125		0.032		-0.058
		(0.167)		(0.166)		(0.165)
Mother's schooling		0.046		0.007		-0.034
		(0.029)		(0.029)		(0.033)
ln (household income/capita)		-0.048		-0.037		0.047
		(0.055)		(0.054)		(0.077)
Mother's age at birth		-0.288***		-0.178+		-0.222**
		(0.093)		(0.092)		(0.105)
(Mother's age at birth)^2		0.004***		0.002+		0.003+
		(0.001)		(0.001)		(0.002)
Constant	3.038***	7.996***	2.993***	6.249***	2.903***	6.580***
	(0.116)	(1.398)	(0.114)	(1.322)	(0.120)	(1.472)
N	84	84	86	86	78	78
R-squared	0.009	0.330	0.007	0.241	0.050	0.279

Standard errors in parentheses

*** p<0.01, ** p<0.05, + p<0.10

Table 5. Bootstrap Sampling (100 replicates): OLS Estimates of the Effect of Materenal Stress on Birth Weight by Trimester

	Randomized Experiment			Quasi-Experiment		
	T1 vs. Control	T2 vs. Control	T3 vs. Control	T1 vs. Control	T2 vs. Control	T3 vs. Control
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Born before SARS (T0=1)						
First trimester (T1=1)	0.111	1.019+	1.012**	0.053	0.983+	0.983+
	(0.140)	(0.530)	(0.470)	(0.159)	(0.561)	(0.530)
Second trimester (T2=1)	-0.170	-1.100+	-0.248	-0.197	-1.054+	-0.276
	(0.182)	(0.571)	(0.173)	(0.191)	(0.596)	(0.173)
Third trimester (T3=1)	1.063**	1.064**	0.232+	1.032**	1.059+	0.382**
	(0.505)	(0.533)	(0.119)	(0.447)	(0.579)	(0.168)
Covariates controlled						
Constant	5.894**	5.773***	5.814**	7.996***	6.249**	6.580+
	(2.378)	(2.222)	(2.608)	(2.576)	(2.828)	(3.510)
N	114	115	111	84	86	78
R-squared	0.210	0.188	0.196	0.330	0.241	0.279

Standard errors in parentheses

*** p<0.01, ** p<0.05, + p<0.10

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