

APPENDIX
Intergenerational Effects of Early-Life Advantage:
Lessons from a Primate Study

Amanda M. Dettmer *
Yale Child Study Center

James J. Heckman †
Center for the Economics of Human Development, University of Chicago
and American Bar Foundation

Juan Pantano ‡
Department of Economics, University of Arizona

Victor Ronda §
Department of Economics and Business Economics
and TrygFonden's Centre for Child Research, Aarhus University

Stephen J. Suomi ¶
National Institute of Child Health and Human Development

* amanda.dettmer@yale.edu.

† jjh@uchicago.edu.

‡ jpanta@email.arizona.edu.

§ ronda@econ.au.dk.

¶ stephen.suomi@nih.gov.

1 Additional Specifications

In the main text, we report intergenerational treatment effects estimated by augmented inverse probability weighting (AIPW) estimates. In this section, we test for the robustness of our results under different assumptions. We focus on two main issues. One concern is selection into treatment. This shouldn't be a significant issue in our study since the researchers at the lab had complete control over the treatment assignment. Differently than human studies, the monkeys could not select out of the assignment treatment. However, we know that researchers were more likely to assign first-born monkeys to the maternal-rearing condition. It is also possible for the treatment assignment to change over the years. A second concern is that of sample selection. We do not observe the outcomes of every monkey in the study. For example, we only observe the health outcomes for 109 out of the 656 offspring in our sample. Often, the reason for the missing outcomes is that the outcome was only collected for a subset of the cohorts or during a restricted period. For example, we only have access to veterinarian health records from 2002 to 2009, limiting the sample with observable health outcomes. While this is not a cause for concern, it is also possible for the treatment assignment to influence the sample selection. For example, we find that the offspring of maternal-reared females were more likely to survive the first month of life than offspring of nursery-reared females.

For these reasons, we check for the validity of the results presented in Section 4. We re-estimate the parameters in Table 6 under four alternative specifications. We present these in Tables 1-2. In column (1), we present unconditional mean estimates by comparing the outcomes between monkeys assigned to two different rearing sequences. This approach exploits the random assignment to the rearing sequences to compute the treatment effects of interest but does not account for selection into treatment or sample selection. In column (2), we present conditional mean estimates, where we control for sex, birth order, and cohort effects in the outcome regression. In column (3), we present inverse probability weighting

(IPW) estimates, where we control for sex, birth order, and cohort effects in the propensity score model. In column (4), we present augmented inverse probability weighting (AIPW) estimates, where we control for sex, birth order, and cohort effects in the outcome regression and the propensity score model. The AIPW combines the advantages of the IPW and conditional mean estimates. The AIPW estimator had the advantage of being ‘doubly-robust,’ and it is consistent for the ATE if either the propensity score model or the outcome regression is properly specified. We report the AIPW estimates in the main paper. These latter three methods account for the presence of selection into treatment. To account for the possibility of sample selection, in column (5), we estimate a modified version of the IPW estimator proposed by Huber (2014). The approach weights the observations by the inverse of a nested propensity score that characterizes both the selection probability into the treatment and the observable sample. This approach accounts for both the fertility selection and mortality selection discussed in Sections 4.1 and 4.2.

We do not find any systematic differences in the estimates across the five models. Parameter estimates across the five models are not statistically different from each other. These results are reassuring. The results provide evidence that the estimates presented in Section 4 are not driven by either selection into treatment or sample selection.

Table 1: ALTERNATIVE SPECIFICATIONS: % IN GOOD HEALTH

Generation 2 Outcome: Specification	% in Good Health				
	(1)	(2)	(3)	(4)	(5)
$\Psi(s_{0,1}) - \Psi(s_{0,0})$	-0.019 (0.033)	0.007 (0.034)	-0.009 (0.031)	-0.019 (0.034)	-0.009 (0.029)
$\Psi(s_{1,1}) - \Psi(s_{1,0})$	0.122*** (0.039)	0.102** (0.040)	0.100*** (0.037)	0.078** (0.033)	0.099** (0.039)
$\Psi(s_{1,0}) - \Psi(s_{0,0})$	-0.088** (0.041)	-0.034 (0.037)	-0.054 (0.040)	-0.030 (0.031)	-0.053 (0.042)
$\Psi(s_{1,1}) - \Psi(s_{0,1})$	0.053* (0.029)	0.061* (0.034)	0.055* (0.029)	0.067** (0.034)	0.054* (0.028)
$\Psi(s_{1,0}) - \Psi(s_{0,1})$	-0.068 (0.044)	-0.041 (0.043)	-0.045 (0.042)	-0.011 (0.040)	-0.044 (0.043)
Outcome mean:	0.904	0.904	0.904	0.904	0.904
Generation 2 obs.	109	109	109	109	109
Generation 1 obs.	59	59	59	59	59

Notes: This table compares the treatment effects estimates for the probability of being in good health under different specifications. Column (1) presents unconditional mean estimates. Column (2) presents conditional mean estimates, where we control for the offspring's sex, primiparous status and birth cohort, and maternal primiparous status and birth cohort. Column (3) presents IPW estimates, where we allow the offspring sex and maternal or offspring year of birth trends and primiparous status to influence the propensity score for intergenerational and intragenerational models, respectively. Column (4) AIPW estimates, where we control for the offspring's sex, primiparous status, and birth cohort in the regression model in addition to the propensity score controls. Model (5) presents IPW estimates that control for sample selection. We report the AIPW estimates in the main paper. In model (5), we allow for the probability of having the outcome observed to influence the propensity score, in addition to the other controls. The inference is under the null that the parameter of interest is zero. Standard errors clustered at the mother level are reported in parenthesis. The stars correspond to the following p-value levels: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 2: ALTERNATIVE SPECIFICATIONS: ELO PERCENTILE RANK

Generation 2 Outcome: Specification	Elo Percentile Rank				
	(1)	(2)	(3)	(4)	(5)
$\Psi(s_{0,1}) - \Psi(s_{0,0})$	0.008 (0.093)	-0.101 (0.074)	-0.127* (0.070)	-0.109 (0.071)	-0.155** (0.066)
$\Psi(s_{1,1}) - \Psi(s_{1,0})$	0.216*** (0.076)	0.142* (0.077)	0.135* (0.074)	0.134** (0.066)	0.142* (0.079)
$\Psi(s_{1,0}) - \Psi(s_{0,0})$	-0.062 (0.074)	-0.077 (0.071)	-0.067 (0.069)	-0.075 (0.068)	-0.075 (0.066)
$\Psi(s_{1,1}) - \Psi(s_{0,1})$	0.147 (0.090)	0.167* (0.087)	0.196** (0.078)	0.168** (0.079)	0.222*** (0.077)
$\Psi(s_{1,0}) - \Psi(s_{0,1})$	-0.069 (0.083)	0.025 (0.080)	0.060 (0.079)	0.034 (0.076)	0.080 (0.075)
Outcome mean:	0.540	0.540	0.540	0.540	0.540
Generation 2 obs.	106	106	106	106	106
Generation 1 obs.	68	68	68	68	68

Notes: This table compares the treatment effects estimates for the within-cohort percentile social-rank under different specifications. Column (1) presents unconditional mean estimates. Column (2) presents conditional mean estimates, where we control for the offspring's sex, primiparous status and birth cohort, and maternal primiparous status and birth cohort. Column (3) presents IPW estimates, where we allow the offspring sex and maternal or offspring year of birth trends and primiparous status to influence the propensity score for intergenerational and intragenerational models, respectively. Column (4) AIPW estimates, where we control for the offspring's sex, primiparous status, and birth cohort in the regression model in addition to the propensity score controls. Model (5) presents IPW estimates that control for sample selection. We report the AIPW estimates in the main paper. In model (5), we allow for the probability of having the outcome observed to influence the propensity score, in addition to the other controls. The inference is under the null that the parameter of interest is zero. Standard errors clustered at the mother level are reported in parenthesis. The stars correspond to the following p-value levels: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.