

Can Increased Recovery Rates from Coronavirus be explained by Prevalence of ADHD? An Analysis at the US Statewide Level

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Abstract

Previous research demonstrates that *ADHD* is considered a risk factor for COVID-19. The current study attempts to investigate the relationships between infection, mortality and recovery rates from coronavirus and the prevalence of *ADHD* at the US statewide level. Based on information from 2011 regarding the prevalence of *ADHD* across the US by state, findings suggest that, while there are no correlations between *ADHD* and population size, infection and mortality rates from coronavirus, recovery rates (recovery-population ratio) rise with the prevalence of *ADHD*. Consequently, a possible explanation is that in coping with the disease, *ADHD* might provide an evolutionary advantage. An example of this phenomenon can be found in the gene that causes sickle-cell disease, which, as a non-dominant gene, helps cope with infection from malaria. If corroborated, research findings may support the conclusion that coronavirus limitations in special educational frameworks for *ADHD* would not be required or could be relaxed. (*J. of Att. Dis.* 2021; 25(14) 1951-1954)
JEL Codes: H75, I12

Keywords

COVID-19, ADHD

Introduction

Attention-Deficit / Hyperactivity Disorder (*ADHD*) is considered one of the most common neurodevelopmental disorders of childhood, often extending into adulthood. Children with *ADHD* typically have trouble paying attention, controlling impulsive behavior, acting without fully considering expected results, or exhibit over-active behavior (Center for Disease Control and Prevention (CDC), available at: <https://www.cdc.gov/ncbddd/adhd/facts.html>).

Following the COVID-19 pandemic, the recent emerging research views *ADHD* solely as an additional risk factor for COVID-19. This is explained by the difficulties of those with *ADHD* to comply fully with recommendations to prevent infection from the virus (Merzon et al., 2020). Yet, one possibility that was not considered is the presence of an evolutionary advantage that *ADHD* might provide, similar to the natural immunity that sickle-cell disease provides from infection from malaria.¹ This approach stresses the theory that hemoglobinopathies protect from severe life-threatening manifestation of malaria and view one of the most important examples as: “the mutation that causes sickle cell disease (SCD) which leads to a 90% risk reduction of severe Plasmodium falciparum malaria in

sub-Saharan African children”. (p. 2—background section). In that context, some of the *ADHD* literature indicates an evolutionary advantage to *ADHD*, such as: creativity, high energy levels and preference to risk. Referring to these advantages, Shelley-Tremblay and Rosén (1996) mention that: “This is a dramatic departure from viewing ADHD only as a set of behaviors that are disruptive to academic, social and career success” (pp. 443–444).

Following Arbel et al. (2020), we test the correlation between infection, mortality and recovery from coronavirus (divided by the total population)—based on information from August 11, 2020; and prevalence of *ADHD* in 2011 at a US statewide-level. Findings suggest that, while contrary to previous studies, there are no correlations

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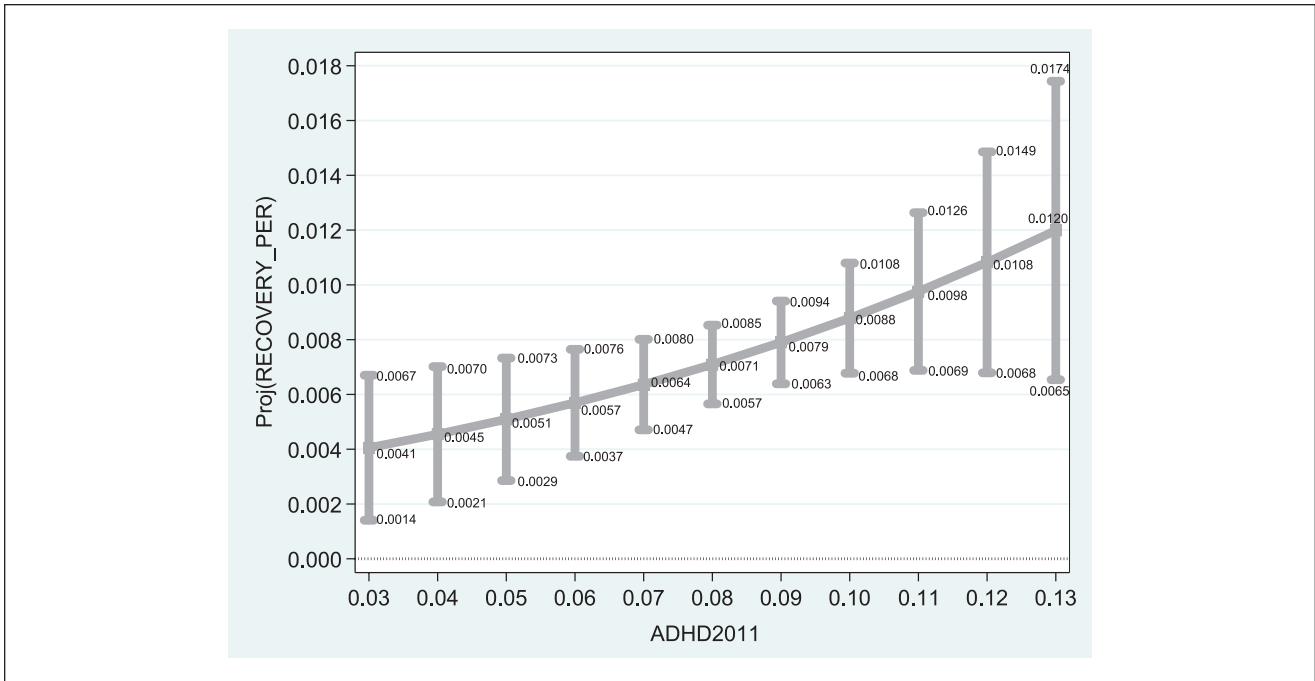


Figure 1. Projected recovery rates versus prevalence of ADHD in 2011.

Note: The graph is based on projections obtained from column (4) in Table 1, where $\hat{Y}_2 = proj(RECOVERY_PER) = proj\left(\frac{RECOVERY_CASES}{POPULATION}\right)$ in probability terms. Due to limited information regarding recovery cases from coronavirus, only 34 US states are included. The $X = ADHD2011$ variable (the horizontal axis) runs between the minimum (approx. 0.03 of the state's population) and the maximum (approx. 0.13 of the state's population).

between *ADHD* and population size, and infection and mortality rates from coronavirus, recovery rates (recovery-population ratio) rise with the prevalence of *ADHD*. These outcomes support the theory that *ADHD* may promote evolutionary advantages, which provide assistance in recovery.

Methodology

Consider the following two competing fractional probit models (e.g., Johnston & DiNardo, 1997, pp. 424–426; Papke & Wooldridge, 1996; Wooldridge, 2010):

$$Pr(0 < Y_j < 1) = F(\hat{\alpha}_{1j} + \hat{\alpha}_{2j}X) \tag{1}$$

$$Pr(0 < Y_j < 1) = F(\hat{\beta}_{1j} + \hat{\beta}_{2j}X + \hat{\beta}_{3j}X^2) \tag{2}$$

where $j=1,2,3$; $Y_1 = INFECTED = \frac{CORONA_CASES}{POPULATION}$; $Y_2 = RECOVER = \frac{RECOVERY_CASES}{POPULATION}$; $Y_3 = MORTAL = \frac{DEATH_CASES}{POPULATION}$ and all the dependent variables are

based on information obtained in August 11, 2020; X =prevalence of *ADHD* (2011), calculated as $\frac{ADHD_CASES}{POPULATION}$ in each state; $F(Y_j) = Z = \frac{exp(-\mu / 2\sigma^2)}{\sqrt{2\pi\sigma^2}}$

(the cumulative normal distribution function); α_{1j}, α_{2j} and $\beta_{1j}, \beta_{2j}, \beta_{3j}$ are parameters; and the circumflex denotes estimated parameters. Unlike equation (1), equation (2) permits non-monotonic change of the corona indicator with the X value.

Results

Figure 1 describes the projected probability of recovery vs. the prevalence of *ADHD* in 2011 based on the regression outcomes reported in column (4) of Table 1, the only column that yields a significant coefficient ($p = .0391$). Due to missing observations regarding recovery cases from coronavirus, 34 US states are included. The $X = ADHD2011$ variable (the horizontal axis) runs between the minimum (approx. 0.03 of the state's population) and the maximum (approx. 0.13 of the state's population).

Findings demonstrate that the projected probability of recovery rises from 0.41% for states with the minimum prevalence of *ADHD* (3%) to 1.20% for states with the

Table 1. Relationship between Coronavirus Indicators and Prevalence of ADHD.

Variables	(1)	(2)	(3)	(4)	(5)	(6)
	Pr. (INFECTED)	Pr. (INFECTED)	Pr. (RECOVER)	Pr. (RECOVER)	Pr. (MORTAL)	Pr. (MORTAL)
Constant	-2.133*** (<.001)	-2.309*** (<.001)	-3.194*** (8.74×10^{-9})	-2.765*** (<.001)	-3.287*** (9.52×10^{-11})	-3.322*** (<.001)
ADHD2011	-3.417 (.662)	1.040 (.409)	14.05 (.284)	3.900** (.0391)	-1.219 (.916)	-0.325 (.865)
ADHD2011 ²	26.37 (.575)		-56.56 (.456)		5.360 (.933)	
Observations	51	51	34	34	51	51
Calculated Wald-Chi ²	0.892	0.682	6.297**	4.256**	0.0300	0.0289
Degrees of freedom	2	1	2	1	2	1
5% Critical Chi ²	5.991	3.841	5.991	3.841	5.991	3.841

Note: Estimation outcomes are based on the fractional probit model and are given in terms of \mathbb{Z} = normalized standard distribution function. Robust p-values are given in parentheses.

p < .05. *p < .01.

Table 2. Pearson Correlation Matrix.

	ADHD2011	INFECTED	RECOVER	MORTAL	POPULATION
ADHD2011	1				
	51				
INFECTED = (CORONA_CASES ÷ POPULATION)	0.1177 (.4109)	1			
	51	51			
RECOVER = (RECOVERY ÷ POPULATION)	0.3893** (.0229)	0.7440*** (<.001)	1		
	34	34	34		
MORTAL = (DEATH_CASES_CORONA ÷ POPULATION)	-0.0251 (.861)	0.5722*** (<.001)	0.235 (.181)	1	
	51	51	34	51	
POPULATION	-0.1037 (.4688)	0.2962** (.0348)	0.1633 (.356)	0.1927 (.1756)	1
	51	51	34	51	51

Note: p-values for testing the null hypotheses of zero correlations are given in parentheses. **p < .05. ***p < .01.

maximum prevalence of ADHD (13%). Referring to Table 1, and in contrast to the conventional classification of ADHD as a coronavirus risk factor (e.g., Merzon et al., 2020), no correlation was found between elevated prevalence of ADHD and infection or mortality from coronavirus with respect to the population size of the state.

Finally, we test the possibility that these outcomes are not spurious and may be simply attributed to potential positive relationships between each of the two variables *per se* and the population size in the state.² To rule out this possibility, we provide a Pearson correlation matrix in Table 2 and test the possibility to reject the null hypothesis of zero correlation for each pair of variables. Findings support the possibility of zero correlations between ADHD2011 and POPULATION ($p = .4688$) and RECOVER and POPULATION ($p = .356$). Moreover, despite the high Pearson correlations between coronavirus indicators, ADHD2011 is found to be correlated only with RECOVER ($p = .029$).

Summary and Conclusions

Coronavirus disease 2019 (COVID-19) is a declared global pandemic with multiple risk factors (WHO report: coronavirus). Referring to the relationship between ADHD and COVID-19 pandemic, and based on the assumption ADHD

limits the ability to comply with COVID-19 prevention recommendations (Merzon et al., 2020), the conventional wisdom among researchers is that ADHD poses an additional risk factor for COVID-19.

The current study suggests an intriguing possibility. Rather than being a risk factor, when coping with coronavirus, ADHD also provides evolutionary advantages. This possibility was previously proposed both in the ADHD literature (e.g., Shelley-Tremblay and Rosén (1996) suggest that ADHD promotes creativity, high energy and willingness to take risks) as well as in a different context—the same gene that causes sickle cell anemia—also provides a natural vaccination against the Plasmodium parasite causing malaria (Eleonore et al., 2020).

Following Arbel et al. (2020), the objective of the current study is to test the correlation between infection, mortality and recovery from coronavirus (divided by the total population)—based on information from August 11, 2020; and prevalence of ADHD in 2011 in the US at the state-wide-level. Findings suggest that, while contrary to previous studies, there are no correlations between ADHD and population size, and infection and mortality rates from coronavirus, recovery rates (recovery-population ratio) rise with the prevalence of ADHD. These outcomes support the theory that ADHD may promote evolutionary advantages, which are of assistance in promoting recovery.

Our findings may be of assistance in dealing with coronavirus by public health systems. If corroborated, research findings might lead to the conclusion that coronavirus limitations in special educational frameworks for *ADHD* might not be required or could be reduced.

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Notes

1. Following the COVID-19 pandemic, malaria was mentioned in another context: the debate over whether hydroxychloroquine or chloroquine—prior medicines that failed to cure malaria (e.g., Trape et al., 2014, who demonstrated that the greatest changes in overcoming malaria were associated with the replacement of chloroquine and the introduction of ITNs)—are efficient in COVID-19 prevention. In this context, Negahdaripour (2020) mention that: “The arm receiving hydroxychloroquine, a formerly promising candidate, which was approved for the emergency use in COVID-19 patients by the FDA, was stopped on 17 June 2020 based on some internal and external evidence failing to reduce the mortalities.” (page 231, second paragraph).
2. For a discussion concerning spurious regressions, see, for example, Johnston and DiNardo (1997, pp. 9–11, 259–263).

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