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Effects of Biological Versus Psychosocial Explanations on Stigmatization of Children With ADHD

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Abstract

Objective: Previous studies have found biological conceptualizations of psychopathology to be associated with stigmatizing attitudes and prognostic pessimism. This research investigated how biological and psychosocial explanations for a child's ADHD symptoms differ in affecting laypeople's stigmatizing attitudes and prognostic beliefs. **Method:** Three experiments were conducted online with U.S. adults, using vignettes that described a child with ADHD and attributed his symptoms to either biological or psychosocial causes. Dependent measures gauged social distance and expectations about the child's prognosis. **Results:** Across all three studies, the biological explanation yielded more doubt about treatability but less social distance—a result that diverges from previous research with other disorders. Differences in the amount of blame ascribed to the child mediated the social distance effect. **Conclusion:** The effects of biological explanations on laypeople's views of ADHD seem to be a "double-edged sword," reducing social rejection but exacerbating perceptions of the disorder as relatively untreatable. *(J. of Att. Dis. 2012; XX(X) 1-XX)*

Keywords

ADHD, stigma, attitudes, causal attributions, biological explanations

ADHD is one of the most common childhood mental disorders, affecting an estimated 8.7% of children in the United States (Froehlich et al., 2007). Despite ADHD's relatively high prevalence, research suggests that children suffering from the disorder are subject to considerable stigmatization and misunderstanding in society (Martin, Pescosolido, Olafsdottir, & Mcleod, 2007; McLeod, Fettes, Jensen, Pescosolido, & Martin, 2007; Pescosolido, Fettes, Martin, Monahan, & McLeod, 2007; Pescosolido et al., 2008; J. S. Walker, Coleman, Lee, Squire, & Friesen, 2008). The current study examines the differential effects of biological and psychosocial accounts of ADHD on stigmatization of those with the disorder.

The stigma of mental illness is harsh and widespread, imposing a host of social misfortunes on individuals with psychiatric disorders (Hinshaw & Stier, 2008). It also has important clinical implications; among youngsters with ADHD, perceptions of the disorder as highly stigmatized negatively predict treatment utilization (Bussing, Zima, Mason, Porter, & Garvan, 2011).

One potential determinant of stigmatizing attitudes toward people with mental disorders that has received considerable attention is the extent to which psychiatric symptoms are seen as resulting from biological causes, such as genetic and neurobiological factors. Although such biomedical conceptualizations were once seen as a promising tool to combat stigma, empirical research has tended to show little benefit in this regard, and various studies have actually found that attributing psychopathology to biological causes is associated with increased stigmatization (Pescosolido et al., 2010; Read, Haslam, Sayce, & Davies, 2006). Among the stigmatizing attitudes that seem to be exacerbated by such biological attributions are prognostic pessimism (the perception that mental disorders are relatively immutable and unlikely to remit) and desire for social distance (reluctance to interact with mentally ill individuals; Haslam, 2011). Despite this, biological construals of mental disorders continue to increase in importance and prevalence, fueled by cutting-edge advances in psychiatric genetics and neuroscience (Hyman, 2007).

Although some debate whether ADHD is best understood as a cultural construct or a medical disorder (Timimi

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& Taylor, 2004), and research suggests a complex of interacting causes that defies simple etiological explanation (Sonuga-Barke & Halperin, 2010), the disorder has nonetheless been increasingly understood in biological terms. In recent years, research has implicated a variety of genetic and neurobiological factors in ADHD (Konrad & Eickhoff, 2010; Liston, Cohen, Teslovich, Levenson, & Casey, 2011; Nikolas & Burt, 2010).

Indeed, at least among some subsets of the population, biomedical conceptualizations of the disorder appear to predominate. For example, an Australian study of parents and professionals in fields related to health and rehabilitation found that neurological and genetic factors were the most commonly endorsed causal attribution for ADHD (Dryer, Kiernan, & Tyson, 2011). In this sample, genetic and neural causal attributions were especially strong among parents of children with ADHD. This finding mirrors those of an earlier study of American children with ADHD and their parents, in which 72.7% of the children and 91.3% of the parents reported believing that ADHD was inborn (Bowen, Fenton, & Rappaport, 1991).

Empirical research examining how the stigma of ADHD might be related to biological attributions for the disorder is relatively scarce. A national survey of U.S. adults revealed that respondents' reluctance to interact with a child displaying symptoms of ADHD, depression, asthma, or "normal troubles" was statistically unrelated to their endorsement of genetic or neurochemical causal attributions (Martin et al., 2007). However, Martin et al. (2007) did find that attributing the symptoms to bad character or to psychosocial factors like lack of discipline in the home or violent media was associated with increased social distance. Although these findings are valuable, they are correlational and thus uninformative about whether providing information about the etiology of mental disorders might actually *cause* changes in social distance. For example, survey participants' responses indicating their endorsement of certain causal attributions might be based on conjecture or beliefs that are vague and not firmly held, in which case they might change easily with exposure to an authoritative explanation. In addition, Martin et al. did not examine the role of causal beliefs in attitudes toward the child with ADHD separately from those concerning the children with other types of symptoms, which limits the possibility of drawing conclusions about attitudes toward ADHD per se. To our knowledge, no published study has examined how stigmatizing attitudes are affected by systematically manipulating whether or not ADHD symptoms are attributed to biological causes. Here, we report the results of our attempts to address this gap in the literature by comparing participants' stigmatizing attitudes toward a child with symptoms of ADHD as a function of whether the presented information suggested psychosocial/environmental or neural/genetic causes for the symptoms.

As explained earlier, previous studies have documented an association between stigmatizing attitudes and endorsement of biomedical conceptualizations for mental disorders (Haslam, 2011; Read et al., 2006). These findings would seem to indicate that information suggesting neural and genetic causes of ADHD could yield more negative attitudes toward a child with the disorder. However, the existing literature may also provide reasons to suspect that ADHD could be an exception to this trend.

First, most research on the stigma of psychopathology has examined attitudes toward disorders whose prototypical sufferers are adults, whereas conceptions of ADHD as primarily a childhood disorder may mean that its stigmatization is driven by unique factors. For example, children with mental health problems may be less likely to be viewed as threatening or dangerous than adults with similar symptoms. Studies that have manipulated causal attributions for psychopathology have frequently found perceptions of dangerousness to be a form of stigma that tends to be exacerbated by biological explanations (Bennett, Thirlaway, & Murray, 2008; I. Walker & Read, 2002). This may result from a perception that people whose psychiatric problems are caused by biological factors have limited control over their actions and are therefore unpredictable and potentially dangerous (Read et al., 2006). But adults may be disinclined to regard children with ADHD as dangerous or menacing, both because of their youth and because people whose disorders are characterized mainly by cognitive deficits seem to be stereotyped as incompetent but not ill-willed or threatening (Sadler, Meagor, & Kaye, 2012). This absence of dangerousness perceptions could render biological attributions less stigmatizing than they otherwise might be.

Although perceptions of dangerousness might play a smaller role in the stigma of pediatric ADHD than in that of other mental disorders, ascriptions of personal responsibility and blame might actually play a *larger* role. This, in turn, could position biological attributions as an effective measure to counteract negative attitudes. Indeed, perhaps the only positive consequence of biomedical explanations for psychopathology that has been consistently demonstrated is their capacity to reduce the blame and personal accountability attributed to individuals with mental disorders (Haslam, 2011; Mehta & Farina, 1997; Phelan, Cruz-Rojas, & Reiff, 2002). This beneficial effect likely forms the basis of popular assumptions that biomedical accounts should reduce stigma generally (Read et al., 2006). However, contrary to this conventional wisdom, the public generally does not seem to consistently blame people with mental disorders for their psychiatric illnesses, which may explain why promoting biomedical conceptualizations has failed to reduce stigmatizing attitudes overall (Angermeyer, Holzinger, Carta, & Schomerus, 2011). Unlike other mental disorders, however, the public may perceive ADHD more as a matter of personal responsibility. Many of the symptoms that form the

diagnostic criteria for ADHD can be interpreted as mere naughtiness or lack of effort. These include not listening when spoken to, not following instructions, avoiding cognitively effortful tasks, talking excessively, failing to remain seated when expected to do so, difficulty taking turns, and interrupting others. If these symptoms are seen as mere misbehavior or laziness, observers may be more likely to blame them on the child exhibiting them or attribute them to poor parenting. This would suggest that reducing ascriptions of blame and personal responsibility could be a way to diminish the stigmatization of ADHD. Because biological attributions can effectively reduce blame, biological information may actually *decrease* the stigma of ADHD.

Finally, there is now widespread understanding that ADHD often responds favorably to medications and relatively little understanding of how psychosocial interventions may be beneficial against the disorder, which may promote the notion that biological factors are most relevant in the treatment of ADHD (Hinshaw, 2005). Laypeople tend to view medications as more effective—and psychosocial interventions as less effective—for disorders that are portrayed as biologically caused (Iselin & Addis, 2003). Because the only well-known treatments effective against ADHD are medications, portraying the disorder as a result of biological factors could lead people to view it as more treatable. Some research has shown that the combination of biological causal attributions and information about treatability can reduce social distance toward people with mental disorders (Lebowitz & Ahn, 2012). Information about treatment can also effectively counter negative stereotypes about people with mental disorders (Romer & Bock, 2008). Thus, if biological explanations of ADHD are more compatible with laypeople's notions of how the disorder can be treated, they may be more likely to diminish stigmatizing attitudes.

In combination, these factors lend plausibility to the notion that biological attributions for ADHD could decrease negative attitudes. However, given the prior studies showing that biological accounts can increase the stigma of other mental disorders, it also seems conceivable that they could do the same in the case of ADHD. Thus, we sought to investigate whether participants exposed to a biological causal explanation for ADHD would show more or less negative attitudes toward a child with the disorder than would participants exposed to a psychosocial explanation. In addition, we wished to extend previous findings (e.g., Iselin & Addis, 2003) that biological attributions trigger the perception of biomedical treatments as more effective and psychosocial treatments as less effective, by demonstrating the same pattern in the case of ADHD. In three experiments, we examined whether the prognostic expectations, perceptions of treatment effectiveness, and preferences for social distance of participants exposed to a biological causal explanation for ADHD would differ from those of participants exposed to a psychosocial explanation.

Experiment 1

Method

Participants. A total of 245 adults in the United States were recruited via Amazon.com's Mechanical Turk website, which allows individuals to sign up for short tasks in exchange for small payments (Buhrmester, Kwang, & Gosling, 2011). In answering demographic questions presented at the end of the study procedure, 33 participants indicated that they had been diagnosed with ADHD or had a child with an ADHD diagnosis. These 33 participants were excluded from all analyses to standardize the sample, as extra knowledge and firmly held beliefs stemming from direct experience with ADHD might have prevented any effects of our experimental manipulations from taking hold. The remaining 212 participants were approximately 63% female and ranged in age from 18 to 64.

Procedure. The study was administered via the Internet, using Qualtrics.com online survey software. After providing informed consent, participants were randomly assigned to one of two conditions. All participants were first presented with a vignette describing a child named Andrew. Each vignette began with "Symptoms" paragraph that described Andrew and a variety of his behaviors, which were designed to meet diagnostic criteria for ADHD. Because we wished to measure the effects of our experimental manipulations rather than participants' preconceptions regarding particular diagnostic labels, the vignette did not explicitly identify Andrew's problem as ADHD. The "Symptoms" paragraph read as follows:

> Andrew is an 8-year-old boy who has been having some difficulties both in school and at home. In class, he often has difficulty staying focused and paying attention. He fidgets and squirms in his seat constantly, and the teacher often has to repeat directions for him multiple times. He becomes easily distracted, missing details and forgetting to turn in homework assignments. While the rest of the children are quietly working, Andrew is often up and out of his seat, roaming around the classroom. At home, Andrew is often impatient and often is unable to control his emotions. He becomes bored with tasks easily and frequently interrupts his parents' conversations and activities.

This description was accompanied by a paragraph of "background" information, which suggested either a biological explanation for Andrew's behavior (biological condition) or a psychosocial explanation for his behavior (psychosocial condition). In the biological condition, the "Background" paragraph read as follows:

> Andrew's father experienced similar difficulties as a child, indicating that there probably is a genetic basis

> for Andrew's problems. Andrew had his brain scanned several years ago. MRI scans of Andrew's brain revealed that he had thinner brain tissue in areas of the brain associated with attention. Andrew also appeared to have an imbalance of neurotransmitters (chemicals that act on the brain).

The "Background" component for the psychosocial condition read as follows:

> Andrew's parents have always worked long hours, and he has had a babysitter at home for as long as he can remember. This babysitter rarely disciplines Andrew and lets him watch as much television and play as many videogames as he wants. He rarely exercises. Andrew's house is always disorderly and chaotic, with stacks of papers and toys everywhere. There have never been any rules in the home, because Andrew's parents believe in hands-off parenting. His parents fight constantly and his mother has been threatening to leave the family for several years. Andrew has lived in five different cities since he was born, due to his father's business.

As a manipulation check, after reading the vignette, participants were presented with a list of three biological factors and three psychosocial factors and asked to rate how likely it was that each factor was causing Andrew's symptoms. The biological factors were "abnormal brain structure/development," "genetics," and "brain chemistry or other biochemical imbalance"; the psychosocial factors were "poor parenting (e.g., lack of discipline in the home)," "day-to-day problems or stress," and "overstimulation (e.g., videogames, violent TV, excessive Internet)." The response scales ranged from 1 (*very unlikely*) to 7 (*very likely*).

Participants then provided ratings of their desire for social distance, their prognostic expectations regarding Andrew's symptoms, and their perceptions of the potential for medication and psychotherapy to effectively treat him (see below for details). Each participant completed these three sets of dependent measures in a randomized order, and both the "Symptoms" and "Background" paragraphs were repeated with each block of questions so that participants could reference the vignette for information.

To gauge desire for social distance, we asked participants five questions related to interactions with Andrew, his family, and children similar to him. These items were as follows: (a) "How likely would you be to have Andrew and his parents over for dinner?" (b) "How likely would you be to let your child play with Andrew?" (c) "How supportive would you be of a school for children like Andrew in your neighborhood?" (d) "How likely would you be to agree to carpool with Andrew?" and (e) "How likely would you be to babysit Andrew?" All of the response scales ranged from 1 to 7, with higher scores indicating a stronger desire for social distance.

Prognostic expectations were measured with three questions. The first two measured expectations about how long Andrew's symptoms would persist. These items were "How long do you think Andrew will continue to exhibit these symptoms?" and "How long will it take for Andrew's symptoms to completely go away?" Participants answered these questions on a 9-point scale. The scale points were "less than 1 month" (coded as 1), "1 month to 6 months," "6 months to 1 year," "1 to 2 years," "2 to 5 years," "5 to 7 years," "7 to 10 years," "more than 10 years but not indefinitely/forever," and "indefinitely/forever" (coded as 9). The third item measuring prognostic expectations was "To what extent do you believe that Andrew's symptoms are treatable?" Possible responses on this treatability rating ranged from 1 (*not at all treatable*) to 7 (*very treatable*).

To measure perceptions of treatment effectiveness, we asked participants two questions about the efficacy of medication and psychotherapy as treatment for Andrew's symptoms. They were "How likely do you think it is that Andrew's symptoms will be effectively treated with medication (e.g., taking medication like Ritalin)?" and "How likely do you think it is that Andrew's symptoms will be effectively treated with therapy (e.g., seeing a professional therapist)?" Both questions had response scales ranging from 1 (*not at all likely*) to 7 (*very likely*).

At the end of the procedure, participants were asked optional questions about basic demographic information. They were then fully debriefed as to the fictitious nature of the vignettes in the study.

Results and Discussion

As expected, participants in the biological condition endorsed "abnormal brain structure/development," "genetics," and "brain chemistry or other biochemical imbalance" as causes of Andrew's behavior ($M = 5.69$, $SD = 0.93$) more strongly than those in the psychosocial condition ($M = 3.63$, $SD = 1.39$), $t(210) = -12.71$, $p < .01$. Participants in the psychosocial condition endorsed "poor parenting (e.g., lack of discipline in the home)," "day-to-day problems or stress," and "overstimulation (e.g., videogames, violent TV, excessive Internet)" ($M = 5.72$, $SD = 1.03$) more strongly than participants in the biological condition ($M = 3.74$, $SD = 1.23$), $t(210) = 12.71$, $p < .01$.

Cronbach's alpha for the five social distance items was .85, indicating a high degree of internal consistency, so they were averaged together to create an overall social distance score for each participant. Responses on the two items related to the duration of symptom persistence items were significantly correlated, $r = .69$, $p < .01$, so they were averaged to create a "duration" rating for each participant.

Table 1. Means, *n*s, and Standard Deviations for all Dependent Variables, by Condition, in Experiment 1

Dependent variable	Condition	*M*	*n*	*SD*
Treatability	Psychosocial	5.67	105	1.31
	Biological	5.37	106	1.35
Psychotherapy effectiveness	Psychosocial	4.89	105	1.56
	Biological	4.19	105	1.48
Medication effectiveness	Psychosocial	3.73	104	1.75
	Biological	5.00	106	1.45
Duration eating	Psychosocial	7.31	106	1.63
	Biological	7.27	106	1.46
Social distance	Psychosocial	4.37	106	1.24
	Biological	3.41	106	1.32

Means, *n*s, and standard deviations for all dependent variables, by condition, are displayed in Table 1.

To examine the effects of our experimental manipulations, we first compared participants in the biological and psychosocial conditions on their ratings of desire for social distance from Andrew, his family, and children similar to him. Compared with those in the psychosocial condition, participants in the biological condition reported significantly less desire for social distance, indicating less stigmatizing attitudes, $t(210) = 5.47$, $p < .01$. This finding diverges from previous studies, which have tended to find that presenting biological explanations for other mental disorders does not reduce social distance (Bennett et al., 2008; Breheny, 2007; Jackson & Heatherington, 2006; Phelan, 2005). Indeed, numerous studies have found that believing in biological causes for psychopathology is associated with increased social distance (Read et al., 2006). However, as previously stated, these earlier studies did not examine ADHD, a disorder with unique characteristics that might make biological explanations more likely to reduce social distance.

Next, we compared the treatment-related opinions and prognostic expectations of participants in the two conditions regarding Andrew's symptoms. An independent-samples Mann–Whitney *U* test revealed that our measures of expected symptom duration did not yield a significant difference between the conditions ($p = .49$). However, replicating previous studies that have linked biological explanations to doubt about the responsiveness of mental disorders to treatment (e.g., Phelan, Yang, & Cruz-Rojas, 2006), participants in the biological condition viewed Andrew's symptoms as marginally less treatable, $t(209) = 1.63$, $p = .10$. Also as expected, they rated psychotherapy as significantly less likely to be effective than did those in the psychosocial condition, $t(208) = 3.31$, $p < .01$, and they rated medication as significantly more likely to be effective than did those in the psychosocial condition, $t(208) = –5.72$, $p < .01$. Overall, these results concerning treatment beliefs and prognostic expectations conform with prior findings that biological explanations of psychiatric symptoms can lead them to be perceived as less treatable in general and especially less likely to respond to nonmedical interventions.

Because we found that participants in the biological condition, compared with those in the psychosocial condition, viewed Andrew's symptoms as less treatable, our results do not appear to support the possibility that the biological explanation's beneficial effect on social distance stemmed from increased belief in treatability. It is still possible, however, that the biological explanation positively affected social distance by reducing blame. One way in which blame could play a role in explaining our findings is evident in the psychosocial explanation presented in Experiment 1, which seemed to paint Andrew's parents as responsible for his symptoms. They were described as favoring "hands-off parenting," spending little time at home with their son, employing a highly permissive babysitter, and failing to ensure that their son exercises frequently. Thus, the difference between conditions on social distance could have been caused by negative attitudes toward Andrew's parents engendered by the psychosocial explanation. We investigated this possibility in Experiment 2, by creating a revised psychosocial explanation designed to avoid ascribing personal responsibility to Andrew's parents for his symptoms.

Experiment 2

Method

Participants. We recruited 84 participants from Mechanical Turk, screening them to ensure that none had participated in Experiment 1. Of these, 10 reported having a diagnosis of ADHD or a child with ADHD and were thus excluded to standardize the sample. The remaining participants were approximately 51% female and ranged in age from 19 to 70.

Procedure. The procedure for Experiment 2 was identical to that of Experiment 1, except that instead of the original psychosocial condition, we used a psychosocial-blameless condition in which the vignette was worded to avoid ascribing blame or personal responsibility to Andrew's parents. The "Symptoms" paragraph and the "Background" paragraph for the biological condition were identical to those used in Experiment 1. The "Background" paragraph for the psychosocial-blameless condition read as follows:

> Andrew's parents both work out of financial necessity, and they are forced to work long hours to make ends meet. Therefore, they are unable to spend much time with Andrew. Andrew is looked after by his grandmother after school, because his parents cannot afford outside child care. His grandmother has many

health problems and is unable to engage in active afterschool activities with Andrew. Andrew often ends up watching television and playing videogames by himself. Andrew's house is always disorderly and chaotic, with stacks of papers and toys everywhere.

This language was carefully chosen so that the psychosocial factors described would mirror those mentioned in the psychosocial vignette from Experiment 1 but would portray them as unavoidable misfortunes rather than the result of poor parenting. This paragraph was also closer in length to the biological "Background" paragraph.

We used the same manipulation check and dependent measures as in Experiment 1.

Results and Discussion

As expected, participants in the biological condition endorsed biological factors as causing Andrew's symptoms ($M = 5.80$, $SD = 0.99$) more strongly than did those in the psychosocial-blameless condition ($M = 3.94$, $SD = 1.34$), $t(72) = -6.83$, $p < .01$. Also as expected, participants in the psychosocial condition endorsed psychosocial factors as causing Andrew's symptoms ($M = 5.31$, $SD = 0.91$) more strongly than did those in the biological condition ($M = 3.21$, $SD = 1.23$), $t(72) = 8.21$, $p < .01$.

Cronbach's alpha for the social distance items was .84, so these were again averaged to create a social distance score for each participant. Also replicating Experiment 1, the two items asking how long participants expected Andrew's symptoms to persist were significantly correlated, $r = .50$, $p < .01$, so these were again averaged to compute a duration rating for each participant.

Our data-analysis approach was the same as the one used in Experiment 1. Means, *n*s, and standard deviations for all dependent variables, by condition, are displayed in Table 2.

As in Experiment 1, a Mann–Whitney independent-samples *U* test revealed that duration ratings did not differ significantly among participants in the two conditions ($p = .17$). Also replicating Experiment 1, however, participants in the biological condition again viewed Andrew's condition as less treatable than those in the psychosocial-blameless condition, and in Experiment 2 this difference reached significance, $t(72) = 4.43$, $p < .01$.

In a further replication of Experiment 1, participants in the biological condition saw medication as marginally more efficacious than did those in the psychosocial-blameless condition, $t(72) = 1.73$, $p = .09$, and participants in the psychosocial-blameless condition rated psychotherapy as more effective than did those in the biological condition, $t(72) = 2.44$, $p = .02$.

More importantly, mirroring the results of Experiment 1, participants in the biological condition of Experiment 2 reported less desire for social distance than did participants in the psychosocial-blameless condition, $t(72) = 2.22$, $p = .03$. This suggests that the patterns of social distance observed in Experiment 1 were likely not caused by implications of parental blame in the psychosocial condition. Indeed, de-emphasizing Andrew's parents' personal responsibility in Experiment 2 did not substantially change the pattern of differences in stigmatizing attitudes between people presented with a biological explanation of Andrew's ADHD symptoms and people presented with a psychosocial explanation.

Table 2. Means, *n*s, and Standard Deviations for all Dependent Variables, by Condition, in Experiment 2

Dependent variable	Condition	*M*	*n*	*SD*
Treatability	Psychosocial blameless	6.03	35	0.95
	Biological	4.95	39	1.12
Psychotherapy effectiveness	Psychosocial blameless	4.94	35	1.24
	Biological	4.13	39	1.59
Medication effectiveness	Psychosocial blameless	4.20	35	1.91
	Biological	4.87	39	1.42
Duration rating	Psychosocial blameless	5.83	35	1.56
	Biological	6.50	39	1.87
Social distance	Psychosocial blameless	3.63	35	1.25
	Biological	2.98	39	1.24

While the results of Experiment 2 seem to disqualify parental blame as the cause of the observed between-conditions difference in social distance, they do not eliminate the possibility that differences in the blame attributed to the child himself could be the relevant factor. Because, as previously discussed, reductions in blame have been the most consistently demonstrated benefit of biological explanations, we sought in Experiment 3 to directly test whether differences in ascription of blame to Andrew would mediate the effect of condition on social distance.

Experiment 3

Method

Participants. We recruited an additional 112 participants from Mechanical Turk, again screening them to ensure that none had participated in Experiments 1 or 2. Of these, 8 reported having a diagnosis of ADHD or a child with ADHD and were thus excluded to standardize the sample. The remaining participants were 55% female and ranged in age from 18 to 69.

Procedure. In Experiment 3, we rearranged the text of the vignettes to increase the generalizability of the findings beyond the specific manipulations used in Experiments 1 and 2 (i.e., by testing whether the pattern of results would remain even if the causal background information was not presented at the end of the vignettes). Here, the vignettes were each one paragraph, which contained the same wording as the "Symptoms" paragraph used in the prior two experiments but displayed it at the end of the vignettes instead of the beginning. In the biological condition, this symptom information was preceded by the same biological information used in Experiments 1 and 2, such that the vignette read as follows:

> Andrew is an 8-year-old boy who has been having some difficulties both in school and at home. Andrew's father experienced similar difficulties as a child, indicating that there probably is a genetic basis for Andrew's problems. Andrew had his brain scanned several years ago. MRI scans of Andrew's brain revealed that he had thinner brain tissue in areas of the brain associated with attention. Andrew also appeared to have an imbalance of neurotransmitters (chemicals that act on the brain). In class, Andrew often has difficulty staying focused and paying attention. He fidgets and squirms in his seat constantly, and the teacher often has to repeat directions for him multiple times. He becomes easily distracted, missing details and forgetting to turn in homework assignments. While the rest of the children are quietly working, Andrew is often up and out of his seat, roaming around the classroom. At home, Andrew is often impatient and often is unable to control his emotions. He becomes bored with tasks easily and frequently interrupts his parents' conversations and activities.

In the psychosocial condition, the symptom information was preceded by a revised version of the psychosocial explanation, which included neither Experiment 1's language blaming Andrew's parents for their son's problems nor Experiment 2's language explicitly absolving them of culpability. The resulting paragraph was intended to contain minimal information about personal responsibility for Andrew's symptoms, so that the extent to which participants blamed Andrew for his problems would be unaffected by information about his parents' role in the etiology of his difficulties. The new psychosocial vignette read as follows:

> Andrew is an 8-year-old boy who has been having some difficulties both in school and at home. Andrew's parents both work long hours, so he has had a babysitter at home for as long as he can remember. Andrew watches as much television and plays as many video games as he wants. He rarely gets exercise. Andrew's house is always disorderly and chaotic, with stacks of papers and toys everywhere. Andrew has lived in five different cities since he was born. In class, Andrew often has difficulty staying focused and paying attention. He fidgets and squirms in his seat constantly, and the teacher often has to repeat directions for him multiple times. He becomes easily distracted, missing details and forgetting to turn in homework assignments. While the rest of the children are quietly working, Andrew is often up and out of his seat, roaming around the classroom. At home, Andrew is often impatient and often is unable to control his emotions. He becomes bored with tasks easily and frequently interrupts his parents' conversations and activities.

As the purpose of Experiment 3 was to test whether blame could account for the differences in social distance observed in Experiments 1 and 2, we included only measures of blame and social distance as dependent variables. We planned to perform a mediation analysis, for which the preferred temporal sequence is to measure mediating variables before measuring outcome variables (Kenny, Kashy, & Bolger, 1998). As such, all participants were presented with measures of blame first, followed by measures of social distance.

To measure the extent to which participants blamed Andrew for his condition, we used three items. Because we suspected that participants might be reluctant to blame a child for his own problems per se (i.e., due to social desirability effects), each item included a hypothetical scenario outlining negative consequences of Andrew's behavior and then asked a question intended to gauge how much participants believed Andrew should be held accountable. The items were as follows:

- "Suppose that Andrew receives the treatment that is standard for problems like his but no improvement occurs. To what extent is this due to Andrew's lack of self-discipline or willpower?" (rated from 1 = *not at all* to 9 = *completely*).
- "Suppose that other children who sit near Andrew in class are distracted by Andrew's behavior, which negatively impacts their learning. To what extent should Andrew be reprimanded or punished?" (rated from 1 = *definitely not* to 9 = *definitely*).
- Suppose that Andrew is failing in school. To what extent is this due to Andrew's lack of self-discipline or willpower? (rated from 1 = *not at all* to 9 = *completely*).

We used the same social distance items from Experiments 1 and 2, but we altered the wording so that it would be

Table 3. Means, *n*s, and Standard Deviations for all Dependent Variables, by Condition, in Experiment 3

Dependent variable	Condition	*M*	*n*	*SD*
Blame	Psychosocial	4.94	55	0.23
	Biological	3.61	49	0.22
Social distance	Psychosocial	4.33	55	0.18
	Biological	3.12	49	0.19

consistent across all five items. Thus, participants rated the likelihood, from 1 (*very likely*) to 7 (*very unlikely*), that they would have Andrew and his parents over for dinner, let their child play with Andrew, support a school for children like Andrew in their neighborhood, agree to carpool with Andrew, and be willing to babysit Andrew.

Results and Discussion

Scores on the three blame items were highly consistent, yielding a Cronbach's alpha of .83, so they were averaged to compute a blame score for each participant. The social distance items also showed high reliability (Cronbach's $\alpha = .86$), so they were averaged to compute a social distance score for each participant as in the previous experiments.

Means, *n*s, and standard deviations for all dependent variables, by condition, are displayed in Table 3. Responses of participants in the biological condition indicated that they ascribed significantly less blame to Andrew than did those in the psychosocial condition, $t(102) = -4.11$, $p < .01$. Replicating Experiments 1 and 2, participants in the biological condition again reported significantly less desire for social distance (i.e., significantly less social rejection), $t(102) = 4.72$, $p < .01$. Blame and social distance scores were positively correlated ($r = .31$, $p < .01$).

To test whether the differences in blame mediated the relationship between condition and social distance, we used Preacher and Hayes's (2008) bootstrapping dialog for SPSS with 5,000 resamples. The mediation analysis is diagrammed in Figure 1. The bias-corrected 95% confidence interval for the indirect effect of condition on social distance through blame ranged from .01 to .50; mediation is indicated by the fact that 0 does not fall within this interval. Thus, differences in blame mediated the difference in social distance between participants who received a biological explanation for Andrew's symptoms and those who received a psychosocial explanation.

General Discussion

The goal of the present research was to examine whether people's stigmatizing attitudes toward a child with ADHD would differ as a function of whether they were presented with a biological or psychosocial account of the syndrome's etiology. The findings of Experiments 1 and 2 appear to depict biological explanations as a "double-edged sword" with respect to attitudes toward a child with symptoms of ADHD. Compared with a psychosocial explanation, the biological explanation yielded less desire for social distance—a departure from previous studies of causal explanations for psychopathology. However, much as in research with other disorders, it led to more pessimism about the disorder's treatability. Also consistent with previous studies of causal attributions for mental disorders, the biological explanation led to more faith in the effectiveness of medical interventions and significantly less confidence in the effectiveness of psychotherapy.

The current results clearly ruled out one potential explanation for the reduction in social distance that resulted from attributing ADHD to biological causes—namely, that a biomedical explanation might be more congruent with laypeople's understanding of how ADHD is treated and thus make the child's symptoms seem more curable. In both studies, participants who read the biological explanation saw the child's symptoms as less treatable, not more treatable, than did participants who read a psychosocial explanation. In addition, the biological condition yielded comparatively less social distance regardless of whether the psychosocial explanation portrayed the parents of the child in question as blameless or responsible for his problems. This suggests that implications of parental responsibility did not account for the stronger preferences for social distance among people exposed to a psychosocial account of the child's symptoms. Thus, in Experiment 3, we tested and found support for the hypothesis that the observed difference in social distance resulted from greater ascription of blame to Andrew himself among participants in the psychosocial condition.

One limitation of our findings is that we only measured participants' reactions to somewhat simplistic etiological accounts in which biological and psychosocial factors were never integrated. This was done to experimentally isolate the consequences of each type of explanation. Yet, given that ADHD, like other mental disorders, likely stems from complex interactions of genetic, biochemical, and environmental factors, future research should consider the implications for stigma of causal explanations that incorporate both biological information and psychosocial history. In addition, although existing research suggests that Mechanical Turk samples are geographically diverse (Buhrmester et al., 2011), we did not collect information about participants' geographical locations beyond requiring that they be in the United States. As such, our data cannot provide any insight into the role that regional differences might have played in shaping participants' reaction to the hypothetical child with ADHD. Regional differences in the diagnosis and treatment of ADHD in children have been documented (Stevens,

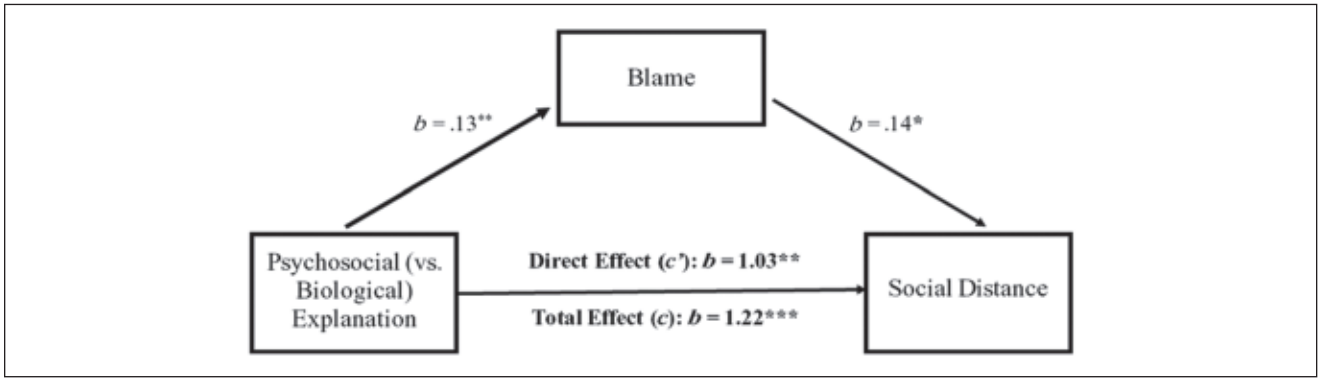

Figure 1. Results from a mediation model of blame as a mediator of the effect of explanation type (condition) on participants' desire for social distance from Andrew, a child with ADHD, in Experiment 3
Note: Characters in superscript indicate significance levels (*$p < .10$, **$p < .001$, ***$p < .0001$). The bias-corrected 95% confidence interval for the indirect effect of condition on social distance through blame ranged from .01 to .50; mediation is indicated by the fact that 0 does not fall within this interval.

Harman, & Kelleher, 2004), and U.S. regions may differ in attitudes and beliefs about children with the disorder as well. Furthermore, the majority of participants in all three of the present experiments were female, and previous research has found women's attitudes toward children with mental health problems to be less stigmatizing than those of men (Martin et al., 2007). However, we randomly assigned participants to conditions in the present studies, so gender and regional differences are highly unlikely to have confounded the effects of our experimental manipulations. Nonetheless, future research could explore these topics by directly examining the influence of geographic region and respondent gender on attitudes and beliefs about children with ADHD as well as how these factors might interact with the effects of different etiological explanations.

Our results paint a novel picture of the relationship between biological explanations for ADHD and attitudes toward children with the disorder, including the ways in which this relationship both resembles and diverges from those observed with other mental disorders. Our findings provide more evidence of one of the most consistently documented downsides of biomedical conceptualizations of psychopathology—namely, that they tend to be associated with prognostic pessimism and reduced faith in psychosocial treatments. However, our finding that preferences for social distance were relatively less pronounced among individuals presented with a biological explanation appears inconsistent with previous studies concerning the relationship of stigma to causal explanations of mental disorders. Perhaps this inconsistency should not be seen as surprising, though—after all, ADHD is a very different disorder from those that have typically been the targets of similar previous research. The assumption that all mental disorders are subject to uniform stigma may itself be stigmatizing, as has been noted in the literature (Hinshaw, 2005). By the same token, there is no reason to assume that the same antistigmatization measures will be effective for all forms of negative attitudes toward all sufferers of all disorders. In the case of children with ADHD, at least, our findings suggest that biological attributions may be effective in reducing desire for social distance by decreasing blame, although they risk engendering pessimistic beliefs about the efficacy of treatment.

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