

# A Mixed-Methods Investigation of Adolescents' Beliefs About the Causes of Depression

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## Abstract

The goal of the present study was to better understand adolescents' beliefs about what causes depression and how these beliefs relate to other clinical constructs. This study explored the causal beliefs about depression held by a diverse sample of U.S. adolescents with elevated depression symptoms ( $N=281$ ; 55% White non-Hispanic; 53% cisgender girl; 78% LGBTQ\*). Qualitative methods were used to identify causal beliefs from open-ended survey responses. Quantitative methods compared the perceived causes of one's own depression versus others' depression, compared causal beliefs across groups, and measured the association between causal beliefs and additional clinical constructs. The most common causal beliefs were dysfunctional home and family relationships (52%) and stress from school (42%). Several causal beliefs were expressed more in regard to one's own depression than others' depression (e.g., adverse childhood events, 11% vs.

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3%,  $p = .004$ ) and vice versa (e.g., social media use, 12% vs. 2%,  $p < .001$ ). Few significant relationships emerged between causal beliefs and demographic and clinical variables. Adolescents' causal beliefs about depression are diverse and multifaceted, and their causal beliefs endorsed about their own depression differ substantially from those endorsed about depression generally. However, associations between adolescents' causal beliefs and clinical variables appear limited.

### **Keywords**

depression, adolescents, causal beliefs, illness beliefs, common-sense model, explanatory model

Depression is among the most common and debilitating mental health problems worldwide (Vos et al., 2020) and is associated with a variety of adverse physical and mental health outcomes, including suicide (Brådvik, 2018). Depression commonly onsets during adolescence (Kessler et al., 2003), and the prevalence of depression and suicide has been rising significantly among adolescents worldwide (Collishaw, 2015). For any individual experiencing depression, decisions about how to manage their depression (e.g., whether or not to seek formal mental health treatment) can have major effects on the course and consequences of their condition. Understanding how adolescents manage their depression and why they select certain self-management strategies over others is of particular interest, not only because of the number of adolescents experiencing depression, but also because self-management patterns established during adolescence may have far-reaching consequences for their depression symptoms over the course of their lives.

One potentially important factor in how adolescents manage their illness is their belief about what causes that illness. Mechanisms for this relationship are articulated by the Common-Sense Model of Self Regulation (CSM, sometimes referred to as CD-SRM or SRM). The CSM posits that individuals' beliefs about their illness—including beliefs about its cause, timeline, consequences, identity, and controllability—inform how individuals manage their symptoms, which in turn impact their clinical outcomes (Leventhal et al., 2016). This theory has found support with physical illnesses (Hagger et al., 2017), and has more recently been applied to mental illnesses as well, including depression (Glattacker et al., 2018; Lynch et al., 2011). In depression, the link between self-management strategies and clinical outcomes has strong empirical support; not only can seeking treatment be an effective means of

managing one's depression symptoms, but everyday strategies for coping with emotional distress are also prospectively linked with depression outcomes (Dickson et al., 2012; Herman-Stabl et al., 1995). The link between depression illness beliefs—in particular causal beliefs—and self-management strategies has received less attention (Cannon et al., 2022).

Among adolescents experiencing depression, causal beliefs may impact help-seeking, treatment response, and even the coping strategies they select. Research with adults has shown that individuals are more likely to prefer and engage with treatments that are logically connected to their causal beliefs about depression (e.g., people show a preference for medication when they believe their depression is due to biological factors; Barnwell et al., 2022) and less likely to adhere to treatments that are not logically connected to their causal belief about depression (e.g., people show lower medication adherence when they attribute their depression to relationship problems; Lynch et al., 2006). Confidence in the effectiveness of, and engagement with, treatment for depression is subsequently related to greater symptom reduction during treatment, both in adults and adolescents (Greenberg et al., 2006; Rutherford et al., 2010). Another line of research has demonstrated the link between biogenetic causal beliefs (i.e., beliefs that depression is caused by genetic predispositions and/or “chemical imbalances”) and stronger *prognostic pessimism*: the belief that depression is unlikely to improve, even in the presence of external support or personal effort (Lebowitz, 2014). Preliminary research on prognostic pessimism in adolescents has found that adolescents who believe depression is less likely to improve are more likely to engage in maladaptive or depressogenic coping strategies (e.g., avoidance; Pinder et al., 2024), pointing to another potential link between causal beliefs and clinical outcomes. The belief that depression is malleable is also associated with more positive attitudes toward treatment (Zimmermann et al., 2020).

Previous attempts to measure causal beliefs about depression symptoms and disorders have netted a variety of different results. For example, a recent quantitative study of a non-clinical, adult, U.S. sample ( $N=319$ ) identified three dimensions of causal beliefs: biological, environmental, and psychological (Barnwell et al., 2022). Another study, which sampled New Zealand adults taking antidepressants ( $N=1,829$ ), identified biogenetic, adulthood stress, and childhood adversity dimensions (Read et al., 2014). Further studies have found additional dimensions, such as characterological (Fu & Parahoo, 2009; Murphy & Hankerson, 2018; Thwaites et al., 2004). While relatively little research has explored adolescents' causal attributions for depression, one study that did so found themes of bewilderment about the reasons for depression, depression as a result of external factors such as rejection and stress, and depression as a result of internal factors such as

psychological disposition or genetics (Midgley et al., 2017). In sum, many attempts have been made to describe the landscape of causal beliefs among different groups, and these attempts have netted a variety of results.

There are a number of reasons why different studies may uncover different patterns of causal beliefs; these reasons can be characterized as either *population-based* (owing to differences in the group being studied) or *methods-based* (owing to differences in research methods). Population-based differences reflect real differences in the distribution of causal beliefs across cultures and groups (Karasz, 2005). For example, a study on the beliefs of Indians living in Malaysia found endorsement of supernatural causal beliefs for depression (e.g., punishment from God, possession), which are generally not endorsed in studies conducted in the United States (Loo & Furnham, 2013). In addition, differences in causal beliefs between adolescents and adults may reflect actual differences in the factors that contribute to depression in these age groups (e.g., the role of school-specific stressors in adolescence; Midgley et al., 2017). Those who have experienced depression may also have different causal beliefs than those who have not, perhaps because their causal beliefs are informed by unique personal experiences (Read et al., 2014). All of these population-based factors may contribute to differences in causal beliefs found across studies.

Other differences in the types of identified causal beliefs may result from methodological differences across studies. Among these differences is the decision to query about beliefs about what causes *one's own* depression versus beliefs about what causes depression *generally*. While these are distinct constructs that may vary substantially, very little research in this area actually distinguishes between the two (Larkings et al., 2017; Read et al., 2015). Another difference is in the decision to either provide participants with a pre-specified list of causes to endorse, which constrains participants' responses, or allow participants to come up with causes on their own. Much research in this area uses "top-down" measures either created ad hoc or based on existing measures such as the Illness Perception Questionnaire (IPQ; Weinman et al., 1996) or Illness Perception Questionnaire—Revised (IPQ-R; Moss-Morris et al., 2002). While this approach can facilitate statistical methods like factor analysis, it also relies on the assumption that researchers know in advance what causal beliefs are relevant for the population being studied (Bhui & Bhugra, 2002)—whereas, to date, the literature is largely mixed with respect to which causal beliefs are held in particular samples or contexts. It is critical to attend to both population- and methods-based differences across studies when investigating perceived causes of depression: Failing to account for (and distinguish between) these differences may yield invalid conclusions about the landscape of causal beliefs across groups.

Thus, while the study of causal beliefs has the potential to explain important behaviors like help-seeking and coping strategies, doing so in a way that is valid (and, thus, more likely to explain such behaviors) requires close attention to not only the population-based factors informing causal beliefs, but also the methodological factors that inform the results of studies. Methodologically speaking, research may benefit from more qualitative, “bottom-up” approaches and from closer attention paid to the difference between causal beliefs about one’s own depression versus that of others. Attempts to learn more about the causal beliefs held by adolescents with depression may be bolstered by the use of such methods.

## **Present Study**

Causal beliefs about depression may inform how adolescents attempt to manage their symptoms (e.g., via help-seeking and coping strategies), and consequently, may impact clinical outcomes. However, the causal beliefs held by adolescents are not well understood; more research is needed to accurately assess causal beliefs about depression in adolescents and their relationship to other clinically-relevant constructs. In the present study, we utilized a mixed-methods approach to measure and analyze causal beliefs in a sample of adolescents ( $N=281$ ) living in 43 states across the United States during the COVID-19 pandemic. With these methods, we sought to achieve four related research aims that will inform our understanding of beliefs about what causes depression. First, using open-ended qualitative methods, we assessed what factors depressed adolescents believe cause depression in themselves and others. Second, using quantitative methods, we examined whether adolescents provide different reasons for their own depression than those of people generally. Third, we examined the extent to which these causal beliefs vary across demographic variables. Lastly, we explored the potential relationship between each identified causal belief, depression symptom severity, and other constructs associated with depression outcomes, such as hopelessness (Liu et al., 2015) and the perceived permanence of depression (Schroder et al., 2016).

## **Method**

### *Procedures*

All study procedures, measures, and analytic methods were pre-registered at <https://osf.io/d7wyn/> and approved by the University of Denver Institutional Review Board. This study’s sample was recruited from the participant pool of a previous study (original study  $N=2,452$ ), which was conducted 6 months

earlier (Schleider et al., 2022). Participants in the original study disproportionately held minoritized gender and sexual identities, a pattern that persists in the current sample. The original study did not specifically recruit for sexual and gender minority youth; instead, the authors suggest that sexual and gender minority youth may be disproportionately drawn to take part in online self-help activities for depression (Schleider et al., 2022). Participants in the previous study were recruited via advertisements posted online to web-based forums and social media (e.g., Instagram); participants in this study were drawn at random from this study pool via email invitations to participate in a follow-up study. A total of 925 emails were sent, and 348 participants responded (37.6% response rate). Of those 348, 41 responses were incomplete, and an additional 26 responses were duplicates, leaving a total of  $N=281$  participants in the present study. All participants received a \$10 Amazon gift card in exchange for their participation. All participants were aged 13 to 16, fluent in English, and screened positive for elevated symptoms of depression in the recruitment period of the initial study (defined as a score of  $\geq 2$  on the PHQ-2; Richardson et al., 2010).

We cannot preclude the possibility that participants' beliefs about depression might have been shaped by their participation in the original study. However, the activities participants engaged in during that study were not expected to significantly affect their beliefs about depression (e.g., none of the online interventions to which adolescents were randomized explicitly endorsed specific causal beliefs about depressive symptoms or disorders). We conducted statistical tests to assess any bias that might have resulted from the previous study, detailed below. Furthermore, we explored the possibility that responders to recruitment emails were systematically different from non-responders, detailed further below.

## Measures

For the first and second research aims, we asked the following open-ended questions: "If you experience depression, what do you think is the cause of your depression?" and "What do you think causes depression for most people your age?" Qualitative coding steps are outlined in the following subsection.

For the third research aim, we collected the following demographic information via adolescent self-report: race/ethnicity, gender, sexual identity, and age. Racial-ethnic identity was divided into the following five categories: White non-Hispanic, Black or African American non-Hispanic, Asian non-Hispanic, other non-Hispanic, and Hispanic. We opted to use a single-variable approach to operationalizing race/ethnicity to avoid conflating responses

from White non-Hispanic respondents and Hispanic respondents who do not belong to a second community of Color, which would be done in an analysis using only race, and to avoid conflating responses from White non-Hispanic respondents and non-Hispanic respondents of Color, which would be done in an analysis using only ethnicity. Gender was coded as girl/woman, boy/man, or transgender/gender diverse (TGD). Sexual orientation was coded dichotomously, as either heterosexual or lesbian, gay, bisexual, queer, or another sexual identity (LGBQ\*). In addition, gender and sexual identity frequencies are reported descriptively for all categories measured. Age was measured continuously, in years (13–16).

For the fourth research aim, we measured the following clinical constructs: depression symptom severity, the perceived permanence of depression, hopelessness, the perceived helpfulness of therapy, and the perceived helpfulness of medication. Depression symptom severity was measured using the Children's Depression Inventory 2nd Edition Short Form (CDI-2-SF), a 12-item version of the CDI-2 (Kovacs, 2011). The CDI-2-SF is a valid, reliable measure of depression symptoms among adolescents (Allgaier et al., 2012). Cronbach's alpha for the CDI-2-SF in this sample was  $\alpha = .85$ . Participants' perceived permanence of depression (prognostic pessimism) was assessed with a single item, consistent with prior studies assessing this construct (Lebowitz & Ahn, 2015): "Imagine you go through depression in the future. . . how permanent do you think the depression would be?" on a scale from 1 (not permanent at all) to 10 (completely permanent). Hopelessness was measured via the Beck Hopelessness Scale Four Item Version (BHS-4; Perczel Forintos et al., 2013). Cronbach's alpha for the BHS-4 in this sample was  $\alpha = .86$ . The perceived helpfulness of therapy was assessed with a single item, also consistent with prior studies assessing this construct (Khalsa et al., 2011): "How helpful do you think talking with a therapist would be when it comes to your emotional or mental health problems?" on a scale from 1 (not helpful at all) to 10 (extremely helpful). The perceived helpfulness of medication was assessed the same way but asked participants about "taking medication" instead of "talking with a therapist." While we pre-registered that we would examine self-hate as an outcome, our self-hate measure was programmed incorrectly in our survey, so we do not report it.

### *Qualitative Coding*

Using the coding reliability approach of thematic analysis (Braun et al., 2019), researchers identified categories of causal beliefs from two open-ended, free-response questions, to which participants were able to respond with as many or as few details as they preferred: (1) "If you experience

depression, what do you think is the cause of your depression?” and (2) “What do you think causes depression for most people your age?” Responses to both questions were coded using one study-wide codebook (described below, and available at <https://osf.io/d7wyn/>), to allow researchers to compare responses across items. Our brief approach to measuring causal illness beliefs fits with prior approaches, which have yielded reliable thematic coding schemes based on as few as one free-response question (e.g., “why do you have [your illness]?”; Stern & Kirmayer, 2004; G. Williams, 1984).

To identify causal beliefs, three independent coders (the first, second, and third authors) took the following steps. In the first step (code development), coders independently reviewed a random subset of 60 responses (two each for 30 participants); each coder then independently brainstormed potential coding categories. Next, the three coders met to draft an initial consensus codebook based on their initially-identified themes. In the second step (code revision), each coder used the draft codebook to independently code a second randomly-selected subset of 60 responses. After this, coders met again to revise the codebook, based on themes identified during the second independent coding phase. In the third step (code validation), each coder used the revised codebook to independently code a third random subset of 60 responses. Codes with reliability estimates of  $k = .8$  or higher were kept in the final codebook, while codes with reliability estimates of  $k < .8$  were either dropped or revised and subject to further rounds of validation until reliability reached  $k = .8$  (McHugh, 2012). Once the codebook was finalized, coders independently coded all responses ( $N = 562$ , two open-ended responses per participant). All disagreements were resolved via discussion to achieve a consensus dataset of coded responses. A causal belief could be coded in multiple ways, if it fit the definition of both codes; however, each causal belief was only counted once for the purpose of calculating the total number of causal beliefs endorsed by each respondent. The complete codebook is available alongside the study’s pre-registration at <https://osf.io/d7wyn/>.

### *Data Analytic Plan*

Analytic code for quantitative analyses is available alongside the study’s pre-registration at <https://osf.io/d7wyn/>. Data processing and analysis were conducted in R. All analyses were limited to complete cases, dropping 36 responses (of 562) where participants did not provide causal beliefs about their own depression (generally because they did not identify as having depression).

*Aim 1: Description of Causal Beliefs.* After qualitatively identifying the categories of causal beliefs endorsed by respondents, we reported these categories and their frequencies. For each category of causal belief, we reported (a) the proportion of respondents who endorsed this factor as a cause of their own depression (per the first open-ended prompt), (b) the proportion of respondents who endorsed this factor as a cause of depression in general (per the second open-ended prompt), and (c) the proportion of respondents who endorsed this factor in response to either the first or the second free-response prompt.

*Aim 2: Causal Beliefs for the Self Versus Others.* To compare the types of causal beliefs endorsed for one's own depression versus those endorsed for depression in general, we conducted a series of McNemar chi-square tests, a chi-square test appropriate for use with paired (e.g., within-subjects) data. A paired *t*-test (not pre-registered) was also conducted to compare the total number of causes endorsed for one's own depression versus depression in general. To account for the large number of statistical tests, the Benjamini-Hochberg method was used to limit the false discovery rate to .05 (Benjamini & Hochberg, 1995).

*Aim 3: Demographic Predictors of Causal Beliefs.* A series of logistic regressions were conducted to determine if participants' endorsement of a given causal belief (in regards to one's own depression or depression generally, examined separately) differed significantly by demographic variables. The initial pre-registration specified that causal beliefs about one's own depression and beliefs about depression generally would be conflated for the sake of this analysis, but we chose to examine each separately to better align with the aims of this study. One logistic regression was conducted per causal belief/demographic variable pair. Demographic variables included race/ethnicity, gender, sexual orientation, and age. For categorical demographic variables (i.e., race/ethnicity, gender, and sexual orientation), we excluded subgroups with  $n < 25$  respondents from logistic regressions to reduce the risk of reporting unreliable estimates (as specified in the pre-registration). As a result, logistic regressions using race/ethnicity as the independent variable were limited to the categories of White non-Hispanic, Asian non-Hispanic, and Hispanic, with White non-Hispanic as the reference category, and logistic regressions using gender as the independent variable were limited to the categories of cisgender girl/woman and TGD, with cisgender girl/woman as the reference category. Logistic regressions using sexual orientation as the independent variable included the categories of heterosexual and LGBTQ\*, with heterosexual as the reference category, and logistic regressions using age as

the independent variable treated age (in years) as a continuous predictor. While the study's pre-registration stated that symptom severity would also be used as an independent variable for this aim, this analysis was dropped as it was redundant with the analyses in Aim 4. To account for the large number of statistical tests, the Benjamini-Hochberg method was again used to limit the false discovery rate to .05.

*Aim 4: Associations with Clinical Variables.* To assess the relationship between each causal belief (endorsed either in regard to one's own depression or depression generally, examined separately) and relevant clinical variables, we conducted a series of Pearson correlation tests. The initial pre-registration specified that causal beliefs about one's own depression and beliefs about depression generally would be conflated for the sake of this analysis, but we chose to examine each separately to better align with the aims of this study. Clinical variables included depression symptom severity, the perceived permanence of depression, hopelessness, the perceived helpfulness of therapy, and the perceived helpfulness of medication. Correlation tests were also conducted between the total count of causal beliefs endorsed and the aforementioned clinical variables. The Benjamini-Hochberg method was used to limit the false discovery rate to .05.

*Robustness Checks.* To assess the possibility that participants' responses in the present study might have been affected by their participation in the previous study (Schleider et al., 2022), we conducted chi-square tests of independence to determine if causal beliefs endorsed by respondents varied significantly by the intervention to which they were randomized in the initial study (i.e., whether they received one of two single-session online interventions, or an active control program), with the Benjamini-Hochberg procedure set to limit the false discovery rate to .05. Furthermore, we conducted chi-square and *t*-tests to explore differences between responders and non-responders on demographic characteristics (age, race, gender identity, sexual orientation) and depression symptom severity collected in the original study. These checks were not included in the pre-registration, but they were added afterwards for the sake of thoroughness.

## Results

### *Sample Characteristics*

Table 1 reports sample characteristics. A total of 281 adolescents participated in this study. Participants were 55% White non-Hispanic, 17% Hispanic,

16% Asian non-Hispanic, 6% Black non-Hispanic, and 6% another race/ethnicity. The mean age was 15.20 years and the standard deviation was 0.87. Participants were 53% girls/women, 9% boys/men, and 39% TGD (broken down further in Table 1). Most participants (78%) reported a sexual orientation other than heterosexual (broken down further in Table 1), while 22% identified as heterosexual. Scores on the CDI-2-SF were distributed with a mean of 11.30 and a standard deviation of 5.00. Approximately two-thirds (63%) of participants scored at least 10 on the CDI-2-SF, which has been one clinical cutoff proposed for this measure (Cho et al., 2022). The demographic profile of the present sample approximately reflects the composition of the original study's sample, which included a relatively large proportion of LGBTQ\* (79%) and TGD participants (22%) and 67% White non-Hispanic participants.

### *Aim 1: Description of Causal Beliefs*

Qualitative coding of responses identified 27 categories of causal beliefs that could be meaningfully and reliably distinguished from one another. A complete breakdown of causal beliefs endorsed by the participants in this study, including their definitions and examples, can be found in Table 2. A visual representation of the frequency of each theme (endorsed as either a cause of one's own depression or a cause of depression generally) is presented in Figure 1.

The most highly cited cause for depression was dysfunctional home or family relationships. More than half (52%) of respondents cited a cause for depression that fell in this category. Examples included "abusive parents," "unsupportive family," and "family issues." Dysfunctional peer relationships were also among the most commonly cited causes for depression (28%). Examples of this included "toxic friends," "bullying," and the "social aspects of teenage years."

Stress from school was the second most highly cited cause, with 42% of respondents endorsing a cause that fell in this category. Examples included "school load," "academic burnout," and simply "school." The weight of pressure and external expectations—largely regarding, but not limited to, school—was also a commonly cited cause, as 23% of respondents identified this cause. Examples of this included "pressure to do well," "having to keep up with unrealistic goals," and "pressure to fit in." Many participants also endorsed "stress" as a cause of depression without providing a specific source of this stress (e.g., "stress"). This cause was identified in 17% of responses.

Biogenetic causes (e.g., "chemical imbalance," "hormones," and "genetic clinical depression") were endorsed by 19% of respondents. Trauma, for

**Table 1.** Sample Characteristics.

Variable	<i>n</i>	Percent	<i>M (SD)</i>
<b>Race/ethnicity</b>			
White non-Hispanic	154	54.80	
Black non-Hispanic	17	6.05	
Asian non-Hispanic	44	15.66	
Other non-Hispanic	17	6.05	
Hispanic	49	17.44	
<b>Gender</b>			
Cisgender girl/woman	148	52.67	
Cisgender boy/man	24	8.54	
Trans/gender diverse	109	38.79	
Transgender	26	5.99	
Female to male transgender	22	5.07	
Male to female transgender	1	0.23	
Trans male/masculine	28	6.45	
Trans female/feminine	3	0.69	
Genderqueer	17	3.92	
Gender expansive	6	1.38	
Androgynous	17	3.92	
Nonbinary	46	10.60	
Two-spirited	2	0.46	
Third gender	1	0.23	
Agender	10	2.30	
Not sure	27	6.22	
Other/not listed	8	1.84	
<b>Sexual orientation</b>			
Heterosexual	63	22.42	
LGBQ*	218	77.58	
Gay/lesbian/homosexual	31	11.03	
Bisexual	75	26.69	
Pansexual	27	9.61	
Queer	28	9.96	
Asexual	15	5.34	
Other/not listed	3	1.07	
Unsure/questioning	23	8.19	
Do not use a label	16	5.69	
Age			15.20 (0.87)
Depression symptom severity (CDI-2-SF)			11.30 (5.00)

Note. Trans/Gender Diverse categories are not mutually exclusive; total value exceeds 281.

**Table 2.** Perceived Causes of Depression.

Cause	Definition	Examples	Percent endorsed (%)		
			Self	Others	Either
Adverse childhood events	Something that happened in a person's early life history, generally childhood	"I was never allowed to truly show or process a lot of the traumas and personal problems I face. . . I don't talk and ignore my issues and traumas"	10.61	2.49	11.03
Biogenetic	Something biological, including a "chemical imbalance" or genetics	"I think that's caused by something wrong in your brain, chemical imbalance maybe"	12.65	15.66	18.86
Body image	Body image issues or body dissatisfaction	"Being insecure about my appearance"	2.86	2.85	4.27
Community/systems	Dysfunction or problems between an individual and their community	"Well for LGBTQIA+ youth and people a lack of acceptance and validation from the people around you can play a part in it"	3.67	4.63	5.69
Different for everybody	There is no way to say what causes depression since it's different for everybody	"There is no answer because everyone is different and it happens to anyone"	0.00	2.14	2.14
Dysfunctional home/family relationships	Present/ongoing dysfunction or problems in one's home situation or relationship with one's family	"Parents also make a huge impact for teenagers suffering with depression. If they aren't supportive it really messes with you"	33.06	40.93	51.96
Dysfunctional peer relationships	Present/ongoing dysfunction or problems in one's relationships with one's peers	"Bullying," "feelings of inferiority and inadequacy caused by peers," "fake friends"	8.57	24.20	28.11

(continued)

**Table 2. (continued)**

Cause	Definition	Examples	Percent endorsed (%)		
			Self	Others	Either
Global/social problems	Feelings that the world is in crisis or in decline	"Many teens have been traumatized by current events," "stress and fear for the world (i.e., climate change)"	6.53	13.17	16.01
Grief	Experiencing grief or the loss of a loved one	"The loss of a loved one"	0.82	1.42	2.14
Hereditary	Inheriting depression from one's parents, either via genetics or non-biological means	"Mine is hereditary. My parents both have depression, and I do too"	11.43	8.90	12.46
Identity-related stressors	Stressors related to one's identity, especially minority identities	"My existence is being debated and even outlawed by people who don't do their research and are blinded by religion"	6.94	5.69	9.25
Just "happens"	Depression just happens "out of the blue"	"It can also just happen out of nowhere," "sometimes it isn't caused by anything in particular"	0.41	2.14	2.49
Lack of control	Not having control over one's present or future situation	"Lack of control over my life"	1.22	1.07	1.78
Lack of hope or certainty about the future	Not knowing where to go in one's life, what to do next, not having a purpose, or being worried or pessimistic about where one's life is going	"Being unsure about the future," "I think we can't see the point in life sometimes"	4.08	6.05	7.12

(continued)

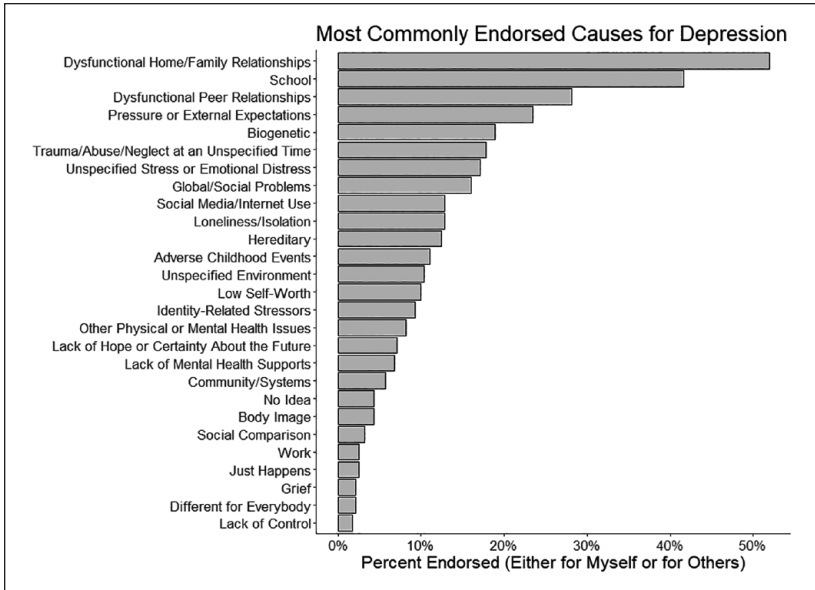
**Table 2. (continued)**

Cause	Definition	Examples	Percent endorsed (%)		
			Self	Others	Either
Lack of mental health supports	Not having the support—social, professional, or otherwise—to avoid depression (response distinguishes from loneliness or isolation in general)	“Lack of accessible therapy,” “lack of empathy or care from others”	3.27	6.05	6.76
Loneliness/isolation	Present/ongoing lack of connections to one’s family or peers	“Being isolated a lot,” “having little to no friends”	9.80	6.41	12.81
Low self-worth	Having a low opinion of oneself or feeling that one is worthless	“Not feeling like I’m worth anyone’s time or energy”	8.57	5.37	9.96
No idea	Respondent doesn’t know what causes depression	“Hard to pinpoint,” “I don’t know”	3.27	1.78	4.27
Other physical or mental health issues	A comorbid physical or mental health issue	“Early undiagnosed ADHD symptoms making school difficult,” “untreated for physical ailments that cause me pain”	7.35	2.14	8.19
Pressure or external expectations	Failure to live up to either one’s own or someone else’s expectations for oneself	“High standards presented by families, friends, and social media”	11.02	20.64	23.49
School	The stress of school, primarily one’s responsibilities	“School stress,” “pressures of school work, extra curriculars, getting into a good college”	22.86	38.79	41.64

(continued)

**Table 2.** (continued)

Cause	Definition	Examples	Percent endorsed (%)		
			Self	Others	Either
Social comparison	Self-comparison to other individual or groups; social comparison is interpersonal in nature and does not capture pressure to fit into society at large	"I feel like a failure at times, comparing myself to peers who've accomplished so much more and are practically certain to be accepted to prestigious colleges"	2.45	2.14	3.20
Social media/ internet use	Overuse of social media and/or the internet	"Overuse of social media has caused us to be over reliant on our phones as a source of dopamine"	2.45	12.81	12.81
Trauma/abuse/ neglect at an unspecified time	Trauma (response does not specify time)	"Abuse, neglect, and trauma takes a high place for many"	11.43	11.74	17.79
Unspecified environment	One's environment (response does not specify beyond that)	"Harsh environments," "living in a bad environment"	6.53	7.47	10.32
Unspecified stress or emotional distress	Stress and/or stressful emotions (response does not specify from where)	"Stress," "major life stressors"	10.61	13.88	17.08
Work	The stress of non-school work, primarily one's responsibilities	"Work life has been very tense," "the stress of jobs"	0.82	2.14	2.49



**Figure 1.** Most commonly endorsed causes for depression.

example, from victimization, abuse, or neglect, was cited as a cause by 18% of respondents, without stating specifically whether the trauma had to occur immediately before the depression or earlier in one's life. An additional 11% attributed depression to trauma (or other adverse events) experienced in childhood specifically. Global and social problems (e.g., "world events," "the pandemic," and "the monotonous grind of surviving under capitalism") were cited as a cause of depression for 16% of respondents.

### *Aim 2: Causal Beliefs for the Self Versus Others*

A number of causal beliefs were endorsed more or less by respondents when talking about their own depression, relative to when they cited causes for depression in general. After adjusting for multiple comparison testing, seven causal beliefs showed statistically significant differences. Participants were significantly more likely to cite the following five causes when talking about depression *in general* than when talking about their own depression: dysfunctional peer relationships (24% vs. 9%;  $X^2=21.97$ ,  $p < .001$ ); global/social problems (13% vs. 7%;  $X^2=7.76$ ,  $p = .020$ ); pressure or external expectation (21% vs. 11%;  $X^2=11.61$ ,  $p = .004$ ); school (39% vs. 23%;  $X^2=22.67$ ,

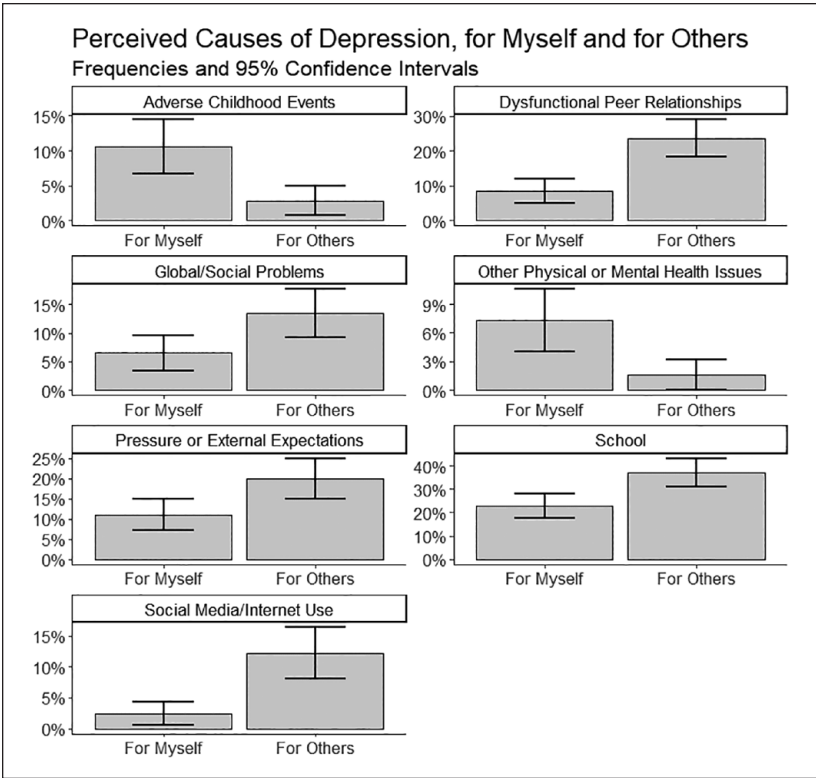


Figure 2. Perceived causes of depression, for myself and for others.

$p < .001$ ); and social media/internet use (13% vs. 2%;  $\chi^2=22.04, p < .001$ ). There were two causes that participants were more likely to cite when discussing *their own* depression than when talking about depression in general: adverse childhood events (11% vs. 2%;  $\chi^2=11.17, p=.004$ ) and comorbid physical or mental health issues (7% vs. 2%;  $\chi^2=8.45, p=.016$ ). Figure 2 presents comparisons of these frequencies for causal beliefs with statistically significant differences.

Respondents also reported more causal beliefs when asked about the causes of depression for others ( $M=2.32, SD=1.64$ ) than when asked about the causes of their own depression ( $M=1.78, SD=1.08$ ). This difference was statistically significant ( $p < .001$ ).

### *Aim 3: Demographic Predictors of Causal Beliefs*

Only one demographic factor emerged as a statistically significant predictor of causal beliefs in this sample, after adjusting for multiple comparisons. Asian non-Hispanic respondents were more likely to endorse pressure or external expectations as a cause of their own depression (33%) when compared to White non-Hispanic respondents (7%), with an odds ratio of 7.06 ( $p = .030$ ). Complete results for Aim 3 are available in Supplemental Appendix A.

### *Aim 4: Associations with Clinical Variables*

After adjusting for multiple hypothesis testing, two causal beliefs were associated with the clinically-relevant variables studied. Endorsement of adverse childhood events as a cause of one's own depression was associated with greater depression symptom severity ( $r = .29, p = .001$ ), and endorsement of hereditary factors as a cause of others' depression was associated with a greater belief in the helpfulness of medication ( $r = .23, p = .024$ ). Complete results for Aim 4 are available in Supplemental Appendix B.

### *Robustness Checks*

Regarding differences in causal beliefs by intervention assignment, all  $X^2$  tests resulted in  $p$ -values of above .05, indicating that participants' causal beliefs about depression were not significantly associated with intervention assignment in the original study, conducted 6 months earlier. These tests are reported in full in Supplemental Appendix C. Regarding differences in demographic characteristics by response to recruitment emails, we found no differences by gender, age, or depression symptom severity ( $p$ 's  $> .05$ ), but significant differences by race/ethnicity (Asian non-Hispanic: 49.5%, Black non-Hispanic: 41.0%, White non-Hispanic: 36.5%, Hispanic: 35.4%, other non-Hispanic: 23.8%;  $p < .001$ ) and sexual orientation (heterosexual: 42.6%, LGBTQ\*: 34.1%;  $p = .035$ ).

## **Discussion**

This study examined causal beliefs about depression in a diverse U.S. sample of  $N = 281$  adolescents with elevated depression symptoms. From adolescents' qualitatively coded responses, 27 distinct causal beliefs were reliably coded, with beliefs covering a wide range of causal factors (e.g., school, dysfunctional relationships, biogenetic causes, trauma). Despite the diversity of

causal beliefs expressed by adolescents, certain beliefs emerged among large proportions of respondents. Our findings further demonstrate large, significant differences in the causal beliefs adolescents hold about their own depression and the causal beliefs adolescents hold about others' depression. However, we did not find that causal beliefs were significantly associated with demographic characteristics or clinical variables in the vast majority of cases.

The wide range of causal beliefs identified via open-ended qualitative methods stands in contrast to certain top-down tools used to measure causal beliefs, which may query about only a few causal beliefs (e.g., 11, as in Barnwell et al., 2022). Not only did adolescents report a wide range of causal beliefs, a majority (52%) provided more than one cause for their own depression, with some reporting as many as seven causes. While this study is not the first to observe the complexity of causal beliefs (B. Williams & Healy, 2001), it is an important reminder that top-down approaches that impose constraints on the causes endorsed by respondents may fail to accurately reflect their causal beliefs. Moreover, the large number of respondents who endorsed multiple causes challenges theories that people tend to essentialize mental illness, reducing it to a single cause (Ahn et al., 2017).

Further illustrating the complexity of adolescents' causal beliefs about depression, our findings demonstrate large, significant differences in the causal beliefs adolescents hold about their own depression versus the causal beliefs adolescents hold about depression in general. Participants were significantly more likely to identify adverse childhood events, as well as comorbid physical or mental health issues, as causes of their own depression than as causes of others' depression. For other causal beliefs (dysfunctional peer relationships, global/social problems, pressure or external expectations, school, and social media/internet use), participants were significantly more likely to endorse them in regard to others' depression than in regard to their own. One potential explanation is that participants base their beliefs about depression *in general* on the socially constructed knowledge about depression they have internalized, while they base their beliefs about *their own* depression more so on their own personal experience. For example, the belief that social media or internet use can lead to depression (especially among adolescents) has received significant media attention in the United States, and was endorsed by 13% of participants when asked about depression in general. However, participants in this study were much less likely to volunteer this as a cause of their own depression, with only 2% believing their own depression was caused by social media or internet use. A similar "me versus them" phenomenon has also been found with the effect of mental health stigma on adolescents' help-seeking behaviors (Villatoro et al., 2022). Future

research should be clear about which causal belief construct is being studied, given the significant differences between the two. Moreover, future studies may seek to explain why this discrepancy occurs.

Despite the wide range of causal beliefs endorsed, many were still shared by a large proportion of adolescents. Many of the most commonly endorsed causal beliefs align with existing research on developmental risk factors for depression. For example, dysfunctional home and family relationships were the most commonly cited causes of depression in this sample (52%), and these have also been empirically identified as risk factors for depression in adolescence (Beardslee et al., 2012). Some participants in this sample, which was largely comprised of sexual and gender minority adolescents, also reported identity-related stressors (e.g., not being accepted because of their sexual or gender identity) as a cause of depression (9%), aligning with research on minority stress as a risk factor for depression (Baams et al., 2015). However, other causal beliefs identified in this study do not align with research on developmental risk factors for depression. For example, while school-related stressors were endorsed by many adolescents (42%, replicating a finding from Midgley et al., 2017), stress from school is rarely considered a developmental risk factor for depression (Beardslee et al., 2012). Future research on developmental risk factors for depression may benefit from examining the role of school stress more closely.

Notably, we found a large discrepancy between the number of respondents in this study endorsing biogenetic causal beliefs and estimates found by previous studies. Biogenetic beliefs were endorsed by around a fifth of respondents (19%), many fewer than found in other studies also conducted in the United States (e.g., 67% reported in Pescosolido et al., 2010). Given the ascendancy of biogenetic causal beliefs in recent decades (Schomerus et al., 2012), this is not likely due to our study being conducted more recently. One possible explanation for this result is that adolescents are less likely to endorse this belief than adults, perhaps because they have been less exposed to the biogenetic model at this stage. However, this discrepancy may also be explained by methodological differences across studies; studies asking participants whether or not they agree that a biogenetic factor may cause depression may present a “lower bar” than studies requiring participants to generate this belief without prompting. Research on the population prevalence of causal beliefs must pay close attention to this difference and how it may influence results.

Only one demographic variable was significantly associated with causal beliefs, as Asian non-Hispanic respondents were more likely to endorse pressure or external expectations as a cause of their own depression than White non-Hispanic respondents. This result is consistent with prior research finding

a relationship between maladaptive perfectionism, parent-driven perfectionism, and depression symptoms among Asian college students in the United States (Yoon & Lau, 2008). To build on this result, future research may explore the role of perceived external pressure in how Asian American adolescents navigate the experience of depression. Notably, however, no other demographic characteristics were significantly associated with causal beliefs. While our results do not preclude such differences, our findings are limited by our often small per-group sample size—they suggest that cultural variation in causal beliefs for depression among U.S. adolescents may be relatively limited.

With respect to links of causal beliefs to clinically-relevant variables, adolescents who endorsed adverse childhood events as a cause of their own depression reported significantly greater depression symptom severity, and those who endorsed hereditary causes for others' depression (e.g., genetic susceptibility) rated medication-based treatment as more likely to be effective. The former relationship has not been found in research on causal beliefs to date, and may reflect a relationship between the experience of adverse events in early childhood and more severe depression symptoms within this sample, which was selected on the basis of elevated depression symptoms. The latter relationship replicates earlier research demonstrating a relationship between individuals' causal beliefs about depression and their treatment preferences (e.g., Barnwell et al., 2022; Lebowitz, 2014). However, we did not find further significant correlations between causal beliefs and clinical variables. While these null results do not preclude such relationships, they do suggest that causal beliefs may not be especially strong predictors of hopelessness, timeline beliefs about depression, and attitudes toward treatment among U.S. adolescents.

## Limitations

Several limitations and design features suggest directions for future research in this domain. First, while theory suggests that causal beliefs may influence illness management behaviors such as help-seeking and coping, we did not measure these constructs. Future work is needed to unpack how causal beliefs influence these behaviors. Moreover, as a cross-sectional study, we were unable to assess the stability of causal beliefs over time, or the longitudinal relationship of causal beliefs with other clinical variables. This is notable as previous research has found that causal beliefs vary substantially over time (McCabe & Priebe, 2004), which may limit the strength of their relationship to the other clinical constructs assessed here. The size of our sample may further limit our ability to identify meaningful relationships between causal beliefs and clinical variables, or differences in causal beliefs across demographic groups. For example, this study was not well-equipped to draw

generalizable results about Black non-Hispanic ( $n=17$ ) or cisgender male adolescents ( $n=24$ ). In addition, some constructs were measured using single-item measures; while this is consistent with prior research in this area, it may limit the reliability of such constructs in our sample and, therefore, the strength of their relationships with other variables. Lastly, two characteristics of the present study are reasons for caution regarding generalizability: first, that the sample (as with the sample of the original RCT) disproportionately consisted of sexual and gender minority youth, and second, that there were significant differences between respondents and non-respondents in terms of race/ethnicity and sexual orientation.

Further, the qualitative approach to measuring causal beliefs can be considered both a strength and limitation for this study. Measuring these beliefs as dichotomous variables hides differences in the degree of belief, and may thus limit our ability to identify significant relationships between beliefs and other variables. This approach also makes investigating the factor structure of our participants' beliefs significantly more challenging, as our data is dichotomous, frequencies are generally low, and correlations between beliefs are generally small.

## **Conclusions and Future Directions**

This study sheds light on the causal beliefs held by a diverse group of U.S. adolescents experiencing elevating depression symptoms. Moreover, this study highlights significant differences in causal beliefs elicited via open-ended versus closed-ended methods, and when asking about one's own depression versus that of others. Researchers investigating causal beliefs must be aware of both population- and methods-based differences across studies, and the implications for their own research, or risk making invalid comparisons across studies. Our design and approach may offer a roadmap for research hoping to minimize this risk. First, by inquiring about causal beliefs using open-ended methods, future studies would not risk excluding unexpected causal beliefs. Second, by drawing a distinction between beliefs about one's own depression and those regarding depression generally, future studies can hone in on the relationships that truly matter for how individuals manage their depression.

Public health messaging campaigns (as well as corporate advertising campaigns) have been used to promote biogenetic causal beliefs in the United States in recent decades (Lebowitz & Appelbaum, 2019). Although research suggests that biogenetic beliefs are linked to prognostic pessimism in many populations (Dinos et al., 2017; Lebowitz, 2014), we did not find this to be the case in our sample of high-symptom adolescents.

For clinicians working with depressed adolescents, this research provides the following insights: first, when assessing clients' causal beliefs, expect complexity. Clients are likely to hold multiple (or multidimensional) causal beliefs, and may hold different causal beliefs for their own depression than they hold regarding depression in general. At the same time, clinicians may reasonably expect school-related stressors, problems with family, and problems with friends to figure prominently based on these results. Lastly, clinicians should be aware that biological causal beliefs may predispose clients toward favoring medication as an appropriate treatment for their depression.

Lastly, this study suggests many future directions for research in this area. First, while theory suggests that causal beliefs play a role in the management of depression, further empirical research is needed to identify the extent to which this is accurate, and the mechanisms by which this happens. Future research should also explore the role of other components of the common-sense model of illness management for depression, such as what defines depression and beliefs about its prognosis (Leventhal et al., 2016). With regards to adolescent causal beliefs about depression, studies may seek to explore causal beliefs among non-depressed adolescents and/or compare these beliefs to those of depressed adolescents. Future research may also explore the clinical implications of this work; while existing research demonstrates the utility of exploring clients' causal beliefs for the sake of the therapeutic alliance (James et al., 2014) and aligning intervention design to the causal beliefs of its recipients (Hinton et al., 2005), little work has been done on the potential of causal beliefs as a treatment target. Lastly, future research may take adolescents' causal beliefs about depression at face value and explore the role of under-studied causes like school stress in the development of depression.

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## Supplemental Material

Supplemental material for this article is available online.

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