

CORRELATES AND PREDICTORS OF CHRONICITY AMONG ADOLESCENTS LIVING IN PUERTO RICO WITH A HISTORY OF DEPRESSIVE SYMPTOMS

CORRELATOS Y PREDICTORES DE CRONICIDAD EN ADOLESCENTES RESIDENTES EN PUERTO RICO CON HISTORIAL DE SÍNTOMAS DEPRESIVOS

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ABSTRACT

Chronic depression (CD) among Hispanic/Latina(o) youths has been understudied, although chronicity is the biggest risk factor for treatment-resistant depression. We examined CD correlates and predictors among 291 youths (aged 12–18 years) living in Puerto Rico with a history of depressive symptoms. They completed the Children's Depression Inventory (CDI), the Depressive Symptoms Spectrum Assessment Inventory (DSSAI), and the Brief Structured Diagnostic Measure for Depression. We explored CD correlates using odds ratios adjusted for CDI-Total scores. With multiple logistic regression, we identified optimal predictors of a history of chronic depressive symptoms (HCDS) or any chronic depressive disorder (HACDD). Living zone (rural), history of depressive disorder, household size (< 4), age of onset of symptoms (< 13 years), death/suicidal thoughts at the first episode, antidepressants use, and scores $\geq$ 84th percentile in the DSSAI-Anhedonia subscale, accounted for 37% of HCDS variance. The latter five variables and socioeconomic status (lower middle/low) best distinguished HACDD and episodic disorders ($R^2 = .331$). Identifying factors that distinguish chronic and episodic depression among Hispanic/Latina(o) youths may help to improve their diagnosis, access to and quality of care, as well as treatment selection, tailoring, and outcomes.

KEYWORDS: Adolescents, anhedonia, chronic depression, Hispanics/Latinas(os).

RESUMEN

La depresión crónica (DC) juvenil se ha estudiado poco entre hispanas(os)/latinas(os), aun siendo factor de riesgo principal para la resistencia al tratamiento. Examinamos los correlatos y predictores de DC entre 291 jóvenes (entre 12–18 años) residentes en Puerto Rico con historial de síntomas depresivos. Estas(os) completaron el *Children's Depression Inventory* (CDI), el Inventario de Evaluación del Espectro de la Sintomatología Depresiva (INEESD) y la Evaluación Diagnóstica Estructurada Breve para la Depresión. Exploramos los correlatos de DC utilizando *odds ratios* ajustados por el CDI-Total. Utilizando regresión logística múltiple, identificamos predictores óptimos del historial de síntomas depresivos crónicos (HSDC) o cualquier trastorno depresivo crónico (HCTDC). Variables como zona de vivienda (rural), historial de trastornos depresivos, tamaño del hogar (< 4), edad de inicio de síntomas (< 13 años), pensamientos suicidas/mórbidos en el primer episodio, usar antidepressivos y puntuaciones $\geq$ al percentil 84 del INEESD-Subescala de Anhedonia explicaron 37% de la varianza del HSDC. Las últimas cinco variables y el nivel socioeconómico (medio-bajo/bajo) distinguieron mejor entre HCTDC y trastornos episódicos ($R^2 = .331$). Identificar factores discriminadores de DC vs. episódica entre jóvenes hispanas(os)/latinas(os) puede ayudar a mejorar su diagnóstico, acceso y calidad de servicios, así como la selección, adaptación y rendimiento del tratamiento.

PALABRAS CLAVE: Adolescentes, anhedonia, depresión crónica, origen hispano o latino.

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Depression is a disabling disorder that affects all age groups. Even if some people have acute episodes, many experience chronic depression (CD), defined by symptoms lasting ≥ 2 years in adults and ≥ 1 year in minors (American Psychiatric Association [APA], 2013). Some people with a history of chronic depressive symptoms have never met criteria for a depressive disorder (e.g., they lack the impairment criterion). However, most CD cases were diagnosed as chronic major depressive disorder (MDD), dysthymia, or combined MDD and dysthymia until 2013. Other CD cases showed impairing subthreshold depressive episodes of chronic duration (chronic minor depression). In 2013, the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; APA, 2013) defined persistent depressive disorder (PDD) as the category for CD. PDD may occur with or without a major depressive episode (MDE). In adults, the combined rate of all forms of CD previously mentioned is about 6% (Garland & Scot, 2008). Point prevalence of dysthymia among children in epidemiologic studies ranges from 0.5 to 5.5% (Waslick et al., 2003). Data from a United States (US) national survey showed a 4.8% rate for persistent depression in youth aged 12–17 years (Breaslau et al., 2017).

Essau and Chang (2009) reviewed studies on the course of pediatric depression and found that about 21%–41% of youths were still depressed a year after, and 8%–10% were depressed after 2 years. Transition to adolescence is a critical period for the development of CD (Morken et al., 2021). Recently, Chi et al. (2020) reported a 46.4% rate of persistent symptoms among 7th grade students with depression. About 45% of participants in the Treatment of Adolescent with Depression Study had CD (Lorenzo-Luaces et al., 2020). Compared to acute episodic cases, CD leads to higher costs, burden, and suffering (Köhler et al., 2019; Waslick et al., 2003), as well as reduced recovery chances, lower spontaneous remission, and higher relapse rates (Essau & Chang, 2009; Klein & Black, 2017). The

various forms of CD are similar in clinical features, demographics, social functioning, coping style, comorbidity, negative cognitions, early adversity, and treatment response, but differ from episodic depression (Klein & Black, 2017).

Researchers propose three psychosocial models to explain CD (Lara & Klein, 1999). From a cognitive perspective, there is a reciprocal relationship between depressed moods, the way people process their symptoms and life events, as well as how they respond to them. In this view, maladaptive schema, response styles (e.g., rumination), hopelessness and helplessness, self-criticism, and suicide ideation play an important role in the persistence of depression (Riso & Newman, 2003). From an interpersonal view, depression produces negative social reactions, which reduce social support, reinforce depressive behaviors, and perpetuate maladaptive relational and communication styles (Pettit & Joiner, 2006). A third model affirms that CD arises from a poor early home environment (e.g., maltreatment, abuse, neglect, or poor parenting) and chronic stress related to it (Brown et al., 1994). An approach that assumes the interaction of cognitive, interpersonal, and early adversity factors may provide a more comprehensive explanation of the origin, maintenance, and persistence of CD (Lara & Klein, 1999).

Research has shown that increased guilt, somatic symptoms, anhedonia, symptom severity, suicidality, recurrence, comorbid physical/mental disorders, negative beliefs, greater functional impairment, childhood adversity, parental depression, interpersonal/social problems, low involvement with fathers, lack of positive youth development, worse family functioning, earlier age of onset, bully victimization, greater use of treatment services, and longer episodes before treatment, all are related to CD in youth (Chi et al., 2020; Essau & Chang, 2009; Hill et al., 2017; Klein & Black, 2017; Sanford et al., 1995; Vance & Winther, 2020). Results about sex differences in episode duration or rates of

CD are inconsistent (Essau & Chang, 2009). Shain et al. (1991) found a higher rate of antidepressant use in CD cases. In most studies, episodic depression and CD do not differ in sex, socioeconomic status (SES), and age, or their results have been inconclusive (e.g., Chi et al., 2020; Essau, 2007; Salk et al., 2016; Vance & Winther, 2020). However, two studies have found a lower parental education in the chronically depressed group (Chi et al., 2020; Wickrama et al., 2009).

The course of CD in youth relates to many complications. These youths suffer long-term consequences on social skills and psychosocial functioning, but also a higher risk for relapse and for adult substance use, suicidality, anxiety, CD, other mental illnesses, marginalization from the labor market, longer use of antidepressants, and criminal offenses (Alaie et al., 2021; Fombonne et al., 2001; Jonsson et al., 2011; Schubert et al., 2017; Weavers et al., 2021). Kovacs et al. (1994) followed outpatients aged 8–13 years old whose index diagnosis was dysthymia (with or without a superimposed MDE at study entry), and a comparison group with MDD only, during a 3- to 12-year period. Since their index diagnosis, 76% of youth with dysthymia developed an MDE. These participants met criteria for a mood disorder during 52% of the follow-up period, which was higher than for youth with MDD. In another study, only 7% of youth with dysthymia showed evidence of recovery 2 years after the onset of a first episode (Kovacs et al., 1997). Despite similar admission and discharge depression scores, inpatient adolescents with CD have a slower initial rate of improvement (Shain et al., 1991). In Brent's (2018) words, "the biggest single risk factor for treatment-resistant depression is chronicity" (p. 5).

Some studies have reported higher rates of CD among adolescents from ethnic minorities, including Hispanics/Latinas(os). In a representative U.S. sample, Alaimo et al. (2002) found a lifetime rate for dysthymia of 4.9% for non-Hispanic Whites aged 15–16 years, but of 7.2% for Mexican-Americans. In

a longitudinal study, minority ethnicity increased the likelihood of following high-depression trajectories, compared to White Americans (Costello et al., 2008). Wickrama et al. (2009) found a higher proportion of Hispanic/Latina(o) adolescents in the trajectory group with chronically high depressive symptoms, compared with three other trajectories. Differences in help-seeking behavior, reduced access and quality of care, and low use of antidepressants, may contribute to CD and suffering among Hispanics/Latinas(os) (Riolo et al., 2005).

In a study conducted in Puerto Rico (PR) with youths aged 4–17 years, Canino et al. (2004) reported a 0.5% past-year rate for dysthymia. However, no research study has reported the combined rate of all forms of CD (including chronic MDD and chronic minor depression) among youth living in the island. In addition, no published study has examined the correlates or predictors of CD among Hispanic/Latina(o) youth from PR or the US. Given that an earlier age of onset and longer episodes indicate a poorer prognosis, identifying CD in youth could help to contain or prevent complications, as it may help to provide early and improved treatment (Klein & Black, 2017). Identifying predictors of CD could ease their inclusion on evaluation protocols for depressed youth, help to better define patients' prognosis, and inform the selection of techniques to target these predictors in treatments. The latter may help in reducing chronicity, its complications (e.g., medical, functional, and relational), and its related health care costs.

In this study, we aimed to examine the correlates of CD in a sample of Hispanic/Latina(o) youth living in PR who presented a history of depressive symptoms and to identify multiple logistic regression models that best explain the occurrence of CD in this sample, after adjusting for the severity of current depressive symptoms. We expected that the severity of past depressive symptoms, a history of suicidal ideation/behavior, an earlier age of onset, and the pre-

sence of multiple impairment areas would be among the strongest predictors of CD in these youths.

METHOD

Participants

Participants were 291 youths (68.04% girls) aged 12–18 years ($M = 15.37$; $SD = 1.59$). They were enrolled in school grades 7–12. We conducted secondary analyses on cases reporting a history of at least one lifetime episode of unusual depressed mood and/or anhedonia, within a larger convenience sample of 621 youths from a major study (the Depressive Symptoms Spectrum Assessment Inventory [DSSAI] project). We recruited them from San Juan public schools or private schools in the San Juan Metropolitan Area of Puerto Rico (SJMA-PR), which also includes Bayamón, Guaynabo, Trujillo Alto, and Carolina. Youths must read and write in Spanish and not show any neurological, sensory, cognitive, or physical problems that could prevent participation. Recruitment took place from November 2010 to December 2012.

Primary caregivers only completed the Socio-Demographic Data Form (SDDF). Most children (77.32%) lived in urban zones and 88.32% lived in the SJMA-PR. Most primary caregivers were women and perceived that their family had a upper-middle (34.02%) or lower-middle (56.70%) SES, while 9.28% reported a low SES. About 59.11% of primary caregivers worked full-time and 13.40% part-time. Their mean schooling was 14.86 years ($SD = 2.87$) with a mean age of 43.04 years ($SD = 7.22$). The mean household size was 3.95 ($SD = 1.26$). In 69.76% of the homes, adolescents lived with at least two adult relatives (aged ≥ 21 years).

Measures

Children's Depression Inventory (CDI)

This is a 27-item self-report inventory for youth 7–17 years of age that measures the

presence of depressive symptoms in the past 2 weeks. Items have three statements of different severity levels for each indicator (e.g., sadness, guilt, tiredness, etc.). Options are scored as 0 (absent), 1 (mild), or 2 (severe). We used the published Spanish CDI (Kovacs, 2001). Its internal reliability in this sample was .87. Scores ≥ 13 suggest a need for a diagnostic assessment for depression. The sample mean CDI score was 11.01 ($SD = 7.39$). The median, skewness, and kurtosis were 9.00, 1.35, and 2.86, respectively.

Depressive Symptoms Spectrum Assessment Inventory (DSSAI)

The DSSAI (INEESD by its Spanish acronym) is a self-report measure of depressive symptoms in youth aged ≥ 12 years. It provides total scores for the last 2 weeks (L2W) and the last 6 months (L6M). It contains 120 items in Likert-type format with options from 0 (*Never or rarely*) to 3 (*Very frequently*). DSSAI items classify into 10 clinical themes (number of items in parenthesis): Activity Alterations (8), Cognitive Alterations (10), Hopelessness/Pessimism (8), Helplessness (9), Anhedonia (9), Undervaluing/Self-reproach (12), Mood Disturbances (13), Interpersonal Alterations (20), Suicidality/Self-destructiveness (14), and Somatic Alterations (17). Scores at or above the 84th percentile for these subscales correspond to values at or above 9, 14, 8, 8, 9, 12, 15, 20, 6, and 18, respectively. An initial report of the DSSAI psychometric properties is available in Feliciano-López and Cumba-Avilés (2014). The internal consistency of both DSSAI total scores was .98. Alpha values for its subscales ranged from .78 to .90 (L2W) and from .75 to .89 (L6M) in that report. Values for the final sample ($N = 621$) ranged from .78 to .91 (L2W) and from .77 to .91 (L6M). Test-retest values were above .85 for both total scales and above .70 for all subscales (Cumba-Avilés & Feliciano-López, 2015). For this study, we also used a Rumination Composite score comprised of four DSSAI items. The internal and temporal reliability of this composite score was .75 for both the L6M and the L2W.

Brief Structured Diagnostic Measure for Depression (BSDMD)

We developed the BSDMD for its use in the DSSAI project. It assesses the presence of MDD, dysthymia, or depressive disorder not otherwise specified (DDNOS) diagnostic criteria ever in a lifetime, including the first and latest occurrence of symptoms, according to the DSM. Besides symptoms of disorders, the BSDMD assesses impairment and exclusion criteria, frequency and duration of episodes, depression's age of onset, and lifetime history of depression treatment and suicide attempts. Its questions allow the distinction between primary depressive disorders and adjustment disorder with depressed mood (ADDM). Youths answered the BSDMD in self-report format. Using its data, two clinical psychologists derived decisions for each diagnostic category. During its validation process (which occurred as part of the DSSAI project), we computed diagnostic agreement corrected by chance (Cohen's κ) between two clinicians (raw agreement percent in parenthesis). About lifetime disorders, κ was .976 (99.36) for MDD, .854 (99.36) for dysthymia, .915 (98.71) for DDNOS (i.e., minor depression), .996 (99.84) for any depressive disorder (either MDD, dysthymia, or DDNOS), and .898 (99.93) for ADDM (all at $p \leq .001$). The average κ across categories was .92. Lower bounds in the 95% confidence interval were all $\geq .70$. Diagnoses based on the BSDMD were significantly related to external criteria.

Suicide Risk Interview for Adolescents (FERSA by its Spanish acronym)

We used this interview to assess the lethality of suicidal ideation/behavior. The development of this interview and details regarding the protocol for suicide risk assessment used in the main study are described elsewhere (Cumba-Aviles & Feliciano-López, 2013).

Procedure

After the Institutional Review Board approval (#0910-111), we met with school staff to explain study procedures and obtain their

cooperation. At informative meetings with students, we explained study procedures and gave them an envelope with the SDDF, consent/assent Forms, and information sheets. We asked youths to deliver the envelope to their guardians to authorize their participation and complete the SDDF. Teens signed the forms if assented to participate and handed over documents in the envelope to school staff, who called research staff to pick them up at school. We coordinated informative meetings and assessment dates with school staff. The assessment session was in self-report format and lasted about 60 minutes. As in our pilot study (Cumba-Avilés & Feliciano-López, 2013), we conducted risk assessments with youths who reported suicidal ideation. In these cases, we contacted parents by phone to inform them about adolescents' suicidal thoughts, assessed their commitment to support youths, and provided them with referrals for youths' treatment. When adolescents presented with depressive symptoms only, we recommended them to seek help from a mental health professional and provided them with contact information from several mental healthcare providers. Then, we informed their caregivers about the presence of depressive symptoms in their children, and provided them with referrals, as needed. We also informed authorized school personnel (usually social workers or school counselors) about the status of cases as soon as possible to ease their monitoring of youths' emotions or behaviors during school hours.

Data Analyses

We analyzed data with SPSS 24.0. First, we divided the sample into two groups: cases with a history of chronic (≥ 1 year) depressive symptoms (HCDS; $n = 83$) and those with non-chronic symptoms (No HCDS; $n = 208$). The HCDS group included cases who reported a history of at least one episode of unusual chronic depressed mood and/or anhedonia, with or without other symptoms, irrespectively of diagnostic status. Impairment criterion was not required to be in this group. Its members could have met (or not) criteria for any lifetime depressive disorder or ADDM. We used

adjusted odds ratios (AOR) to identify individual variables significantly related to HCDS ($p \leq .05$), after adjusting for CDI scores. Potential correlates included demographics and variables related to youth mental health history, health status, characteristics of past depressive episodes, functional impairments, and specific symptom areas within 6 months of enrollment. Then, using data on diagnostic decisions, we identified cases with a history of any chronic depressive disorder (HACDD; $n = 65$) or without such history (No HACDD; $n = 226$). Members of the HACDD group must have met full criteria for a lifetime history of at least one of these disorders: chronic MDD, dysthymia, or chronic DDNOS. We classified all other cases into the No HACDD for this comparison. We used the procedures described before to identify correlates of HACDD vs. No HACDD. We also used AORs to detect which potential correlates distinguished cases in the HACDD group against two subgroups from the No HACDD cases: those with a history of non-chronic depressive disorders (HNCDD; $n = 95$) and cases with no history of primary depressive disorders (NHDD; $n = 131$). The latter subgroup included cases with depressive symptoms but no disorder and those who only had a history of ADDM.

After identifying correlates of an HCDS, we used multiple logistic regression to build a model that best explained membership in the HCDS vs. No HCDS group, adjusting for CDI scores and the other variables in the model. We focused our next logistic regression analyses on identifying optimal predictors of membership in the HACDD vs. the HNCDD group, on the one hand, and of membership in the HACDD vs. the NHDD group, on the other hand. We selected candidate variables for each logistic regression from the pool of significant correlates (Table 1) found in their respective AOR analysis with individual variables. Regarding demographics, we considered for regressions those with significant or marginally significant ($p < .10$) AORs in at least one of the analyses conducted to identify correlates. We estimated a 95% confidence interval (CI) for all analyses and used the backward method (exit criterion of $p = .10$) to

facilitate the identification of the best combination of predictors of group membership. Using analysis of covariance, we compared the mean number of impaired areas by group adjusting for CDI scores. We adjusted for CDI scores because current symptoms may induce a recall bias on retrospective reports about depressive symptoms or episodes, their impairment, and even their duration (which defines chronicity) among people with a history of depression (Patten, 2003; Urban et al., 2018).

The rationale for dividing our sample into the selected groups were as followed. First, no published study has examined if there are differences in the correlates and predictors of CD considering the distinction between these two ways (chronic symptoms vs. chronic disorders) to operationalize the concept. Second, only analyses per diagnostic category allow distinctions between cases with CD and more specific subgroups that are mutually exclusive (such as the so-called HNCDD and NHDD). Third, in clinical practice, clinicians receive cases that are known to have presented chronic depressive symptoms, but it is unknown whether at some point in their lives they have met the criteria for a primary depressive disorder. Finally, analyses by diagnostic subgroups may contribute in a better way to distinguish between episodic and chronic disorders.

RESULTS

Descriptive Analyses and Diagnostic Agreement

Eighty-three (28.52%) cases had symptoms lasting ≥ 1 year. About 13.40% (39) reported symptoms lasting ≥ 2 years. Sixty-five (22.34%) youths met criteria for an HACDD lasting ≥ 1 year. Besides, 9.97% (29) met criteria for a disorder that lasted ≥ 2 years. CD diagnoses were chronic MDD only (39), dysthymia only (11), chronic DDNOS only (12), chronic DDNOS followed by chronic MDD (1), chronic MDD followed by dysthymia (1), and initial dysthymia followed by chronic MDD (1). Other 95 youth had HNCDD (i. e., non-chronic MDD or non-chronic DDNOS), 25

had a history of ADDM, and 106 had none of these disorders. Raw inter-rater agreement about the presence of a CD disorder was 100%. Raw agreement when classifying cases into one of these specific categories (dysthymia only, chronic MDD only, chronic DDNOS only, history of two CD disorders, HNCDD only, history of ADDM only, and no disorder), was 95.88%, with a Cohen's κ of .944 [.912 – .975], $p \leq .001$.

Adjusted Relationship Among Individual Predictors and Membership in CD Groups

Socio-Demographic Variables

In Table 1, we showed AORs for several variables, reflecting their relationship with the HCDS or HACDD groups after adjusting for CDI scores. Youths in one-caregiver homes had 82% and 90% more odds of presenting HCDS and HACDD than those who lived with two or more potential caregivers, respectively. When comparing the HACDD group against HNCDD cases, the AOR increased to 2.61,

but this value was non-significant against cases with NHDD. Perceived SES (low middle or low) and residential zone (rural) showed significant AORs for HACDD vs. the No HACDD group and the latter obtained a marginally significant AOR value ($p \leq .10$) for the HCDS. When splitting the No HACDD group for a more specific examination, the AOR value for perceived SES was only significant when comparing HACDD vs. HNCDD cases and the AOR value for the residential zone was only significant for the HACDD vs. NHDD comparison. Youths in smaller households (less than four members) showed a twofold increase in their odds for being in the HCDS group ($p \leq .05$) but only a marginal increase in their odds for HACDD compared to HNCDD. Youths' sex, current age, and type of school attended, as well as primary caregivers' education, age, or employment status, were unrelated with membership in any of these groups (not shown in Table 1).

TABLE 1.

Adjusted Odds Ratios (with 95% CI) for a History of Chronic Depressive Symptoms or a Chronic Depressive Disorder.

Variable	HCDS ($n = 83$) vs. No HCDS ($n = 208$)	HACDD ($n = 65$) vs. No HACDD ($n = 226$)	HACDD ($n = 65$) vs. HNCDD ($n = 95$)	HACDD ($n = 65$) vs. NHDD ($n = 131$)
Residential zone (Rural)	1.78 [†] [0.96 – 3.21]	1.98 [†] [1.03 – 3.80]	1.46 [0.71 – 2.96]	2.96 ^{**} [1.33 – 6.59]
Socioeconomic status (LM/L)	1.39 [0.78 – 2.49]	2.05 [*] [1.03 – 4.10]	2.42 [*] [1.14 – 5.13]	2.11 [†] [0.94 – 4.70]
One-caregiver home	1.82 [*] [1.04 – 3.18]	1.90 [*] [1.03 – 3.51]	2.61 ^{**} [1.28 – 5.32]	1.55 [0.76 – 3.15]
Household size (< 4)	2.07 [*] [1.20 – 3.55]	1.57 [0.87 – 2.85]	1.75 [†] [0.90 – 3.41]	1.43 [0.72 – 2.87]
Hx of any depressive disorder	3.26 ^{***} [1.75 – 6.08]			
Hx of suicide attempt	2.70 ^{**} [1.32 – 5.51]	2.27 [*] [1.08 – 4.79]	2.37 [*] [1.03 – 5.49]	2.38 [†] [0.95 – 5.96]
Hx of depression psychotherapy	1.51 [0.81 – 2.81]	1.90 [†] [0.98 – 3.68]	1.54 [0.75 – 3.15]	2.35 [*] [1.03 – 5.37]
Hx of depression pharmacotherapy	2.87 [*] [1.05 – 7.82]	3.19 [*] [1.14 – 8.89]	4.85 [*] [1.24 – 19.08]	1.94 [0.57 – 6.60]
Death/suicidal ideation (FE)	3.24 ^{***} [1.68 – 6.25]	3.56 ^{***} [1.80 – 7.05]	2.88 ^{**} [1.36 – 6.10]	5.26 ^{***} [2.16 – 12.80]
Age of onset of DEP (< 13 years)	2.81 ^{***} [1.63 – 4.85]	2.31 ^{**} [1.27 – 4.22]	3.32 ^{***} [1.69 – 6.55]	1.75 [0.87 – 3.49]
DSM symptoms in FE (≥ 4)	1.97 [*] [1.10 – 3.53]	2.40 [*] [1.22 – 4.71]	0.99 [0.46 – 2.11]	4.82 ^{***} [2.29 – 10.11]
Presence of at least one EP 5+	1.62 [0.90 – 2.91]	2.53 ^{**} [1.28 – 5.00]	0.88 [0.41 – 1.90]	5.74 ^{***} [2.67 – 12.33]
Recurrence of EP 5+	1.42 [0.77 – 2.70]	1.44 [0.75 – 2.77]	0.97 [0.48 – 1.94]	2.37 [*] [1.07 – 5.24]
Two or more impairment areas	1.07 [0.62 – 1.86]	1.92 [*] [1.04 – 3.55]	0.64 [0.32 – 1.29]	5.41 ^{***} [2.59 – 11.27]
Impairment at home	1.33 [0.76 – 2.35]	1.89 [*] [1.02 – 3.49]	1.01 [0.52 – 1.97]	3.73 ^{***} [1.77 – 7.84]
Emotional impairment	1.20 [0.70 – 2.05]	2.17 [*] [1.20 – 3.94]	1.05 [0.55 – 2.02]	4.31 ^{***} [2.09 – 8.91]
Rumination Composite $\geq$ P84	1.34 [0.74 – 2.42]	2.26 [*] [1.20 – 4.27]	1.66 [0.84 – 3.27]	2.78 ^{**} [1.31 – 5.93]
DSSAI-UVSR L6M $\geq$ P84	3.11 ^{***} [1.65 – 5.86]	3.29 ^{***} [1.68 – 6.44]	2.86 ^{**} [1.38 – 5.93]	3.54 ^{**} [1.59 – 7.90]
DSSAI-INTA L6M $\geq$ P84	2.06 [*] [1.07 – 3.95]	2.18 [*] [1.09 – 4.37]	1.82 [0.87 – 3.82]	2.34 [†] [0.98 – 5.58]
DSSAI-MDIS L6M $\geq$ P84	1.42 [0.74 – 2.74]	1.97 [†] [0.98 – 3.96]	1.40 [0.67 – 2.90]	2.88 [*] [1.23 – 6.76]
DSSAI-COGA L6M $\geq$ P84	1.85 [†] [0.98 – 3.56]	2.18 [*] [1.10 – 4.30]	2.07 [†] [1.00 – 4.30]	2.09 [†] [0.92 – 4.78]
DSSAI-HOPL L6M $\geq$ P84	2.24 [*] [1.16 – 4.34]	2.16 [*] [1.06 – 4.36]	1.72 [0.81 – 3.65]	2.37 [†] [0.97 – 5.78]
DSSAI-HELP L6M $\geq$ P84	2.65 ^{**} [1.34 – 5.26]	3.02 ^{**} [1.45 – 6.26]	2.11 [†] [0.98 – 4.54]	4.31 ^{**} [1.74 – 10.68]
DSSAI-ANHE L6M $\geq$ P84	3.82 ^{***} [2.00 – 7.29]	3.30 ^{***} [1.66 – 6.54]	2.59 [*] [1.24 – 5.41]	3.93 ^{***} [1.70 – 9.07]
DSSAI-SUIC L6M $\geq$ P84	3.37 ^{***} [1.78 – 6.38]	3.52 ^{***} [1.79 – 6.95]	2.87 ^{**} [1.37 – 6.01]	4.37 ^{***} [1.95 – 9.81]

Variable	HCDS (<i>n</i> = 83) vs. No HCDS (<i>n</i> = 208)	HACDD (<i>n</i> = 65) vs. No HACDD (<i>n</i> = 226)	HACDD (<i>n</i> = 65) vs. HNCDD (<i>n</i> = 95)	HACDD (<i>n</i> = 65) vs. NHDD (<i>n</i> = 131)
DSSAI-ACTA L6M $\geq$ P84	1.26 [0.67 – 2.39]	1.47 [0.74 – 2.90]	1.21 [0.59 – 2.45]	1.36 [0.55 – 3.33]
DSSAI-SOMA L6M $\geq$ P84	2.70*** [1.49 – 4.92]	2.96*** [1.56 – 5.60]	2.03* [1.01 – 4.05]	4.08*** [1.81 – 9.17]

Note. Odds ratios were adjusted for scores in the Children's Depression Inventory. CI = Confidence interval; HCDS = History of chronic depressive symptoms; HACDD = History of any chronic depressive disorder; HNCDD = History of a non-chronic depressive disorder; NHDD = No history of a primary depressive disorder (includes cases with adjustment disorder with depressed mood and those with no disorder); LM/L = Lower middle or low; Hx = History; FE = First Episode; DEP = Depression; DSM = Diagnostic and Statistical Manual; EP 5+ = Episode of 5 or more depressive symptoms; P84 = 84th percentile; DSSAI = Depressive Symptoms Spectrum Assessment Inventory; UVSR = Undervaluing/Self-reproach; L6M = Last 6 months; INTA = Interpersonal Alterations; MDIS = Mood Disturbances; COGA = Cognitive Alterations; HOPL = Hopelessness/Pessimism; HELP = Helplessness; ANHE = Anhedonia; SUIC = Suicidality/Self-destructiveness; ACTA = Activity Alterations; SOMA = Somatic Alterations.

† $p \leq .10$; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$.

Youth Mental Health History and Physical Health Status

Mental health indicators significantly related to membership in CD groups included a history of antidepressants use and suicide attempt. Their AOR values ranged from 2.87 to 4.85 ($p \leq .05$) in the first case and from 2.27 ($p \leq .05$) to 2.70 ($p \leq .01$) in the second, depending on the specific comparison. These variables did not discriminate between HACDD and NHDD cases. Having met criteria for a depressive disorder related significantly with HCDS ($p \leq .001$). Yet, we found no relationship between CD groups and adolescents' last-year physical health status or history of substances use, history of any non-depressive mental disorder, history of comorbid physical/mental disorders, or attributing depression to stressful life events (not shown).

Characteristics of Past Depressive Episodes

The odds for HCDS or HACDD were significantly higher for youths whose age of onset of depression was < 13 years, and for those who had ≥ 4 MDD symptoms in their first episode. The latter variable, however, did not distinguish HACDD from HNCDD cases. Experiencing one or more lifetime episodes of ≥ 5 MDD symptoms (MDE) related with belonging to the HACDD group compared with No HACDD cases (AOR = 2.53), but particularly if compared to the NHDD subgroup (AOR = 5.74). Recurrence of this type of episode only distinguished between HACDD and NHDD cases (AOR = 2.37). Yet, the feature of past episodes with the highest odds for CD in youths across comparisons was having death/suicidal thoughts in the first episode.

History of Functional Impairments

Youth informed about depression-related impairment in several areas: school/work, home, social life, emotions, sports, or any other area. Impairment in ≥ 2 areas related with a 92% increase in the odds for HACDD vs No HACDD, and with an AOR of 5.26 when comparing HACDD and NHDD groups. Impaired emotions and impairment at home related with increased odds for HACDD, but not HCDS. This link was considerably higher when comparing HACDD with NHDD ($p \leq .001$), but not significant if compared with HNCDD cases. After adjusting for CDI scores, the mean number of impaired areas was higher among HACDD [$M = 2.46$, $SD = 1.30$] than among No HACDD cases [$M = 1.50$, $SD = 1.46$, $F(1, 288) = 9.34$, $p = .003$, $\eta_p^2 = 0.03$]. We found a 36% increase in the odds for HACDD vs No HACDD for each impaired area ($p = .003$; not shown). Yet, if examining specific subgroups, only the HACDD vs. NHDD comparison [$M = 0.82$, $SD = 1.23$] was significant [$F(1, 193) = 35.53$, $p \leq .001$, $\eta_p^2 = 0.16$].

Specific Symptom Areas During the Past 6 Months

When examining AOR values for HCDS in youth with scores $\geq 84^{\text{th}}$ percentile in the DSSAI subscales, we observed significant values ranging from 2.06 (Interpersonal Alterations) to 3.82 (Anhedonia). We found non-significant values for three subscales. When comparing HACDD vs. No HACDD, these values ranged from 2.16 (Hopelessness) to 3.52 (Suicidality), with non-significant findings for two subscales. Adolescents with scores $\geq 84^{\text{th}}$ percentile in

the Rumination Composite also showed higher odds for HACDD but not for HCDS cases. The latter association was significant only if comparing HACDD with the NHDD subgroup. When comparing HACDD vs HNCDD cases, only four DSSAI subscales show significant AORs. The latter was true for six subscales when comparing HACDD vs. NHDD cases.

Independent Predictors of a History of Chronic Depressive Symptoms (HCDS)

As described previously, we used multiple logistic regression to identify the optimal predictors of membership in the HCDS group (as opposed to No HCDS), adjusting for CDI scores. An optimal model would account for the highest variance amount with the lower number of predictors from candidates identified as correlates in the AOR analyses conducted for individual variables.

TABLE 2.
Multiple Logistic Regression Models for HCDS and HACDD Adjusted for CDI Scores.

Variable	B	SE	Adjusted OR (95% CI)	R ²
HCDS vs No HCDS (N = 291)				
Residential zone (Rural)	0.63†	0.36	1.88† [0.92 – 3.83]	.106
Household size (< 4)	1.06***	0.33	2.89*** [1.53 – 5.47]	.144
DEP onset (Before 13 years old)	1.14***	0.32	3.14*** [1.68 – 5.85]	.199
Death/suicidal ideation (FE)	1.00**	0.37	2.71** [1.30 – 5.64]	.251
Hx of any depressive disorder	1.12**	0.36	3.05** [1.50 – 6.22]	.301
Hx of DEP pharmacotherapy	1.37*	0.59	3.94* [1.23 – 12.60]	.317
DSSAI-ANHE L6M ≥ P84	1.37***	0.37	3.93*** [1.90 – 8.14]	.370
HACDD vs. HNCDD (N = 160)				
Perceived SES (LM/L)	0.85*	0.36	2.34* [1.01 – 5.48]	.093
Household size (< 4)	0.93*	0.40	2.54* [1.17 – 5.54]	.124
DEP onset (Before 13 years old)	1.17**	0.38	3.22** [1.52 – 6.82]	.208
Death/suicidal ideation (FE)	0.92*	0.42	2.51* [1.10 – 5.75]	.246
Hx of DEP pharmacotherapy	1.78*	0.78	5.96* [1.29 – 29.54]	.282
DSSAI-ANHE L6M ≥ P84	1.15**	0.42	3.15** [1.37 – 7.22]	.331
HACDD vs. NHDD (N = 196)				
Perceived SES (LM/L)	0.86†	0.51	2.35† [0.87 – 6.41]	.316
Household size (< 4)	1.62***	0.49	5.07*** [1.94 – 13.22]	.355
Presence of at least one EP 5+	1.65***	0.49	5.21*** [1.99 – 13.62]	.463
Death/suicidal ideation (FE)	1.15*	0.55	3.17* [1.07 – 9.40]	.509
Impairment at home	1.10*	0.49	3.00* [1.14 – 7.87]	.547
Emotional impairment	1.11*	0.48	3.02* [1.17 – 7.77]	.568
DSSAI-HELP L6M ≥ P84	1.74**	0.59	5.69** [1.78 – 18.21]	.605

Note. R² values are Nagelkerke estimate of variance accounted by step after adjusting for CDI scores and variables in previous steps. HCDS = History of chronic depressive symptoms; CDI = Children's Depression Inventory; CI = Confidence interval; HACDD = History of any chronic depressive disorder; HNCDD = History of non-chronic depressive disorder; NHDD = No history of a primary depressive disorder; FE = First Episode; SES = Socioeconomic status; LM/L = Lower middle or low; Hx = History; DEP = Depression; L6M = Last 6 months; DSSAI = Depressive Symptoms Spectrum Assessment Inventory; ANHE = Anhedonia subscale; P84 = 84th percentile; EP 5+ = Episode of 5 or more depressive symptoms; HELP = Helplessness subscale. †p ≤ .10; *p ≤ .05; **p ≤ .01; ***p ≤ .001

We presented results of incremental variance accrued by step, but regression coefficients, standard errors, and AORs belong to the final step. Residential zone (rural), household size (< 4 members), age of onset of symptoms, death/suicidal thoughts in the first episode, history of any depressive disorder, history of antidepressant use, and DSSAI-Anhedonia scores (≥ 84th percentile)

in the L6M integrated the optimal model. The latter explained 37% of variance for HCDS, LR = 86.82, $p \leq .001$. No other variable added significant variance. The highest AORs for HCDS belonged to a history of antidepressant use, Anhedonia scores, and age of onset.

Independent Predictors of a History of Any Chronic Depressive Disorder (HACDD)

We also used multiple logistic regression to identify which variables best predicted membership in the HACDD group, adjusting for CDI scores. One model compared the HACDD against the HNCDD group. Perceived SES, household size, age of onset, death/suicidal thoughts in the first episode, a history of antidepressant use, and Anhedonia scores ($\geq 84^{\text{th}}$ percentile) integrated the optimal model, explaining 33.1% of the variance for HACDD, $LR = 45.01$, $p \leq .001$. The highest AORs belonged to the same predictors mentioned in the previous section.

The next model examined predictors of HACDD against NHDD. SES, household size, history of at least one MDE, death/suicidal thoughts in the first episode, being impaired at home, showing emotional impairment, and DSSAI-Helplessness scores ($\geq 84^{\text{th}}$ percentile) in the L6M formed the optimal model, accounting for 60.5% of the variance, $LR = 112.05$, $p \leq .001$. DSSAI-Helplessness scores, history of an MDE, and a smaller household size showed the highest AORs.

DISCUSSION

Our data showed that 28.52% of youths reported HCDS lasting at least 1 year and 22.34% met criteria for HACDD of this duration. About 13.40% and 9.97% showed a course lasting at least 2 years for HCDS and HACDD, respectively. These rates are consistent with those reported by Essau and Chang (2009). When computing lifetime CD rates for the entire DSSAI project sample ($N = 621$) applying adult criteria (2 years), we found rates from 4.67% (for HACDD) to 6.28% (for HCDS). Such results are impressively similar to rates reported in the general population of adults and adolescents (Breaslau et al., 2017; Garland & Scot, 2008; Murphy & Byrne, 2012), and provide indirect support for the validity of our clinical diagnoses.

We examined correlates and predictors of CD among Hispanic/Latina(o) youth in PR and

identified correlates in most domains considered. As expected, a history of suicidal ideation/behavior, an earlier age of onset, indicators of symptom severity, and having multiple areas of impairment, related significantly to CD after adjusting for CDI scores. Emotions and impairment at home were the specific impaired areas related to increased odds of CD, which agree with findings of prolonged suffering and conflicted family relationships among these patients. As meeting criteria for a depressive disorder increases the odds for HCDS, it is clear that any impairment accompanying those symptoms could help to prolong their duration in youth, but especially when the number of impaired areas increases.

Several correlates of CD reported in the youth research literature were also significantly associated with chronicity in our sample. The latter included variables such as previous depressive episodes, interpersonal problems, greater use of treatment services, somatic symptoms, feelings of guilt, anhedonia, suicidality, and negative beliefs (e.g., hopelessness and helplessness). These findings are consistent with the integrative theoretical view of CD proposed by Lara and Klein (1999), particularly supporting the role of interpersonal and cognitive aspects in maintaining depression persistence. Yet, suffering depression in response to stressful life events (such as childhood adversity) did not distinguish episodic and CD in this sample. On the other hand, sociodemographic factors such as living in one-caregiver homes, small household size, lower SES, and rural living zone were related with CD in this study after adjusting for CDI scores. Youths who lived in homes with these characteristics may have experienced reduced access to family and external social support, particularly if three or all of the characteristics were present. Since some socio-demographic factors may operate as social determinants of health, further studies should examine if the accumulative presence of these factors could relate to an additional increase in the odds of CD in youths living in PR. Among households in which only one caregiver was present, it

was the father figure who was absent in most cases. This finding may relate to reports linking low involvement with fathers and CD (Sanford et al., 1995). In addition, the association of low SES with increased odds for HACDD may reflect the relationship between family adversity and CD (Klein & Black, 2017; Murphy & Byrne, 2012).

Self-reports about specific symptom domains in the L6M were among correlates with the highest adjusted associations with CD. Suicidality, Undervaluing/Self-reproach (a measure of self-esteem/guilt problems), Somatic Alterations, Anhedonia, and Helplessness scores were the strongest and most consistently related with increased odds for HCDS and HACDD. The latter two subscales were independent predictors of CD in the multiple regression models (Table 2). The Anhedonia subscale was the only one that contributed independently and significantly to models accounting for membership in both HCDS and HACDD groups. Its items target poor satisfaction with life, indifference about things, lack of interest in activities and relationships, low motivation, reduced ability to enjoy or have pleasure, and lack of reactivity when faced with positive events or social reinforcement. Intense anhedonia may not only prolong the duration of MDD episodes in non-referred adolescents, as reported by Essau (2007), but also the chronicity of sub-threshold episodes and/or dysthymia, as seems to be the case in the current study. We need more research to understand the mechanisms that underlie these findings since previous research identified anhedonia as a negative prognostic indicator among treatment-resistant depressed adolescents (McMakin et al., 2012). There is a long historical track of authors linking anhedonia to CD (see Kessel & Klein, 2016). Interestingly, anhedonia, as well as suicidality and helplessness, are not currently included among symptoms that define a PDD (APA, 2013).

Previous studies identified self-esteem problems and feelings of guilt as predictors of CD (Randenborgh et al., 2016; Rubio et al.,

2011). Related feelings and thoughts may arise or be reinforced by a history of personal failure, childhood adversity, and chronic interpersonal stress, all of which have been identified as predictors of CD. A negative view of the self and propensity to guilt, self-criticism, and self-devaluation frequently coexist with death wishes and suicidal thoughts in youths. This cluster of symptoms, initially known as *self-deprecatory ideation*, was considered as one of two core indicators of pediatric depression years before the publication of the DSM-III. Such presentation reflects a severe type of negative self-referent thinking and there is evidence that suggests it is consistent across episodes (Williams et al., 2006). Our findings suggest that this cluster may deserve further examination as a possible anticipatory signal for chronicity, at least among Hispanic/Latina(o) youth from schools in the SJMA-PR.

Detecting CD in youth must be a public health priority. As Hispanics/Latinas(os) may be more prone to CD than non-Hispanic Whites, identifying predictors of CD in this group may help to improve their clinical assessment, diagnostic precision, as well as access to quality of care. Improved assessment and diagnosis may allow an earlier referral to appropriate acute treatment. Instead of reinforcing the practice of referring youths only if presenting physical symptoms, or when symptoms cause school-related difficulties, our results suggest that guardians and school staff should consider emotional impairment, especially if concurrent with impairment at home, as a sufficient indicator of a need for treatment among Hispanic/Latina(o) youth in the SJMA-PR. Consistent with findings from Shain et al. (1991), youth with HACDD had higher odds of antidepressants use. Some received help only after years of suffering and, still, 66.62% remained untreated, which is similar to other reports (Essau, 2007). Early detection of emotional problems and appropriate referral could result in a sooner reduction of suffering since CD relates to a slower initial rate of improvement.

The limitations of our study must be considered when interpreting our results. Our sample was self-selected, which may have introduced bias in the type of participants. Our assessment of diagnostic criteria and clinical history relied on self-reports, which made it necessary to adjust analyses for depression severity at assessment. Moreover, our cross-sectional design, which relies heavily on retrospective reports, did not allow us to distinguish the nature of changes in variables over time nor assess other possible confounds or predictors (e.g., chronic interpersonal stress, family history of depression, etc.). Further research with Hispanic/Latina(o) youth should examine predictors of CD using longitudinal designs and diagnostic measures that do not rely exclusively on self-reports. Other potential predictors of CD should be included.

On the other hand, our analyses were conducted on a sample recruited from 2010–2012. By then, people in PR have lived the first 5–7 years of a long economic crisis, which included a government closure in 2006, the firing of thousands of public employees in 2009, and significant budget costs, particularly in government services (see Special Act to Declare a State of Fiscal Emergency and to Establish a Comprehensive Fiscal Stabilization Plan to Salvage the Credit of Puerto Rico, 2009). Subsequently, residents of the island faced a 64.3% increase in their local sales tax, from 7.0% to 11.5% (Ley Núm. 72 del 29 de mayo de 2015) and a prolonged drought period (2014–2016), which included intermittent water rationing and severe economic losses in many municipalities (Department of Natural and Environmental Resources, 2016). This was followed by the imposition of a Financial Oversight and Management Board by the U.S. Congress over the local government, under the Puerto Rico Oversight, Management, and Economic Stability Act (PROMESA, 2016). The latter initiated a second series of austerity public policies that have further affected health services and the economy of local families. In addition, our island experienced the catastrophic

consequences of hurricanes Irma and Maria in 2017 (see Orengo-Aguayo et al., 2019), a political and governance crisis that resulted in the resignation of the local governor in July 2019, several earthquakes from December 2019 to May 2020, and the current COVID-19 pandemic. It is unknown to what extent our results would have been (or could be) replicated if similar studies had been (or were to be conducted) after this cumulative series of stressful and potentially traumatic circumstances. Unfortunately, although some studies have reported on the rates of significant depressive symptoms in youths after some of these traumatic events (e.g., Orengo-Aguayo et al., 2019), as far as we know, no study has even reported data on the prevalence of CD among children or adolescents in PR since or before our data collection.

In the context of the COVID-19 pandemic, depressive symptoms have increased and persisted over time. A meta-analysis of studies conducted with children and adolescents during the first year of the pandemic showed a pool estimated of 25.2% of clinically elevated depressive symptoms, which are double of pre-pandemic estimates (Racine et al., 2021). Youth's sensitivity to mitigation measures, deterioration of family health and economic conditions, increased screen time and social media dependence, domestic violence/abuse, academic worries, or bereavement for losing normal routines may contribute to an acceleration of their mental health deterioration (Cousien et al., 2021). Ongoing and future studies in the (post)pandemic era should assess the impact of stressors related to the COVID crisis and explore its potential role as correlates of CD in youth. We should allocate clinical resources to address this age group given its higher risk for depression as the COVID pandemic evolves (Racine et al., 2021). An evidence-based assessment of risk can focus resources on those at greatest risk for a CD trajectory (Hankin, 2017).

As argued by Klein and Black (2017), "the persistent/nonpersistent distinction is an important source of heterogeneity in depressive disorders that should be considered in both clinical practice and research" (p. 233). Examining CD correlates and predictors may help to identify cases that might need a higher number of psychotherapy sessions or longer continuation pharmacotherapy (Melrose, 2017). As chronicity relates to relapse and resistance to treatment, knowledge about CD correlates and predictors may facilitate tailoring interventions to patients' needs. For example, cognitive therapy with these patients requires a greater emphasis on targeting hopelessness, helplessness, and perfectionism, addressing early-life adverse experiences, and modifying maladaptive schemas (Riso & Newman, 2003). When targeting this clinical course, evidence also suggests some interventions or combinations of treatments (e.g., combined psychotherapy and antidepressants) are more efficacious than others are (Brent, 2018; Lorenzo-Luaces et al., 2020). CD could also affect treatment dose. For instance, among youths with treatment-resistant depression, a therapeutic dose of more than nine sessions and the use of social skills and problem-solving components have shown to be efficacious when using cognitive-behavioral therapy (Hamill-Skoch et al., 2012).

As the distinction between episodic and CD is relatively stable over time (Klein & Black, 2017), research that facilitates such distinction may help to appropriately select treatment type, dose, course, goals, components, and outcome measures, reducing patient and family burden, promoting psychosocial functioning and contributing to decrease treatment costs. Our study is just a first step in that direction and a modest but significant contribution to help in closing research gaps about CD among Hispanic/Latina(o) youth, particularly if living in the SJMA-PR.

Research Ethical Standards

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