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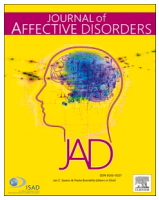

Research paper

Affective dysregulation, familial psychopathology, and suicidal behavior in transgender individuals: A structural equation model

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ABSTRACT

Background: Transgender individuals present disproportionately high rates of suicidal behavior, yet little is known about the affective and familial mechanisms that explain this vulnerability. This study examined an explanatory model of suicidal behavior grounded in affective dysregulation, impulsive aggression, and familial psychopathology.

Methods: A clinical sample of 506 transgender individuals attending a gender identity unit in Spain completed structured assessments of mood disorders, impulsive aggression, exposure to violence, childhood abuse, and suicidal behaviors. Structural equation modeling (SEM) using maximum likelihood estimation was applied to test direct and indirect pathways linking familial and affective variables to suicide attempts.

Results: The final model demonstrated excellent fit (χ^2/df = 1.692, GFI = 0.955, IFI = 0.956, RMSEA = 0.037) and explained 48% of the variance in suicide attempts. Affective dysregulation ($\beta = 0.520$, $p < .001$), impulsive aggression ($\beta = 0.173$, $p = .015$), and environmental adversity ($\beta = 0.420$, $p < .001$) were significant predictors of suicidal behavior. Parental mood disorders exerted indirect effects on suicide attempts through their influence on offspring mood disturbances.

Limitations: The cross-sectional design precludes causal inference. Mood disorder diagnoses relied on clinical interviews rather than standardized diagnostic instruments. The wide age range may limit generalizability to adolescent-specific samples.

Conclusions: Findings indicate that suicidal behavior in transgender individuals arises from interrelated affective, familial, and environmental mechanisms. Early detection of mood disturbances, assessment of impulsive aggression, and family-based interventions may help reduce suicide vulnerability in this population.

1. Introduction

Suicide is a leading cause of death worldwide and represents a critical endpoint of affective dysregulation, impulsivity, and maladaptive stress responses. Central models in the field conceptualize suicidal behavior as the convergence of affective vulnerability, impaired emotion regulation, and exposure to environmental adversity (O'Connor and Nock, 2014; Klonsky and May, 2015). Affective disturbances, particularly depressive and anxiety symptoms, hopelessness, and mood instability, are among the most robust predictors of suicidal ideation and attempts. Understanding how these affective mechanisms interact with familial and contextual factors remains a crucial challenge for suicide research. Beyond categorical mood diagnoses, contemporary research emphasizes transdiagnostic affective processes, including emotion regulation deficits, rumination, defeat and entrapment cognitions, and heightened stress reactivity (Franklin et al., 2017; O'Connor and Kirtley, 2018). These processes may be particularly salient in populations exposed to chronic social stress, where repeated invalidation and discrimination amplify emotional vulnerability (Hatzenbuehler, 2009; Thoits, 2011). Such mechanisms have been associated not only with suicidal ideation but also with the progression from ideation to attempts (Klonsky and May, 2015; O'Connor, 2011).

Transgender individuals constitute a population with disproportionately elevated rates of suicidal behavior, often several times higher than in the general population (Grant et al., 2011; Toomey et al., 2018). While the heightened prevalence is well established, less is known about why this vulnerability emerges. Existing research suggests that transgender individuals frequently experience chronic stressors such as rejection, discrimination, victimization, and emotional invalidation (Meyer, 2003; Plöderl and Tremblay, 2015), which may potentiate

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underlying affective vulnerabilities. Consistent with stress–diathesis models, exposure to persistent social adversity may activate or exacerbate affective dysregulation, increasing the likelihood of suicidal behavior. However, few studies have examined the joint contribution of affective, familial, and environmental mechanisms within a unified explanatory framework. Large epidemiological surveys have consistently reported markedly elevated rates of suicidal ideation and attempts among transgender individuals compared to cisgender populations. Lifetime suicide attempt rates ranging from 30% to 45% have been documented in national samples (Grant et al., 2011; Haas et al., 2011), with recent population-based studies confirming significantly higher odds of suicidality among transgender individuals even after controlling for psychiatric comorbidity (Becerra-Culqui et al., 2018; Toomey et al., 2018). These disparities are not restricted to specific countries but have been observed across diverse sociocultural contexts, underscoring the global relevance of the phenomenon (Bauer et al., 2015). Importantly, the psychological and behavioral mechanisms examined in this study, such as affective dysregulation, impulsive aggression, and exposure to environmental adversity, are not specific to transgender populations, but represent transdiagnostic processes consistently implicated in suicidal behavior across diverse groups. The present study does not aim to identify mechanisms unique to transgender identity, but rather to examine how these well-established pathways operate within a clinical population that is disproportionately exposed to psychosocial stressors. In this context, transgender individuals may show a higher prevalence or intensity of these mechanisms, not as a function of identity per se, but as a consequence of increased exposure to environmental adversity and minority-related stress.

A large body of evidence highlights the role of family context in shaping affective development and suicide risk. Parental mood disorders have been linked to emotional dysregulation, negative affectivity, and internalizing symptoms in offspring, both through genetic transmission and through the effects of caregiving environments characterized by inconsistency, emotional unavailability, or heightened conflict (Brent and Mann, 2006). Similarly, parental impulsive–aggressive traits may influence the emergence of reactive aggression or impaired volitional control in adolescents and young adults, two pathways associated with the transition from suicidal ideation to suicidal action (Gvion and Apter, 2011; Brent and Mann, 2006). These intergenerational processes suggest that familial psychopathology contributes to suicide risk not only directly but also indirectly through affective vulnerability.

Environmental adversity, including exposure to violence, emotional abuse, or hostile social climates, further amplifies suicide risk (McLaughlin et al., 2019; Teicher and Samson, 2016). Such experiences can disrupt emotion regulation systems, foster hyperreactivity to stress, and reinforce maladaptive interpersonal schemas. Previous studies have shown that discrimination, victimization, and social threat are associated with depressive symptoms, social anxiety, and self-injurious behaviors among transgender individuals (Clements-Nolle et al., 2006; Hatzenbuehler, 2009). These findings align with contemporary ideation-to-action theories of suicide, which posit that affective drivers (e.g., dysphoria, hopelessness) and volitional moderators (e.g., impulsive aggression, habituation to pain or threat) jointly determine the likelihood of suicidal behavior (O'Connor, 2011). Minority Stress Theory provides a useful explanatory framework for understanding these disparities (Meyer, 2003). According to this model, stigma, discrimination, and structural marginalization create chronic stress exposure that accumulates over time, increasing vulnerability to affective disorders and suicidal behavior. Empirical studies have shown that experiences of rejection, misgendering, and victimization are directly associated with depressive symptoms and suicidality among transgender individuals (Clements-Nolle et al., 2006; Hatzenbuehler, 2009; Plöderl and Tremblay, 2015). Importantly, minority stress processes operate through affective dysregulation and interpersonal vulnerability, rather than through intrinsic characteristics of gender identity.

Despite robust evidence linking minority stress, affective vulnerability, and suicidality in transgender populations, few studies have simultaneously examined familial psychopathology, affective dysregulation, impulsive aggression, and environmental adversity within a unified multivariate framework. Most prior studies have relied on bivariate associations or regression-based approaches that do not permit simultaneous estimation of indirect pathways or reciprocal influences. Prior research in Spain, including clinical work by Guzmán-Parra et al. (2015) and an exploratory study on social anxiety and gender dysphoria (Bergero-Miguel et al., 2016), highlighted elevated affective symptoms and psychosocial vulnerability in this group. However, these studies did not test the structural pathways linking familial and affective factors to suicidal behavior.

Structural equation modeling (SEM) provides a robust methodological tool for testing complex explanatory models of suicide risk (Lei and Wu, 2012). SEM allows simultaneous estimation of direct and indirect effects, enabling a nuanced examination of how parental mood disorders, environmental adversity, and emotional dysregulation interact to predict suicidal outcomes. Applying SEM to a large transgender clinical sample offers an opportunity to clarify the affective and contextual determinants of suicidal behavior within a group that faces substantial mental health disparities. By testing a theoretically grounded structural model, the present study seeks to move beyond prevalence documentation toward mechanistic clarification of suicide risk in transgender individuals.

Importantly, the present study does not aim to examine the developmental origins or determinants of transgender identity. While factors such as family environment, affective vulnerability, or exposure to adversity have been examined in other areas of developmental psychopathology, the current model is exclusively focused on mechanisms underlying suicidal behavior within a transgender clinical population. Accordingly, the variables included in this study are conceptualized as risk factors for suicidality and should not be interpreted as explanatory factors for gender identity. Although prior studies have examined associations between affective symptoms, family context, and suicidality among transgender individuals, these investigations have primarily relied on regression-based or bivariate approaches. Such methods do not permit simultaneous estimation of intergenerational, affective, and contextual pathways or the testing of indirect effects within a unified explanatory framework. The present study therefore seeks to clarify the structure of these associations rather than merely apply a specific statistical technique. Based on developmental and intergenerational models of psychopathology (Brent and Mann, 2006; Weissman et al., 2005), parental mood disorders are expected to influence offspring suicidality primarily through the transmission of affective vulnerability rather than through direct behavioral pathways. Similarly, impulsive aggression has been conceptualized as a volitional facilitator of suicidal action rather than a primary driver of suicidal ideation (Klonsky and May, 2015; O'Connor, 2011). We hypothesized that: (1) parental mood disorders would indirectly predict suicide attempts through their impact on offspring mood disturbances; (2) impulsive aggression would serve as a volitional factor increasing the likelihood of suicidal behavior; (3) environmental adversity would exert both direct and indirect effects on suicide attempts through affective dysregulation; and (4) the integrated model would demonstrate adequate fit and explain a substantial proportion of variance in suicidal behavior.

By focusing on affective pathways and their intersection with familial and environmental contexts, this study provides a comprehensive test of core mechanisms that underlie suicidal behavior in a transgender clinical sample. The findings aim to contribute to the broader literature on affective disorders and suicide by clarifying how emotional vulnerability, impulsive tendencies, and intergenerational risk interact within a high-risk population.

2. Method

2.1. Participants

Participants were 506 individuals diagnosed with gender dysphoria according to DSM-5 criteria who were consecutively assessed at the Transgender and Gender Identity Unit (UTIG) of the Carlos Haya Regional University Hospital in Málaga, Spain. The sample comprised 284 transgender men (56.1%) and 222 transgender women (43.9%), with a mean age of 27.81 years (SD = 8.88; range = 14–61). Recruitment occurred during routine diagnostic and clinical evaluation procedures prior to gender-affirming interventions. Inclusion criteria were: (a) age ≥14 years, (b) diagnosis of gender dysphoria confirmed by specialized clinicians, and (c) ability to provide informed consent. Exclusion criteria were: (a) active psychotic disorder, (b) severe cognitive impairment preventing reliable assessment, and (c) acute intoxication at the time of evaluation. No participants were excluded based on current mood disorder severity, substance use history, or prior suicide attempts. All participants were evaluated at their first psychiatric intake visit to the specialized gender identity clinic. The study did not include variables assessing the development or formation of gender identity, as the focus was restricted to clinical and psychosocial factors associated with suicidal behavior. Importantly, all clinical assessments were conducted prior to the initiation of any medical or surgical gender-affirming interventions, ensuring that participants were assessed at a comparable stage of clinical presentation. The study received approval from the institutional ethics committee (protocol MH-UTIG/PSY/14-03), and all participants provided informed consent. For minors, assent was obtained alongside parental consent. The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Carlos Haya Regional University Hospital. All participants (and parents, when applicable) provided informed consent. Participation was voluntary and did not affect the diagnostic or clinical services received. All eligible patients attending their first psychiatric intake visit during the study period were invited to participate. Consecutive recruitment was used; the vast majority of approached patients consented to participate. Exact refusal rates were not systematically tracked as part of the routine intake protocol.

2.2. Procedure

The intake evaluation was conducted before any hormonal or surgical gender-affirming treatment was initiated. This ensured that psychiatric diagnoses and environmental risk assessments were not influenced by subsequent medical transition processes. Participants completed a structured clinical assessment conducted by trained psychologists and psychiatrists specializing in gender identity and affective disorders. The assessment took place during two scheduled sessions within the diagnostic protocol of the unit. In the first session, clinicians conducted a detailed interview evaluating psychiatric history, mood symptoms, impulsive–aggressive behaviors, exposure to violence, and prior suicidal behaviors. In the second session, participants completed standardized questionnaires and validated measures of emotional and behavioral functioning. The standardized instruments administered included: the Beck Depression Inventory (BDI; Beck et al., 1961) and the Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983) to assess mood and anxiety symptoms; the Affect Scale (EA-Hijos) and the Norms and Demands Scale (ENE-Hijos; Bersabé et al., 2001) to assess perceived parental style dimensions (affective communication, criticism-rejection, inductive, rigid, and indulgent parenting); the Duke-UNC Functional Social Support Questionnaire (Broadhead et al., 1988) to assess perceived social support; the Fear of Negative Evaluation scale (FNE) and Social Avoidance and Distress scale (SAD; Watson and Friend, 1969) to assess social anxiety; the SCID-II personality disorder screening questionnaire (First et al., 1997); and the Violence Exposure Questionnaire (CEV; Orue and Calvete, 2010), a 21-item validated instrument capturing victimization and witnessing of violence across home, school, neighborhood, and media contexts. Diagnoses of current and past mood disorders (major depressive disorder, dysthymia, bipolar disorder, and anxiety disorders) in both participants and their parents were established using a structured clinical interview based on DSM-5 criteria. Although structured instruments such as the SCID-5 were not administered, clinicians followed a uniform diagnostic checklist used in the unit to ensure consistency across evaluations. Inter-rater agreement was monitored during regular team meetings, and discordant cases were reviewed until consensus was reached. All assessments were conducted prior to medical or surgical gender-affirming interventions, ensuring that participants were evaluated at a comparable stage of clinical transition. The variables included in the structural model were derived from routine clinical intake assessments and were not collected exclusively for research purposes. No additional research-only diagnostic instruments were administered.

2.3. Measures

All constructs were assessed as part of the routine psychiatric intake protocol conducted at the specialized gender identity clinic. All assessments were conducted through clinician-administered structured psychiatric interview, supplemented where available by review of clinical records. No measure relied exclusively on self-report; all assessments were conducted by trained mental health professionals specializing in gender identity and affective disorders. Variables were coded to match their specification within the structural equation model (SEM). Several indicators were dichotomous (0/1), reflecting the presence versus absence of clinically meaningful phenomena. The inclusion of categorical variables in SEM is methodologically appropriate and widely supported in the literature when robust estimators are employed (Lei and Wu, 2012; Muthén, 1984). Robust weighted least squares estimators (e.g., WLSMV) are specifically recommended for modeling dichotomous or ordinal indicators within SEM frameworks (Li, 2016; Muthén and Muthén, 2017).

2.3.1. Parental mood disorder

Parental mood disorder was assessed via clinician-administered structured interview with the participant, during which detailed family psychiatric history was systematically elicited. Where available, this information was corroborated through the patient's own clinical records, which included documented family history. Direct access to parents' independent medical records was not obtained. The presence of a lifetime mood disorder (major depressive disorder and/or bipolar disorder) in either parent was coded dichotomously (0 = absent; 1 = present). In the SEM, this variable was specified as an exogenous predictor of participant mood disorder.

2.3.2. Parental impulsive aggression

Parental impulsive aggression was assessed through structured psychiatric evaluation and clinical documentation of impulsive-aggressive behavioral patterns. The variable was coded dichotomously (0 = absent; 1 = present). In the SEM, parental impulsive aggression was treated as an exogenous predictor of participant impulsive aggression.

2.3.3. Suboptimal family environment

Suboptimal family environment was restricted to intra-family relational dynamics (e.g., chronic conflict, emotional invalidation, instability within the household). This construct was operationalized using information from the clinician-administered interview and participant responses on the EA- Children (Affect Scale) and ENE- Children (Norms and Demands Scale; Bersabé et al., 2001), which assess perceived parental affective communication, criticism-rejection, and degree of parental control. A dichotomous composite was derived (0 = no suboptimal environment; 1 = present) based on clinician judgment integrating these sources. School and neighborhood violence were coded

separately under environmental adversity. These two constructs are conceptually and operationally distinct in the model: Suboptimal Family Environment captures intrafamilial dysfunction, while Environmental Adversity encompasses extra-familial contextual stressors (school and neighborhood violence). This variable was coded dichotomously (0 = no suboptimal environment; 1 = present). In the SEM, suboptimal family environment was modeled as an exogenous predictor of abuse/neglect, participant mood disorder, participant impulsive aggression, and suicide attempt.

2.3.4. Life stressors

Life stressors were assessed as the number of clinically significant stress-inducing events reported at intake. Examples of included events are: job loss, divorce or major relationship breakdown, serious illness of a significant other, financial crisis, housing instability, bereavement, and legal problems. Childhood abuse, neglect, and school and neighborhood violence were assessed and coded separately and were explicitly excluded from the life stressors count to avoid redundancy across constructs. Life stressors were assessed using the "Significant Psychological Events" section of the UTIG structured intake questionnaire, which records the occurrence, age at occurrence, and recency (within the last 12 months) of a broad range of adverse life events across multiple domains (interpersonal losses, family disruptions, occupational and legal difficulties, severe illness, among others). The life stressors score reflects a weighted composite across event categories and developmental periods, where multiple independent events across the lifespan each contribute to the total. The observed range (0–58, M = 5.76, SD = 4.6) reflects this cumulative structure, with high values concentrated in a small subset of participants with complex multilevel psychosocial histories. This variable was treated as a count variable and entered into the SEM as an observed predictor of suicide attempt. Because this variable represents a count of discrete events rather than a multi-item scale, internal consistency reliability (e.g., Cronbach's alpha) is not applicable. These variable and Suboptimal family environments represent distinct dimensions of environmental exposure: chronic family dysfunction versus acute life stressors.

2.3.5. Childhood abuse and neglect

History of abuse and neglect was assessed separately for childhood (before age 12) and adolescence (ages 12–17) during clinical interview. Experiences included physical, emotional, or sexual abuse occurring within the family or broader social environment. Broader exposure to violence in contextual settings (school, neighborhood, media) was assessed using the CEV (Orue and Calvete, 2010) and contributed to the Environmental Adversity construct rather than to the abuse/neglect composite. For modeling purposes, a dichotomous composite variable (0 = no abuse; 1 = any abuse across developmental periods) was used. In the SEM, abuse/neglect was specified as a mediator linking suboptimal family environment to affective and behavioral vulnerabilities. Intrafamilial abuse (perpetrated by a parent or primary caregiver, including physical, emotional, or sexual abuse) was captured within this composite and is conceptually distinct from extra-familial victimization (e.g., school or neighborhood violence), which was coded separately under Environmental Adversity. Neglect and physical abuse were included in this composite variable alongside emotional and sexual abuse.

2.3.6. Participant mood disorder

Participant mood disorder was diagnosed by an experienced psychiatrist according to DSM-based criteria during structured clinical evaluation. In the structural model, participant mood disorder was specified as a latent variable indicated by two dichotomous clinical indicators derived from validated psychometric instruments: (1) the Beck Depression Inventory (BDI; Beck et al., 1961), coded as 0 = below clinical threshold/1 = clinical caseness based on established cut-off scores; and (2) the Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983), coded as 0 = below threshold/1 = probable mood/anxiety disorder based on established cut-off scores. Both indicators were coded dichotomously (0 = absent; 1 = present) reflecting the presence of clinically significant affective symptomatology. The use of two observed indicators to define a latent variable is consistent with standard SEM practice for constructs assessed via multiple instruments (Lei and Wu, 2012). The factor loadings of the BDI indicator ($\beta = 0.614$, $p < .001$) and the HADS indicator ($\beta = 0.605$, $p < .001$) confirmed adequate indicator reliability. Within the SEM, participant mood disorder functioned as a central latent mediator between familial/environmental risk factors and suicide attempt.

2.3.7. Participant impulsive aggression

Participant impulsive aggression was assessed during structured psychiatric intake and coded dichotomously (0 = absent; 1 = present). The following clinician-rated item was used: "Has the patient displayed a history of impulsive or explosive aggressive behaviors, including verbal or physical outbursts that are disproportionate to the precipitating stimulus and inconsistent with the patient's general level of functioning?" Coding was based on the clinician's overall judgment and review of behavioral history. Assessment integrated multiple converging sources: (1) the impulsivity and aggression-related items of the SCID-II screening questionnaire (items 96–103), covering explosive anger, loss of behavioral control, and impulsive behavior patterns; (2) the "Assaults and Abuse" section of the UTIG structured intake questionnaire, which directly asks whether the participant has ever felt so angry or enraged as to have verbally, physically, or sexually aggressed another person; and (3) the clinician's integrated judgment synthesizing these sources. In the SEM, this variable served both as a mediator and as a direct predictor of suicide attempt.

2.3.8. Suicidal ideation

Suicidal ideation was assessed during the structured psychiatric intake interview conducted by an experienced clinician. Participants were asked whether they had experienced recurrent thoughts of death, wishes to die, or thoughts of killing themselves at any point in their lifetime. When clinically indicated, additional probing was conducted to clarify frequency and intensity of ideation. Suicidal ideation was coded dichotomously (0 = no lifetime suicidal ideation; 1 = presence of lifetime suicidal ideation). Although not included as a dependent variable in the structural equation model, suicidal ideation was examined descriptively to characterize the severity of suicidal phenomena within the sample and to contextualize the prevalence of suicide attempts. Suicidal ideation was excluded from the structural model due to its high collinearity with the primary outcome (suicide attempt) and the theoretical focus on behavioral rather than ideational outcomes.

2.3.9. Suicide attempt

Lifetime suicide attempt was assessed during the structured psychiatric intake interview conducted by an experienced clinician. Participants were asked whether they had ever engaged in self-injurious behavior with at least some intent to die. This criterion distinguishes suicide attempts from non-suicidal self-injury (NSSI); behaviors without suicidal intent were not coded as positive on this variable. When available, responses were corroborated through review of clinical records. Suicide attempt was coded dichotomously (0 = no lifetime attempt; 1 = at least one lifetime suicide attempt). This variable served as the primary outcome in the structural equation model. Given its dichotomous nature, it was modeled as a categorical indicator within the SEM framework using robust estimation procedures appropriate for binary outcomes.

Several study variables were coded dichotomously (0/1) based on structured clinical interview. Because these indicators do not represent multi-item psychometric scales, internal consistency reliability indices are not applicable. Although minority stress processes such as discrimination and stigma are highly relevant in transgender mental health research, specific measures of discrimination were not included in the

present clinical intake protocol. Environmental adversity variables in this study primarily reflected family and contextual instability rather than direct assessment of minority stress experiences.

2.4. Statistical analysis

2.4.1. Rationale for structural equation modeling

Structural equation modeling (SEM) was selected because it allows simultaneous estimation of complex interrelationships among affective, familial, and environmental variables, distinguishing direct and indirect effects. This approach is consistent with contemporary theoretical models of suicidal behavior, which conceptualize risk as the result of interconnected mechanisms rather than independent predictors. SEM allows distinguishing clinically overlapping risk factors that are often collinear in psychiatric samples.

2.4.2. Model specification

The hypothesized structural model included: Parental mood disorders and parental impulsive aggression as exogenous variables, participant mood disorders and impulsive aggression as mediating variables, environmental adversity as both a mediating and direct predictor, and suicide attempts as the endogenous outcome. Indirect effects tested the pathways from parental psychopathology to suicidal behavior through affective vulnerability and impulsivity. Childhood abuse and neglect were included as an additional risk factor but were not expected to contribute directly after accounting for affective pathways.

2.4.3. Model estimation

Analyses were conducted using AMOS 20.0 with maximum likelihood estimation (MLE). This estimation method is robust for moderate deviations from normality and is widely used in psychiatric and affective research. Prior to model estimation, the distribution of the life stressors variable was examined to assess potential zero inflation. Although a proportion of participants reported no recent stressors, the frequency distribution did not indicate excessive zero inflation beyond what would be expected in a clinical intake sample. Therefore, the variable was modeled as an observed count predictor within the SEM without zero-inflated specification. Model fit was evaluated using standard indices: χ^2/df ratio (<3 acceptable), Goodness-of-Fit Index (GFI > 0.90), Incremental Fit Index (IFI > 0.90), and Root Mean Square Error of Approximation (RMSEA < 0.06). A priori power analysis for SEM using the semPower framework (Moshagen and Bader, 2024). The analysis demonstrates that, given the sample size (N = 506), the proposed model is adequately powered to detect small-to-moderate effects (RMSEA-based power > 0.90).

2.4.4. Missing data

Missing data were minimal (<3%) and handled using full information maximum likelihood (FIML), which provides unbiased parameter estimates under missing-at-random (MAR) assumptions.

2.4.5. Sensitivity analyses

To evaluate the stability of the model across subgroups, sensitivity analyses were conducted by gender identity (trans men vs. trans women) and age (≤25 vs. >25). These analyses examined whether model pathways varied across demographic strata. No significant differences in structural paths were observed, and thus the final model was estimated using the full sample.

3. Results

3.1. Sample characteristics

The final sample consisted of 506 transgender individuals (56.1% transgender men; 43.9% transgender women) with a mean age of 27.81 years (SD = 8.88) (see Table 1). Lifetime prevalence of mood disorders in participants was 58.7%. Parental mood disorders were present in 20.3% of cases. Impulsive–aggressive behavior was reported by 27.5% of participants. A total of 24.1% of the sample reported at least one lifetime suicide attempt. Suboptimal family environment before age 18 was common: 61.8% experienced recurrent discrimination or violence in at least one context (family, school, or community). The mean number of reported life stressors was M = 5.76 (SD = 4.6), ranging from 0 to 58. For descriptive purposes, 62% of participants reported at least one life stressor. Childhood abuse or neglect was reported by 8.1%. No significant demographic differences were observed between transgender men and transgender women in mood disorder prevalence, impulsive aggression, or suicide attempt rates (all p > .10). Age was weakly but significantly correlated with suboptimal family environment (r = −0.14, p = .004), indicating slightly higher adversity among younger participants.

Table 1
Descriptive statistics of sociodemographic and clinical variables used in the SEM (N = 506).

Variable	Category/coding	N (%)/M (SD)
Gender identity	Trans men	284 (56.1)
	Trans women	222 (43.9)
Age group	13–17	33 (6.5)
	18–24	195 (38.5)
	25–34	166 (32.8)
	35–44	80 (15.8)
	≥45	32 (6.3)
Education	Primary	138 (27.3)
	Secondary	307 (60.7)
	University	61 (12.1)
Marital status	Married	6 (1.2)
	Living with partner	105 (20.8)
	Single	395 (78.1)
Occupational status	Employed	256 (50.6)
	Unemployed	250 (49.4)
Suicidal ideation (<18 years)	Yes	188 (37.2)
Suicide attempt (<18 years)	Yes	56 (11.1)
Parent mood disorder	0 = No/1 = Yes	153 (30.2%)
Parent impulsive aggression	0 = No/1 = Yes	139 (27.5%)
Suboptimal family environment	0 = No/1 = Yes	222 (43.9%)
Abuse and neglect of child	0 = No/1 = Yes	41 (8.1%)
Life stressors	Count variable	M = 5.76 (SD = 4.6)
Participant mood disorder	0 = No/1 = Yes	297 (58.7%)
Participant impulsive aggression	0 = No/1 = Yes	234 (66%)
Suicide ideation (lifetime)	0 = No/1 = Yes	267 (52.8%)
Suicide attempt (lifetime)	0 = No/1 = Yes	122 (24.1%)

Note. Binary variables were coded as 0 = absence and 1 = presence of the clinical condition. Life stressors represent the number of significant stress-inducing events reported at intake.

3.2. Bivariate associations

As expected, participant mood disorders were positively correlated with impulsive aggression (r = 0.29, p < .001), suboptimal family environment (r = 0.33, p < .001), and suicide attempts (r = 0.36, p < .001). Impulsive aggression was also correlated with suicide attempts (r = 0.23, p < .001). Parental mood disorders were associated with participant mood disorders (r = 0.31, p < .001) and, to a lesser extent, with impulsive aggression (r = 0.17, p = .002). These correlations supported the hypothesized configuration of affective, impulsive, and familial pathways.

3.3. Structural equation model

To test the hypothesized relationships among parental, familial, and psychological factors associated with suicidal behavior, a structural equation model (SEM) was estimated using maximum likelihood estimation in AMOS 20. The final model demonstrated an excellent overall fit χ^2/df = 1.692, GFI = 0.955, IFI = 0.956, RMSEA = 0.037. The model

explained 48% of the variance ($R^2 = 0.48$) in suicide attempt. About direct effects, significant direct effects were observed between several constructs (see Table 2). Mood disorder (β = 0.520, p = .001), impulsive aggression (β = 0.173, p = .015), and suboptimal family environment (β = 0.420, p = .001) showed significant positive associations with suicide attempt. Parent mood disorder significantly predicted mood disorder (β = 0.379, p = .023). Suboptimal family environment positively predicted abuse and neglect (β = 0.318, p = .002). No significant direct relationship emerged between life stressors and suicide attempt (β = −0.049, p = .512).

Simultaneously in the indirect effects, bootstrapped indirect effects revealed that parent mood disorder and parent impulsive aggression influenced suicide attempt indirectly through their effects on mood disorder, impulsive aggression, and suboptimal family environment. Notably, parent mood disorder had a substantial indirect influence on suicide attempt (β = 0.379), while parent impulsive aggression showed a smaller, negative indirect effect (β = −0.143). The mediational structure suggested a chain of influence whereby parental psychopathology contributes to a maladaptive family climate and subsequent emotional dysregulation (see Table 3).

3.4. Model interpretation

Fig. 1 illustrates the final model with standardized path coefficients. The analysis confirmed that the risk of suicide in transgender individuals is not attributable to a single factor but to the cumulative and interacting effects of emotional disorders, impulsive traits, and family adversity. While mood disorder emerged as the strongest direct predictor of suicidal behavior, family environment and parental psychopathology contributed indirectly through mediating pathways. These findings align with Brent and Mann's (2006) developmental model, emphasizing the intergenerational and psychosocial transmission of risk for suicidal behavior. However, the model also highlights the particular salience of environmental adversity in transgender individuals, consistent with evidence linking discrimination and victimization to suicidality in this population (Clements-Nolle et al., 2006; Haas et al., 2011).

3.5. Sensitivity analyses

To evaluate the stability and generalizability of the structural model, a series of multi-group analyses were conducted. These analyses examined whether the measurement and structural parameters of the model were invariant across (1) gender identity (trans men vs. trans women) and (2) age groups (≤25 years vs. >25 years). Invariance testing followed standard procedures using a hierarchical sequence of nested models, beginning with configural invariance and progressing to increasingly restrictive equality constraints.

3.5.1. Gender identity

In configural invariance, a baseline model allowing all parameters to vary freely across groups demonstrated adequate fit for both trans men and trans women, indicating that the overall pattern of relationships among variables was similar across groups. This step confirmed that the same conceptual model was appropriate for both subgroups. Next, metric and scalar invariance, factor loadings were constrained to be equal across gender identity groups. The resulting metric model did not differ significantly from the freely estimated model ($\Delta\chi^2$ nonsignificant; p > .10), supporting equality of factor loadings. Scalar invariance was then tested by constraining item intercepts across groups. Again, model fit did not worsen significantly, indicating that constructs were measured equivalently. Next, structural invariance, the structural paths, error variances, and latent variable variances/covariances were constrained to equality across groups. The constrained model showed no significant degradation in fit relative to the unconstrained model (all $\Delta\chi^2$ < critical values; p > .10). None of the individual structural paths differed significantly across groups when tested using Wald χ^2 difference tests. Taken together, these results demonstrate full invariance across gender identity. The affective–familial pathways predicting suicidal behavior operate similarly for trans men and trans women, with no evidence of differential structural associations between risk factors and suicide attempts.

Table 2
Standardized direct effects in the final SEM.

Predictor	β	p
Parent mood disorder → participant mood disorder	0.379	0.023
Parent impulsive aggression → participant impulsive aggression	0.019	0.671
Suboptimal family environment → abuse and neglect	0.318	0.002
Abuse and neglect → participant mood disorder	0.205	0.093
Abuse and neglect → participant impulsive aggression	0.142	0.111
Suboptimal family environment → participant mood disorder	−0.072	0.638
Suboptimal family environment → participant impulsive aggression	0.112	0.156
Participant mood disorder → suicide attempt	0.520	0.001
Participant impulsive aggression → suicide attempt	0.173	0.015
Suboptimal family environment → suicide attempt	0.420	0.001
Life stressors → suicide attempt	−0.049	0.512

Table 3
Standardized indirect effects.

Predictor	β	p
Parent mood disorder → participant suicide attempt	0.117	0.011
Parent impulsive aggression → participant suicide attempt	0.143	0.003
Suboptimal family environment → participant suicide attempt	−0.029	0.011
Abuse and neglect → participant suicide attempt	0.131	0.014

3.5.2. Age group

Parallel invariance tests were conducted for younger (≤25 years) and older (>25 years) participants. In configural and metric invariance, the configural model showed acceptable fit in both age groups, supporting the same underlying factor structure. Equality constraints on factor loadings (metric invariance) did not significantly worsen model fit (p > .10), suggesting that the constructs were interpreted similarly across age groups. Next, scalar and structural invariance, constraining intercepts and structural paths also resulted in no significant decline in model fit relative to the unconstrained model (all p > .10). Wald tests indicated no age-related differences in the strength of associations between mood disorders, impulsive aggression, environmental adversity, and suicide attempts. Finally, variance stability, latent variable variances and residual terms were comparable across age groups, indicating similar levels of variability in affective and environmental predictors. Model invariance across age groups suggests that the same affective and familial mechanisms are relevant across the broad age range represented in the sample. Neither younger nor older participants showed unique structural pathways leading to suicidal behavior.

4. Discussion

This study examined affective, familial, and environmental pathways to suicidal behavior in a large clinical sample of transgender individuals. Using structural equation modeling, we found that mood disorders, impulsive aggression, and environmental adversity were the primary predictors of suicide attempts. These findings align with extensive evidence identifying affective dysregulation as a central mechanism driving suicidal thoughts and behaviors (Franklin et al., 2017; Hawton et al., 2013; Ribeiro et al., 2018) and support contemporary models that emphasize multi-level interactions among genetic, psychological, and contextual risk factors. This is one of the largest clinical SEM studies on transgender suicide risk conducted to date.

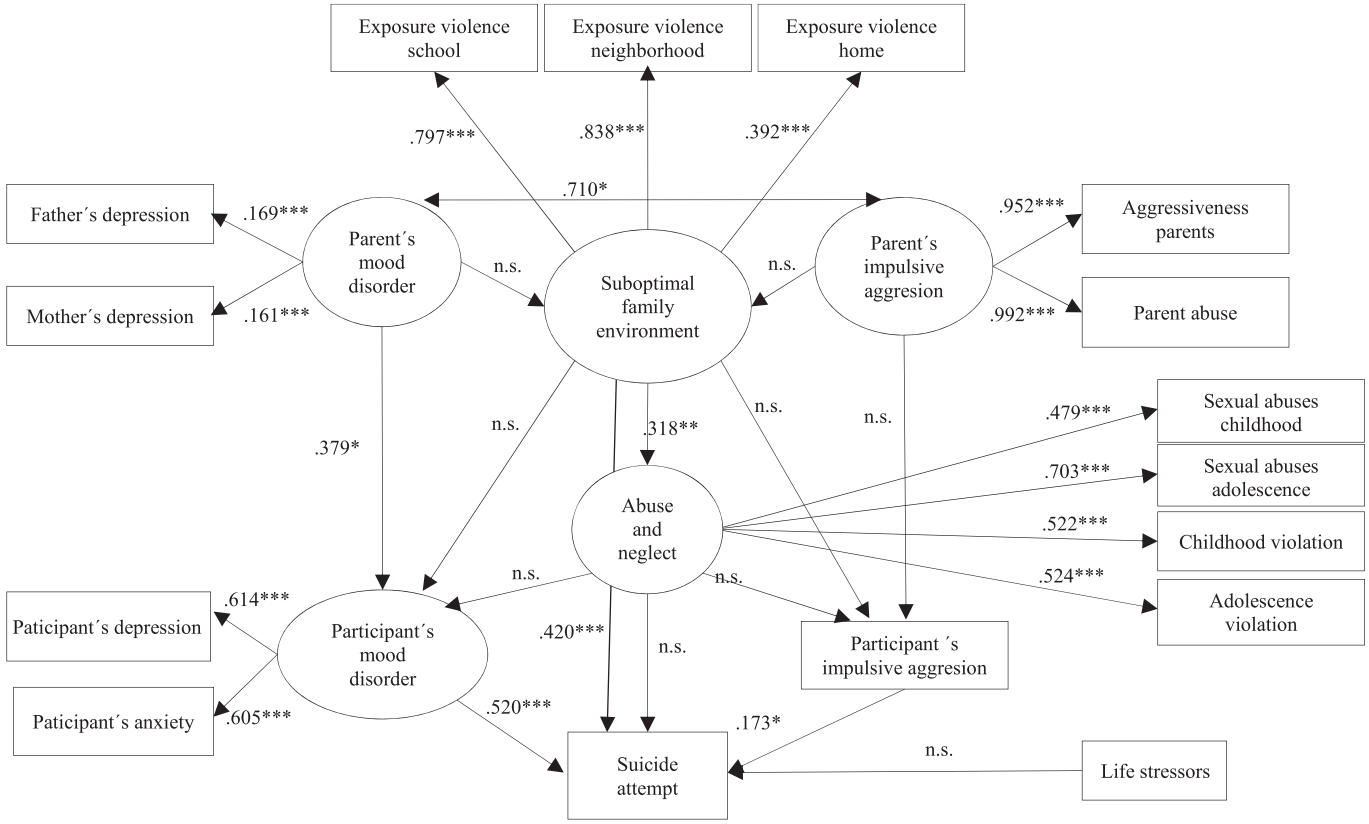

Fig. 1. Structural equation model of suicide risk in transgender individuals.
Note: To be recreated for publication — standardized paths, latent variables as ovals, observed variables as rectangles, significant paths bolded. Model fit: $\chi^2/df = 1.692$; GFI = 0.955; IFI = 0.956; RMSEA = 0.037. Explained variance (R^2): Suicide attempt = 0.48.

4.1. Affective dysregulation as the core mechanism

Mood disorders emerged as the strongest single predictor of suicide attempts. This is consistent with meta-analytic work demonstrating that depression, anxiety, hopelessness, and rumination are among the most reliable clinical correlates of suicidal ideation and behavior (Batterham et al., 2019; Gvion and Apter, 2011). Neurobiological evidence highlights structural and functional abnormalities in the amygdala, anterior cingulate cortex, and ventromedial prefrontal cortex among individuals with mood disorders (Mann and Rizk, 2020; Schmaal et al., 2020). Disturbances in these regions impair top-down regulation of negative affect and intensify emotional reactivity to stressors, thereby increasing risk for suicidal crises.

Our findings are consistent with the notion that mood dysregulation functions as a final common pathway, integrating distal vulnerabilities (e.g., genetic predispositions, family psychopathology) and proximal stressors (e.g., victimization, interpersonal conflict). The mediating role of mood disorders in this study parallels evidence indicating that emotional instability predicts both the onset and persistence of suicidal ideation across psychiatric diagnoses (Bryan et al., 2013). Moreover, affective dysregulation is strongly associated with maladaptive cognitive processes, such as catastrophic thinking, attentional biases to threat, and deficits in problem-solving, which increased risk for suicidal behavior (Miranda et al., 2013; Williams et al., 2005). These mechanisms likely contribute to the pathway identified in our model. Importantly, the present findings should not be interpreted as indicating mechanisms that are specific to transgender identity. Rather, the pathways identified in this study, particularly affective dysregulation, impulsive aggression, and exposure to environmental adversity, represent transdiagnostic processes that have been consistently documented in both clinical and non-clinical populations, including cisgender individuals. The contribution of the present study lies not in proposing transgender-specific mechanisms, but in examining how these well-established processes operate within a transgender clinical sample. This distinction is critical. Transgender individuals are disproportionately exposed to chronic stressors, including social rejection, victimization, and environmental instability, which may increase the prevalence, severity, or interaction of these risk mechanisms. Accordingly, the elevated rates of suicidal behavior observed in this population should not be interpreted as intrinsic to gender identity, but as reflecting increased exposure to psychosocial adversity and minority-related stress. This interpretation is consistent with minority stress frameworks and avoids pathologizing gender diversity while still acknowledging clinically relevant risk processes. Importantly, the present model does not address, nor does it allow inferences regarding, the origins or determinants of transgender identity. The variables included in this study, such as affective dysregulation, familial psychopathology, and exposure to adversity, were examined exclusively as predictors of suicidal behavior within a transgender clinical sample. Given the cross-sectional design and the absence of any variables related to identity development, the findings cannot be interpreted as suggesting that transgender identity is caused by psychological dysregulation, family factors, or traumatic experiences. Such interpretations would extend beyond the scope of the data and are not supported by the present analyses. Instead, the model should be understood as identifying mechanisms that may increase vulnerability to suicidal behavior after gender identity has already been established, particularly in contexts characterized by elevated psychosocial stress. This distinction is essential to avoid misinterpretation and to ensure that the findings are framed within an appropriate clinical and theoretical context. Rather, the identified pathways reflect mechanisms, such as affective dysregulation, impulsive aggression, and exposure to environmental adversity, that are

consistently implicated in suicidal behavior across diverse clinical and non-clinical populations, including cisgender individuals (Franklin et al., 2017; O'Connor and Kirtley, 2018). The elevated risk observed in transgender populations likely reflects increased exposure to stressors and minority-related adversity rather than identity per se. This interpretation aligns with contemporary models emphasizing psychosocial stress processes rather than essentialist explanations.

4.2. Environmental adversity as both direct and indirect contributor

The environmental adversity examined in the present study primarily reflected family and contextual instability rather than direct measurement of minority stress or discrimination. Environmental adversity exerted significant direct and indirect effects on suicidal behavior. The findings suggest that chronic family dysfunction, rather than acute stress exposure, emerged as the more robust contextual predictor of suicide attempts in this sample. This is consistent with longitudinal evidence demonstrating that exposure to violence, discrimination, and chronic interpersonal stress increases vulnerability to both mood disorders and suicidality (Hatzenbuehler, 2009; Liu and Miller, 2014; Turner et al., 2017). Developmental trauma research indicates that chronic exposure to threat disrupts HPA-axis functioning, increases inflammation, alters reward sensitivity, and reshapes brain networks involved in emotion regulation (McLaughlin et al., 2019; Miller et al., 2009; Teicher and Samson, 2016). Such alterations may create enduring neurobiological vulnerability that heightens the probability of suicidal thinking during emotionally intense states. The persistence of a direct effect suggests that adversity also contributes to volitional processes that facilitate suicidal behavior. According to ideation-to-action theories (Klonsky and May, 2015; May and Klonsky, 2016) and Joiner's interpersonal theory (Joiner, 2005), individuals repeatedly exposed to painful or fear-inducing experiences may develop heightened pain tolerance, reduced fear of death, and a sense of acquired capability for suicide (Bryan et al., 2015; Smith et al., 2013). Although this study did not directly measure these constructs, the structural effect of adversity is consistent with these theoretical mechanisms. The role of adversity is further supported by cross-cultural evidence showing that minority stress, stigma, and discrimination are potent determinants of suicidal behavior independent of psychiatric diagnoses (Meyer, 2003; Plöderl and Tremblay, 2015). This situates environmental adversity as a central mechanism, not merely a background variable.

4.3. Impulsive aggression as a volitional moderator of suicidal action

Impulsive aggression significantly predicted suicide attempts above and beyond mood dysregulation. This finding is consistent with research indicating that impulsivity and reactive aggression are key volitional factors that increase the likelihood of acting on suicidal thoughts (Brodsky et al., 2001; Gvion and Apter, 2011; McGirr et al., 2008). Biological studies link impulsive aggression to serotonergic dysregulation, dysconnectivity between limbic and prefrontal regions, and impaired inhibitory control, mechanisms repeatedly associated with suicidal behavior (Asberg et al., 1976; Oquendo et al., 2014; Van Heeringen and Bijttebier, 2005). Within the framework of the IMV model (O'Connor, 2011), impulsive aggression corresponds to volitional moderators that influence whether suicidal ideation is translated into behavior. The significant pathway in our model reinforces the distinction between drivers of ideation (affective dysregulation) and facilitators of action (impulsivity, habituation to pain). The indirect effect of parental impulsive aggression aligns with behavioral genetics findings suggesting that impulsive and aggressive temperaments have moderate heritability and are transmitted across generations through both genetic and environmental mechanisms (Bevilacqua and Goldman, 2013; Hicks et al., 2004; Krueger et al., 2002). These intergenerational processes highlight the relevance of addressing both emotional and behavioral regulation in suicide prevention.

4.4. Parental mood disorders and intergenerational vulnerability

Parental mood disorders indirectly predicted suicide attempts through participants' mood disorders. This result is consistent with longitudinal research showing that offspring of parents with depression or bipolar disorder are at substantially elevated risk for mood dysregulation, internalizing symptoms, and suicidal behavior (Beardslee et al., 2011; Rice et al., 2002; Weissman et al., 2005). Emerging evidence suggests that intergenerational transmission occurs through multiple interacting mechanisms including genetic susceptibility (Hyde et al., 2016), epigenetic alterations linked to stress regulation (Yehuda and Lehrner, 2018), modeling of maladaptive affective responses, and exposure to dysregulated interpersonal climates. Our findings extend prior Spanish clinical research documenting heightened affective symptoms, interpersonal distress, and suicidality among transgender individuals (Guzmán-Parra et al., 2015; Bergero-Miguel et al., 2016), by clarifying that familial psychopathology contributes via mood dysregulation rather than through direct behavioral pathways.

4.5. Childhood maltreatment: mediated rather than direct effects

Childhood abuse and neglect did not independently predict suicide attempts once affective dysregulation and environmental adversity were included. This is consistent with studies showing that maltreatment confers suicide risk primarily through its effects on emotional regulation, cognitive vulnerabilities, and interpersonal sensitivity (Infurna et al., 2016; Liu et al., 2017; Miller et al., 2017). Meta-analytic findings indicate that childhood maltreatment is strongly linked to later depression, anxiety, and emotion regulation deficits (Norman et al., 2012; Teicher and Samson, 2016), suggesting that its effects on suicidality are largely mediated by affective pathways. Our results reinforce this view.

4.6. Integration with theoretical models of suicidal behavior

The pattern of results identified in this study can be meaningfully situated within several contemporary theoretical models of suicidal behavior, each of which provides a distinct but complementary perspective on how vulnerabilities translate into suicidal risk. Mapping the findings onto these frameworks strengthens the explanatory validity of the affective–familial model tested and highlights the multideterminant nature of suicidal behavior. First, the results align closely with the Stress–Diathesis Model (Mann et al., 1999), which proposes that suicidal behavior emerges from the interaction between preexisting vulnerabilities and environmental stressors. In our study, parental mood disorders operate as a diathesis, contributing to inherited or early acquired tendencies toward affective dysregulation. Environmental adversity, including exposure to violence and chronic social threat, functions as a persistent source of stress capable of activating these latent vulnerabilities. The strong mediating role of mood disorders reflects the model's assertion that affective dysregulation represents a proximal mechanism linking stress and diathesis to suicidal behavior. The indirect pathways identified, particularly those connecting familial mood disorders to suicide attempts, provide empirical support for this theoretical structure.

The findings also correspond with the Integrated Motivational–Volitional (IMV) Model (O'Connor, 2011; O'Connor and Kirtley, 2018), which distinguishes between factors that generate suicidal ideation and those that facilitate the transition from ideation to action. Within the motivational phase, our results show that environmental adversity predicts mood dysregulation, consistent with the IMV emphasis on defeat, entrapment, and affective collapse as precursors to suicidal thinking. The volitional phase is reflected in the significant role of impulsive aggression, a behavioral tendency that lowers the threshold

for enacting suicidal behavior. The direct effect of environmental adversity on suicide attempts may similarly relate to volitional moderators such as habituation to threat or diminished fear responses. Taken together, these findings support central IMV propositions regarding the differentiation of ideation drivers and volitional facilitators.

Finally, the results are compatible with the Ideation-to-Action Framework (Klonsky and May, 2015; May and Klonsky, 2016), which argues that the causes of suicidal ideation differ from those of suicidal behavior. Consistent with this distinction, mood disorders and affective dysregulation acted as the primary drivers of ideation, while impulsive aggression and the direct influence of adversity appeared to facilitate the enactment of suicidal behavior. This pattern mirrors extensive evidence that behavioral disinhibition, heightened pain tolerance, and acquired capability play unique roles in differentiating ideators from attempters.

Taken together, the convergence of evidence across these three frameworks underscores the robustness of the affective–familial model identified in this study. Affective dysregulation emerges as the central mechanism integrating familial vulnerability and environmental stress, while volitional factors such as impulsive aggression help explain why certain individuals progress from suicidal ideation to suicidal behavior. This theoretical consistency reinforces confidence in the structural pathways identified and provides a coherent conceptual foundation for clinical interpretation and intervention design.

4.7. Clinical and preventive implications

From a clinical perspective, the findings point to several actionable targets for prevention. First, early detection of mood and anxiety symptoms in transgender individuals is essential. Screening for depressive symptoms in gender identity clinics and primary care settings could allow timely referral to evidence-based psychological treatments. Cognitive-behavioral and dialectical-behavioral interventions have shown promise in reducing suicidal ideation and improving emotional regulation in youth at risk (Linehan et al., 2015). Second, family-based interventions should be prioritized. Given the indirect influence of parental psychopathology on suicidality, psychoeducational programs addressing parental mood disorders, emotional communication, and acceptance of gender identity could mitigate intergenerational risk transmission. Supportive family relationships have been identified as among the strongest protective factors against suicide in transgender adolescents (Ryan et al., 2010). Third, the significant role of environmental adversity underscores the importance of systemic and community-level interventions. Anti-bullying policies, inclusive educational programs, and training for healthcare professionals in gender-affirmative practices may help reduce exposure to violence and discrimination two of the most modifiable contextual risk factors identified in this study. Public health frameworks must therefore consider not only clinical treatment but also societal transformation as an essential component of suicide prevention in transgender populations. The mechanisms identified in this study are not unique to transgender individuals but mirror those documented in broader suicide research. Accordingly, clinical assessment should focus on affective instability and environmental stress exposure rather than assuming inherent vulnerability linked to gender identity.

4.8. Limitations and future directions

Several limitations should be considered when interpreting these findings. First, the cross-sectional design precludes causal inference. Although SEM permits the testing of theoretically driven pathways, temporal precedence and reciprocal influences among affective, familial, and environmental variables cannot be established. Longitudinal studies following transgender individuals across critical developmental or transition-related periods are needed to clarify whether fluctuations in mood dysregulation, impulsive and aggressive tendencies, or family functioning predict subsequent changes in suicidal ideation or behavior. Second, the clinical nature of the sample may limit generalizability. Participants were recruited from a specialized gender identity unit, which may reflect a subgroup with greater access to structured clinical care, diagnostic evaluation, and psychosocial resources compared with community-based transgender populations. Replication in non-clinical, population-based, and cross-cultural samples, particularly in settings with varying levels of healthcare access and minority stress exposure, would strengthen external validity. Third, the reliance on self-report and retrospective clinical interviews may introduce recall or reporting biases, especially for sensitive constructs such as childhood maltreatment or prior suicidal behavior. Future research would benefit from multimethod assessment strategies combining validated psychometric instruments, structured diagnostic interviews, and, where feasible, corroborating information from family members or medical records to enhance reliability and reduce subjectivity. Fourth, preliminary bivariate associations were explored using Pearson correlations for descriptive purposes. Because several study variables were dichotomous, Pearson coefficients may not optimally capture associations between categorical indicators. However, the primary analyses relied on structural equation modeling with appropriate estimation procedures for categorical data, thereby minimizing potential bias in the main findings. Future research could consider alternative correlation metrics (e.g., tetrachoric or polychoric correlations) when exploring associations among categorical variables. Fifth, the present study focused primarily on risk mechanisms and did not assess protective or resilience-promoting factors. Incorporating constructs such as social support, emotion regulation skills, community connectedness, perceived belongingness, and gender affirmation experiences would allow for a more comprehensive and balanced understanding of suicide risk and may help identify buffers that mitigate the impact of adversity and affective vulnerability. The study did not include a direct measure of gender-related discrimination or minority stress. Given the central role of discrimination in theoretical models of transgender mental health (Meyer, 2003), future research should incorporate validated measures of enacted stigma and internalized transphobia to better differentiate identity-related stressors from broader familial and contextual adversity.

Finally, additional work is needed to integrate biological and neurocognitive markers into models of suicidal behavior in transgender populations. Potential avenues include the assessment of impulsivity using behavioral tasks, examination of stress-system biomarkers (e.g., cortisol reactivity), and neuroimaging studies targeting networks involved in emotion regulation and inhibitory control. Such multimodal approaches would help bridge psychological and neurobiological levels of analysis and support the development of more precise, biopsychosocial models of suicide risk tailored to gender-diverse individuals. Despite these limitations, the large sample size and theoretically grounded modeling approach provide a robust platform for future longitudinal research.

5. Conclusions

The present study developed and validated an explanatory structural model of suicide risk in transgender individuals, demonstrating that familial psychopathology, environmental adversity, and emotional vulnerability jointly predict suicidal behavior. These results highlight the intergenerational and contextual nature of suicide, emphasizing that effective prevention must extend beyond individual treatment to include family and community interventions. Early identification of mood disturbances and family-based psychoeducation may reduce suicide vulnerability, while broader social and institutional changes are necessary to mitigate stigma and violence against transgender individuals. Future longitudinal and multimethod research will be critical for deepening our understanding of these complex pathways and for designing integrative, evidence-based prevention programs. Targeting mood dysregulation and environmental adversity simultaneously may

yield the most effective suicide-prevention strategies in transgender populations.

CRediT authorship contribution statement

Nicolás Sánchez-Álvarez: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Informed consent

Participants were informed about the anonymous study. Completion of the study implied agreement to participate in the study.

Compliance with ethical standards

The present research was conducted in compliance with IRB standards.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: reports a relationship with that includes: has patent pending to. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2026.121793.

Data availability

For information regarding access to the data associated with the present study, please contact the author.

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