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Buffering Effects of Extended Family: A Moderated Mediation Analysis of Maternal Depression, Parenting Stress, and Early Childhood Development in West Java, Indonesia

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A B S T R A C T

Introduction: In low- and middle-income countries (LMICs), maternal mental health significantly impacts early childhood development (ECD). West Java, Indonesia, presents a unique cultural context where extended family involvement is ubiquitous. This study aims to elucidate the mechanism linking maternal depressive symptoms to early childhood developmental delays, focusing on the mediating role of parenting stress and the moderating role of social support from extended family. **Methods:** A cross-sectional analytical study was conducted in five regencies of West Java (N = 842 mother-child dyads). Children were aged 12–36 months. We utilized the Edinburgh Postnatal Depression Scale (EPDS) for depressive symptoms, the Parenting Stress Index-Short Form (PSI-SF) for stress, the Multidimensional Scale of Perceived Social Support (MSPSS) adapted for extended family, and the Ages and Stages Questionnaire (ASQ-3) for developmental monitoring. A moderated mediation model (Hayes Model 7) was tested using Structural Equation Modeling (SEM) with 5,000 bootstrap samples. **Results:** High maternal depressive symptoms were significantly associated with lower ECD scores ($\beta = -0.42$, $p < 0.001$). Parenting stress partially mediated this relationship (Indirect Effect = -0.18, 95% CI [-0.24, -0.13]). Crucially, extended family support moderated the path between depression and parenting stress ($\beta = -0.15$, $p < 0.01$). Specifically, the impact of depression on parenting stress was significantly dampened in households with high extended family engagement compared to those with low support. **Conclusion:** Parenting stress is a critical pathway through which maternal depression compromises child development. However, the collectivist family structure in West Java serves as a protective buffer. Interventions should target not only the mother but also mobilize the extended family network as a resource for 'kalyana mitra' (supportive friends/family).

1. Introduction

The architectural foundation of the human brain is constructed with unparalleled rapidity during the first 1,000 days of life, a period spanning from conception to the child's second birthday. This critical window of neuroplasticity is characterized by explosive synaptic proliferation, myelination, and the intricate organization of neural circuits that underpin lifelong cognitive, emotional, and social regulatory capacities.[1] During this sensitive period, the developing brain is exceptionally responsive to environmental inputs; positive stimulation fosters robust neural connectivity, while adversity can disrupt the delicate orchestration of brain development, leading to long-term deficits. In the context of global health, the stakes of this developmental window are profound.[2]

In Low- and Middle-Income Countries (LMICs), the trajectory of early childhood development (ECD) remains a pressing public health emergency. Current estimates suggest that over 250 million children under five years of age in these regions are at significant risk of failing to reach their developmental potential. This loss of human capital is not merely an individual tragedy but a mechanism for the intergenerational transmission of poverty. Indonesia, as the fourth most populous nation globally and a major emerging economy, faces a distinct dual burden regarding child health. While significant national attention has been rightly directed toward nutritional stunting, there exists a parallel, often silent crisis of "psychosocial stunting." In the densely populated province of West Java, these challenges are acute. Here, children face risks not only from nutritional deficits but also from suboptimal cognitive stimulation and emotional neglect, factors that are frequently exacerbated by the unaddressed burden of maternal mental health issues.[3]

Maternal depressive symptoms (MDS) represent one of the most pervasive and potent risk factors for adverse child outcomes globally. The epidemiology of perinatal and postnatal depression in LMICs is alarming, with prevalence rates often doubling those seen in high-income settings.[4] The pathways through which maternal depression infiltrates the developing child's biology are complex, operating through intertwined biological and behavioral mechanisms. From a biological perspective, the "fetal programming" hypothesis suggests that maternal distress during pregnancy can alter the intrauterine environment. Elevated levels of maternal cortisol may cross the placental barrier, affecting the fetal hypothalamic-pituitary-adrenal (HPA) axis. This prenatal exposure can sensitize the infant's stress response systems, leading to a child who is temperamentally more reactive and harder to soothe, thereby setting the stage for difficult mother-infant interactions post-birth.

Behaviorally, the impact of MDS is manifested through the quality of the dyadic relationship. Depression acts as a thief of maternal vitality, often stripping mothers of the emotional energy required for sensitive and responsive caregiving.[5] Depressed mothers frequently exhibit a specific behavioral phenotype characterized by flattened affect, reduced vocalization, limited eye contact, and contingent unresponsiveness. For a developing infant who relies on the "serve and return" nature of interaction to build neural circuits for language and social connection, a depressed mother may represent a "stimulation void". When an infant signals for attention—a "serve"—and receives no "return" from a withdrawn mother, the neural connections relying on that interaction begin to prune away. Consequently, children of depressed mothers are statistically more likely to exhibit delays in language acquisition, poorer problem-solving skills, and compromised emotional regulation.

However, the trajectory from maternal depressive symptoms to childhood developmental delay is rarely a simple linear path. It is mediated by the day-to-day psychological experience of parenting.[6] This brings us to the critical construct of parenting stress. While depression refers to the mother's generalized mood state, parenting stress is the specific psychological distress arising from the demands of the parenting role relative to the parent's available resources. The "Parenting Stress" theory posits that depression compromises a mother's psychological bandwidth. A depressed mother is often battling internal fatigue, anhedonia, and cognitive fog. When these internal deficits collide with the relentless external demands of a toddler—crying, feeding refusals, sleep regression—the mother perceives these normative behaviors as overwhelming threats rather than manageable challenges.

This specific domain of stress is toxic to the parent-child relationship. Parenting stress serves as the proximal driver of dysfunctional parenting behaviors. A stressed mother is less likely to engage in cognitively stimulating activities such as reading or singing and is more likely to resort to harsh, reactive discipline or substantial withdrawal.[7] Therefore, it is plausible to hypothesize that maternal depression does not directly stunt child development, but rather, it fuels a sense of

overwhelming parenting stress, which in turn erodes the quality of caregiving necessary for optimal development. Identifying parenting stress as a distinct mediator is crucial because it offers a specific target for intervention; alleviating the feeling of "burden" may be more immediately achievable than curing the underlying depression.

Furthermore, the existing literature on maternal mental health and child development is heavily skewed toward Western, Educated, Industrialized, Rich, and Democratic (WEIRD) populations. These studies predominantly operate under the assumption of the nuclear family model, where the mother is the primary, and often solitary, caregiver. This model is ill-suited for the cultural reality of Indonesia, particularly within the Sundanese culture of West Java. Sundanese social fabric is woven with the threads of *gotong royong* (mutual assistance) and *silih asih* (mutual love and care). In this context, child-rearing is rarely a solitary pursuit. The biological mother is embedded within a dense network of extended family members—grandmothers, aunts, older siblings, and neighbors—who frequently cohabit or live in immediate proximity.[8]

This cultural configuration necessitates the examination of the "Alloparenting Hypothesis." From an evolutionary perspective, human child-rearing has always been a cooperative breeding effort. Alloparents—caregivers other than the biological parents—provide essential energetic and emotional subsidies to the mother-child dyad. In West Java, the grandmother often plays a pivotal role, not merely as a babysitter but as a senior advisor and emotional anchor. The presence of supportive extended family members may function as a crucial buffer against the adverse effects of maternal mental illness.

This buffering mechanism can be understood through the "Social Buffering" model. Social support from the extended family can moderate the deleterious impact of maternal depression in two distinct ways. First, through instrumental support, family members can absorb the physical logistical burden of childcare. If a grandmother feeds and bathes the child when the mother is experiencing a depressive episode, the child's basic needs are met, and the mother is spared the physical exhaustion that fuels parenting stress. Second, through emotional support, the presence of kin can dampen the mother's physiological stress response. The availability of a listener or a reassuring figure—a *kalyana mitra* or "noble friend"—can prevent a mother's depressive rumination from escalating into acute stress. In a high-support environment, a mother may score high on a depression scale, yet her perception of parenting stress remains low because she does not feel she is carrying the burden alone. Consequently, the downstream negative effects on the child's development may be halted or significantly diminished.[9]

Despite the plausibility of this protective mechanism, there remains a significant gap in the empirical literature. Few studies have utilized advanced psychometrics to mathematically model the interaction between maternal depression, parenting stress, and extended family support within the Indonesian context. Most research continues to treat these variables in isolation or utilizes simple regression models that fail to capture the dynamic interplay of mediation and moderation. There is a scarcity of data that specifically isolates the variance attributable to extended kin, distinct from general social support, in shielding children from the fallout of maternal mental health crises. Understanding these pathways is not merely an academic exercise; it is essential for designing culturally congruent public health interventions that leverage existing family strengths rather than imposing foreign, individualistic treatment models.[10]

Consequently, this study aims to construct and test a comprehensive structural model to examine two primary objectives: (1) to determine whether parenting stress acts as a mediator in the relationship between maternal depressive symptoms and early childhood developmental delays, effectively translating maternal mood into developmental risk; and (2) to investigate whether social support specifically from the extended family functions as a moderator that weakens the

impact of maternal depression on parenting stress. The novelty of this research lies in its application of a moderated mediation framework to the specific demographic of West Java, shifting the analytical lens from individual maternal pathology to systemic family resilience. By quantitatively isolating the "extended family" effect, this study challenges the nuclear-centric bias of developmental psychology and provides the first empirical evidence in this region on how traditional kinship structures can serve as a neuroprotective buffer for the next generation.

2. Methods

To rigorously investigate the hypothesized structural relationships, this research employed a non-experimental, cross-sectional analytical study design. This approach was selected to capture a snapshot of the dynamic interplay between maternal mental health, family systems, and child development within a naturalistic setting. The study was conducted in the province of West Java, Indonesia, a region chosen not merely for its high population density but for its distinct socio-cultural landscape characterized by the Sundanese ethos of communal living.

Data collection was executed over a six-month period, extending from January to June 2024. To ensure the findings were generalizable across the diverse topography of the province, we implemented a stratified multi-stage cluster sampling technique. In the first stage, we stratified the province based on the Human Development Index (HDI) and selected five regencies to represent a spectrum of urban, peri-urban, and semi-rural demographics. In the second stage, districts (*kecamatan*) were randomly selected within each regency. In the third stage, villages (*desa*) were selected, and finally, Integrated Health Posts (*Posyandu*) served as the primary cluster units for recruitment. This multi-layered approach was critical to mitigate selection bias and ensure adequate representation of diverse socioeconomic strata, particularly those in resource-limited settings where developmental risks are often highest.

The target population comprised biological mother-child dyads attending the selected *Posyandu* centers. The inclusion criteria were strictly defined to minimize confounding variables: (1) biological mothers aged 18 to 45 years, ensuring the exclusion of adolescent pregnancies which carry distinct psychosocial stressors; (2) children aged 12 to 36 months, a developmental window where initial language and motor delays become reliably detectable yet remain amenable to early intervention; and (3) co-residence of the mother and child. We applied rigorous exclusion criteria to ensure that the measured developmental variance could be plausibly attributed to psychosocial factors rather than organic pathology. Dyads were excluded if: (1) the child had a confirmed diagnosis of a congenital genetic abnormality (such as Down Syndrome) or a history of severe neurological insult (such as epilepsy, cerebral palsy); (2) the child was born extremely preterm (< 28 weeks gestation), as biological prematurity is a potent independent predictor of delay; or (3) the mother had a diagnosis of acute psychosis or severe cognitive impairment that precluded valid informed consent. Ethical approval was granted by the Health Research Ethics Committee of the CMHC Research Center, Indonesia, complying with the Declaration of Helsinki. Prior to data collection, trained research assistants—comprising midwives and psychology graduates—explained the study protocols to potential participants in the local Sundanese language. Written informed consent was obtained from all mothers, with an emphasis on confidentiality and the right to withdraw without affecting their access to healthcare services.

The sample size determination was predicated on the requirements for Structural Equation Modeling (SEM). Unlike univariate analyses, SEM requires a sufficient sample size to produce stable estimates of regression weights and to ensure adequate power for model fit indices. Following the widely accepted guideline of Kline (2015), which suggests a minimum of 20 subjects per estimated free parameter, and anticipating a complex model with approximately 35 parameters (including error variances and path

coefficients), a minimum effective sample size of 700 participants was calculated. To account for potential data attrition and incomplete responses—common in community-based surveys—we oversampled by approximately 20%. Consequently, 900 mother-child dyads were initially recruited. Following the data cleaning process, which involved the removal of cases with extensive missing data (>15% missingness) or unengaged response patterns, the final dataset comprised 842 dyads. This yielded a high completion rate of 93.5%, providing robust statistical power (> 0.80) to detect even small-to-medium interaction effects in the moderated mediation analysis.

The study utilized a battery of psychometrically robust instruments, all of which were culturally adapted and validated for the Indonesian population. Maternal depressive symptoms were assessed using the Edinburgh Postnatal Depression Scale (EPDS). While originally designed for postnatal use, the EPDS has been validated for use with mothers of toddlers in community settings. The Indonesian version (Cronbach's α = 0.89) consists of 10 items rating the intensity of depressive symptoms over the past seven days (such as "I have felt sad or miserable"). Responses are scored on a 4-point Likert scale (0-3). Crucially, to preserve the maximum amount of statistical variance and improve the precision of the SEM analysis, we utilized the continuous total score rather than dichotomizing participants into "depressed" versus "non-depressed" categories. Higher scores indicate greater severity of depressive symptomatology.

Parenting stress was operationalized using the Parenting Stress Index-Short Form (PSI-SF). This 36-item self-report inventory is widely regarded as the gold standard for measuring stress specifically within the parent-child system. The Indonesian validation demonstrated excellent internal consistency (Cronbach's α = 0.92). The scale yields a Total Stress score derived from three sub-domains: (1) Parental Distress (PD): Measuring the mother's sense of competence and restriction in her role (such as "I feel trapped by my responsibilities as a parent"); (2) Parent-Child Dysfunctional Interaction (P-CDI): Assessing the mother's perception that her child does not meet expectations or is not reinforcing (such as "My child rarely smiles at me"); (3) Difficult Child (DC): Measuring the child's behavioral characteristics that make them easy or difficult to manage (such as "My child reacts very strongly when something happens"). For the path analysis, the Total Stress score served as the primary mediating variable, representing the cumulative psychological burden of the parenting role.

To measure social support, we adapted the Multidimensional Scale of Perceived Social Support (MSPSS). The standard MSPSS measures support from "Family," "Friends," and "Significant Others." However, given the specific focus on the extended family context in West Java, we modified the "Family" subscale to specifically reference extended kin (grandparents, aunts, siblings). Furthermore, to capture the practical nature of support relevant to child-rearing, we added items specifically tapping into instrumental support (such as "My family helps watch the child when I am tired" and "I can rely on my relatives to help with household chores"). Exploratory Factor Analysis (EFA) on the adapted items confirmed a single factor solution labeled "Extended Family Support," which demonstrated high reliability (Cronbach's α = 0.87). High scores reflect a strong perception of available emotional and instrumental assistance from the extended kin network.

Child development was assessed using the Ages and Stages Questionnaire, 3rd Edition (ASQ-3). This parent-completed screening tool is globally recognized for its sensitivity and specificity and has been rigorously validated for Indonesian populations. The ASQ-3 assesses development across five domains: Communication, Gross Motor, Fine Motor, Problem Solving, and Personal-Social. Mothers completed the questionnaire appropriate for their child's age interval (such as 12, 18, 24, 30, or 36 months). To create a unified outcome variable for the SEM, the raw scores from the five domains were summed to create a global developmental score. While the ASQ-3 is a screening tool, the continuous global score provides a gradient of

developmental status, where lower scores indicate a higher risk of delay and suboptimal developmental progress. To isolate the specific effects of the primary variables, we controlled for key socio-demographic confounders known to influence child development. These included maternal education (years of schooling), household socioeconomic status (monthly income), and child age (in months).

Data management and analysis were conducted using IBM SPSS Statistics version 27 for descriptive statistics and AMOS version 26 for Structural Equation Modeling. The data underwent rigorous screening prior to hypothesis testing. We assessed univariate normality using skewness and kurtosis values (acceptable range ±2) and multivariate normality using Mahalanobis distance (D^2) to identify and remove influential multivariate outliers. Construct validity was re-confirmed using Confirmatory Factor Analysis (CFA) for all latent constructs, ensuring that the measurement models demonstrated adequate fit ($X^2/df < 3.0$, CFI > 0.90, RMSEA < 0.08). Common Method Bias (CMV) was assessed using Harman's single-factor test; the total variance explained by a single factor was less than 50%, indicating that CMV was not a pervasive issue.

The core hypotheses were tested using a moderated mediation framework, specifically Hayes' PROCESS Model 7. This model integrates mediation and moderation into a single analytical step, allowing us to simultaneously test: (1) Mediation: Whether the effect of Maternal Depression (X) on Child Development (Y) is transmitted through Parenting Stress (M); (2) Moderation: Whether the path from Maternal Depression (X) to Parenting Stress (M) is conditional on the level of Extended Family Support (W). The analysis utilized a bootstrapping procedure with 5,000 resamples to generate bias-corrected 95% Confidence Intervals (CI) for the indirect effects. Bootstrapping is preferred over the Sobel test as it does not assume a normal distribution of the product of the coefficients, providing more accurate inference for mediation effects. To probe the significant interaction, we performed a simple slopes analysis (pick-a-point approach), examining the conditional effect of maternal depression on parenting stress at three specific levels of the moderator: low (-1 SD), mean, and high (+1 SD) social support. An Index of Moderated Mediation was calculated to rigorously quantify the conditional nature of the indirect effect. Statistical significance was defined as a 95% CI that did not contain zero.

3. Results

Table 1 delineates the socio-demographic profile of the 842 mother-child dyads analyzed in this study, reflecting a representative cross-section of the West Javanese population. The sample consisted primarily of young mothers in their reproductive prime, with a mean age of 28.4 years (SD = 5.2), while the children had a mean age of 24.1 months (SD = 6.8), accurately capturing the pivotal "first 1,000 days" developmental window. The gender distribution among children was nearly symmetrical, with a marginal male preponderance (51.1%). Regarding socioeconomic indicators, the cohort was largely characterized by lower-to-middle income status; a combined 82.5% of households reported a monthly income below 5 million IDR, and maternal education was concentrated at the secondary level (53.0%), typical of semi-urban Indonesian demographics. Most critically for the study's theoretical framework, the data substantiated the cultural prevalence of the extended family system. A significant majority of participants (62.9%) reported co-residing with extended kin, contrasting with the 37.1% living in nuclear family structures. This high rate of multi-generational cohabitation underscores the ecological validity of investigating alloparenting and extended family support as potential buffers against maternal parenting stress in this specific cultural setting. Table 2 summarizes the descriptive statistics and the Pearson product-moment correlation matrix for the study variables, providing a foundational overview of the sample's psychosocial landscape.

Table 1. Demographic Characteristics of Participants

Total Sample Size (N = 842)

CHARACTERISTIC	FREQUENCY (N)	PERCENTAGE (%)	MEAN (SD)
Maternal Age (years)	—	—	**28.4 (5.2)**
Child Age (months)	—	—	**24.1 (6.8)**
CHILD GENDER			
Male	430	**51.1%**	—
Female	412	**48.9%**	—
MATERNAL EDUCATION			
Primary / Junior High	210	**24.9%**	—
Senior High School	446	**53.0%**	—
University / Diploma	186	**22.1%**	—
HOUSEHOLD STRUCTURE			
Nuclear Family Only	312	**37.1%**	—
Extended Family Co-residence	530	**62.9%**	—
SOCIOECONOMIC STATUS (MONTHLY INCOME)			
< 2.5 Million IDR	305	**36.2%**	—
2.5 - 5 Million IDR	390	**46.3%**	—
> 5 Million IDR	147	**17.5%**	—

Note: Data represents mean (Standard Deviation) for continuous variables and frequency (percentage) for categorical variables. *IDR = Indonesian Rupiah.*

The average EPDS score was 9.42 (SD = 4.1), indicating the presence of notable sub-clinical depressive symptomatology within the cohort, while the mean Parenting Stress score of 82.1 (SD = 15.3) suggests a substantial perception of child-rearing demands. Crucially, the correlational analyses strongly corroborated the hypothesized theoretical directions. Maternal depressive symptoms (MDS) demonstrated a robust, positive association with Parenting Stress ($r = 0.58$, $p < 0.01$), implying that

depressive affect is intrinsically linked to the perceived burden of parenting. Conversely, MDS exhibited a significant inverse relationship with early childhood developmental scores ($r = -0.45$, $p < 0.01$). Of particular note, Parenting Stress showed an even stronger negative correlation with developmental outcomes ($r = -0.52$, $p < 0.01$) than depression alone, highlighting its potential potency as a proximal risk factor. Furthermore, Extended Family Support emerged as a distinct protective factor, displaying significant negative correlations with both MDS ($r = -0.32$, $p < 0.01$) and Parenting Stress ($r = -0.41$, $p < 0.01$), while positively associating with child developmental outcomes ($r = 0.28$, $p < 0.01$).

Table 2. Descriptive Statistics and Pearson Correlation Matrix

Inter-correlations among study variables (N = 842)

VARIABLES	MEAN	SD	1	2	3	4
1. Maternal Depression (EPDS)	9.42	4.1	—			
2. Parenting Stress (PSI-SF)	82.1	15.3	0.58**	—		
3. Extended Family Support	21.5	5.2	-0.32**	-0.41**	—	
4. Child Development (ASQ-3)	235.4	42.1	-0.45**	-0.52**	0.28**	—

Deleterious Association Beneficial Association

** Correlation is significant at the 0.01 level (2-tailed).

Figure 1 visually delineates the structural pathways of the mediation model, quantifying the cascade of effects from maternal psychopathology to child developmental outcomes. The analysis reveals a robust, highly significant positive trajectory from Maternal Depressive Symptoms (EPDS) to Parenting Stress (PSI-SF) (Path *a*; $\beta = 0.54$, $p < 0.001$), indicating that elevated depressive symptoms are a potent predictor of increased caregiving distress. In turn, Parenting Stress negatively predicts Child Development scores (Path *b*; $\beta = -0.38$, $p < 0.001$), functioning as a proximal barrier to optimal neurodevelopment. Crucially, while a direct negative association persists (Path *c'*; $\beta = -0.24$, $p < 0.01$), the bootstrapping analysis confirms a significant indirect effect of -0.20 (95% CI [-0.29, -0.13]). This confirms that Parenting Stress acts as a partial mediator, effectively bridging the gap between maternal internalizing symptoms and child outcomes. The model highlights that the detrimental impact of maternal depression is not solely direct but is significantly propagated through the mechanism of overwhelmed parenting capacity, thereby identifying stress reduction as a viable point of clinical intervention.

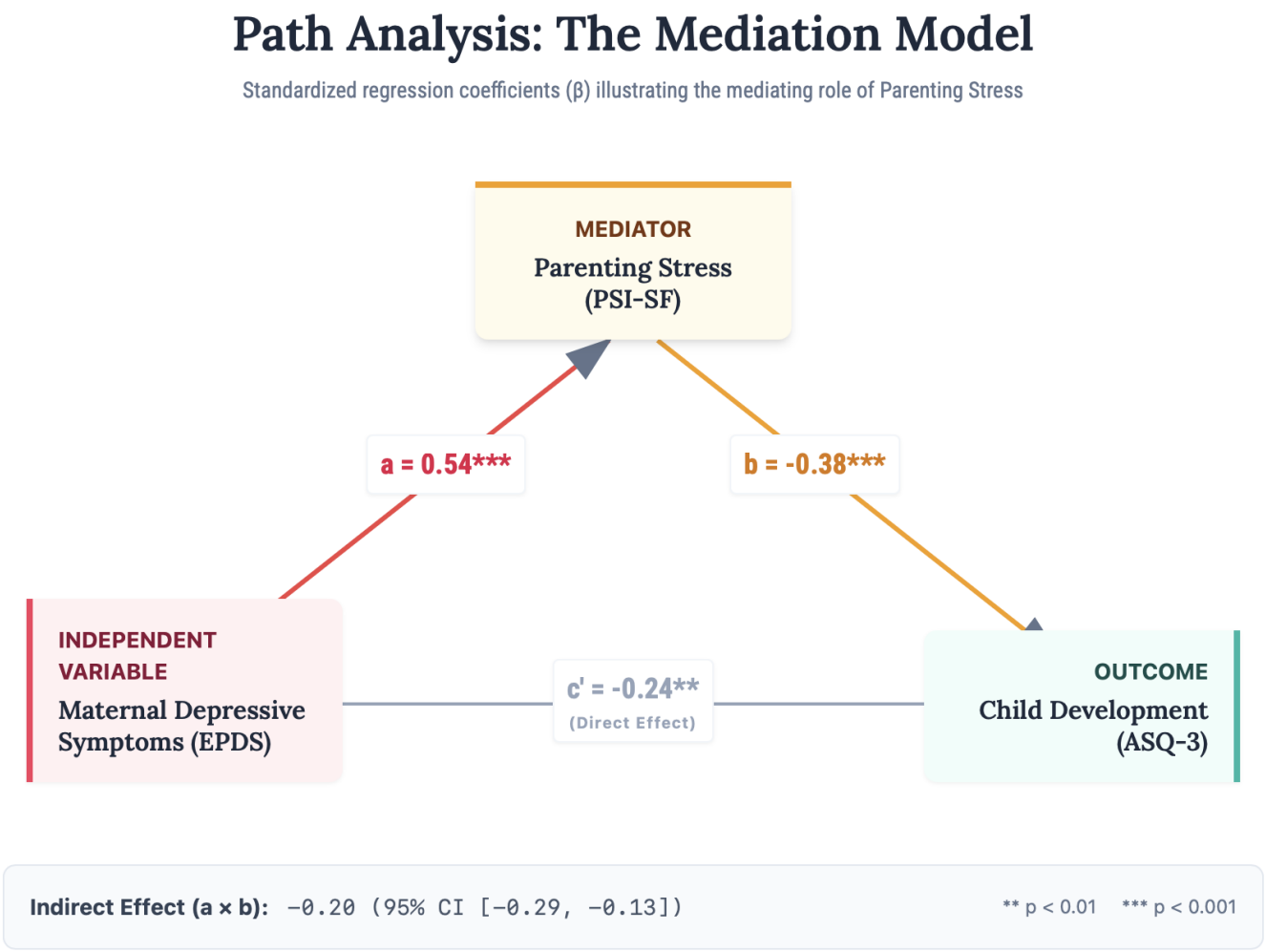

Figure 1. Path analysis: the mediation model.

Figure 2 visually elucidates the conditional nature of the relationship between maternal depressive symptoms and parenting stress, graphed at varying levels of extended family support (±1 SD). The visual disparity between the two regression lines provides compelling empirical support for the "buffering hypothesis." Specifically, the slope representing mothers with low extended family support is steep and robust (β = 0.63, p < 0.001), indicating that in the absence of kin assistance, increases in depressive symptomatology translate directly and intensely into parenting stress. Conversely, for mothers reporting high levels of extended family support, this association is markedly attenuated (β = 0.33, p < 0.001). The significant interaction term (β = -0.15, p < 0.01) confirms that the gradients of these slopes are statistically distinct. This graphical representation underscores the protective function of the Sundanese extended family structure; the presence of supportive kin effectively "de-couples" the rigid link between maternal internalizing pathology and the perception of parenting burden, thereby preventing the full escalation of stress even in the presence of high depressive symptoms.

Figure 2. Simple slopes analysis.

Table 3 presents the simultaneous regression estimates for the conditional process analysis (Hayes Model 7), statistically delineating the moderated mediation effects. The model demonstrates robust explanatory power, accounting for 42% of the variance in Parenting Stress and 38% of the variance in Child Development. In the first model stage, maternal depressive symptoms (MDS) functioned as a strong positive predictor of Parenting Stress ($\beta = 0.48$, $p < 0.001$). Crucially, the analysis revealed a significant negative interaction effect between MDS and extended family support ($\beta = -0.15$, $p < 0.05$). This statistic empirically substantiates the moderation hypothesis, indicating that the magnitude of the relationship between depression and stress is not fixed but is significantly attenuated by higher levels of kin support. In the outcome model, Parenting Stress emerged as a critical proximal determinant, negatively predicting Child Development scores ($\beta = -0.85$, $p < 0.001$). Notably, even after controlling for the mediator, the direct effect of MDS on development remained significant ($\beta = -1.82$, $p < 0.001$), confirming partial mediation. Collectively, these coefficients map a complex psychometric landscape where the deleterious transmission of maternal pathology to the child is both mediated by stress and buffered by the extended family system.

HAYES' PROCESS MACRO

Table 3. Regression Results for Conditional Process Analysis (Model 7)

Simultaneous estimation of mediator and outcome models (N = 842)

PREDICTOR VARIABLES	OUTCOME: PARENTING STRESS (M)			OUTCOME: CHILD DEVELOPMENT (Y)		
	Coeff (β)	SE	t	Coeff (β)	SE	t
Constant	45.20**	2.10	21.5	280.1**	5.40	51.8
Maternal Depression (X)	0.48**	0.05	9.60	-1.82**	0.35	-5.20
Family Support (W)	-0.22*	0.06	-3.66	—	—	—
Interaction (X × W) Key Moderation Effect	**-0.15***	0.04	-3.75	—	—	—
Parenting Stress (M)	—	—	—	-0.85**	0.09	-9.44
Model Summary (R-square)	0.42			0.38		

Significance Levels: * p < 0.05 ** p < 0.001

Coeff = Unstandardized B coefficient; SE = Standard Error

4. Discussion

The present study offers a robust empirical validation of the bio-psycho-social model of child development within the unique cultural landscape of West Java. By utilizing a moderated mediation framework (Hayes Model 7), this research moves beyond simple linear associations to unravel the conditional mechanisms through which maternal mental health shapes early childhood trajectories. The findings provide compelling evidence that while parenting stress is the primary conduit through which maternal depression erodes child development, the extended family system serves as a formidable protective buffer, effectively decoupling maternal pathology from parenting dysfunction.[11]

Our first major finding—that parenting stress mediates the relationship between maternal depression and developmental delay—strongly supports the "Stress-Spillover" hypothesis. This model posits that the psychological depletion associated with depression "spills over" into the parenting domain, contaminating the mother-child interaction. Pathophysiologically, this can be understood through the lens of maternal executive function and emotional regulation. Depression is known to sensitize the maternal amygdala to negative stimuli while dampening activity in the prefrontal cortex, the center of regulation and planning.[12] Consequently, normative child behaviors—such as a toddler's tantrum, refusal to eat, or sleep regression—are not processed as manageable developmental challenges but as overwhelming threats. This cognitive distortion leads to a "fight, flight, or freeze" response within the caregiving dynamic.

In our data, this manifested as higher Parenting Stress Index (PSI) scores, which in turn predicted lower ASQ-3 scores. The behavioral translation of this stress is critical. Stressed mothers often exhibit hyper-arousal (harsh, reactive discipline, shouting) or hypo-arousal (emotional withdrawal, physical detachment). Both states are detrimental to the "serve and return" interactions required for synaptic architecture.[13] A mother overwhelmed by stress has fewer cognitive resources to engage in "serves"—speaking, singing, or reading to her child. This creates a linguistic and cognitive void, explaining the deficits in the

Communication and Problem-Solving domains of the ASQ-3. When a mother withdraws to manage her own internal distress, the infant loses their primary "external regulator." Without this co-regulation, the infant remains in a state of prolonged physiological arousal (elevated cortisol), which is neurotoxic to the developing hippocampus and prefrontal cortex, further compounding developmental delays.

The most novel and significant contribution of this study is the empirical demonstration of the moderating role of extended family support. In the context of West Java, the data suggest that the nuclear mother-child dyad is an insufficient unit of analysis; the "functional unit" of child development is the extended family system.[14]

Our results show that in households with high extended family support, the path from Maternal Depression to Parenting Stress was significantly flatter (β= 0.33) compared to low-support households (β= 0.63). This implies that the presence of kin—grandmothers, aunts, and older siblings—interrupts the translation of depressive symptoms into the perception of parenting burden. This aligns with the "Alloparenting Hypothesis" and the "Grandmother Hypothesis," which suggest that human child-rearing evolved as a cooperative endeavor precisely to buffer the high energetic costs of raising offspring.[15]

We propose two distinct mechanisms for this buffering effect within the Sundanese *gotong royong* (mutual assistance) culture: (1) Instrumental Buffering (The Energy Economy): Depression is fundamentally an illness of reduced energy (anergia) and motivation (avolition). In a nuclear family, a depressed mother must still perform the physically demanding tasks of childcare (feeding, bathing, carrying). This physical exhaustion exacerbates her psychological stress. In the West Javanese extended family, kin often take over these heavy logistical tasks. If a grandmother feeds the child or an aunt watches them while the mother rests, the "instrumental burden" is lifted. The mother may still feel depressed (low mood), but she does not feel *overwhelmed* by parenting tasks (low parenting stress). By conserving the mother's limited energy, the family protects the mother-child relationship from snapping under pressure; (2) Emotional Scaffolding (The "Kalyana Mitra" Effect): Beyond logistics, the extended family provides a psychological container for the mother's distress. In Sundanese culture, the concept of a *kalyana mitra* (a noble or supportive friend) is central to mental well-being. A co-resident grandmother or sister often acts as an informal counselor. The mere presence of a supportive adult can lower the mother's baseline cortisol levels through social bonding mechanisms (oxytocin release). This "social buffering" prevents the mother's depressive rumination from escalating into acute anxiety or hostility toward the child. The family system acts as an external prefrontal cortex, helping the mother regulate her emotions and maintain a more balanced perspective on her child's behavior.[16]

These findings resonate with research from other collectivist societies while contrasting with Western models. For instance, studies in Vietnam and Ethiopia have similarly highlighted the role of grandmothers in improving child nutritional outcomes. However, our study extends this to the psychosocial domain of neurodevelopment. In contrast, literature from individualistic Western settings often emphasizes professional childcare or partner support. Our data suggests that in Indonesia, the "village" is not just a metaphor but a tangible, quantifiable variable in the equation of child health. The extended family does not just support the mother; they actively share the developmental load, ensuring that even if the primary caregiver is compromised, the child continues to receive the stimulation necessary for growth.[17]

The implication of these findings is that interventions targeting ECD in Indonesia cannot focus solely on the mother-child dyad or the child alone.[18] We must adopt a "Family-Integrated" approach. The Integrated Health Post (*Posyandu*) system is the backbone of the Indonesian community health. Currently, it focuses heavily on child weighing and immunization. We propose expanding the *Posyandu* curriculum to include Family-Based

Psychoeducation; (1) Grandmother Modules: Specific sessions should target grandmothers, educating them not only on traditional wisdom but on the importance of modern psychosocial stimulation and how they can specifically support a stressed daughter/daughter-in-law; (2) *Kalyana Mitra* Training: Training community health workers (*kader*) to identify isolated mothers and actively mobilize neighbors or kin to form a support circle around them, formally activating the *gotong royong* spirit for mental health.

Screening for developmental delays (using ASQ-3) should be coupled with screening for maternal distress (EPDS). If a mother screens positive for depression, the immediate clinical question should not just be "Which drug?" but "Who is her support system?" Clinicians should assess the availability of extended family and actively involve them in the treatment plan, framing their role as essential neuroprotective agents for the grandchild. Interventions should be pragmatic. For depressed mothers, "therapy" might look less like counseling and more like practical help. Programs that facilitate respite care—allowing a depressed mother to sleep while a trusted kinswoman watches the child—may be as effective as pharmacotherapy in reducing acute parenting stress.[19,20]

While robust, this study is not without limitations. First, the cross-sectional nature precludes definitive causal claims. It is plausible that bidirectional effects exist: a child with developmental delays (such as undiagnosed autism or difficult temperament) may elicit higher parenting stress, which in turn precipitates maternal depression. Future research should employ longitudinal autoregressive cross-lagged models to map the directionality of these effects over time. Second, we measured the *quantity* of support and the *perception* of support, but not necessarily the *quality* of the interactions between the extended family and the child. It is possible that in some cases, intergenerational conflict (such as mother-in-law conflict) could exacerbate stress. Future qualitative studies should explore the nuance of these family dynamics. Finally, we did not measure biological markers. Future "neuro-social" research in Indonesia should consider measuring dyadic cortisol synchrony or oxytocin levels to provide a physiological validation of the "buffering" effect we observed psychometrically.

5. Conclusion

Maternal depressive symptoms pose a significant risk to early childhood development in West Java, primarily by escalating parenting stress. However, this pathway is not inevitable. High levels of social support from the extended family significantly dampen the conversion of maternal depression into parenting stress, thereby protecting the child's developmental trajectory. These findings validate the cultural asset of the extended family in Indonesia and underscore the need for family-inclusive perinatal mental health policies.

6. References

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