

Basic Environmental Supports for Positive Brain and Cognitive Development in the First Year of Life

Joan L. Luby, MD; Max P. Herzberg, PhD; Caroline Hoyniak, PhD; Rebecca Tillman, MS; Rachel E. Lean, PhD; Rebecca Brady, PhD; Regina Triplett, MD; Dimitrios Alexopoulos, MS; David Loseille, BS; Tara Smyser, MS; Cynthia E. Rogers, MD; Barbara Warner, MD; Christopher D. Smyser, MD; Deanna M. Barch, PhD

[+ Supplemental content](#)

IMPORTANCE Defining basic psychosocial resources to facilitate thriving in the first year of life could tangibly inform policy and enhance child development worldwide.

OBJECTIVE To determine if key environmental supports measured as a thrive factor (T-factor) in the first year of life positively impact brain, cognitive, and socioemotional outcomes through age 3.

DESIGN, SETTING, AND PARTICIPANTS This prospective longitudinal cohort study took place at a Midwestern academic medical center from 2017 through 2022. Participants included singleton offspring oversampled for those facing poverty, without birth complications, congenital anomalies, or in utero substance exposures (except cigarettes and marijuana) ascertained prenatally and followed up prospectively for the first 3 years of life. Data were analyzed from March 9, 2023, through January 3, 2024.

EXPOSURES Varying levels of prenatal social disadvantage advantage and a T-factor composed of environmental stimulation, nutrition, neighborhood safety, positive caregiving, and child sleep.

MAIN OUTCOMES & MEASURES Gray and white matter brain volumes and cortical folding at ages 2 and 3 years, cognitive and language abilities at age 3 years measured by the Bayley-III, and internalizing and externalizing symptoms at age 2 years measured by the Infant-Toddler Social and Emotional Assessment.

RESULTS The T-factor was positively associated with child cognitive abilities ($\beta = 0.33$; 95% CI, 0.14-0.52), controlling key variables including prenatal social disadvantage (PSD) and maternal cognitive abilities. The T-factor was associated with child language ($\beta = 0.36$; 95% CI, 0.24-0.49), but not after covarying for PSD. The association of the T-factor with child cognitive and language abilities was moderated by PSD ($\beta = -0.32$; 95% CI, -0.48 to -0.15 and $\beta = -0.36$; 95% CI, -0.52 to -0.20 , respectively). Increases in the T-factor were positively associated with these outcomes, but only for children at the mean and 1 SD below the mean of PSD. The T-factor was negatively associated with child externalizing and internalizing symptoms over and above PSD and other covariates ($\beta = -0.30$; 95% CI, -0.52 to -0.08 and $\beta = -0.32$; 95% CI, -0.55 to -0.09 , respectively). Increasing T-factor scores were associated with decreases in internalizing symptoms, but only for children with PSD 1 SD above the mean. The T-factor was positively associated with child cortical gray matter above PSD and other covariates ($\beta = 0.29$; 95% CI, 0.04-0.54), with no interaction between PSD and T-factor.

CONCLUSIONS AND RELEVANCE Findings from this study suggest that key aspects of the psychosocial environment in the first year impact critical developmental outcomes including cognitive, brain, and socioemotional development at age 3 years. This suggests that environmental resources and enhancement in the first year of life may facilitate every infant's ability to thrive, setting the stage for a more positive developmental trajectory.

Author Affiliations: Department of Psychiatry, School of Medicine, Washington University in St. Louis, St. Louis, Missouri (Luby, Herzberg, Hoyniak, Tillman, Lean, T. Smyser, Rogers, Barch); Department of Neurology, School of Medicine, Washington University in St. Louis, St. Louis, Missouri (Brady, Triplett, Alexopoulos, Loseille, C. D. Smyser); Department of Pediatrics, School of Medicine, Washington University in St. Louis, St. Louis, Missouri (Warner); Department of Psychological & Brain Sciences, School of Medicine, Washington University in St. Louis, School of Medicine, St. Louis, Missouri (Barch).

Corresponding Author: Joan Luby, MD, Department of Psychiatry, Washington University School of Medicine, 660 S Euclid Ave, Campus Box 8511, St. Louis, MO 63110 (lubyj@wustl.edu).

JAMA Pediatr. doi:10.1001/jamapediatrics.2024.0143
Published online March 18, 2024.

New evidence estimates that 43% of the world's children younger than age 5 years in low- and middle-income countries do not achieve their developmental potential due to poverty.¹ Notably, poverty rates for US children are the highest among all developed countries, with 1 in 5 lacking the basic environmental supports necessary for healthy growth and development.² In 2023, the poverty rate among children in the US doubled following congressional failure to renew the Child Tax Credit.³ Relevant to these staggering statistics, decades of empirical data have now made clear that the human infant's capacity for healthy brain, socioemotional, and cognitive development is in part dependent on the availability of key environmental conditions and basic resources. A rich literature documents the advantages of breast milk for the enhancement of cognitive and health outcomes.⁴ However, in addition to the obvious essential role of nutrition, a number of other environmental factors have proven critical. Over 75 years ago,⁵ observation of human infants' failure to thrive without a consistent nurturing caregiver, despite sufficient nutrition, was reported, leading to empirical studies documenting the essential role of psychosocial support for infant thriving.^{6,7} Similar to a plant's need for light, water, and nutrient-rich soil for healthy growth, the developing infant's brain—along with the socioemotional and cognitive skills that they subservise—requires psychosocial supports to proceed on a positive developmental trajectory. However, despite remarkable advances in science, medicine, and technology, almost half of the world's infants and young children still do not have access to these basic resources.

An emerging literature has begun to detail the material and psychosocial factors that drive the development of a robust neural infrastructure supporting cognitive and emotional flourishing. Beginning in utero, fetal neurodevelopment is impacted by maternal psychopathology and stress.⁸⁻¹¹ Recent data suggest that maternal experiences of psychosocial adversity during pregnancy (poverty, discrimination, low social support, and exposure to neighborhood crime) impact fetal structural and functional brain development.¹²⁻¹⁴ Such early developmental experiences powerfully impact brain development in ways that, while adaptive for expected early adverse contexts, may prove maladaptive later in development. Furthermore, emerging data suggest that the way in which biological factors shape brain development (eg, cytokine levels influencing tract development) may be different in the context of low vs high resources.¹⁵ If further validated, such findings raise the possibility that there may be a biological poverty line above or below which brain development is informed by experience in unique ways. Such a biological poverty line would represent the threshold at which physiology responds along distinct trajectories to psychosocial influences for best adaptation to future expected environments.

Building on these principles and an extensive body of international work focusing on essential supports for developing children, we hypothesized that the necessary basic conditions for healthy early development, dubbed the thrive factor (T-factor), include (1) adequate shelter and protection from danger, (2) a supportive and reliable primary caregiver, (3) healthy sleep and daily rhythms, (4) sufficiently nutritious food, and (5) appropriate environmental stimulation.^{16,17} We aimed to empirically test

Key Points

Question How do key environmental resources support healthy cognitive, socioemotional, and brain development in the first 3 years of life?

Findings In this longitudinally assessed sample of 232 mother-infant dyads oversampled for those facing poverty, greater levels of psychosocial environmental supports in the first year were associated with improved cognitive, socioemotional, and brain development.

Meaning The provision or enhancement of psychosocial environments in the first year of life may facilitate every child's ability to thrive.

whether these supports in the first year of life, combined as a T-factor (**Figure 1**), facilitate a child's cognitive, socioemotional functioning, and structural brain outcomes at age 3 years, well-established predictors of later childhood functioning.¹⁸ This model builds on the proven pervasive negative impacts of low socioeconomic status on cognitive, behavioral, and neural outcomes in children¹⁹⁻²¹ to identify the basic environmental supports developing human infants require to thrive. While components of the T-factor will be impacted by poverty and related structural inequities, they may also serve as pathways through which poverty impacts child developmental outcomes in distinct negative (by their absence) or promotive (by their presence) ways, suggesting that enhancement of the T-factor could buffer the detrimental impacts of poverty. The objective of investigating this T-factor is to create a biologically validated, developmentally pragmatic, and public health-relevant index to guide and prioritize the provision of basic environmental supports in the first year of life. If such necessary environmental supports early in life can be clarified, quantified, and validated, it could provide a roadmap for practitioners and actionable public policy reform aimed at facilitating every child's ability to achieve their potential in the first year of life.

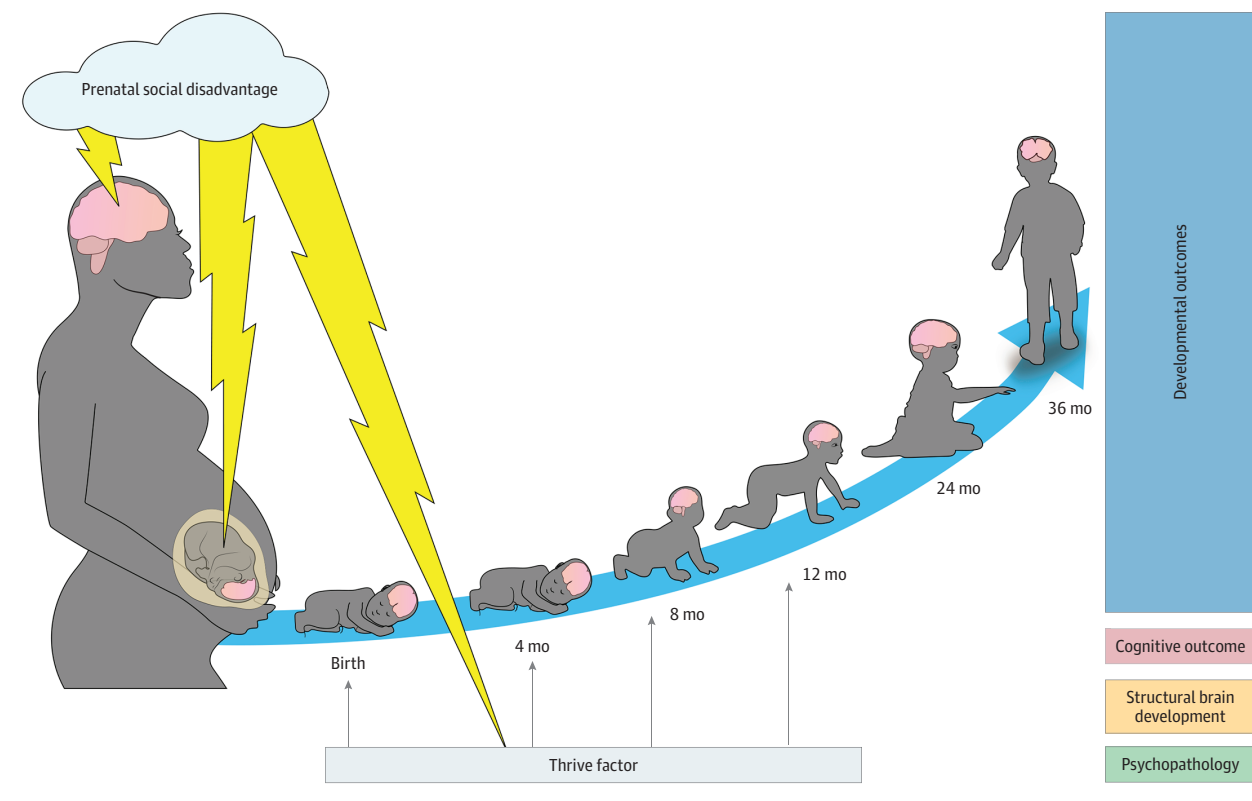
To begin to test these ideas, we used data from an ongoing longitudinal study, Early Life Adversity Biological Embedding, and Risk for Developmental Precursors of Mental Disorders (eLABE) that collected comprehensive measures of social disadvantage (income, education, insurance, neighborhood adversity) beginning in utero (prenatal social disadvantage [PSD]) and then measured the components of the T-factor in early life. We investigate associations between the T-factor and cognitive, socioemotional, and structural brain development in early childhood, including whether the T-factor accounts for variance in these outcomes over and above the impact of PSD. We further assess whether the T-factor functions consistently across levels of PSD and/or mediates the associations between disadvantage and neurobehavioral outcomes in early childhood.

Methods

Participants

Participants were mother-infant dyads recruited during pregnancy and enriched for exposure to adversity as part of eLABE,

Figure 1. Conceptual Diagram of the Primary Predictors and Outcomes of Interest



a multiwave, longitudinal study investigating the effects of early adversity on neurodevelopment and risk for mental disorders. Pregnant women who were participants in a study of preterm birth within the Prematurity Research Center at Washington University in St Louis with no alcohol or substance use during pregnancy (except for tobacco or marijuana) and without known pregnancy complications or fetal congenital problems were invited for eLABE participation. The study recruited 395 pregnant women and their subsequent 399 singleton offspring (4 mothers had 2 singleton births). Inclusion criteria for the present study included English speaking, maternal age 18 years or older, and singleton birth. Additional exclusion criteria included maternal congenital infections and known fetal abnormalities. Written informed consent was obtained from mothers prior to study participation. All study procedures were approved in advance by the Washington University institutional review board. EQUATOR reporting guidelines were followed.

Measures

PSD was quantified using a structural equation model-derived factor previously described and validated,²² composed of household income, caretaker education, insurance status, area deprivation index, and diet (Supplement 1). Derived based on a priori hypotheses building on the extant literature, the T-factor was composed of (1) the Child Opportunity Index²³ education domain at birth and 4, 8, and 12 months, as a measure of environmental stimulation, (2) Infant Feeding Questionnaire-Revised²⁴ at 4, 8, and 12 months and the eat-

ing subscale from Infant-Toddler Social and Emotional Assessment (ITSEA)²⁵ at 12 months as a measure of nutrition, (3) violent crime rates²⁶ based on US census block information from 4, 8, and 12 months as a measure of neighborhood safety, (4) objective coding of parent-child interaction tasks at 12 months as a measure of positive caregiving,²⁷ and (5) sleep problems measured using the ITSEA sleep subscale at 12 months²⁵ as a measure of child sleep (Figure 2). The 3 domains with more than 1 measure (ie, environmental stimulation, nutrition, and neighborhood safety) were estimated using Confirmatory Factor Analysis (eFigure 1 in Supplement 1).

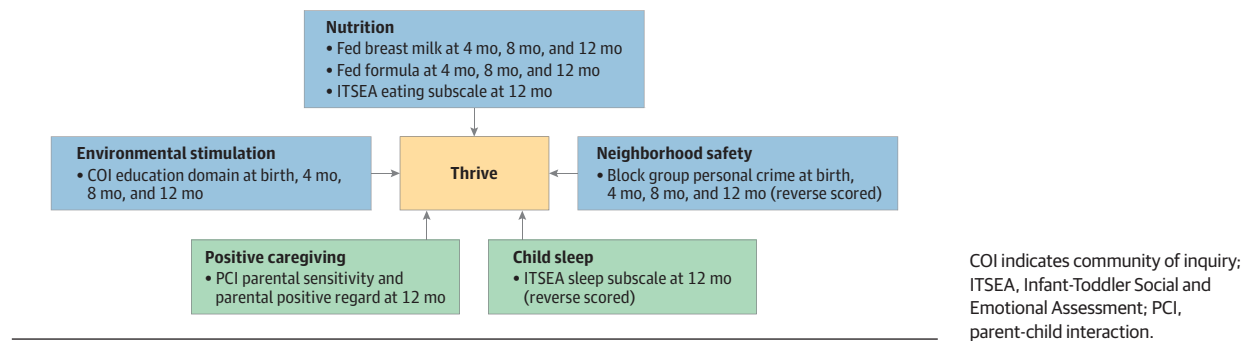
Cognitive and Language Outcomes

Cognitive and language outcomes were measured by Bayley Scales of Infant Development, 3rd edition (Bayley-III)²⁸ at age 36 months with separate analyses for the cognition and language. Internalizing and externalizing behaviors were measured using the ITSEA²⁵ at age 24 months. Covariates included infant sex, birth weight, age at magnetic resonance imaging (MRI) scan, and gestational age (GA) at birth. Maternal cognitive ability was assessed using age-normed standard scores from the Test of Premorbid Function.²⁹ The Test of Premorbid Function demonstrates concurrent validity with the Wechsler Adult Intelligence Scale-IV ($r = 0.70$).²⁹

MRI Scanning

MRI scans occurred at ages 24 and 36 months (24 months was used for participants with data at both ages) on a Siemens

Figure 2. Thrive Model



Prisma 3-T scanner and a 64-channel Siemens head coil while children slept. Details about data acquisition and processing are in the eMethods in [Supplement 1](#). Brain volumes of interest included total cortical gray and total white matter. Cortical folding was measured using the mean gyrification index across both hemispheres.

Analytic Plan

To include all children who had outcome data at age 3 years, we used multiple imputation, which is regarded as best practice for handling missing data and improves accuracy and statistical power relative to other techniques (eg, list-wise deletion). Incomplete variables were imputed under a fully conditional specification, using the default settings of the mice 3.0 package³⁰ in R (The R Project)³¹ and stabilized after 5 imputations.

The T-factor was created by averaging the environmental stimulation, nutrition, and neighborhood safety factor scores with standardized values of the positive caregiving and child sleep indexes from each imputed dataset and then pooling across datasets. Statistical parameters of interest were estimated in each imputed data set separately and pooled.³²

Stepwise linear regressions were used to examine the association between the T-factor and our 3 main outcomes of interest (ie, cognitive abilities, psychopathology, and brain structure). First, the simple association between the T-factor and outcome of interest was established. Second, we regressed the outcome of interest on the T-factor and child sex (for Bayley-III and MRI outcomes only), age at the time of MRI (for MRI outcomes only), and birth weight. We next added the PSD factor score. Robustness checks included covarying for GA in all models and maternal cognitive ability in the models investigating cognitive and language outcomes. To better understand the interaction between PSD and the T-factor, moderation models were fit for each outcome of interest in follow-up analyses. Multiple comparison correction was subsequently completed using the false discovery rate method³³ within sets of primary main-effect models using the same outcome instrument. The same approach was taken to the moderation follow-up analyses.

Lastly, we fit separate mediation models with PSD as the predictor and the T-factor as the mediator for each outcome. Mediation models were fit according to the approach recommended by Hayes³⁴ to test for statistical mediation. Indirect

paths between predictor and outcome through the proposed mediator were estimated using a bootstrapping approach (with 1000 iterations) using the lavaan package for R (The R Project).³⁵

Results

Descriptive statistics for the predictors and outcomes of interest are included in eTable 1 in [Supplement 1](#) with correlations in eTable 2 in [Supplement 1](#). PSD and the T-factor were highly correlated ($r = -0.78$; $P < .001$), though unique variance remained for each predictor when included in regression models. See eTable 2 in [Supplement 1](#) for correlations among T-factor components, as well as outcomes.

Main Effects and Moderation

Cognitive Abilities

As shown in the [Table](#), the T-factor was significantly positively associated with child cognitive abilities at age 3 years ($\beta = 0.33$; 95% CI, 0.14-0.52) even when controlling for child sex, birth weight, and PSD. This effect remained significant when GA and maternal cognitive abilities were included. The T-factor also was significantly associated with child language abilities at age 3 years when covarying for child sex and birth weight ($\beta = 0.36$; 95% CI, 0.24-0.49), but not when PSD was added to the model ($\beta = 0.11$; 95% CI, -0.08 to 0.30) nor when GA and maternal cognitive abilities were included ([Table](#); eTable 3 in [Supplement 1](#)). The association of the T-factor with child cognitive and language abilities was moderated by PSD ($\beta = -0.32$; 95% CI, -0.48 to -0.15 and $\beta = -0.36$; 95% CI, -0.52 to -0.20, respectively; eTable 4 in [Supplement 1](#)). Increases in the T-factor were positively associated with child cognitive and language abilities at low and mean levels of PSD (ie, less disadvantage), but not in the context of high PSD ([Figures 3A and 3B](#)).

Socioemotional Outcomes

The T-factor was negatively associated with child externalizing and internalizing symptoms when child sex and birth weight, as well as PSD, were included in the regression models ($\beta = -0.30$; 95% CI, -0.52 to -0.08 and $\beta = -0.31$; 95% CI, -0.55 to -0.08, respectively; [Table](#); eTable 3 in [Supplement 1](#)). No moderating effects of PSD were observed on the

Table. Pooled Estimates From Multiply Imputed Linear Regression Models of Thrive Factor Associations With Year 3 Bayley, Year 2 Infant-Toddler Social and Emotional Assessment (ITSEA), and Year 2 or 3 Brain Measures

| Covariates | None | | Child sex, MRI age, birth weight ^{a,b} | | Child sex, MRI age, birth weight, PSD ^{a,b} | | Child sex, MRI age, birth weight, PSD, GA ^{a,b} | | Child sex, MRI age, birth weight, PSD, GA, maternal IQ ^{a,b} | |
|--|------------------------|---------|---|---------|--|---------|--|---------|---|---------|
| | β (95% CI) | P value | β (95% CI) | P value | β (95% CI) | P value | β (95% CI) | P value | β (95% CI) | P value |
| Outcome | | | | | | | | | | |
| Year 3 Bayley Cognitive | 0.42 (0.30-0.55) | <.001 | 0.41 (0.28-0.54) ^c | <.001 | 0.33 (0.14-0.52) ^c | <.001 | 0.33 (0.13-0.52) ^c | .001 | 0.33 (0.13-0.52) ^c | .001 |
| Year 3 Bayley Language | 0.38 (0.26-0.50) | <.001 | 0.36 (0.24-0.49) ^c | <.001 | 0.11 (-0.08 to 0.30) | .26 | 0.11 (-0.09 to 0.30) | .27 | 0.08 (-0.12 to 0.27) | .44 |
| Year 2 ITSEA externalizing | -0.39 (-0.52 to -0.25) | <.001 | -0.36 (-0.49 to -0.22) ^c | <.001 | -0.30 (-0.52 to -0.08) ^c | .01 | -0.30 (-0.52 to -0.08) ^c | .01 | NA | NA |
| Year 2 ITSEA internalizing | -0.25 (-0.39 to -0.11) | <.001 | -0.24 (-0.38 to -0.09) ^c | .002 | -0.31 (-0.55 to -0.08) ^c | .01 | -0.32 (-0.55 to -0.09) ^c | .01 | NA | NA |
| Total gray matter volume (relative to ICV) | 0.40 (0.23-0.58) | <.001 | 0.43 (0.25-0.61) ^c | <.001 | 0.30 (0.05-0.55) | .02 | 0.29 (0.04-0.54) ^c | .02 | NA | NA |
| Cerebral white matter volume (relative to ICV) | 0.26 (0.08-0.45) | .01 | 0.19 (0.00-0.37) | <.05 | 0.06 (-0.20 to 0.32) | .64 | 0.06 (-0.20 to 0.33) | .64 | NA | NA |
| Mean gyri-fication index | 0.18 (-0.01 to 0.37) | .06 | 0.11 (-0.07 to 0.30) | .23 | -0.11 (-0.37 to 0.15) | .41 | -0.13 (-0.38 to 0.12) | .32 | NA | NA |

Abbreviations: GA, gestational age; ICV, intracranial volume; IQ, intelligence quotient; MRI, magnetic resonance imaging; NA, not applicable; PSD, prenatal social disadvantage.

^a Child sex only included as a covariate in Bayley models.

^b MRI age only included as a covariate in brain metric models.

^c Remains significant after false discovery rate method correction.

association between the T-factor and child externalizing behavior. However, there was a significant moderation of the association between the T-factor and internalizing symptoms ($\beta = -0.25$; 95%CI, -0.44 to -0.06 ; eTable 4 in Supplement 1). Increasing T-factor scores was associated with decreases in internalizing symptoms, but only for individuals with PSD scores above the mean (ie, more disadvantage) (Figure 3C).

Brain Structure

The T-factor was positively associated with child cortical gray matter ($\beta = 0.43$; 95% CI, 0.25 - 0.61) (Table; eTable 3 in Supplement 1) when covarying for child sex, age at scan, and birth weight. Furthermore, the association with cortical gray matter remained significant when PSD and GA were added to the model ($\beta = 0.29$; 95% CI, 0.04 - 0.54 ; Table; eTable 3 in Supplement 1). Neither white matter volume nor mean gyri-fication index were associated with the T-factor after false discovery rate method correction. PSD did not moderate the association between the T-factor and child cortical gray matter ($\beta = 0.12$; 95% CI, -0.11 to 0.36 ; eTable 5 in Supplement 1).

Mediation Models

Cognitive Abilities

PSD was a strong, negative predictor of child cognitive abilities at age 3 years ($\beta = -0.38$; 95% CI, -0.50 to -0.26). The T-factor mediated this association between PSD and child cognitive abilities at age 3 years (Figure 4A). No mediation was observed for child language abilities.

Socioemotional Outcomes

PSD was positively associated with child externalizing ($\beta = 0.34$; 95% CI, 0.21 - 0.48) and internalizing ($\beta = 0.16$; 95%

CI, 0.02 - 0.30) symptoms at age 2 years. Both of these associations were mediated by T-factor scores (eFigures 2 and 3 in Supplement 1), such that higher T-factor scores were associated with fewer externalizing and internalizing problems.

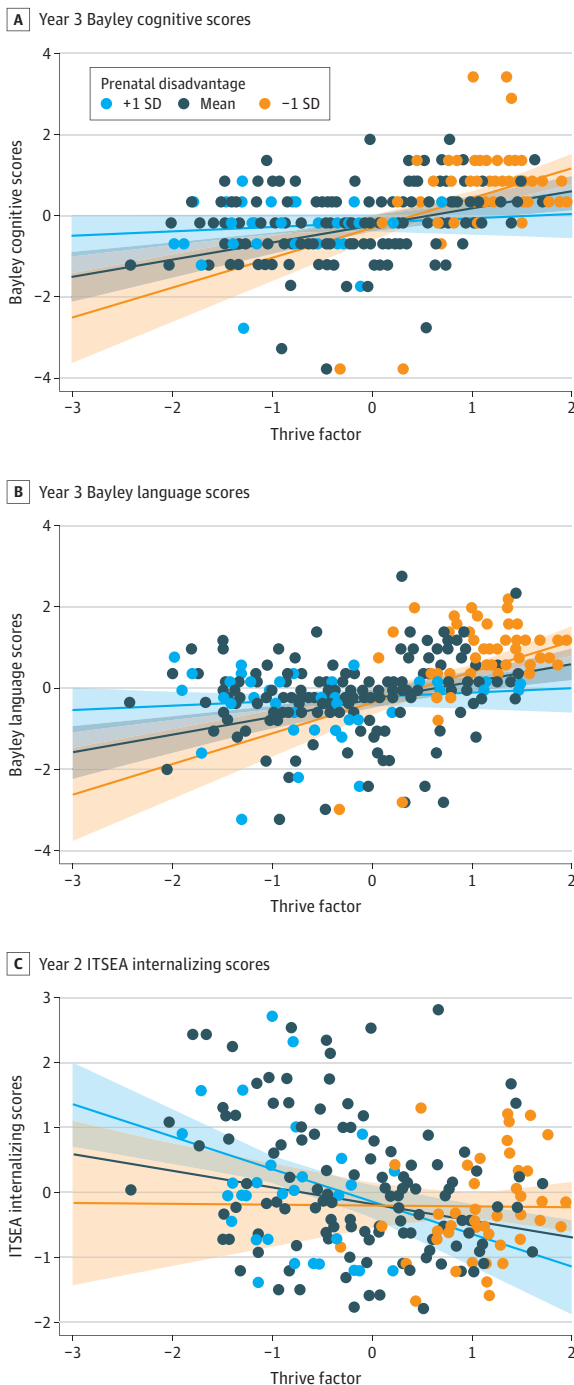
Brain Structure

The negative association between PSD and child cortical gray matter volume ($\beta = -0.40$; 95% CI, -0.59 to -0.21) was mediated by T-factor scores (Figure 4B). T-factor scores did not mediate the association between PSD and child white matter volume (indirect effect $\beta = -0.05$; 95% CI, -0.27 to 0.17 ; eFigure 4 in Supplement 1).

Discussion

Findings from this longitudinal study beginning in utero demonstrate important links between higher T-factor scores in the first year of life, a reflection of high-quality developmental environments and improved cognitive, emotional, and neural outcomes at ages 2 and 3 years. Notably, both main effects and moderation analyses indicate that the T-factor explains unique variance relative to that attributable to PSD. Our results indicate that the T-factor is positively associated with global cognition, assessed using the Bayley-III, above and beyond PSD and maternal cognitive abilities. Similar effects were identified for child language outcomes, though these associations did not survive correction for PSD or maternal cognitive abilities. Importantly, these associations are only significant in children with low and mean levels of PSD, suggesting that a basic level of social advantage is required for the T-factor to confer promotive effects in cognitive development. Conversely, the

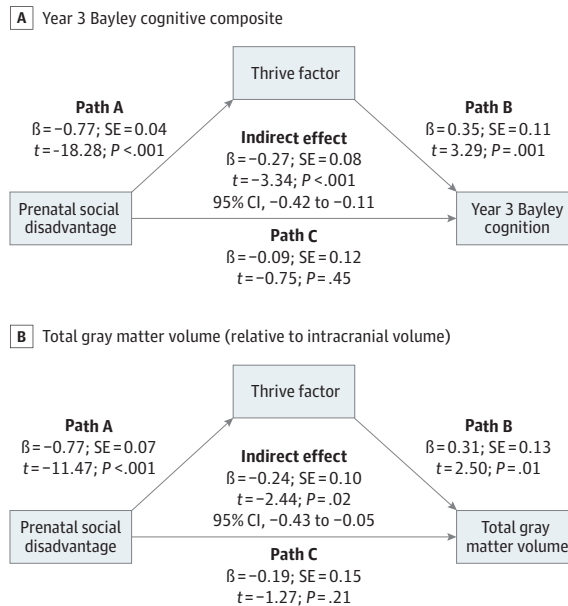
Figure 3. Prenatal Social Disadvantage Moderates the Association Between Thrive Factor and Year 3 Bayley Cognitive Scores, Year 3 Bayley Language Scores, and Year 2 Infant-Toddler Social and Emotional Assessment (ITSEA) Internalizing Symptoms



Cognition and language scores were age normed and moderation models included sex and birth weight. Internalizing symptom scores were normed for age and sex with birth weight included as a covariate. Each moderation survived false discovery rate correction for multiple comparisons.

T-factor was a robust predictor of child externalizing and internalizing symptoms when all covariates and robustness

Figure 4. Thrive Factor as a Mediator of the Association Between Prenatal Social Disadvantage and Year 3 Bayley Cognitive Composite and Total Gray Matter Volume



checks were included, and moderation analyses suggested that the promotive effect of the T-factor on internalizing symptoms was most beneficial for children at high levels of PSD (in contrast to cognitive findings).

These effects of the T-factor in the reduction of internalizing symptoms, especially in those facing high levels of PSD, is notable. These findings suggest that the internalizing symptoms arising in toddlers in high-disadvantage environments could be directly related to experiences of threat and deprivation that arise in that setting and that adding enrichment directly mitigates these symptoms, whereas internalizing symptoms arising in more resourced environments may be related to other factors that require different kinds of interventions.³⁶ Notably and relevant to public policy, these collective findings suggest that a baseline level of social advantage may be necessary for the T-factor to promote cognitive development, but that additional T-factor supports are beneficial for reducing some aspects of psychopathology risk even at high levels of PSD. Lastly, the T-factor was associated with child cortical gray matter at age 2 or 3 years, both when covarying for PSD and at all levels of PSD. This suggests that brain development is powerfully impacted by the environment in the first year of life—a finding of high public health relevance. These effects may be operational biologically through alterations in stress systems, inflammatory responses, and the gut microbiome, which have a particularly powerful impact on brain development during this period of high neuroplasticity.

Current study findings are notable for the consistency of the promotive effects of the T-factor across multiple developmental domains. Perhaps most notable are the associations with cognitive abilities, as changes in cognitive development in the preschool period have been difficult to achieve despite

some intensive interventions.³⁷ The T-factor was also associated with improved externalizing and internalizing symptoms in early childhood, consistent with the known positive impacts of early childhood nurturance and positive supports on emotion regulation and behavioral control.³⁸ Importantly, these associations with the T-factor account for variance not accounted for by PSD. This pattern of results suggests that the T-factor has the potential to make specific contributions to healthy development during early sensitive periods over and above the contributions of risk, and highlight the importance of the early psychosocial environment as a promotor of improved neurobehavioral outcomes.

Taken together, the results suggest that enhancing the components of the T-factor in the first year of life should become a priority for infants in general, as well as those exposed to adversity in utero. Additional intervention or policy intended to decrease exposure to PSD, such as financial assistance for pregnant individuals in high-disadvantage contexts, could also be beneficial as it could create the necessary conditions for the T-factor to promote improved cognitive ability and might limit early psychopathology symptoms associated with prenatal maternal stress.

Limitations

While this study had many strengths, including the prospective assessment of a variety of child outcomes, there were also limitations. These included the fact that we did not have direct measures of some components of the T-factor, such as direct assessment of sleep in children, which could be addressed in future studies using actigraphy. Furthermore, more nuanced measures of nutrition, as well as exposures to/ protections from environmental toxins, would also be ideal to clarify and refine the most important and/or differential effects of the elements of the T-factor, including some elements not accounted for in the current model. Additionally,

findings were based on a high-risk Midwestern cohort and should be replicated in geographically diverse populations. Future studies in larger samples with more intensive and objective measures of all components of the T-factor are necessary to further establish the validity of T-factor, and the differential contribution of its component indices, to healthy child development. These studies would ideally be intervention designs targeting specific domains of the T-factor, necessary to inform causality and identify rigorously which components have the largest effects on outcomes.

Conclusions

In summary, the finding that these basic psychosocial supports in the first year of life, which should be in reach for every human infant, positively impact cognitive, psychosocial, and brain development at age 3 years above and beyond, effects of PSD and maternal cognitive abilities suggests that the assurance of these resources should be among the highest public health priorities. Basic resources in the first year of life that make up what has been conceptualized as the postnatal womb are perhaps as important as clean water to protect behavioral and cognitive health. If further validated, ensuring that the components of the T-factor are available to infants in the first year of life could and should become a focus for primary care and other settings designed to promote positive development in early childhood. Furthermore, these findings from a sample enriched for experiences of PSD suggest that the negative effects of PSD may be mitigated by fortification of the postnatal environment. While focus on the health and safety of the prenatal environment remains crucial for offspring health, the finding that positive developmental outcomes can arise at age 2 and 3 years with enhancements in the first postnatal year is promising news for developing children and toward ensuring every child's right to thrive.

ARTICLE INFORMATION

Accepted for Publication: January 17, 2024.

Published Online: March 18, 2024.

doi:10.1001/jamapediatrics.2024.0143

Author Contributions: Dr Luby had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Concept and design: Luby, Herzberg, Hoyniak, Warner, C. Smyser, Barch.

Acquisition, analysis, or interpretation of data: Luby, Herzberg, Hoyniak, Tillman, Lean, Brady, Triplett, Alexopoulos, Loseille, T. Smyser, Rogers, C. Smyser, C. Barch.

Drafting of the manuscript: Luby, Herzberg, Hoyniak, Tillman, Barch.

Critical review of the manuscript for important intellectual content: Luby, Herzberg, Tillman, Lean, Brady, Triplett, Alexopoulos, Loseille, T. Smyser, Rogers, Warner, C. Smyser, Barch.

Statistical analysis: Herzberg, Hoyniak, Tillman, Barch.

Obtained funding: Luby, Brady, Rogers, Warner, C. Smyser, Barch.

Administrative, technical, or material support: Lean, Triplett, Loseille, T. Smyser, C. Smyser, Barch.

Supervision: Luby, Rogers, Warner, Barch.

Conflict of Interest Disclosures: Dr Luby reported grants from the National Institute of Mental Health and the American Foundation for Suicide Prevention. Dr Lean reported grants from the National Institute of Mental Health during the conduct of the study and grants from Brain & Behavior Research Foundation, the National Institute of Mental Health, and the National Institute of Neurological Disorders and Stroke Grant outside the submitted work. Dr Brady reported grants from the National Institute of Health and the Center for Race, Ethnicity, and Equity at Washington University during the conduct of the study. Dr Triplett reported grants from the National Institute of Mental Health and the National Institutes of Health during the conduct of the study. Dr Rogers reported grants from the National Institutes of Health during the conduct of the study and grants from the National Institutes of Health outside the submitted work. Dr C. Smyser reported grants from the National Institutes of Health and grants from the Gates Foundation during the conduct of the study. No other disclosures were reported. Dr Barch reported grants from the National Institute of Mental Health, the National Institute on Drug Abuse, and the American

Foundation for Suicide Prevention. No other disclosures were reported.

Funding/Support: All phases of this study were supported by the National Institute of Mental Health (5 R01 MH13883-04).

Role of the Funder/Sponsor: The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Data Sharing Statement: See [Supplement 2](#).

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