A PRACTICE BRIEF FOR CHILD WELFARE AND CHILDREN'S MENTAL HEALTH PRACTITIONERS

The Reform You Have Been Waiting Your Whole Career for Isn't Coming

Ten Concepts from Science to Reform Your Practice Instead

"Relationships are the oxygen of human development."
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This brief is for practitioners in child welfare and children's mental health, and others who work with children and families during times of severe adversity and hardship. When a child has no parent or close kin to buffer adversity, the unbuffered stress can do lasting biological harm, because the systems it disrupts are still forming. Children are not affected equally: some are more sensitive to their conditions than others, and the more sensitive a child is, the more those conditions shape them. That sensitivity is not only a vulnerability, because the children hurt most by bad conditions are often the ones who gain most from good ones. Where the harm occurs, it is a lasting impairment, not passing distress or a behavior problem to manage, and it may limit functioning in major life areas: learning, attention, communication, emotion, work, housing, health, reproductive health, parenting, and relationships. The single most important factor is whether the child keeps at least one relationship their stress system already recognizes as safe, and more than one is better.

THE TEN CONCEPTS

- **Disrupted childhood development produces disability.** Severe neglect, whether by a parent or in a non-kin placement, and repeated placement instability produce developmental disabilities that affect major life areas and functioning. The DSM labels the symptoms and ignores the causes.
- **A stable relationship with a parent or kin matters more than the adversity itself.** Whether a parent or close kin helps a child recover from stress does more to shape the outcome than how severe the adversity was. With that support, the stress stays tolerable. Without it, the same adversity becomes toxic.
- **The harm becomes biological and shows up later.** HPA-axis and epigenetic changes make the harm last, producing vulnerability to early chronic disease decades later through biology rather than behavior alone.
- **It is not destiny.** Children stay responsive to good conditions, and recovery is possible when a stable relationship with a parent or kin is preserved or quickly restored.
- **Resilience is a measure of what is around the child, not inside them.** It is not a trait some children have, and others lack. The relationships and conditions around a child produce it, and it can be built at any age.
- **Early puberty may open a second window.** The stress-response system can reset during adolescence, giving placement and reunification decisions real biological weight.
- **Healing stays possible after the windows close.** Recovery is partial rather than a reset, so the impairment and its accommodations remain. A recognized, co-regulating relationship can still build new regulation capabilities at any age, which is why support should not end at emancipation.
- **Struggles in many areas can come from one cause.** Problems with learning, school, self-care, work, housing, health, reproductive health, parenting, and relationships can trace to the same early harm, so they are not separate failings to be treated within multiple sub-specialties.
- **Severely disrupted child development produces disabilities, not disorders.** The removal from a parent and non-kin placement most often comes first. The DSM disorder label quickly follows, to qualify the child to fund a therapist, a prescriber, and other services, despite a lack of scientific validity, long admitted by the DSM authors themselves.
- **Foster placement itself qualifies a child, like prematurity.** Current AAP guidance recommends that foster placement itself qualify a child for early intervention, on the same logic as prematurity. The experience is the marker of developmental risk, before any diagnosis.

DECADES OF SCIENCE, ACROSS DISCIPLINES

This is the mainstream position in pediatric medicine, not a fringe or contrarian one. Decades of research support it, in developmental neuroscience, pediatrics, epidemiology, and psychology. The American Academy of Pediatrics put it into policy in 2012, reaffirmed and extended it in 2021, and in 2026 applied the framework to young children in foster care, in a policy statement and a clinical report. The National Academies came to the same conclusions in their 2025 report on early relational health.

PART ONE: THE MECHANISM OF PRODUCTION

1. Development is a construction process on a fixed schedule

The visible milestones of childhood, such as holding up the head, crawling, walking, pointing, naming, and speaking in sentences, are the outward signs of a deeper process. Beneath them, the nervous system, the stress-response system, the immune system, the early reproductive system of girls (oogonia and primary oocytes), and the cardiovascular infrastructure are being built from environmental inputs during **sensitive periods**. These are windows of heightened neuroplasticity and somatic growth, when developing systems respond strongly to experience, for better or worse, shaped in part by oxidative stress (Do et al., 2015).

These windows open and close on a schedule that does not wait for a child's circumstances to improve. The ear's sensitivity to speech sounds closes in infancy. The prefrontal-limbic circuitry behind executive function and emotional regulation does most of its wiring in the first three years and continues more slowly through adolescence. The HPA axis, the body's central stress system, sets its baseline from the relationships of early childhood. Whatever a window closes on becomes permanent. Disrupt the child during that building and the result is developmental disability: systems built differently than they would have been, with lasting, predictable effects that depend on what was disrupted and when. This is a physical fact, not a label a clinician assigns.

WHAT COUNTS AS A DEVELOPMENTAL DISABILITY

When people hear "developmental disability," they usually picture the visible, recognized conditions present from early life, such as intellectual disability, autism, cerebral palsy, mobility problems, and severe vision or hearing impairments. Each of these is a developmental disability because it produces lasting impairment in one or more major life areas, and that impairment is the definition. This is the legal definition too, not a loose usage. Federal law defines disability by substantial limitation in major life activities (Americans with Disabilities Act), and defines a developmental disability as such an impairment, arising during development, that substantially limits three or more of them, among them self-care, language, learning, mobility, and independent living (DD Act). So, the category is wider than the visible conditions: it includes any lasting impairment in major life areas that comes from development being disrupted while the brain and body are still being built. The disabilities in this brief meet that definition but are less visible: impaired executive function (planning, attention, working memory, and impulse control), difficulty regulating emotion, delayed language, learning difficulty, and early physical illness. Like any disability, they call for support and accommodation, not blame. Part Two takes each one in turn, showing where it impairs a life area and what helps.

2. The buffering relationship decides whether stress is tolerable or toxic

The decisive factor is whether the child keeps a relationship their stress system already recognizes as safe, usually with the parent or close kin who raised them, so that a familiar adult's calm can bring the child's stress back to a tolerable baseline. Stress that stays elevated instead disrupts the child's developing systems and accelerates cellular aging. As a result, the presence of the co-regulating adult matters more than how severe the adversity was, and more than the health, training, or license held by any substituted caregiver the child does not recognize. The science calls this **co-regulation**.

> **HEALTHY, TOLERABLE, AND TOXIC STRESS**
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> **Healthy stress** is a brief, manageable challenge, a first day at a new school or a shot at the doctor: it spikes, comes back down, and the child comes out a little more capable. This is how the system is built to work. **Tolerable stress** is something serious, a death in the family, an injury, a frightening event; the stress response rises sharply, but a co-regulating adult is there, so it still comes back down, and the system recovers. **Toxic stress** is serious adversity that is strong, frequent, or prolonged, with no one to bring the stress response back down: the response goes up and does not come back down, and the developing systems adjust to a higher resting level or set point that never fully switches off. Whether stress stays tolerable or turns toxic depends on whether someone the child recognizes as a co-regulator was there to bring the stress response back to a regulated baseline, not on how severe the adversity was.

This is a specific relationship, not any caring adult, and the reason is biological. Co-regulation only works through a relationship the child's nervous system already recognizes as safe, the one it formed with the parent or kin who raised the child. A newly placed foster parent may be stable and caring, but at first the child's stress system does not recognize them as a co-regulator. Building that recognition takes time, if it forms at all, and each change of placement or caregiver starts that recognition over. In the meantime, the child's stress response goes unbuffered, unable to be reset by the co-regulator. The absence of a recognized co-regulator moves the hardship from tolerable to toxic: the stress activates and does not come back down. Because this falls during the sensitive windows when the child's systems are still being built, the toxic stress disrupts the healthy development underway there, and that disrupted development produces the impairment. This is why removal itself, even into a stable home, causes harm: it takes away the co-regulator the child recognizes and imposes an indefinite exposure to toxic stress while those systems are being built in response to relationships, experiences, and the environment.

The long-term outcome data agree, and the strongest designs control for the selection objection directly. Swedish sibling studies, comparing one sibling the state placed against one who remained in the same household, found the placed sibling had worse adult outcomes across physical health and all-cause mortality, with hazard ratios in the three-to-five-times range (Brannström, Vinnerljung and Hjern, 2020). In California's own records, adolescents and young adults who exited foster care showed elevated mortality (McNellan et al., 2026). In the sibling comparisons, both children lived through the same family hardship. Only one was also separated from the co-regulator, placed with non-kin, and exposed to the system's own dysfunction. The comparison isolates that added layer, not the hardship both shared. The AAP's 2026 clinical report notes that kinship placements are associated with better outcomes than non-kin foster care. A UK national study found that adults who had been in out-of-home care as children had all-cause mortality about 1.6 times higher than those who had not, up to forty-two years later, mainly from self-harm, accidents, and mental and behavioral

causes (Murray et al., 2020a).

The field usually explains these outcomes one case at a time, as mental health disorders, missing skills, or bad character. Disrupted development is the better explanation. Non-kin placement, repeated moves, staff changes, and separation from family remove the co-regulating relationship at the age the child's systems are being built, and the disrupted development that follows is what produces the impairment in major life areas that those labels are trying to name.

The evidence also nulls the beliefs the system is built on: that a healthy stranger is better for a child than a struggling parent, and that a healthy stranger with professional training is better still. The outcome data do not support it. Outcomes worsen along a gradient, from a child's own parents to kin to non-kin strangers to institutions, tracking how much of a recognized relationship the placement preserves rather than the health or credentials of the adult. A UK national cohort found the same gradient in adult health. Compared with adults who grew up with a parent, the odds of reporting poor health rose across kinship, non-relative foster, and residential care, and held across ten, twenty, and thirty years of follow-up, with residential care three to four times higher and kinship care the least affected (Murray et al., 2020b). The Swedish sibling studies make the point: the sibling left home did better than the one the state placed. Better, not well. Staying in a hard home was not good; however, for most children it was less bad than removal, because it kept the relationship the child's biology was built around. That is the deeper meaning of prevention. It does not mean swapping a failing adult for a healthier one. It means protecting and strengthening the child's own relationships and the conditions around them, which is what a developing child recovers through.

Harm here is not only what is done to a child; it is also what is withheld. The National Scientific Council on the Developing Child finds that the persistent absence of responsive care damages developing brain structure more than overt physical abuse does. Adversity that piles up with no one to buffer it does more damage than any single terrible event. The AAP's ecobiodevelopmental framework (2012, reaffirmed and extended in 2021) turns this into practice guidance: since stable, nurturing relationships build resilience and prevent or treat toxic stress, the evidence-based response is to keep those relationships intact, not to escalate symptom management instead. Resilience is not a trait inside the child. It is produced by the environment of relationships and material conditions around the child. That is why it can be built at any age, and why its loss does lasting harm.

3. How the disruption becomes permanent

Two things turn exposure to toxic stress into something harmful to lifelong health.

HPA-axis dysregulation.

Stress that never resolves resets the HPA axis to activate faster and recover slower. Cortisol stays high, which wears on the immune system and on how the body handles blood sugar. The autonomic system settles into fight-or-flight, keeping heart rate, blood pressure, and inflammation up. A stress system that should have been tuned for safety gets tuned for threat instead.

Epigenetic embedding.

Early adversity also leaves chemical marks on the genes while those circuits are still being built. Methylation of the glucocorticoid-receptor gene (NR3C1) thins out the receptors in the hippocampus that would normally switch the stress response off, so the off-switch works less well. These marks are not inherited genes; the environment produces them. And some of them pass to the next generation, through the prenatal, infant, and childhood environments, through early caregiving, and directly. So the way disability recurs across generations has a biological route, alongside the material and behavioral ones we already know about. It matters most for girls, whose reproductive development is sensitive to elevated oxidative stress both before and after birth (Richardson et al., 2014).

4. From developmental disruption to chronic adult disease

The damage does not stay in the brain. The immune, cardiovascular, metabolic, and endocrine systems all adapt to early adversity together, so the change is body wide. The raised inflammation, the reset stress axis, the altered immune response are the *upstream start* of what later gets diagnosed as ordinary adult disease: high blood pressure, type 2 diabetes, atherosclerosis, autoimmune conditions. They begin as disrupted childhood development and take decades to appear.

The Felitti and Anda ACE Study put this on an epidemiological scale, tracking 17,337 adults. More childhood adversity meant more adult disease, and the curve steepened at the high end: seven or more ACEs went with a life expectancy about **twenty years shorter** than none. Most of the harm came from the direct biological effect of chronic toxic stress on organs that were still being built, not from smoking or drinking. Parental separation, non-kin placement, and system dysfunction are additional adversities layered onto those a child experienced in the family before removal, increasing the burden and changing its composition in ways that are uniquely disruptive to health and social functioning.

> **WHY THIS IS NOT DESTINY**
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> The children hit hardest by bad conditions are frequently the same ones who gain the most from good ones. Researchers call it differential susceptibility. Positive Childhood Experiences are the measurable version of the good side: in a sample of 6,188 adults, those with high ACE scores but six or seven PCEs had 72 percent lower depression risk (adjusted OR 0.28) than those with none to two. The disability shows up most reliably when the high adversity and the loss of those protective relationships occur together, in the same windows.

5. A second window in early puberty

Timing matters beyond early childhood. The second decade is not the end of development. It is a second period of significant reorganization of the prefrontal cortex, the limbic system, and the HPA axis (Lerner et al., 2018; Campbell and Wendel, 2026). That reorganization opens the same biological systems to experience again, in both directions: harmful conditions can deepen the damage, and supportive ones can undo part of it. The second decade is therefore both a risk and an opportunity.

The specific opportunity is a stress-response system that can still be reset. In a supportive relational context, the adolescent HPA axis remains open to recalibration. DePasquale, Donzella, and Gunnar (2019) found that cortisol reactivity in previously institutionalized youth normalized across pubertal stages, moving from blunted

at early stages to indistinguishable from never-institutionalized peers by later stages. Following the same cohort, Gunnar and colleagues (2019, PNAS) reported pubertal stress recalibration that reversed the effects of early-life stress on HPA reactivity, a finding DePasquale (2021) set out as the pubertal stress recalibration hypothesis. Some evidence suggests that supportive relational conditions in adolescence affect the stress-response system as much as, or more than, the same conditions earlier in childhood.

None of this is automatic; the recalibration only happens if the relationship is restored. And an adolescent's behavior can mislead us. Looking independent is not the same as being able to self-regulate: a teenager can ramp the stress response up alone but still cannot bring it back down without a parent or close kin. So a non-kin or congregate placement, where that help is least likely, is a developmental mismatch, not just a suboptimal setting.

> **THE SECOND WINDOW IS A CHANCE FOR REPAIR**
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> Adulthood does not offer this window. Bringing a parent or close kin back during adolescence is not just giving a young person stability through a hard stretch; it engages an active biological repair. Do the same thing in adulthood and you are working on accumulated wear, not on developmental plasticity, because the architecture has set. It is one mechanism seen from either side. A co-regulating relationship present during stress produces the good outcome, and its absence produces the harm. The years around early puberty are a second, time-limited chance to change the course, and what the young person is given during those years does most to set it.

Healing is always possible

The second window is time-limited, and not every young person still has a parent or close kin when it opens. In time it narrows for everyone. The developmental windows do not reopen; the altered stress calibration does not return to what buffering would have produced, and the epigenetic marks and raised cardiometabolic risk persist. That lasting alteration is the disability this brief describes, and it is why support must continue. Recovery is still possible, but it is not a return to the original baseline. Rather than rebuilding the circuits the early windows set, it adds new structure alongside them.

The Center on the Developing Child, whose neglect research this brief draws on above, describes a plasticity that lasts across the lifespan: the brain's ability to reorganize is greatest early and lessens with age, but is never fully lost, so the capabilities that underlie resilience can be strengthened at any age (National Scientific Council on the Developing Child, 2015). The system that formed under chronic adversity can, once conditions change, begin building again: new connections, new regulation, and new relationships that take on the co-regulatory role the early ones never filled. An adult can form a secure attachment they did not have as a child. When children were moved out of institutional deprivation into families, their stress and attachment systems partially reorganized (McLaughlin et al., 2015), though the earlier alteration was not erased. Recovery follows the differential-susceptibility pattern described earlier: the children whose systems were shaped most by adversity often respond most once the relational conditions improve (Pluess and Belsky, 2013).

This matters most for the young people least likely to enter adolescence with a co-regulator, who are disproportionately those the system has moved repeatedly. For them recovery cannot wait on structural reform, which will not arrive in time; it depends instead on a recognized relationship, and that relationship can be

formed by whoever is available to stay, whether found kin, a mentor, a teacher, a coach, or a foster parent who remains long enough for the young person's stress system to register them as safe. Because recovery depends on a relationship rather than on an open developmental window, it can begin at any age, and the support it requires does not end at emancipation. The practice implication extends past the second window: continuity of a recognized, co-regulating relationship is the intervention, and it stays available for as long as such a relationship can be formed and kept.

PART TWO: IMPAIRMENTS ACROSS MAJOR LIFE AREAS

6. Where the impairment shows up

Assessment already looks at the areas this harm affects: learning, concentrating, thinking, communicating, managing emotion, working, maintaining housing, keeping relationships, and physical health. The mechanism adds that trouble across several of these areas is usually one problem, not many, and rarely a choice or "non-compliance." It is the same early disruption appearing in different places at different ages. It should also make us slow to treat a parent's impairments as proof of unfitness, when those impairments may be what an earlier system did to them or failed to give them.

What a diagnosis leaves out

A DSM diagnosis describes a cluster of symptoms and, by design, says nothing about cause. That puts the problem inside the child and leaves out the developmental process that made the presentation. The DSM's own field has said as much. Its categorical borders did not appear in the brain-imaging and genetics research meant to confirm them, and in 2026 the American Psychiatric Association set out a plan to overhaul the manual, one that would change the 'S' from 'Statistical' to 'Scientific' and move diagnosis toward dimensions, context, and biological measures rather than fixed categories (Parshall, 2026). This brief names the developmental cause directly: biological systems disrupted during sensitive windows, with no buffering relationship there to protect them. Naming the cause also changes where the response belongs. The problem is not inside the child. It is in what surrounds the child, the relationships and the early opportunities to strengthen development that are present or missing across the developmental windows. The remedy is there too: prevention and repair work on those conditions, not on the child.

The evidence on each side

The two accounts are not supported by the same evidence.

Toxic stress and disrupted development	The DSM categories

Dose-response epidemiology (the ACE studies). Causal designs that rule out selection (within-family sibling comparisons). A randomized trial (the Bucharest project). A measurable biological pathway (HPA-axis and epigenetic markers). Agreement across pediatrics, the National Academies, neuroscience, and epidemiology.	Poor reliability for several categories in the DSM's own field trials. Borders that do not appear in brain imaging or genetics. A verdict from the federal mental-health institute that the categories lack validity, and a turn in its research toward biology instead (Insel et al., 2010). The manual's own authors, from the chair of DSM-III to a contributor to the current text revision, have called the categories working hypotheses rather than validated entities (Frances, 2013; Kendler, 2021; APA, 2022). An overhaul begun by the APA in 2026, for the same reason (Parshall, 2026).

None of this must wait on reform. The DSM is being revised, and policy may follow, but the evidence is already strong enough to use. A practitioner can change how they understand a child's experiences and what they recommend now, without waiting for the new manual or the Medicaid and state financing system to catch up with the evidence.

Reform your standard of evidence, not your state of mind. That is where the reform in this brief is located. It is built throughout on a substantially stronger evidence standard, and when used, it reforms your practice. The standard comes from developmental neuroscience, pediatrics, epidemiology, and psychology, not from social work and industry insiders alone.

The coding is a concrete example. Alongside the DSM-aligned F-codes, which name a mental or behavioral disorder, the ICD offers codes that name the situation. Z-codes record the psychosocial and environmental circumstances (Z55–Z65, among them problems related to upbringing and to the primary support group), T-codes record confirmed or suspected maltreatment (T74, T76), R-codes record the symptoms and signs themselves, and Q-codes record developmental and congenital origins such as fetal alcohol syndrome. A clinician can use them to record what happened to the child.

The barrier to using them is not federal. CMS has pressed states on this in a series of EPSDT guidance, most recently its 2026 toolkit, which encourages states to allow children's behavioral health services without a formal diagnosis and points to states already doing so (CMS, 2024; CMS, 2026). The obstacle is at the state level. Legacy reimbursement pays the most for the F-codes, and providers bill the codes that pay. This will not change until states follow the science and the law, reprice the etiology-informed codes, and rewrite their managed-care contracts to match. So far none have, and even where the codes are permitted, they are recorded for under two percent of Medicaid enrollees (Ubri et al., 2022). That part is a financing decision, and it belongs to the states. But a practitioner need not wait on the state. Understanding a case differently, and recording it differently, is a choice they can make now.

Name the presentation an invalidated DSM disorder, and a parent's impairments become proof of unfitness, and the response is aimed at the individual: a therapist in an office, a prescriber, and the child's removal and placement. Recognize it as the developmental disability it is, in part produced by the legacy system, and the response is the one health care and federal law already provide. Rather than DSM-related treatment and removal, it is early support that builds capability. It works toward school success and community access, it supports the parent alongside the child, and it protects the civil rights, dignity, and agency of both. It treats them

as people to accommodate and value, not as a case opened to fund an agency, a program, or a professional.

The same federal protections extend to parents, whose own impairments are often what an earlier system produced or failed to prevent. And those protections are not only about services. A person with a federally recognized disability is also protected from having their rights trampled or their freedom taken because of it, which is what removal, and the loss of parental rights, can amount to when the impairment behind them is itself a produced disability. Federal law names this protection directly. In federally funded child welfare proceedings, a 2024 Section 504 rule extends disability nondiscrimination to biological and adoptive parents, and the ADA's integration mandate treats the family home as the setting a parent's impairment should be accommodated in, rather than removed from (Section 504 rule, 2024; Olmstead v. L.C., 1999). A permanent disability is not something we medicate away; it is something we accommodate and build around. Same child, same behavior, two different plans, depending on which story you believe about where it came from.

Major life area	How it impairs, and what helps
Learning & thinking	Prefrontal-limbic circuitry built around chronic threat leads to impaired executive function, working memory, and attention. This often appears as reading difficulty, school failure, and trouble concentrating. **What helps:** structure and routine, checklists and reminders, extra time, smaller steps, low-distraction settings, and IEP or 504 accommodations.
Educational achievement	Achievement depends not only on cognitive skill but on regular attendance and continuity. Health problems and repeated placement or school changes add absence and lost instruction on top of the executive-function and language deficits above, so measured achievement falls further behind with each grade. It shows up as grade retention, special-education referral, and low rates of high-school and college completion. **What helps:** keeping the child in one school through placement changes, catch-up instruction and tutoring, and special-education services.
Communication & language	Language-sensitive windows that close in infancy, when disrupted during construction, produce lasting language and communication impairment. **What helps:** speech-language therapy, early intervention, and communication accommodations.
Emotional regulation	An HPA axis and autonomic system calibrated for threat produce affect dysregulation, meaning stress that activates readily and recovers slowly. This limits the capacity to regulate emotional states under pressure. **What helps:** co-regulation and predictable settings, de-escalation rather than punishment, and support to build regulation skills.
Hygiene & self-care	Everyday self-care, such as bathing, grooming, dressing, eating regularly, sleep, and managing appointments and medication, draws on the same executive-function and regulation systems that adversity disrupts, and on routines a co-regulating adult would normally model. When that support is missing, self-care can lapse. In a youth this may be taken as laziness, and in a parent as neglect, when it often reflects limited regulatory capacity rather than unwillingness. **What helps:** routines and prompts, hands-on coaching, and treating a lapse as a support need rather than proof of neglect.
Work & economic participation	Executive-function deficits together with emerging cardiometabolic disease create a combined profile that makes employment precarious and, at mid-life, can force a person out of the workforce decades after the original disruption. **What helps:** workplace accommodation, supported employment, and benefits counseling.

Major life area	How it impairs, and what helps
Housing & stability	The same regulatory impairments, made worse by the loss of relational support, show up later as housing instability and a raised risk of mid-life homelessness. **What helps:** housing support, case management, and supported or independent-living services.
Physical health & bodily function	Systemic inflammatory and endocrine dysregulation cause premature cardiovascular, metabolic, immune, and neurodegenerative disease, along with accelerated biological aging, all of which impair major bodily functions. **What helps:** early screening and ongoing medical care, and treating early adversity as part of the health history.
Reproductive health	A dysregulated maternal HPA axis alters the intrauterine hormonal environment, for example through sharply elevated preterm-birth risk, passing programmed vulnerability to the next generation before birth. **What helps:** prenatal care and support to reduce stress in pregnancy.
Relationships & belonging	The loss of co-regulatory and community relationships removes the support structure people rely on across life, which can produce social isolation that persists into old age. **What helps:** building and preserving relationships, community connection, and mentoring.

7. An unfolding pattern across a lifespan, not a single childhood episode

The harm is not confined to one developmental window; it compounds, and it starts before birth, in a stress axis already programmed in the womb, and continues from there: an infancy without a consistent co-regulator, preschool years when executive function should be forming without anyone to scaffold it, school age without protective relationships, adolescence without family or community. By young adulthood the body already shows early cardiometabolic disease; by mid-life, several conditions at once and a shrinking capacity for work; by old age, accelerated aging and isolation. What was laid down in childhood keeps doing harm long after the circumstances that caused it are gone.

> **THE MECHANISM IN PLAIN TERMS**
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> Severe, chronic adversity, occurring during the sensitive windows without a relationship to buffer it, can produce real impairment through known biological pathways: in learning, language, attention, and emotional regulation, and in cardiovascular, immune, metabolic, and reproductive function. These impairments limit major life areas, they last, and they build on each other over a lifetime. The science behind this is peer-reviewed, and because the harm rises with the dose, we can describe both how likely it is and how bad.

8. What to do differently

A few priorities follow directly from the mechanism.

- **Protect the buffer.** Because the buffering relationship is the factor that matters most, continuity of the child's relationship with a parent or close kin is not a "nice to have." Every avoidable disruption of that relationship removes the one resource that turns adversity from toxic into tolerable.

- **See behavior as biology, not defiance.** Dysregulation, inattention, and difficulty following through are often the output of systems built under threat. Treating them as willful weakens both accurate assessment and the working relationship.
- **Weigh what a placement removes, not only what it provides.** Moving a child away from a parent or close kin can reproduce the same loss of buffering that causes the harm. The relationships and community ties that would be lost belong in the decision, and a stable non-kin placement does not, on the outcome data, make up for them.
- **Build in layers of protective experience.** One buffering relationship keeps stress tolerable and improves a child's odds, but by itself it tends toward survival more than thriving. Better, not well. Doing well tracks instead with how many protective experiences a child can draw on. The Positive Childhood Experiences that predict better adult mental health are several, not one: being able to talk to family, a sense of belonging at school, supportive friends, at least two non-parent adults who take a genuine interest and feeling safe at home. They are layers of relationship, and preserving or creating them counts as intervention, not enrichment.
- **Treat early adolescence as a second window.** The second decade reopens the stress-response system to recalibration. Restoring a stable, co-regulating relationship with a parent or close kin during these years engages an active repair mechanism that adulthood no longer offers, so adolescent placement and reunification decisions have biological weight.
- **Keep realistic hope.** Children who are highly sensitive to harm are often just as responsive to improvement, so better conditions can still change the course. Recovery is possible when a stable relationship with a parent or kin, and their community, is restored.

Some jurisdictions are already working this way, preventing a large share of removals and, when a child cannot safely stay home, placing with kin rather than strangers. The mechanism explains why that matters. Preventing separation keeps the co-regulating relationship intact, so the child's stress stays tolerable, and development is never disrupted. When separation cannot be avoided, kin are far more likely to be a relationship the child's stress system already recognizes as safe, which shortens or prevents the stretch of unbuffered toxic stress a non-kin placement imposes while recognition is built, if it is built at all. Both protect the buffer the science identifies as decisive, which is why they prevent produced disability rather than generate it.

9. Where this aligns with current AAP guidance (2026)

In July 2026 the American Academy of Pediatrics published a paired policy statement and clinical report, *Developmental Challenges of Young Children in Foster Care* (Waite, Spinks-Franklin, and Simkins, with the AAP Council on Foster Care, Adoption, and Kinship Care and the Section on Developmental and Behavioral Pediatrics). It updates the AAP's earlier guidance on the developmental effects of severe or chronic stress from neglect, abuse, family disruption, poverty, and racism, and focuses on children from birth to five, the peak period of early development.

The AAP describes the same mechanism this brief does. It presents the nurturing, responsive caregiver as the critical buffer against stress, notes that early stress without that caregiver alters the neurobiology and architecture of the developing brain and warns that developmental challenges in these children are often seen as behavioral problems. Between 32 and 58 percent of young children in foster care have developmental delays, compared with 13 percent of peers, yet only 10 to 26 percent of those who need developmental services receive them. One recommendation states the paper's argument in the AAP's own voice. The AAP recommends that

foster placement itself be a qualifying condition for early-intervention services, on the same logic used for premature infants. Developmental and emotional challenges are common enough in this population that the placement, not a later diagnosis, is reason enough to refer a child for support. A child who has been in foster care, like a premature infant, carries developmental risk by virtue of the experience, and the clinical report ties that risk to the disruption of the child-parent attachment that placement so often causes. The statement also calls for teaching foster and biological parents that these are developmental rather than willful behaviors, prioritizing kinship placement and timely permanency, and addressing the poverty and structural racism that led to placement. Its call for developmental, social-emotional, and trauma screening is a reminder, not a new standard. That screening has been required for years, set by the AAP's Bright Futures schedule (2017) and mandated for children in Medicaid under the EPSDT benefit (42 U.S.C. § 1396d(r)), and almost no state delivers it on the more frequent schedule set for children in out-of-home care. The recommendation exists because the practice does not.

The companion clinical report reinforces the biology. It states that toxic stress dysregulates the HPA axis and can alter the epigenetic regulation of the stress-response system, that a secure attachment to a responsive caregiver supports self-regulation and brain development, and that kinship placements are associated with better outcomes than non-kin foster care. Each of these points confirms the mechanism and the practice implications above, now with the authority of formal AAP guidance.

Source & scope. This brief draws on the primary literature and frameworks it cites. Its empirical anchors include the AAP ecobiodevelopmental framework (Shonkoff et al., 2012; reaffirmed 2021), the National Scientific Council on the Developing Child working papers, the Felitti & Anda ACE Study, Bethell's Positive Childhood Experiences research, the Belsky/Ellis differential-susceptibility literature, and the adolescent HPA recalibration research (DePasquale, Donzella & Gunnar, 2019; Gunnar et al., 2019, PNAS; DePasquale, 2021). For current AAP guidance on this population, see the 2026 AAP policy statement and clinical report on the Developmental Challenges of Young Children in Foster Care, together with the National Academies' 2025 Early Relational Health report. The legal definitions and protections are set by the ADA, the DD Act, Section 504 and its 2024 rule, and Olmstead; all sources are listed in the bibliography. It is a plain-language brief for practitioners; specific figures should be verified against the original publications before use in formal assessments, reports, or training.

APPENDIX

Plain-language glossary

The brief keeps the scientific terms because they are the terms used in assessments, records, and the research. This glossary gives a plain one-line meaning for each, so the language is a tool rather than a barrier.

Term	Plain-language meaning
ACE (Adverse Childhood Experience)	A potentially harmful childhood experience, such as abuse, neglect, or a household affected by violence, addiction, or separation. Often totaled as an "ACE score."
Adjusted odds ratio (OR)	A figure comparing the odds of an outcome between two groups after accounting for other factors. Below 1 means lower odds; OR 0.28 is about a 72 percent reduction.
Allostatic load	The cumulative wear on the body from stress that stays switched on too long without recovery. "Allostatic overload" is the heavier end of this.
Attachment (secure attachment)	The emotional bond between a child and a caregiver. A secure attachment is one in which the child reliably experiences the caregiver as a safe base to return to under stress.
Autonomic nervous system	The automatic system that controls heart rate, breathing, and the stress response. "Sympathetic dominance" means it stays tilted toward fight-or-flight.
Cardiometabolic disease	Conditions of the heart and metabolism, such as high blood pressure, heart disease, and type 2 diabetes.
Co-regulation	The process by which a calm, attuned adult helps a child's stress system return to baseline. The child borrows the adult's regulation until able to do it alone.
Cortisol (glucocorticoids)	The body's main stress hormones. Helpful in short bursts, harmful when elevated for long stretches. "Cortisol reactivity" is how strongly they rise in response to a challenge.
Differential susceptibility	The finding that the children most harmed by poor conditions are often the same ones who gain the most from good conditions. Sensitivity works both ways.
Dose-response	A pattern in which more of something (here, adversity) produces proportionally more of an effect (here, disease or disability).
Ecobiodevelopmental framework	A pediatric model, adopted by the American Academy of Pediatrics, describing how a child's environment, biology, and development together shape lifelong health.
Epigenetic change (methylation)	Chemical marks that turn genes up or down without changing the genes themselves. Early stress can leave these marks, and some pass to the next generation.
Executive function	The brain's management skills: planning, focusing attention, holding instructions in mind, controlling impulses, and following through.
HPA axis	The hypothalamic-pituitary-adrenal axis, the body's central stress-response system that releases cortisol and, ideally, switches it back off.
Inflammatory tone (cytokines)	The body's baseline level of inflammation. Cytokines are signaling molecules; chronically high levels contribute to disease.

Term	Plain-language meaning
Neuroplasticity	The brain's capacity to be shaped by experience. It is highest during sensitive periods.
PCE (Positive Childhood Experience)	A protective experience, such as feeling supported by family, belonging in a community, or feeling safe at school, that buffers the effects of adversity.
Prefrontal-limbic circuitry	The wiring between the thinking and decision-making brain (prefrontal cortex) and the emotion and threat centers (limbic system). It governs self-control and emotional regulation.
Recalibration	A resetting of the stress-response system. "Pubertal stress recalibration" is a second chance for a threat-tuned system to normalize under supportive conditions during puberty.
Relational health	The health that grows out of safe, stable, nurturing relationships. A growing focus in pediatrics, treated as a foundation for lifelong physical and mental health.
Resilience	Not a trait inside a child. It is the capacity to do well despite adversity, produced by the supportive relationships and conditions around the child, and it can be built at any age.
Sensitive period (developmental window)	A limited window when a developing system is especially open to being shaped by experience, in either direction. Once it closes, change is much harder.
Setpoint	The resting level a body system settles at. Early experience sets the stress system's baseline toward safety or toward threat.
Toxic vs. tolerable stress	Tolerable stress is hard but buffered by a supportive relationship, so the system recovers. Toxic stress is prolonged and unbuffered, so it stays activated and the strain becomes biological.
Vantage sensitivity	A companion idea to differential susceptibility: some people respond especially strongly to positive experiences and support.
Congregate care	Group residential settings such as group homes or institutions, rather than family-based placement.

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