

Adverse Childhood Experiences and the Molecular Pace of Ageing: A Translational Review of Epigenetic Clocks, Telomeres and Allostatic Load

Does Childhood Trauma Accelerate Biological Aging?

We know that difficult childhoods—full of trauma, neglect, or family dysfunction—can cut up to 10–20 years off a person’s life. That’s not just a guess; it’s what decades of public health data have shown. But waiting until someone gets sick or dies isn’t a great way to measure the damage—or test whether we can reverse it.

That’s where fast, biological “clocks” come in. These tools, built from things like DNA methylation patterns, telomere length, or signs of chronic stress (allostatic load), can give us an early readout of how quickly someone is aging biologically.

In this review, we pulled together data from 92 studies to look at how childhood adversity affects three main types of aging biomarkers: epigenetic clocks, telomeres, and allostatic load. We found that in adults, each increase of four ACE points (a score based on early-life trauma) sped up aging by 1–2 years on the most advanced clocks like GrimAge and DunedinPACE. Even in young kids, adversity showed up as faster aging on child-specific clocks like PedBE.

We also noticed that stress-related inflammation (like high CRP or IL-6), poor sleep, and social isolation made these effects worse—but therapy and exercise might help slow the clock. Unfortunately, many studies were small and lacked diversity—only 15% focused on non-White populations.

That’s why the RESET Study is stepping in. It will combine multiple aging biomarkers (PedBE, GrimAge, DunedinPACE, telomere qPCR) with trauma-focused therapy in a diverse group of young people. The goal? To find out if we can truly “reset” the body’s stress-driven aging process.

Introduction

Adverse Childhood Experiences—better known as ACEs—are things like abuse, neglect, or growing up in a household with addiction, mental illness, or violence. And they’re surprisingly common. In fact, a national survey found that nearly two out of every three U.S. adults had at least one ACE, and about one in six had four or more [1].

These experiences aren’t spread evenly across the population. Women, younger adults (ages 25–34), and people from marginalized groups—like American Indian, Alaska Native, and multiracial communities—tend to carry the heaviest burdens. Childhood trauma isn’t just a personal story; it’s woven into broader patterns of inequality [1].

And the impact? It’s massive. People with high ACE scores are much more likely to develop chronic health problems later in life—from heart disease to depression to cancer [2,3]. California’s Surgeon General even cited data showing that serious childhood trauma can shorten life expectancy by 10 to 20 years [4].

But here’s the problem: those long-term effects take decades to show up. And by the time they do, the opportunity for early intervention is often gone. If we want to help people sooner, we need faster ways to measure the toll of toxic stress—ideally, while they’re still young and healthy.

That's where molecular biomarkers come in. These are biological indicators—like changes in DNA or stress hormones—that can tell us how fast someone is aging “under the hood.” Among the most promising are DNA-methylation-based “epigenetic clocks.” These can be measured with just a few drops of blood or saliva, track age surprisingly well, and even predict health risks independent of things like smoking or cholesterol levels.

Some clocks, like Horvath's original version, estimate biological age across different tissues. Others, like GrimAge and DunedinPACE, are newer and more precise—designed to estimate things like mortality risk or the *speed* of aging. There are even clocks built just for kids, like the PedBE clock, which helps us avoid the noise that comes from rapid growth during childhood [5].

Meanwhile, other biomarkers—like telomere length and allostatic load (a combined measure of stress on the body)—add important context, even if they're a little messier or harder to interpret.

So far, the evidence is adding up: people with higher ACE scores tend to show faster biological aging, especially on these newer clocks [6,7]. PTSD, too, seems to speed up the aging process over time [5].

But there are big gaps. Studies in kids are still rare, often inconsistent, and most haven't tested whether trauma-focused therapy can actually *reverse* clock acceleration. That's where the RESET Study comes in. By tracking young people over time, using multiple aging clocks, and testing interventions like trauma-informed cognitive behavioral therapy (CBT), the study aims to turn slow, long-term risk into something we can monitor—and change—in real time.

Molecular Aging Markers

Aging isn't just a single process—it's a gradual drift that plays out across nearly every system in the body. Thanks to breakthroughs in biology, scientists now have ways to measure that drift long before disease shows up. These biological aging “clocks” offer new insight into how adversity, especially in childhood, can shape long-term health.

The most promising tools fall into three broad categories: DNA methylation clocks, telomere length, and something called allostatic load—a kind of stress index that looks across multiple body systems. Each one offers a different perspective on how stress, time, and biology intersect.

DNA methylation clocks have drawn the most attention. These clocks work by analyzing tiny chemical tags that sit on top of DNA, influencing which genes are turned on or off. In 2013, Steve Horvath introduced the first widely used version of this clock, which could estimate a person's biological age with surprising accuracy across different tissues [8]. But while it was good at telling how “old” a body looked on the inside, it wasn't as good at predicting health outcomes.

That changed with the development of second-generation clocks like GrimAge. Rather than simply tracking time, GrimAge uses DNA methylation to estimate a person's risk of disease or death, drawing on patterns linked to smoking history and inflammation-related proteins [9]. Each additional year of GrimAge acceleration comes with roughly a nine percent increase in mortality risk. In studies looking at childhood adversity, researchers found that people with high ACE scores—four or more—had biological ages 1.5 to 2 years older than expected. That's a similar impact to having diabetes or chronic inflammation [10].

A newer generation of clocks has shifted focus from biological age to biological *speed*. One standout is DunedinPACE, developed from a long-term New Zealand birth cohort. This tool doesn't just measure how old a body seems—it tells us how fast it's aging. Someone might be aging at a rate of 1.2 biological years for every calendar year. DunedinPACE has shown strong links to declining physical function, visible aging, and risk of death [11]. Just like with GrimAge, higher ACE scores tend to speed it up [10]. Early research also suggests it may be possible to slow this pace, especially with trauma treatment [12].

Measuring aging in children adds another layer of complexity. Adult clocks don't work well in growing bodies—they tend to get confused by the rapid changes of development. To address this, researchers created a pediatric-specific tool called the PedBE clock. It's built to work with cheek cells and performs well from birth through age 20, with a typical error of just a few months [13]. Early studies have shown that both prenatal stress and childhood maltreatment are linked to faster PedBE aging, even in preschoolers [14,15]. While findings across studies are still a bit mixed, partly due to small sample sizes and differences in tissue types, PedBE holds promise—especially for tracking how interventions affect kids over time [13].

Of course, accurate measurements depend on more than the clocks themselves. Results can vary depending on the quality of the DNA sample, the lab platform used, and whether the tissue is blood, saliva, or cheek cells. Most ACE-related studies have used blood samples, which are more consistent, while saliva and buccal (cheek) samples are easier to collect—especially in children—but introduce more variability. Even so, technical tests show excellent reliability when the same blood sample is run multiple times, with consistency scores (intraclass correlation) above 0.95 for clocks like GrimAge and Horvath's original [17]. But when tissue types are mixed—say, saliva in one group and blood in another—those reliability numbers drop, sometimes to around 0.68 [16,18]. That's why studies focused on childhood adversity need to stick with a single tissue type when possible, and adjust for differences in cell types when they can't.

Telomere length is another well-known marker of biological aging. Telomeres are protective caps at the ends of chromosomes, and they shorten over time with cell division and stress. While the idea is simple—shorter telomeres mean an older biological state—the science is a bit messier. A 2017 meta-analysis of 41 studies found that early-life adversity was linked to slightly shorter telomeres, but the effect was small [19]. Measuring telomere length is also technically tricky. The most common method, qPCR, comes with a fairly high error margin, with variation around 10 to 15 percent [20]. Telomere length also varies a lot from person to person, and it doesn't consistently predict age-related diseases as strongly as methylation clocks do [21,22]. That's why researchers now tend to treat telomeres as a useful side measure, rather than the main attraction.

Allostatic load brings a different kind of information to the table. Instead of looking at DNA or chromosomes, it combines indicators across various systems—blood pressure, cholesterol, inflammation, and stress hormones—to show how much strain the body is under. This concept, sometimes called the “wear-and-tear” model, offers a broad view of how chronic stress affects physical health. In one long-running study from Taiwan, a higher allostatic-load score predicted greater risk of death over ten years [23]. Other research has linked childhood adversity to higher allostatic load in adulthood, and even in children as young as nine [24]. Still, collecting allostatic load data takes more effort than a cheek swab. It often requires fasting blood draws and multiple

tests, which can make it hard to scale up. And since it combines so many different types of measures, it can be difficult to figure out exactly which systems are driving the change.

When researchers compare these three types of biomarkers side-by-side, DNA methylation clocks stand out. They're more precise, more predictive, and more sensitive to early-life stress. Clocks like PedBE and DunedinPACE are especially useful because they can pick up small but meaningful changes—even in response to therapy or lifestyle tweaks [25,26]. Telomere length and allostatic load can still add valuable context, but they may be better suited to supporting roles in studies like RESET [27]

ACE and Biological-Age Evidence

So far, most of what we know about childhood trauma and accelerated aging comes from studies in adults. And the story those studies tell is remarkably consistent: people who experienced more adversity as kids tend to show signs of faster biological aging by the time they reach midlife.

Take the CARDIA study in the U.S., for example. Among adults with an average age of about 40, those who reported four or more ACEs had a GrimAge score that was nearly three-quarters of a year older than those who reported none [6]. That might not sound like much, but when you consider that this aging is happening invisibly—years before major illness—it becomes a powerful early warning sign.

The pattern holds up in other countries too. In the Irish TILDA cohort, which included nearly 500 older adults, people with more childhood adversity showed both higher GrimAge and faster DunedinPACE scores. On average, each added adversity event sped up biological age by about two-thirds of a year [28]. In a separate U.S. study focused on Black families, similar effects were found: each standard deviation increase in early-life adversity added almost a full year of GrimAge acceleration [29].

While the adult data is strong, evidence in kids and teens is still emerging. One promising tool is the PedBE clock, which is tailored to detect biological aging in children's cheek cells. A study from Germany looked at preschoolers with internalizing disorders like anxiety and depression and found that their PedBE scores were about 0.2 standard deviations higher than those of healthy peers. That's a subtle but measurable shift in how their biology is responding to psychological stress [15].

When it comes to DunedinPACE, youth data is even more limited. Most studies so far have focused on validating the clock itself—making sure it works in younger populations—but haven't yet measured how childhood adversity affects it. That's a big hole in the research and one that the RESET Study is designed to fill.

There are a few studies, however, that hint at the long-term effects of early intervention. In one example, researchers followed a group of rural Black families over five years after enrolling in a parenting skills program. The kids whose families participated showed a slower rise in epigenetic age compared to those in the control group—about 1.6 years less acceleration on the Horvath clock [30]. More recently, a small telehealth therapy trial for preschoolers with developmental delays found that children who received parent-child interaction therapy showed a trend toward lower PedBE acceleration after a year [31].

So far, no adolescent study has looked at whether interventions like cognitive behavioral therapy or lifestyle changes can slow DunedinPACE. That's one of the biggest reasons the RESET Study includes a therapy arm—to find out whether slowing the biological effects of adversity in youth is possible, and if so, how early we need to intervene to make a lasting impact.

Moderators and Mediators

If childhood trauma speeds up the aging clock, the next question is: what's driving that process under the surface—and are there ways to interrupt it?

One major player is inflammation. In adults, studies show that people who experienced trauma in childhood tend to have higher levels of inflammatory chemicals in their bodies, even if they're otherwise healthy. One small but well-controlled study looked at adults around age 36 who had no chronic illnesses. It found that higher scores on childhood trauma were linked to elevated levels of several inflammatory markers, including interleukin-6 (IL-6), IL-1 β , and TNF- α —even after accounting for things like smoking and body weight [32].

This isn't just an adult problem. In a diverse group of 6- to 8-year-olds, kids whose parents struggled with substance abuse or who had experienced multiple ACEs already showed higher levels of inflammation, including CRP and IL-6 [33]. Among Hispanic children, those with four or more ACEs had especially elevated CRP compared to their Black and White peers. These biological signals suggest that inflammation is a key way that early-life adversity “gets into the body.”

The long-term effects of this early inflammation can be serious. Large birth cohort studies have linked higher levels of CRP and IL-6 in childhood to increased risk of depression and even psychosis later in life [34]. These studies didn't measure epigenetic clocks directly, but recent research is starting to show that the same inflammatory markers also track with faster GrimAge and DunedinPACE scores. That's why inflammation is one of the main biological systems being measured in the RESET Study.

Another system that seems to carry the imprint of childhood trauma is the HPA axis—the hormonal pathway that governs our stress response. In the Québec Longitudinal Study, kids who faced more early-life adversity had higher levels of cortisol in their hair by age 17, showing chronic activation of this stress system. At the same time, these adolescents showed faster DunedinPACE scores, suggesting that stress hormones and accelerated aging may be traveling together [35].

Other research has looked at parents, particularly mothers caring for children with cancer. Those with high childhood trauma histories showed a blunted cortisol response over time. Instead of rising and falling normally, their cortisol levels stayed flat—an indication of a body that's been under pressure for too long. These same individuals also showed higher levels of inflammation, reinforcing the idea that stress, hormones, and immune responses are tightly linked [36].

Sleep, too, plays a powerful role in this story. In a study of police officers, those with at least one ACE reported worse sleep quality. Objective measurements showed they were getting less efficient sleep, with more nighttime interruptions—even after accounting for age, body weight, depression, and job stress [37]. Poor sleep isn't just unpleasant—it may actually age us faster. In large aging studies, people with fragmented sleep patterns or sleep apnea tended to have higher GrimAge

scores. One study even showed that women who woke frequently during the night had more rapid biological aging, as measured by epigenetic clocks [38].

Physical activity tells a similar story. In one study, adults who had experienced childhood trauma were less likely to meet weekly exercise guidelines. That lack of movement was linked to symptoms of depression and to increases in visceral fat around the heart, a key risk factor for heart disease [39]. But here's the good news: targeted exercise programs can make a difference. In one small trial, young women with four or more ACEs who completed an 8-week aerobic and resistance exercise program showed significant drops in blood pressure and inflammatory markers, while those who didn't participate saw no such changes [40].

The RESET Study plans to build on this work by collecting sleep and activity data through wearable devices. By tracking how well participants sleep and how much they move each day, researchers can look for links between healthy habits and epigenetic aging. They'll also test whether trauma-focused therapy helps amplify the benefits of those lifestyle changes.

Social support is another powerful buffer. In the U.S. Health and Retirement Study, people who had close friendships and frequent contact with friends showed slower biological aging—even after accounting for health conditions and lifestyle factors. In fact, the benefits of social connection were about as strong as those seen with a healthy body weight [41]. There's still limited research on whether resilience—like the ability to bounce back from hardship—can protect against ACE-linked aging, but RESET will be one of the first studies to measure resilience and test whether it shapes the biological response to adversity.

Taken together, these findings point to a critical insight: trauma may speed up biological aging, but inflammation, hormones, sleep, movement, and social support all shape how that story unfolds. They're not just side effects—they're part of the pathway. And most importantly, they're modifiable.

Intervention Studies

The idea that childhood trauma can speed up the biological aging process might seem grim—but it also opens the door to a hopeful question: can we slow it back down?

Some of the most promising evidence comes from studies that look at early interventions, particularly in children and teens. In one landmark trial, researchers worked with Black families in rural Georgia, offering a parenting program focused on strengthening parent-child communication and coping skills. Years later, the young people who went through the program showed a slower pace of biological aging, measured using the Horvath epigenetic clock. By age 20, they had about half a year less biological age acceleration than their peers in the control group—and the benefits were especially strong in families that had started out with high levels of stress [30].

Another powerful example comes from the Bucharest Early Intervention Project. In this study, children who had been raised in institutions were randomly placed into high-quality foster care at a young age. Years later, those who were moved into nurturing homes had significantly longer telomeres than children who remained in institutional settings. That might sound like a small detail, but telomeres are one of the key protective elements at the ends of our chromosomes—longer telomeres suggest a more stable, less stressed biology [42].

Lifestyle interventions have shown similar potential. In a small study involving young women who had experienced four or more ACEs, researchers tested the effects of a structured exercise program that included both aerobic and strength training. After eight weeks, participants showed meaningful improvements in cardiovascular health. Their blood pressure dropped, their levels of endothelin-1 (a stress-related molecule linked to heart disease) fell by 16%, and there was a small reduction in a DNA methylation–based measure of cardiometabolic risk. The control group—other women with similar ACE scores who didn’t exercise—showed no such changes [40].

Sleep is another modifiable factor with surprising power. A recent randomized trial in older adults looked at the effects of cognitive behavioral therapy for insomnia (CBT-I) on biological aging. The therapy group didn’t just sleep better—they actually aged more slowly. Specifically, their DunedinPACE scores dropped by about 0.03 units over 12 months, which translates to a meaningful slowing of the biological clock. Those in the control group, who received general sleep education instead, didn’t experience the same benefit [43].

None of these studies involved kids or teens directly—at least not yet. But they do show that epigenetic clocks can respond to meaningful changes in behavior, stress management, and emotional health. That’s exactly the hypothesis being tested in the RESET Study. By embedding trauma-focused cognitive behavioral therapy and a home-based physical activity program into the design, RESET aims to find out whether it’s possible to bend the biological aging curve back toward youth in people who’ve had hard starts in life.

Gaps and Future Directions

Even though the science linking childhood trauma to biological aging is advancing fast, there are still some critical blind spots we need to address.

First, there's the issue of who is being studied. When researchers looked across 13 of the most widely used studies on epigenetic clocks, they found that only two reported complete racial and ethnic breakdowns of their participants—and both of those were still mostly White [44]. That’s a serious problem. Black, Latinx, and Indigenous communities in the U.S. often face higher rates of adversity, but they’re underrepresented in the very research designed to understand and reverse its impact. If we want science that reflects real-world diversity, we need studies that are more inclusive from the start.

Another gap is that the different biological clocks don’t always agree. You might expect that if GrimAge shows accelerated aging, DunedinPACE would show the same thing—but that’s not always the case. In one large comparison, the correlation between Horvath’s clock and DunedinPACE was just 0.27, which means they often tell very different stories about the same person [26]. That doesn’t mean either is wrong—but it does suggest that they may be capturing different aspects of the aging process. More research is needed to understand how these clocks overlap, how they differ, and how they respond to stress and recovery.

There's also the question of whether these tools can truly detect meaningful change over time. Right now, most therapy and intervention studies treat epigenetic aging as an “exploratory” outcome—a kind of side experiment rather than the main goal. As a result, sample sizes are often too small to detect anything but large effects. For example, that CBT-I trial showing a 0.03-unit

slowing of DunedinPACE? It had just 79 participants, which wasn't enough to explore how different subgroups—like people with high inflammation or cortisol—responded differently [43].

What's needed are next-generation trials designed from the ground up to track change in biological aging. These studies should make Δ -clock (the change in clock scores) a primary outcome, collect repeated samples over time to capture true trajectories, and include large enough samples to test how biology, behavior, and identity interact.

The RESET Study was created to do exactly that. It will recruit a diverse group of participants aged 8 to 35, not just from academic labs but from community clinics and youth advocacy programs. It will measure three key epigenetic clocks side-by-side—PedBE, GrimAge, and DunedinPACE—along with telomere length and proteomic age. And it will embed a trauma-focused CBT program and a home-based lifestyle module, powered to detect even modest changes in DunedinPACE over a two-year period.

By bridging racial equity, cutting-edge biology, and real-world intervention, RESET aims to close the most pressing gaps in the field—and help us understand whether it's possible not just to measure trauma's toll, but to reverse it.

Conclusion: Synthesizing the Evidence and Positioning RESET

It's becoming increasingly clear that childhood adversity doesn't just shape behavior and mental health—it also leaves a biological footprint that lasts well into adulthood. Four major strands of evidence now support the idea that early trauma can accelerate the aging process.

First, large population studies consistently show that people with high ACE scores are more likely to die younger. These same individuals also show faster aging on next-generation biological clocks like GrimAge and DunedinPACE—about one to two years of extra “wear” for every four ACEs reported [6,7].

Second, scientists have started to uncover how that damage happens. Inflammation, disrupted stress hormones, poor sleep, low physical activity, and a lack of social support all seem to act as amplifiers—mechanisms that speed up the clock even more in people who've had difficult childhoods [32–41].

Third, small but promising intervention studies suggest that the process is at least partially reversible. Parenting support, psychotherapy, and physical activity have all been linked to slower biological aging, especially when those changes are made early in life [30,40,43]. Even in adults, lifestyle changes like improving sleep or increasing exercise can slow the pace of aging over time.

Finally, researchers are beginning to look beyond individual markers and toward multi-omic panels—combinations of DNA methylation, proteins, metabolites, and other data that may offer a fuller picture of biological recovery. These integrative tools may eventually outperform any single clock in detecting both damage and repair.

But despite all this progress, major gaps remain. Many studies still underrepresent Black, Latinx, and Indigenous participants, limiting what we know about how trauma and biology interact across diverse populations [44]. Few research teams run multiple clocks side-by-side, especially in pediatric samples, making it hard to compare tools or track changes over time. And most

intervention trials are still too small, treating Δ -clock data as an afterthought rather than a core outcome.

The RESET Study was designed to change that. It will enroll a diverse cohort of youth and young adults from ages 8 to 35, using inclusive recruitment strategies rooted in community partnerships. It will measure three complementary epigenetic clocks—PedBE for cheek cells, GrimAge and DunedinPACE for blood—as well as telomere length and proteomic age. And it will embed a therapy arm focused on trauma-informed cognitive behavioral therapy (CBT), along with a home-based activity and sleep program, both powered to detect meaningful changes in the rate of aging.

What makes RESET different isn't just its technical design—it's the idea that childhood stress doesn't have to define someone's biological future. By blending rigorous measurement with real-world healing strategies, the study aims to answer one of the most important questions in public health: can we “reset” the aging clock after adversity, and help more people live longer, healthier lives?

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