



Benevolent Childhood Experiences (BCEs) and Neuropsychological Outcomes in Former Professional American-style Football Players

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Benevolent Childhood Experiences (BCEs) and Neuropsychological Outcomes in Former
Professional American-style Football Players

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A Thesis in the Field of Biology
for the Degree of Master of Liberal Arts in Extension Studies

Harvard University

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Abstract

Research indicates that childhood experiences significantly shape neuropsychological function, impacting mood and cognitive function (e.g., deficits in memory, executive function, and mood regulation) in adulthood. Benevolent childhood experiences (BCEs) such as emotional support, stable caregiving, and safe, nurturing environments are shown to be associated with better mental health outcomes in general population studies.

Mood and cognition have been shown to be affected by previous exposure to repetitive head impacts, often experienced by contact sport athletes and military personnel. While previous research has shown that former American-style football (ASF) players experiencing many Adverse Childhood Experiences (ACEs) have reported worse neuropsychological outcomes, the relationship between BCEs and neuropsychological outcomes in populations exposed to repeated traumatic brain injuries, such as former professional ASF players, remains largely unexplored.

This study utilized data from the Football Players' Health Study at Harvard University (FPHS) to examine BCEs among former professional ASF players, and how BCEs are associated with neuropsychological function. This data set included self-reported measures of BCEs, ACEs, and neuropsychological outcomes in former professional ASF players, in addition to demographic and football-related data. Specifically, this study evaluated the extent to which BCEs are associated with mood and cognition, and how these associations may change after adjusting for ACEs. BCEs and

ACEs were assessed using the Adverse Childhood Experiences Questionnaire and the Benevolent Childhood Experiences Questionnaire, and neuropsychological outcomes including depression, anxiety, and self-reported cognition were measured using the Patient Health Questionnaire-9 (PHQ-9), Generalized Anxiety Disorder-7 (GAD-7), and the Neuro-Quality of Life in Neurological Disorders (Neuro-QoL), respectively.

Multivariable generalized estimating equation models were used to examine associations between BCEs and neuropsychological outcomes, with sequential adjustment for demographic characteristics, football-related exposures, and ACEs. I hypothesized that BCEs would provide a protective mechanism against neuropsychological health in former professional ASF players. The principal finding of this thesis was that BCEs were highly prevalent in this population and were associated with more favorable neuropsychological outcomes in minimally adjusted models, but these associations were attenuated after adjustment for ACE burden and adult health-related covariates. This suggests that, in this cohort, BCEs did not demonstrate strong independent protective associations in fully adjusted models. Furthermore, the persistence of ACE associations for depression and anxiety was consistent with prior literature in former ASF players. Understanding these associations could provide insights into protective factors that enhance resilience within this high-risk group.

Author's Biographical Sketch

Ayesha Zahrah is a master's candidate with interdisciplinary interests in neuroscience, community health, and Islamic finance. At Harvard Medical School, she conducts research through the Football Players' Health Study (FPHS) at Harvard University, alongside supporting multiple initiatives focused on long-term outcomes among former professional athletes. Her scientific training was begun at Massachusetts General Hospital and Harvard Medical School, where she worked on research examining autism spectrum disorders and gastrointestinal comorbidities. In parallel, she has a longstanding interest in Islamic finance and its ethical foundations. She aspires to build a career in neuroscience research that strengthens patient care and expands understanding of brain health across diverse populations.

Dedication

Thank you to my family and friends, whose love and prayers carried me through the hardest moments and that made this journey at Harvard possible.

Most importantly, I'd like to thank God for blessing me the health and strength to continue my thesis despite all the trials and tribulations.

Acknowledgments

I would like to express my deepest gratitude to the many individuals who have guided, supported, and inspired me throughout the course of this research and my graduate studies. This thesis represents not only months of academic work, but years of mentorship, encouragement, and personal growth, for which I am sincerely grateful.

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I would also like to thank my Research Advisor, Dr. James Morris, for your guidance, thoughtful insight, and steadfast support from the earliest stages of this project to its completion. Your expertise and encouragement have shaped my development as a researcher, and I am profoundly appreciative of the trust and intellectual space you gave me to explore this work.

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project. Thank you for helping me think through analytic decisions, for troubleshooting with patience, and for ensuring this work was as thoughtful and rigorous as possible.

I extend heartfelt thanks to my Academic Advisor, Joan Short, as well as Trudi Goldberg Pires and Gail Dourian, for the academic support, advising, and direction across my coursework and thesis preparation. Your careful guidance, willingness to help, and genuine investment in my success made this process smoother, more attainable, and deeply meaningful. Thank you for building a framework that allows students like me to feel supported and empowered at every step.

My sincere appreciation also extends to the staff, collaborators, and advisors of the Football Players Health Study at Harvard University. I am especially and wholeheartedly grateful to Cheyenne Brown and Heather DiGregorio, whose support and generosity with their time sustained me throughout this process. I would also like to sincerely thank Robyn Keske for her mentorship, guidance, and constant encouragement throughout my work at FPHS. I also extend my gratitude to the participants, whose time, insight, and willingness to share their experiences made this research possible. Working within FPHS has not only informed this thesis but has also shaped my professional identity. I am deeply thankful for the opportunity to learn from and contribute to such meaningful and impactful work.

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Chapter I.

Introduction

Benevolent childhood experiences (BCEs) and adverse childhood experiences (ACEs) are both types of childhood experiences that can impact adult health in many ways. Previous research suggests that positive and negative childhood experiences can be associated with later physical health, mental health, cognitive function, and longevity (Zhou et al., 2025). BCEs, such as nurturing and supportive relationships (Narayan et al., 2018), may promote resilience and offer long-term protection against adverse neuropsychiatric outcomes, whereas ACEs, including childhood abuse and neglect (Felitti et al., 1998), are expected to increase risk for these outcomes (Hughes et al., 2017). While both types of experiences are important priorities for future research, the literature on ACEs exceeds the current evidence for BCEs, leaving important questions about how BCEs and ACEs each contribute to adult health still unanswered.

ACEs are not rare or just occurring in one geographical region; rather, where you grow up shapes your risk of experiencing adversity (Merrick et al., 2018). The Behavioral Risk Factor Surveillance System (BRFSS) highlights the high prevalence of ACE exposure in the United States. Using 2019–2020 BRFSS data, Aslam et al. (2024) found that 62.8% of U.S. adults experienced at least one ACE. Importantly, this burden is not randomly distributed but instead reflects broader social and demographic inequalities. Differences in ACE exposure by socioeconomic status and race or ethnicity indicate that adversity is shaped not just by what happens within a family, but also by broader social conditions like poverty, racism, and a lack of resources. State-level variation, with

estimates for at least one ACE ranging from approximately 54.9% in Connecticut to 72.5% in Maine, provides additional evidence that ACEs are patterned across populations rather than randomly dispersed (Aslam et al., 2024). ACEs are not evenly distributed and can fall harder on children who already face the most disadvantage.

ACEs and the Cumulative Framework

Importantly, not all ACEs are equally common, and this matters for research that seeks to link childhood experiences to specific adult outcomes. Aslam et al. (2024) found that emotional abuse was among the most frequently reported ACE type, while household member incarceration was among the least common. However, ACEs are not simply one exposure, but a constellation of experiences that vary across populations and may affect adults differently. A single ACE score captures the overall burden of adversity a person has experienced from birth up until age 18. Most research has measured cumulative burden of adversity, rather than any single type, that most strongly predicts poor adult health outcomes (Felitti et al., 1998; Aslam et al., 2024). Therefore, this thesis measures ACE exposure as a cumulative total score, capturing the overall burden of childhood adversity rather than isolating exposure types.

Alley et al. (2025) further demonstrate that childhood adversity is associated not only with adult mental health, but also with a broad range of physical health dysfunctions. In their analysis of adult health, childhood adversity was associated with differences across 25 disease-related biomarkers and 20 health conditions, further supporting the idea that early stress exposure may become biologically embedded across multiple systems. Their findings showed a dose-response pattern, where the more adversities a person experienced resulted in greater risk for health problems. The original

ACE study, Felitti et al. (1998), found that these exposures frequently co-occurred with one another, and that if a person experienced one type of adversity, it increased the likelihood of them also reporting others. These findings both reinforce that a cumulative ACE score would be the best way to measure impact and to raise questions about whether BCEs may serve as a protective factor.

A cumulative framework raises an important question for BCE research: why do outcomes vary so widely among individuals with similar ACE burdens? Not all adults with high ACE exposure show severe impairment, and many demonstrate meaningful resilience. This raises an important question about what protective experiences might explain why some people cope better than others despite similar levels of adversity.

BCE Frameworks

Narayan et al. (2018) introduced the BCE scale to measure positive and supportive experiences during childhood, designed to mirror the same cumulative approach used for ACEs. Their findings showed that higher BCE scores were associated with lower depression, PTSD, and stress in adulthood, even after accounting for ACEs (Narayan et al., 2018). This suggests that positive childhood experiences have their own direct and lasting benefits on mental health, not just as a buffer against adversity, but as protective resources.

Researchers have increasingly emphasized the need to measure BCEs: supportive relationships, feelings of safety, predictable routines, and other positive experiences that may promote adaptive development. BCEs sometimes framed as “counter-ACEs,” are one way of capturing these kinds of positive experiences as a cumulative resource. Importantly, BCEs are not simply the inverse of ACEs, a person may experience

significant childhood hardship while also having people who cared about them. That matters because it means resilience is not just about avoiding adversity, but about having support even when adversity is present.

Crandall et al. (2019) extend this work into a general adult sample by conceptualizing BCEs as “counter-ACEs” and situating them in Resiliency Theory. In their survey study, higher counter-ACE scores were associated with improved adult well-being across multiple domains (including lower stress, depression indicators, and better psychosocial functioning), and many associations persisted after accounting for ACEs and demographics (Crandall et al., 2019). Notably, their results aligned most strongly with the compensatory model they used. BCEs appeared to have their own independent protective effect on adult outcomes, rather than simply canceling out the harm of ACEs. They also observed that BCE benefits were reduced among those with very high ACE exposure (e.g., ≥ 4 ACEs), suggesting that protection may have limits under severe adversity (Crandall et al., 2019).

BCEs as Protective Resources: Main Effects vs Buffering

The BCE literature shows that BCEs having their own promotive effects do not necessarily mean they will also buffer or cancel out the effects of ACEs. Doom et al. (2021) studied ACEs during the COVID-19 pandemic and found that ACEs were associated with higher depressive symptoms, while BCEs were associated with fewer depressive symptoms, even after accounting for factors like current stressors, social support, socioeconomic status, and other proximal predictors (Doom et al., 2021). BCEs were also linked to lower perceived stress and loneliness, suggesting BCEs may shape not only clinical symptom severity but broader social and emotional well-being (Doom et

al., 2021). Yet importantly, the study did not find evidence that BCEs moderated the association between ACEs and mental health outcomes. This pattern reinforces that BCEs should not be assumed to function primarily as buffers. Instead, they may operate as independent positive resources that contribute to better outcomes regardless of adversity level, with buffering appearing in some samples but not others (Doom et al., 2021).

Hou et al. (2022) used a four-group framework (high/low ACEs $\times$ high/low BCEs) and observed that greater ACE exposure was associated with higher distress while greater BCE exposure was associated with lower distress, with some evidence that high BCEs reduce risk among those with high ACEs. However, the protective pattern was not consistent across all outcomes, especially for more severe outcomes such as suicidal ideation, aligning with the idea that buffering may weaken at higher levels of risk or symptom severity (Hou et al., 2022).

Similarly, Nahar et al. (2025) found that BCEs moderated the relationship between ACEs and adult depression, anxiety, and stress symptoms, such that higher BCE scores were associated with weaker ACE associations. Using Johnson–Neyman probing, they found that this moderation was statistically evident up to approximately nine BCEs, after which further increases in BCEs did not produce additional detectable attenuation of ACEs. This suggests that while BCEs can buffer the effects of adversity, that protection may have a limit at higher ACE exposures.

How Early Experiences Shape Adult Health

Beyond identifying associations, an increasingly important area of study in the BCE/ACE literature concerns the biological and psychological mechanisms through which early experiences shape adult mental health outcomes. Research has shown that

adversity can affect multiple biological systems, including stress hormones and inflammation, in ways that contribute to mental health problems later (Koss & Gunnar, 2018). Feiler et al. (2023) examined BCEs and ACEs in relation to psychopathology symptoms and tested emotion dysregulation as a pathway. In a large sample of adults, ACEs were associated with higher PTSD, depressive, and anxiety symptoms and greater likelihood of positive symptom screens, whereas BCEs were associated with lower symptom levels and reduced likelihood of positive screens (Feiler et al., 2023). Difficulty regulating emotions helped explain part of the link between early experiences and adult mental health for both BCEs and ACEs. Although Feiler et al. (2023) also tested BCEs as moderators, buffering effects accounted for relatively little, suggesting that BCEs work more consistently as their own promotive factor rather than as a direct offset to ACEs. From this perspective, BCEs are important as they function to provide safe and stable experiences that help people regulate their emotions and may benefit mental health long-term.

BCE Measurement and Distribution Issues

One important finding from BCE research is that positive childhood experiences are common but not evenly distributed. Using a nationally representative sample of young people ages 11–19 in Northern Ireland, Redican et al. (2022) reported high BCE exposure overall, with most participants reporting the most common items including having at least one good friend, opportunities for enjoyment, and predictable home routines. At the same time, a small but important subgroup reported no BCEs, indicating that some youth lack even basic protective resources (Redican et al., 2022). The BCE scores were heavily skewed toward the top of the scale in this sample, which can make it

harder to detect meaningful differences between people and limit what the data can tell us statistically. These features make it harder to detect differences between people statistically, especially when BCEs cluster at high levels.

Redican et al. (2022) also demonstrate that BCE exposure is socially patterned. Lower BCE scores were associated with socioeconomic disadvantage (e.g., household receipt of social welfare) and higher area-level deprivation, whereas higher BCE scores were associated with markers of stability and resources such as living with both biological parents and higher parental education. This is important for adult health research because it suggests BCEs may reflect access to supportive relationships and stable environments that not everyone has.

Early Life Experiences in Former Professional ASF Players

ACEs and BCEs are worth studying in populations such as former professional ASF players, who already face elevated risks for neuropsychiatric and cognitive issues. Former professional ASF players are one such population, given documented concerns related to cognitive symptoms, mental health burden, and neurologic health (Alosco et al., 2023).

Evidence suggests cognitive concerns are common among former players, and objective neuropsychological testing identifies areas where they tend to struggle (Alosco et al., 2023). In a cohort of former college and professional football players from the DIAGNOSE CTE project, Alosco et al. (2023) reported that issues in verbal episodic memory, was among the most frequently observed impairments. This shows why it is important to measure cognition both through objective testing and through how players subjectively report their daily functioning.

Within this context, Roberts et al. (2022) also provided evidence linking childhood adversity to later neuropsychiatric and cognition-related outcomes in former NFL players. Using data from 1,755 former professional players in the Football Players Health Study (FPHS), they assessed 10 ACEs and examined associations with dementia symptoms screening (AD8), cognition-related quality of life (Neuro-QoL cognitive function short form), depression (PHQ-9), anxiety (GAD-7), and pain outcomes. Their results indicated that players reporting ≥ 4 ACEs showed elevated risk across multiple neuropsychiatric outcomes relative to those reporting none, including increased risk of screening positive for dementia symptoms and worse scores across several symptom domains (Roberts et al., 2022). This is especially meaningful because it looks at childhood adversity in men who also spent careers absorbing head impacts and other physical traumas.

Equally important, Roberts et al. (2022) examined how football-related exposures might intersect with developmental risk. Adjusting for concussion symptoms during playing years attenuated some associations between ACEs and outcomes, though several remained significant. This suggests that childhood adversity may affect adult outcomes in two ways, which can be directly through its effects on development and stress, and indirectly through its relationship with head injury exposure during a football career. Moreover, players with ≥ 4 ACEs were more likely to be in the highest quartile of concussion symptoms (Roberts et al., 2022). These findings motivate further research examining how early life adversity and adult sports exposures together shape neuropsychiatric health trajectories.

Roberts et al. (2022) established the prevalence of ACEs in this population but did not test whether benevolent experiences are associated with better adult mental health and cognitive quality of life. This represents a clear gap in the literature: if ACEs are linked to poorer outcomes among former players, BCEs may help explain variability in outcomes and may identify promotive experiences that are associated with better adult functioning.

PHQ-9, GAD-7, and Neuro-QoL

The primary outcomes examined in this study were depressive symptoms, anxiety symptoms, and cognition-related quality of life. These outcomes were assessed using the Patient Health Questionnaire–9 (PHQ-9), Generalized Anxiety Disorder–7 (GAD-7), and Neuro-Quality of Life Applied Cognition instruments (Neuro-QOL), which are established and widely used measures in clinical and research settings.

Depression: PHQ-9

Kroenke et al. (2001) validated the PHQ-9 as a brief self-administered measure of depressive symptom severity that asks about nine symptoms over the past two weeks with each item scored from 0 (“not at all”) to 3 (“nearly every day”). In their validation work, increasing PHQ-9 scores corresponded to meaningful declines in daily functioning and increased symptom-related impairment, supporting strong validity (Kroenke et al., 2001). Importantly for research, the PHQ-9 provides clinically interpretable thresholds and a widely used screening cutoff: a PHQ-9 score of ≥ 10 demonstrated strong sensitivity and specificity for major depression, and standard severity thresholds (5, 10, 15, 20) correspond to mild through severe depression (Kroenke et al., 2001). Prior studies from the FPHS show that depression is meaningfully associated with multiple downstream

factors in this population. For example, FPHS analyses link depressive symptoms with greater concussion-symptom burden (Roberts et al., 2019) and with broader post-career health burden, including pain interference and other potentially modifiable correlates such as sleep apnea (Roberts et al., 2021). This makes the PHQ-9 an appropriate tool to study former professional ASF players, where depression is prevalent.

Anxiety: GAD-7

Spitzer et al. (2006) developed the GAD-7 as a brief self-report tool designed to identify anxiety symptom severity over the prior two weeks. In a multicenter primary care sample, the GAD-7 demonstrated strong reliability (including high internal consistency) and multiple forms of validity, and performed well against clinical diagnoses (Spitzer et al., 2006). A score of ≥ 10 was identified as an efficient cutoff and increasing GAD-7 scores were associated with progressively worse functioning across multiple domains (Spitzer et al., 2006). Importantly, Spitzer et al. also demonstrated that anxiety and depression symptoms are related but distinct dimensions. This supports the value of measuring anxiety directly rather than assuming it is the same thing as depression (Spitzer et al., 2006). This distinction is particularly relevant in populations like former athletes, where symptom profiles may vary across depression, anxiety, and cognitive concerns. For this reason, anxiety symptoms are routinely assessed in former professional ASF research using the GAD-7. For example, FPHS survey analyses have assessed past-2-week anxiety symptoms with the GAD-7 alongside other common post-career health domains and football-related exposures (e.g., pain, sleep apnea, and career characteristics) (Grashow et al., 2024), and independent clinical research in former NFL

retirees has similarly used the GAD-7 to characterize anxiety symptom burden (Kumar et al., 2018).

Cognition-related quality of life: Neuro-QoL

For cognition-related outcomes, the present study used the Neuro-QoL Applied Cognition measure, a self-report measure developed to capture how neurological symptoms affect everyday functioning. Cella et al. (2012) described Neuro-QoL as a standardized, patient-centered health-related quality of life measurement framework supported by the National Institute of Neurological Disorders and Stroke, designed to fill a gap on how health is measured, as many of the tools focus on clinical phenotypes rather than how a person feels and functions on a day to day basis. Neuro-QoL uses carefully developed question sets covering physical, mental, and social domains—including applied cognition, anxiety, and depression—and reports results as standardized T-scores anchored to reference populations, making scores easy to interpret and compare across different groups (Cella et al., 2012). Validation work indicates that Neuro-QoL short forms are brief, reliable, and able to detect meaningful differences in how much symptoms affect daily life, supporting their use for detecting meaningful variation in self-reported cognitive functioning (Cella et al., 2012). In populations of former professional ASF players where subjective cognitive concerns are common, Neuro-QoL provides a way to assess cognitive symptoms experienced in daily life (Alosco et al., 2023; Cella et al., 2012).

Existing research provides strong evidence that ACEs contribute to later-life mental health and cognitive vulnerability, including among former professional football players, but far less is known about whether BCEs are associated with better adult

neuropsychiatric functioning in this population. By using validated symptom measures and a quality-of-life instrument, the present thesis aims to address a clear gap in the athlete neuropsychiatric health literature.

In summary, this study examined BCEs among former professional ASF players and evaluated how BCEs relate to adult mood and cognition, both independently and in combination with ACEs. The research questions were: (1) What is the distribution of BCEs in this cohort? (2) Are BCEs associated with depressive symptoms (PHQ-9), anxiety symptoms (GAD-7), and cognition-related quality of life (Neuro-QoL Applied Cognition) in models adjusted for demographic and football-related characteristics? and (3) In models that include both exposures simultaneously, how do BCEs and ACEs jointly relate to these outcomes? The overall aim was to estimate associations between BCEs and neuropsychological outcomes adjusting for ACEs and other covariates. I hypothesized that higher BCEs operationalized in primary analyses as endorsing the maximum BCE score ($BCE = 10$ vs. $BCE < 10$), would be associated with lower depressive and anxiety symptom severity and better self-reported cognitive functioning. Further, I hypothesized that positive associations of BCEs would remain after inclusion of ACEs.

Chapter II.

Methods

This study utilized data from the Football Players' Health Study at Harvard University, an ongoing longitudinal cohort of former professional ASF players (Zafonte, R. et al., 2019). All individuals who had signed a contract with a professional football league since 1960 were eligible to participate. The FPHS study is IRB approved. All participants provided informed consent before participation. Analyses included in this study utilized a de-identified dataset from the larger epidemiological cohort and is therefore considered not to be Human Subjects Research. This designation of non-human subjects' research was determined and confirmed by an IRB Administrator Team Lead at Harvard University.

Measures

Sociodemographic characteristics included age at assessment, self-identified race (Black/White/Other), player education, and maternal education.

Football-related exposures and health covariates included player position (lineman vs. non-lineman), age started playing football, number of seasons played (0–7, 8–12, 13+), professional playing body mass index (pro-BMI), and total concussion symptom score. These variables were chosen based on prior research and were available in the FPHS cohort to account for football exposure and related health burden that may be associated with both childhood experience measures and adult outcomes.

Positive early-life experiences were assessed using the 10-item Benevolent Childhood Experiences (BCEs) scale (Narayan et al., 2018), which captures protective

and supportive experiences from birth to age 18. Participants retrospectively indicated whether each experience occurred during childhood (“Yes” = 1, “No” = 0). Items include having a caregiver with whom they felt safe, having at least one good friend, holding beliefs that provided comfort, enjoying school, having a teacher/coach/mentor who cared, having good neighbors, having a supportive adult outside the immediate caregiver, engaging in enjoyable activities, feeling comfortable with oneself, and having a predictable home routine. Endorsements were summed to create a total BCE score ranging from 0 to 10, with higher scores indicating greater exposure to BCEs. BCEs were distributed as $BCE = 10$ vs. $BCE < 10$ to help the highly skewed BCE distributions in this population.

ACEs were assessed using a standard ACE inventory reflecting exposure to childhood abuse, neglect, and household dysfunction prior to age 18 (Felitti et al., 1998). Endorsed ACE categories were summed to create a total ACE score, with higher values indicating greater adversity burden. ACE total score was included alongside BCE exposure in models to estimate associations of BCEs and ACEs with adult outcomes.

Depressive symptom severity was assessed using the PHQ-9 total score (range 0–27), with higher scores indicating more severe depressive symptoms. Anxiety symptom severity was measured using the GAD-7 total score (range 0–21), with higher scores indicating greater anxiety symptoms. Cognition-related quality of life was evaluated using the Neuro-QoL Applied Cognition short form (Cella et al., 2012), which yields standardized T-scores (mean = 50, SD = 10), where higher T-scores indicate better perceived cognitive functioning. I analyzed an inverse Neuro-QoL T-score, such that higher inverse scores indicate worse perceived cognitive functioning.

For each outcome, participants were included if they had non-missing outcome data and complete data on all covariates required for the fully adjusted model. As a result, the analytic N was 366 for PHQ-9 models, 368 for GAD-7 models, and 366 for Neuro-QoL cognition models. Differences in N across outcomes reflect outcome-specific missingness and, in some cases, missingness in covariates included in the fully adjusted models (e.g., BMI or concussion symptom burden), reflecting complete-case analysis.

Statistical analysis

Statistical analyses were conducted to summarize participant characteristics and examine associations between childhood experience measures and neuropsychological outcomes.

Descriptive analyses

Participant characteristics were divided by BCE < 10 vs BCE = 10.. Continuous variables were reported using mean (SD) and median (Q1, Q3), and categorical variables were reported using counts and percentages.

Associations between childhood experience measures and neuropsychological outcomes were estimated using multivariable generalized estimating equations (GEE) with a Gaussian distribution and identity link, with robust sandwich standard errors. Separate models were fit for PHQ-9, GAD-7, and Neuro-QoL inverse T-score. Higher Neuro-QoL T-scores indicate better perceived cognition; therefore, we analyzed an inverse Neuro-QoL T-score (multiplied by -1) so that higher values consistently reflected worse symptoms across outcomes and coefficients could be interpreted in the same direction as PHQ-9 and GAD-7. Multivariable GEE models included BCE group (BCE =

10 vs BCE < 10) and ACE total score simultaneously, along with covariates to evaluate how the inclusion of ACEs changed the relationship between BCEs and neuropsychological outcomes.

To evaluate how associations changed with progressive adjustment, models were built sequentially. The Basic model included BCE group only. The Demographic model added age at assessment, race, and player education. The Football Exposure model added lineman status and number of seasons played. The Full model added in pro-BMI and total concussion symptom score. Finally, the ACEs model added ACE total score to the Full model. This approach helps interpret how BCEs associations change as additional covariates are added and help to further understand if BCEs persist after accounting for ACEs.

Model Diagnostics

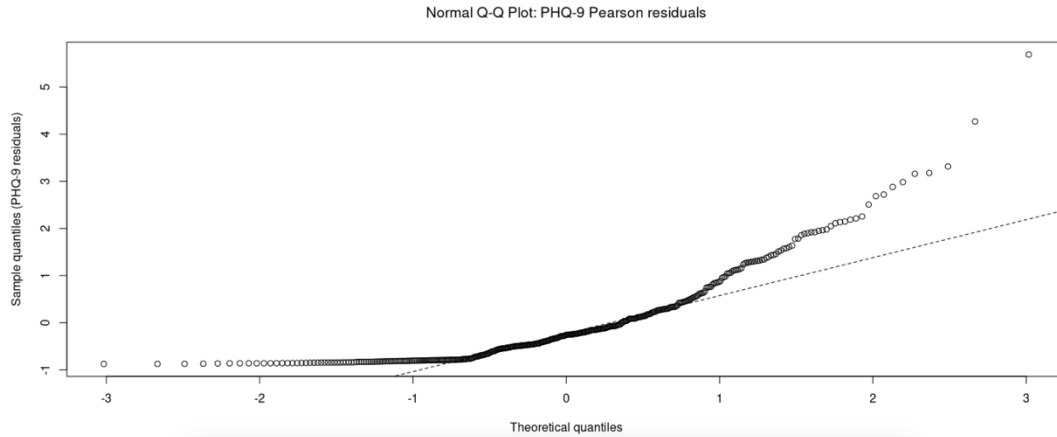


Figure 1. Normal Q–Q Plot of Pearson Residuals for the PHQ-9 Gaussian GEE Model.

This figure presents a normal Q–Q plot of Pearson residuals from the PHQ-9 Gaussian GEE model. The plot is used as a diagnostic to assess how closely the residuals follow a normal distribution.

The normal Q-Q plot showed departures from normality, particularly in the right tail, indicating right-skewness. This pattern is consistent with the expected distribution of PHQ-9 scores, in which many participants reported low symptom levels and relatively few reported high depressive symptom burden. Visual inspection of the outcome distribution also suggested clustering near the lower bound of the scale. Because generalized estimating equations provide population-averaged estimates with robust sandwich standard errors, the Gaussian identity-link GEE model was used.

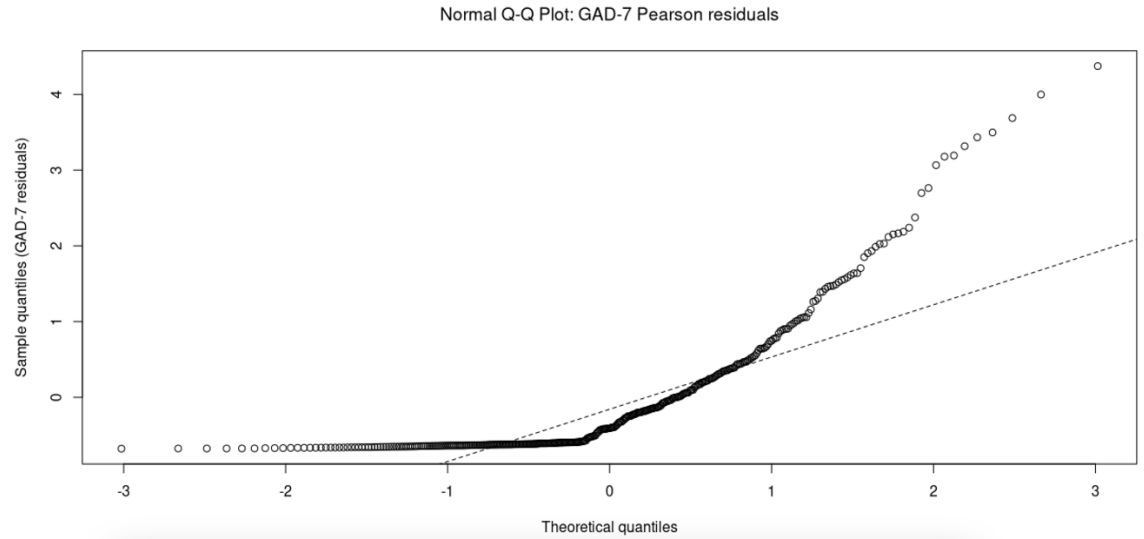


Figure 2. Normal Q–Q Plot of Pearson Residuals for the GAD-7 Gaussian GEE Model.

This figure presents a normal Q – Q plot of Pearson residuals from the GAD-7 Gaussian GEE model. The plot is included as a diagnostic to assess how closely the residuals follow a normal distribution.

The normal Q–Q plot of Pearson residuals from the GAD-7 Gaussian GEE model showed departures from normality in the upper tail, indicating a right-skewed residual distribution with an extended positive tail. This pattern is consistent with the distribution expected for anxiety symptom scores, where many participants report low symptom burden and fewer report high scores. Because generalized estimating equations provided population-averaged estimates with robust sandwich variance estimators, results were interpreted as valid despite non-normal residuals.

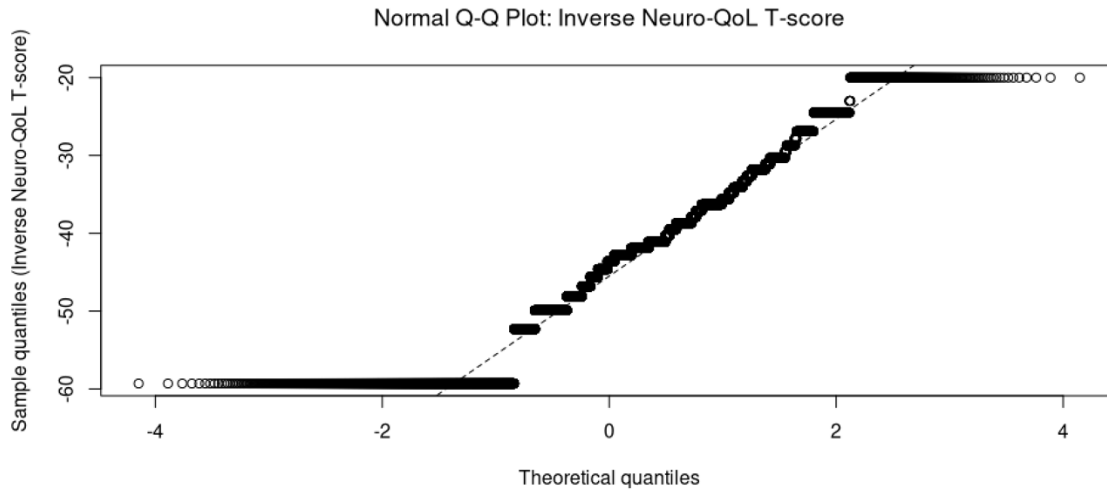


Figure 3. Normal Q–Q Plot of Pearson Residuals for the Neuro-QoL Cognition Inverse T-Score Gaussian GEE Model.

This figure presents a normal Q–Q plot of Pearson residuals from the Gaussian GEE model for Neuro-QoL cognition inverse T-scores.

Diagnostic assessment of the Gaussian GEE model for Neuro-QoL cognition inverse T-scores used a normal Q–Q plot to assess whether residuals followed a normal distribution. The Q–Q plot demonstrated good agreement with the reference line across the central portion of the distribution, supporting the use of a Gaussian model for this outcome. Minor departures from normality were observed in the tails, likely reflecting the nature of the Neuro-QoL scale. Given that generalized estimating equations provide population-averaged estimates with robust (sandwich) standard errors, results were interpreted as valid despite minor tail deviations.

Chapter III.

Results

This chapter presents the study findings and describes the demographic characteristics of the analytic sample.

Cohort Demographics

To be included in this analysis, respondents were required to have completed the BCE items within the Life Course Questionnaire. Based on this inclusion criterion, 415 former professional ASF players were included in the analytic sample. Table 1 summarizes demographic, educational, and football-related characteristics of former professional football players (N=415) stratified by BCE score <10, (n=96) vs BCE = 10, (n=319). Overall, the sample had a mean age of 59 years (SD=13.88) at the time of BCE assessment, with a median age of 61.0 (Q1=46.7, Q3=69.35), with participants' age ranging from 31 to 87 years (Table 1). Players were predominantly White (62.9%), followed by Black (35.1%), with 2.0% identifying as other racial groups.

Across BCE strata, most demographic and background characteristics were similar, and differences were generally not statistically significant. Mean age was slightly lower in the BCE <10 group (57.63, SD=13.93) compared with the BCE =10 group (58.95, SD=13.87), though this difference did not reach statistical significance ($p=0.104$). Race distributions were also comparable ($p=0.492$); Black players comprised 45.3% of the BCE <10 group and 32.1% of the BCE =10 group.

Educational attainment did not differ significantly by BCE category ($p=0.457$). Overall, most players reported graduating college (55.0%), and over one-third reported

education beyond college (35.5%). Maternal education was similarly distributed across groups ($p=0.664$), with the largest proportion reporting mothers who graduated high school (40.9% overall).

Football-related characteristics were also broadly comparable. Player position (lineman vs non-lineman) did not differ across BCE groups ($p=0.922$). Age at starting football was similar between groups (overall mean=11.40 years, $SD=3.07$; $p=0.560$), as was the distribution of professional seasons played ($p=0.561$). Total concussion symptom score did not differ significantly by BCE category (overall mean=29.91, $SD=25.44$; $p=0.435$).

In contrast, childhood adversity and symptom outcomes differed by BCE category in bivariable unadjusted analyses. ACE burden was higher among players with BCE <10 (mean=2.61, $SD=2.65$) compared with those with BCE =10 (mean=1.01, $SD=1.33$; $p<0.001$). Mood symptoms were also higher in the BCE <10 group, with higher PHQ-9 scores (mean=5.35, $SD=5.41$ vs 3.62, $SD=4.94$; $p=0.003$) and higher GAD-7 scores (mean=3.93, $SD=5.11$ vs 2.60, $SD=4.03$; $p<0.001$). Finally, Neuro-QoL cognition T-scores were lower in the BCE <10 group (mean=42.70, $SD=9.50$) compared with the BCE =10 group (mean=45.10, $SD=9.55$; $p=0.004$), indicating worse self-reported cognitive functioning among participants reporting fewer BCEs.

Table 1. Demographic Characteristics in Former Players.

Characteristic	Total (N = 415)	BCE < 10 (n = 96)	BCE = 10 (n = 319)	<i>p</i> -value
Age				0.104
Mean (SD)	58.64 (13.88)	57.63 (13.93)	58.95 (13.87)	
Min–Max	31.40–87.00	31.40–85.10	31.40–87.00	
Median (Q1, Q3)	61.00 (46.70, 69.35)	58.75 (45.72, 68.48)	61.40 (47.10, 69.60)	
Race				0.492
White	256 (62.9%)	50 (52.6%)	206 (66.0%)	
Black	143 (35.1%)	43 (45.3%)	100 (32.1%)	
Other	8 (2.0%)	2 (2.1%)	6 (1.9%)	
Missing, n	8	1	7	
Player education				0.457
Graduated high school	1 (0.2%)	0 (0.0%)	1 (0.3%)	
Some college	37 (9.2%)	15 (16.3%)	22 (7.1%)	
Graduated college	220 (55.0%)	43 (46.7%)	177 (57.5%)	
More than college	142 (35.5%)	34 (37.0%)	108 (35.1%)	

Characteristic	Total (N = 415)	BCE < 10 (n = 96)	BCE = 10 (n = 319)	<i>p</i> -value
Missing, n	15	4	11	
Maternal education				0.664
Some high school or less	39 (9.8%)	11 (12.1%)	28 (9.2%)	
Graduated high school	162 (40.9%)	41 (45.1%)	121 (39.7%)	
Some college	64 (16.2%)	14 (15.4%)	50 (16.4%)	
Graduated college	91 (23.0%)	18 (19.8%)	73 (23.9%)	
More than college	40 (10.1%)	7 (7.7%)	33 (10.8%)	
Missing, n	19	5	14	
Player position				0.922
Non-lineman	210 (50.6%)	49 (51.0%)	161 (50.5%)	
Lineman	205 (49.4%)	47 (49.0%)	158 (49.5%)	
Age started football				0.560
Mean (SD)	11.40 (3.07)	11.40 (2.62)	11.40 (3.20)	
Min–Max	5.00–23.00	5.00–16.00	5.00–23.00	
Median (Q1, Q3)	12.00 (9.00, 14.00)	12.00 (9.75, 14.00)	12.00 (9.00, 14.00)	
Missing, n	3	0	3	

Characteristic	Total (N = 415)	BCE < 10 (n = 96)	BCE = 10 (n = 319)	<i>p</i> -value
NFL career BMI				0.388
Mean (SD)	30.99 (5.02)	31.38 (5.16)	30.87 (4.98)	
Min–Max	17.33–49.99	22.31–49.44	17.33–49.99	
Median (Q1, Q3)	30.00 (27.38, 32.99)	30.91 (27.38, 34.29)	29.99 (27.38, 32.49)	
Missing, n	2	0	2	
Number of seasons				0.561
0–7	290 (70.9%)	65 (68.4%)	225 (71.7%)	
8–12	101 (24.7%)	24 (25.3%)	77 (24.5%)	
13+	18 (4.4%)	6 (6.3%)	12 (3.8%)	
Missing, n	6	1	5	
Total concussion symptom score				0.435
Mean (SD)	29.91 (25.44)	31.68 (25.37)	29.36 (25.48)	
Min–Max	0.00–130.00	0.00–117.00	0.00–130.00	
Median (Q1, Q3)	24.00 (10.50, 41.00)	26.25 (14.88, 42.38)	23.00 (10.50, 39.00)	
Missing, n	6	0	6	
ACE score				< .001

Characteristic	Total (N = 415)	BCE < 10 (n = 96)	BCE = 10 (n = 319)	<i>p</i> -value
Mean (SD)	1.38 (1.85)	2.61 (2.65)	1.01 (1.33)	
Min–Max	0.00–9.00	0.00–9.00	0.00–7.00	
Median (Q1, Q3)	1.00 (0.00, 2.00)	1.00 (0.00, 4.00)	1.00 (0.00, 2.00)	
Total PHQ-9 score				0.003
Mean (SD)	4.02 (5.10)	5.35 (5.41)	3.62 (4.94)	
Min–Max	0.00–27.00	0.00–25.00	0.00–27.00	
Median (Q1, Q3)	2.00 (0.00, 6.00)	4.00 (1.00, 8.00)	2.00 (0.00, 5.00)	
Missing, n	6	2	4	
Total GAD-7 score				< .001
Mean (SD)	2.91 (4.33)	3.93 (5.11)	2.60 (4.03)	
Min–Max	0.00–21.00	0.00–19.00	0.00–21.00	
Median (Q1, Q3)	1.00 (0.00, 5.00)	1.00 (0.00, 7.00)	1.00 (0.00, 4.00)	
Missing, n	6	2	4	
Neuro-QoL T score				0.004
Mean (SD)	44.55 (9.58)	42.70 (9.50)	45.10 (9.55)	

Characteristic	Total (N = 415)	BCE < 10 (n = 96)	BCE = 10 (n = 319)	<i>p</i> -value
Min–Max	20.00–59.30	24.50–59.30	20.00–59.30	
Median (Q1, Q3)	43.60 (37.90, 49.90)	41.90 (34.80, 49.00)	44.60 (38.70, 52.30)	
Missing, n	16	5	11	

Note. Values are presented as n (%) for categorical variables and as mean (SD), min–max, or median (Q1, Q3) for continuous variables. p values compare participants with BCE < 10 versus BCE = 10. ACE = adverse childhood experiences; BCE = benevolent childhood experiences; BMI = body mass index; GAD-7 = Generalized Anxiety Disorder 7-item scale; Neuro-QoL = Quality of Life in Neurological Disorders; PHQ-9 = Patient Health Questionnaire-9.

Table 2: Distribution of Individual BCE Items in the FPHS cohort

Benevolent childhood experience	n / N (%)
Teacher/coach who cared	412/414 (99.5%)
Had at least one good friend	410/413 (99.3%)
Opportunities to have a good time	406/412 (98.5%)
Beliefs that gave comfort	400/413 (96.9%)
Had good neighbors	397/412 (96.4%)
Felt safe with a caregiver	393/413 (95.2%)
An adult for support/advice	388/412 (94.2%)
Enjoyed school	388/414 (93.7%)
Liked myself / felt comfortable with myself	385/412 (93.4%)
Predictable home routine (meals/bedtime)	380/410 (92.7%)

This table summarizes endorsement of each BCE item, presented as n/N (%) among participants with non-missing responses for that item. Denominators vary slightly across items due to item-level missing data.

Overall, endorsement of individual BCE items was very high in this cohort. Across the 10 BCE items shown, endorsement ranged from 92.7% to 99.5%, indicating that most participants reported multiple benevolent experiences in childhood. The most frequently endorsed items were having a teacher/coach who cared (412/414, 99.5%), having at least one good friend (410/413, 99.3%), and having opportunities to have a good time (406/412, 98.5%). The least frequently endorsed item was a predictable home routine (meals/bedtime) (380/410, 92.7%), though it remained highly prevalent. Taken together, these item-level distributions suggest a strong overall concentration of BCE endorsement and limited variability across items.

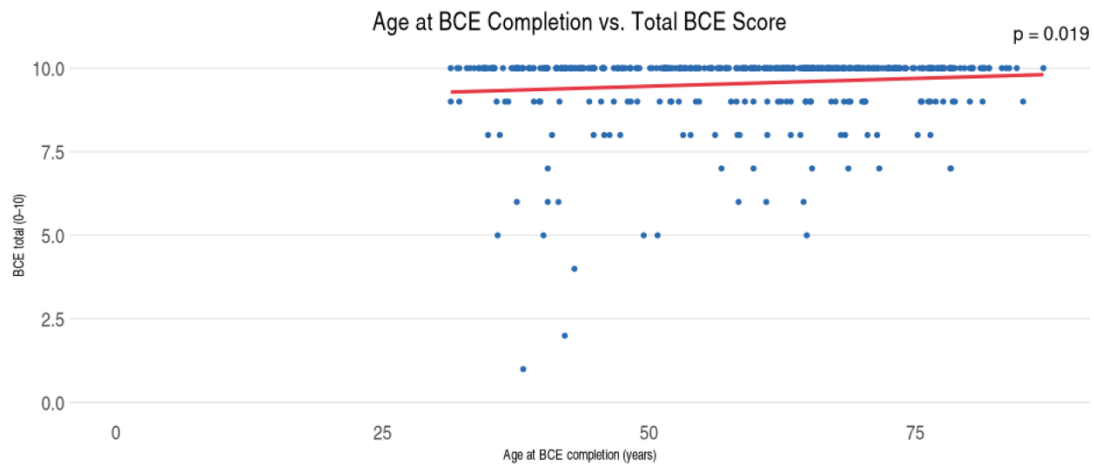


Figure 4. Age at BCE Completion and Total BCE Score.

This scatterplot shows the association between age at BCE questionnaire completion and total BCE score (0–10) among former professional football players in the FPHS (n = 415). Each point represents an individual participant, and the line represents the fitted linear regression.

In bivariate analyses, age at survey completion showed a small positive association with total BCE score (Pearson $r = 0.116$, 95% CI 0.019–0.209; $p = 0.019$; $n = 415$). Specifically, older age at survey completion was associated with modestly higher BCE totals across the observed range of ages, as reflected by the upward linear trend. However, BCE scores were strongly concentrated at the highest end of the distribution, with a large proportion of participants reporting the maximum BCE score of 10, suggesting a pronounced ceiling effect.

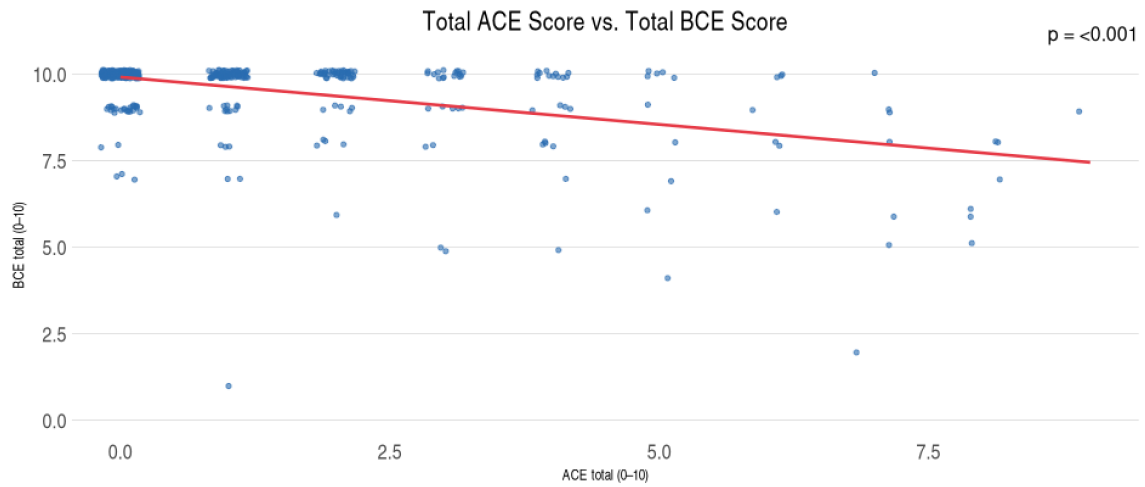


Figure 5: Total ACE Score and Total BCE Score.

This scatterplot shows the relationship between total ACE score and total BCE score (0–10) among former professional ASF players ($n = 415$). Each point represents an individual participant, and the line represents the fitted linear regression.

Total ACE score was moderately and significantly inversely correlated with total BCE score (Pearson $r = -0.451$, 95% CI -0.525 to -0.371 ; $p < 0.001$; $n = 415$), indicating that participants reporting higher childhood adversity tended to report fewer BCEs. The scatterplot showed a negative trend across the ACE range, with BCE totals decreasing as ACE burden increased. BCE scores were also strongly clustered at the maximum value (10/10).

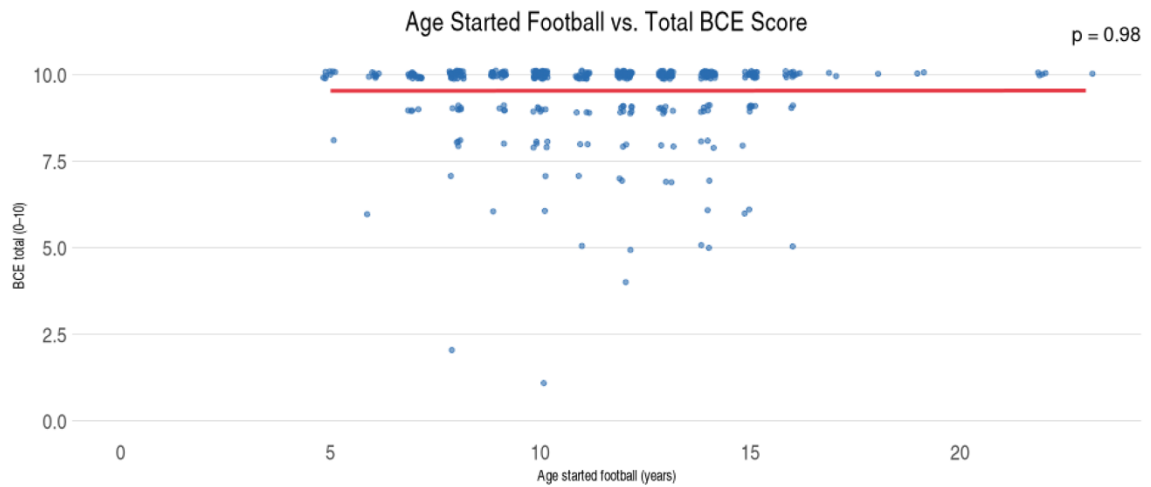


Figure 6: Age Started Football and Total BCE score.

This scatterplot shows the association between age at starting football (years) and total BCE score (0–10) among former professional ASF players ($n = 412$). Each point represents an individual participant, and the line represents the fitted linear regression.

Age started playing football was not associated with total BCE score in bivariate analyses. There was no evidence of a linear relationship between age started football and BCE total (Pearson $r = 0.001$, 95% CI -0.096 to 0.098 ; $p = 0.98$; $n = 412$). Visual inspection of the scatterplot showed substantial clustering of BCE totals at the maximum score (10/10), consistent with a ceiling effect, with BCE values broadly overlapping across the range of reported ages started football. Overall, these findings suggest that age at football initiation was not related to endorsement of BCEs in this cohort.

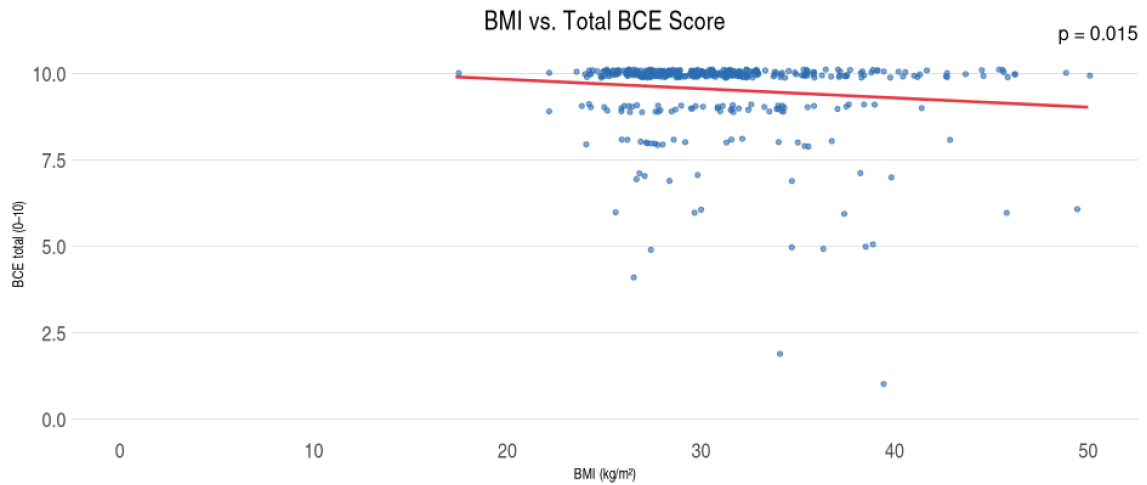


Figure 7: Body Mass Index and Total BCE Score

This scatterplot shows the association between body mass index (BMI; kg/m²) and total BCE score (0–10) among former professional ASF players (n = 413). Each point represents an individual participant, and the line represents the fitted linear regression.

Total BCE score was examined in relation to professional playing body mass index (BMI) using Pearson correlation. Pro-BMI was weakly and inversely correlated with total BCE score (Pearson $r = -0.120$, 95% CI -0.214 to -0.023 ; $p < 0.05$; $n = 413$), indicating that higher pro-BMI was associated with slightly lower BCE totals. The scatterplot showed substantial clustering of BCE totals at the upper limit of the scale (10/10), consistent with a ceiling effect, with BCE scores largely overlapping across the BMI range.

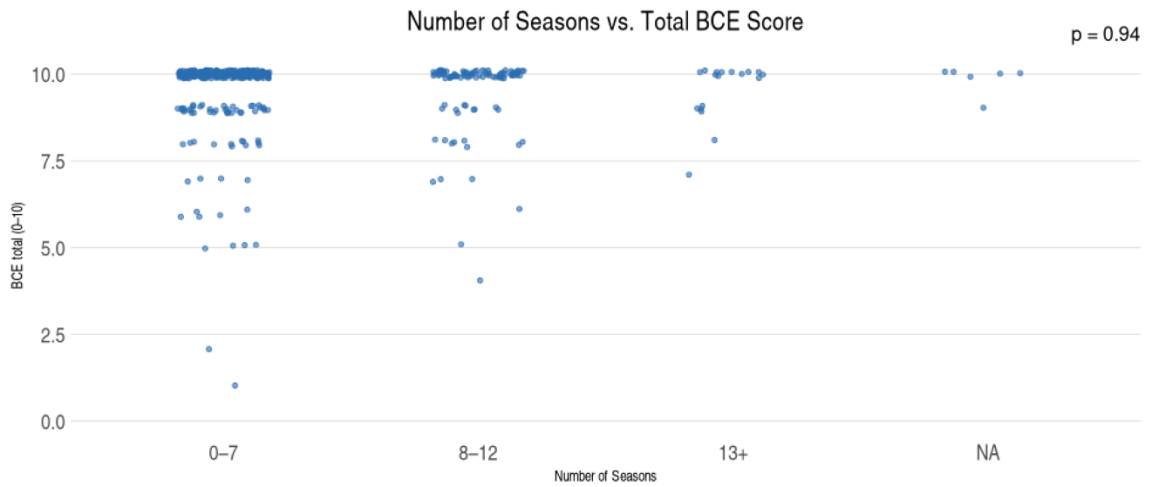


Figure 8. Number of Professional Seasons and Total BCE Score.

This scatterplot shows total BCE score (0–10) across categories of professional seasons played (0–7, 8–12, 13+, and missing) among former professional ASF players ($n = 415$).

Total BCE scores were consistently high across all categories of seasons played, with minimal variation between groups. Participants with 0–7 seasons ($n = 290$) had a mean BCE total of 9.55 ($SD = 1.16$), those with 8–12 seasons ($n = 101$) had a mean of 9.50 ($SD = 1.10$), and those with 13+ seasons ($n = 18$) had a mean of 9.50 ($SD = 0.86$). Median BCE totals were 10 across all season categories, with interquartile ranges concentrated at the upper end of the scale (0–7: $Q1$ – $Q3 = 10$ – 10 ; 8–12: 10 – 10 ; 13+: 9 – 10), consistent with a pronounced ceiling effect. BCE totals did not significantly differ by season category (one-way ANOVA $p = 0.94$), suggesting that football career duration was not associated with BCEs in this cohort.

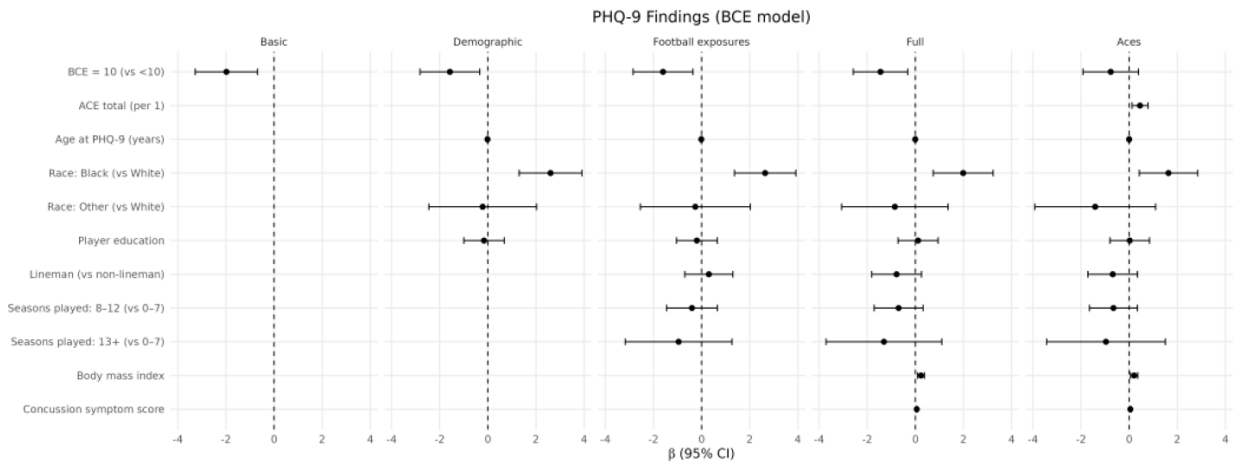


Figure 9. Forest plot of the association between BCEs and PHQ-9 scores (Gaussian GEE).

Points represent β coefficients and horizontal lines represent 95% confidence intervals from Gaussian generalized estimating equation (GEE) models. Models are sequentially adjusted: Basic (BCE only); Demographic (BCE + age at PHQ-9, race, player education); Football exposures (Demographic + lineman status and seasons played); Full (Football exposures + body mass index and concussion symptom score); and ACEs (Full + ACE total score). The vertical dashed line indicates $\beta = 0$. Reference groups: BCE < 10, White race, non-lineman, 0–7 seasons played. $N = 366$.

Table 3. Association between BCEs and PHQ-9 scores (Gaussian GEE)

Predictor	Basic	Demographic	Football exposures	Full	ACEs
BCE = 10 (vs <10)	-1.98 (-3.28, -0.69); p = .003	-1.58 (-2.82, -0.34); p = .013	-1.60 (-2.85, -0.36); p = .011	-1.45 (-2.58, -0.32); p = .012	-0.76 (-1.92, 0.39); p = .194
Age at PHQ-9 (years)	—	-0.02 (-0.05, 0.02); p = .399	-0.01 (-0.05, 0.03); p = .540	-0.00 (-0.04, 0.03); p = .854	0.00 (-0.03, 0.04); p = .935
Race: Black (vs White)	—	2.60 (1.30, 3.91); p < .001	2.64 (1.37, 3.92); p < .001	1.99 (0.75, 3.23); p = .002	1.64 (0.42, 2.85); p = .008
Race: Other (vs White)	—	-0.21 (-2.45, 2.03); p = .851	-0.26 (-2.54, 2.02); p = .823	-0.85 (-3.07, 1.36); p = .450	-1.41 (-3.92, 1.10); p = .271
Player education	—	-0.16 (-1.00, 0.68); p = .711	-0.20 (-1.04, 0.65); p = .647	0.11 (-0.72, 0.94); p = .790	0.03 (-0.80, 0.85); p = .951
Lineman (vs non-lineman)	—	—	0.30 (-0.70, 1.30); p = .553	-0.78 (-1.81, 0.25); p = .139	-0.68 (-1.71, 0.35); p = .196
Seasons played: 8–12 (vs 0–7)	—	—	-0.40 (-1.45, 0.65); p = .456	-0.70 (-1.71, 0.32); p = .179	-0.65 (-1.65, 0.34); p = .200
Seasons played: 13+ (vs 0–7)	—	—	-0.96 (-3.17, 1.26); p = .399	-1.31 (-3.71, 1.10); p = .287	-0.96 (-3.43, 1.50); p = .445

Predictor	Basic	Demographic	Football exposures	Full	ACEs
Body mass index	—	—	—	0.24 (0.09, 0.38); p = .002	0.21 (0.06, 0.36); p = .006
Concussion symptom score	—	—	—	0.06 (0.04, 0.08); p < .001	0.05 (0.03, 0.08); p < .001
ACE total (per 1)	—	—	—	—	0.45 (0.12, 0.78); p = .008

Note. Models are sequentially adjusted: Basic (BCE only); Demographic (adds age at assessment, race, and player education); Football exposures (adds lineman status and seasons played); Full (adds BMI and concussion symptom score); ACEs (adds ACE total score). Reference groups: BCE < 10, White race, non-lineman, and 0–7 seasons played.

In Gaussian GEE models evaluating depressive symptoms (PHQ-9; N = 366), endorsing all 10 BCEs (BCE = 10 vs <10) was associated with lower PHQ-9 scores in the Basic model, and this inverse association persisted after adjustment for demographics in the Demographic model. The BCE association was modestly attenuated with the addition of football-related exposures and health-related factors but remained statistically significant through the Full model. After inclusion of ACE total score in the final ACEs model, the BCE association was further attenuated and was no longer statistically significant. In the final model, ACE total score showed a positive association with PHQ-9, indicating that higher ACE burden corresponded to higher depressive symptom scores. Across adjusted models, Black race (vs White) was associated with higher PHQ-9 scores, while football exposure variables were not consistently associated with PHQ-9. In the Full and ACEs models, higher body mass index and higher concussion symptom score were associated with higher PHQ-9 scores, with concussion symptom severity showing the strongest and most consistent positive association.

In Gaussian GEE models assessing depressive symptoms (PHQ-9; N = 366), endorsing all 10 BCEs (BCE = 10 vs <10) was associated with lower PHQ-9 scores in the Basic model ($\beta = -1.98$, 95% CI: -3.28 to -0.69 ; $p = 0.003$) and remained significant after adjustment for demographics (Demographic: $\beta = -1.58$, 95% CI: -2.82 to -0.34 ; $p = 0.013$) and football exposure variables (Football exposures: $\beta = -1.60$, 95% CI: -2.85 to -0.36 ; $p = 0.011$). The BCE association remained significant in the Full model that additionally included health-related factors ($\beta = -1.45$, 95% CI: -2.58 to -0.32 ; $p = 0.012$) but was attenuated and no longer statistically significant after inclusion of ACE

total score in the ACEs model ($\beta = -0.76$, 95% CI: -1.92 to 0.39 ; $p = 0.194$). In the ACEs model, ACE total score was positively associated with PHQ-9 ($\beta = 0.45$, 95% CI: 0.12 to 0.78 ; $p = 0.008$). Black race (vs White) was associated with higher PHQ-9 across models (ACEs: $\beta = 1.64$, 95% CI: 0.42 to 2.85 ; $p = 0.008$), while age at assessment, race categorized as “Other,” player education, and football exposure variables were not significantly associated with PHQ-9 in the fully adjusted models. Higher body mass index (Full: $\beta = 0.24$, 95% CI: 0.09 to 0.38 ; $p = 0.002$; ACEs: $\beta = 0.21$, 95% CI: 0.06 to 0.36 ; $p = 0.006$) and higher concussion symptom score (Full: $\beta = 0.06$, 95% CI: 0.04 to 0.08 ; $p < 0.001$; ACEs: $\beta = 0.05$, 95% CI: 0.03 to 0.08 ; $p < 0.001$) were associated with higher PHQ-9 scores.

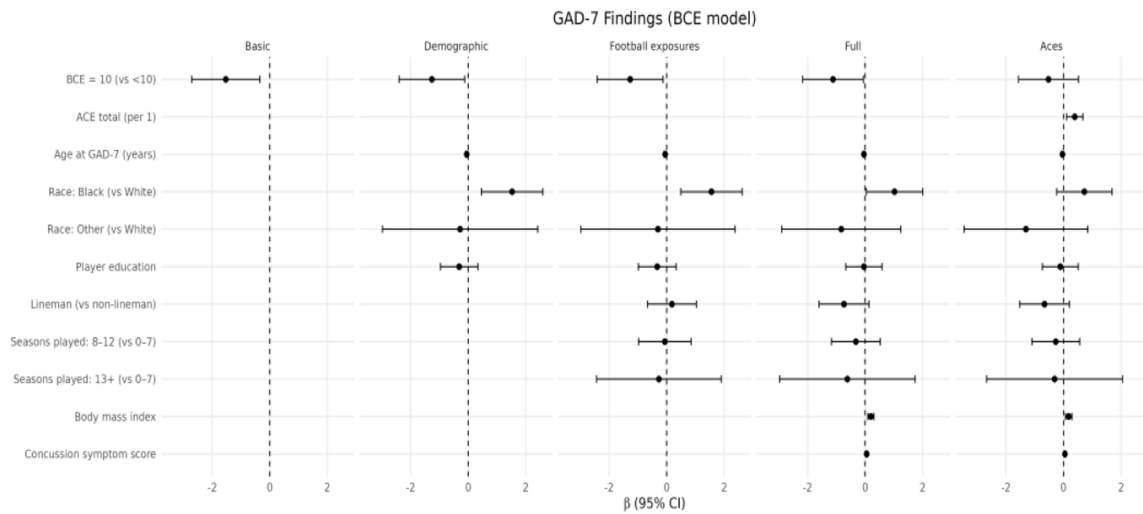


Figure 10. Forest plot of the association BCEs and GAD-7 scores (Gaussian GEE).

Points represent β coefficients and horizontal lines represent 95% confidence intervals from Gaussian generalized estimating equation (GEE) models. Models are sequentially adjusted: Basic (BCE only); Demographic (BCE + age at GAD-7, race, player education); Football exposures (Demographic + lineman status and seasons played); Full (Football exposures + body mass index and concussion symptom score); and ACEs (Full + ACE total score). The vertical dashed line indicates $\beta = 0$. Reference groups: BCE < 10, White race, non-lineman, 0–7 seasons played. $N = 368$

Table 4. Association between BCEs and GAD-7 scores (Gaussian GEE)

Predictor	Basic	Demographic	Football exposures	Full	ACEs
BCE = 10 (vs <10)	-1.53 (-2.71, -0.34); p=0.011	-1.26 (-2.40, -0.12); p=0.030	-1.27 (-2.41, -0.12); p=0.030	-1.11 (-2.17, -0.06); p=0.038	-0.52 (-1.57, 0.52); p=0.325
Age at GAD-7 (years)		-0.05 (-0.09, -0.02); p=0.004	-0.05 (-0.08, -0.02); p=0.004	-0.04 (-0.07, -0.01); p=0.012	-0.04 (-0.07, -0.01); p=0.021
Race: Black (vs White)		1.53 (0.46, 2.59); p=0.005	1.56 (0.49, 2.62); p=0.004	1.02 (0.04, 2.00); p=0.041	0.72 (-0.24, 1.69); p=0.141
Race: Other (vs White)		-0.28 (-2.98, 2.41); p=0.838	-0.30 (-2.98, 2.37); p=0.825	-0.83 (-2.89, 1.24); p=0.433	-1.31 (-3.46, 0.84); p=0.233
Player education		-0.31 (-0.96, 0.34); p=0.348	-0.33 (-0.98, 0.33); p=0.330	-0.04 (-0.67, 0.59); p=0.895	-0.11 (-0.74, 0.51); p=0.727

Predictor	Basic	Demographic	Football exposures	Full	ACEs
Lineman (vs non-lineman)			0.18 (-0.67, 1.03); p=0.673	-0.73 (-1.60, 0.13); p=0.097	-0.66 (-1.53, 0.20); p=0.133
Seasons played: 8–12 (vs 0–7)			-0.06 (-0.97, 0.85); p=0.895	-0.32 (-1.17, 0.53); p=0.458	-0.27 (-1.10, 0.56); p=0.523
Seasons played: 13+ (vs 0–7)			-0.27 (-2.43, 1.90); p=0.808	-0.62 (-2.97, 1.74); p=0.608	-0.31 (-2.67, 2.05); p=0.796
Body mass index				0.20 (0.09, 0.31); p=<0.001	0.18 (0.07, 0.29); p=0.002
Concussion symptom score				0.05 (0.03, 0.08); p=<0.001	0.05 (0.03, 0.07); p=<0.001
ACE total (per 1)					0.39 (0.10, 0.67); p=0.007

Note. Values are reported as β (95% CI); p-value. BCE = benevolent childhood experiences; GEE = generalized estimating equations; ACE = adverse childhood experiences. Values are β coefficients with 95% confidence intervals; p-values shown after the semicolon. Models are sequentially adjusted: Basic (BCE only); Demographic (adds age at assessment, race, player education);

Football exposures (adds lineman status and seasons played); Full (adds BMI and concussion symptom score); ACEs (adds ACE total score). Reference groups: BCE < 10, White race, non-lineman, 0–7 seasons played

In Gaussian GEE models evaluating anxiety symptoms (GAD-7; N = 368), endorsing all 10 BCEs (BCE = 10 vs <10) was associated with lower GAD-7 scores in the bivariable Basic model, and this inverse association persisted after adjustment for demographics, football-related exposures, and health-related factors through the Full model. However, after inclusion of ACE total score in the final ACEs model, the BCE association was attenuated and no longer statistically significant. In the final ACEs model, ACE total score was positively associated with GAD-7, indicating that greater ACE burden corresponded to higher anxiety symptom scores. Older age at assessment was consistently associated with lower GAD-7 scores across adjusted models. Black race (vs White) was associated with higher GAD-7 scores in less adjusted models, but this association diminished with additional covariate adjustment. Football exposure variables were not significantly associated with GAD-7, whereas higher body mass index and higher concussion symptom score were associated with higher GAD-7 scores in the Full and ACEs models.

In Gaussian GEE models evaluating anxiety symptoms (GAD-7; N = 368), endorsing all 10 BCEs (BCE = 10 vs <10) was associated with lower GAD-7 scores in the Basic model ($\beta = -1.53$, 95% CI: -2.71 to -0.34 ; $p = 0.011$) and remained significant after adjustment for demographics (Demographic: $\beta = -1.26$, 95% CI: -2.40 to -0.12 ; $p = 0.030$), football exposures (Football exposures: $\beta = -1.27$, 95% CI: -2.41 to -0.12 ; $p = 0.030$), and health-related factors (Full: $\beta = -1.11$, 95% CI: -2.17 to -0.06 ; $p = 0.038$). However, after inclusion of ACE total score in the final ACEs model, the BCE association was attenuated and no longer statistically significant ($\beta = -0.52$, 95% CI:

−1.57 to 0.52; $p = 0.325$), while ACE total score was positively associated with higher GAD-7 ($\beta = 0.39$, 95% CI: 0.10 to 0.67; $p = 0.007$). Older age at assessment was consistently associated with lower GAD-7 across adjusted models (ACEs: $\beta = -0.04$, 95% CI: −0.07 to −0.01; $p = 0.021$). Black race (vs White) was associated with higher GAD-7 in models without health/ACE adjustment (Demographic: $\beta = 1.53$, 95% CI: 0.46 to 2.59; $p = 0.005$; Football exposures: $\beta = 1.56$, 95% CI: 0.49 to 2.62; $p = 0.004$), but this association was attenuated and not significant in the ACEs model ($\beta = 0.72$, 95% CI: −0.24 to 1.69; $p = 0.141$). Football exposure variables were not significantly associated with GAD-7 in the fully adjusted models. Higher body mass index (Full: $\beta = 0.20$, 95% CI: 0.09 to 0.31; $p < 0.001$; ACEs: $\beta = 0.18$, 95% CI: 0.07 to 0.29; $p = 0.002$) and higher concussion symptom score (Full: $\beta = 0.05$, 95% CI: 0.03 to 0.08; $p < 0.001$; ACEs: $\beta = 0.05$, 95% CI: 0.03 to 0.07; $p < 0.001$) were associated with higher GAD-7 scores.

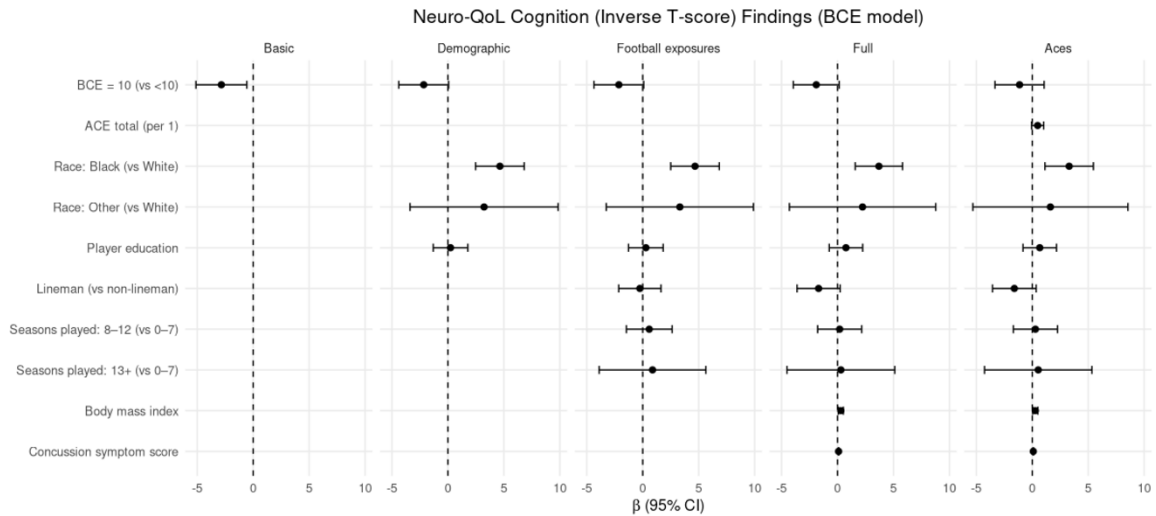


Figure 11. Forest plot of the association between BCEs and Neuro-QoL Cognition inverse T-scores (Gaussian GEE).

Points represent β coefficients and horizontal lines represent 95% confidence intervals from Gaussian generalized estimating equation (GEE) models. Higher inverse T-scores indicate worse cognitive function. Models are sequentially adjusted: Basic (BCE only); Demographic (BCE + race, player education); Football exposures (Demographic + lineman status and seasons played); Full (Football exposures + body mass index and concussion symptom score); and ACEs (Full + ACE total score). The vertical dashed line indicates $\beta = 0$. Reference groups: BCE < 10, White race, non-lineman, 0–7 seasons played. $N = 366$.

Table 5: Association between BCEs and Neuro-QOL Cognition inverse T-scores (Gaussian GEE)

Predictor	Basic	Demographic	Football exposures	Full	ACEs
BCE = 10 (vs <10)	-2.84 (-5.12, -0.57); p=0.014	-2.17 (-4.39, 0.06); p=0.056	-2.13 (-4.36, 0.09); p=0.060	-1.90 (-3.95, 0.15); p=0.070	-1.14 (-3.33, 1.06); p=0.311
Race: Black (vs White)		4.64 (2.47, 6.82); p=<0.001	4.67 (2.50, 6.84); p=<0.001	3.70 (1.59, 5.81); p=<0.001	3.30 (1.13, 5.46); p=0.003
Race: Other (vs White)		3.23 (-3.39, 9.85); p=0.339	3.32 (-3.26, 9.89); p=0.323	2.24 (-4.30, 8.78); p=0.502	1.62 (-5.31, 8.55); p=0.647
Player education		0.23 (-1.31, 1.77); p=0.772	0.28 (-1.27, 1.83); p=0.723	0.76 (-0.74, 2.25); p=0.321	0.67 (-0.83, 2.16); p=0.381
Lineman (vs non-lineman)			-0.26 (-2.14, 1.63); p=0.790	-1.69 (-3.61, 0.24); p=0.086	-1.60 (-3.55, 0.35); p=0.107
Seasons played: 8–12 (vs 0–7)			0.58 (-1.46, 2.63); p=0.576	0.19 (-1.77, 2.16); p=0.847	0.28 (-1.69, 2.25); p=0.779
Seasons played: 13+ (vs 0–7)			0.88 (-3.89, 5.64); p=0.719	0.30 (-4.51, 5.12); p=0.901	0.53 (-4.27, 5.33); p=0.827

Predictor	Basic	Demographic	Football exposures	Full	ACEs
Body mass index				0.30 (0.08, 0.52); p=0.009	0.27 (0.04, 0.50); p=0.022
Concussion symptom score				0.10 (0.06, 0.14); p=<0.001	0.09 (0.05, 0.13); p=<0.001
ACE total (per 1)					0.48 (-0.06, 1.02); p=0.081

Values are β coefficients with 95% confidence intervals; p-values shown after the semicolon. Higher inverse T-scores indicate worse cognitive function. Models are sequentially adjusted: Basic (BCE only); Demographic (adds race and player education); Football exposures (adds lineman status and seasons played); Full (adds BMI and concussion symptom score); ACEs (adds ACE total score). Reference groups: BCE < 10, White race, non-lineman, 0–7 seasons played.

In Gaussian GEE models examining Neuro-QoL Cognition inverse T-scores (higher scores indicating worse cognitive function; N = 366), endorsing the maximum number of BCEs (BCE = 10 vs <10) was associated with lower inverse cognition scores (better cognitive outcomes) in the bivariable Basic model, with a similar direction of association after adjustment for demographics in the Demographic model. The BCE association was attenuated in models that additionally included football-related exposures (Football exposures) and health-related factors (Full) and remained non-statistically significant in the most adjusted models. In the final ACEs model, ACE total score was positively associated with worse cognitive outcomes in the expected direction, while the BCE association remained attenuated. Football exposure variables were not consistently associated with cognition. In contrast, higher concussion symptom scores and to a lesser extent higher body mass index, were associated with worse cognitive function in the Full and ACEs models, with concussion symptom severity showing the strongest association across adjusted models.

In Gaussian GEE models examining Neuro-QoL cognition inverse T-scores (higher scores indicating worse cognitive function; N = 366), endorsing all 10 BCEs (BCE = 10 vs <10) was associated with lower inverse cognition scores (better cognitive outcomes) in the Basic model ($\beta = -2.84$, 95% CI: -5.12 to -0.57 ; $p = 0.014$). This association was attenuated and borderline in the Demographic model ($\beta = -2.17$, 95% CI: -4.39 to 0.06 ; $p = 0.056$), remained similar after inclusion of football exposures (Football exposures: $\beta = -2.13$, 95% CI: -4.36 to 0.09 ; $p = 0.060$), and was further attenuated in the Full model ($\beta = -1.90$, 95% CI: -3.95 to 0.15 ; $p = 0.070$). After inclusion of ACE

total score in the ACEs model, the BCE association was no longer statistically significant ($\beta = -1.14$, 95% CI: -3.33 to 1.06 ; $p = 0.311$), and ACE total score showed a positive but non-statistically significant association with worse cognitive outcomes ($\beta = 0.48$, 95% CI: -0.06 to 1.02 ; $p = 0.081$). Black race (vs White) was consistently associated with higher inverse cognition scores across models (ACEs: $\beta = 3.30$, 95% CI: 1.13 to 5.46 ; $p = 0.003$), while football exposure variables were not significantly associated with cognition in the fully adjusted models. In the Full and ACEs models, higher body mass index (Full: $\beta = 0.30$, 95% CI: 0.08 to 0.52 ; $p = 0.009$; ACEs: $\beta = 0.27$, 95% CI: 0.04 to 0.50 ; $p = 0.022$) and higher concussion symptom score (Full: $\beta = 0.10$, 95% CI: 0.06 to 0.14 ; $p < 0.001$; ACEs: $\beta = 0.09$, 95% CI: 0.05 to 0.13 ; $p < 0.001$) were associated with worse cognitive outcomes.

Chapter IV.

Discussion

This chapter interprets the study findings in relation to the existing literature and considers their methodological and substantive implications.

Overview and principal findings

This thesis examined BCEs conceptualized as cumulative, protective, and supportive resources among former professional ASF players. Further, I investigated whether BCEs were associated with adult neuropsychological outcomes in models adjusted for demographic, football exposures, and health-related factors. Finally, I evaluated whether these associations persisted after accounting for ACEs in adjusted models.

One of the first things that stood out to me was the BCE distribution in this population. As shown in Table 2, the BCE endorsement in this cohort of ASF players is extremely high. Every BCE item was endorsed by about 92% of the population. Some items reported highly including having a teacher or coach who cared (99.5%) and having at least one good friend (99.3%). Statistically, these high endorsements created a ceiling effect. When looking at this from a non-statistical lens, I could infer that these players had some sort of strong baseline to reach the success of being a former professional football player. Being in a team sport may offer substantial support. This is consistent with Lee et al. (2022), who found that individuals with high ACEs and low social support had significantly worse psychological and physical health compared to those with high ACEs and high social support. Notably, support from friends, family, and community

was associated with lower odds of depression and health risk behaviors. If football provided that kind of sustained support for these players, it may help explain why BCE endorsement was so uniformly high in this cohort.

Due to BCE endorsement clustered at the maximum, operationalizing BCE as 10 vs <10 likely reduced power to detect independent effects in this sample. This is consistent with population-based work showing that BCE scores can be skewed upward and subject to ceiling effects (Redican et al., 2022; Narayan et al., 2023). Part of the reason why the BCE data may be skewed was the use of the original BCE measure (Narayan et al., 2018) rather than the revised version developed by Narayan et al. (2023), which took out more universally endorsed items such as having at least one safe caregiver, at least one good friend, at least one teacher who cared, and opportunities to have a good time and replaced them with more discriminative items such as feeling accepted for who you were, feeling treated fairly, and having regular time in nature. Using the revised scale in future work may yield more variability and stronger power to detect BCE effects in future research.

Depression, Anxiety, Cognition-Related Quality of Life

Across the three outcomes that I studied (PHQ-9, GAD-7, and Neuro-QoL applied cognition), BCEs were shown to be protective in the minimally adjusted models, but after including ACEs and other covariates, I saw these associations attenuate.

As seen in Roberts et al. (2022), they found that ACE total score was significantly associated with depression. My findings align with this, where each one-point increase in ACE total score corresponded with a 0.45-point higher PHQ-9 score, even after adjusting for BCEs and the other covariates. When adding BCEs to the model

of depression, I saw that it was significant in the basic model ($\beta = -1.98$, $p = .003$), in the demographic model ($\beta = -1.58$, $p = .013$), football exposures model ($\beta = -1.60$, $p = .011$), full model ($\beta = -1.45$, $p = .012$), but the association disappeared once ACEs were added in ($\beta = -0.76$, $p = .194$). What I find meaningful here is that once ACEs were added to the BCEs model, BCEs were no longer protective over depressive symptoms. This suggests that ACEs may carry the stronger independent association with depression when both adverse and benevolent experiences are considered together.

One difference I found between my research and Roberts et al., (2022) came when studying anxiety. In my analysis, I found that anxiety symptoms were significant with ACEs ($\beta = 0.39$, $p = .007$). However, Roberts et al. (2022) did not find ACEs to be associated with anxiety in their main models. Their sample was considerably larger ($n = 1,755$ vs. my $n = 368$), which could have influenced their results in either direction.

In my research, anxiety was significant in the basic model ($\beta = -1.53$, $p = .011$), the demographic model ($\beta = -1.26$, $p = .030$), the football exposure model ($\beta = -1.27$, $p = .030$), but not quite significant in the full model ($\beta = -1.11$, $p = .060$), and not significant in the ACEs model ($\beta = -0.52$, $p = .325$). This further shows the pattern that BCEs are associated with lower anxiety symptoms in the simpler models, but this seems to disappear in the fully adjusted and after ACEs are included.

For cognition-related quality of life, I found that BCEs were only significant in the basic model ($\beta = -2.84$, $p = .014$) and not significant in the rest of the models (demographic, football exposures, full, and ACEs model). Roberts et al. (2022) observed a similar pattern, though the factors responsible differed. In my analyses, the association weakened after adjustment for broader adult health-related factors including BMI and

concussion symptoms. As shown in Table 5, concussion symptom score was significantly associated with worse cognition-related quality of life in the full model ($\beta = 0.10$, $p < .001$), as was BMI ($\beta = 0.30$, $p = .009$), whereas in Roberts et al. (2022) attenuation was observed after adjustment for concussion symptoms alone. This is not entirely surprising, as these players spent most of their careers suffering from repeated head impacts, and that accumulated burden leaves less power for childhood experiences to independently explain variation in cognition.

Integration with Prior Literature

Taken together, these findings suggest that ACEs may have a larger impact on adult outcomes in this cohort than BCEs. Prior work by Nahar et al. (2025) found an interaction effect suggesting that BCEs moderate the relationship between ACEs, depression, and anxiety symptoms, although this buffering effect diminished at higher levels of ACE exposure. Doom et al. (2021) similarly tested whether BCEs buffered the effects of ACEs, but did not find a significant $ACE \times BCE$ interaction for depression or anxiety, and noted that ACEs and BCEs were mostly, but not strongly, negatively correlated. This is consistent with the scatterplot in this thesis showing ACEs vs BCEs (Figure 5) having a modest negative association. This may also emphasize a substantial co-occurrence: high BCE endorsement can occur at any level of ACE exposure, including among individuals with high ACE burden. This co-occurrence is consistent with the BCE literature suggesting that benevolent experiences are not merely the inverse of adversity (Narayan et al., 2018) and further shows that ACEs and BCEs are mostly independent experiences. Crandall et al. (2019) similarly used resilience theory as a framework in a cohort of 246 adults and found that when both BCEs and ACEs were in the same model,

BCEs largely neutralized the effects on ACEs. However, they found that in the face of 4 or more ACEs, this protective effect had diminished. That this pattern appears consistently across general population studies and now in a cohort of former ASF players suggests that the weight of childhood adversity may be difficult to offset regardless of the population being studied.

Methodological Considerations

Another methodological consideration involves model choice. This thesis used Gaussian GEE models with robust standard errors. Outcome measures such as PHQ-9 and GAD-7 are often right-skewed, and future work could explore alternative distributions or link functions, such as negative binomial models, to assess the robustness of these findings.

Finally, the sequential modeling framework used in this thesis, building from demographics, through football exposures, health-related factors, and then ACEs—supports transparent interpretation of how associations change with adjustment. This matters especially when studying childhood experiences in an adult cohort where factors like BMI and concussion symptoms have their own real effects on health regardless of what happened in childhood and including them in models is appropriate rather than excessive.

Biological and Developmental Context

One dimension this thesis did not directly study was the biological impact of ACEs and BCEs. However, I do find it is worth briefly acknowledging, because it may help explain the bigger picture as to why ACEs seem to leave such a lasting imprint even

among those with high BCE scores. Research has shown that early adversity has been associated with structural changes in the amygdala: stress may initially enlarge it, but severe or prolonged maltreatment has been linked to reduced development over time (Alley et al., 2025). There was another study conducted by Herzog et al. (2018) that showed that different time periods of exposures to ACEs may affect a person's response and impact both biologically and psychologically. For instance, earlier sexual abuse exposure was associated with greater risk for depressive symptoms, while later exposure was linked to more severe PTSD. Trauma exposure between ages 3 and 5 has been associated with elevated risk for PTSD, depression, and suicidality compared to exposure at other developmental windows. I think this opens an important direction for future research: examining not just whether BCEs and ACEs occurred, but when they occurred, and how the timing of both adverse and benevolent experiences may interact to shape long-term outcomes.

Strengths

Several features of this study strengthen its findings. First, the study drew on a well-characterized cohort of former professional ASF players. Second, outcomes were measured using validated instruments that are widely used and easy to interpret: PHQ-9 for depressive symptoms (Kroenke et al., 2001), GAD-7 for anxiety symptoms (Spitzer et al., 2006), and Neuro-QoL applied cognition for cognition-related quality of life (Cella et al., 2012). Third, building the models sequentially made it easier to see exactly where and why the BCE association changed as more variables were added. Finally, by explicitly modeling both ACEs and BCEs together, this thesis is among the first to test whether

BCEs contribute independently to neuropsychological outcomes in a former professional athlete population.

Limitations

Several limitations should be considered when interpreting these findings. First, BCEs and ACEs were assessed retrospectively through self-report, which introduces the potential for recall bias. Especially in a cohort where head trauma is common and the median age was 61 at the time of survey, participants were reflecting on childhood experiences from several decades prior. Time may have softened or blurred those memories, meaning experiences may not be reported with full accuracy. Additionally, current life circumstances and symptom burden may have influenced recollections such that participants with worse mood or cognitive symptoms may have differentially recalled adverse or benevolent experiences.

Second, BCE endorsement was highly skewed, with many participants reporting the maximum score. This may have limited my power to detect independent BCE associations. Instead of using the original BCE scale, future studies should consider using the revised BCE scale (Narayan et al., 2023) to address this.

Third, I may not have adjusted for other important factors such as childhood socioeconomic conditions, neighborhood environment, family structure across the life course. Bethell et al. (2019) found that lower-income adults reported fewer levels of positive experiences, suggesting that socioeconomic context may have shaped BCE exposure in ways this analysis could not fully capture. These unmeasured factors may have contributed to the observed associations.

Fourth, selection bias and limited generalizability should be considered.

Volunteer-based study samples may underrepresent certain groups of former players, particularly with respect to racial composition. For example, ACE exposure has been shown to be higher among Black, Hispanic, and multiracial adults, as well as among those with lower income and lower educational attainment (Merrick et al., 2018), while positive childhood experiences also appear to vary across racial and ethnic groups (Crouch et al., 2021). Limited diversity in a volunteer cohort may affect not just who is represented, but also how the childhood experiences are distributed across the sample.

Conclusion

This thesis addressed a key gap in athlete neuropsychiatric health research by evaluating BCEs alongside ACEs in relation to adult depression, anxiety, and cognition-related quality of life among former professional ASF players. BCEs demonstrated protective associations in minimally adjusted models for depression and anxiety, and a borderline association for cognition-related quality of life, but these associations were attenuated after accounting for ACE burden. In contrast, ACE total score remained a consistent correlate of depressive and anxiety symptom burden across progressively adjusted models, showing that what happened in childhood still matters decades later for this cohort.

What these findings ultimately suggest is that BCEs matter, but they may not fully insulate against the effects of adversity. For players in the cohort studied, many of whom are now living with the aftermath of neurological and mental health burden, it's important to study what these players experienced long before they stepped on the field. This makes a case to emphasize how important it is to mitigate ACEs as these experiences may even

undermine the benefits of BCEs. And more broadly, it makes a case for prevention: if we want to improve long-term health outcomes in this population and beyond, reducing childhood adversity in the first place may matter more than we have previously appreciated.

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