Introduction
- Adult health problems can be traced to environmental influences in childhood, and a specific cluster of these influences are called Adverse Childhood Experiences (ACEs).
- Evidence increasingly traces the origins of disparities in educational, socioeconomic, and crime outcomes to ACEs.
- ACEs can exert graded impacts through both physiological and psychosocial avenues.
- Adverse experiences are amplified by poverty, but limited research has generalized ACE effects to underserved populations.
- As evidence for these connections builds, it is also important to investigate mechanisms of resilience.

Research Questions
1. Do cumulative ACEs predict well-being in a primarily African American sample?
2. Are associations between cumulative ACEs and outcomes in adulthood strongest for participants with the highest levels of cumulative ACEs, males and for participants attending school in the highest poverty neighborhoods?
3. Do 5 Hypothesis Model (5HM) mediator variables buffer against the effects of ACEs?
4. Are these buffering effects stronger for males and for participants attending schools in the highest poverty neighborhoods?

Methods
- Setting & Participants: The sample is from the Chicago Longitudinal Study, an investigation of the effects of the CPC program. Urban, low-income, primarily African-American (94.1%) children who attended CPC beginning in preschool (n=777) were included in the analyses (n = 1202, 45.9% male).
- ACEs from 0-18: (1) prolonged absence or divorce of parents; (2) death of a parent, sibling, or close friend; (3) frequent family conflict; (4) parental substance abuse; (5) witnessing a violent crime; (6) victim of a violent crime; (7) physical abuse*; (8) sexual abuse; (9) neglect; (10) child welfare reports (abuse or neglect) from 0-3, (11) financial insecurity in 4th grade
- Note: Abuse and neglect information was drawn from administrative (DCFS) records. Financial insecurity information was drawn from parent survey records.
- 5 HM mediator variables: (1) Cognitive Advantage (2) Motivation (3) Family Support (4) Socioemotional Competencies (5) School Support
- Additional mediator variables: Juvenile arrest and high school graduation

Measures
- Subgroups: (1) Sex (2) Neighborhood poverty
- Outcomes: (1) High school graduation (2) Occupational prestige (3) Smoking status (4) Juvenile arrest (5) Adult felony charge

Results
- Associations between ACEs 0-18 and later well-being were stronger for males and participants in higher poverty neighborhoods.
- Associations between ACEs 0-5 and later well-being were only stronger for participants in higher poverty neighborhoods.

Questions 3 and 4
- 5HM mediators buffered against the effects of high ACEs. Buffering effects were stronger for males and higher poverty participants for ACEs experienced from birth to age 5, and for higher poverty participants for ACEs from birth to 18.

Discussion
- ACEs in early childhood and across childhood and adolescence impacted outcomes across domains of well-being for a low-income African-American population.
- Males and participants in poorer neighborhoods were disproportionately impacted by high ACE scores.
- 5HM mediators accounted for significant portions of the relations between ACEs and outcomes.
- Family support and socioemotional mediators specifically appeared to be driving many of the mediation effects.

Implications
- Our results suggest that research on ACE effects generalizes to low income and minority populations, and that poverty and male sex can render individuals more vulnerable to these effects.
- It should be noted that the majority of participants were living in poverty; the “higher poverty” designation is relative. The reduced impacts of ACEs on the lower poverty participants suggests that even a small decrease in neighborhood poverty may have meaningful effects.
- Fostering resources such as family and school support, socioemotional competencies, and school motivation through programs like CPC can buffer individuals from the effects of ACEs.

Future Directions
- Do levels of concurrent/retrospective distress or the type or timing of ACEs affect outcomes?
- Why and how do specific mediators differentially impact outcomes?
- Does early childhood education program attendance affect ACEs? And if so, do these effects differ by subgroups (e.g., gender, risk, and dosage)?
- Are population-specific ACEs different from traditionally measured ACEs?
- What are the relations between ACEs and physiological indicators of biological processes and health outcomes (e.g., cortisol, lipid panels, telomere length)?

References
2. Shenkoff et al. JAMA. 2009, 301(21), 2252-2259