May 2016

Response Modulation Moderates the Effect of Agonistic Striving on Cardiovascular Disease Risk in Children

Sarah Rose LaFont
Syracuse University

Follow this and additional works at: https://surface.syr.edu/etd

Part of the Social and Behavioral Sciences Commons

Recommended Citation
https://surface.syr.edu/etd/475

This Thesis is brought to you for free and open access by the SURFACE at SURFACE. It has been accepted for inclusion in Dissertations - ALL by an authorized administrator of SURFACE. For more information, please contact surface@syr.edu.
Abstract
Psychological and physiological risk indices in youth may mark early trajectories to adult cardiovascular disease. The present study tested the hypotheses that: (1) children who exhibit agonistic striving (struggling to influence, control, or change other people) exhibit increased cardiovascular risk relative to their peers; (2) children whose parents exhibit agonistic striving exhibit similar risk; and (3) the associations between agonistic striving and cardiovascular risk are moderated by the child’s response modulation abilities. Participants were 100 children aged 9 to 11 years ($M = 10.61, SD = 0.86$) and 100 parents/caregivers. Agonistic striving was measured with the Social Competence Interview; response modulation abilities were measured with: (a) a self-report index of emotion regulation, and (b) heart rate variability. Cardiovascular risk was indexed by levels of resting blood pressure, cardiovascular reactivity, arterial stiffness, and left ventricular mass of the heart. The results replicated the striving profiles observed previously in adolescents and adults. Indices of increased cardiovascular risk were associated with parent but not child agonistic striving. The degree of a parent’s agonistic goal focus interacted with the level of the child’s emotion regulation abilities and autonomic vagal control to predict higher levels of resting blood pressure and arterial stiffness in the child. Those at greatest risk were children with lower emotion regulation abilities or lower autonomic vagal control, and whose parents exhibited a high degree of agonistic focus. This study offers important new evidence that a psychological mechanism of parental stress may interact with regulatory mechanisms in the child to impair the child’s health.

Keywords: Agonistic Striving (AS), Cardiovascular Disease (CVD), emotion regulation
Response Modulation Moderates the Effect of Agonistic Striving on Cardiovascular Disease Risk in Children

by

Sarah R. LaFont

B.A., SUNY Institute of Technology, 2013

Master’s Thesis

Submitted in partial fulfillment of the requirements for the

Masters of Science in Psychology

Syracuse University

May 2016
# Table of Contents

Table of Contents .................................................................................................................. iv  
List of Tables .......................................................................................................................... v  
List of Figures ........................................................................................................................ vi 
List of Appendices ................................................................................................................ vii  

Chapters  
I. Introduction .......................................................................................................................... 1  
II. Methods .............................................................................................................................. 13  
III. Results ............................................................................................................................... 25  
IV. Discussion .......................................................................................................................... 31  

Tables .................................................................................................................................. 39  
Figures .................................................................................................................................. 40  
Appendices .............................................................................................................................. 48  
References ............................................................................................................................... 54  
Vita ........................................................................................................................................ 64
List of Tables

1. Means, Standard Deviations, and Bivariate Correlations among the Study Variables............ 39
List of Figures

1. Response Modulation Moderates the Effect of Agonistic Striving on Cardiovascular Disease (CVD) Risk .................................................................................................................. 40

2. Adult Sample Striving Profile Taxonomy ........................................................................... 41

3. Child Sample Striving Profile Taxonomy ........................................................................... 42

4. Differences in Child Resting Systolic Blood Pressure (SBP) by Parent Motive Profile ........ 43

5. Children’s Cognitive Emotion Regulation Moderates the Association between Their Parents’ Agonistic Goals and the Children’s Resting Diastolic Blood Pressure (DBP) ....................... 44

6. Children’s Cognitive Emotion Regulation Moderates the Association between Their Parents’ Agonistic Goals and the Children’s Resting Systolic Blood Pressure (SBP) ......................... 45

7. Children’s Cognitive Emotion Regulation Moderates the Association between Their Parents’ Agonistic Goals and the Presence of Arterial Stiffness in the Child ....................................................................................... 46

8. Children’s Tonic Heart Rate Variability Moderates the Association between Their Parents’ Agonistic Goals and the Presence of Arterial Stiffness in the Child .......................................................... 47
List of Appendices

A. Social Competence Interview Observer Rating Scale – Expressiveness Scale .................. 48
B. Social Competence Interview Observer Rating Scale – Self Defense and Acceptance-Affiliation Scales ........................................................................................................................................49
C. Social Competence Interview Observer Rating Scale – Approval Seeking and Self-Improvement Scales........................................................................................................................................51
D. Shift-and-Persist Questionnaire (SAPQ) - Shift Subscale .................................................. 53
Response Modulation Moderates the Effect of Agonistic Striving on Cardiovascular Disease Risk in Children

One of the greatest public health concerns facing the world today is cardiovascular disease, which includes an array of pathologies that are rooted in hypertensive and atherosclerotic processes leading to coronary heart disease and stroke (Matthews, 2013). Despite the four decade long trend of decreasing rates of cardiovascular disease mortality, cardiovascular disease continues to be the leading cause of death in the U.S. and around the world, and is estimated to cost the U.S. healthcare system $150 billion annually (Capewell et al., 2010; Lloyd-Jones et al., 2010). As the population ages and obesity and diabetes continue to increase in prevalence in younger and younger age groups, cardiovascular diseases continue to threaten the health of individuals across the life span (Lloyd-Jones et al., 2010). A better understanding of the complex and multifaceted factors that contribute to cardiovascular disease is greatly needed if we hope to counter these inauspicious trends.

Psychological Factors in Cardiovascular Disease Risk

Today it is widely accepted that psychological factors contribute to cardiovascular disease risk. The research that led to this discovery began in the 1960s and 70s, largely thanks to the work of cardiologists Meyer Friedman and Ray Rosenman. Friedman and Rosenman reported an association between coronary heart disease and a set of behavioral tendencies that included hostility, competitiveness, achievement orientation, and impatience. They labeled this cluster “Type A” personality (Friedman & Rosenman, 1959). As this research continued to advance, it encouraged the rise of new research fields such as behavioral medicine and health psychology, and helped revive a longstanding interest in understanding the link between psychological phenomena and physical illnesses (Matthews, 2013). As cardiovascular research continued within these new fields, scientists discovered that other personality traits, including
neuroticism, conscientiousness, and optimism, were associated with cardiovascular disease risk. Evidence for a connection between personality and illness became more persuasive when demonstrated prospectively in longitudinal research, and with designs that measured actual disease outcomes, not just illness behaviors (e.g., somatic complaints) that have substantial overlap with the personality constructs themselves (Chida & Steptoe, 2009; Lloyd-Jones et al., 2010).

Despite these early studies’ strengths and their seminal contributions to a new era of behavioral medicine, they were not without limitations. A major limitation was the fact that in much of this research, psychological influences were defined as personality traits, or broad behavioral averages (e.g., hostility, dominance, optimism, anxiety, depression). Yet, statistical averages do not afford causal explanations of the psychological mechanisms that contribute to disease. Trait research suggested that individual differences in cardiovascular disease risk were related to individual differences in personality, but trait research was unable to specify exactly what the connection was, or explain why it existed (Suls & Bunde, 2005). These limitations have led health scientists to argue that mechanistic research is greatly needed (Matthews, 2013). Not only does the study of causal mechanisms afford a better understanding of the causal relationships between psychological factors and disease, but it also has implications for clinical and preventive intervention (Lloyd-Jones et al., 2010).

The other major focus of psychological research in cardiovascular disease has been to identify disruptive psychological events, or stressors, that elicit large cardiovascular responses, or reactivity, and to link those events and responses to concurrent or future disease. A recent meta-analysis determined that greater cardiovascular reactivity to laboratory stressors is prospectively associated with future cardiovascular risk indices (atherosclerosis and
hypertension) and future cardiac events (Chida & Steptoe, 2010). Studying cardiovascular reactivity affords two important insights into psychological mechanisms that may affect cardiovascular health. First, this research has shown that cardiovascular reactivity increases when one is uncertain of one’s ability to predict or control a stressor. This suggests that stress is a psychological construct resulting from a perceived inability to control or influence important events (Sapolsky, 2004). Reactivity research links perceived control—a non-trait psychological causal construct—to a cardiovascular response pattern which then predicts future cardiovascular health. Second, the study of cardiovascular reactivity emphasizes the importance of preclinical cardiovascular indicators that can be linked to psychological processes. That is, although cardiac events are clear indicators of cardiovascular disease, earlier pathophysiological processes, such as cardiovascular reactivity, the stiffening of the arteries (Asmar et al., 1995), and the excessive growth of the left ventricle of the heart (i.e., increased left ventricular mass; Daniels, 2004; Gump, Matthews, & Räikkönen, 1999), can serve as early markers of a trajectory toward cardiovascular disease in childhood, long before diagnosable clinical-level pathologies emerge.

The quest to understand causal mechanisms must also address two other considerations. First, psychological and physiological processes of health and illness do not function in isolation but are influenced by and embedded within social, economic, and other environmental contexts. This implies that understanding psychological influences in cardiovascular disease requires a multilevel approach to detecting causal pathways (Matthews, 2013). Second, it is important to consider mounting evidence that atherosclerotic processes begin early in life. Studies of cardiovascular disease within a developmental perspective suggest there are increasing rates of cardiovascular disease and cardiovascular risk factors in children (Asmar et al., 1995; Lloyd-Jones et al., 2010). This evidence indicates the need for a lifespan approach to studying the
development of cardiovascular disease and psychological risk indices in youth that mark early trajectories to adult cardiovascular disease. A conceptual approach designed specifically to advance a mechanistic, multilevel, lifespan understanding of psychological factors in cardiovascular disease risk is Social Action Theory. This theory formed and guided the research reported in this thesis.

Social Action Theory

Social action theory is a social-ecological theory of self-regulation derived from the notion that to survive and flourish, humans must act upon their environment to obtain resources while modulating the environment’s internal impact (Ewart, Elder, Jorgensen, & Fitzgerald, 2016). Individuals meet this challenge by engaging in self-regulation, which in social action theory is comprised of two distinct components: self-direction and response modulation. Self-direction involves cognitive problem solving activities that generate goals, defined as cognitive representations of desired events (Ewart, 1991; Ewart, 2011). Response modulation is supported by cognitive-affective processes of attention and appraisal (Gross, 2013) as well as autonomic control (Thayer, Hansen, Saus-Rose, & Johnsen, 2009). By repeatedly engaging in self-directive problem-solving and response modulation activities in everyday situations, people form habitual routines, shared activities, and social networks that generate important resources for oneself and others. One’s routines, contacts, and networks comprise a protective social-ecological niche. Persistent threats to niche-building and maintenance foster persistent psychological states of uncertainty and generate recurring physiological and physiologic stress. Self-directive problem solving and response modulation activities that support niche-building foster resilience.

The social action theory approach to stress and resilience differs from older conceptions of stress and coping that have guided research on stress and illness. Much research has been
guided by a model of stress and coping that subdivides self-regulation into a two-stage sequence comprised of *primary* control, or “problem-focused” coping, and *secondary* control, or “emotion-focused” coping (Lazarus & Folkman, 1984; Heckhausen, Wrosch, & Schulz, 2010; Rothbaum, Weisz, & Snyder, 1982). In this model, one reacts to a stressor initially by seeking to control one’s environment; if that fails, one then attempts to dampen one’s internal reaction to the problem. Secondary control is but a fallback option to be tried only after primary control has proved impossible (Ewart, 2011). However, this conception does not adequately reflect the fact that environmental and internal control mechanisms operate in concert as different aspects of the same self-regulatory process. Problem solving and emotion regulation are not dichotomous either-or choices, but rather form two inseparable sides of the self-regulatory coin. Nor does the older dichotomous two-step model recognize that humans are self-directing social animals who select and shape their environments as well as react to them. In social action theory, people generate many of the stressors (often unknowingly) to which they must respond.

**Implicit Strivings.** Social action theory proposes that self-directive problem solving operates at different levels of consciousness. Hence, the goals that serve ecological niche-building—and often increase stress exposure—frequently are *implicit* or nonconscious (Bargh, Gollwitzer, Lee-Chai, Barndollar, & Trötschel, 2001; Custers & Aarts, 2010). This implies that the goals that foster chronic stress can be difficult to identify and measure via self-report (Ewart, 2011). Social action theory proposes that implicit stress-inducing goals can be observed and measured reliably by activating social problem-solving processes in a controlled laboratory setting using a structured interview protocol known as the Social Competence Interview (SCI; Ewart, Jorgensen, Suchday, Chen, & Matthews, 2002). The SCI is an experiential problem-solving protocol that involves re-living an ongoing personal stressor, imagining it is happening to
another person like oneself, and then inventing a desirable yet realistic resolution to the problem, and a problem-solving (coping) strategy that could lead to the desired outcome. Trained observers code the participant’s verbal responses to the SCI to identify the person’s implicit self-directive goals.

The implicit goals assessed with the SCI are characterized on two basic dimensions: direction of goal focus (change self vs. change others) and level of goal immersion (emotional investment in the goal). Social action theory proposes that recurring threats to important goals foster regulatory struggles that focus either on controlling/changing aspects of the self, or on controlling/changing aspects of the one’s social environment. These ongoing struggles gradually form self-organizing, goal-directed strivings that either involve persistently seeking to control or alter the self, or seeking to influence or control other people in one’s social milieu. Either type of persistent regulatory striving is characterized by a high level of goal immersion; that is, one is emotionally invested in achieving personal or social control. The combination of goal focus and goal immersion yields a natural taxonomy of self-organizing regulatory strivings in which an individual may be: (1) immersed in striving to control or change other people but not the self; (2) immersed in trying to control or change the self but not other people; or (3) not immersed in either goal (e.g., distressed but unable to envisage a desired outcome and problem-solving strategy). These patterns or motive profiles are known, respectively, as agonistic striving, transcendence striving, and dissipated striving (Ewart, Elder, Laird, Shelby, & Walker, 2013; Ewart, Elder, Smyth, Sliwinski, & Jorgensen, 2011; Ewart & Jorgensen, 2004).

Social action theory predicts that agonistic striving, compared to transcendent and dissipated striving, is the motive profile that is most likely to induce large cardiovascular stress responses and hence is most harmful to cardiovascular health. This hypothesis stems from
evidence that people experience greater stress when they are unable to predict and control important outcomes (Sapolsky, 2004). Since the behavior of others is inherently less predictable than one’s own behavior, agonistic striving represents investment in a goal more likely to expose a person to more stress (e.g., compared to the self-change focus of transcendence striving). Further, efforts to influence or control other people easily evoke hostile reactions and lead to ongoing power struggles that are a source of continuing unpredictability and psychological stress. As a consequence, given its orientation towards changing others, agonistic striving is more likely to induce sustained, hypervigilant states of alert attention toward others in the social environment. These hypervigilant states are associated with physiologic changes (increased heart rate, blood pressure) that, over time, foster the development of cardiovascular disease (Ewart & Jorgensen, 2004). In support of this prediction, research evidence indicates that adolescents and adults who exhibit the agonistic striving profile tend to have higher ambulatory blood pressure, especially during social interactions, than peers with the transcendence or dissipated striving profiles, even after controlling for gender, body size, and race (Ewart et al., 2016; Ewart, et al., 2011; Ewart, Elder, & Smyth, 2012b; Ewart & Jorgensen, 2004).

**Response modulation.** Whereas goal pursuit increases or lowers stress exposure, response modulation activities regulate stress responding. Social action theory holds that response modulation is accomplished both by cognitive-affective mechanisms (e.g., emotion regulation) as well as physiological autonomic mechanisms (Thayer & Lane, 2000). Important cognitive-affective mechanisms of emotion regulation include attention deployment and appraisal (Gross, 2013). Evidence indicates that these abilities are supported by autonomic regulatory mechanisms in adults and children (Di Simplicio et al., 2012; Hastings et al., 2008; Park, Van Bavel, Vasey, & Thayer, 2013; Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp,
One important marker of physiological autonomic self-regulatory ability is flexible vagal control of the heart, indexed by heart rate variability. Heart rate variability is a measure of autonomic nervous system activity and an indicator of the adaptability of an individual’s parasympathetic system, which is capable of producing quicker changes in heart rate than is the sympathetic system (Di Simplicio et al., 2012). This measure is contingent on the cardiac vagal system, which functions to maintain homeostasis and resource preservation while adapting to internal and external demands (Gentzler, Santucci, Kovacs, & Fox, 2009). Much like psychological self-regulatory abilities, having a high level of heart rate variability protects health. For example, low heart rate variability is associated with all-cause mortality and mortality from chronic heart failure (Dekker et al., 1997; Nolan et al., 1998).

Research suggests that the relationship between agonistic striving and higher blood pressure can be attenuated by response modulation abilities, such as pain tolerance and social and emotional self-regulation assessed in the laboratory and in the classroom (Ewart et al., 2013; Ewart, Elder, & Smyth, 2012a). This suggests that the ability to modulate one’s own responses is an important protective factor for cardiovascular health. Research by Ewart and associates indicates, moreover, that motive profiles and response modulation abilities represent independent and qualitatively different constructs. Individuals with the agonistic, transcendent, or dissipated striving profiles do not differ in their ability to regulate anger in the laboratory (Ewart et al., 2011), exhibit self-control in the classroom (Ewart et al., 2012a), or regulate their response to graduated thermal pain stimuli (Ewart et al., 2013). In the SAT framework, motives and response modulation processes operate together to increase or lower risk. Goals increase or decrease stress exposure, and response modulation capabilities (emotion regulation, heart rate variability) regulate stress responding (Ewart et al., 2011).
Thus, unlike older dichotomous models that viewed problem solving (or primary control) and response modulation (or secondary control) as either-or alternatives, social action theory proposes that a useful understanding of self-regulation and coping can be achieved only by examining how self-directive problem solving and response modulation mechanisms combine and interact. The theory thus encourages researchers to frame their hypotheses in terms of *interactions*, which are more difficult to detect and reliably replicate than are statistical main effects.

**The Present Study**

Social action theory supports a mechanistic, multilevel, lifespan approach to studying psychological risk factors for cardiovascular disease. Specifically, the theory is designed to facilitate multilevel analyses linking self-directive goals and response modulation mechanisms at the level of the person to social-ecological niche building processes in families, social networks, neighborhoods, and larger communities. The theory provides concepts and methods to specify and measure self-regulatory processes in children, adolescents, and adults of different ages, and thus facilitates research investigating how these processes function across the lifespan. Up to now, however, the theory has been tested only in samples of adults and adolescents—not in children. No studies have investigated social action theory constructs or mechanisms in children below the age of 13 years. Thus it is not known if the social action theory taxonomy of regulatory strivings characterizes the stress experiences of pre-pubertal youth, or if the relationships among implicit strivings, response modulation capabilities, and cardiovascular outcomes that have been observed repeatedly in previous research also are evident earlier in the lifespan.
A social action analysis of environmental engagement in ecological niche-building, for example, affords reasons to suspect that relationships among self-directive processes, response modulation abilities, and cardiovascular responding that have been observed in high school students may not characterize children in elementary or middle schools. Prior to adolescence, a child’s social-ecological niche consists of immediate family members; parents and guardians are the central actors shaping the child’s social network, daily routines, and contacts with peers. Beginning in adolescence, however, peers replace parents as the key niche actors and drivers of day-to-day concerns and social relationships (Berger, 2008). During this time, approval by one’s friends is valued more than approval by one’s parents. Adolescents, compared to younger children, also have much greater latitude to form goals and construct social networks. This ability increases further in adulthood. Thus, striving profiles and regulatory processes replicated in prior studies of high school students and adults may not typify the younger child.

Parents’ central role in shaping younger children’s social worlds and daily routines suggests another key question: Might parents’ regulatory strivings represent risk factors for their children? Might having a parent who is persistently caught up in agonistic struggles evoke psychological and physiologic responses in a child that are harmful to the child’s cardiovascular health? Might a child in this situation grow hypervigilant to social control cues that induce physiologic stress responses? Thus a question with important lifespan developmental implications is whether parental agonistic striving serves as an additional health risk factor for children. Could agonistic striving in a parent lead to higher blood pressure levels in the child? Might it contribute to defective arterial development or cardiac growth? Social action theory’s transactional analysis of personal and environmental influences suggests that parent and child regulatory processes reciprocally influence each other. A parent’s regulatory struggles shape—
and are shaped by—the child’s behavior. Child behaviors that are challenging to manage may foster parental agonistic striving, and such striving on a parent’s part may adversely affect the child’s health (Ewert, 2011). When agonistic parent-child contests persist, they may give rise to continuing coercive struggles that gradually escalate over time (Granic & Patterson, 2006), generating stressful home environments that may increase a child’s cardiovascular risk. Investigating how agonistic goals facilitate these transactional family influences could advance a mechanistic, multilevel, developmental understanding of children’s cardiovascular risk.

The present study tested the hypothesis that agonistic striving in (a) the child, and (b) the child’s parent, interacts with the child’s capacity for response modulation (cognitive emotion regulation and autonomic vagal control) to predict four indices of cardiovascular risk and preclinical disease: higher levels of resting blood pressure, increased cardiovascular reactivity to psychological stressors, the presence of increased arterial stiffness, and abnormal heart growth. Research has shown that high blood pressure in children is an important marker of increased cardiovascular disease risk. The prevalence estimates for pediatric hypertension range from 0.8% to 5%, with the most common type of hypertension among children being primary hypertension (Dekker et al., 1997; Daniels, 2004). Pediatric hypertension is among the strongest predictors of hypertension in adulthood, and thus is a critical marker for a trajectory into adult cardiovascular disease. However, sub-clinical blood pressure elevations and pathophysiological indicators also predict adult hypertension, and are thus worthy of attention (Morgenstern & Butani, 2004). Other blood pressure-related cardiovascular risk indices commonly assessed in children include cardiovascular reactivity to psychological stressors, increased arterial stiffness indexed by pulse wave velocity, and abnormal heart growth indexed by increased left ventricular mass of the heart. Cardiovascular reactivity, defined as the magnitude of the cardiovascular response in
reaction to stress and often represented by change ($\Delta$) scores of cardiovascular indices from resting/baseline to stressful tasks, is an important indicator that predicts risk of future hypertension, arterial damage, and related cardiovascular disease (Allen, Matthews, & Sherman, 1997; Matthews, Salomon, Brady, & Allen, 2003). Pulse wave velocity is an index of arterial stiffness that measures the speed at which pulse pressure travels from the carotid artery to the femoral artery (Spartano et al., 2014). Indexing arterial stiffness in children is important because arterial stiffness contributes to eventual organ damage, yet can be detected before blood pressure increases manifest. Finally, left ventricular mass of the heart, defined as the mass of the walls of the left ventricle in $g/m^2$, is another important indicator of pediatric cardiovascular health (Malcolm, Burns, Mahoney, & Lauer, 1993). The left ventricle grows naturally throughout the life span, yet increased blood pressure can expand the muscles of this chamber, making an increased left ventricular mass an early indicator of high blood pressure in children as well as a marker for future blood pressure increases (Daniels, 2004). Given the relatively low prevalence of pediatric hypertension, it is critical to investigate earlier, sub-clinical indicators, such as pulse wave velocity and left ventricular mass, in order to better discern risk trajectories among children.

The present study tested the hypothesis that agonistic striving is associated with cardiovascular disease risk indices in children living in low- to middle-income Syracuse neighborhoods, and that this association is moderated (attenuated) by the child’s response modulation abilities, indexed by self-reported cognitive emotion regulation (distraction, appraisal) abilities and autonomic vagal control indexed by tonic (resting) heart rate variability. Further exploratory analyses tested a corresponding ancillary hypothesis that agonistic striving in parents is associated with increased cardiovascular disease risk in their children, and that this
association is moderated (attenuated) by the child’s response modulation abilities. These analyses used data from the Environmental Exposures and Child Health Outcomes (EECHO) study, a research project investigating the relationship among exposures to toxic environmental substances and cardiovascular disease risk indices in children living in low- to middle-income neighborhoods of Syracuse, NY. Data collected from the children and their parents was used to test the theoretical model shown in Figure 1.

**Ancillary analyses.** In the event that the tests of the study hypotheses yielded statistically significant findings that supported the proposed theoretical models, additional post-hoc ancillary analyses were planned to identify factors that might explain the link between agonistic striving by the parent and cardiovascular risk indices in the child. These factors included characteristics of the child, the parent, and the larger community environment that may foster agonistic parent-child struggles with the potential to affect the child’s health. Child characteristics included externalizing behavior and symptoms of autistic spectrum disorder. Parent characteristics included perceived stress and depression. Environmental characteristics included the family’s socioeconomic status and exposure to perceived neighborhood stress.

**Methods**

**Sample**

Study participants were 100 children and 100 parents/guardians from the ongoing EECHO study, a research project investigating the relationships among exposures to environmental toxicants and cardiovascular risk indices in 9-11 year old children living in low- to middle-income neighborhoods in Syracuse. The EECHO is designed to generate a sample containing equal numbers of African American and Caucasian, male and female children;
participants are recruited from the following U.S. postal zip-codes: 13202, 13203, 13204, 13205, 13206, 13207, 13208, 13210 or 13224. The child participants had an average age of 10.61 (SD = 0.86), 52% were Black, 48% were White, and 46% were female. Children were eligible for the study if they met the EECHO race, age, and zip code residence selection criteria, did not have serious medical or developmental disabilities, and were not taking medications that might affect their cardiovascular system. Data for the present thesis study was obtained from the first 100 children and 100 parents who completed the Social Competence Interview.

Measures

**Agonistic striving.** Agonistic striving in both parent and child participants was assessed using the 10-minute Social Competence Interview (SCI; Ewart et al., 2002). In the Social Competence Interview, the interviewer first asked a participant to select and then describe an ongoing problem (chronic stressor) in detail. The structured interview protocol is divided into two five-minute phases. In the first “hot” (emotionally evocative) phase of the interview, the interviewer helped the participant re-live a particular stressful incident that exemplified the recurring problem. The participant was encouraged to re-experience and describe the thoughts and feelings that he or she experienced during the stressful event. In the second “cool” (reflective) phase of the interview, the interviewer asked the participant to imagine that he or she was a film director who was making a film about a person like the participant who had a similar problem. The participant was asked to invent a desirable but realistic ending for the film, and to describe what the characters in the story might do to make this ending happen. Participants then were asked to apply the film story to themselves by explaining how they might apply the film ending and plot to their own problem situation. They were asked to describe the actions they might take to achieve their desired outcome and the consequences might follow after they took
the necessary action. The film ending and action plan (problem-solving strategy) reveal the participant’s implicit action goals (agonistic, transcendent) when confronting the chronic stressor. Interviewers were female graduate students in clinical psychology who were trained by SCI developer, Ewart following a detailed manual (Ewart, Suchday, & Sonnega, 1997).

Audio recordings of the interviews were coded by independent observers using a reliable and valid behavioral coding system that measures the implicit goals that are associated with a chronic stressor, as well as the person’s degree of immersion (emotional investment) in those goals (Ewart et al., 2002). Goal immersion is indexed by the participant’s degree of emotional expressiveness when reliving the stressful incident during the initial hot phase of the SCI. Expressiveness was calculated using observer ratings of participants’ expressive style, including speech characteristics such as “Speaks loudly,” “Speaks rapidly,” “Gives detailed responses,” and “Voice easily expresses emotion.” A copy of the SCI Expressiveness scale is provided in Appendix A. Agonistic and transcendent goal scores were computed from observer ratings of participants’ film narratives. Agonistic Goal scores were derived from ratings of the SCI Self-Defense and Acceptance-Affiliation goal scales (Appendix B). Transcendence Goal scores were derived from ratings of the SCI Approval Seeking and Self-Improvement goal scales (Appendix C). All goal scale ratings were made using a 5-point Likert-type scale (1 = Not at all; 5 = Very much). Each interview was coded by two independent coders in order to monitor interrater reliability. Any significant discrepancies (ratings that differed by more than 1 point) were resolved by the author. These resolved ratings were used to calculate interrater reliability and were used in the final analyses. Inter-rater reliability for the Expressiveness, Agonistic Goals, and Transcendence Goal scales was excellent for both adults and children (rs ranging from .89 to .96). The internal consistencies of the scales, indexed by Cronbach’s alpha, were the following in
children and parents, respectively: Expressiveness, .93, .88; Agonistic Goals (Self-Defense, .70, .81; Acceptance-Affiliation, .79, .75); Transcendence Goals (Approval-Seeking, .89, .86; Self-Improvement, .86, .65). A previous study found these scales to have adequate temporal stability (rs ranging from .40 to .79) over a 3-month period (Ewart et al., 2002).

**Response modulation abilities.** Participants’ response modulation abilities were assessed by measuring: (a) self-reported use of cognitive emotion regulation techniques, and (b) tonic heart rate variability. The use of cognitive emotion regulation techniques was measured with the Shift subscale of The Shift and Persist Questionnaire (Chen, McLean, & Miller, 2015; Chen & Miller, 2012), a 12-item scale that assesses the ability to regulate negative emotions by refocusing one’s attention on positive outcomes (i.e., “shifting”; Appendix D). The Shift scale has been used previously with participants aged 9-18 years (Chen et al., 2011), and has good internal consistency (Cronbach’s α = .88 in the current sample).

A physiologic index of participants’ ability to modulate negative emotions was afforded by the assessment of autonomic vagal control of the heart indexed by resting (tonic) heart rate variability. Heart rate variability is the autonomically-regulated fluctuation/variation of heart rate (Dekker et al., 1997). Evidence suggests that very early in life, stable individual patterns of cardiac vagal tone emerge (Fracasso, Porges, Lamb, & Rosenberg, 1994), making heart rate variability a viable area of study in child populations. The high frequency spectral component of heart rate variability was measured with electrocardiographic (ECG) data that generate measurements of the interbeat interval, or the amount of time between consecutive heart beats. The sampling rate used to generate interbeat interval files from the ECG data is 500Hz, which is within the sampling rate recommended by the Task Force of the European Society of Cardiology (Heart rate variability, 1996). The interbeat interval files were generated by the COP-WIN
computer program; these files were used to derive heart rate variability using Nevrokard™ software. The high frequency spectral component of heart rate variability affords a relatively pure index of parasympathetic activity (Heart rate variability, 1996). Heart rate variability data were log transformed due to non-normality.

**Cardiovascular disease risk indices.** Indications of the child’s cardiovascular disease risk were obtained from four indices: Resting blood pressure and heart rate level, cardiovascular reactivity, arterial stiffness, and abnormal heart growth. The participant’s resting level of heart rate, systolic and diastolic blood pressure, and total peripheral resistance were obtained at 30-second intervals during the final three minutes of the resting baseline that preceded the laboratory stress task protocol. The mean of these six resting baseline readings was used for analyses. The readings were obtained with an automated Vasotrac device (APM 205A; Medwave, Danvers, MA). Total peripheral resistance is defined as the overall resistance to blood flow throughout systemic blood vessels (Mattoo & Gruskin, 2004). It was calculated as follows: First, cardiac output (CO) and mean arterial pressure (MAP) were computed from the resting systolic blood pressure (SBP) and diastolic blood pressure (DBP) readings using the following formulas: MAP = ((SBP - DBP)/3) + DBP and CO = stroke volume (SV) x heart rate (HR). Finally, total peripheral resistance (TPR), which is a function of cardiac output and mean arterial pressure, were calculated using the following formula: TPR (dyne-seconds X cm⁻⁵) = (MAP/CO) x 80.

The participant’s cardiovascular reactivity to psychological stressors was indexed by measuring changes in heart rate, blood pressure, total peripheral resistance, and mean arterial pressure during three stress tasks. A reactivity (difference) score for each variable was calculated by subtracting the mean resting baseline level of the variable from the mean level recorded
during the three stress tasks. To enhance the reliability of these reactivity measures, each participant’s cardiovascular reactivity indices were defined as the average z-score of their cardiovascular reactivity to the three stressor tasks (Jackson, Treiber, Turner, Davis, & Strong, 1999; Kamarck, Jennings, & Manuck, 1992). The three tasks, which were presented in counterbalanced order, consisted of reaction-time, mirror image tracing, and signal detection challenges. The reaction time task was developed in prior studies (Gump et al., 2011). Reaction time is an auditory choice reaction time task with two trials (2.5 minutes each). In the first “easy” trial, participants are presented with either a 1000 Hz (target) or 2000 Hz (distracter) tone, and in the second “hard” trial, they are presented with either a 1000 Hz (target) or 1500 Hz (distracter) tone on a variable interval schedule of 10 seconds. This type of task elicits a β-adrenergic response in many participants (Allen, Boquet, & Shelley, 1991). The mirror image tracing task, is a 3-minute task in which a subject traces an outline of a star on a computer program in which the cursor movement “mirrors” the movement of the mouse. This task has reliably demonstrated an ability to increase total peripheral resistance due to increased α-adrenergic activity (Kasprowicz, Manuck, Malkoff, & Krantz, 1990). The 10-minute signal detection task involves 5 separate signal detection conditions. In these conditions, the presentation rate of the target stimulus (the number 9) occurs at a varying rate (of 10%, 30%, 50%, 70%, or 90% with 200 trials per condition). The numerical stimuli (numbers 0-9) are presented in a rapid random sequence, with a 250 ms stimulus interval and a 500 ms interstimulus interval. The 5 separate conditions are presented in randomized blocks. This task elicits both β-adrenergic and α-adrenergic responses (Hahn & Stolerman, 2005). Using this particular combination of tasks is generally regarded as desirable in cardiovascular reactivity.
assessment because it widens the range of stressors and thus maximizes response variability (Kamarck & Lovallo, 2003).

The third cardiovascular disease risk index, arterial stiffness, was indexed by arterial pulse wave velocity, which was measured via applanation tonometry (AtCor Medical, SphygmoCor Technology, Sydney, Australia). A high fidelity pressure transducer was used to measure pressure waveforms between the right common carotid artery and the right femoral artery over a 10 second period. The distance between the two sites was measured and pulse wave velocity was calculated using the difference in the distances between sites and the measured time delay between proximal and distal waveforms. Pulse wave velocity is measured in m/s (Augustine et al., 2016). This system has demonstrated excellent inter-observer agreement and intra-observer reliability (Asmar et al., 1995). A greater pulse wave velocity is indicative of greater arterial stiffness.

The fourth cardiovascular disease risk indicator, abnormal heart growth, was indexed by excessive growth of the heart’s left ventricle. This was measured by calculating the ventricle’s size or mass. Left ventricular mass was computed using two-dimensionally directed M-mode echocardiograms performed and digitally recorded using a Sonos 5500 Phillips cardiac ultrasound unit according to the American Society of Echocardiography. Raw left ventricular mass (in units of g/m²) was converted into a left ventricular mass index by dividing left ventricular mass by participant height in centimeters (Daniels, Kimball, Morrison, Khoury, & Meyer, 1995). Twenty participants received a second echocardiogram at least 1 month after their first echocardiogram in order to calculate temporal reliability. These repeat echocardiograms were not identified to the sonographer or physician. The temporal reliability of the left ventricular mass index was good ($r = .72, p < .001$).
**Psychosocial variables for ancillary analyses.** Eight variables representing child, parent, and environmental characteristics were assessed to determine in post-hoc ancillary analyses if these variables accounted for any significant interactions between agonistic striving, response modulation, and the cardiovascular outcomes.

*Child* characteristics included *autism spectrum symptoms* and *externalizing behaviors*. Child autism spectrum symptoms were measured using the Autism Spectrum Quotient – Adolescent Version (Baron-Cohen, Hoekstra, Knickmeyer, & Wheelwright, 2006). The Autism Spectrum Quotient is a 50-item parent-rated questionnaire that assesses symptoms of autism spectrum disorder in children. This questionnaire has previously exhibited construct validity as indicated by its significant association with clinical diagnoses (Baron-Cohen et al., 2006). This questionnaire also demonstrated internal consistency in the current sample (Cronbach’s alpha = .72). Child externalizing behaviors were measured using the Child Behavior Checklist (Achenbach, 1991), a parent-reported questionnaire that measures a number of behavioral and emotional problems among children aged 6-18, including externalizing behaviors. The Child Behavior Checklist externalizing behaviors scale evidenced excellent internal consistency in the current sample (Cronbach’s alpha = .94).

*Parent* characteristics included *perceived stress* and *depression*. A 5-item version of The Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983) was used to assess parent perceived stress over the past month. The Perceived Stress Scale evidenced good internal consistency in the current sample (Cronbach’s alpha = .83). Parent depression was measured using the Center for Epidemiologic Studies Depression Scale (Radloff, 1977). The Center for Epidemiologic Studies Depression Scale asks participants to rate the frequency in which they experienced 20 symptoms of depression in the past week on a 1 (less than 1 day) to 4 (5-7 days)
scale. This scale evidenced excellent internal consistency in the present sample (Cronbach’s alpha = .92).

*Environmental* characteristics included *socioeconomic status and neighborhood stress*. Socioeconomic status was calculated based on parental occupation, income, and education data. Level of parental education was based on a 1-7 scale and parental occupation was based on a 1-9 scale, as described in Hollingshead (1975). Parental income was based on a 1-10 scale and adjusted by dividing income by the square root of the number of people living in the household (cf. Rognerud & Zahl, 2006). Occupation, income, and education data from each of these three indices was averaged across parents for each participant if data from both parents was available. Income, education, and occupation data was then converted into a z-score and SES score was calculated as the mean of these three z-scores. Neighborhood stress in parents and children was measured with the City Stress Inventory (Ewart & Suchday, 2002). The City Stress Inventory is an 18-item questionnaire that assesses the frequency of adverse events in one’s neighborhood on a 4-point scale ranging from “1 = Never.” to “4 = Often.” This scale evidenced excellent internal consistency for parent (Cronbach’s alpha = .93) and child (Cronbach’s alpha =.91) participants.

**Data Analysis**

Descriptive analyses, multiple regressions, and *k*-means cluster analyses were performed using SAS version 9.4. In the multiple regression analyses (Wampold & Freund, 1987), cardiovascular disease risk indices were regressed on agonistic striving, response modulation abilities, and their interactions. The predicted modulating effects of cognitive emotion regulation and heart rate variability were tested in separate analyses. Each model included race, age, body mass index (BMI) percentile, and gender as covariates. Continuous independent and moderating variables were mean centered for the regression analyses.
In these regression models the central independent variable, agonistic striving, was represented in two different ways. In the primary tests of the study hypotheses, agonistic striving was defined as a dummy coded group or categorical motive profile variable, consisting of agonistic, transcendence, and dissipated striving groups. To identify the three implicit striving profiles, $k$-means cluster analyses were performed, according to procedures used in previous research (Ewart & Jorgensen, 2004). Significant effects of motive profile ($p < .05$) were examined with planned follow-up contrasts to determine if the direction of the group differences matched the predicted pattern (e.g., higher cardiovascular risk for agonistic striving). Significant interaction effects of the continuous agonistic goal score and response modulation were examined with follow up simple slope analyses in order to clarify the nature of the interactions.

To examine the simple slopes, participants were separated into three response modulation groups. The “high” group represented children whose response modulation score was more than one standard deviation above the mean. The “moderate” group represented children whose response modulation score was within one standard deviation (above/below) of the mean. The “low” group represented children whose response modulation score was more than one standard deviation below the mean.

The Low slope represents the children whose emotion regulation (Shift) score was equal to or less than 1 standard deviation (SD) below the mean. The Moderate slope represents the children whose emotion regulation score was above -1 SD and below +1 SD. The High slope represents children whose emotion regulation score was 1 SD above the mean or greater.

In a secondary set of analyses, the agonistic striving construct was represented as the participant’s score on the Agonistic Goal scale, which is a continuous variable representing the sum of the individual’s scores on the SCI Self-Defense and Acceptance-Affiliation subscales.
The categorical agonistic striving variable affords a *person-centered* analysis that uses the social action theory striving taxonomy to identify groups of individuals whose distinctive patterns of goal focus and goal immersion may be associated with different illness risks. The secondary Agonistic Goal analysis affords a *variable-centered* analysis testing the social action theory hypothesis that one’s risk of cardiovascular disease rises as one’s agonistic goal focus increases (and one’s response modulation ability declines). The person-centered, taxonomy-based analysis is regarded as more theoretically and clinically informative, as it allows an investigator to examine how different configurations of goals are related to different illnesses, and has practical implications for identifying subgroups of higher-risk individuals (Maisto et al., in press).

However, the variable-centered analysis, which replaces a categorical (motive profile group) predictor variable with a continuous (agonistic goal score) predictor variable, may afford greater statistical sensitivity to the health consequences of having an agonistic goal focus (focusing on controlling others). This consideration is important in the present study given the number of predictor variables (e.g., independent variable, moderator, and four covariates) in the current models and the sample size of 100 participants in each regression analysis.

To address the ancillary hypothesis, models similar to those described above were analyzed, but with parent agonistic striving as the predictor variable.

In the event of a significant interaction between agonistic striving and response modulation in predicting any cardiovascular disease risk index, ancillary post-hoc analyses were planned. In these analyses, post-hoc covariates (child, parent, environment) entered separately into each significant model to determine whether the predicted relationships were better explained by the covariates. These variables included environmental, child, and parent variables (see above) that could potentially contribute to agonistic struggles between parents and children.
Power analyses. Prior to the study, calculations were performed to determine if the proposed research design would have sufficient statistical power to test the primary hypotheses. Statistical “power” refers to the probability of correctly rejecting the null hypothesis. Power is a function of sample size, the probability of a Type I error (α), standard error, and the magnitude of the difference between the two sample means (Howell, 2010). The statistical power of the present design was estimated by considering the predicted effect size, defined as the predicted magnitude of the differences between the agonistic striving and transcendence striving group mean scores on cardiovascular disease risk indices.

The predicted effect size was determined by examining the effect sizes reported in previous studies of agonistic striving and transcendence striving profiles, and their interactions with emotion regulation variables. In two previous studies with different adolescent samples, the magnitude of the difference between the ambulatory blood pressure levels of persons with the agonistic striving profile, contrasted with persons who exhibited the transcendence striving profile, was about two-thirds a standard deviation higher for waking diastolic blood pressure (d = .69), and about three-quarters a standard deviation higher for diastolic blood pressure during social interactions (d = .79; Ewart & Jorgensen, 2004). Further, studies investigating the interaction of motive profiles with emotion regulation have shown that, after controlling for sex, body mass index, emotion regulation, and motive profiles (agonistic and transcendence striving), the interaction of agonistic striving and emotion regulation accounts for about 32% of the variance in ambulatory systolic blood pressure (η² = .32; Ewart et al., 2012a). Based on these data, effect size estimates of these magnitudes were used to calculate the statistical power of the proposed design using G*Power, Version 3.1.5. The calculations indicated that statistical power of .80 at p = .05 would be achieved with a total sample size of 68. Thus, the current study sample
of 100 child and 100 parent participants should afford more than adequate statistical power to test the primary study hypotheses.

In the current analyses, effect sizes of the study’s primary hypotheses were tested using the Cohen’s $d$ formula: \( \frac{(M_2 - M_1)}{SD_{\text{pooled}}} \) in which \( M_2 \) and \( M_1 \) represent the means of the agonistic and transcendence groups and \( SD_{\text{pooled}} = \sqrt{\frac{(SD_1^2 + SD_2^2)}{2}} \) (Howell, 2010).

**Results**

Of the 110 parents and 117 children who initially consented to participate, 10 adults and 17 children did not complete the data collection protocols. Reasons for noncompletion included: (a) SCI interviewer not available (children = 9, adults = 6); (b) adult is not the child’s primary guardian = 3 adults; (c) SCI not possible due to hearing impairment (adults = 1); (d) participant could not complete the SCI due to fatigue or emotional distress (children = 8). The data reported in the present analyses are from the first 100 parents and 100 children who completed the SCI. Table 1 shows the means, standard deviations, and bivariate correlations among the study variables.

**Replicating the Motive Profile Taxonomy**

First, $k$-means cluster analyses were performed separately in the child sample and in the parent sample to determine if the resulting clusters corresponded to the predicted motive profiles. The social action theory taxonomy of implicit strivings predicted that the analysis would yield three clusters, each with a distinctive profile of scores on the SCI Expressiveness, Agonistic Goal focus, and Transcendence Goal focus scales as follows: (1) high Expressiveness, low Agonistic Goal focus, high Transcendence Goal focus (transcendence striving); (2) high Expressiveness, high Agonistic Goal focus, low Transcendence Goal focus (agonistic striving); and (3) low Expressiveness, low Agonistic Goal focus, and low Transcendence Goal focus (dissipated
striving). In the adult sample, the cluster analysis supported a three-cluster solution and revealed the predicted pattern of agonistic, transcendence, and dissipated motive profiles that closely matched the patterns obtained in previous studies (Ewart et al., 2013; Ewart et al., 2011; Ewart & Jorgensen, 2004; Maisto et al., in press). As in previous studies, the distribution of agonistic, transcendent, and dissipated striving groups was relatively equivalent, with 36% transcendent, 28% agonistic, and 36% dissipated in the present adult sample. Figure 2 displays the adult cluster profiles, represented as T-scores on the Expressiveness, Agonistic Goal focus, and Transcendence Goal focus scales in the present adult sample (top), juxtaposed with a the cluster profiles obtained in an earlier study of adults (bottom; Ewart et al., 2016).

In the child sample, the cluster analysis again supported a three-cluster solution and revealed the predicted pattern of agonistic, transcendence, and dissipated motive profiles that closely matched the patterns obtained in earlier studies with adolescents and adults. The percentages of individuals in the different motive profile groups differed somewhat from the percentages that have been observed in earlier studies. More than half of the participants exhibited the agonistic striving profile (55%), approximately one-third exhibited the dissipated striving profile (31%), and a minority exhibited the transcendence striving profile (14%). Children in the agonistic, transcendent, and dissipated groups did not differ in age, gender, race, or BMI (all ps > .05). Figure 3 displays the child profiles (top), juxtaposed with figures of two previous adolescent samples (Ewart et al., 2011).

**Does Agonistic Striving Predict Cardiovascular Disease Risk?**

Having replicated the motive profile groups in adults and children, it now was possible to perform person-centered analyses to determine if a child’s cardiovascular disease risk is greater
if the child, or the child’s parent, exhibits the agonistic striving profile rather than the
transcendent striving or the dissipated striving profile.

**Child sample.** Regression models testing study hypotheses indicated that the motive
profiles in the child sample were not significantly associated with any cardiovascular disease risk
indices, nor did they interact with response modulation (emotion regulation or heart rate
variability) to predict any cardiovascular disease risk indices (all $p > .05$).

**Adult sample.** Regression analyses testing the study hypotheses revealed that a parent’s
motive profile was significantly associated with their child’s resting systolic blood pressure ($b =
4.40, SE = 1.60, t = 2.75, p < .01$). Children whose parents exhibited the dissipated striving
profile ($M = 121.23; SD = 13.96$) had the highest resting systolic blood pressure, followed by
children whose parents exhibited the agonistic striving profile ($M = 117.95; SD = 12.64$), and
children whose parents exhibited the transcendence striving profile ($M = 111.89; SD = 12.25$)
had the lowest resting systolic blood pressure. Follow up contrasts (see Figure 4) revealed that
significant group differences emerged between the DS and TS groups ($F = 7.36, p < .01$) and the
combined DS/AS groups and TS group ($F = 7.02, p < .01$). No other significant group
differences emerged. The association between parent motive profile and child resting systolic
blood pressure was not moderated by either the child emotion regulation or the heart rate
variability indices of response modulation ability. Parent motive profile did not predict any of the
other child cardiovascular disease risk indices (all $p > .05$).

Given the results of the contrasts, the agonistic and dissipated parent groups were
collapsed and compared to the transcendence parent group in order to determine the effect size of
the difference in child resting systolic blood pressure between the groups. The effect size
(Cohen’s $d$) of the difference in the combined agonistic/dissipated and transcendence parent
groups for child’s resting systolic blood pressure was .61. This indicates that the Agonistic + Dissipated groups’ children differed from the Transcendence groups’ children in resting systolic blood pressure by approximately two-thirds of a standard deviation.

Do Agonistic Goals Predict Cardiovascular Disease Risk?

Variable-centered analyses that replaced the agonistic striving profile (categorical) predictor variable with the Agonistic Goal focus score (continuous) variable were performed to determine if the level of a child’s cardiovascular disease risk rose as the degree of the child’s or the parent’s agonistic goal focus increased. These analyses substituted the Agonistic Goal focus score for the Motive Profile variable in the regression models described above.

Child sample. Regression analyses using the child’s agonistic goal focus score as the predictor variable indicated that the degree of a child’s aagnostic goal focus was negatively associated with the child’s arterial stiffness, indexed by pulse wave velocity ($b = -0.03, SE = 0.01, t = -2.49, p < .05$). Although the observed association was statistically significant, it was in a direction opposite to that predicted by the study hypotheses. No other significant associations emerged between children’s agonistic goal focus and their cardiovascular disease risk indices, nor did any significant interaction of child agonistic goal focus and response regulation indices emerge in predicting cardiovascular disease risk.

Adult sample.

Blood pressure. Regression analyses using the parent’s agonistic goal focus score as the predictor variable indicated that that the degree of a parent’s agonistic goal focus interacted with their child’s use of cognitive emotion regulation techniques (Shift score) to predict the child’s resting diastolic blood pressure ($b = -0.06, SE = 0.02, t = -2.57, p < .05$). The simple slopes of the
Low (m = 5.44) and Moderate (m = 2.45) emotion regulation groups were statistically significant ($p < .01$), whereas the slope for the High emotion regulation group (m = -0.55) was not ($p > .05$; see Figure 5). The pattern of slopes indicated that, as predicted, the positive association between a parent’s agonistic goal focus and the child’s level of resting diastolic pressure increased as the child’s cognitive emotion regulation abilities declined. At the highest level of cognitive emotion regulation ability (i.e., + 1 SD or more above the mean), the level of the child’s diastolic pressure was unrelated to the level of parent’s agonistic goal focus.

Results also disclosed that the parent’s agnostic goal focus score interacted with their child’s use of cognitive emotion regulation to predict the child’s resting systolic blood pressure ($b = -0.09$, $SE = 0.03$, $t = -2.54$, $p < .05$). The simple slopes of the Low (m = 7.10) and Moderate (m = 2.36) emotion regulation groups were statistically significant ($p < .01$ and $p < .05$, respectively), whereas the slope for the High emotion regulation group (m = -2.38) was not ($p > .05$; see Figure 6). The pattern of slopes indicated that, as predicted, the positive association between a parent’s agonistic goal focus and the child’s level of resting systolic pressure increased as the child’s cognitive emotion regulation abilities declined. At the highest level of cognitive emotion regulation ability (i.e., + 1 SD or more above the mean), the level of the child’s systolic pressure was unrelated to the level of parent’s agonistic goal focus.

**Arterial stiffness.** The regression analysis indicated that the degree of a parent’s agonistic goal focus interacted with the child’s use of cognitive emotion regulation techniques to predict the increased presence of arterial stiffness indexed by pulse wave velocity ($b = -0.005$, $SE = 0.002$, $t = -2.42$, $p < .05$). An examination of the simple slopes showed that the (negative) slope of the High emotion regulation group (m = -0.28) was statistically significant ($p < .05$), whereas the slopes for the Low (m = 0.17) and Average (m = -0.05) emotion regulation groups were not
The pattern of slopes is generally consistent with the prediction that cognitive emotion regulation capabilities protect children from the stressful impact of parental agonistic striving. A high level of emotion regulation ability (+ 1 SD or more above the mean) is protective if the child’s parent exhibits a very high level of agonistic goal focus. However, the nonsignificant slopes evident at Moderate and Low levels of emotion regulation ability indicate that the health-protective effect of cognitive emotion regulation becomes detectable only when a child’s response modulation capabilities and a parent’s agonistic goal focus are both high.

A parent’s degree of agonistic goal focus also interacted with their child’s level of tonic heart rate variability to predict increased arterial stiffness in the child, indexed by pulse wave velocity (b = -0.03, SE = 0.01, t = -2.82, p < .01). The (negative) simple slope of the High heart rate variability group (m = -0.32) was statistically significant (p < .05), whereas the slopes for the Low (m = 0.06) and Moderate (m = -0.13) heart rate variability groups were not (p > .05; see Figure 8). The pattern of slopes is generally consistent with the prediction that higher levels of heart rate variability protect children from the stressful impact of parental agonistic striving. A high level of heart rate variability (+ 1 SD or more above the mean) is protective if the child’s parent exhibits a very high level of agonistic goal focus. However, the nonsignificant slopes evident at Moderate and Low levels of heart rate variability indicate that the health-protective effect of enhanced autonomic vagal control becomes detectable only when a child’s level of heart rate variability and a parent’s agonistic goal focus both are very high.

**Cardiovascular reactivity.** The degree of a parent’s agnostic goal focus was significantly associated with their child’s cardiovascular responsiveness to laboratory stress tasks indexed by diastolic reactivity (b = -0.04, SE = 0.01, t = -3.42, p < .01), heart rate reactivity (b = -0.04, SE = 0.02, t = -2.32, p < .05), and mean arterial pressure reactivity (b = -0.03, SE = 0.01, t = -2.70, p < .01).
However, the direction of these associations was opposite to the directions predicted by the study hypotheses. These three main effects were not moderated by either of the response modulation indices.

*Abnormal heart growth.* Significant associations or interactions between the degree of a parent’s agonistic goal focus and abnormal heart growth in their child indexed by increased left ventricular mass were not detected.

*Ancillary analyses controlling for environmental, child, and parent variables.* Post-hoc ancillary analyses of all significant interactions disclosed that none of the above significant interactions became nonsignificant ($p > .05$) when including socioeconomic status, parent-rated city stress, child-rated city stress, parent-rated child autism spectrum symptoms, parent-rated child externalizing behavior, parent perceived stress, or parent depression as covariates in the above models.

**Discussion**

Present findings offer important new evidence supporting a social action theory approach to chronic stress and related illnesses. This evidence suggests that social action theory’s concepts and methods may help advance a mechanistic, multilevel, life span approach to understanding how psychological processes contribute to cardiovascular disease.

First, the present results again replicated the social action theory taxonomy of motive profiles in a new sample of adults, while for the first time replicating this taxonomy in younger children aged 9 to 11 years. Before this, the youngest participants in previous tests of social action theory were 13 years old. Thus the present results represent an important extension of this theory into a younger sample of children (Ewart et al., 2002). It now appears that the agonistic,
transcendent, and dissipated striving profiles that have been observed in five community-based studies of adults and adolescents are evident earlier in life. Children, too, tend to cluster into three groups whose stress-induced regulatory struggles match the agonistic, transcendent, and dissipated patterns of goal focus and immersion. Interestingly, compared to the present sample of adults and to previous samples of adults and adolescents (Ewart et al., 2011; Ewart et al., 2016), the proportion of children who exhibited the agonistic striving profile (55%) was somewhat larger, and the proportion who exhibited the transcendence striving profile was somewhat smaller (14%). These frequencies prompt important questions warranting further research. For example, do children tend to be more susceptible to agonistic struggles than adolescents and adults? Do the more equivalent distributions among adolescents and adults suggest that some originally agonistically-focused children become transcendence-focused as they grow older? Future longitudinal research could elucidate the developmental trajectory of agnostic striving.

Although the taxonomy of motive profiles was replicated in the present sample of children, tests of the study’s hypotheses did not detect significant associations between these three striving profiles and the children’s indices of cardiovascular disease risk. Thus the study did not offer evidence that agonistic striving in a child may impair the child’s cardiovascular health. The one significant association linking a child’s agonistic goal focus to arterial stiffness was in a direction opposite to that predicted. As this unexpected result has not been observed previously, and is inconsistent with existing theory, it is not possible to draw reliable conclusions from it. One potential explanation for the overall lack of significant associations between children’s goals and their cardiovascular risk is that children’s goals are less temporally stable than the goals of adolescents and adults. The present data do not allow for an examination of temporal reliability of child goals; hence, it is not known whether child goals are less stable, thus
less likely to affect the child’s cardiovascular health. The overall lack of significant associations between child goals and cardiovascular risk indices is also consistent with an observation offered in the Introduction concerning differences in the ways that goals shape stress exposure in adolescents or adults, on the one hand, and in younger children on the other. Younger children’s exposure to stressors is shaped extensively by their parents, who organize and determine children’s everyday activities and routines while also controlling the child’s social environment. Hence a child’s self-directive strivings may affect the child’s health less than do the self-directive strivings of the child’s parent.

In line with this potential explanation, indices of parents’ agonistic striving and degree of agonistic goal focus predicted several important child cardiovascular risk indicators. A parent’s agonistic striving represented as a categorical motive profile variable was associated with their child’s resting systolic blood pressure. Follow-up contrasts revealed that children whose parents exhibited the agonistic and dissipated striving profiles had higher levels of resting systolic blood pressure than did children whose parents exhibited the transcendence striving profile. Transcendence striving in parents, compared to agonistic and dissipated striving, was associated with lower resting systolic blood pressure in their children. Interestingly, the effect size of this difference (approximately two-thirds of a standard deviation) was similar to effect sizes reported in previous research that measured the association between one’s own motive profile and one’s own blood pressure. Hence, the magnitude of difference in children’s resting systolic blood pressure by parent motive profile is approximately as large as the effect that adolescents’ and adults’ own motive profiles have on their own blood pressure. Furthermore, as in previous literature, the transcendence profile was most protective for cardiovascular health.
Indexing parent agnostic striving with the continuous agonistic goal-focus score afforded additional insights into the association between a parent’s motives and their child’s cardiovascular health. The interaction of parent agnostic goal focus and their child’s emotion regulation ability was associated with the child’s resting diastolic blood pressure, resting systolic blood pressure, and increased presence of arterial stiffness. The interaction of parent agonistic goals and the child’s heart rate variability predicted the presence in the child of increased arterial stiffness. These findings demonstrate that, whereas a parent’s degree of agonistic goal focus may be a risk factor for higher blood pressure and the presence greater arterial stiffness in their child, the child’s cognitive/emotional and physiological ability to regulate stress responding may afford important protection. Simple slope analyses revealed that in children with well-developed cognitive emotion regulation capabilities, there was no association between the degree of a parent’s agonistic goals and their child’s resting systolic or diastolic blood pressure. However, as a child’s cognitive emotion regulation capabilities decreased, (e.g., moderate and low levels), the magnitude of the positive association between parent agonistic goals and resting systolic and diastolic blood pressure became increasingly larger. These patterns are consistent with previous findings in samples of adolescents and adults (Ewart et al., 2016; Ewart et al., 2011).

The findings for arterial stiffness (pulse wave velocity) also indicate that robust response modulation abilities (cognitive emotion regulation and tonic heart rate variability) may protect cardiovascular health. Here, the association between a parent’s degree of agonistic goal focus and the increased presence in their child of greater arterial stiffness was nonsignificant in the moderate and low groups of response modulation ability, but the slope was statistically significant and negative as the child’s response modulation abilities increased (see Figures 7 and 8). Overall, these results suggest that a high level of emotion regulation and physiological
autonomic self-regulation is protective. However, these protective effects may only be beneficial when a child’s response modulation capabilities and a parent’s agonistic goal focus are both high.

Post-hoc ancillary analyses were performed to evaluate the possibility that certain characteristics of the child, the parent, and the community environment might make parenting more difficult, and thereby contribute to ongoing power struggles and agonistic contests that could affect the child’s health. Indeed, characteristics of the child, parent, or environment that contributed to agonistic striving might better account for any observed associations between agonistic striving and child health outcomes. To evaluate these possibilities, in each post-hoc analysis a regression model that indicated a significant interaction between parent agonistic goal focus and child response modulation (cognitive emotion regulation, tonic heart rate variability) in predicting a child cardiovascular outcome was modified by including one of the child, parent, or environmental variables that could contribute to parent-child agonistic regulatory struggles. Variables evaluated in these regressions included child externalizing and autistic spectrum behaviors, parent perceived stress and depression symptoms, and the indices of the family’s SES and perceived neighborhood stress. The results disclosed that none of the significant interaction effects reported above were affected by any of the additional child, parent, or environmental covariates. This instills confidence in the present findings by suggesting that the significant interactions between parent agnostic goals and child response modulation in predicting cardiovascular disease risk in the child were not likely due to, or confounded, by the effects of the child, parent, or environmental variables examined. It is important to note that these post-hoc analyses do not alter the possibility that the influences examined here, or other similar
influences, may contribute to agonistic struggles in families even if they do not explain the findings reported here.

In addition to these interactions, three main effects emerged between parent agnostic goals and the child’s heart rate, diastolic blood pressure, and mean arterial pressure reactivity. Contrary to hypotheses, these main effects were negative in direction, indicating that parent agonistic goals were associated with less cardiovascular reactivity. One possible explanation for this effect is that children whose parents have higher agonistic goals also have higher resting levels of heart rate and blood pressure, and as such necessarily have a smaller potential for cardiovascular reactivity because it is calculated by subtracting the resting level from mean level during a stressor. Having a higher resting level implies that this difference score will be smaller even with the same mean cardiovascular response to the stress tasks. Furthermore, laboratory and medical settings may serve as stressors themselves, raising resting blood pressure and heart rate (Lurbe & Redon, 2004). Hence, parents who exhibit agonistic striving may become more hypervigilant in the unfamiliar, evaluative laboratory setting, resulting in higher resting heart rate and blood pressure in their children.

The present study design has some important limitations. One limitation is the use of laboratory blood pressure assessment on a single occasion, rather than ambulatory blood pressure assessment over several days. By indicating the level and variations of blood pressure throughout the course of one’s normal daily activities, ambulatory blood pressure readings more accurately reflect prevailing blood pressure levels, and thus afford a more reliable index of hypertension risk than do “casual” blood pressure readings obtained in a clinic setting, regardless of age (Lurbe & Redon, 2004). The present study includes a resting blood pressure index that is the average of blood pressure over three minutes, as well as blood pressure change that provides an
index of subjects’ cardiovascular reactivity to three stressors, helping to overcome some of the limitations of casual blood pressure readings. Furthermore, the assessment of structural cardiovascular disease risk indices (left ventricular mass, pulse wave velocity) expanded the cardiovascular risk indices measured, and accordingly extended the implications that can be drawn.

Another limitation is that these analyses used data from the first 100 adults and 100 children who completed the Social Competence Interview. Several parent and child participants did not complete the interviews. While some of this missing data is random (e.g., no interviewer was available), other missing data may not be random, such as some children’s inability to complete the interview when they became too emotional. It is unknown whether excessive emotionality during the interview is systematically linked to the data in a way that would bias or limit the present results. Furthermore, the study only allowed an examination of how one parent/guardian’s agonistic striving affected their child’s cardiovascular risk. Hence, the present study cannot afford insights into the dynamics of implicit parent motives and child cardiovascular risk within triadic groups (e.g., between two parents/guardians and a child) that may more accurately represent a child’s daily experiences and interactions at home.

Finally, the present findings may be limited by the sample size. Previous studies testing social action theory hypotheses about implicit motive profiles have relied on larger samples of adolescents and adults. Nonetheless, the power analyses suggested that the current sample size should afford adequate statistical power to detect the hypothesized differences and effects, supporting confidence in the present results.

These findings yield important implications for the understanding of cardiovascular health risk in children as well as important clinical implications, especially given that both
agonistic striving and emotion regulation are thought to be modifiable. This is suggested by previous research within social action theory that finds that goals are moderately temporally stable (Ewart et al., 2002) as well as research that suggests that children’s emotion regulation can be enhanced via psychotherapy (Hannesdottir & Ollendick, 2007). Information from the present findings could influence both parent and child cognitive behavior therapy-type interventions. Parent interventions could reflect on the protective nature of transcendence goals both for their health (as previous research has indicated) and for their children’s health by helping parents modify their goals. Interventions for the child can focus on strengthening emotion regulation abilities with psychoeducation, individual skills training in attention deployment and re-framing emotional experiences, and parent-level interventions that provide psychoeducation and behavioral parent training (Hannesdottir & Ollendick, 2007). Such tailored interventions may hold promise for improving both psychological and physical health.
Table 1. Means, Standard Deviations, and Bivariate Correlations among the Study Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>$M$</th>
<th>$SD$</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Child Agonistic Goals</td>
<td>2.03</td>
<td>0.65</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Parent Agonistic Goals</td>
<td>1.68</td>
<td>0.66</td>
<td>.17</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Emotion Regulation (Shift)</td>
<td>2.74</td>
<td>0.63</td>
<td>.05</td>
<td>.16</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Heart Rate Variability</td>
<td>7.77</td>
<td>1.22</td>
<td>.01</td>
<td>-.02</td>
<td>-.05</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Resting Heart Rate</td>
<td>82.21</td>
<td>9.92</td>
<td>-.01</td>
<td>.22</td>
<td>-.07</td>
<td>-.32</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Resting Systolic Blood Pressure</td>
<td>116.92</td>
<td>13.11</td>
<td>.05</td>
<td>.11</td>
<td>-.02</td>
<td>-.02</td>
<td>-.11</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Resting Diastolic Blood Pressure</td>
<td>61.86</td>
<td>9.29</td>
<td>.06</td>
<td>.19</td>
<td>.16</td>
<td>-.09</td>
<td>.01</td>
<td>.77</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Resting Total Peripheral Resistance</td>
<td>1226.32</td>
<td>385.12</td>
<td>.15</td>
<td>.05</td>
<td>.11</td>
<td>-.09</td>
<td>-.12</td>
<td>.45</td>
<td>.46</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Heart Rate Reactivity</td>
<td>0.03</td>
<td>4.43</td>
<td>-.14</td>
<td>-.22</td>
<td>.06</td>
<td>.00</td>
<td>.46</td>
<td>-.10</td>
<td>-.06</td>
<td>-.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Systolic Reactivity</td>
<td>3.53</td>
<td>6.58</td>
<td>-.01</td>
<td>-.05</td>
<td>-.08</td>
<td>-.08</td>
<td>.16</td>
<td>-.34</td>
<td>-.24</td>
<td>-.19</td>
<td>.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Diastolic Reactivity</td>
<td>2.67</td>
<td>3.70</td>
<td>-.13</td>
<td>-.32</td>
<td>-.18</td>
<td>-.14</td>
<td>.03</td>
<td>-.30</td>
<td>-.51</td>
<td>-.13</td>
<td>.13</td>
<td>.49</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Total Peripheral Resistance Reactivity</td>
<td>70.19</td>
<td>113.89</td>
<td>-.08</td>
<td>-.20</td>
<td>-.18</td>
<td>.09</td>
<td>.07</td>
<td>-.19</td>
<td>-.27</td>
<td>-.26</td>
<td>.12</td>
<td>.52</td>
<td>.60</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Mean Arterial Pressure Reactivity</td>
<td>2.84</td>
<td>4.61</td>
<td>-.09</td>
<td>-.26</td>
<td>-.18</td>
<td>-.15</td>
<td>.09</td>
<td>-.37</td>
<td>-.47</td>
<td>-.24</td>
<td>.11</td>
<td>.81</td>
<td>.89</td>
<td>.67</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Left Ventricular Mass</td>
<td>61.90</td>
<td>13.48</td>
<td>.01</td>
<td>-.15</td>
<td>.08</td>
<td>-.02</td>
<td>-.22</td>
<td>.17</td>
<td>-.02</td>
<td>-.11</td>
<td>.14</td>
<td>.07</td>
<td>.17</td>
<td>.32</td>
<td>.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. Pulse Wave Velocity</td>
<td>4.44</td>
<td>0.73</td>
<td>-.20</td>
<td>-.11</td>
<td>-.12</td>
<td>-.11</td>
<td>.11</td>
<td>.05</td>
<td>-.04</td>
<td>-.04</td>
<td>.02</td>
<td>.07</td>
<td>.14</td>
<td>.10</td>
<td>.14</td>
<td>.21</td>
<td></td>
</tr>
</tbody>
</table>

*Note: N = 100 parents and 100 children. All variables except Parent Agonistic Goals are from the child. Significant correlation coefficients at p < .05 are bolded.*
Figure 1. Response modulation moderates the effect of agonistic striving on Cardiovascular Disease (CVD) risk.
Figure 2. Three motive profiles (Agonistic Striving \((N = 28)\), Transcendent Striving \((N = 36)\), and Dissipated Striving \((N = 36)\)) generated by cluster analyses of \(T\)-scores on the Social Competence Interview for Expressiveness (EX), Agonistic Goals (AG), and Transcendent Goals (TG) scales in the present study of Syracuse adults (above) and in an earlier study of Baltimore adults (below; Ewart et al., 2016).
Figure 3. Three motive profiles (Agonistic Striving ($N = 55$), Transcendent Striving ($N = 14$), and Dissipated Striving ($N = 31$)) generated by cluster analyses of $T$-scores on the Social Competence Interview for Expressiveness (EX), Agonistic Goals (AG), and Transcendent Goals (TG) scales in the present study of Syracuse children (above) and in two earlier studies of Baltimore and Syracuse adolescents (below; Ewart et al., 2011).
Figure 4. Differences in child resting systolic blood pressure (SBP) by parent motive profile (TS = Transcendent Striving; DS = Dissipated Striving; AS = Agonistic Striving).
Figure 5. Children’s cognitive emotion regulation moderates the association between their parents’ agonistic goals and the children’s resting diastolic blood pressure (DBP). The Low slope represents the children whose emotion regulation (Shift) score was equal to or less than 1 standard deviation (SD) below the mean. The Moderate slope represents the children whose emotion regulation score was above -1 SD and below +1 SD. The High slope represents children whose emotion regulation score was 1 SD above the mean or greater.
Figure 6. Children’s cognitive emotion regulation moderates the association between their parents’ agonistic goals and the children’s resting systolic blood pressure (SBP). The Low slope represents the children whose emotion regulation (Shift) score was equal to or less than 1 standard deviation (SD) below the mean. The Moderate slope represents the children whose emotion regulation score was above -1 SD and below +1 SD. The High slope represents children whose emotion regulation score was 1 SD above the mean or greater.
Figure 7. Children’s cognitive emotion regulation moderates the association between their parents’ agonistic goals and the presence of arterial stiffness in the child indexed by pulse wave velocity. The Low slope represents the children whose emotion regulation (Shift) score was equal to or less than 1 standard deviation (SD) below the mean. The Moderate slope represents the children whose emotion regulation score was above -1 SD and below +1 SD. The High slope represents children whose emotion regulation score was 1 SD above the mean or greater.
Figure 8. Children’s tonic heart rate variability moderates the association between their parents’ agonistic goals and the presence of arterial stiffness in the child indexed by pulse wave velocity. The Low slope represents the children whose heart rate variability was equal to or less than 1 standard deviation (SD) below the mean. The Moderate slope represents the children whose heart rate variability was above -1 SD and below +1 SD. The High slope represents children whose heart rate variability was 1 SD above the mean or greater.
Appendix A
Social Competence Interview – Observer Rating Scales
Expressiveness Scale

1 = Not at all; 3 = Moderately; 5 = Very much

Is poised, at ease, self-assured . . . . 1 2 3 4 5
Speaks emphatically . . . . . . . . . 1 2 3 4 5
Gives detailed responses . . . . . . . 1 2 3 4 5
Speaks loudly . . . . . . . . . . . . . 1 2 3 4 5
Gives short, monosyllabic responses . . 1 2 3 4 5
Voice (inflection, tone, quality) easily expresses emotion . . 1 2 3 4 5
Speech is slow and halting . . . . . . 1 2 3 4 5
Speaks rapidly . . . . . . . . . . . . . 1 2 3 4 5
Speaks very softly . . . . . . . . . . . 1 2 3 4 5
Is open, easy to get to know . . . . . 1 2 3 4 5
Is likeable . . . . . . . . . . . . . . . . . 1 2 3 4 5
Appendix B
Social Competence Interview – Observer Rating Scales
Self-Defense and Acceptance-Affiliation Scales

1 = Not at all; 3 = Moderately; 5 = Very much

**Self-Defense:** To what extent was he/she:

Wanting someone to stop being critical of
him / her? . . . . . . . . . . . . . 1 2 3 4 5

Wanting someone to stop being
demanding of him / her? . . . . . . . 1 2 3 4 5

Wanting to get even with someone,
to get revenge? . . . . . . . . . . . . 1 2 3 4 5

Wanting someone to stop doing
or saying mean, hurtful or annoying
things? . . . . . . . . . . . 1 2 3 4 5

Striving to protect or defend oneself
(e.g., trying to correct an unfair situation,
stop hostile criticism/ rumors/ abuse, get
even with someone?) . . . . . . . . . . 1 2 3 4 5

**Acceptance-Affiliation:** To what extent was he/she:

Wanting someone to like her / him? . . 1 2 3 4 5
Wanting someone to show they understand, to sympathize? . . . . . 1 2 3 4 5

Wanting someone to stop ignoring or excluding her / him? . . . . . . . 1 2 3 4 5

Wanting to be closer to someone? . . . 1 2 3 4 5

Striving for affiliation (e.g., to get someone to appreciate her / his feelings or needs, achieve intimacy, become closer to someone, obtain sympathetic understanding / support)? 1 2 3 4 5
Appendix C
Social Competence Interview – Observer Rating Scales
Approval Seeking and Self-Improvement Scales

1 = Not at all; 3 = Moderately; 5 = Very much

**Approval Seeking**: To what extent was he/she:

Wanting to pursue an activity
(e.g., course, club, sport) just to
please someone else?  .   .   .   .   .   . 1  2  3  4  5

Wanting to avoid disappointing
an important individual? .................. 1  2  3  4  5

Wanting to accomplish a difficult
goal or task just to satisfy a
respected person?  .   .   .   . 1  2  3  4  5

Wanting to live up to someone else’s
high expectations?  .   .   .   . . . . 1  2  3  4  5

Striving to attain a difficult standard or goal
(e.g., high grade, make team) or engage in an
activity merely to avoid disappointing a
parent or other respected figure?  .   .   . .1  2  3  4  5

**Self-Improvement**: To what extent was he/she:
Wanting to achieve a self-standard
that’s important to him / her personally? 1 2 3 4 5

Wanting to develop a good behavior pattern
(study habits, diet, exercise, etc)? 1 2 3 4 5

Wanting to improve her / his skills in
a favorite/important activity (sport, music, school
subject, etc)? 1 2 3 4 5

Wanting to improve him/herself as a
person (to be nicer, smarter, healthier) 1 2 3 4 5

Striving for self-mastery, or for personal
achievement (e.g., attain a personally valued
goal, master a skill) because the achievement
is important personally—not just to satisfy
someone else? 1 2 3 4 5
Appendix D
Shift-and-Persist Questionnaire (SAPQ)
Shift Subscale

1 = not at all
2 = a little
3 = some
4 = a lot

Next you will see a list of things that people sometimes do, think, or feel when something stressful happens. Everybody deals with problems in their own way. Please rate how much you do each of the following things when something stressful happens in your life:

1. I think about what I can learn from the situation
2. I work to change the problem for better
3. I do something to calm myself down
4. I think about the positive aspects, or the good that can come from the situation
5. I try to think of different ways to change the problem or fix the situation
6. I tell myself that everything will be all right
7. I keep my feelings under control and only let them out when they won’t make things worse

In life, things don’t always go the way that we want. Everyone has different preferences for how they deal with situations in which something doesn’t turn out the way that they want, and they are not able to change it. Please rate how much you do each of the following things in that kind of situation:

8. I think about other new goals that I could pursue
9. I think about what good things could come from the situation
10. I tell myself that everything will be all right
11. I start working on other new goals
12. I think about what I can learn from the situation
References


56


disease: The problems and implications of overlapping affective dispositions.
*Psychological Bulletin, 131*(2), 260-300.

prefrontal neural function and cognitive performance: The neurovisceral integration
perspective on self-regulation, adaptation, and health. Annals of Behavioral Medicine,
37*(2)*, 141-153.

Thayer, J. & Lane, R. (2000). A model of neurovisceral integration in emotion regulation and

Correspondence between physiological and self-report measures of emotion
dysregulation: A longitudinal investigation of youth with and without

372-382.
**Curriculum Vitae**
Sarah R. LaFont

**Education**

SYRACUSE UNIVERSITY, Syracuse, NY

**M.S. Clinical Psychology**
Spring 2016

**Ph.D. Clinical Psychology (APA Accredited)**
Anticipated 2019

SUNY INSTITUTE OF TECHNOLOGY, Utica, NY

**B.A. Psychology & Community and Behavioral Health**
2013

Summa Cum Laude

**Clinical Experience**

SYRACUSE UNIVERSITY
Psychological Services Center, Syracuse, NY
January 2015- Present

**Therapist**

• Conduct intake, clinical, ADHD, and Learning Disorder assessments
• Provide interpretations, feedback, and recommendations on assessments
• Provide weekly psychotherapy to a caseload of 8 clients
• Receive supervision on cases from psychodynamic, interpersonal, humanistic, and cognitive-behavioral approaches to therapy
• Participate in didactic learning of CBT and DBT techniques
• Schedule weekly appointments in Titanium and maintain client billing
• Supervisors: Afton Kapuscinski, Ph.D., Kevin M. Antshel, PhD, Deborah Pollack, PhD, Amy Olszewski, PhD, Robbi T. Saletsky, PhD, Deborah Pollack, PhD, Jessica Costosa-Umina, PhD, Whitney Wood, PhD, Sarah Felver, PhD

SYRACUSE UNIVERSITY
Psychological Services Center, Syracuse, NY

**Group-Co Facilitator, Social Skills Training group**

• The Social Skills Training group (SST) at Syracuse University is a 10-week CBT intervention that focuses on conversation skills and social problem solving skills for
children with autism spectrum disorders (ASDs) and common comorbid disorders.
- Co-facilitated weekly SST group therapy sessions.
- Supervisor: Kevin M. Antshel, PhD

UPSTATE CEREBRAL PALSY  
February 2012-August 2012

Community Health & Behavioral Services, Utica, NY

Undergraduate Intern / Volunteer

- Co-facilitated group therapies, including Dialectical Behavioral Therapy
- Created Powerpoint presentations and handouts on various types of therapies
- Completed paperwork, including attendance sheets, filling out the monthly mandatory Medicaid review paperwork, discharge & withdrawal paperwork
- Performed outreach tasks (phone calls and letters) and their involved paperwork
- Attended weekly staff meetings

Publications


Conference Presentations


**Research Experience**

SYRACUSE UNIVERSITY August 2013- Present

Falk College of Public Health, Syracuse, NY

**Graduate Research Assistant for the Environmental Exposures and Child Health Outcomes (EECHO) study**

Supervisors: Brooks B. Gump, PhD, MPH & Craig K. Ewart, PhD

- Environmental toxicants pose grave health risks for children and adults alike. The EECHO aims to examine the impact that such toxicants (i.e., lead) have on the health of black and white children ages 9 - 11 who live in Syracuse area zip codes.
- I conducted standardized, semi-structured qualitative interviews (the Social Competence Interview) with primary caregivers and their children. Interview topics included chronic stressors and coping strategies.
- I served as the project’s data manager from August 2014 to May 2015.

SYRACUSE UNIVERSITY August 2013- Present

Department of Psychology, Syracuse, NY

**Research Assistant for Project Heart**

Supervisors: Craig K. Ewart, PhD

- Cardiovascular disease remains the #1 cause of death in the United States. Through a series of longitudinal studies (Project Heart) spanning over 20 years, the Project Heart Lab has been studying determinants of CVD risk in low-income, largely minority populations.
- Currently we are examining relationships between biomarkers of health (e.g., interleukin-6, c-reactive protein, blood lipids) and development of metabolic syndrome and other indices of risk for CVD.
Undergraduate Research Assistant

Supervisors: Joanne Joseph, PhD & Veronica Tichenor, PhD

- Using data from the 2011 Oneida County Teen Assessment Project (TAP) survey, we examined Adverse Childhood Experiences (ACEs) as a risk factor for bullying behaviors. Our model also included social support scales (school, family, & community) as moderating variables.
- I presented our findings on 5/22/13 to the Oneida County Youth Council in New Hartford, NY.

Teaching Experience

SYRACUSE UNIVERSITY

Department of Psychology, Syracuse, NY

Instructor

Health Psychology PSY 382

This course explored the relationship between behavior and health. All topics were covered from a biopsychosocial perspective, illustrating the interaction among variables within an individual's environment, body, and behavior. Topics discussed within the course included: medical adherence, psycho-neuroimmunology, anger/hostility and health, weight control, eating and exercise, health care systems, heart disease, HIV/AIDS, stress, and complementary/alternative treatments.

SYRACUSE UNIVERSITY

Department of Psychology, Syracuse, NY

Teaching Assistant to Tibor Palfai, PhD

Foundations of Human Behavior, PSY 205

This position involved teaching four weekly recitation classes of approximately 20 students each, leading classroom lecture and discussion, and grading students assignments, quizzes, and papers.
Service

CENTRAL NEW YORK PSYCHOLOGICAL ASSOCIATION September 2015 – April 2016
Syracuse, NY

Worked with CNYPA President Dr. Deborah Pollack and other graduate students to develop CNYPA’s webpage, including researching and writing about various mental health careers and psychological disorders.

SYRACUSE UNIVERSITY September 2015 – May 2016
Department of Psychology, Syracuse, NY

Clinical Area Student Representative

- Conduct surveys of graduate students on important decisions/issues
- Attend monthly Clinical faculty meetings and vote on behalf of graduate students on agenda items
- Serve as liaison between Clinical faculty and graduate students
- Attend Psychology Action Committee meetings and update students from other areas on matters/business of the Clinical area

SYRACUSE UNIVERSITY September 2014– December 2014
Psychology Action Committee, Syracuse, NY

Mentor

Served as a mentor for an incoming first year student in the Syracuse University Clinical psychology PhD program

Professional Memberships

Psi Chi Honor Society April 2012 – Present
Future Professoriate Program August 2014 – Present
Women in Science & Engineering (WiSE) August 2014 – Present
American Psychosomatic Society March 2014 – Present
American Psychological Association – Division 38 (Health Psychology) June 2014 – Present
Central New York Psychological Association December 2015 – Present
Society of Behavioral Medicine March 2016 – Present