

The Mediating Role of Executive Functioning in the Relation Between Difficult Temperament and Physical Aggression

Peter R. Giancola,^{1,4} Robert M. Roth,² and Dominic J. Parrott³

Accepted October 3, 2005

Published online: 11 July 2006

The present investigation tested the hypothesis that executive functioning (EF) would mediate the relation between difficult temperament (DT) and aggressive behavior. This model was tested in 310 adult men and women. DT was measured using the *Dimensions of Temperament Scale—Revised*, EF was measured using 7 well-established neuropsychological tests, and aggression was assessed using the *Buss–Perry Aggression Questionnaire*. EF successfully mediated the DT–aggression relation for men, however, the model did not hold for women. Results are discussed with regard to how they influence current models of aggressive behavior as well as their implications for future violence prevention efforts.

KEY WORDS: physical aggression; temperament; executive functioning; risk factors.

Deviations in temperament are related to aggressive and antisocial behavior in children and adults (Tarter, 1988; Tarter & Vanyukov, 1994). Temperament can be defined as a latent construct comprising a series of trait dimensions depicting individual differences in various types of behavioral and affective response and self-regulatory styles (Rothbart, 1989; Rothbart & Ahadi, 1994; Thomas & Chess, 1977). Examples of such dimensions include the degree of regularity in performing various behavioral activities and biological functions, the tendency to respond with an approach or an avoidance style to novel situations, a predisposition to experience positive or negative affect, and the degree of flexibility and adaptability in reacting to unfamiliar situations and environments (Thomas & Chess, 1977). The term difficult temperament (DT) reflects behaviors and affective states characterized by withdrawal from novel stimuli, intense reactions to stimuli, low adaptability to change, irritability, negative mood, distractibility, irregularities in biological functions, as well as

poor attention and persistence (Tarter & Vanyukov, 1994; Thomas & Chess, 1984; Windle, 1991).

DT is linked with a greater degree of behavior problems, aggression, and delinquency in young children (Jansen, Fitzgerald, Ham, & Zucker, 1995; Kingston & Prior, 1995) and adolescents (Fox & Calkins, 1993; Giancola, Mezzich, & Tarter, 1998; Sanson & Prior, 1999; Tarter, Blackson, Martin, Loeber, & Moss, 1993; Windle, 1992a). It is also related to greater levels of violence and attempted suicide as well as a hostile interpersonal style in adult men (Engstrom, Persson, & Levander, 1999; Patrick, 1994; Windle, 1994). DT, measured in 3- to 5-year-old boys, has also been shown to predict convictions for violent offenses at age 18 (Henry, Caspi, Moffitt, & Silva, 1996) and a recent study determined that DT was a risk factor for intoxicated aggression for men but not for women (Giancola, 2004a).

There is a wealth of data demonstrating that poor cognitive functioning, specifically, executive functioning (EF), is also a risk factor for aggressive behavior (reviewed in Fishbein, 2000; Hawkins & Trobst, 2000; Morgan & Lilienfeld, 2000; Paschall & Fishbein, 2002; Stevens, Kaplan, & Hesselbrock, 2003). EF is defined as a higher-order cognitive construct involved in the planning, initiation, and self-regulation of goal-directed behavior (Luria, 1980; Milner, 1995; Stuss & Alexander, 2000; Tranel, Anderson, & Benton, 1994). The cognitive abilities

¹Department of Psychology, University of Kentucky, Lexington, Kentucky.

²Department of Psychiatry, University of Dartmouth Medical School, Lebanon, New Hampshire.

³Department of Psychology, Georgia State University, Atlanta, Georgia.

⁴To whom correspondence should be addressed at Department of Psychology, University of Kentucky, Kastle Hall, Lexington, Kentucky 40506-0044; e-mail: peter@uky.edu.

subsumed within this construct include attentional control, strategic goal planning, abstract reasoning, set shifting, temporal response sequencing, previewing ability, cognitive flexibility, self-monitoring, inhibition, hypothesis generation, and the ability to organize and adaptively utilize information contained in working memory (Kimberg, D'Esposito, & Farah, 1998; Stuss & Alexander, 2000). When considered in a more functional context, it becomes clear that the ability to correctly appraise a problematic situation, determine a plan of action to adaptively cope with that situation, and then adroitly carry out that plan, making appropriate changes when necessary, is heavily dependent upon possessing good EF (Damasio, 1994; Stuss & Alexander, 2000).

A large literature indicates that EF contributes, in part, to the expression of aggressive and violent behavior (reviewed in Fishbein, 2000; Hawkins & Trobst, 2000; Paschall & Fishbein, 2002; Stevens et al., 2003). In fact, a recent meta-analytic study reported that the effect size of EF on antisocial behavior is in the "medium" to "large" range (Morgan & Lilienfeld, 2000). More specifically, studies with clinical samples indicate that adult (Lapierre, Braun, & Hodgins, 1995; Smith, Arnett, & Newman, 1992) and adolescent psychopaths (Roussy & Toupin, 2000), adolescent sex offenders (Kelly, Richardson, Hunter, & Knapp, 2002), adults with antisocial personality disorder (Gorenstein, 1987; Malloy, Noel, Longabaugh, & Beattie, 1990), and adolescents with conduct disorder (Moffitt, 1993; Moffitt & Henry, 1989), all exhibit poorer performance on neuropsychological measures of EF compared with controls. The results of more recent studies further document a negative relation between EF and aggression. Notably, EF has been shown to be negatively associated with verbal aggression in adolescents (Santor, Ingram, & Kusumakar, 2003), fighting in normal preadolescent boys (Seguin, Pihl, Harden, Tremblay, & Boulerice, 1995), impulsive aggression in college students (Villemarette-Pittman, Stanford, & Greve, 2002), violent and nonviolent conduct disorder symptoms in adolescent girls (Giancola et al., 1998), as well as physical aggression measured in a laboratory setting (Giancola & Zeichner, 1994; Hoaken, Shaughnessy, & Pihl, 2003; Lau & Pihl, 1996).

The prefrontal cortex and aspects of its striatal and limbic connections are thought to be the primary neuroanatomical substrates that subservise EF (Cabeza & Nyberg, 2000; Fuster, 1997; Luria, 1980; Milner & Petrides, 1984; Stuss & Levine, 2002). Acquired focal lesions to the prefrontal cortex can manifest overtly as cognitive rigidity, impulsivity, and disorganized, disinhibited, and at times aggressive behavior in both humans and primates (Blair, 2004; Brower & Price, 2001;

Chow, 2000; Kalin, 1999; Kim, Choi, Kwon, & Seo, 2002; Miller & Cummings, 1999; Seguin, 2004; Stuss, Gow, & Hetherington, 1992). In fact, a recent study using positron emission tomography found reduced blood flow in the orbital region of the prefrontal cortex in healthy young adults who were imagining scenarios involving aggressive behavior (Pietrini, Guazzelli, Basso, Jaffe, & Grafman, 2000). From a functional perspective, it has been theorized that impaired EF facilitates aggressive behavior due to decreased behavioral inhibition, increased cognitive rigidity, and deficiencies in generating alternative nonaggressive socially appropriate responses in provocative situations (Giancola, 1995). However, it is important to note that low EF or damage to the prefrontal cortex does not inevitably lead to aggressive behavior in all cases. Although such individuals can be antisocial by virtue of violating societal norms in some instances, the majority of persons with damage to the prefrontal cortex are not necessarily physically violent (Benson & Miller, 2000; A. James, S. James, Smith, & Javaloyes, 2004; Osmon, 1996; Taylor et al., 2004).

The prefrontal cortex also plays a significant role in expression and regulation of different aspects of temperament (Beer, Heerey, Keltner, Scabini, & Knight, 2003; Damasio, 1995; Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Heller, 1993; Kolb & Taylor, 1981; Tarter, Alterman, & Edwards, 1985). For example, the prefrontal cortex has a multitude of direct and indirect reciprocal connections with various subcortical regions involved in processing affective information such as the amygdala (Barbas, 2000; Davidson, 2002; Phan et al., 2003). These connections permit some degree of "top-down" control over emotional experiences (e.g., Beauregard, Levesque, & Bourgoin, 2001; Jackson et al., 2003). Developmental studies in children have also reported significant relations between asymmetries of frontal electroencephalographic activations and temperament characteristics such as hostility and reactivity to novelty (Dawson et al., 1999; McManis, Kagan, Snidman, & Woodward, 2002). These findings strongly indicate that the cognitive functions subserved by prefrontal circuitry directly influence individual differences in temperament. This contention is supported by empirical evidence indicating that EF deficits are related to DT in children and adults (reviewed in Giancola, 1995; Tarter, 1988; Tarter & Vanyukov, 1994) and by clinical data showing that individuals who have sustained damage to the prefrontal cortex display a behavioral profile resembling that of non-brain damaged individuals with DT (Starkstein & Robinson, 1991; Stuss et al., 1992). Given this, one should not be surprised that alterations in mood, increased irritability, and emotional as well as behavioral dysregulation have been observed

subsequent to damage to the prefrontal cortex (Blair, 2004; Seguin, 2004; Stuss et al., 1992).

On the basis of the preceding review, one could argue that EF is involved in the regulation of temperament. The Russian neuropsychologist Luria (1961, 1980) first advanced this hypothesis by concluding that the cognitive regulation of affect and behavior is governed predominantly by the prefrontal cortex that, as noted above, is the primary neural substrate for EF. Following Luria, Tarter and colleagues (Tarter, 1988; Tarter et al., 1985) put forth a similar theoretical stance that has its foundation in an extensive body of empirical work aimed at delineating the neurobehavioral underpinnings of alcoholism. Within his model, Tarter made it clear that the overt manifestations of DT (e.g., overactivity, poor soothability, low sociability) were regulated by EF. Finally, this hypothesis was further elaborated upon by Moffitt (1993) who postulated that neuropsychological disturbances in EF can underlie behavioral and affective manifestations of DT such as irritability, emotional dysregulation, and poor impulse control. Collectively, these theorists all seem to conclude that EF plays an important role in regulating temperament.

The theoretical formulations and empirical data reviewed above can be summarized as follows: (a) deviations in temperament are related to aggressive behavior, (b) EF is related to aggressive behavior, and (c) EF is involved in the regulation of temperament. Therefore, the purpose of this study was to test the hypothesis that EF plays a mechanistic role underlying the relation between DT and aggression. This hypothesis was originally theorized by Moffitt (1993) who argued that neuropsychological deficits, particularly deficits in EF, can underlie a dysregulation of temperament, which can then predispose toward aggressive behavior. Only one published study has tested this model. Giancola et al. (1998) found that EF mediated the relation between DT and physical aggression in a sample of adolescent girls with comorbid diagnoses of conduct disorder and substance use disorder. Insofar as this is the only study that has tested this model and given that it was conducted on a sample of adolescents with psychiatric diagnoses, further research is required in order to better determine whether EF underlies the relation between DT and aggression. As such, the present investigation examined the mediating effects of EF on the DT–aggression relation in a large sample of normal adult men and women. DT was measured using a validated self-report instrument, EF was measured using seven well-established neuropsychological tests, and aggression was assessed using a self-report inventory that inquired about previous acts of physical aggression.

METHOD

Participants

Participants were 310 men ($n = 152$) and women ($n = 158$) between 21 and 35 years of age ($M = 23.03$; $SD = 2.85$). Participants were recruited through advertisements placed in various newspapers in Lexington, KY. Respondents were initially screened by telephone. Individuals reporting any past or present drug- or alcohol-related problems, serious head injuries, learning disabilities, or serious psychiatric symptomatology were excluded from participation. The sample consisted of 286 Caucasians, 23 African Americans, and 1 Hispanic. Eighty-nine percent of the participants were never married, 31.3% had a high-school degree and were not pursuing further education, 43.9% had a high-school degree and were working on a bachelor's or an associate's degree, 21.6% had a bachelor's or an associate's degree, 1.9% had a graduate degree, and 1.3% did not graduate from high-school. Forty-eight percent of the sample supported themselves financially and earned approximately \$18,500 per year; the remainder were supported by a parent or by a spouse. Participants received \$50 at the completion of the study as compensation.

Neuropsychological Assessment

Selection of the EF test battery was guided by functional and neuroanatomical considerations according to guidelines put forth by Diamond (1991). Specifically, from a functional perspective, tests of EF were chosen to reflect a wide variety of skills encompassed by this construct such as attentional control, previewing ability, strategic goal planning, abstract reasoning, cognitive flexibility (set shifting), hypothesis generation, inhibition, and the ability to organize and adaptively utilize information contained in working memory. From a neuroanatomical perspective, tests of EF were selected on the basis of being generally accepted as measures of functions that are subserved primarily by the prefrontal cortex. The prefrontal cortex and its subcortical circuits are thought to be the primary neurological substrates that subserve EF (Fuster, 1995; Luria, 1980; Stuss & Alexander, 2000). There are extensive neuroimaging data with normals and ample neuropsychological evidence from patients with acquired brain lesions demonstrating that, although some generalized cerebrocortical patterns of activation have been noted, the EF tests selected for this investigation primarily assess prefrontal cortical functions (e.g., Casey et al., 1997; Demakis, 2003; Goel & Grafman, 1995; Karnath, Wallech, & Zimmermann, 1991; Petrides, Alivisatos,

Evans, & Meyer, 1993; Rezai et al., 1993; Sasaki, Gemba, Nambu, & Matsuzaki, 1993; Stuss, Floden, Alexander, & Katz, 2001).

Porteus Maze Test

Participants were required to navigate their way through eight mazes (Porteus, 1965). They were instructed to not lift their pencil from the paper until each maze was completed. The Impulsive Errors score (i.e., Qualitative Score), was used to index EF (Porteus, 1965). This type of error reflects a lack of foresight, poor judgment, difficulty learning from experience, as well as poor planning and organizational abilities (Crown, 1952; Porteus & Kepner, 1944).

Go/No-Go Task

Participants completed a computerized version of this task (Newman & Kosson, 1986). Participants were informed that a series of numbers were going to be presented, one at a time, in the center of a computer screen. They were told that they had an opportunity to win money on the basis of their performance on the task. Participants were informed that each time a number appeared on the screen they had to choose whether or not they were going to press the spacebar on the keyboard and that their choice would result in either winning or losing money. They were given no further instructions. Prior to beginning the task, \$5.00 in quarters was placed on the table in front of the participant. The experimenter kept a large stack of quarters on his/her side of the table. Each time the participant won or lost a trial, the experimenter would respectively give or take away a quarter from the participant. Participants did not win or lose money if they made no response at all.

The task had a total of 85 trials. A total of 10 numbers were used. Five numbers were “winners” (37, 96, 78, 53, 29) and five were “losers” (43, 82, 64, 73, 31). The numbers were presented on the computer screen for 2 s with an intertrial interval of 1 s. The first five trials were all winning numbers (to establish a dominant response set) and the remainder of the trials were randomly ordered with no consecutive win or lose sequence exceeding three trials. Participants had to learn, by trial and error, when to respond and when not to respond. Trials were presented in eight continuous blocks of 10, excluding the first five. EF was indexed by the total errors of commission (i.e., pressing the spacebar when incorrect) for the last 40 trials of the task. Such errors reflect an inability to inhibit incorrect responding under circumstances involving sustained attention (Newman & Kosson, 1986).

Trails B of the Trail Making Test

Participants were given a sheet of paper randomly arranged with the numbers “1” through “13” and the letters “A” through “L.” They were told that they had to connect the numbers and letters in an alternating sequence (e.g., 1-A-2-B-3-C. . .) as quickly as possible using a pencil. If an error was made, the experimenter quickly informed the participant so that it could be corrected. Performance on this task was measured by the amount of time taken to complete all of the connections (Reitan, 1992). Success on this task requires good cognitive flexibility and set shifting skills in order to quickly and repeatedly alternate between two different tasks.

Stroop Task

Participants were presented with three stimulus cards. For the first card, they were instructed to read a list of words (red, blue, green, yellow) printed in black ink as quickly as possible. For the second card, they were asked to name the color (red, blue, green, yellow) in which a series of “X”s were printed as quickly as possible. These first two parts of the task respectively measure verbal and nonverbal perceptual processing speed. For the third card, participants reported the color of the ink in which words were printed as quickly as possible; however, the word names were incongruent with the colors in which they were printed. EF was indexed by the “interference score,” derived by subtracting the response time of the second portion of the task (color naming) from the response time of the third portion of the task (incongruent color-word naming; MacLeod, 1991). Poor performance on this task reflects an inability to inhibit the effects of a distracting stimulus as well as poor attentional skills (MacLeod, 1991; Perret, 1974).

Conditional Associative Learning Test

Seven black squares (1 in. × 1 in.) were printed on a laminated 3 in. × 11 in. card and placed before the participant. Seven small lights were fixed, in a random arrangement, onto a 10 in. × 8 in. metal box that was placed anterior to the card. Participants were asked to learn the manner in which the squares and the lights were associated. The experimenter illuminated the lights in a fixed random order and the participant’s task was to point to the square that s/he believed was associated with the particular illuminated light. Participants learned the associations by trial and error based on feedback from

the experimenter. The task ended when 17 consecutive correct responses were achieved or when 210 trials were exhausted. Performance was indexed by the number of errors committed. This test measures the ability to organize and utilize information contained in working memory (Petrides, 1985).

Tower of Hanoi

Participants were presented with a wooden platform mounted with three vertical rods. Five rings (differing in circumference) were stacked on the left-most rod (smaller rings were always stacked on top of a larger ring). Participants were instructed to reproduce the same stacking configuration of rings on the right-most rod by moving the rings according to the following three rules: (1) Only one ring can be moved at a time; (2) a larger ring cannot be placed on a smaller ring; and (3) unless actively being moved, no ring can be removed from a rod. Three trials were conducted. The first involved four rings and the second and third involved all five. Performance was indexed by the number of moves taken to complete the first and third trials (Goel & Grafman, 1995). Scores from the second trial were not used because the test was so difficult that most participants could not solve the problem. This task measures strategic planning and the organization and use of information contained in working memory reflected as the ability to sequentially order a series of responses to achieve a particular goal (Goel & Grafman, 1995).

Wisconsin Card Sorting Test

A computerized version of this task was administered (Heaton, 1993). Participants were presented with four sample “cards” at the top of the screen. The cards depicted between one and four stimulus shapes (i.e., circle, triangle, cross, and square) that were printed in one of four colors (i.e., red, blue, yellow, and green). At the bottom of the screen was a “deck” of 128 cards each printed with different combinations of these shapes and colors. Participants were asked to match each card from the deck to one of the sample cards. The cards could be matched according to their similarity in color, shape, or number of stimuli. However, participants were not informed of the matching principles. Each time 10 consecutive correct matches were achieved, the computer changed the matching principle without notifying the participants. The test proceeded until six sorting categories were completed or until all 128 cards were used. Performance on this task was indexed by the number of errors committed. Success on this task requires the ability to abandon a previous

sorting principle and then generate and test new hypotheses about other solutions thus capitalizing on cognitive flexibility and set shifting skills.

All tests were administered according to standard procedures. In accordance with the results of a previously published confirmatory factor analysis on these data (Giancola, 2004b), scores from the neuropsychological tests were z-transformed and then summed to create an *executive functioning* variable. Higher scores indicate better EF.

Temperament

Temperament was assessed using the *Dimensions of Temperament Survey—Revised* (DOTS-R; Windle & Lerner, 1986), upon which participants rated themselves on a 4-point Likert scale ranging from “usually false” to “usually true.” The DOTS-R is a 54-item self-report inventory assessing various aspects of temperament (Windle & Lerner, 1986). The DOTS-R comprises 10 scales: (1) Activity Level—General (energy and motor activity); (2) Activity Level—Sleep (motor sleep activity); (3) Approach—Withdrawal (an approach or withdrawal style to new objects and persons); (4) Flexibility—Rigidity (degree of adaptability to changes in the environment); (5) Mood (quality of mood); (6) Rhythmicity—Sleep (regularity in sleep behavior); (7) Rhythmicity—Eating (regularity in eating behavior); (8) Rhythmicity—Daily Habits (regularity in performing daily habits); (9) Distractibility (attention and distractibility); and (10) Persistence (persistence in performing tasks). It is important to note that none of the DOTS-R items assessed aggression in any way.

A difficult temperament index was created by summing all of the subscale scores. Scoring was adjusted so that lower scores reflect a more difficult temperament. The 10 subscales of the DOTS-R possess moderate to high levels of internal consistency (Cronbach’s α range: .62–.89) and good 6-week test–retest reliability (range: .59–.75) in adolescents (Windle & Lerner, 1986). The 10 subscales also possess good convergent and discriminant validity (Windle, 1992b). The total temperament score also possesses high levels of internal consistency in both children ($\alpha = .83$) and adults ($\alpha = .89$; Blackson, Tarter, Loeber, Ammerman, & Windle, 1996).

Physical Aggression

The *Buss–Perry Aggression Questionnaire* (BPAQ; Buss & Perry, 1992) was used to measure aggression.

Table I. Gender differences

Measure	Men		Women	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age (years)	23.34	3.07	22.72	2.58
Years of education	15.92	2.02	16.31	2.00
Salary (\$)	18.86K ^a	12.72K	17.80K	9.80K
Executive functioning				
Porteus maze	14.09	10.61	16.16	12.93
Go/No-Go	5.37	4.42	6.38	4.94
Trails B	57.14	15.44	55.77	14.23
Stroop	36.50	13.39	38.49	14.11
Conditional associative learning	53.21	43.53	52.85	38.57
Tower of hanoi	58.70	18.95	59.69	17.04
Wisconsin card sorting	20.75	15.74	18.35	11.85
Executive functioning total score ^b	0.00	1.00	-0.01	1.00
Difficult temperament				
General activity	15.52	4.68	14.55	4.84
Sleep activity	9.25	3.59	8.88	3.92
Approach-withdrawal	20.55	3.32	20.97	3.56
Flexibility-rigidity	14.93	2.56	14.72	3.12
Mood	24.98	3.56	25.61	3.25
Sleep rhythmicity	13.35	4.29	13.84	4.55
Eating rhythmicity	12.93	4.10	13.13	3.94
Daily habits rhythmicity	11.58	3.09	11.88	3.44
Distractibility	12.64	2.93	11.37	3.05
Persistence	8.80	1.67	8.45	1.72*
Difficult temperament total score	144.52	16.51	143.41	17.36
BPAQ physical aggression	21.47	6.84	15.87	5.67*

Note. BPAQ: Buss-Perry Aggression Questionnaire.

^aK = \$1,000.

^bData represented as *z* scores.

**p* < .05.

The BPAQ is a well-known 29-item inventory with four subscales (i.e., Physical Aggression, Verbal Aggression, Anger, and Hostility). The BPAQ is scored on a 5-point Likert scale and has been shown to have excellent psychometric properties (Buss & Perry, 1992; Tremblay & Ewart, 2005; Williams, Boyd, Cascardi, & Poythress, 1996). Given the nature of the present study, aggression was indexed by participants' scores on the Physical Aggression subscale.

Procedure

Upon arrival to the laboratory, all participants provided informed consent and demographic data were collected. Participants then completed seven neuropsychological tests measuring EF. The seven tests were administered in a fixed order across participants and took approximately 1.5 hr to complete. Participants were given a 10-min break after the first hour of testing. After completing the neuropsychological tests, they completed the

DOTS-R and BPAQ. Participants were then debriefed, compensated, and thanked.

RESULTS

Gender Differences

Gender differences were examined using *t* tests. Results indicated that men and women did not differ significantly with respect to age, years of education, salary, EF, or DT. However, as would be expected, men reported greater physical aggression than did women (see Table I).

Mediation Analyses

The aim of this study was to determine whether EF mediates the relation between DT and aggression. Baron and Kenny (1986) have argued that mediation can be tested by regressing (a) the proposed mediator (EF) on

the independent variable (DT), (b) the dependent variable (aggression) on the independent variable, and (c) the dependent variable on both the mediator and the independent variable. They noted that mediation is present if (a) the relations in the first two equations are significant, (b) the mediator is significantly related to the dependent variable in the third equation, (c) and the influence of the independent variable on the dependent variable is substantially reduced following the inclusion of the mediator in the model. These tests were carried out separately for men and for women.

The conditions described above were satisfied for men by regressing (a) EF onto DT ($\beta = -.15, p < .05$); (b) aggression onto DT ($\beta = -.17, p < .05$); and (c) aggression onto EF and DT (β for DT = $-.12, p = ns$; β for EF = $.31, p < .001$). As can be seen, adding EF to the equation reduced the relation between DT and aggression by 30% and rendered it nonsignificant. These data indicate that EF mediated the relation between DT and aggression for men. Although in the correct direction, the relation between DT and aggression was not significant for women ($\beta = -.09, p = ns$), and therefore, mediation could not be tested. Nevertheless, it should be noted that the relation between EF and aggression was significant for women ($\beta = .24, p < .05$).

Alternative Model

It is important to note that the conceptual model put forth in this paper maintains that EF mediates the relation between DT and aggression. However, from a statistical perspective, one could easily make the case for the reverse being true. Specifically, that DT might mediate the relation between EF and aggression. This alternative model was not supported.

DISCUSSION

The results of this study indicate that EF mediated the relation between DT and self-reported aggression. However, this model was upheld for men only. Indeed, although EF predicted aggression among women in this study, it did not sufficiently explain the relation between DT and self-reported aggression. One possible explanation for this gender difference is that women do not exhibit physical aggression to the same degree as men because they may possess a higher threshold for such behavior. In other words, women may necessitate a greater intensity or number of risk factors, whether biological, psychological, or environmental, in order to exhibit violence (Cloninger,

Reich, & Guze, 1975; Cloninger, Christiansen, Reich, & Gottesman, 1978). This hypothesis might explain why EF mediated the DT–aggression relation among adolescent girls in a previous study (Giancola et al., 1998) but not for women in the current study. Further consideration of this hypothesis requires an examination of key differences in the nature of the samples used in these two investigations.

Although the participants in the current investigation were healthy adults, those in the Giancola et al. (1998) study were conduct disordered adolescent girls. As such, they had what is referred to as early-onset variants of their disorders, which denote significantly more virulent etiologies and more severe courses of psychopathology compared with persons who are diagnosed with later-onset variants of these disorders (Lahey et al., 1998; Lynam, 1996; Ridenour et al., 2002). In fact, Moffitt (1993) has argued that the etiology of the early onset variant of conduct disorder is characterized by neuropsychological problems and DT, whereas the later occurring type is more likely to be caused by environmental influences (see Donnellan, Ge, & Wenk, 2000; Moffitt, Lynam, & Silva, 1994). Finally, it should also be noted that the dependent variable in the Giancola et al. (1998) study was a composite index of symptoms of conduct disorder as well as other indices of serious physical violence. Taken together, all of these factors converge upon the conclusion that the girls in the Giancola et al. (1998) study possessed a significantly stronger liability for violent and antisocial behavior than the women in the present investigation. It is argued herein that it is this differential liability that most likely accounted for the contrasting findings for women between the two investigations.

In summary, the results of the present investigation, in conjunction with the Giancola et al. (1998) report, are in keeping with the theoretical formulations described earlier postulating that the cognitive skills subsumed under the EF rubric are involved in regulating temperament (Luria, 1961, 1980; Moffitt, 1993; Tarter et al., 1985). In addition to this, the findings also bolster Moffitt's (1993) theory that neuropsychological deficits, particularly EF deficits, underlie a dysregulation of temperament that can predispose toward aggressive behavior. These theoretical formulations, as well as the results of the present study, are consistent with research showing that patients with acquired lesions to the prefrontal cortex often show a symptom complex consisting of cognitive and behavioral inflexibility, impulsivity, emotional dysregulation, as well as heightened aggression (McAllister, 1992; Tateno, Jorge, & Robinson, 2003). It is important to note these symptoms are also common features of DT.

Taken as a whole, the results of the current investigation and the Giancola et al. (1998) study suggest that EF

may be a possible underlying mechanism for the relation between DT and aggression. The findings also indicate that whereas this mechanistic role of EF occurs for both men and women, at present, the data suggest that the effect for women is restricted to those with more severe forms of antisociality. This avenue of inquiry clearly requires further investigation. Furthermore, the mediating role of EF in the DT–aggression relation has now been demonstrated using very different dependent variables (i.e., symptoms of conduct disorder denoting physical violence and self-reported aggression on a questionnaire) that lends credence to the model reported herein.

Before concluding, some limitations of the current investigation should be discussed. First, this investigation would have been strengthened by including individuals, especially women, with a clinical diagnosis associated with some type of disinhibitory psychopathology such as antisocial or borderline personality disorder or even substance use disorders. This would have broadened the empirical and theoretical generalizability of the results as well as their implications and also helped reconcile the Giancola et al. (1998) findings with those of the present investigation. Second, the present findings would have been bolstered had additional measures of aggressive behavior been utilized. For example, administering additional self-report instruments and clinical interviews to measure aggression as well as obtaining participants' history of violence by collateral report would have provided the opportunity to assess the reliability and increase the external validity of the findings.

In summary, taken within the context of the above theoretical formulations, these findings are important because they have significant clinical implications regarding the treatment and prevention of violence. Specifically, the model tested in this investigation suggests that DT may be improved by enhancing EF. In practical terms, one can see how deviations in temperament such as low adaptability to change as well as rigid behavior/thinking styles can be ameliorated by possessing strong EF skills such as cognitive flexibility and hypothesis generation abilities. Impulsivity, distractibility, and impersistence can be improved with sound cognitive appraisal and attentional skills as well as good working memory capacity. Distorted and depressogenic thinking styles that can lead to negative affect, irritability, and worry can be regulated with intact previewing and abstract reasoning abilities. Furthermore, dysregulated daily habits and functions can also be normalized by adaptively employing key EF skills such as planning, sequencing, and organization. As such, the present findings imply that by habilitating or strengthening EF skills, one can help regulate temperament that will, in turn, have the effect of decreasing the likelihood

of violence. Indirect support for this proposal is found in research showing that cognitive remediation targeting EF not only improves performance on tests of EF, but it also reduces behavioral disinhibition (Cicerone & Giacino, 1992).

ACKNOWLEDGMENT

This research was supported by grant 1R29-AA-11691 from the National Institute on Alcohol Abuse and Alcoholism.

REFERENCES

- Barbas, H. (2000). Connections underlying the synthesis of cognition, memory, and emotion in primate prefrontal cortices. *Brain Research Bulletin*, *52*, 319–330.
- Baron, R., & Kenny, D. (1986). The moderator–mediator variable distinction in social psychology research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1182.
- Beauregard, M., Levesque, J., & Bourgouin, P. (2001). Neural correlates of conscious self-regulation of emotion. *Journal of Neuroscience*, *21*, 165.
- Beer, J. S., Heerey, E. A., Keltner, D., Scabini, D., & Knight, R. (2003). The regulatory function of self-conscious emotion: Insights from patients with orbitofrontal damage. *Journal of Personality and Social Psychology*, *85*, 594–604.
- Benson, D. F., & Miller, B. L. (2000). Issues in clinical and cognitive neuropsychology. In T. E. Feinberg & M. J. Farah (Eds.), *Patient-based approaches to cognitive neuroscience* (pp. 309–316). Cambridge, MA: MIT Press.
- Blackson, T., Tarter, R., Loeber, R., Ammerman, R., & Windle, M. (1996). The influence of paternal substance abuse and difficult temperament in fathers and sons on sons' disengagement from family to deviant peers. *Journal of Youth and Adolescence*, *25*, 389–411.
- Blair, R. J. R. (2004). The roles of orbital frontal cortex in the modulation of antisocial behavior. *Brain and Cognition*, *55*, 198–208.
- Brower, M. C., & Price, B. H. (2001). Neuropsychiatry of frontal lobe dysfunction in violent and criminal behaviour: A critical review. *Journal of Neurology, Neurosurgery and Psychiatry*, *71*, 720–726.
- Buss, A., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology*, *63*, 452–459.
- Cabeza, R., & Nyberg, L. (2000). Imaging cognition II: An empirical review of 275 PET and fMRI studies. *Journal of Cognitive Neuroscience*, *12*, 1–47.
- Casey, B., Trainor, R., Orendi, J., Schubert, A., Nystrom, L., Giedd, J., et al. (1997). A developmental functional MRI study of prefrontal activation during performance of a go-no-go task. *Journal of Cognitive Neuroscience*, *9*, 835–845.
- Chow, T. W. (2000). Personality in frontal lobe disorders. *Current Psychiatry Reports*, *2*, 446–451.
- Cicerone, K. D., & Giacino, J. T. (1992). Remediation of executive function deficits after traumatic brain injury. *Neuro Rehabilitation*, *2*, 12–22.
- Cloninger, C., Christiansen, K., Reich, T., & Gottesman, I. (1978). Implications of sex differences in the prevalence of antisocial personality, alcoholism, and criminality for familial transmission. *Archives of General Psychiatry*, *35*, 941–951.
- Cloninger, C., Reich, T., & Guze, S. (1975). The multifactorial model of disease transmission: II. Sex differences in the familial transmission of sociopathy (antisocial personality). *British Journal of Psychiatry*, *127*, 11–22.

- Crown, S. (1952). An experimental study of psychological changes following prefrontal lobotomy. *Journal of General Psychology*, *47*, 3–41.
- Damasio, A. (1994). *Descartes' error*. New York: Avon Books.
- Damasio, A. (1995). Toward a neurobiology of emotion and feeling: Operational concepts and hypotheses. *Neuroscientist*, *1*, 19–25.
- Davidson, R. J. (2002). Anxiety and affective style: Role of prefrontal cortex and amygdala. *Biological Psychiatry*, *51*, 68–80.
- Davidson, R., Ekman, P., Saron, C., Senulis, J., & Friesen, W. (1990). Approach-Withdrawal and cerebral asymmetry: Emotional expression and brain physiology I. *Journal of Personality and Social Psychology*, *58*, 230–341.
- Dawson, G., Frey, K., Self, J., Panagiotides, H., Hessel, D., Yamada, E., et al. (1999). Frontal brain electrical activity in infants of depressed and nondepressed mothers: Relation to variations in infant behavior. *Development and Psychopathology*, *11*, 589–605.
- Demakis, G. (2003). A meta-analytic review of the sensitivity of the Wisconsin Card Sorting Test to frontal and lateralized frontal brain damage. *Neuropsychology*, *17*, 255–264.
- Diamond, A. (1991). Guidelines for the study of brain-behavior relationships during development. In H. Levin, H. Eisenberg, & A. Benton (Eds.), *Frontal lobe function and dysfunction* (pp. 339–378). New York: Oxford University Press.
- Donnellan, M. B., Ge, X., & Wenk, E. (2000). Cognitive abilities in adolescent-limited and life-course-persistent criminal offenders. *Journal of Abnormal Psychology*, *109*, 396–402.
- Engstrom, G., Persson, B., & Levander, S. (1999). Temperament traits in male suicide attempters and violent offenders. *European Psychiatry*, *14*, 278–283.
- Fishbein, D. (2000). Neuropsychological function, drug abuse, and violence: A conceptual framework. *Criminal Justice and Behavior*, *27*, 139–159.
- Fox, N., & Calkins, S. (1993). Pathways to aggression and social withdrawal: Interactions among temperament, attachment, and regulation. In K. Rubin & J. Asendorpf (Eds.), *Social withdrawal, inhibition, and shyness in childhood* (pp. 81–100). Hillsdale, NJ: Erlbaum.
- Fuster, J. (1995). Memory and planning: Two temporal perspectives of frontal lobe function. In H. Jasper, S. Riggio, & P. Goldman-Rakic (Eds.), *Epilepsy and the functional anatomy of the frontal lobe* (pp. 9–19). New York: Raven Press.
- Fuster, J. (1997). *The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe* (3rd edn.). New York: Raven Press.
- Giancola, P. R. (1995). Evidence for dorsolateral and orbital prefrontal cortical involvement in the expression of aggressive behavior. *Aggressive Behavior*, *21*, 431–450.
- Giancola, P. R. (2004a). Difficult temperament, acute alcohol intoxication, and aggressive behavior. *Drug and Alcohol Dependence*, *74*, 135–145.
- Giancola, P. R. (2004b). Executive functioning and alcohol-related aggression. *Journal of Abnormal Psychology*, *113*, 541–555.
- Giancola, P. R., Mezzich, A., & Tarter, R. E. (1998). Executive cognitive functioning, temperament, and antisocial behavior in conduct disordered adolescent females. *Journal of Abnormal Psychology*, *107*, 629–641.
- Giancola, P. R., & Zeichner, A. (1994). Neuropsychological performance on tests of frontal-lobe functioning and aggression in human males. *Journal of Abnormal Psychology*, *103*, 832–835.
- Goel, V., & Grafman, J. (1995). Are the frontal lobes implicated in “planning” functions? Interpreting data from the Tower of Hanoi. *Neuropsychologia*, *33*, 623–642.
- Gorenstein, E. (1987). Cognitive-perceptual deficit in an alcoholism spectrum disorder. *Journal of Studies on Alcohol*, *48*, 310–318.
- Hawkins, K., & Trobst, K. (2000). Frontal lobe dysfunction and aggression: Conceptual issues and research findings. *Aggression and Violent Behavior*, *5*, 147–157.
- Heaton, R. (1993). *Wisconsin Card Sorting Test: Computer version—2*. Odessa, FL: Psychological Assessment Resources.
- Heller, W. (1993). Neuropsychological mechanisms of individual differences in emotion, personality, and arousal. *Neuropsychology*, *7*, 476–489.
- Henry, B., Caspi, A., Moffitt, T., & Silva, P. (1996). Temperamental and familial predictors of violent and nonviolent criminal convictions: Age 3 to age 18. *Developmental Psychology*, *32*, 614–623.
- Hoaken, P., Shaughnessy, V., & Pihl, R. O. (2003). Executive cognitive functioning and aggression: Is it an issue of impulsivity? *Aggressive Behavior*, *29*, 15–30.
- Jackson, D. C., Mueller, C. J., Dolski, I., Dalton, K. M., Nitschke, J. B., Urry, H. L., et al. (2003). Now you feel it, now you don't: Frontal brain electrical asymmetry and individual differences in emotion regulation. *Psychological Science*, *14*, 612–617.
- James, A., James, S., Smith, D. M., & Javaloyes, A. (2004). Cerebellar, prefrontal cortex, and thalamic volumes over two time points in adolescent-onset schizophrenia. *American Journal of Psychiatry*, *161*, 1023–1029.
- Jansen, R., Fitzgerald, H., Ham, H., & Zucker, R. (1995). Pathways into risk: Temperament and behavior problems in three- to five-year-old sons of alcoholics. *Alcoholism: Clinical and Experimental Research*, *19*, 501–509.
- Kalin, N. H. (1999). Primate models to understand human aggression. *Journal of Clinical Psychiatry*, *15*, 29–32.
- Karnath, H., Walleesch, C., & Zimmermann, P. (1991). Mental planning and anticipatory processes with acute and chronic frontal lobe lesions: A comparison of maze performance in routine and non-routine situations. *Neuropsychologia*, *29*, 271–290.
- Kelly, T., Richardson, G., Hunter, R., & Knapp, M. (2002). Attention and executive function deficits in adolescent sex offenders. *Child Neuropsychology*, *8*, 138–143.
- Kim, J. S., Choi, S., Kwon, S. U., & Seo, Y. S. (2002). Inability to control anger or aggression after stroke. *Neurology*, *58*, 1106–1108.
- Kimberg, D. Y., D'Esposito, M., & Farah, M. J. (1998). Cognitive functions in the prefrontal cortex—Working memory and executive control. *Current Directions in Psychological Science*, *6*, 185–192.
- Kingston, L., & Prior, M. (1995). The development of patterns of stable, transient, and school-age onset aggressive behavior in young children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 348–358.
- Kolb, B., & Taylor, L. (1981). Affective behavior in patients with localized cortical excisions: Role of lesion site and side. *Science*, *214*, 89–91.
- Lahey, B. B., Loeber, R., Quay, H., Applegate, B., Shaffer, D., Waldman, I., et al. (1998). Validity of DSM-IV subtypes of conduct disorder based on age of onset. *American Academy of Child and Adolescent Psychiatry*, *37*, 435–442.
- Lapierre, D., Braun, C., & Hodgins, S. (1995). Ventral frontal deficits in psychopathy: Neuropsychological test findings. *Neuropsychologia*, *33*, 139–151.
- Lau, M., & Pihl, R. O. (1996). Cognitive performance, monetary incentive, and aggression. *Aggressive Behavior*, *22*, 417–430.
- Luria, A. (1961). *The role of speech in the regulation of normal and abnormal behavior*. New York: Basic Books.
- Luria, A. (1980). *Higher cortical functions in man*. New York: Basic Books.
- Lynam, D. R. (1996). Early identification of chronic offenders: Who is the fledgling psychopath? *Psychological Bulletin*, *120*, 209–234.
- MacLeod, C. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin*, *109*, 163–203.
- Malloy, P., Noel, N., Longabaugh, R., & Beattie, M. (1990). Determinants of neuropsychological impairment in antisocial substance abusers. *Addictive Behaviors*, *15*, 431–438.
- McAllister, T. W. (1992). Neuropsychiatric sequelae of head injuries. *Psychiatric Clinics of North America*, *15*, 395–413.
- McManis, M. H., Kagan, J., Snidman, N. C., & Woodward, S. A. (2002). EEG asymmetry, power, and temperament in children. *Developmental Psychobiology*, *41*, 169–177.
- Miller, B. L., & Cummings, J. L. (1999). *The human frontal lobes*. New York: Guilford Press.

- Milner, B. (1995). Aspects of human frontal lobe function. In H. Jasper, S. Riggio, & P. Goldman-Rakic (Eds.), *Epilepsy and the functional anatomy of the frontal lobe* (pp. 67–84). New York: Raven Press.
- Milner, B., & Petrides, M. (1984). Behavioural effects of frontal-lobe lesions in man. *Trends in Neurosciences*, 7, 403–407.
- Moffitt, T. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review*, 100, 674–701.
- Moffitt, T., & Henry, B. (1989). Neuropsychological assessment of executive functions in self-reported delinquents. *Developmental Psychopathology*, 1, 105–118.
- Moffitt, T. E., Lynam, D. R., & Silva, P. A. (1994). Neuropsychological tests predicting persistent male delinquency. *Criminology*, 32, 277–299.
- Morgan, A., & Lilienfeld, S. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review*, 20, 113–136.
- Newman, J., & Kosson, D. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology*, 95, 252–256.
- Osmon, D. C. (1996). Understanding symptoms of medial frontal lobe disorder: A clinical case study. *Journal of Clinical Psychology in Medical Settings*, 3, 23–39.
- Paschall, M., & Fishbein, D. (2002). Executive cognitive functioning and aggression: A public health perspective. *Aggression and Violent Behavior*, 7, 215–235.
- Patrick, C. (1994). Emotion and psychopathy: Startling new insights. *Psychophysiology*, 31, 319–330.
- Perret, E. (1974). The left frontal lobe of man and the suppression of habitual responses in verbal categorical behaviour. *Neuropsychologia*, 12, 323–330.
- Petrides, M. (1985). Deficits on conditional associative-learning tasks after frontal- and temporal-lobe lesions in man. *Neuropsychologia*, 23, 601–614.
- Petrides, M., Alivisatos, B., Evans, A., & Meyer, E. (1993). Dissociation of human mid-dorsolateral from posterior dorsolateral frontal cortex in memory processing. *Proceedings of the National Academy of Sciences of the United States of America*, 90, 873–877.
- Phan, K., Taylor, S. F., Welsh, R. C., Decker, L. R., Noll, D. C., Nichols, T. E., et al. (2003). Activation of the medial prefrontal cortex and extended amygdala by individual ratings of emotional arousal: A fMRI study. *Biological Psychiatry*, 53, 211–215.
- Pietrini, P., Guazzelli, M., Basso, G., Jaffe, K., & Grafman, J. (2000). Neural correlates of imaginal aggressive behavior assessed by positron emission tomography in healthy subjects. *American Journal of Psychiatry*, 157, 1772–1781.
- Porteus, S. (1965). *Porteus Maze Test: Fifty year's application*. Palo Alto: Pacific Books.
- Porteus, S., & Kepner, R. (1944). Mental changes after bilateral prefrontal lobotomy. *Genetic Psychological Monographs*, 29, 3–115.
- Reitan, R. (1992). *Trail making test: Manual for administration and scoring*. Tucson, AZ: Reitan Neuropsychological Laboratory.
- Rezaei, K., Andreasen, N., Alliger, R., Cohen, G., Swayze, V., & O'Leary, D. (1993). The neuropsychology of the prefrontal cortex. *Archives of Neurology*, 50, 636–642.
- Ridenour, T. A., Cottler, L. B., Robins, L. N., Compton, W. M., Spitznagel, E. L., & Cunningham-Williams, R. M. (2002). Test of the plausibility of adolescent substance use playing a causal role in developing adulthood antisocial behavior. *Journal of Abnormal Psychology*, 111, 144–155.
- Rothbart, M. (1989). Temperament in childhood: A framework. In G. Kohnstamm, J. Bates, & M. Rothbart (Eds.), *Temperament in childhood* (pp. 59–73). New York: Wiley.
- Rothbart, M., & Ahadi, S. (1994). Temperament and the development of personality. *Journal of Abnormal Psychology*, 103, 55–66.
- Roussy, S., & Toupin, J. (2000). Behavioral inhibition deficits in juvenile psychopaths. *Aggressive Behavior*, 26, 413–424.
- Sanson, A., & Prior, A. (1999). Temperament and behavioral precursors to oppositional defiant disorder and conduct disorder. In H. Quay & A. Hogan (Eds.), *Handbook of disruptive behavior disorders* (pp. 397–417). Dordrecht, The Netherlands: Kluwer.
- Santor, D., Ingram, A., & Kusumakar, V. (2003). Influence of executive functioning difficulties on verbal aggression in adolescents: Moderating effects of winning and losing and increasing and decreasing levels of provocation. *Aggressive Behavior*, 29, 475–488.
- Sasaki, K., Gemba, H., Nambu, A., & Matsuzaki, R. (1993). Go no-go activity in the frontal association cortex of human subjects. *Neuroscience Research*, 18, 249–252.
- Seguin, J. R. (2004). Neurocognitive elements of antisocial behavior: Relevance of an orbitofrontal cortex account. *Brain and Cognition*, 55, 185–197.
- Seguin, J. R., Pihl, R. O., Harden, P., Tremblay, R., & Boulerice, B. (1995). Cognitive and neuropsychological characteristics of physically aggressive boys. *Journal of Abnormal Psychology*, 104, 614–624.
- Smith, S., Arnett, P., & Newman, J. (1992). Neuropsychological differentiation of psychopathic and nonpsychopathic criminal offenders. *Personality and Individual Differences*, 13, 1233–1243.
- Starkstein, S., & Robinson, R. (1991). The role of the frontal lobes in affective disorder following stroke. In H. Levin, H. Eisenberg, & L. Benton (Eds.), *Frontal lobe function and dysfunction* (pp. 288–303). New York: Oxford University Press.
- Stevens, M., Kaplan, R., & Hesselbrock, V. (2003). Executive-cognitive functioning in the development of antisocial personality disorder. *Addictive Behaviors*, 28, 285–300.
- Stuss, D., & Alexander, M. (2000). Executive functions and the frontal lobes: A conceptual view. *Psychological Research*, 63, 289–298.
- Stuss, D., Floden, D., Alexander, M., & Katz, D. (2001). Stroop performance in focal lesion patients: Dissociation of processes and frontal lobe lesion location. *Neuropsychologia*, 39, 771–786.
- Stuss, D., Gow, C., & Hetherington, C. (1992). “No longer Gage”: Frontal lobe dysfunction and emotional changes. *Journal of Consulting and Clinical Psychology*, 60, 349–359.
- Stuss, D. T., & Levine, B. (2002). Adult clinical neuropsychology: Lessons from studies of the frontal lobes. *Annual Review of Psychology*, 53, 401–433.
- Tarter, R. (1988). Are there inherited behavioral traits that predispose to substance abuse? *Journal of Consulting and Clinical Psychology*, 56, 189–196.
- Tarter, R., Alterman, A., & Edwards, K. (1985). Vulnerability to alcoholism in men: A behavior-genetic perspective. *Journal of Studies on Alcohol*, 46, 329–356.
- Tarter, R., Blackson, T., Martin, C., Loeber, R., & Moss, H. (1993). Characteristics and correlates of child discipline practices in substance abuse and normal families. *American Journal on Addictions*, 2, 18–25.
- Tarter, R., & Vanyukov, M. (1994). Alcoholism: A developmental disorder. *Journal of Consulting and Clinical Psychology*, 62, 1096–1107.
- Tateno, A., Jorge, R. E., & Robinson, R. G. (2003). Clinical correlates of aggressive behavior after traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 15, 155–160.
- Taylor, W. D., MacFall, J. R., Payne, M. E., McQuoid, D. R., Provenzale, J. M., Steffens, D. C., et al. (2004). Late-life depression and microstructural abnormalities in dorsolateral prefrontal cortex white matter. *American Journal of Psychiatry*, 161, 1293–1296.
- Thomas, A., & Chess, S. (1977). *Temperament and development*. New York: Brunner/Mazel.
- Thomas, A., & Chess, S. (1984). Genesis and evolution of behavioral disorders: From infancy to early adult life. *American Journal of Psychiatry*, 141, 1–9.
- Tranel, D., Anderson, S. W., & Benton, A. L. (1994). Development of the concept of ‘executive function’ and its relationship to the frontal lobes. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology* (Vol. 9, pp. 125–148). Amsterdam: Elsevier Science.
- Tremblay, P. R., & Ewart, L. A. (2005). The Buss and Perry Aggression Questionnaire and its relations to values, the big five, provoking

- hypothetical situations, alcohol consumption patterns, and alcohol expectancies. *Personality and Individual Differences*, 38, 337–346.
- Villemarette-Pittman, N., Stanford, M., & Greve, K. (2002). Language and executive function in self-reported impulsive aggression. *Personality and Individual Differences*, 34, 1533–1544.
- Williams, T., Boyd, J., Cascardi, M., & Poythress, N. (1996). Factor structure and convergent validity of the aggression questionnaire in an offender population. *Psychological Assessment*, 8, 398–403.
- Windle, M. (1991). The difficult temperament in adolescence: Associations with substance use, family support, and problem behaviors. *Journal of Clinical Psychology*, 47, 310–315.
- Windle, M. (1992a). Temperament and social support in adolescence: Interrelations with depressive symptoms and delinquent behaviors. *Journal of Youth and Adolescence*, 21, 1–21.
- Windle, M. (1992b). Revised Dimensions of Temperament Survey (DOTS-R): Simultaneous group confirmatory factor analysis for adolescent gender groups. *Psychological Assessment*, 4, 228–234.
- Windle, M. (1994). Temperamental inhibition and activation: Hormonal and psychosocial correlates and associated psychiatric disorders. *Personality and Individual Differences*, 17, 61–70.
- Windle, M., & Lerner, R. (1986). Reassessing the dimensions of temperamental individuality across the life span: The Revised Dimensions of Temperament Survey (DOTS-R). *Journal of Adolescent Research*, 1, 213–230.