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It's in Our Nature: Verbal Aggressiveness As Temperamental Expression

Michael J. Beatty and James C. McCroskey

Over the past ten years, a substantial body of research focusing on verbal aggressiveness has accumulated. One major observation emerging from this literature is that some people are more disposed toward aggressive symbolic action than are others. Despite this considerable research effort, why individuals vary in their predispositions toward aggressive communication in interpersonal contexts is not well understood. Current speculation about the origin of verbally aggressive predispositions reflects a long standing paradigm which assigns importance to various learning processes. However, communication scholars have ignored the work of psychobiologists that strongly points to inborn neurobiological bases for human behavior and a trivial impact of environment. In this essay, we propose a theory of verbal aggressiveness supported by the work of psychobiologists as articulated in the temperament literature. We contend that verbal aggressiveness represents expressions of inborn, biological functioning, which is antecedent to social experience and, therefore, independent of social learning processes. In formulating our position, we (1) delineate a metatheoretic rationale for a temperament-based model of verbal aggressiveness, (2) integrate neurologically-based temperament functions into an explanation of research findings regarding verbal aggression, (3) present a working model of verbal aggression, and (4) discuss the implications of our theoretical position.

KEY CONCEPTS communibiology, genetics, traits, verbal aggression, temperment

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A scorpion once asked a frog for a ride across a lake. The frog pointed out that he feared the scorpion's deadly sting. "Now why would I do that?" retorted the scorpion "after all if I sting you we both drown." Having won the argument, the scorpion hopped on the frog's back and into the water they went. Halfway across the lake the scorpion stung the frog."Why did you do that?" screamed the frog "now we're both going to die!" "I

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can't help it," said the scorpion apologetically, "it's in my nature."

Over a decade ago, Infante and Wigley (1986) proposed a conceptualization and measure of verbal aggressiveness, which was firmly anchored in a personality approach to communication behavior. According to Infante and Wigley (1986), trait verbal aggressiveness refers to the predisposition to attack "the self-concept of another person instead of, or in addition to, the person's position on a topic of communication" (p. 61). In the past ten years, numerous studies validating the trait-like qualities of verbal aggressiveness and documenting the generally destructive interpersonal consequences of aggressive messages have been published (For a review, Infante & Rancer, 1996). Despite an already huge and rapidly growing body of research directed at verbal aggressiveness, relatively little progress has been made regarding the development of trait verbal aggressiveness. Put simply, after a decade of research, why there are individual differences in the predisposition toward aggressive communication remains a matter for speculation.

While communication scholars tend not to take strong stances regarding the origins of aggressive behavior, the explanations that have been offered reflect variants of social learning theory and hypothetical constructs to explain communicators' processing of situational information (Canary, Spitzberg, & Semic, 1996; Infante, 1987; Infante & Rancer, 1996). A review of the literature cited by communication scholars when musing about the reasons for verbal aggressiveness reveals a glaring inattentiveness to basic neurobiological principles. This is unfortunate indeed since psychobiologists have, in recent years, made profound advances in the understanding of human behavior. We believe that theory regarding communicative behavior should be informed by the massive body of research that has identified strong effects of inborn, individual differences in neurobiological processes underlying major dimensions of social behavior (Appleton & Mishkin, 1986; Bates & Wachs, 1994; Buss, 1989; Buss & Plomin, 1975, 1984; Collins & Depue, 1992; Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Davis, 1992; Depue & Achene, 1989; Eysenck, 1991; Eysenck & Eysenck, 1985; Farb, Aoki, Milner, Kaneko, LeDoux, 1992; Fowles, 1980; Fox, 1989, 1991; Fox, Bell, & Jones, 1992; Gray, 1982, 1987, 1990, 1991; Grillon, Ameii, Woods, Merikangas, & Davis, 1991; Kagan, 1992; Kagan, Reznick, & Snidman, 1988; Kagan & Snidman, 1991; LeDoux, 1986; LeDoux, Cicchetti, Xagoraris, & Romanski, 1990; Reiman, Fusselman, Fox, & Raichle, 1989; Reiman, Raichle, Butler, Herscovitch, & Robins, 1984; Rolls, 1990; Rothbart, 1989; Rothbart, Derryberry, & Posner, 1994; Sears & Steinmetz, 1990; Smith & DeVito, 1984; Steinmetz, 1994; Steinmetz & Thompson, 1991; Stelmack, 1990; Stelmack & Geen, 1992; Strelau, 1989; Thomas & Chess, 1977; Wachs, 1992; Zuckerman, 1991a, 1991b, 1995; Zuckerman, Kuhlman, & Camac, 1988).

Almost without exception, this work has been conducted under the rubric of *temperament*, which Bates (1989) defined as "biologically rooted individual differences in behavioral tendencies that are present early in life and are relatively stable across various kinds of situations and over the course of time" (p. 4). While a temperament-based conception of human behavior is traceable to the early ideas of Diamond (1957), recent years have witnessed striking progress in mapping inborn, neurobiological processes that are (1) foundational to behavior patterns we commonly refer to as "traits," and (2) antecedent to socialization processes. In contrast to communication scholars' speculation about social learning and situational origins of verbally aggressive behavior, the findings of psychobiologists strongly point to trivial influences of environment, which when observed are mediated by inborn, individual

differences in neurobiological functioning.

In this essay, we propose a theory of trait verbal aggressiveness supported by the principles of psychobiology (e.g., neurology, neuroanatomy, and endocrinology) as articulated in the temperament literature. We argue that trait verbal aggressiveness represents an individuals' expression of inborn, biological characteristics (described in sufficient detail later). Under our view, individual differences in verbal aggressiveness are due to parallel individual differences in neurobiology. In formulating our theoretic position, we (1) delineate a metatheoretic rationale for a biologically-based theory of verbal aggressiveness, (2) integrate neurobiological principles into the concept of verbal aggressiveness, (3) present a working model, and (4) address the implications of our theoretical position.

RATIONALE FOR A TEMPERAMENT-BASED THEORY OF VERBAL AGGRESSIVENESS

Metatheoretic Principles

Before advancing our theoretic perspective on verbal aggressiveness, it is necessary to delineate two guiding principles, which after twenty years of psychobiological research findings are taken as axiomatic in the temperament literature but, as the literature in our field suggests, remain unfamiliar to most communication scholars. In view of our interest in interpersonal communication, a considerably narrower focus than the broader field of psychology, the following principles are more precisely referred to as basic tenets of *communibiology*.

1. *All human psychological experience, both cognitive and affective, depends on brain activity, making necessary a neurobiology of temperament* (Gray, 1991; Strelau, 1994). Theoretical speculation about thinking and feeling must be consistent with neurological functioning. If we posit the existence of particular types of cognitive processes (e.g., attributing, appraising, construing, etc.), we are obligated to specify the neurological activity responsible for those processes. Scholars working from cognitive perspectives involving information processing must either identify the neurological processes antecedent to cognition or argue that cognition occurs somewhere other than the brain. If we insist that communicators are goal-oriented, we must tackle the question of first cause: Where do intention, motives, and goals originate if not within the neurobiological structures of the brain? If we insist that we can exert control over our cognitive processes or make choices, where does the control and the decision to exert it or make choices originate if not in brain structures? The alternative position is to posit the existence of some entity in control of brain processes, capable of independent cognition, perhaps like the old man behind the curtain in the *Wizard of OZ*. Scholars have long wrestled with the "mind-brain" problem (Churchland, 1986; Popper & Eccles, 1977; Squire, 1987). Our position is decidedly reductionistic. Simply stated, (1) cognition does not exist independent of neurological operations, and (2) all cognition is triggered by neurological activity.

Considering extant neurobiological knowledge, we believe that the time has past when it was sufficient to advance hypothetical constructs and processes without being attentive to neurological facts. It is instructive to recognize that conceptual terms such as "attributing," "assembling," "planning," "constructing," "selecting," and "implementing" are merely metaphors or shorthand for neurobiological operations. At best, such constructs are inferences about those underlying neurobiological processes; at worst, hypothetical constructs are misleading, misrepresenting

neurobiological reality. Quite clearly, theories that posit cognitive or emotional processes that do not correspond to neurological functioning are probably wrong, regardless of how intuitively appealing the theories might seem.

We would make a similar argument regarding conceptualizations of anger, fear, anxiety, humiliation, embarrassment, guilt, and all other expressions of affect, endorsing Zajonc and McIntosh's (1992) suggestion that "The key to rapid theoretical development is understanding the connection between the subjective realm of the emotions and their neurobiological substrate" (p. 70). Although configuring and reconfiguring written accounts of emotional experiences into prototypes provide opportunities for creative expression among researchers (not to mention justifying scores of masters theses and doctoral dissertations), any serious attempt to describe emotional features of communicative processes must embrace fundamental neurobiological functioning. More generally, credible explanations of why and how people interact in social situations in the ways they do requires adequate attention to the neurobiology of interpersonal communication, or communibiology.

From the start of Infante and Wigley's (1986) work to the present (e.g., Infante & Rancer, 1996), the conceptualization of verbal aggressiveness has involved both cognitive and affective components. Following the line of thinking set forth thus far, there must by definition be a neurobiology of verbal aggressiveness. Therefore, we are obligated to describe the neurobiological processes responsible for aggressive symbolic behavior and represented by individuals' responses to the Verbal Aggressiveness Scale (Infante & Wigley, 1986).

2. *The presence of temperament traits and individual differences since infancy cannot be explained by environmental factors* (e.g., Nelson, 1994; Strelau, 1994). Under this view, people react to environmental stimuli but environment does not "shape" traits in any direct or significant way. Instead, temperament, which is mostly inherited (other influences on temperament include prenatal care, drug and alcohol abuse, and disease, Chess & Thomas, 1989), mediates reactions to environmental stimuli. As we will show later, the inborn, neurobiological circuitry underlying individual temperament traits also influences selective attention and the regulation of attentional processes, providing the inborn, biological mechanisms necessary for selective perception and processes on environmental stimuli (Ball & Zuckerman, 1992; Brown & Kulik, 1977; Derryberry & Rothbart, 1989; MacLeod & Mathews, 1988; Mathews, 1990; Nelson, 1994; Posner, 1990; Posner & Peterson, 1987; Posner & Presti, 1987; Vogt, Finch, & Olsen, 1992).

Consistent with the proposition that social experience only minimally affects temperament, studies of twins who were raised apart and those who were raised together provide strong evidence that individual differences are largely attributable to heredity (e.g., Bouchard, 1993; Horvath, 1995; Lykken & Tellegen, 1996; Myers & Dierer, 1995; Zuckerman, 1991b, 1994). A recent study, for example, of several thousand twins found that nearly eighty percent of the variance in subjective well-being is attributable to heredity. Factors such as socioeconomic status, marital status, religious commitment, family income and educational attainment each accounted for less than three percent of the variance, with most accounting for less than two percent. In that study, the single best predictor of whether a person was generally happy (the presence of positive affect and the absence of negative affect) was the happiness of that person's twin, whether or not they were raised apart.

Lykken and Tellegen's (1996) findings regarding the heritability of happiness is relevant to our treatment of verbal aggressiveness since the chronic experience of

negative affect is associated with hostility (Zuckerman, 1995), the global construct of which verbal aggressiveness is seen as a subset (Infante, 1987; Infante & Rancer, 1996). Similar findings to those of Lykken and Tellegen have been found in studies of twins for a number of traits such as "communicator style" (Horvath, 1995), "sensation-seeking" (Zuckerman, 1994), "constraint" (Bouchard, 1993), a potential regulatory inhibitor of aggression, as well as aggressiveness itself (Rushton, Fulker, Neal, Nias, & Eysenck, 1986). Although the shared variance due to common biological origin is quite large, especially compared to effect sizes typically observed in communication studies, critics might point out that in some twin studies up to forty-percent of the variance is not explained by biology. As with all social research, however, the magnitude of effects is limited by a variety of methodological imperfections. Environmental effects can more accurately be teased out by comparing the correlations between personality scores for twins raised together with those of twins reared apart. In reviewing the evidence gleaned from such studies, Zuckerman (1994) observed that "There is little difference between the correlations for identical twins who were raised apart and those who were raised together, which indicated that shared environment is of little importance for these traits" (p. 245).

Consistent with the proposition that neurobiological structures underlying individual differences in temperament are inborn, numerous scholars have observed behavioral markers of these neurobiological processes during infancy (Bates, 1987; Calkins & Fox, 1992; Eaton, 1983; Fox, 1980, 1989: Fuster, 1990; Gunnar, 1990; Kagan, Reznick, & Snidman, 1988; Kagan & Snidman, 1991; Matheny, Riese, & Wilson, 1985; Nelson, 1993; Porter & Collins, 1982; Riese, 1987; Stifter & Fox, 1990; Thomas, Chess, & Birch, 1968; Torgerson & Kinglen, 1978; Wachs, Morrow, & Slabach, 1990). From a communibiological perspective, situational or environmental explanations of verbal aggressiveness, or any other interpersonal process for that matter, should be proffered as a last resort, only after all neurobiological explanations have failed.

NEUROBIOLOGICAL FOUNDATION OF VERBAL AGGRESSIVENESS

Neurobiological Bases of Individual Differences in Trait Verbal Aggressiveness

Several published studies have established the existence of individual differences in a reasonably stable predisposition to employ aggressive messages in interpersonal contexts (Infante & Rancer, 1996). The empirical foundation for this conclusion resides mostly in the descriptive statistics observed for Infante and Wigley's (1986) self-report instrument, the Verbal Aggressiveness Scale (VAS). As verbal aggressiveness scholars already know, the VAS consists of items focused on respondents' typical and preferred ways of dealing with others. Like most other trait-based measures, accurate VAS scores require respondents to reflect about previous interactions. High VAS scores indicate a consistent tendency to deploy aggressive messages.

At the most fundamental level, then, the validity of our view that verbal aggressiveness represents the expression of temperament requires that we first posit a neurological foundation for stable, individual differences in the *inclination* to deploy aggressive messages. We will later propose neurological processes that either inhibit or channel neurobiological inclinations into manifest behavior. As a starting point, the neurobiology of verbal aggressiveness can be examined within the circuitry underlying human aggression and hostility in general, since the former is a narrower subset of the latter set. Although the neurobiological circuits involved in aggression are not

completely understood, enough is known to inform and substantially augment our understanding of verbal aggressiveness. Perhaps the most detailed model of the neurobiology of temperament was proposed by Gray (1982, 1987, 1990, 1991). Evolving across time and consistently under refinement, Gray's model serves as a prominent conceptual model for temperament researchers (Bates & Wachs, 1994).

Gray's (1991) model is particularly relevant to the conceptualization of verbal aggressiveness because it integrates the neurobiological structures into three interconnected behavior systems, all of which are involved in the instigation and inhibition of aggressive behavior. One set of neurological circuits, described by Gray (1991) as the *fight or flight system* (FFS), interconnects the basolateral and centromedial nuclei of the amygdala, the ventromedial nucleus of the hypothalamus, the central gray region of the midbrain, and the somatic and motor nuclei of the lower brain stem. Other scholars studying "rage" have identified similar systems (Adams & Victor, 1993; Marieb & Mallatt, 1992; Panksepp, 1982, 1986). From a purely anatomical perspective, the hypothalamus can be viewed as the seat of "rage" (Marieb & Mallatt, 1992) and studies of the effects of lesions of the ventromedial hypothalamus indicate that this region inhibits aggressive behaviors (Panksepp, 1982, 1986). The anatomical description of the circuitry is supported by biochemical analyses. For example, according to Panksepp (1986), prosocial behaviors such as friendliness, bonding, and comforting behavior, depend on opiate projections to the ventromedial hypothalamus from the amygdala and associated limbic structures. More importantly, however, withdrawal of opiate results in irritability and aggression. Spoont (1992) points out that serotonergic projections from the midbrain suppresses aggression by constraining information processes within the aggressive circuitry of the FFS.

Although humans share in common the basic anatomical structures just described, psychologists have observed individual differences in the reactivity of neurobiological systems (Eysenck, 1991; Gray, 1991; Nelson, 1994; Steinmetz, 1994; Strelau, 1994). As Gray (1991) puts it "temperament reflects parameter values . . . that determine for any individual, the operating characteristics of our three emotional systems" (p. 23). Moreover, "the major dimensions of personality. . . are created by individual differences in such parameter values' (Gray, 1991, p. 23). Commenting on individual variation in neurobiological functioning, Strelau (1994) points out that individual differences may represent "sensitivity to neuron's postsynaptic receptors or sensitivity in their synaptic transmission, the amount of neurotransmitters being released, the activity of the neural structures (including receptors) to different kinds of stimuli, all taking part in the determination of individual differences in traits" (p. 135). In a recent review of research, Zuckerman (1995) identified biochemical features associated with aggressive-hostility, including low levels of monoamine oxidase.

According to Gray (1991), detection of painful or frustrating input stimulates amygdaloid, hypothalamic, and midbrain functioning, which combine to coordinate the brain stem effectors in producing defensive and aggressive behavior. In addition, Gray (1991) contends that individual differences in the reactivity of the FFS account for individual predispositions toward aggressive behavior. Following Gray's lead, we propose that individual differences in FFS reactivity and functioning account for inclinations to engage in aggressive symbolic activity. Whether aggressive action is taken, however, depends on the activation of other neurobiologically based systems, which either inhibit or facilitate aggressive behavior.

Neurobiological Bases of Facilitation and Inhibition of Aggression

A second challenge to positing a viable temperament-based view of verbal aggressiveness regards laying down the neurobiological foundation of the forces that propel individuals toward acting on aggressive impulses. In his initial conceptual work, Infante (1987) underscores the "energizing" role of frustration, which often results when interaction goals are blocked. Indeed, studies conducted from a variety of perspectives have demonstrated that aggression escalates across interactions when participants fail to achieve their interaction goals (Beatty, Burant, Dobos, & Rudd, 1996; deTurck, 1987; Harris, Gergen, & Lannamann, 1986; Infante, Chandler, & Rudd, 1989; Infante, Sabourin, Rudd, & Shannon, 1990; Infante, Trebling, Sheperd, & Seeds, 1984; Lim, 1990).

In addition to the FFS described earlier, Gray (1991) posited a second set of neurobiological circuitry, termed the *behavioral activation system* (BAS), which describes the processes by which neurobiological systems energize certain kinds of goal-directed behavior and convert the energy into irritable aggression when efforts are blocked. As such, Gray's (1991) BAS serves as a potentially important component of our model. According to Gray (1991) the BAS is activated by potential rewards or opportunities to stifle punishment. Anatomically, the BAS consists of the basal nuclei, the neocortical regions that connect to it, the dopaminergic fibers that ascend from the midbrain, and the thalamic nuclei (Gray, 1991). As with the FFS, reactivity of BASs varies across individuals and these individual differences are instrumental in defining various personality types. For example, low thresholds for BAS activation include impulsives, neurotic extraverts (Gray, 1991), and psychopaths (Arnett, Howland, Smith, & Newman, 1993).

As mentioned, the significance of the BAS to aggressiveness resides in its potential to channel FFS activity into manifest aggressive behavior. Depue and Iacono (1989) suggested that BAS activation energizes behavior directed at acquiring rewards or eliminating punishment. Attention is selectively focused on the goal. However, when individuals' efforts are thwarted, BAS activation is converted into irritative aggression aimed at the source of frustration. This particular aspect of BAS functioning lays a neurobiological foundation for Infante's (1987) contention that frustration "energizes" verbally aggressive behavior. Furthermore, the processes related to BAS activity provide the neurobiological dynamics of escalating aggression in conflict filled interpersonal interactions. Viewed from a temperament perspective, BAS activation initially fuels instrumental behavior designed to attain interaction goals (either gain rewards or terminate aversive stimuli) but when interaction partners obstruct goal attainment BAS activity is converted into aggression directed at the interfering partner.

The theoretical work of Infante and his colleagues (Infante, 1987; Infante & Rancer, 1996) also directs our attention to potential inhibitors of verbal aggression. Even among individuals who are high in trait verbal aggressiveness, concerns about punishment or loss of rewards as consequences of verbal aggression are thought to inhibit aggressive behavior. Thus, a third requirement of a temperament-based theory of verbal aggressiveness is to identify the neurobiological mechanisms capable of exerting inhibitory effects on aggression. The *behavioral inhibition system* (BIS), also described by Gray (1991), is well-suited for our purpose. The BIS consists of a set of holistically functioning neurobiological circuits linking the hippocampus, the subiculum, and the septum with the limbic system, which is comprised of the medial

wall of the limbic lobe, the olfactory cortex, the cingulate and subcallosal gyri, and the subcortical areas of the amygdala, hypothalamus, epithalamus, anterior thalamic nuclei and a portion of the basal nuclei. The BIS responds to novel stimuli and those associated with potential punishment and cessation of reward. When activated, the BIS produces increased arousal due to its connection with the limbic system, increased attentional focus on threatening stimuli and halting of behavior. Not surprisingly, low thresholds for BIS activation are common to anxiety prone individuals.

Stimulation of the BIS, concurrent with FFS activation, due to the perception of potentially punishing stimuli or loss of rewards for acting on urges produced by the combination of FFS and BAS activation tends to inhibit aggression. The antagonistic function of the BIS in the context of aggression provides neurobiological evidence for Infante's (1987) proposition that aggressive behavior could be inhibited even for individuals high in trait aggressiveness, thereby corroborating Infante's rejection of the cross-situational consistency argument.

A WORKING MODEL OF VERBAL AGGRESSIVENESS AS TEMPERAMENTAL-EXPRESSION

We propose that individual differences in FFS, BAS, and BIS reactivity account for the way people score on Infante and Wigley's (1986) VAS. High levels of trait verbal aggressiveness can be described in terms of (1) a low threshold for BAS activity, (2) a low threshold for FFS activity, but (3) a high threshold for BIS activity. In concrete terms, individuals high in verbal aggressiveness are highly motivated to achieve goals through interpersonal interaction, quickly turn to aggressive tactics when initial attempts fail, and without sufficient inhibition, become highly aggressive. The attentional focus, which accompanies system activation, promotes persistent focus on the goal and minimizes focus on potential negative consequences of aggressive symbolic action.

Within our proposed model, we would expect verbal aggression from people who are low in trait verbal aggressiveness when stimuli from the environment are sufficient to activate BAS and FFS but insufficient to trigger BIS involvement. People high in verbal aggressiveness are not expected to behave aggressively when stimuli are insufficient to activate BAS and FFS or are sufficient to trigger BIS functioning. In this regard, our position is consistent with Infante's (1987) initial delineation of the interactionist perspective of trait verbal aggressiveness. However, verbal aggression as temperamental expression implies that compared to individuals low in trait verbal aggressiveness, high scorers on verbal aggression instruments engage in aggressive communication more because the neurobiological circuitry underlying their behavioral systems requires comparatively less stimulation to facilitate and more stimulation to inhibit aggressive responses. When persons high in verbal aggression are thwarted in the pursuit of highly valued goals and at the same time precluded from acting aggressively, we would expect them to experience high levels of internal stress.

Why verbal expressions of aggression are manifest rather than violence remains a puzzle. Certainly, many scholars have argued that verbal aggression can escalate into violence during interpersonal conflicts (deTurck, 1987; Infante, 1987; Infante & Rancer, 1999; Roloff, 1996). At this juncture, we believe that the mode of temperamental expression depends on the degree of activation of the FFS and BAS relative to the BIS. Although we have discussed the three systems as though they were either activated or dormant, most (but not all) functions operate along a continuum

(e.g., limbic activation as indexed by heart rate). Perhaps, interpersonal conflicts escalate to violence because verbal aggression represents a manifestation of the current state of relative balance among the three behavior systems. Interpersonal encounters become violent when the balance is tipped too far toward BAS and FFS functioning and away from BIS dominance. In this way, individuals who go straight to violence without warning may represent the extreme low bounds of BAS and FFS thresholds and the extreme high end of BIS thresholds.

Research indicates, for instance, that the neurobiological systems, as determined by biochemical analyses, of psychopaths (Arnett, et al., 1993) and compulsive repeat offenders (Zuckerman, 1995) are overstimulated by potential rewards (overactive BAS) and are comparatively indifferent to potential punishment (underactive BIS). The potential for violence increases for individuals when a reactive FFS is in place. A similar neurobiological profile might be found for individuals who are high in verbal aggressiveness. Of course, the communicative behavior of interaction partners also serves as stimuli processed by a set of efferent neurobiological systems (Infante, et al., 1984; Lim, 1990). Research into the neurobiological mechanisms responsible for specific modes of expression would seem worthwhile.

IMPLICATIONS OF A TEMPERAMENT-BASED CONCEPTUALIZATION OF VERBAL AGGRESSION

The position advanced is this essay has four implications for research and theory pertaining to verbal aggressiveness. First, as mentioned in the metatheoretic principles guiding our thinking, trait verbal aggressiveness is presented as an expression of inborn, neurobiological structures, leaving little variation in the trait due to environment. How then do we account for the apparent linkage reported by some scholars between parenting and subsequent aggressiveness of offspring (e.g., Baumrind, 1971; Brook, Brook, Whiteman,& Gordon, 1983; Straus & Gelles, 1980)?

Bearing in mind that scholars have pointed out that the strength of association for the intergenerational hypothesis is not large, and is greatly overstated (In addition to the studies of twins reviewed in this essay see Kaufman & Zigler, 1993 for commentary on the evidence for intergenerational models), consider that studies thought to document the effects of family environment have not controlled for the effect of neurobiological systems common to parents and off-spring. As an illustration, a longitudinal study by Brook, Brook, Whitehead, and Gordon (1983) found that fathers' parenting style correlated with sons' impulsivity and interpersonal sensitivity during college-aged years. Specifically, impulsive, insensitive sons were most likely to have been reared by authoritarian fathers. Beatty and his colleagues (1996), for example, interpreted these findings as support for parenting effects, a reasonable conclusion when psychobiological research is ignored. However, we now know that impulsivity is largely inherited (Zuckerman, 1994, 1995) and that the behaviors coded as "sensitive" may well be expressions of genetically determined social traits (see the earlier cited studies of twins). We also know that parenting behavior is associated with parents' trait verbal aggressiveness such that aggressive parents are more likely to engage in authoritarian tactics (Bayer & Cegala, 1992; Beatty, Burant, Dobos, & Rudd, 1996). Thus, prior to drawing inferences about direct effects of parenting style or behavior on children's trait development, it is necessary to first remove the variance due to neurobiological systems common to parents and children. We must also recognize that genetic transfers are complex, involving the families of both parents

and often skipping generations. Therefore, we do not contend that children's traits are simple functions of parents' biological traits but only that traits such as verbal aggressiveness are due to inborn, neurobiological systems representing the mingling of family genes and the impact of physical stimuli (e.g., prenatal care, trauma, etc.). In light of the rapidly growing body of psychobiological evidence, we contend that the observed correlations between sets of family environment variables and indices of children's social development are spurious, amounting to correlations among dependent variables: Both sets of variables are effects of common biological origins. Following this line of reasoning, we propose that trait verbal aggressiveness is relatively free from environmental effects in its development.

Second, our purpose in advancing a temperament-based model of verbal aggressiveness was two-fold: (1) to reformulate the construct in light of advances in neurobiology, and (2) to stimulate thinking and research activity. We recognize that our treatment is necessarily incomplete. As further research accumulates some of our ideas will be confirmed, some will be revised, and some will be falsified. However, we believe that the infusion of knowledge from the temperament literature provides biological evidence for the legitimacy of the vast majority of Infante and his colleagues' theorizing about verbal aggressiveness.

Third, while we do not envision verbal aggression researchers conducting CAT scans, analyzing blood samples, conducting gene splicing experiments, or boring holes into the heads of verbally aggressive communicators (although if current trends are any indication the related disciplines of neurobiology and psychobiology might be regarded as appropriate cognate areas for graduate study in communication), we do maintain that theoretical speculation about aggressive communication must be consistent with neurological functioning. As mentioned earlier, all cognitive and affective processes take place in the brain and must be describable in terms of neurobiological functioning. Although proposing hypothetical constructs without reference to their biological existence might have been useful in the early development of the discipline when data were in short supply, that time has past. Psychobiologists are rapidly increasing our knowledge of how and why we behave as we do. It is time that our theories of communicative behavior are informed by that growing body of knowledge about human functioning.

Finally, the position advocated in this essay represents a radical departure from traditional and current thinking in the field of communication. However, we view such a paradigm shift as not only healthy but necessary. It is perhaps useful to remember that the purpose of scientific theory is not only to perform a descriptive function but also to provide explanations. In recent years, unfortunately, most of the theoretical work in interpersonal communication has focused on description. Embracing a temperament-based perspective, on the other hand, leads to an explanation for why we behave as we do. It's in our nature.

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