Human Communication
A Journal of the Pacific and Asian Communication Association
Vol. 11, No.1, pp. 115 – 131

Nature Nor Nurture:

Understanding Verbal Aggression in the Military

Scott Johnson
University of Alabama at Birmingham

Jean Bodon
University of Alabama at Birmingham

James C. McCroskey
University of Alabama at Birmingham

Scott Johnson (M.A., University of Alabama at Birmingham, 2007) is a member of the National Guard of Alabama. He is currently on active duty in Iraq. Jean Bodon (Ph.D., Florida State University, 1983) is a Professor of Communication Studies at the University of Alabama at Birmingham. James C. McCroskey (Ed.D., Pennsylvania State University, 1966) is Professor Emeritus of Communication Studies at West Virginia University and currently a Scholar in Residence in the College of Humanities and Arts at the University of Alabama at Birmingham.

Abstract

A content analysis was conducted on various studies involving hormonal effects on verbal and physical aggression in animals ranging from "old world monkeys" to humans in order to explore whether genetics might be responsible for verbal aggressiveness used in communication exchanges by military personnel. The prevailing stereotype is that the military personnel are more aggressive than other social and vocational groups. The analysis shows that military personnel do not differ in neurophysiology from other groups or species. Military personnel are no more aggressive in communication or otherwise than any other social or vocational group. Military personnel do not learn to be verbally aggressive as a function of military training. Neither nature nor nurture produces increased verbal aggression in military personnel. The stereotype of members of the military is not valid.

Introduction

During the first half-century (1909-1959) of the field of Speech (now Human Com munication) it was dominated teachers of public speaking, argumentation, and debate. The re were few true research programs in this field. What scientific research on human comm unication that existed was conducted mostly by social psychologists and a very small num ber of scholars who had been educated in the areas of public speaking and argumentation who were concerned with the problem of stage fright. There was no paradigm for this rese arch to adhere to, hence progress was very limited.

In Search of a Paradigm: Modeling Theory

The first paradigm that attracted large numbers of communication scholars emerged over the decades of 1960 to 2000. This was what has come to be called the "social learning paradigm." It's best know version is known as "modeling theory." This theory emerged from the work of scholars in Psychology and Education employing the learning paradigm. Before the 1990s this had become the dominant explanation in theories concerned with communication behavior. Social learning theory is mainly based on the premise that individuals learn behavior by situational responses, observation, and imitation of other individuals' behavior. For example, someone who uses this kind of reasoning would believe that violence on television causes people to act violently. This is basically pre-90s thinking, but there are still many researchers in academia that believe that social learning is the sole factor that influences the way we communicate.

When someone learns from imitating another, they are shaping their behavior by a process known as "modeling." If I observe my father communicating aggressively, I will learn to communicate aggressively. Modeling is the most popular term used by those who use social learning to explain communication. There are two variations of this thinking: (1) The Stimulus-Response Model (S-R) and (2) The Stimulus-Organism-Response Model (S-O-R). The S-R is simple. If I watch violence, I am stimulated and I react/respond violently. The S-O-R model acknowledges the mediating influence organisms have on stimuli. The S-O-R model was a response to the S-R model and offers a more in depth explanation than the latter. Since these models could not explain all behavioral happenings, processes were introduced to explain the variance in individuals' behavior.

The processes thought to explain the variances in our behavior were attentional, retention, motor reproduction, reinforcement, and motivational processes. The attentional process posits that an individual can not respond to stimuli if they are not paying attention to the stimulus. Retention process refers to the fact that "A person cannot be much influenced by the behavior of a model if he has no memory of it." Motor Reproduction Processes assumes that if one does not possess the ability to perform the motor functions of the observed behavior, they will not be able to reproduce it. The next two, reinforcement and motivational processes, assume that if one is not outwardly motivated,

positively or negatively, they will not reproduce the observed behavior.

What is most problematic about the social learning model is that results of real scie ntific research on human communication using this paradigm has accounted for little varia nce. This has led some communication researchers to believe that is necessary that a new p aradigm for communication research be employed since the learning paradigm has so little predictive power. Social learning theory does not have the scientific value that academia once thought. However, researchers have not dismissed social learning completely. They a rgue the learning theory cannot be ruled out when individuals learn nonverbal behaviors at a young age. By definition, an individual's culture is also learned (There are some who wo uld argue against this). Another element of human communication can be explained by the ories based on the learning paradigm–the acquisition of language. Little children will learn whatever language (or languages) to which they are extensively exposed. Generalizing th ese phenomena to other elements of communication behavior, however, has not been supp orted by the research.

In Search of an Alternative Paradigm: Communibiology

The most recently advanced and more scientifically sound paradigm is the communibiological paradigm (Beatty, McCroskey, & Heisel, 1998; McCroskey, 1997). Communibiology is the study of the role of biology in the communication process. It states that communication is "driven by inborn, neurobiological processes." Different behaviors that individuals exhibit are actually stable differences in neurobiological functioning. Themes are borrowed from biology in this paradigm because "we are biological beings." This is quite a shift from modeling because communibiology states that how we behave is dictated by genetics, not environmental learning.

The two reasons that this paradigm came about in communication literature were (1) Beatty and McCroskey (2001) decided that the literature that has been accumulating since the 1970s about "temperament" and "psychobiology" indicates a great deal of our social behavior, including the way we communicate, is not under our conscious control. Biology had not, to that point, played a central role in explaining human communication. Reason number (2) is that their thinking was that neurobiological structures are completely responsible for behavior. Although those structures interact in complex ways, they are principally inherited structures. In research situations communibiology has shown very strong predictive power and "empirical relevance." This theory does not totally dismiss its predecessor, social learning, however, it estimates that temperament is 80% inherited while environmental learning and other (not yet identified) factors account for the other 20% (Beatty & McCroskey, 2001). It is important that it is noted that this is their "best guess" based on their research on communication apprehension and verbal aggression, not a scientific established fact (Beatty & McCroskey, 1997; Beatty, McCroskey, & Heisel, 1998). It is quite possible that some not-yet-studied human communication behaviors are dominated by genetics while others are not.

To further explain the new paradigm, it is known that communication is driven by

the neurological processes of the brain. In order to understand communibiology, the above mentioned term "temperament" must be defined. Temperaments are biologically rooted individual differences present early in life that remain stable throughout the course of life. Each temperament is made up of biologically based traits which, by themselves, are personality variables. Communibiology has five basic assumptions: (1) All psychological processes including cognitive, affective and motor-involved in social interaction depend on brain activity, which thereby, necessitates a neurobiology of communication traits. (2) Brain activity precedes psychological experience. (3) The neurobiological structures underlying temperamental traits and individual differences, such as those associated with communication apprehension are mostly products of genetic inheritance. (4) Environment has only a negligible effect on trait development. (5) Differences in interpersonal behavior are principally a consequence of individual differences in neurobiological functioning (Beatty & McCroskey, 2001).

Communibiology takes on a unique view under the neurophysiological perspective that posits that "media use is primarily a biological function, rather than a social one (Beatty & McCroskey, 2001)." Beatty and McCroskey also state that biology situates human behavior along with complex animal behavioral systems science, rather than occupying a privileged place resulting from non-material forces. And true understanding of human behavior lies with understanding of the organ that gives rise to these behaviors, the brain. This new research paradigm goes away from traditional mass media use because other models, agenda setting for example, state that there exists a relationship between media and communication but does not explain why and how. The way the new perspective achieves the why and how is by challenging communication professionals to study brain development, sensation and perception, learning and memory, movement, sleep, stress, aging, and neurological and psychiatric disorders.

For scholars to understand how this paradigm works Beatty and McCroskey explain in their book, the "hardware" of our brain is composed of neurons (nervous cells) embedded in a matrix of so called "glial cells." The main principle of information processing in our brains is based on neurons that are able to create electrical action potentials which are transmitted rapidly over their entire cell body and its extension- the so called "axon." The axons can connect with a number of synapses to other neurons. These synaptic connections, however, are chemical in nature. Therefore, the synaptic connections are slower (in the area of milliseconds) but allow for modulation and plasticity of connections and a counterbalance of excitatory and inhibitory signals. This hybrid system in our brain enables us to simultaneously process information in distributed sub-systems and, at the same time, ensures high cognitive plasticity to adapt flexibility to changing environments. Under most natural conditions, our brain with its complex and distributed information processing capabilities is still outperforming artificial systems." As you can see, this information is very complex and has, not surprisingly, caused an unfavorable reaction to many communication professionals due to their worrying that they will have to "relearn there trade."

What is also important to know is that anatomically, the human brain is

characterized by a folded outside layer--the cerebral cortex. All higher cognitive functions are linked to activity in this structure which is organized in a modular fashion. For instance, visual recognition flows through a hierarchy of cortical areas which represent more and more complex features. Primary areas are sensitive to brightness changes and edges in small receptive fields. Higher order areas are responsive to the perception of faces in the entire visual field. This "principle of compartmentalization" is reflected by different cell structures of these areas and a rather consistent localization within the brain. Moreover, these different cortical patches are connected in a consistent way with each other and with other neuronal structures such as the midbrain and the brainstem, the cerebellum and sensory organs. Since all higher cognitive functions can be localized in the cerebral cortex (including all limbic/emotional structures) and many brain imaging technologies-including functional neuroimaging target cortical structures, the paradigm concentrates on this part of the brain (Beatty & McCroskey 2001).

It is important to know how the cortex is placed in order to better understand the communibioligical paradigm. The cortex is separable into a left and a right hemisphere; each structured by almost symmetric gyri and sulci. Gyri can be imagined as ridges and sulci as fissures on the cerebral cortex. A gyrus is generally surrounded by one or more sulci. The gross anatomical brain structure allows distinguishing frontal, parietal, occipital, and temporal lobes in each hemisphere. The central sulcus separates the frontal lobe from the parietal lobe. The sylvian fissure (also called lateral sulcus) extending almost horizontally from front to back separates the upper frontal and parietal lobes from the lower temporal lobe. The occipital lobe is located most back (Beatty & McCroskey, 2001).

One of the more important cognitive functions as it relates to communication is the visual system. The primary visual cortex is located in the calcarine sulcus between the left and the right occipital lobes. The most information flows from the eye's retina via midbrain structures into the primary auditory cortex and from there to higher order centers. Interestingly, an exception to this flow of information seems to be the direct connections into the brain's emotional system. These "short-cuts"-bypassing visual and auditory cortices- directly influence affective processing in limbic (emotional) areas and particularly in the amygdale. This might explain why subliminal visual stimuli in mass media messages can not be consciously perceived due to their short presentation time (below fifty miliseconds) but are able to affect emotions and emotional learning (Beatty & McCroskey, 2001). Other systems linked to communication are the auditory and the tactile system. Both measure mechanical effects either sound waves in the air or mechanical changes in the skin.

Conflicting Paradigms

The two paradigms available (learning and communibiology) are not very comparable. This is because of the fourth assumption of the communibiological paradigm that states, "Environment has only a negligible effect on trait development." In order for a paradigm shift to occur the following criterion must be met: It presents a radically

different conceptualization of phenomena of interest. It suggests new research strategies. although dramatic new research procedures are not required. It suggests new research questions. It may explain events previously unexplained. Communibiology does all of this to the social learning paradigm. Due to these facts, the new paradigm sweeps modeling theory to the side. There are a few exceptions; however, and Beatty and McCroskey do not claim their theories based on the communibiological paradigm to be absolute.

A major contrast of the two paradigms is that communibiology requires almost solely the use of stating data in quantitative terms. Historically the field of communication has a lack of quantitative analysis in its research methodology. This is because communication study was mainly thought to be observatory in nature. The problem with that assumption parallels the problem with modeling theory. It can be explained by this analogy: If someone were to look down a railroad track they would tell you that the track curves. This is even so with a track that is set perfectly straight. This phenomenon is called an optical illusion which presents itself with "optical observation." Scientific research is based upon the "search" for truth. Qualitative research deals more with speculation. "The numbers don't lie." An example of this fact surfaces in twin research. Even when identical twins are separated at birth, they still exhibit identical temperaments. Modeling has only a negligible effect on identical twin's personalities. A large reason why such a paradigm shift has occurred is due to the goal of communibiology being based on a scientific theory of communication.

The Roots of Aggressive Communication

Aggressive behavior is extremely complex, and is influenced by a variety of genetic, physiological, social, and environmental factors. Although aggressive behavior has been described in a wide variety of species, there has been relatively little communication between clinical and basic researchers, and also among basic researchers studying different species. This may stem, in part, from diversity in how species handle aggressive interactions. In many species vocal, visual, or chemical displays are used to resolve conflicts, frequently without resorting to fighting. When aggressive displays do not settle disputes, the specific patterns of fighting can include various combinations of biting, wrestling, and chasing. Undoubtedly there are many important species differences in the mechanisms that underlie aggressive behavior. However, studies in fish, amphibians, birds, and mammals (including humans) indicate that a common set of the hypothalamic and limbic brain regions is activated during aggressive behavior. These data suggest that there are homologies in the neuroendocrine mechanisms regulating aggression. Neuronal activity in the so-called "social behavior circuit" may regulate the probability that an individual will behave aggressively (Trainor & Marler, 2001). Mechanisms regulating the decision to engage in aggression may be more likely to be genetically wide-spread, as opposed to mechanisms controlling species-specific aggressive behaviors (Trainor, 2003).

In support of this hypothesis, testosterone (T) is known to affect aggression in a

wide variety of species and was one of the first physiological mechanisms discovered to regulate aggression. Over time, many studies have demonstrated that promotes aggression in a wide variety of species. However, it has also become apparent that the simple hypothesis that high levels result in elevated aggression has limited explanatory power, as aggressive behavior can be expressed in conjunction with low circulating . Efforts to relate aggressive behavior with plasma levels in humans have yielded inconsistent results. However, the lack of a correlation between plasma levels and a behavioral outcome does not rule out as a contributor to behavioral variation.

For example, there is a growing literature indicating that dynamic changes in response to social interactions which cannot be reliably assessed by a baseline blood sample may have important effects on aggressive behavior (Trainor, Bird & Marler, 2004).

Introduction to Aggressive Hormones

Testosterone (T) is converted to estradiol by the aromatase enzyme. In birds and amphibians aromatase is distributed throughout the brain whereas in mammals, the distribution of aromatase is limited to hypothalamic and limbic brain areas. Interestingly, many of these brain areas are known to regulate sexual and aggressive behaviors (Trainor, 2003).

The most detailed studies of the effects of aromatase on behavior examine sexual behavior. Studies in birds, rodents, and primates indicate that estrogen formed within the brain can affect various aspects of male sexual behavior (although the evidence is less clear in humans). There is growing evidence that estrogen produced in the brain regulates aggression as well. Thus if aromatization of T has important effects on behavior, individuals with similar T levels could behave very differently if they differed in central aromatase activity or estrogen sensitivity (estrogen receptors) (Trainor, Bird & Marler, 2004).

In this content analysis, we will consider the evidence that aromatization within the brain may mask relationships between androgens and aggressive behavior. First, we will consider the effects of aromatase and its estrogenic products on aggressive behavior in a variety of species. Second, because many of these species have complex social systems, we will consider the likelihood that human reaction is similar to these species on estrogenic mechanisms of aggression. Finally, we consider how studies of aromatase and aggression conducted with animal model systems may relate to human behavior.

A limited number of clinical studies suggest that estrogens modulate aggression or its components (hostility) (Toda, 2001). Animal studies clearly show that previous social experience (especially reproductive experience) regulates aromatase activity within the brain, and could significantly increase the effects of aromatization on aggression (Trainor & Marler, 2001). Humans and non-human animals exhibit similar steroid hormone responses to certain types of social stimuli, suggesting that they are similar in aromatase function (Toda, 2001).

Defense of territories is one of the most well studied contexts of aggressive

behavior. In a wide variety of species, estrogen has been found to regulate aggressive defense of a territory. However, in some species estrogen facilitates aggression whereas in other species estrogen appears to dampen aggression. Thus, there is no simple rule that increased aromatase activity leads to increased aggression in regards to estrogen. This does not, however, mean that estrogenal aromatase patterns mirror those of T and that there are no general patterns. Factors such as the source of androgen, expression of different estrogen receptor types, and social experience can play an important role in how aromatization affects aggressive behavior. Understanding how these factors work together should reveal the bases for species similarities in the estrogenic control of behavior (Toda, 2001).

Cross-Species Similarities

Estrogens increase aggression in several species of birds, although the methodology used to measure aggression differs among studies. In Japanese quail, aromatase activity in the preoptic area (POA) of males is positively correlated with aggressive pecking responses towards a stimulus male in an adjacent cage, and males that are treated with an aromatase inhibitor exhibit reduced aggressive pecking responses (Zupac & Maler, 1993). While these results were collected in a laboratory environment, several field studies have found comparable results by measuring aggressive behavior in response to a caged "intruder" next to a speaker playing taped bird songs. Territorial males usually respond vigorously by producing aggressive song and by darting at the intruder. A study on pied flycatchers (Ficedula hypoleuca) found that aromatase activity in the diencephalon (including the POA) was positively correlated with aggression levels, even though plasma T levels were not correlated with aggression (Zupac & Maler, 1993). Studies on the Pacific Northwest song sparrow found that in the non-breeding season, male song sparrow plasma T levels were low and castration reduced aggressive song production. However, treatment with an aromatase inhibitor decreased aggressive song and chasing behavior in males. The substrate for aromatase during the non-breeding season appears not to be T, but dehydroepiandrosterone (DHEA), an androgen precursor (Zupac & Maler, 1993).

DHEA increased aggressive song in response to intruders. Experiments in zebra finches showed high levels hydroxysteroid dehydrogenase activity (which converts DHEA into the aromatizable androgen, androstenedione) in the brain, which indicates that songbirds can produce estrogen in the brain from plasma DHEA. In birds, increased aromatase activity in the brain is associated with increased aggression and hormone manipulations indicate that aromatization increases aggression. Thus, individual differences in aromatase activity in the brain can be an important factor in determining individual differences in aggressive behavior (Zupac & Maler, 1993).

Aggressive behavior is also found to have more genetic causes than environmental. In Holland, a study done on male members of a certain family found that they were prone

to violent outbursts; one male, criticized by his employer, attempted to run him over with a car, another raped his sister and was sent to a mental hospital, a third coerced his sister into undressing by threatening her with a knife. Such men display retarded motor development, difficulties in task planning, and awkward sexual behavior. Recently, researchers claimed to have found the basis of such aggressive behavior to genetic sources, specifically, a deficiency in the MAOA gene of these males. In Finland, studies were conducted on males who also displayed uncontrollable behavior, and the findings demonstrated that the men possessed a neurotransmitter substance deficiency, particularly in the messenger serotonin. This lack of serotonin has been linked to aggressive behavior: some violence prone individuals did not effectively break down these substances (Toda, 2001).

Neurotransmitters have been suggested as a significant cause of aggressive behavior. Hans Brunner, a geneticist at the University Hospital in Nijmegen, has found that the violent male members of the Dutch family mentioned earlier in this paper, lacked a gene that produces monoamine oxidase-a (MAOA). MAOA is an enzyme that breaks down significant transmitters in the brain. If the MAOA does not break down these transmitters, specifically, serotonin: then buildup of serotonin will occur and could cause a person to act violently. However, it is interesting how contradictory the finding is to several other claims. Studies have shown that serotonin, in normal levels, exerts a calming, inhibitory effect on neuronal firing and that, in low levels of serotonin, aggressive and impulse behaviors increase. This contradicts the previous claim that a built-up, or high levels of serotonin caused by the MAOA mutation, causes violent behavior (Toda, 2001).

Anatomy of Aggressive Communication

What is the neuroanatomy of aggressive behavior? Scientists have linked at least thirty-eight different parts of the brain to various behaviors considered aggressive. Research has mainly concentrated on the limbic area of the brain, which houses the amygdala, an important area in controlling emotions such as fear and anger. For example, amygdalectomy cases reduce violent behavior in individuals, but with the side affect of loss of emotion (Trainor, Bird & Maler, 2004). Temporal lobe epilepsy, which involves the amygdala, can involve aggressive behavior. However, again, problems arise regarding the specificity of aggression and the brain. It has been argued that serotonin levels affect aggressive behavior. Serotonic neurons, which reside in the brainstem, project their axons into many and functionally diverse regions of the brain, including the amygdala, hypothalamus, hippocampus, cerebellum, and temporal and prefrontal regions of the cerebral cortex (Trainor, Bird & Marler 2004). It would be surprising, given this wide spectrum, if abnormalities of the serotonin system affected aggression in a specific way. Furthermore, several humans suffering from a variety of head injuries have displayed aggressive behavior, but never neatly, never in a localized manner which allows for scientists to define a physical pathway of aggression (Trainor, 2003).

Ethologists emphasize four aspects of aggressive behaviors: one related to the

adaptive functions, another related to the neural mechanisms, and a focus on their ontogeny and phylogeny of these behaviors. In fact, ethological analysis relies upon the study of animal behavior from an evolutionary perspective that is, taking into account the adaptive significance of aggressive behavior and the selective pressures that act on a specific type of aggression. This analysis begins with the precise description of different patterns of aggressive behaviors, postures, signals and their sequential and temporal organization (Trainor, 2003). An important assumption of ethologists is that the description of an ethogram implies that species-specific behaviors, in order to be shared by all members of the species, must have, at least in part, inherited characteristics. This assumption is supported by the following observations: (1) the patterns of intraspecific attack are highly stereotyped (fixed action patterns) with little inter-individual variability and fully expressed the first time the animal is exposed to the proper releaser; (2) the fixed action patterns of aggressive behavior are very similar in closely related species and (3) the neurobiological substrates of aggression are highly conservative and homologous in the subphylum of vertebrates, thus supporting the common phylogenetic origin (Trainor, Bird & Marler, 2004).

Early psychologists emphasized the role of environmental variables and experience in the development of aggressive behaviors in a particular individual. These psychological theories were based on evidence that aggression is learned and can be strongly influenced by the experience with models. Furthermore, positive rewards or punishment, as a consequence of an aggressive outburst may strongly affect future aggressive responses. In this view, aggression could be considered self-reinforcing as an individual can experience the "pleasure" of fighting (Zupac & Maler, 1993).

Nature vs. Nurture

This clear distinction between inherited and environmental determinants of aggression deals with the nature vs. nurture debate where the term "nature" means entirely "biologically" determined which often is equated with "genetic," and "nurture" relates to experience and learning and thus entirely environmentally determined. Although these two different approaches have often been depicted as alternatives to model the origins of human and animal aggression, in recent years there is a general recognition of the interaction of these two perspectives in understanding how biological and psychological processes interact in the development of aggressive behavior and how genetic or social determinants could contribute in developing pathological forms of aggression (Bookwala, Frieze, Smith & Ryan, (1992).

Pre-clinical studies of aggression are relevant because they trace the evolutionary origins of human psychopathologies linked to violence and escalated aggression. However, most animal research does not focus on the pathological forms of escalated aggression. Rather, most models of aggression emphasize the adaptive forms of aggressive behaviors such as establishing and maintaining dominance or defending a territory (Toda, 2001).

From a clinical perspective, it would be useful to understand why some forms of aggression exceed the species-normative patterns, often leading to intense harm and injury. From a psychiatric point of view, these forms, of human aggression are those that require intervention both in terms of diagnosis and treatment. We propose that the understanding of the neural mechanisms involved in the expression of adaptive forms of aggressive behaviors may help in understanding how pathological forms can be expressed and which are the determinants that trigger excessive outbursts (Toda, 2001).

Impulsive–hostile–injurious violent outbursts differ fundamentally from the premeditated instrumental calculating attacks in their extreme forms, although many forms of aggressive behavior represent a mixture of both forms, proactive and reactive. In functional terms, it is possible to distinguish between two broad categories of adaptive aggression: one concerned with competition for resources (competitive aggression) and the other concerned with protection of self or offspring from potentially dangerous conspecifics or predators (protective aggression). Intraspecific competitive aggression is generally characterized by "ritualized" or "offensive" patterns of attacks as animals are usually restrained in the use of the deadliest weapons at their disposal; this limits the likelihood of causing serious injuries to their rivals (Trainor, 2003). Example to the contrary is represented by an extremely serious form of aggressive behavior adult males in a socially organized primate species, namely the "killing parties" of chimpanzees. While these deadly attacks by a chimpanzee troop toward their neighbors are rare, they cannot be dismissed as abnormal or accidental. The chimpanzees express their anticipatory excitement behaviorally and physiologically, and during the actual acts of killing they emit pleasurable vocalizations and postural displays that may have parallels in human psychopathology. Another example of deadly aggression observed in numerous rodents and primates species is the killing of infants by conspecific males and females to gain access to mates and resources. This kind of infanticide originated in the context and under the selective forces of intraspecific competition and thus it has been named "sexually selected infanticide" (Trainor, 2003).

Protective aggression against parental attack to protect offspring may be characterized by much less ritualized or "defensive" form of attack. For example, in rodents (mice and rats), offensive and defensive forms of intraspecific attack can be distinguished on the basis of the behavioral phenotypes, since in defensive attack animals persistently direct their bites to vulnerable regions (head) of the opponent. It is important to note that an unambiguous distinction between offensive and defensive intraspecific aggression is impossible because some forms of aggression, depending on the context and sex of interacting animals, may result in a mixture of offensive and defensive types of attack (Trainor, 2003).

The distinction between offensive and defensive forms of attack can be applied to s everal species, and it may be useful to catalogue the acts and postures of species-specific a ggressive behavior in order to evaluate the intensity and the potential harmful features of a specific form of aggression. In clinical practice, it could be essential to detect injurious and excessive forms of aggression in order to carefully evaluate which motivational and conte xtual factors influence its expression (Trainor, 2003).

Implications

he prevailing stereotype of members of the military is that they are more aggressive and carry higher levels of testosterone (T) than other social or vocational groups (Trainor, 2003). Human studies indicate that brain serotonin deficiency is limited to some forms of aggressive behaviors related to impaired impulse control suggested that high T levels tend to be correlated with forms of aggression that escalate out of control and that prompt negative social consequences. Impulsivity can be considered as a personality disorder as it leads to impaired social relationships. In line with the human studies, monkeys with low T levels do not necessarily show high levels of overall aggression but only an increase in those forms that are escalated to excessive levels, i.e. injurious and persistent. For example, in macaques, males with low T levels tend to engage in few social interactions causing them to be more socially isolated. Other studies in different species of Old World monkeys showed that individuals with high T levels take risks during their moves in the forest canopy by jumping long leaps at dangerous heights and repeatedly jumping into baited traps. Laboratory studies indicate that male rhesus macaques with high T levels are quicker to approach a novel black box compared with males with low T levels thus suggesting that they tend to approach more promptly unknown objects or situations that could be a potential risk (Toda, 2001).

A study focused on the immune system and how T levels were affected by a five-day military course following three weeks of combat training in a population of twenty-six male soldiers was compared and showed that the combination of continuous heavy physical activity and sleep deprivation led to energy deficiency. At the beginning of the training program and immediately after the combat course, saliva samples were assayed for T levels. T levels were higher at the onset of the course and decreased rapidly until the end due to sleep deprivation and heavy physical energy output. These results suggest that prolonged and repeated exercise such as that encountered in a military training program induces T level impairment. The impaired secretion of T was thought to be a response to the chronic stressors. Lowered T reflects a general decrease in steroid synthesis as a consequence of the physical and psychological strain. The body is often exposed to combinations of stressors, especially in military operations where the Stressors are often prolonged, hard, continuous physical exercise combined with sleep, energy, and water deficiency, cold, heat, time pressure, or periods of waiting and inactivity. The effects of these various Stressors on the soldier's health are complex but could be deleterious, as it has been shown that chronic stress experienced by soldiers in wartime leads to immunosuppression. However, the study found no increase in verbal or overall aggressive behaviour. In fact lower T levels would suggest the opposite (Zupanac & Maler, 1993).

Another study focused on T levels included blood sampling and took place three weeks before and at the end of a rigorous five week training course. The first set of samples was taken between 7:00 a.m. and 8:00 a.m. Before the sampling the subjects were requested to eat a light breakfast to simulate the conditions of the second sampling. The second sampling, at the end of the course, took place between 5:00 a.m. and 6:00 a.m. For

the second sampling, the subjects were confined to a military barracks for medical and scientific investigations. The results of this study parallelled the results of the saliva sampling mentioned in the study above. T levels declined with the onset of sleep deprivation and rigorous physical exercise (Zupanac & Maler, 1993).

Military testosterone studies provide evidence that a long-lasting exertion, such as military training, induces alterations in the immune system and hormone secretion systems. The combat training program is of particular interest as the uniform and predictable nature of stressors and the effects on the limbic system. Interestingly, the military training schedule has been shown to decrease levels of leptin three, a hormone secreted by adipose tissue, which is regarded as an index of energy availability. Leptin levels are reduced by physical and psychological stressors as a consequence of energy deficiency and lowered leptin is associated with impaired T secretion (Zupanc & Maler, 1993). There are few data on the consequences of repeated and prolonged exposure to multifactorial stressful conditions on the hormone secretion systems.

Several clinical trials have assessed the effects of T or estrogen treatment on aggressive behavior in civilians. A major advantage of clinical trials is that patients are prospectively treated over time, which allows for assessments of cause and effect. Some studies have moved beyond self reports of aggression through the use of observers blind to treatment assignment to assess behavior, but the majority rely on self-reports via questionnaires which may be less accurate. A disadvantage of the clinical trials on civilians is that they are typically conducted in ill populations, such as those with endocrine or psychiatric disorders, so it is uncertain how the results will apply to healthy populations. Finally, it can be difficult to recruit qualified participants, so samples sizes are often low. Despite these problems, clinical studies are an essential tool to determine cause and effect (Zupanc & Maler, 1993).

Studies of aggressiveness in the contemporary operating environment (COE) are al so better predictors due to the nature of military leaders and trainers influencing performan ce, ability, motivation, self-confidence and their perceptions of the competence shown by i ncoming soldiers. The supervisory soldiers' style and behavior have lead civilians to form impressions and attitudes about how military members communicate with each other. Trai ning cycles embody contexts in which controlled aggression occurs. Researchers examinin g marital (Infante, Chandler, & Rudd, 1989) and parent-child relationships (Infante, Kassin g, & Pierce, 1998) have revealed that physical aggressiveness accompanies verbal aggressi veness. These tactics occur in the trainers' attempts to improve overall trainee performance in the COE.

Verbal Aggressiveness as a Training Aid

Studies have illustrated that aggression can seep into Military trainer-trainee relationships. The trainers will experience frustration when they inaccurately interpret the trainee's efforts, when their goals differ from their trainee's goals, and when they mismanage training. These frustrations, in turn, may lead trainers to adopt more aggressive means of soliciting performance from their trainees. Trainers will then use verbal

aggressiveness as a "training aid" to correct the misperceived behavior of the trainees.

Verbal aggressiveness is a destructive aggressive communication trait that involves attacking the self-concept of another person in order to stimulate psychological pain (Infante &Wigley, 1986). Research indicates that verbal and physical aggression are related (Bookwala, Frieze, Smith & Ryan, 1992) and exposure to verbal aggression relates to decreased self-esteem, experiencing less verbal affection, and depression in marital couples (Sergin & Fitzpatrick, 1992). These environmental factors are contained in the learned portion of Beatty and McCroskey's twenty-eighty percent environment to genetics rule of communibiology (Beatty & McCroskey, 2001). Also contained in the same twenty percent is training military "educators" receive to prepare them for instructing the trainees.

Another way to define verbal aggressiveness is by dividing the term into two predispositions: argumentativeness and verbal aggressiveness. These two predispositions are well researched with regard to factors influencing the way individuals behave in situations in which conflict is prevalent (Conflict is always prevalent in the COE), or when advocacy and refutation are necessary (Infante & Rancer, 1993). With respect to the COE, however, much less is known concerning trainee perceptions of both aggressive traits and the influence they have on perceptions of understanding trainers' commands. One of the most fundamental conclusions drawn from the literature on aggressive communication is that the outcomes of argumentativeness are positive and the outcomes of verbal aggressiveness are negative (Rancer, Whitecap, Kosberg, & Avtgis, 1997). According to Infante and Rancer (1982), trait argumentativeness is a generally stable trait which encompasses the characteristics of advocacy for (and defense of positions, while simultaneously refuting the positions that others hold on those issues. In instructional settings, perceived instructor argumentativeness is positively associated with student state motivation (Myers & Knox, 2000), instructor assertiveness and responsiveness (Myers 1998), and classroom satisfaction and affective learning (Myers & Knox, 2000).

What all of these studies have shown is that the anatomy and neurobioligical functi oning of military members does not deviate from other human beings. The fact that overall T levels in soldiers decreases during the duration of training explains why most military s chools have a "relaxation period" after the first three weeks of training. Though it is possib le to remain verbally aggressive when T levels are completed, it is more of a display of "fa lse motivation" than the activation of the amygdala and the secretion of other aggression c ausing hormones. The brain is responsible for what is referred to as "corrective training" (verbal or physical aggression by trainers aimed at trainees). Even the choice to use such a s trategy is biochemically based and is therefore genetic in nature. Choices are situational an d a "situation never chased anyone down the street" (Beatty & McCroskey, 2001). This su ggests that military members are no more aggressive in communication or otherwise than a ny other social or vocational group. Military personnel do not learn to be verbally aggressi ve as a function of military training, and people who join the military are no more genetica lly verbal aggressive than those who do not. Neither nature nor nurture produces increased verbal aggression in military personnel. The stereotype of members of the military is not v alid.

References

Beatty, M. J. & McCroskey, J. C. (1997). It in our nature: Verbal aggressiveness as temperamental expression. *Communication Quarterly, 45,* 446-460.

Beatty, M. J., & McCroskey, J. C. (2001). *The Biology of Communication: A Communibiological Perspective.* Cresskill, NJ: Hampton Press.

Beatty, M. J., McCroskey, J. C., & Heisel, A.. D. (1998). Communication apprehension as temperamental expression: A communibiological paradigm. *Communication Monographs, 64,* 197-219.

Bookwala, J., Frieze, I., Smith, C., & Ryan, K. (1992). Predictors of dating violence: A multivariate analysis. *Violence and Victims, 7,* 297-311.

Infante, D., Chandler, T., & Rudd, J. (1989). Test of an argumentative skill deficiency model of interspousal violence. *Communication Monographs, 56,* 163-177.

Infante, D., Kassing, J. & Pierce, K. (1998, November). Corporal punishment and communication in father-son dyads. Paper presented at the National Communication Association Convention, New York, NY.

Infante, D., & Rancer, A. (1982). A conceptualization and measure of argumentativeness. *Journal of Personality Assessment, 46,* 72-80.

Infante, D., & Rancer, A. (1993). Relations between argumentative motivation, and advocacy and refutation on controversial issues. *Communication Quarterly, 41,* 415-426.

Infante, D., & Wigley, C. (1989). Verbal aggressiveness: An interpersonal model and measure. *Communication Monographs, 53,* 61-69.

Lindlof, T. (1995). *Qualitative Communication Research Methods.* Thousand Oaks, Sage Publications, Inc. (pp 170-194).

Lindlof, T. & Taylor, B. (2004). *Asking, listening, and telling.* Thousand Oaks, Sage Publications, Inc. (pp 170-208).

Martin, M., & Anderson, C. (1997). Aggressive communication traits: How similar are young adults and their parents in argumentativeness, assertiveness, and verbal aggressiveness? *Western Journal of Communication, 61,* 299-314.

Myers, S. (1998). Instructor socio-communicative style, argumentativeness, and verbal aggressiveness in the college classroom. *Communication Research Reports 15,* 141-150.

Myers, S. (2002). Perceived aggressive instructor communication and student state motivation, learning, and satisfaction. *Communication Reports, 15,* 113-121.

Myers, S., & Knox, R. (2000). Perceived instructor argumentativeness and verbal aggressiveness and student outcomes. *Communication Research Reports, 17,* 299-309.

Myers, S., & Rocca, K. (2000). The relationship between perceived instructor communicator style, argumentativeness, and verbal aggressiveness. *Communication Research Reports, 17,* 1-12.

Rancer, A, Whitecap, V., Kosberg, R., & Avtgis T.(1997). Testing the efficacy of a communication-training program to increase argumentativeness and argumentative

behavior in adolescents. *Communication Education, 41,* 375-387.

Segrin, C., & Fitzpatrick, M. (1992). Depression and verbal aggressiveness in different marital couple types. *Communication Studies, 43,* 79-91.

Trainor, B. (2003). Variation in aromatase activity in the medial preoptic area and plasma progesterone is associated with the onset of paternal behavior *Neuroendocrinology 78,* 36–44.

Trainor, B., Bird, I., & Marler, C. (2004). Opposing hormonal mechanisms of aggression revealed through short-lived testosterone manipulations and multiple winning experiences, *Hormonal Behavior 45,* 115–121.

Trainor, B., & Marler, C. (2001) Testosterone, paternal behavior, and aggression in the monogamous California mouse (Peromyscus californicus), *HormonalBehavior, 40,* 3-22.

Toda, K. (2001). A loss of aggressive behavior and its reinstatement by estrogen in mice lacking the aromatase gene, *Journal of Endocrinology, 168,* 217–220.

Zupanc, G., & Maler, L. (1993). Aromatase enzyme activity in the human body: a biochemical analysis of testosterone, estrogen and serotonin on aggressive behavior. *Brain Behavior Evolution, 54,* 171–185.