AGGRESSION

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> War may sometimes be a necessary evil. But no matter how necessary, it is always an evil, never a good. We will not learn how to live together in peace by killing each other's children.
>
> — Jimmy Carter, former U.S. President

If you look at the news, it may seem as if the world is a more violent place now than ever before. But in the media, “if it bleeds it leads.” The media provide a violent, distorted reflection of reality. Television characters are 1,000 times more likely to be murdered than real people are (Robinson, 1992). Quantitative studies of body counts, such as the proportion of prehistoric skeletons with axe and arrowhead wounds, suggest that prehistoric societies were far more violent than our own. Even though one can kill a lot more people with a bomb than with an axe, the death rates per battle were about 20 times higher in ancient tribal wars than in 20th century wars (Pinker, 2007). Even if one compares 20th century wars with more recent wars, such as those fought during the Middle Ages, the death counts were much higher then than now (e.g., Eisner, 2001; Gurr, 1981). For example, estimated murders in England dropped from 24 per 100,000 in the 14th century to 0.6 per 100,000 by the early 1960s. The major decline in violence seems to have occurred in the 17th century during the “Age of Reason,” beginning in the Netherlands and England and then spreading to other European countries (Pinker, 2007). In fact, global violence has been steadily falling since the middle of the 20th century (Human Security Brief, 2007). The number of battle deaths in interstate wars has declined from more than 65,000 per year in the 1950s to less than 2,000 per year in the 2000s. There also are global declines in the number of armed conflicts and combat deaths, the number of military coups, and the number of deadly violence campaigns waged against civilians.

A number of other observations are consistent with the view that human violence is decreasing. Pinker (2007) notes: “Cruelty as entertainment, human sacrifice to indulge superstition, slavery as a labor-saving device, conquest as the mission statement of

government, genocide as a means of acquiring real estate, torture and mutilation as routine punishment,…— all were unexceptionable features of life for most of human history. But, today, they are rare to nonexistent in the West, far less common elsewhere than they used to be, concealed when they do occur, and widely condemned when they are brought to light."

Although we would like to, social psychologists cannot take credit for the significant reduction in violence that has occurred over time. Social psychologists have, however, conducted numerous studies that shed light on specific factors that increase and decrease aggression among present-day humans. We discuss the findings from these studies in this chapter. We begin by defining the terms *aggression* and *violence*. Next, we describe different theoretical explanations for aggression. We describe environmental, pharmacological, physiological, and neuropsychological factors that influence aggression. Next, we discuss different approaches for reducing aggression. Finally, we describe what topics are hot in the area of aggression today, and what topics might be hot in the future.

I. Social Psychological Definitions of Aggression and Violence

In sports and in business, the term "aggressive" is frequently used when the terms "assertive," "enthusiastic," or "confident" would be more accurate. For example, an aggressive salesperson is one who tries really hard to sell you something. The salesperson is not trying to harm you. In social psychology, the term *aggression* is generally defined as any behavior that is intended to harm another person who does not want to be harmed (e.g., Baron & Richardson, 1994). This definition contains several important features. Aggression is an external behavior that you can see. For example, you can see a person hit someone, curse someone, try to destroy someone's reputation by spreading gossip, or leave a really small tip for a waiter. (These behaviors represent different forms of aggression, which we address in detail in the next section.) Aggression is not an emotion that occurs inside a person, such as an angry feeling. Aggression is not a thought that occurs inside someone's brain, such as mentally rehearsing a

murder one would like to commit. Aggression is a social behavior because it involves at least two people. Also, aggression is intentional, though not all intentional behaviors that hurt others are aggressive behaviors. For example, a dentist might intentionally give a patient a shot of Novocain (and the shot hurts!), but the goal is to help rather than hurt the patient.

Social psychologists and laypeople also differ in their use of the term *violence*. A meteorologist might call a storm "violent" if it has intense winds, rain, thunder, and lightning. In social psychology, *violence* is aggression that has extreme physical harm, such as injury or death, as its goal. One child intentionally pushing another child down is an act of aggression but is not an act of violence. One person intentionally hitting, kicking, shooting, or stabbing another person is an act of violence. Violence is a subset of aggression. All violent acts are aggressive, but not all aggressive acts are violent. The U.S. Federal Bureau of Investigation (FBI) classifies four crimes as violent: murder, assault, rape, and robbery. Social psychologists would also classify other physically aggressive acts as violent even if they do not meet the FBI definition of a violent crime, such as slapping someone really hard across the face. But a husband who swears at his wife would not be committing an act of violence by this definition.

II. Forms and Functions of Aggression

a. Different Forms of Aggression: Physical, Verbal, Relational, Direct, Indirect, Displaced, Passive, and Active Aggression. We believe it is useful to distinguish between forms and functions of aggression. By *forms* we mean how the aggressive behavior is expressed, such as physical verses verbal, direct versus indirect, and active versus passive (Buss, 1961). *Physical aggression* involves harming others with body parts or weapons (e.g., hitting, kicking, stabbing, or shooting them). *Verbal aggression* involves harming others with words (e.g., yelling, screaming, swearing, name calling). *Relational aggression* (also called *social aggression*) is defined as intentionally harming another person's social relationships, feelings of acceptance by others, or inclusion within a group (e.g., Crick & Grotpeter, 1995).

Some examples of relational aggression include saying bad things about people behind their backs, withdrawing affection to get what you want, excluding others from your circle of friends, and giving someone the “silent treatment." Relational aggression is similar to the concept of ostracism. *Ostracism* refers to being excluded, rejected, and ignored by others (Williams, 2001).

The different forms of aggression can be expressed directly or indirectly (Lagerspetz, Bjorkqvist, & Peltonen, 1988). With *direct aggression*, the victim is physically present. With *indirect aggression*, the victim is physically absent. For example, physical aggression can be direct (e.g., choking a person) or indirect (e.g., puncturing the tires of a person’s car when they aren’t looking). Likewise, verbal aggression can be direct (e.g., cursing a person face-to-face) or indirect (e.g., spreading rumors about a person who is not present).

In *displaced aggression*, a substitute aggression target is used (e.g., Marcus-Newhall, Pedersen, Carlson, & Miller, 2000). The substitute target has not done anything to provoke an aggressive response, but just happens to be in wrong place at the wrong time. For example, a man is berated by his boss at work and “suffers in silence” rather than retaliating against his boss. When he gets home, he yells at his kids instead. Sometimes the substitute target is not entirely innocent, but has committed a minor or trivial offense. In this case, the aggression is called *triggered displaced aggression* (Pedersen, Gonzales, & Miller, 2000). For example, perhaps the man’s kids left toys in the family room rather than putting them away. Triggered displaced aggression is especially likely to occur when the aggressor ruminates about the initial offense (Bushman, Bonacci, Pedersen, Vasquez, & Miller, 2005), and when the aggressor does not like the substitute target (e.g., Pederson, Bushman, Vasquez, & Miller, 2008). People displace aggression for two main reasons. First, directly aggressing against the initial provoker may not be possible because the source is unavailable (e.g., the provoker has left the area), or because the source is an intangible entity (e.g., hot temperature). Second, fear of retaliation or punishment from the provoker may inhibit direct aggression. For example, the employee who

was reprimanded by his boss may be reluctant to retaliate because he does not want to lose his job.

The form of aggression may be active or passive. With *active aggression*, the aggressor responds in a harmful manner (e.g., hitting, cursing). With *passive aggression*, the aggressor fails to respond in a helpful manner. For example, the aggressor might "forget" to deliver an important message to the person. It is often difficult to establish blame with passive acts of aggression, which frequently is a desirable feature from the aggressor's perspective.

Direct and active forms of aggression can be quite risky, leading to injury or even death. Thus, most people would rather use indirect and passive forms of aggression.

b. Different Functions of Aggression: Reactive and Proactive Aggression. Aggressive acts may also differ in their function. Consider two examples. In the first example, a husband finds his wife and her secret lover together in bed. He takes his rifle from the closet and shoots and kills them both. In the second example, a "hitman" uses a rifle to kill another person for money. The form of aggression is the same in both examples (i.e., physical aggression caused by shooting and killing victims with a rifle). However, the motives appear quite different. In the first example, the husband appears to be motivated by anger. He is enraged when he finds his wife making love to another man, so he shoots them both. In the second example, the "hitman" appears to be motivated by money. The "hitman" probably does not hate his victim and probably is not angry with him. He might not even know his victim, but he kills the person anyway because he wants the money. To capture different functions or motives for aggression, researchers have made a distinction between reactive aggression (also called hostile, affective, angry, impulsive, or retaliatory aggression) and proactive aggression (also called instrumental aggression; e.g., Buss, 1961; Dodge & Coie, 1987; Feshbach, 1964). *Reactive aggression* is "hot," impulsive, angry behavior that is motivated by a desire to harm someone. Harming the person is the end goal. *Proactive aggression* is "cold," premeditated,

calculated behavior that is motivated by some other goal (obtaining money, restoring one's image, restoring justice). Harming the other person is a means to some other end goal. Some social psychologists have argued that it is difficult (if not impossible) to distinguish between reactive and proactive aggression because they are highly correlated and because motives are often mixed (Bushman & Anderson, 2001). For example, what if the husband who finds his wife making love to another man hires a hitman to kill them both? Would this be reactive or proactive aggression?

III. Theoretical Approaches to the Study of Aggression

Although aggression was probably adaptive for our ancient ancestors, it seems maladaptive today. Aggression breeds more aggression, thereby creating a "downward spiral" of aggression (Slater, Henry, Swaim, & Anderson, 2003). Even though aggressive people often get what they want in the short-run, there are many unintended consequences associated with aggression in the long-run (e.g., relationships can be damaged; retaliation can occur). We might therefore ask: Why do humans behave aggressively? Is it because our brains are old and the aggressive tendencies that were so useful for our ancient ancestors are difficult to override now? Is it because of biological abnormalities or poor upbringing? Is it because of frustration? In this section we review the major psychological theories of aggression.

a. Instinctive/psychoanalytic theories. First given scientific prominence by Darwin (1871), the instinct theory of aggression viewed aggressive behavior as motivated neither by the seeking of pleasure nor the avoidance of pain, but rather as an evolutionary adaptation that had enabled our ancient ancestors to survive better. According to this view, aggression is instinctive in humans just as it is in many other animals. Aggression has several adaptive functions, from an evolutionary perspective. Aggression helps to disperse populations over a wide area, thereby ensuring maximum use of available natural resources. Aggression helps animals to

successfully compete for limited resources in their environment and, consequently, is beneficial to their individual survival and to their ability to reproduce. Because it is closely related to mating, aggression also helps ensure that only the strongest individuals will pass their genes on to the next generation. The existence of innate, relatively automatic, aggressive responses has been demonstrated for many species (e.g., Lorenz, 1966). For example, for the male Stickleback fish, a red object triggers attack 100% of the time (Timbergen, 1952). However, no parallel innate aggressive response has been demonstrated for humans (Hinde, 1970).

In his early writings, Sigmund Freud proposed that all human behavior stems from a life or self-preservation instinct, which he called *eros*. Freud did not acknowledge the presence of an independent instinct to explain the darker side of human nature. He wrote: “I cannot bring myself to assume the existence of a special aggressive instinct alongside the familiar instincts of self-preservation and of sex, on an equal footing with them” (Freud, 1909/1961, p. 140). The atrocities of World War I changed his mind. By 1920, Freud had proposed the existence of an independent death or self-destruction instinct, which he called *thanatos*. The life instinct supposedly counteracts the death instinct and preserves life by diverting destructive urges outward toward others in the form of aggressive acts (Freud, 1933/1950).

b. Frustration-aggression theory. In 1939, psychologists from Yale University published an important book titled *Frustration and Aggression* (Dollard, Doob, Miller, Mowrer, & Sears, 1939). In this book, the authors proposed that aggression was due to frustration rather than to an aggressive instinct, as Freud had proposed. Frustration is an unpleasant emotion that arises when a person is being blocked from achieving a goal. Their theory was summarized in two bold statements: (1) “the occurrence of aggressive behavior always presupposes the existence of frustration”, and (2) “the existence of frustration always leads to some form of aggression.” In their view, frustration depended on an “expected” or “hoped for” goal being denied, and was not simply absence of achieving a goal.

This theory seemed to explain a large amount of everyday occurrences of aggression, but it soon became apparent to the authors that not every frustration led to observable aggression. Miller (1941), one of the original authors, was the first to revise frustration-aggression theory. He explained that frustrations actually stimulate a number of different inclinations besides an inclination to aggress, such as an inclination to escape or to find a way around the obstacle to the goal. The inclination that eventually dominates, he proposed, is the one that is most successful in reducing frustration. In other words, people learn through experience to respond to frustrations in a number of different ways. If aggression has been an effective response in the past, then people will tend to use it whenever they become frustrated. This idea opened the door for learning theory explanations of aggression (see next section).

In 1989, Leonard Berkowitz revised frustration-aggression theory by proposing that all unpleasant events — instead of only frustration — deserve to be recognized as important causes of aggression. The idea is that unpleasant events (including frustrations) automatically produce primitive fight or flight reactions. This fight-or-flight response is an adaptive stress-reducing response that occurs in humans and other animals (Cannon, 1915). When we experience an unpleasant event, we want to stop it or leave. Thus, anything that makes us feel bad automatically produces aggressive tendencies. Whether or not aggression occurs depends on how the unpleasant event is interpreted and on the presence of aggressive cues. For example, if a person has just seen a violent movie and is pushed from behind while exiting the theater, he or she may very well act in an aggressive manner.

c. Learning theory models. The earliest learning theory explanations for individual differences in aggressiveness focused on operant and classical conditioning processes. *Operant conditioning theory*, developed by behaviorists such as Edward Thorndike (1901) and B. F. Skinner (e.g., Ferster & Skinner, 1957), proposed that people are more likely to repeat behaviors that have been rewarded and are less likely to repeat behaviors that have been

punished. *Classical conditioning theory*, developed by Ivan Pavlov, proposes that through repeated pairing of an unconditioned stimulus with a conditioned stimulus, the unconditioned stimulus eventually elicits a response similar to the one elicited by the conditioned stimulus (e.g., Pavlov, 1927). Dogs that heard a bell (conditioned stimulus) every time they received food (unconditioned stimulus) eventually salivated when they heard the bell alone (conditioned response). Research showed that children who are reinforced for behaving aggressively learn to behave aggressively. Children also learn to discriminate between situations when aggression pays and when it does not. Through stimulus generalization they apply what they have learned to new situations (Sears, Whiting, Nowlis, & Sears, 1953). These processes explained how aggressive behavior could be learned (e.g., Eron, Walder, & Lefkowitz, 1971).

By the early 1960s, however, it became clear that operant and classical conditioning processes could not fully explain individual differences in aggression. Bandura theorized that people learn to behave aggressively by observing and imitating others (e.g., Bandura, Ross, & Ross 1961; 1963; Bandura, 1977). In several classic experiments, Bandura tested his *observational learning theory* (also called *social learning theory*) by showing that young children imitated specific aggressive acts they observed in aggressive models. Bandura also developed the concept of *vicarious learning* of aggression by showing that children were especially likely to imitate models that had been rewarded for behaving aggressively (Bandura, 1965; Bandura, et al., 1963). Bandura argued that the imitation was the key to social learning. The child doesn't just imitate whatever behaviors he or she observes. What is important is how the child interprets the observed behavior, and how competent the child feels in carrying out the behavior (Bandura, 1986). These cognitions provide a basis for stability of behavior tendencies across a variety of situations. Watching one parent hit the other parent may not only increase a child's likelihood of hitting, but may also increase the child's belief that hitting is OK when someone makes you angry.

More recent research helps us better understand observational learning processes. Human and primate young have an innate tendency to imitate what they observe (Meltzoff, 2005; Meltzoff & Moore, 1977). They imitate expressions in early infancy, and they imitate behaviors by the time they can walk. Thus, the hitting, grabbing, pushing behaviors that young children see around them or in the mass media are generally immediately mimicked unless the child has been taught not to mimic them (Bandura, 1977; Bandura, Ross, & Ross, 1961, 1963). Furthermore, automatic imitation of expressions on others' faces can lead to the automatic activation of the emotion that the other was experiencing. For example, observing angry expressions can stimulate angry emotions in viewers (Prinz, 2005; Zajonc, Murphy, & Inglehart, 1989).

The demonstration in the mid-1990s of the existence of "mirror neurons" that fire either when an action is observed or when it is executed provided a strong basis for understanding why children imitate others (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996; Iacoboni, Woods, Bracc, Bekkering, Mazziotta, & Rizzolatti, 1999; Rizzolatti, 2005). The immediate "mimicry" of aggressive behaviors does not require a complex cognitive representation of the observed act, but only a simple "mirror" representation of it.

d. Theories based on physiological arousal. Many stimuli that increase aggression (e.g., provocation, heat, media violence) also increase arousal levels, suggesting that arousal may have a role in stimulating aggression. But why would arousal increase aggression? There are at least four possible reasons. First, high levels of arousal may be experienced as aversive (e.g., Mendelson, Thurston, & Kubzansky, 2008), and may therefore stimulate aggression in the same way as other aversive stimuli (Berkowitz, 1989). Second, arousal narrows our span of attention (Easterbrook, 1959). If aggressive cues are salient in the situation, then people will focus most of their attention on the aggressive cues, which will facilitate aggression. Third, arousal increases the dominant response**,** which is defined as the most common response in

that situation (Zajonc, 1965). If people are inclined to behave aggressively, they will be more inclined to behave aggressively when aroused. Fourth, arousal may be mislabeled as anger in situations involving provocation, thus producing anger-motivated aggressive behavior. This mislabeling of arousal has been demonstrated in several studies by Dolf Zilimann, who has named it *excitation transfer* (Zillmann, 1979, 1988). Excitation-transfer theory assumes that physiological arousal, however it is produced, dissipates slowly. If two arousing events are separated by a short amount of time, some of the arousal caused by the first event may transfer to the second event. In other words, arousal from the first event may be misattributed to the second event. If the second event increases anger, then the additional arousal should make the person even angrier. Excitation transfer theory also suggests that anger may be extended over long periods of time, if the person has attributes his or her heightened arousal to anger and ruminates about it. Thus, even after the arousal has dissipated the person may remain ready to aggress for as long as the self-generated label of “anger” persists.

e. Social-cognitive, information-processing models of aggression. Two important cognitive information-processing models were proposed in the 1980s. One model, developed by Rowell Huesmann and his colleagues (Huesmann, 1982, 1988, 1998; Huesmann and Eron, 1984), focuses primarily on scripts. In a play or movie, a script tells the actor what to say and do. In memory, a *script* defines situations and guides behavior: the person first selects a script to represent the situation and then assumes a role in the script. One example is a restaurant script (i.e., enter restaurant, go to table, look at menu, order food, eat food, pay for food, leave tip, exit restaurant; see Abelson, 1981). Scripts can be learned by direct experience or by observing others (e.g., parents, siblings, peers, mass media characters). Huesmann proposed that when children observe violence in the mass media, they learn scripts for aggressive behavior.

What determines which of the many scripts in a person's memory will be retrieved on a given occasion? One factor involves the principle of encoding specificity. According to this principle, the recall of information depends in large part on the similarity of the recall situation to the situation in which encoding occurred (Tulving & Thomson, 1973). As a child develops, he or she may observe cases in which violence is used to solve interpersonal conflicts. The observed information is then stored in memory, possibly to be retrieved later when the child is involved in a conflict situation. Whether the script is retrieved will depend partly on the similarity between cues present at the time of encoding and those present at the time of retrieval. If the cues are similar, the child may retrieve the script and use it as a guide for behavior.

The second model, developed by Dodge and his colleagues (Dodge, 1980; 1986, 1993; Dodge & Frame, 1982; Fite, Goodnight, Bates, Dodge, & Pettit, 2008), focuses primarily on attributions. *Attributions* are the explanations people give about why others behave the way they do. Dodge and his colleagues have found that aggressive people have a *hostile attribution bias* — they tend to perceive ambiguous actions by others as hostile, which can lead them to respond in hostile ways themselves. For example, if a person bumps into them, they might infer that the person did it intentionally to hurt or challenge them. A meta-analytic review showed a strong relationship between hostile attribution of intent and aggressive behavior (Orobrio de Castro, Veerman, Koops, Bosch, & Monshouwer, 2002).

Although the two models differ in their details, both view aggression as the outcome of a social problem-solving process in which situational factors are evaluated, social scripts are retrieved or attributions are made, and these scripts or attributions are evaluated (often nonconsciously) until one is selected to guide a response.

f. General Aggression Model. In an attempt to build a broad model of aggression that encompasses other aggression theories, Craig Anderson and his colleagues developed the General Aggression Model (e.g., Anderson & Bushman, 2002). In the model, certain person and

situation *inputs* are risk factors for aggression. Person inputs include anything the person brings to the situation, such as biological sex, genetic predispositions, personality traits, attitudes, beliefs, and values. Situation inputs include all external factors that can influence aggression, such as aggressive cues, unpleasant situations, and external motives for aggression (e.g., money, recognition from others). These personal and situational factors influence the person's internal state, such as aggressive thoughts, angry feelings, physiological arousal levels, and brain activity. These internal states are all interconnected. The internal states influence the decisions the person makes. These decisions influence whether the person will behave aggressively.

IV. Environmental/situational Triggers of Aggression

Often aggression can be triggered by factors external to the person, such as events that occur in one's environment. In the next two sections, we review some of the most common external triggers and the internal states they often produce and that can prompt aggressive responding. Although we have chosen to separate environmental triggers (this section) from internal triggers (next section) given the theoretical distinction between them, it often is difficult to unambiguously assign particular stimuli to only one of these two categories. In addition, although there are numerous external events that can trigger aggression, we have classified them into three categories: (1) *provocations*, (2) *aggression-related cues*, and (3) *intangible entities*.

Most people understand what provocation is, but it is useful to provide a definition. A provocation is any action taken by one person that makes another person angry. Provocations need not be intentional. For example, someone could inadvertently mention a sensitive topic during a conversation without realizing that the remarks might make their partner angry. Whether intentional or not, provocations are perhaps the most reliable predictor of aggression. A considerable amount of research has investigated the influence of provocation on aggression

(e.g., Giancola et al., 2002; Bettencourt, Talley, Benjamin, & Valentine, 2006). In laboratory studies, provocation has been operationalized in a number of ways, including personal insults (e.g., Berkowitz, 1960; Caprara, Passerini, Pastorelli, Renzi, & Zelli, 1986; Caprara & Renzi, 1981); intensity of electric shock or noxious noise administered to a participant (e.g., Bushman, 1995; Giancola & Zeichner, 1995b; Taylor, 1967); magnitude of penalties assessed during a competitive task (e.g., Bjork, Dougherty, & Moeller, 1997; Bjork et al., 2000); and exclusion from some activity (e.g., Geen, 1968; Josephson, 1988; Rule & Percival, 1971). The basic (and unsurprising) conclusion from this work is that people are much more likely to be aggressive if they have been provoked than if they have not. In fact, provocation is generally considered the most consistent and reliable predictor of aggressive behavior (see Anderson & Bushman, 1997; Bettencourt & Miller, 1996).

The second broad category of external triggers includes cues that have been associated with aggression (e.g., weapons, violent media). In an early experiment (Berkowitz & LePage, 1967), participants who had been insulted by a confederate were seated at a table that had a shotgun and a revolver on it, or, in the control condition, badminton racquets and shuttlecocks. The items on the table were described as part of another experiment that the other researcher had supposedly forgotten to put away. The participant was supposed to decide what level of electric shock to deliver to a confederate (aggression measure). The experimenter told participants to ignore the items, but apparently they could not. Participants who saw the guns were more aggressive than were participants who saw the sports items. This so-called "weapons effect" has been replicated numerous times (see Carlson et al., 1990; Turner, Simons, Berkowitz, & Frodi, 1977). The weapons effect appears to be due to increased accessibility of aggressive thoughts (Anderson, Benjamin, & Bartholow, 1998; Bartholow, Anderson, Carnagey, & Benjamin, 2005). Due to their common co-occurrence, strong associations between guns and violence form in long-term memory. Perceiving a gun can activate these associations, temporarily making aggression-related thoughts highly accessible.

Participants in one study responded more quickly to aggressive words after seeing photos of guns than after seeing photos of plants (Anderson et al., 1998). Subsequent work showed that the weapons effect differs for people who have experience with guns (e.g., hunters, target shooters) compared to people who have no experience with guns (Bartholow et al., 2005). Specifically, sport-shooters showed the typical weapon-priming and behavioral weapons effects in the presence of assault guns but not in the presence of hunting guns. In contrast, participants without prior sport-shooting experience did not show this differentiation.

Of course, weapons are just one example of cues that can become associated with aggression in long-term memory. Another example is alcohol-related cues, such as photos of alcohol bottles or words such as “vodka” and “beer.” Research has consistently shown that people – drinkers and nondrinkers alike – associate alcohol consumption with a number of psychological, emotional, and behavioral effects, including increased aggression (e.g., Goldman, 1999; Stacy, Widaman, & Marlatt, 1990). These “alcohol outcome expectancies” are conceptualized as constructs in long-term memory that develop through both direct drinking experience and through indirect experiences (e.g., observing others drinking, the media). Using a variety of cue exposure methods and aggression-related outcome measures, Bartholow and colleagues (e.g., Bartholow & Heinz, 2006; Friedman, McCarthy, Bartholow, & Hicks, 2007) have shown that exposure to alcohol-related cues – even words presented too briefly to be recognized (Friedman et al., 2007) – can elicit increased aggression, particularly among individuals whose alcohol outcome expectancies include the idea that drinking alcohol makes people aggressive. Importantly, these effects occur even though participants do not drink a single drop of alcohol or even a placebo beverage (i.e., one that they believe contains alcohol).

Similarly, hot temperatures are often linked with aggression and violence in memory. This belief has even crept into the English language, as indicated by such common phrases as “hot temper, “hot headed,” “hot under the collar,” and “my blood is boiling.” Recent research has

shown that words associated with hot temperatures (e.g., boiling, roasted) increase aggressive thoughts and hostile perceptions (DeWall & Bushman, in press).

Another important source of external triggers for aggression is mass media. Content analyses have shown that television programs, movies, video games, and other popular forms of entertainment media contain considerable amounts of violence (Gentile & Walsh, 2002; National Television Study, 1998). In 1972, the Surgeon General issued a warning on violent TV programs stating: "It is clear to me that the causal relationship between televised violence and antisocial behavior is sufficient to warrant appropriate and immediate remedial action" (Steinfeld, 1972). In the years since this warning was issued, hundreds of studies have shown a link between violent media exposure and aggression (see Anderson et al., 2003; Bushman & Huesmann, 2006).

Recently, researchers have begun to investigate the boundary conditions for media violence effects, particularly those associated with violent video games. For example, one study showed that violent video games increase aggressive thoughts, feelings and behavior immediately after game play, but that the effects don't last longer than 15 minutes (Sestir & Bartholow, 2009). Although the immediate short term effect of violent media may wear off quickly, the cumulative effects of exposure to media violence can last for many years (e.g., Huesmann et al., 2003).

Intangible entities are triggers that make people feel bad but cannot be attributed to a particular person or obvious cue. One example is hot temperature. In the mid-1700s, Montesquieu wrote of an apparent link between climate differences and crime, noting, "in the northern climates you will find people with few vices…as you move toward the countries of the south, you will believe you have moved away from morality itself: the liveliest passions will increase crime" (1748/1989, p. 234). Montesquieu's observations were correct: Hot temperatures are linked to violent and aggressive behavior (see Anderson, 1989). Other intangible entities are loud noises, including traffic noise (Gaur, 1988). Noise is especially likely

to increase aggression in when it is uncontrollable (Geen, 1978; Geen & McCown, 1984) and when it is paired with other factors that increase aggression, such as provocation (Donnerstein & Wilson, 1976) or violent media (Geen & O'Neal, 1969). Irritants in the air that we breathe can make us more aggressive, such as foul odors (Rotton, 1979), secondhand smoke (Jones & Bogat, 1978), and air pollution (Rotton & Frey, 1985).

V. Internal Triggers

One reason external factors increase aggression is that they increase aggressive thoughts and angry feelings. For example, research suggests that provocation from an external source leads to increased aggression primarily by increasing anger. Why is anger likely to increase aggression? One possible reason is that angry people aggress in the hope that doing so will help them to feel better. Research has consistently shown that people who feel bad often try to remedy or repair their moods (Morris & Reilly, 1987). Because many people believe that venting is a healthy way to reduce anger and aggression (see Bushman, Baumeister, & Phillips, 2001), they might vent by lashing out at others to improve their mood. One series of studies replicated the standard finding that anger increases aggression, but also found an interesting (and revealing) exception: When participants believed that their angry mood would not change for the next hour no matter what they did (ostensibly because of side effects of a pill they had taken), anger did not lead to aggression (Bushman et al., 2001). The implication of this finding is that anger does not *directly* or *inevitably* cause aggression. Rather, angry people attack others because they believe that lashing out will help get rid of their anger and enable them to feel better.

Pain is another internal state that has been linked to the propensity to aggress. Numerous studies conducted on animals (e.g., Azrin, Hutchinson, & McLaughlin, 1965; Hutchinson, 1983; Ulrich, 1966) have shown that experiencing physical pain elicits aggressive

responses. Similar findings have been reported with humans (e.g., Anderson, Anderson, Dill, & Deuser, 1998; Berkowitz, Cochran, & Embree, 1981). A number of hypotheses have been offered to explain why pain increases aggression. Perhaps the most interesting are the contrasting views that pain-induced aggression is (a) merely defensive versus (b) motivated by retribution. Studies using both animal (e.g., Azrin et al., 1965) and human (e.g., Berkowitz et al., 1981) participants support the latter view, showing that, for example, an animal will expend effort (e.g., by pulling a chain) in order to gain access to the target of their aggression.

Not only does physical pain increase aggression, but psychological or emotional pain, such as interpersonal rejection (i.e., feeling as though one's relationship to another person is not valued by or important to that other person) or social exclusion, has similar effects (Buckley, Winkel, & Leary, 2004). The underlying neurocognitive mechanisms of social pain are similar to those for physical pain (see Eisenberger, Lieberman, & Williams, 2003; MacDonald & Leary, 2005). Rejected people aggress for a host of reasons, including to improve their mood, to establish (or re-establish) efficacy, control, or social influence, and to seek revenge (Leary et al., 2006).

Like unpleasant feelings (e.g., anger, frustration), aggressive cognitions hold a prominent place in many theories of aggression (e.g., Dodge, 1986; Huesmann, 1998; Lindsay & Anderson, 2000). As reviewed in the previous section, a number of external triggers (e.g., guns, alcohol, temperature, media violence) increase the accessibility of aggressive thoughts. Aggressive thoughts, in turn, increase the likelihood of aggressive behaviors, either through simple priming (see Bartholow et al., 2005) or via their place in aggressive behavioral scripts (e.g., Huesmann, 1998) or by biasing one's interpretation of others' behaviors (e.g., Dodge, 1986).

VI. Chemical/pharmacological Influences on Aggression

a. Hormones and neurotransmitters. Like virtually all behaviors, aggression is mediated by changes in chemical reactions and interactions within the brain. Two naturally-occurring chemicals in the brain, testosterone and serotonin, have been linked closely with aggression. Testosterone, a male sex hormone, is a simple chemical arrangement of carbon rings, a derivative of the molecule cholesterol. Although both males and females have testosterone, males have much more of it. Testosterone levels are at their lifetime peak during puberty, and they begin to decline around the age of 23. Testosterone has repeatedly been linked to aggression in both sexes. In a review of this work, Sapolsky (1998) provided a concise description of the seemingly direct association between testosterone and aggression: "Remove the source of testosterone in species after species and levels of aggression typically plummet. Reinstate normal testosterone levels afterward with injections of synthetic testosterone, and aggression returns" (p. 150)."

Research indicates both long-term and short-term effects of testosterone on aggression (Archer, 1991). In the long run, testosterone seems to affect the development and organization of various collections of cells in the brain that are associated with sex typed behaviors (ranging from sex to hunting – see Cosmides & Tooby, 2006) as well as affecting bodily structures (e.g., muscles, height) that influence the likelihood and success of aggressive behaviors. In the short run, testosterone may increase aggression by increasing feelings of dominance. Although both effects are well established in animals, only the long-term effects are well established in humans (Book, Starzyk, & Quinsey, 2002; Brain, 1994; Reinisch, 1981).

Serotonin is another naturally-occurring chemical in the brain that is known to influence aggression, particularly impulsive aggression. Serotonin (also known by its chemical name 5-hydroxytryptamine, or 5-HT) is called the "feel good" neurotransmitter. If people don't have enough of it, they feel bad and may therefore behave more aggressively. Although serotonin can act in other parts of the body (e.g., the digestive system), in the brain serotonin is important in modulating a number of emotional and behavioral responses, including anger, mood, and

aggression. In correlational studies, levels of serotonin in the brain have been negatively related to violence in both epidemiological (Moffitt et al., 1998) and clinical samples (Goveas et al., 2004). Similar results have been reported with nonhuman primates (see Higley et al., 1992; Westergaard et al., 1999).

Even more convincing of the influence of serotonin on aggression are experimental laboratory studies showing that short-term reduction in serotonin levels, achieved by decreasing dietary tryptophan, increases aggressive responding, whereas increasing serotonin levels via dietary supplements of tryptophan decreases aggressive responding (e.g., Cleare & Bond, 1995; Marsh et al., 2002; Pihl et al., 1995). Similar results have been obtained by increasing serotonin levels using drugs such as D-fenfluramine (see Cherek & Lane, 2001), and paroxetine (Berman, McCloskey, Fanning, Schumacher, & Coccaro, 2009). Other studies have shown that long-term use of medications that increase serotonin levels reduces impulsive aggression in patients with personality disorders (e.g., Coccaro & Kavoussi, 1997; Salzman et al., 1995).

The question of just *how* serotonin influences aggression has been the subject of considerable debate and theorizing. Most theories agree that serotonin does not decrease aggression directly, but does so indirectly by its effects on other processes such as irritability, impulsivity, and information processing (e.g., Berman et al., 1997). This idea is supported by a research showing that serotonin influences impulsive (but not planned) aggression (see Berman et al., 1997), and the recent idea that factors such as alcohol increase aggression by reducing inhibitory control through serotonin levels (see McCloskey, Berman, Echevarria, & Coccaro, 2009).

b. Alcohol and other drugs of abuse. In addition to considering how naturally-occurring chemicals in the brain influence aggression, it is also important to consider how chemicals that people ingest influence aggression. By far the chemical that has received the most attention is alcohol. Considerable evidence indicates that alcohol consumption increases aggression (for reviews see Bushman & Cooper, 1990; Giancola, 2000; Ito et al., 1996). A

number of theories have been proposed to explain alcohol's aggression-enhancing effects, most of which emphasize effects of the drug on disrupting cognitive processing (see Giancola, 2000; Steele & Josephs, 1990). Perhaps the most influential of these theories has been "alcohol myopia" theory (Steele & Josephs, 1990), which posits that alcohol narrows the range of cues that people pay attention to so that they focus mainly on the most noticeable ones. For example, after a few drinks, a bar patron might be especially likely to focus attention on a highly salient, apparent provocation (e.g., being pushed in the back) and to ignore or poorly process other, more peripheral cues that might inhibit an aggressive response (e.g., that the "push" was accidental, or that the provocateur is much larger and stronger). Some recent experiments have found support for the myopia theory (e.g., Denson, Aviles, Pollock, Earleywine, Vasquez, & Miller, 2008; Giancola & Corman, 2007).

Another, similar theory posits that alcohol disrupts executive functions (Giancola, 2000). Although exactly which processes are considered executive functions is a matter of continuing debate (see Miyake et al., 2000), all models generally agree that the ability to inhibit behavior is central to executive functioning. According to the executive impairment model of alcohol-related aggression, alcohol increases aggression by reducing inhibitory control. In other words, alcohol increases aggression not by "stepping on the gas," but by paralyzing the brakes. Numerous studies have shown that alcohol impairs inhibition (see Bartholow, Dickter, & Sestir, 2006; Fillmore & Vogel-Sprott, 1999, 2000; Giancola, 2000, 2004), and inhibition is critical for withholding aggression (see Berkowitz, 1993).

It is important to note that alcohol consumption is not uniformly associated with increased aggression. A number of factors moderate the effects of alcohol on aggression. For example, alcohol is more likely to increase aggression in men than in women (see Giancola, 2002a; Gussler-Burkhardt & Giancola, 2005; Hoaken & Pihl, 2000), and alcohol is especially likely to increase aggression in men who are predisposed to behave aggressively (Giancola,

2002b; Giancola, Saucier, & Gussler-Burkhardt; Giancola, 2002c) and in individuals who expect alcohol to increase aggression (see Giancola 2006).

Considerably less research has been conducted on the aggression-related effects of other drugs of abuse, particularly in humans. However, those human studies that do exist provide evidence that cocaine exposure, for instance, is associated with increased aggression. For example, preadolescence boys (though not girls) prenatally exposed to cocaine were more aggressive than non-exposed boys and girls (Bennett, Bendersky, & Lewis, 2007). Other work with cocaine-dependent patients found similar results (see Denison, Paredes, & Booth, 1997), though causal relations are not entirely clear in such correlational studies. For obvious ethical and legal reasons it is very difficult for researchers to conducted controlled, randomized laboratory experiments on the effects of cocaine in humans. Still, the available experimental evidence indicates that acute cocaine administration leads to increased aggression in laboratory tasks (e.g., Licata, Taylor, Berman, & Cranston, 1993).

Despite the relative dearth of experimental studies with humans, effects of both acute and chronic cocaine exposure on aggression have been studied extensively with animals. Considerable research has shown that rats, hamsters, and other rodents chronically exposed to cocaine, particularly during adolescence, are more aggressive than non-exposed animals (e.g., DeLeon, Grimes, Connor, & Melloni, 2002; Harrison, Connor, Nowak, & Melloni, 2000; Knyshevski, Ricci, McCann, & Melloni, 2005). A number of studies have linked these effects to systems involving serotonin (e.g., Knyshevski et al., 2005; Ricci, Knyshevski, & Melloni, 2005). This research is consistent with the findings of other research showing that reduced levels of serotonin in humans are associated with increased aggression (e.g., Cleare & Bond, 1995; Marsh et al., 2002; Pihl et al., 1995).

VII. Neuropsychological and Physiological Correlates of Aggression and Violence

In previous sections we have discussed how neuroscience research investigating effects of brain chemicals (e.g., serotonin, testosterone) and ingested substances (e.g., alcohol) has increased understanding of aggression. In this section we extend this review by linking this work with research evidence on the relationships between brain processes, including both brain structure and function, and aggression (for a general overview of the link between brain processes and social processes, see Heatherton and Wheatley, this volume).

a. Frontal lobe function and aggression. We noted previously that alcohol consumption might increase aggression by impairing executive functioning (Giancola, 2000). This hypothesis stems from the more general idea that impaired executive functioning is linked to aggression (Giancola, 1995; Giancola, Mezzich, & Tarter, 1998; Seguin & Zelazo, 2005). Neuropsychological and functional brain imaging research has identified the frontal lobes, and in particular the prefrontal cortex (i.e., the part of the brain located just behind the forehead), as the source of executive functioning (see Roberts, Robbins, & Weiskrantz, 1998). Generally speaking, frontal lobe function is negatively related to aggression and violence, in both normal (e.g., Giancola, 1995; Giancola & Zeichner, 1994) and clinical populations (e.g., Giancola, Mezzich, & Tarter, 1998a, 1998b). Additionally, damage to the pre-frontal cortex has been linked to increased aggression and antisocial behavior (e.g., Grafman et al., 1996). The “frontal lobes” are not, however, a unitary structure. Ongoing research is beginning to specify which structure(s) within the frontal lobes are implicated in aggression, and why.

Aggression and some psychiatric disorders seem to go together like peas in a pod, especially disorders involving poor impulse control (Seo, Patrick, & Kennealy, 2008). The link is particularly strong for disorders involving low serotonin levels. Dysfunctional interactions between serotonin and dopamine systems in the prefrontal cortex appear especially important for understanding links between impulsive aggression and other psychiatric conditions. Abnormally low serotonin function could represent a biochemical trait that predisposes affected individuals to impulsive aggression. The importance of this and related work is in the potential to

identify so-called “endophenotypes” for aggression and violence. An endophenotype is essentially an intermediate phenotype, occurring in between the ultimate causes (e.g., genetic variation) and ultimate outcomes (e.g., psychiatric diagnosis) of a condition of interest. Endophenotypes are thought to be state-independent, meaning they are manifest in affected individuals regardless of whether or not the relevant syndrome or condition (e.g., antisocial personality disorder) has emerged. Thus, identifying endophenotypes for aggression and violence could be very important in the search for ways of identifying people who are at risk for extreme aggression (e.g., school shooters) before they have had a chance to wreak too much havoc, providing opportunities for intervention and treatment.

b. Contributions of electrophysiological, functional brain imaging and genetic research. Recently, some researchers have begun to investigate brain responses elicited by external and internal cues to aggression. Work of this type is important for establishing links between aggression-related triggers and the neural processes that give rise to overt behavioral expression of aggression. One study examined the desensitization effects of violent video games on the brains of young men who either played a lot of violent games or a lot of nonviolent games (Bartholow, Bushman, & Sestir, 2006). Chronic exposure to violent games was expected to be associated with muted brain responses to images depicting violence in the real world, and that this brain response was expected to be related to increased aggressive behavior. Participants completed survey measures of violent media exposure, trait hostility, and irritability, and then viewed a series of violent, negative but nonviolent, and neutral pictures while event-related brain potentials (ERPs) were recorded. Briefly, ERPs represent electrical responses generated by the brain (primarily the cortex) during information processing. A particular component (i.e., voltage deflection) of the ERP, the P300 (which occurs approximately 300 milliseconds, or three tenths of a second, following the onset of a stimulus), has been associated in previous research with the activation of approach and avoidance motivational systems in response to positive and negative images (e.g., Ito, Larsen, Smith, & Cacioppo,

1998; Schupp et al., 2000). Chronic violent video game exposure was expected to be associated with desensitization to violence, as reflected by smaller P300 responses to violent images. As expected, there was a negative association between violent game exposure and the size of the P300 elicited by violent pictures. This relationship remained even after individual differences in trait hostility and irritability were statistically controlled. Moreover, the P300 response elicited by violent pictures predicted aggressive behavioral responses in a subsequent laboratory task, suggesting that desensitization at the neural level is associated with increased aggressive responding (see also Funk et al., 2004).

Functional magnetic resonance imaging (fMRI) has been used to study the specific neural structures involved in processing violence and in regulating aggressive responding. fMRI involves the measurement of blood flow to specific brain structures in response to specific stimuli or events, which can be used as an index of how much activity in those structures is elicited by those stimuli. Recent evidence suggests that exposure to violent media may be linked to decreases in the activity of brain structures needed for regulation of aggressive behavior. For example, the anterior cingulate cortex (ACC), located in the medial frontal lobe, is vital for self-regulation, as it appears to serve as one seat of the interface between affect and cognition during the monitoring of ongoing action (see Bush, Luu, & Posner, 2000). More specifically, the ACC appears to serve an action-monitoring function (see Botvinick et al., 2001), alerting other areas of the prefrontal cortex when increased control is needed to regulate behavior. Recent work used fMRI to test potential links between exposure to violent games, ACC activity, and aggression (Weber, Ritterfeld, & Mathiak, 2006). These researchers found that engaging in virtual violence during game play was associated with decreased activation of the ACC, and in particular the rostral (anterior) part of ACC, which has been linked to integration of emotional information (see Bush et al., 2000). These data are consistent with ERP findings (Bartholow et al., 2006) in suggesting that violence exposure leads to suppression of affective

information processing, which could interfere with regulation of aggressive responding (see also Sterzer, Stadler, Krebs, Kleinschmidt, & Poustka, 2003).

Other brain imaging studies also point to areas in prefrontal cortex as important for regulating anger and aggression. These data are consistent with the neuropsychological data reviewed previously. For example, participants in one study were insulted and induced to ruminate while functional magnetic resonance imaging (fMRI) was used to measure the flow of blood to different parts of their brains (Denson, Pedersen, Ronquillo, & Nandy, 2009). The results showed that activity in areas of prefrontal cortex was positively related to self-reported feelings of anger and to individual differences in self-reported aggression. In another recent study, women received injections of testosterone while viewing slides depicting angry and happy faces (Hermans, Ramsey & Van Honk, 2008). The results showed consistent activation to angry versus happy faces in brain areas known to be involved in reactive aggression, such as the amygdala and hypothalamus. Heightened activation also was found in the orbitofrontal cortex, a region of the brain linked to impulse control. Testosterone appears to enhance responsiveness in neural circuits believed to be involved in interpersonal aggression, providing some of the first direct evidence in humans for the seat of testosterone's effects in the brain.

Recently, Raine (2008) reviewed the genetic and brain imaging literatures related to violent and antisocial behavior, and proposed a model whereby specific genes result in structural and functional brain alterations that, in turn, predispose individuals to behave in an aggressive manner. In the model, the prefrontal cortex (as well as limbic structures, such as the amygdala) is especially important for understanding aggression and violence. The model, however, goes beyond previous work by focusing on how environmental influences may alter gene expression in these areas "to trigger the cascade of events that translate genes into antisocial behavior" (2008, p. 323). For example, a common polymorphism (i.e., an individual difference in the form or expression of a biological process) in the monoamine oxidase A (MAOA) gene, which produces an enzyme important for breaking down neurotransmitters such

as serotonin and dopamine, has been associated both with antisocial behavior (Moffitt et al., 2002) and with reduced volume of brain structures, such as the amygdala and orbitofrontal cortex, important for emotion and self-regulation. These structures are known to be compromised in antisocial people. Future treatments for violent, antisocial behavior could therefore include drug therapy to regulate levels of MAOA activity.

In summary, the available biochemical, neuropsychological, and brain imaging data all point to areas of prefrontal cortex and limbic structures known to be important for self-regulation, impulse control, and processing of emotional information as important for regulating aggressive behavior. Moreover, considerable research in both humans and animals points to serotonin as a key neurotransmitter for this regulatory process, with low levels of serotonin reliably producing high levels of aggression.

VIII. What – if Anything – Can be Done to Reduce Aggression?

People don't have to learn how to behave aggressively -- it comes quite naturally. What people have to learn is how to control aggressive tendencies. Because aggression directly interferes with our basic needs of safety and security, it is important to find interventions that reduce it. The fact that there is no single cause for aggression makes it difficult to design effective interventions. A treatment that works for one person may not work for another. Indeed, some people (e.g., psychopaths) may not respond to any intervention. We don't want to sound pessimistic, but many people have started to accept the fact that aggression and violence may be an inevitable part of our society.

This being said, there certainly are interventions that can reduce aggression and violence. There are two important general points we would like to emphasize. First, successful interventions target as many causes of aggression as possible, and attempt to tackle them collectively. Interventions that are narrowly focused at removing a single cause of aggression, however well conducted, are likely to fail. Second, aggressive behavior problems are best

treated in childhood, when they are still malleable. It is much more difficult to alter aggressive behaviors when they are part of an adult personality than when they are still in development. Thus, interventions should target aggressive children before they grow up to become aggressive adolescents and adults. In this section we discuss some interventions that have been used to reduce aggression. Before we discuss the effective interventions, we first debunk two ineffective ones: catharsis and punishment.

a. Catharsis. The term catharsis dates back to Aristotle, who taught in *Poetics* that viewing tragic plays gave people emotional release from negative emotions such as pity and fear. In Greek drama, the heroes didn't just grow old and die of natural causes —they were often murdered. In modern times, Sigmund Freud revived the ancient concept of catharsis. Freud believed that if people repressed their negative emotions, they could develop psychological systems such as hysteria and neuroses (e.g., Breuer & Freud, 1893-1895). Freud's ideas are the foundation of the hydraulic model of anger, which suggests that frustrations lead to anger. Anger, in turn, builds up inside an individual like hydraulic pressure inside a closed circuit until it is vented. If the anger is not vented, the build-up of anger will presumably cause the individual to explode in an aggressive rage. People can presumably vent their anger by engaging in aggressive activities such as yelling, screaming, swearing, punching a pillow, throwing objects, tearing phone books, kicking trash cans, and slamming doors.

Almost as soon as researchers started testing catharsis theory, it ran into trouble. In one early experiment (Hornberger, 1959), participants who had been insulted by a confederate either pounded nails with a hammer for 10 minutes or did nothing. Next, all participants had a chance to criticize the confederate who had insulted them. According to catharsis theory, the act of pounding nails should reduce anger and subsequent aggression. However, the opposite was true: participants who pounded nails were *more* hostile toward the confederate afterward than were the participants who did nothing. Subsequent research has found similar results (e.g., Geen & Quanty, 1977). Other research has shown that venting doesn't reduce aggression even

among people who believe in the value of venting, and even among people who report feeling better after venting (Bushman, Baumeister, & Stack, 1999). Indeed, venting increases aggression, even against innocent bystanders (Bushman et al., 1999).

One variation of venting is physical exercise. Although physical exercise is good for your heart, it is not good for reducing anger (Bushman, 2002). Angry people are physiologically aroused, and physical exercise just keeps the arousal level high. To reduce anger, people should try to reduce their arousal level.

b. Punishment. Most cultures assume that punishment is an effective way to reduce aggression. *Punishment* is defined as inflicting pain (*positive punishment*) or removing pleasure (*negative punishment*) for a misdeed to reduce the likelihood that the punished individual would repeat the misdeed (or related misdeeds) in the future. Parents use it, organizations use it, and governments use it. But does it work? Today, aggression researchers think punishment does more harm than good. This is because punishment only temporarily suppresses aggression, and it has several undesirable side effects (Baron & Richardson, 1994; Berkowitz, 1993; Eron et al., 1971). Punishment models the behavior it seeks to prevent. For example, suppose a father sees an older brother beating up his younger brother. The father starts spanking the older boy while proclaiming, “I’ll teach you not to hit your little brother!” Yes, the father is indeed teaching the older boy something; he is teaching him that it is okay to behave aggressively as long as you are an authority figure. In addition, because punishment is aversive, it can classically condition children to avoid their parents, and in the short run can instigate retaliatory aggression. Longitudinal studies have shown that children who are physically punished by their parents at home are more aggressive outside the home, such as in school (e.g., Lefkowitz. Huesmann, & Eron, 1978).

c. Developing Nonaggressive Ways of Behaving. Most aggression treatment programs can be divided into one of two broad categories, depending upon whether aggression is viewed as proactive or reactive (Berkowitz, 1993, pp. 358-370). Recall that proactive

aggression is cold-blooded and is a means to some other end, whereas reactive-aggression is hot-blooded and is an end in itself.

d. Approaches to Reducing Proactive Aggression. People often resort to aggression because they think it is the easiest and fastest way to achieve their goals. Psychologists who view aggression as proactive behavior use *behavior modification* learning principles to teach aggressive people to use nonaggressive behaviors to achieve their goals, and it works (e.g., Patterson, Reid, Jones, & Conger, 1975). In behavior modification it is useful to replace an undesirable behavior with a desirable one. A major problem with punishment is that it does not teach the aggressor new, nonaggressive forms of behavior. One way to get rid of an undesirable behavior is to replace it with a desirable behavior (called *differential reinforcement of alternative behavior*). The idea is that by reinforcing nonaggressive behavior, aggressive behavior should decrease. Other effective programs include social skills training, where people are taught how to better read verbal and nonverbal behaviors in social interactions (e.g., Pepler, King, Craig, Byrd, & Bream, 1995). Exposure to prosocial role models also reduces aggression and increases helping (e.g., Spivey & Prentice-Dunn, 1990), even if the models are film or TV characters (for a meta-analytic review see Mares & Woodward, 2005).

e. Approaches to Reducing Reactive Aggression. Other approaches to reducing aggression focus on lessening emotional reactivity using relaxation and cognitive-behavioral techniques (for a meta-analytic review see DiGuiseppe & Tafrate, 2003). Most relaxation-based techniques involve deep breathing, visualizing peaceful images, or tightening and loosening muscle groups in succession. People practice relaxing after imaging or experiencing a provocative event. In this way, they learn to calm down after they have been provoked. Cognitive-based techniques focus on how a potentially provocative event is interpreted and how to respond to such events. For example, people rehearse statements in their mind such as: “Stay calm. Just continue to relax” and “You don’t need to prove yourself.” It is especially effective to combine relaxation and cognitive techniques (e.g., Novaco, 1975).

IX. Aggression Research Today and in the Future

We don't have a crystal ball, and predictions of the future can be hazardous. Indeed, in Dante's Inferno, futurists and fortune-tellers are consigned to the eighth circle of hell. Despite Dante's warning, we will make a few speculations. Social neuroscience is a hot topic today (see Heatherton & Wheatley, this volume), and will probably become even hotter in the future. The link between brain activity and human aggression is a promising area of current and future research, both in terms of understanding those brain structures that are implicated in aggressive responding (e.g., Weber et al., 2006) and in terms of the effects of internal and external triggers on neural responses and how these relate to aggression (e.g., Bartholow et al., 2006). A related area of work that holds considerable promise for vastly improving our ability to predict who will be violent under what circumstances is behavioral genetics. As briefly reviewed in a previous section, researchers are beginning to discover variations in the regulation of neurochemicals linked to aggression and violence, variations that ultimately have genetic causes and that can be targeted for pharmacological and behavioral interventions to reduce their influence on the expression of aggressive behavior (e.g., Seo et al., 2008). Another promising research direction is self-control. Aggression often starts when self-control stops (e.g., DeWall, Baumeister, Stillman, & Gailliot, 2007; Finkel, DeWall, Slotter, Oaten, & Foshee, in press). A third promising research direction is apology and forgiveness (e.g., McCullough, 2008). Hopefully social psychologists will be at the forefront, conducting research on these and other important topics that ultimately have the potential to make the world a less violent, more peaceful place.

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