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A Rat Model Examining Behavioral and Neurochemical Effects of Passive Exposure to Aggression on Observers

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LOYOLA UNIVERSITY CHICAGO

A RAT MODEL EXAMINING
BEHAVIORAL AND NEUROCHEMICAL EFFECTS OF
PASSIVE EXPOSURE TO AGGRESSION ON OBSERVERS

A DISSERTATION SUBMITTED TO
THE FACULTY OF THE GRADUATE SCHOOL
IN CANDIDACY FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

PROGRAM IN APPLIED SOCIAL PSYCHOLOGY

BY

HIDEO SUZUKI

CHICAGO, IL

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TABLE OF CONTENTS

ACKNOWLEDGEMENT	iii
LIST OF TABLES	vii
LIST OF FIGURES	ix
ABSTRACT	xi
CHAPTER ONE: INTRODUCTION	1
Psychological Studies on Exposure to Aggression and Their Limitations	2
Endocrinological Mechanism of Aggression	11
Mechanism of Neurotransmitter Systems in Relation to Aggression	15
Overall Experimental Design and Hypotheses	21
CHAPTER TWO: STUDY #1: BEHAVIORAL EFFECTS OF EXPOSURE TO AGGRESSION	31
Method	31
Results	38
Discussion	41
CHAPTER THREE: STUDY #2: FOLLOW-UP STUDY FOR BEHAVIORAL EFFECTS	53
Method	53
Results	56
Discussion	59
CHAPTER FOUR: STUDY #3: HORMONAL EFFECTS OF EXPOSURE TO AGGRESSION	70
Method	70
Results	74
Discussion	76
CHAPTER FIVE: STUDY #4: RECEPTOR DENSITIES IN RESPONSE TO EXPOSURE TO AGGRESSION	84
Method	84
Results	89
Discussion	91
CHAPTER SIX: GENERAL DISCUSSION	108
Psychological Implications of the Present Research	113
Neurobiological Implications of the Present Research	118
Limitations and Future Research	119
Conclusion	123

APPENDIX A: SOME EPISODES PRIOR TO THE PRESENT RESEARCH	124
REFERENCES	129
VITA	144

LIST OF TABLES

Table	Page
1. Summary of experimental design in study #1	29
2. Summary of experimental design in study #2	29
3. Summary of experimental design in study #3	30
4. Summary of experimental design in study #4	30
5. Mean aggression scores (in seconds) of observer rats and mean defense/submission scores (in seconds) of their opponent rats in priming exposure to non-aggression ($n = 30$), priming exposure to aggression ($n = 30$), acute exposure to non-aggression ($n = 18$), acute exposure to aggression ($n = 18$), chronic exposure to non-aggression ($n = 30$), and chronic exposure to aggression ($n = 30$)	48
6. Multivariate analysis of variance for aggression scores and defense scores between priming conditions and acute exposure conditions ($N = 96$)	49
7. Multivariate analysis of variance for aggression scores and defense scores between acute exposure conditions and chronic exposure conditions ($N = 96$)	49
8. Mean aggression scores (in seconds) of observer rats and mean defense/submission scores (in seconds) of their opponent rats 1 day and 16 days after exposure to aggression ($n = 30$) and non-aggression ($n = 30$)	64
9. Repeated-measures multivariate analysis of variance for aggression scores and defense scores 1 day and 16 days after exposure ($N = 60$)	65
10. Mean concentrations of serum testosterone (ng/dL) and corticosterone (ng/mL) across conditions	80
11. Multivariate analysis of variance for the concentrations of serum testosterone and corticosterone between single and chronic exposure conditions ($N = 30$)	80
12. Multivariate analysis of variance for the concentrations of serum testosterone and corticosterone between night time and morning time ($N = 78$)	81

13. Descriptive statistics of the relative densities of dopamine D ₂ receptors (<i>N</i> = 30)	97
14. Descriptive statistics of the relative densities of 5-HT _{1B} receptors (<i>N</i> = 30)	98
15. Two-way multivariate analysis of variance for the mean density values of dopamine D ₂ receptors (<i>N</i> = 30)	99
16. Two-way multivariate analysis of variance for the mean density values of 5-HT _{1B} receptors (<i>N</i> = 30)	101
17. Individual comparisons of the mean density values of dopamine D ₂ receptors (<i>N</i> = 30)	102
18. Individual comparisons of the mean density values of 5-HT _{1B} receptors (<i>N</i> = 30)	103

LIST OF FIGURES

Figure	Page
1. Mean aggression scores (+ <i>SE</i>) of observer rats in priming exposure to non-aggression ($n = 30$), priming exposure to aggression ($n = 30$), acute exposure to non-aggression ($n = 18$), acute exposure to aggression ($n = 18$), chronic exposure to non-aggression ($n = 30$), and chronic exposure to aggression ($n = 30$)	50
2. Mean aggression scores (+ <i>SE</i>) of observer rats in priming exposure to non-aggression ($n = 30$), priming exposure to aggression ($n = 30$), acute exposure to non-aggression ($n = 18$), acute exposure to aggression ($n = 18$), chronic exposure to non-aggression ($n = 30$), and chronic exposure to aggression ($n = 30$)	51
3. Representative behavioral patterns of observer rats and opponent rats (1 day after exposure to aggression/non-aggression)	52
4. Longitudinal comparisons of mean aggression levels (+ <i>SE</i>) of observer rats 1 day and 16 days after passive exposure to aggression ($n = 30$) and non-aggression ($n = 30$)	66
5. Longitudinal comparisons of mean defense/submission levels (+ <i>SE</i>) of opponent rats 1 day and 16 days after passive exposure to aggression ($n = 30$) and non-aggression ($n = 30$).	67
6. Relationship between the initial aggression scores and the follow-up aggression scores ($N = 60$)	68
7. Representative behavioral patterns of observer rats and opponent rats (16 days after exposure to aggression/non-aggression)	69
8. Mean comparisons in the concentrations of serum testosterone among single/chronic exposure to aggression/non-aggression ($N = 96$)	82
9. Mean comparisons in the concentrations of serum corticosterone among single/chronic exposure to aggression/non-aggression ($N = 96$)	83
10. Mean differences in the densities of dopamine D ₂ receptors in the anterior regions of the rat brain ($N = 30$)	104

11. Selected rat brain sections at the anterior regions	105
12. Mean differences in the densities of 5-HT _{1B} receptors in the posterior regions of the rat brain ($N = 30$)	106
13. Selected rat brain sections at the posterior regions	107

ABSTRACT

Previous studies have consistently reported that passive exposure to aggression is a risk of aggressive inclinations for a human witness. However, it is unclear whether a witness' aggressiveness is semi-permanently socialized or temporarily primed. Furthermore, a neurochemical mechanism of passive exposure to aggression also remains unaddressed in clinical literature. The present research used a rat model to clarify the behavioral and neurochemical effects of passive exposure to aggression. First, rats were screened for their aggressiveness after they were acutely or chronically exposed to aggression or non-aggression. It was found that observer rats chronically exposed to aggression exhibited more aggression than those exposed to non-aggression and even those exposed to aggression only acutely. This behavioral difference was maintained over 16 days. Next, radioimmunoassay and autoradiography were used to test the levels of serum testosterone and corticosterone, as well as the densities of dopamine D₂ receptors and 5-HT_{1B} receptors, among observer rats chronically exposed to aggression or non-aggression. No differences in the hormonal levels were detected between the groups of exposure to aggression and non-aggression, whereas observer rats chronically exposed to aggression showed lower densities of dopamine D₂ receptors and higher densities of 5-HT_{1B} receptors, compared with controls. These suggest that chronic passive exposure to aggression inclined observer rats to be aggressive in the long run, which may be mediated by low densities of dopamine D₂ receptors and high densities of 5-HT_{1B} receptors.

CHAPTER ONE

INTRODUCTION

Although aggression is a universal human phenomenon, aggression level varies across places. The geographic variations in aggression can be roughly seen by making cross-regional comparisons in violent crime rates. According to the United Nations surveys of crime trends in 1980-2000, Columbia and South Africa reported approximately 10 to 12 times higher average homicide rates (per 100,000 inhabitants) than the global average rate; by continent, the total crime rates (per 100,000 population) are steadily higher in North America and European Union than Latin America; and, the United States exclusively shows the highest crime rate among developed nations for recent two decades (Shaw, van Dijk, & Rhomberg, 2003). Within the United States, Western states have revealed the highest rate of aggravated assault since 1984, while murder and non-negligent manslaughter frequently occurs in Southern states during the past 35 years (Pastore & Maguire, 2006).

Why has violence consistently been more pervasive in the same certain areas than the others for a long time? These geographic differences in violent crime, which is an ultimate form of aggression, can be explained by numerous situational factors, such as the transition of modernization (e.g., LaFree & Drass, 2002; Shelley, 1981), population density (e.g., Griffitt & Veitch, 1971), climate (e.g., Anderson, Anderson, Dorr, DeNeve, & Flanagan, 2000; Bell, 2005; Bushman, Wang, & Anderson, 2005a & 2005b; Griffitt,

1970), unemployment rate and economic structure (e.g., Hepworth & West, 1988; Hovland & Sears, 1940; Vinokur, Price, & Caplan, 1996; Catalano, Dooley, Novaco, Wilson, & Hough, 1993), and culture (e.g., Cohen, Nisbett, Bowdle, & Schwarz, 1996; Fraczek, 1985; Fujihara, Kohyama, Andreu, & Ramirez, 1999; Nisbett, 1993; Ramirez et al., 2001; Souweidane & Huesmann, 1999). The present research focused on another possible factor that has been studied for a long time: a cross-regional variation in the degree of passive exposure to aggression.

Psychological Studies on Exposure to Aggression and Their Limitations

It is plausible that, in comparison with people living in “peaceful” areas, those living in “violent” areas could be inevitably exposed to aggression at a frequent rate through the mass media (e.g., violent TV programs), public spheres (e.g., riots, hate crime), and even private space (e.g., date rape, spouse abuse, child abuse). For example, the United States is known as one of the developed countries with a high crime rate (Shaw et al., 2003), whereby American people frequently witness violence in a passive form either indirectly or directly (e.g., Bell & Jenkins, 1993; Osofsky, 1995; Richters & Martinez, 1993). In this respect, Bandura’s (1973, 1977) social learning theory explains that passive exposure to aggression is a risk factor in developing aggressive tendencies among human witnesses because those witnessing aggression tend to imitate and/or learn aggressive behavior through observations.

To demonstrate the principle of social learning theory, Bandura, Ross, and Ross (1961) conducted the Bobo doll experiment where a child was passively exposed to either an aggressive or non-aggressive adult model in a playroom for 10 minutes. The aggressive adult model was engaged in both physical and verbal aggression against a

balloon tumbler doll (i.e., Bobo doll) in the room, whereas the non-aggressive model ignored the Bobo doll. Afterwards, the child was taken to and left alone in another playroom where he/she could find the same Bobo doll and other attractive toys. The child's aggressiveness was measured by his/her aggressive actions against the Bobo doll (e.g., physical, verbal) that an experimenter and school teacher rated on five-point scales. As a result, children who observed the aggressive model were more likely to show imitative physical and verbal aggression against the Bobo doll than those who observed the non-aggressive model. This imitative effect of exposure to aggression was replicated even when children watched the aggressive or non-aggressive adult model through a film (Bandura, Ross, & Ross, 1963).

Bandura et al.'s (1961, 1963) finding that passive exposure to aggression led a child observer to perform imitative aggressive behavior inspired the social psychology of aggression (e.g., Huesmann, Eron, Lefkowitz, & Walder, 1984) and sociological criminology (e.g., Akers, 1990; Akers, Krohn, Lonza-Kaduce, & Radosevich, 1979; Cheung & Ng, 1988). Both psychology and criminology have conducted survey research and investigated psychological consequences of passive exposure to violence in family, school, and community settings. As Bandura's (1973, 1977) social learning theory predicts, some studies reported that child abuse is related to whether a family lives in neighborhood with high crime rates or not (Chalk & King, 1998; Williamson, Bourdin, & Howe, 1991; Tolan, Gorman-Smith, & Henry, 2006). Other studies have particularly focused on children as a target population because they are considered as cognitively and emotionally vulnerable to traumatic violence (Margolin & Gordis, 2000). Those studies have documented that children who chronically witnessed violence tend to behave

violently (Guerra, Huesmann, Tolan, van Acker, & Eron, 1995; Onyskiw & Hayduk, 2001), show child abuse later in life (Widom, 1989), and develop social scripts justifying aggression (Guerra, Huesmann, & Spindler, 2003). Some scholars (e.g., Heise, 1998; Widom, 2000) reasoned that a child's passive observation of aggression might let him/her to accept aggressive manners as a legitimate, appropriate, and/or tactic means in social relationships.

Therefore, previous psychological research, including Bandura et al.'s experiments (1961, 1963) and survey research, has consistently showed that there is a relationship between passive exposure to aggression and the development of observer's aggressive behavior. Nevertheless, this inference needs to be interpreted with caution because of some methodological limitations.

First, survey research often fails to exclude extraneous factors, thus it is uncertain whether an observer's aggressiveness results from a pure source of passive exposure to violence or the combination of it and other risk factors (Margolin & Gordis, 2000). For instance, Osofsky (1995) argues that parents exposed to community violence are likely to experience helpless with their parental control (i.e., perceived inability to protect their child from community violence). Consequently, these parents tend to exhibit authoritarian control, which may be ultimately child abuse. In other words, Osofsky suggests that the relationship between passive exposure to community violence and children's aggressiveness is mediated by parental control and victimization of child abuse.

Chronic exposure to community violence also leads to a high level of arousal and frustration among passive witnesses. According to Zillmann's (1983a, 1983b) excitation transfer theory, these witnesses can misattribute the actual source of their arousal (i.e.,

community violence) to related or unrelated events. For example, in the case of child abuse, parent's arousal and frustration, induced by community violence, might be directed at their child; in the case of bullying at school, a child might transfer their arousal into frustrations with a peer. In these cases, arousal and frustration are combined with a factor of passive exposure to community violence, which might together result in aggression.

In fact, evidence suggests some variables that are confounded with passive exposure to aggression. Among children at social service agency, stress due to isolation from family plays an additional risk factor for their aggressiveness (Egeland, 1997; Lynch & Cicchetti, 1998; Wolfe & McGee, 1994). Heath, Kruttschnitt, and Ward (1986) found that, without the experiences of parental abuse, high exposure to violent (and non-violent) television, which is a special form of passive exposure to aggression, was not associated with a viewer's violent behavior. If Heath et al.'s (1986) finding is applicable to a general form of passive exposure to aggression, the experiences of active involvement in aggression are necessary as a mediating variable to establish the link between passive exposure to aggression and a witness' aggressiveness. In addition to active involvement in aggression, people who are repeatedly exposed to violence are often confronted with poverty, lack of nutrition and medical care, overcrowding, substance abuse, and parents' unemployment and psychopathology (Garmezy & Masten, 1994; Smith & Thornberry, 1995; Vig, 1996).

Therefore, many extraneous factors are potentially confounded with a variable of passive exposure to aggression. However, survey research has difficulty in excluding

these redundant factors. Because of this methodological limitation, it is unclear whether or not passive exposure to aggression solely escalates a witness' aggressive behavior.

The second methodological problem is that survey research relies on subjective self-reports as the measures of aggression (Margolin & Gordis, 2000). That is, objective measures are rarely used to assess how frequently/severely a respondent is exposed to aggressive situations and how aggressive he/she is. Although the validity of self-reported measures of aggression has been controversial, it is often threatened by various forms of biases that are affected by difficulty in remembering, social desirability, and/or ambiguity in understanding of aggression (McCloskey & Coccaro, 2003). Furthermore, there could be an individual variation in the perception of and sensitivity to aggression such that actual aggressive situations around an observer and aggressiveness of the observer might be over-described or under-described by a given respondent. Because of these potential biases, the assessments of aggression by survey are sometimes doubted in terms of their validity and utility.

The third methodological problem is that survey research often uses cross-sectional design or longitudinal design, in which it is difficult to make a causal inference between passive exposure to aggression and an observer's aggressive behavior (Margolin & Gordis, 2000). Moreover, each of these designs has its unique limitation. That is, cross-sectional design cannot assess any changes within the same individuals while longitudinal design faces with a problem of attrition.

In contrast, the experimental method is capable of controlling extraneous variables, measuring aggression objectively, and making a causal inference. In Bandura et al.'s (1961, 1963) Bobo doll experiment, dispositional aggressiveness (i.e., physical,

verbal, and object aggression, aggression inhibition) of the child participants was pre-screened so that there was a homogeneous in dispositional aggressiveness between the experimental group (i.e., exposure to the aggressive model) and control group (i.e., exposure to the non-aggressive model). In addition, all experimental sessions were administered under the same conditions. These strategies helped to reduce potential extraneous variables except for the manipulative variable of passive exposure to the aggressive/non-aggressive model. Bandura et al.'s experiment also measured imitative aggression of the participants objectively; it was based on the ratings given by two third people (i.e., an experimenter and a school teacher). Finally, since the participants' imitative aggression was assessed after they were passively exposed to the model, it could be logically inferred that passive exposure to aggression caused imitative aggression among the child observers.

In spite of these methodological advantages of Bandura et al.'s (1961, 1963) experiment, there were still two unaddressed issues. First, their finding suggests that an observer who is exposed to aggression shows aggression immediately, but a question remains whether or not this observer still behaves aggressively as time passes by. To clarify this question, a follow-up assessment of an observer's aggressiveness is needed, yet Bandura et al.'s experiment did not administer it. As discussed earlier, survey research has reported that high exposure to aggression predicts high level of aggression among a child witness (e.g., Guerra et al., 2003; Guerra et al., 1995; Onyskiw & Hayduk, 2001; Widom, 1989), thus the effect of passive exposure to aggression seems to be long-lasting. But, survey research has difficulty in examining whether passive exposure to aggression has a short-term or long-term effect on an observer's aggressive behavior

exclusively, with controlling all other potential risk factors that may intervene in an observer's aggressiveness between the initial time point (e.g., right after passive exposure to aggression) and following time points (e.g., days/months/years after passive exposure to aggression). Therefore, both survey research and Bandura et al.'s experiment have not clearly answered the length of the effect of passive exposure to aggression.

Second, Bandura et al. (1961, 1963) found that a child witness showed imitative aggression by only one-time exposure to aggression, but they did not extensively examine the effect of chronic exposure to aggression on a child witness. According to Huesmann and Kirwil (2007), there are two psychological processes underlying a mechanism of passive exposure to aggression: the priming/imitative process and the learning/socializing process (also see Huesmann, 1988). A single exposure to aggression, as manipulated by Bandura et al., influences aggressiveness of a witness en route to the priming/imitative process, whereas the learning/socializing process requires repeated observations. In other words, Bandura et al.'s findings probably indicate the priming or imitative effect of a single exposure to aggression, rather than the learning/socializing effect of chronic exposure to aggression.

The priming/imitative process was originally proposed by Berkowitz's (1990, 1998) cognitive neoassociationism. According to this theory, aggression cues exposing to a passive observer induce rudimentary negative affects and then activate aggression-related thoughts in the observer's mind. Subsequently, these activated thoughts become more accessible and ready to be attributed to the negative affects. Thus, anger is more accessible than any other negative feelings and increases the likelihood that the observer chooses aggressive response in a timely manner. But, after situational aggression cues

are removed, priming/imitative effect is weakened. In this way, the priming/imitative effect is short-term and requires automatic retrieval process (Huesmann, 1988; Huesmann & Kirwil, 2007).

To the contrary, the learning/socializing process is a more complex acquisition process where cognition and emotional desensitization interplay (Huesmann, 1988; Huesmann & Kirwil, 2007). When the learning/socializing process occurs, a passive observer incorporates aggressive response into their behavioral repertoire of social scripts through repeated observations. These aggressive scripts eventually become more generalized and serve as cognitive guides to plan future behavior. This may encourage the observer to develop aggressive schema/hostile attributional bias (Dill, Anderson, Anderson, & Deuser, 1997; Dodge, 1980; Dodge & Coie, 1987; Dodge & Frame, 1982; Dodge, Price, Bachorowski, & Newman, 1990; Dodge & Tomlin, 1987; Graham & Hudley, 1994; Nasby, Hayden, & DePaulo, 1979; Slaby & Guerra, 1988; Steinberg & Dodge, 1983), normative beliefs about aggression (Guerra et al., 1995; Huesmann & Guerra, 1997), positive attitudes toward aggression (Bookwala, Frieze, Smith, & Ryan, 1992; Kingery, 1998; Markowitz, 2001), and justification of aggression (Azar & Rohrbeck, 1986; Hyman, 1995), of which all contribute to the readiness for aggressive behavior (for review, see Anderson & Huesmann, 2003). Repeated observations of aggression also leads the passive observer to become less sensitive to empathy toward a victim (i.e., emotional desensitization), thus his/her aggressive tendencies are further accelerated. Compared with the priming/imitative process, the learning/socializing process requires conscious retrieval process of knowledge that is constructed based on repeated experiences/observations (Huesmann & Kirwil, 2007). Moreover, the

learning/socializing effect is long-term; once aggressive behavior is learned through repeated observations, it is difficult to modify it.

Bandura et al. (1961, 1963) provided evidence for the imitative effect, rather than the learning/socializing effect, of passive exposure to aggression because they tested 'imitative' aggression as a result of a 'single' exposure to aggression. On the other hand, survey studies have supported the possible effect of chronic exposure to aggression on an observer's aggressiveness (e.g., Guerra et al., 1995, 2003; Onyskiw & Hayduk, 2001; Widom, 1989), but some methodological issues (i.e., confounding variables, a lack of objectivity, difficulty in making a causal inference) confront these studies. Accordingly, it is necessary to experimentally examine the learning/socializing effect of chronic passive exposure to aggression, which is psychologically an independent process of the imitative process (Huesmann & Kirwil, 2007). Furthermore, analyzing this research topic is socially significant because, in reality, aggressive situations occur repeatedly, rather than only one time, through everyday life (e.g., Bell & Jenkins, 1993; Osofsky, 1995; Richters & Martinez, 1993).

In short, previous psychological studies have consistently suggested that passive exposure to aggression contributes to the development of an observer's aggressive tendencies. Nevertheless, these studies are faced with some methodological problems, that is, confounding variables, validity of self-reported measures of aggression, difficulty of causal inference, a lack of follow-up studies of aggression over time, and/or unclear distinction between the effect of a single exposure (through the priming/imitative process) and one of chronic exposure (through the learning/socializing process). To fully understand the potential risk of passive exposure to aggression, it is important to clarify

whether or not passive exposure to aggression exclusively has a causal link to aggressive behavior of an observer through the priming/imitative process and/or the learning/socializing process.

Endocrinological Mechanism of Aggression

The other uncertain part in the mechanism of passive exposure to aggression is how a passive observer physiologically/neurochemically responds to aggression around him/her, which may, in turn, lead him/her to behave aggressively. For instance, testosterone and corticosterone have been focused as their prominent roles of aggression/subordination (Van Goozen, 2005). Some rat studies showed that the surgical castration of testosterone result in reducing aggression, suggesting that higher levels of testosterone is associated with higher levels of aggression (Albert, Walsh, Gorzalka, Siemens, & Louie, 1986; Giammanco, Tabacchi, Giammanco, Di Majo, & La Guardia, 2005). Consistent with these findings, the administration of testosterone escalated aggression among both male and female rats (Lumia, Thorner, & McGinnis, 1994; Giammanco et al., 2005). Some studies using human subjects also revealed that high levels of testosterone was obtained among individuals with antisocial personality/alcoholism (Aromaki, Lindman, & Eriksson, 1999; Dabbs, Hopper, & Jurkovic, 1990; Dabbs & Morris, 1990; Lindman, Jarvinen, & Vidjeskog, 1987), young men with high behavioral disinhibition scores (Daitzman & Zuckerman, 1980), impulsive young women (Bjork, Moeller, Dougherty, & Swann, 2001), and prisoners with a history of violent crimes (Dabbs, Frady, Carr, & Besch, 1987; Kreuz & Rose, 1972; Kreuz, Rose, & Jennings, 1972). Therefore, there seems to be a positive association between the levels of testosterone and aggression.

The levels of testosterone can be elevated by some specific contexts (Archer, 2006). For example, the levels of testosterone increase when birds are faced with aggressive situations (e.g., territory formation, dominance disputes, mate-guarding; Wingfield et al., 2000), when male rats become dominant (Hardy et al., 2002; Tamashiro, Nguyen, & Sakai, 2005), when humans win in sport competition (Archer, 2006), and when southerners (but not northerners) in the U.S. are insulted (Cohen et al., 1996). These suggest that there may be a relationship between any of these situational factors and aggression, mediated by the levels of testosterone.

The other key hormone in relation to aggression is glucocorticoids, especially corticosterone or cortisol, which are known as stress hormones. Some psychologists believe that an increase in arousal levels is associated with aggression (e.g., Berkowitz, 1993; Geen & O'Neal, 1969; Zillmann, 1979, 1983a, 1983b). If the high levels of arousal are reflected by the high corticosterone levels, it is reasonable that aggression is promoted by the high levels of corticosterone. A rat study conducted by Mikics, Kruk, and Haller (2004) actually found that high dose of corticosterone enhanced aggressive behavior while metyrapone (i.e., corticosterone synthesis inhibitor) reduced aggression.

Mikics et al. (2004) additionally suggest that their findings demonstrated non-genomic effects of corticosterone on aggression. According to their hypothesis, a potentially dangerous situation acutely increases glucocorticoids, which subsequently escalates aggressive behavior through non-genomic mechanisms (within 7 min.). Simultaneously, the non-genomic mechanisms determine second messengers so that genomic mechanisms (i.e., the hypothalamic-pituitary-adrenal, HPA, axis) eventually overtake a role in escalating aggressive behavior. When the HPA axis is continuously

activated, an inhibitory feedback occurs to decrease corticosterone and inhibit aggressive behavior. As a result, acute administration of corticosterone escalates aggression because of non-genomic effects (Mikics et al., 2004), whereas chronic stress and chronic administration of corticosterone conversely reduces aggressive behavior (Politch & Leshner, 1977; Leshner, Korn, Mixon, Rosenthal, & Besser, 1980) probably because of an inhibitory feedback in the HPA axis (i.e., genomic effects).

Those who suggest a positive association between corticosterone and aggression assume that the levels of corticosterone are a physiological indicator of frustration. In contrast, some studies rather found a negative association between corticosterone and aggression, and they reasoned that the levels of corticosterone indicate the degree of fear. For instance, the low levels of plasma glucocorticoids have been found among children with conduct disorder (Kariyawasam, Zaw, & Handley, 2002; McBurnett, Lahey, Rathouz, & Loeber, 2000; Pajer, Gardner, Rubin, Perel, & Neal, 2001; van Goozen et al., 1998; Vanyukov et al., 1993), adults with antisocial personality disorder (Dolan, Anderson, & Deakin, 2001; Virkkunen, 1985), and violent alcoholics (Bergman & Brismar, 1994), suggesting that those disordered individuals may show risk-taking behavior (e.g., aggression) without fear. In previous endocrinological experiments, male dominant rats showed lower levels of corticosterone than male subordinate rats although corticosterone levels for both rats were higher than the baseline (Blanchard, Sakai, McEwen, Weiss, & Blanchard, 1993; Blanchard et al., 1995; Tamashiro et al., 2005). Hardy et al. (2002) found that both dominant and subordinate rats showed similar levels of corticosterone on the fourth day after mixed-sex group housing, but only subordinate rats kept increasing their corticosterone levels on the seventh day; the levels of

corticosterone among dominant rats returned to the basal levels on the seventh day.

Given the fact that subordinate rats show high corticosterone levels and low aggressiveness, these animal studies support evidence for a negative association between corticosterone and aggression. Furthermore, some psychologists believe that low arousal is associated with aggression (for a review, see Anderson & Huesmann, 2003).

Combined with the two contradictory views of the corticosterone-aggression association, Haller and Kruk (2006) hypothesize that both high and low glucocorticoid levels are associated with aggression, depending on a type of aggressive behavior. Hyperarousal or high levels of glucocorticoids may be related to excessive emotional aggression (e.g., fear, anger), characterized by post-traumatic stress disorder, intermittent explosive disorder, and depression. In contrast, hypoarousal or chronically low levels of glucocorticoids may contribute to general and habitual aggression via brain changes, such as some types of post-traumatic stress disorder, schizophrenia, attention deficit/hyperactivity disorder, antisocial personality disorder, and drug abuse.

Therefore, testosterone-aggression associations are positive while corticosterone-aggression associations are either positive or negative, depending on types of aggressive behavior. High levels of corticosterone may lead to affective aggression, but chronically low levels of corticosterone may result in pathological aggression. However, to my knowledge, no studies have investigated the hormonal effects of passive exposure to aggression; all studies discussed in the above demonstrated how active involvement in aggression changes the levels of testosterone and corticosterone.

Mechanism of Neurotransmitter Systems in Relation to Aggression

Van Goozen (2005) argues that the hormone-aggression associations are often weak or none in primates and humans. Her statement implies that aggressive behavior is influenced by not only the hormonal roles but also other neurobiological functions, such as the roles of dopaminergic neurotransmitter systems (Tamashiro et al., 2005) and/or serotonergic systems (Kim & Haller, 2007; Haller & Kruk, 2006). Thus, neurobiological research on aggression also focuses on dopaminergic and serotonergic functions.

Interestingly, Welch and Welch (1971) examined the effects of passive exposure to aggression on brain amines, specifically serotonin (5-hydroxytryptamine or 5-HT) and noradrenaline, of observer mice. In their experiment, some 18-week pre-isolated mice were placed close to (within a few feet away from) a cage of other fighting mice (i.e., experimental group), whereas the other 18-week pre-isolated mice were not given any passive exposure to fighting (control group). Welch and Welch found that mice which heard the fighting for 1 hour increased the concentration of 5-HT and noradrenaline in the whole brain, compared to the control group.

Nevertheless, Welch and Welch did (1) sample from the population of pre-isolated mice (which were hypersensitive to stress), but not healthy mice, (2) not screen aggressiveness of the observer mice, and (3) test the change in the neurotransmitters in response to one-time exposure to fighting for 1 hour, but not repeated chronic exposure to fighting. Accordingly, Welch and Welch's finding did not fully describe how passive exposure to aggression, especially in a chronic form, influences the neurotransmitter system that is related to aggressiveness of observers when the 18-week isolation effect is excluded. To my knowledge, only Welch and Welch's study partly focused on the

effects of passive exposure to aggression; no other studies examine about the neurochemical changes determined by passive exposure to aggression.

However, numerous studies have investigated the neurochemical roles in relation to aggression (e.g., Caramaschi, de Boer, & Koolhaas, 2007; Fish, Faccidomo, & Miczek, 1999; Fish, Faccidomo, DeBold, & Miczek, 2001; Miczek, 1974). To the contrary to Welch and Welch's (1971) finding that the increase in the concentration of 5-HT was observed, those studies, including both human and animal studies, have consistently reported that aggression is associated with the low levels of 5-HT (for a review, see Anderson & Huesmann, 2003; Nelson & Trainor, 2007; Pihl & Benkelfat, 2005). Aggressive behavior is also associated with other neurotransmitters, such as dopamine, noradrenaline (also known as norepinephrine), and gamma-aminobutyric acid (GABA).

Based on these neurochemical studies on aggression, Blum, Cull, Braverman, and Comings (1996) suggest their theory of reward deficiency syndrome. According to their theory, the low levels of 5-HT in the hypothalamus inhibit a release of opioid peptide enkephalin, which allows a release of GABA in the ventral tegmental area. Because GABA plays an inhibitory role to release dopamine in the ventral tegmental area, the excessive release of GABA results in low levels of dopamine in the nucleus accumbens, hippocampus, and amygdala. Consequently, the low levels of dopamine, corresponding to the low levels of 5-HT at the first phase, lead an individual to experience unpleasant emotions, called 'reward deficiency.' To relieve such unpleasant emotions, he/she is motivated to seek addictive, impulsive, and compulsive behavior that can temporarily allow the release of dopamine.

As supportive evidence for the hypothesis of reward deficiency syndrome, alcohol, cocaine, and nicotine promote the release of dopamine temporarily (Blum et al., 1996). More interestingly, an *in vivo* microdialysis study indicated that, during 60 minutes after a fight, aggressive rats showed the high levels of the extracellular concentration of dopamine in the nucleus accumbens, especially the shells of the nucleus accumbens, and prefrontal cortex, compared with the baseline (Van Erp & Miczek, 2000). In contrast, the levels of dopamine remained baseline before and during the incident of a fight. These results imply that the outcome of aggression promptly promotes the release of dopamine. Because substance abuse and aggression can meet the needs of an individual who has a lack of dopamine because of reward deficiency syndrome, low dopaminergic neurotransmissions are considered as neurochemical conditions that potentially trigger aggression.

Blum et al. (1996) further argue that the low densities of dopamine D₂ receptors in the pathway (e.g., the nucleus accumbens, hippocampus, amygdala) also lead to impulsive behavior because the low densities of dopamine D₂ receptors inhibit the neurotransmissions of dopamine. As evidence, White, Morris, Lawford, and Young's (2008) study using human subjects found that young adults with the A1 allele (of the *TaqIA* polymorphism (rs1800497) in the *ANKK1* gene, that is, A1A1 and A1A2 genotypes) showed higher impulsivity on cognitive tasks than those without the A1 allele. Because it has been reported that the A1 allele is related to the reduction of the densities of dopamine D₂ receptor in the striatum and other brain areas (e.g., Jonsson et al., 1999; Noble, Blum, Ritchie, Montgomery, & Sheridan, 1991; Thompson et al., 1997;

Pohjalainen et al., 1998), White et al.'s (2008) study indirectly indicates the association between impulsivity and the low densities of dopamine D₂ receptors.

However, there are some neuropharmacological findings that are contradictory to Blum et al.'s (1996) hypothesis. For instance, studies using human subjects have reported that dopamine D₂ receptor antagonists (e.g., haloperidol, raclopride) are used to treat psychotic aggression disorder, whereas dopamine D₂ receptor agonists (e.g., quinpirole) lead to aggressive behavior (De Almeida, Ferrari, Parmigiani, & Miczek, 2005; Miczek, Fish, de Bold, & de Almeida, 2002).

Therefore, Spont (1992) suggests an alternative hypothesis of aggression in relation to the roles of serotonin-dopamine interaction. According to him, 5-HT plays a role of modulating the signal-to-noise ratio in neural activity within fight/flight system within the septum and periaqueductal gray of the brain. To control aggressive behavior, 5-HT inhibits dopaminergic neurotransmissions, which innervate more than 20 different motivational structures in the fight/flight system (also see Pihl & Benkelfat, 2005). If 5-HT is released at the low levels, an excessive amount of dopamine is released, propagating redundant motivational and motor systems, including food intake, locomotor activity, sexual behavior, and aggression. This might also imply that the pharmacological agonizing effects on the activation of dopamine D₂ receptors (i.e., effects of dopamine D₂ receptors agonists) lead to aggression because the activation of the receptors becomes more sensitive to dopaminergic neurotransmissions. Likewise, the dopamine D₂ receptor antagonists reduce aggression because dopamine D₂ receptors become deactivated and insensitive to dopaminergic neurotransmissions. Therefore, Spont's hypothesis argues that excessive dopaminergic neurotransmissions or the activation of dopamine D₂

receptors contribute to aggression, which is consistent with previous pharmacological findings but contradictory to Blum et al.'s (1996) hypothesis.

Recent studies show that the relationship between 5-HT and dopamine is more complicated (Esposito, Di Matteo, & Di Giovanni, 2008). Whether 5-HT activates or inhibits the neurotransmissions of dopamine depends on a subtype of 5-HT receptor. As Blum et al. (1996) expects, the activation of 5-HT_{2C} receptors indirectly inhibits the release of dopamine, mediated by GABA-nergic neurons. In contrast, as Spooont (1992) argues, the activation of 5-HT_{1A}, 5-HT_{1B}, 5-HT_{2A}, 5-HT₃, and 5-HT₄ receptors directly excites dopaminergic neurons (also see Di Matteo, Di Giovanni, Pierucci, & Esposito, 2008). Thus, studies on aggression need to examine not only dopamine D₂ receptors but also 5-HT receptors to understand serotonin-dopamine interaction that contribute to aggression.

Among these 5-HT subtypes, 5-HT_{1A} and 5-HT_{1B} receptors play an important role in serotonergic system of impulsive aggression. It has been found that both 5-HT_{1A} and 5-HT_{1B} receptor agonists inhibit aggressive behavior among mice/rodents (Chiavegatto et al., 2001) and humans (Cleare & Bond, 2000; also see Miczek et al., 2002; Miczek, Maxson, Fish, & Faccidomo, 2001; Nelson & Trainor, 2007 for a review). In addition, the knockout mice lacking the gene for neuronal nitric oxide synthase (nNOS, an enzyme necessary for the functions of 5-HT_{1A} and 5-HT_{1B} receptors; Chiavegatto et al., 2001) and the knockout mice lacking 5-HT_{1B} receptors (Saudou et al., 1994) show aggression (Chiavegatto et al., 2001). Ferris et al. (1997) found that the microinjection of fluoxetine (i.e., selective 5-HT reuptake inhibitor) in the anterior hypothalamus, where both 5-HT_{1A}

and 5-HT_{1B} receptors are concentrated, inhibited aggression. All of these studies indicate a negative association between aggression and the activation of 5-HT_{1A}/5-HT_{1B} receptors.

However, an association between aggression and the activation of 5-HT_{1A}/5-HT_{1B} receptors may be bidirectional, depending on whether these receptors act postsynaptically or presynaptically. Based on the fact that aggression is expected to be relevant to the low levels of 5-HT (for a review, see Anderson & Huesmann, 2003; Nelson & Trainor, 2007; Pihl & Benkelfat, 2005), aggression can result in the deactivation of 5-HT_{1A}/5-HT_{1B} postsynaptic receptors (i.e., low ability to detect the release of 5-HT from presynaptic neurons), which is consistent with the above studies. In addition, it is also suggested that there are also 5-HT_{1A}/5-HT_{1B} autoreceptors that function as inhibit the levels of 5-HT presynaptically (Sharp, Bramwell, & Grahame-Smith, 1989). For example, Caramaschi et al. (2007) found that aggressive mice showed low serotonergic neurotransmissions and enhanced 5-HT_{1A} autoreceptor activity, suggesting a positive association between aggression and the activation of 5-HT_{1A} inhibitory autoreceptors. Given some findings that low serotonin is associated with not only aggression but also excessive food intake (In fact, both Blum et al. (1996) and Spont (1992) attempt to explain a neural mechanism of obesity by using their hypotheses of ‘serotonin deficiency’), Park, Harrold, Widdowson, and Williams (1999) showed that the densities of 5-HT_{1B} receptors increased in diet-induced obese rats. Thus, Miczek et al. (2007) suggest that somatodendritic 5-HT_{1A} and 5-HT_{1B} inhibitory autoreceptors may be upregulated among highly aggressive rats.

In summary, dopamine D₂ receptors, 5-HT_{1A} receptors, and 5-HT_{1B} receptors play key roles of modulating aggressive behavior. Nevertheless, whether aggression is

associated with low or high densities of each of these receptors still remains unclear, and it may depend on types of receptors (presynaptic vs. postsynaptic), types of aggression, or any other variables. Furthermore, it is still unknown how passive exposure to aggression may influence the densities of those receptors in relation to aggressiveness of observers.

Overall Experimental Design and Hypotheses

Previous psychological studies have found that a passive witness generally tends to show aggression if he or she is exposed to aggression. Because of some methodological limitations, however, some issues on the behavioral effects of passive exposure to aggression still remain questioned. Specifically, it is unclear whether chronic passive exposure to aggression solely predisposes a passive observer to be aggressive through the learning/socializing process and, if it is true, whether this behavioral change lasts permanently or only temporarily. In addition, hormonal and neural mechanisms of passive exposure to aggression have not been sufficiently discussed in clinical literature. Therefore, the main objectives of the present research were to test (1) aggressiveness of an observer over time across various conditions, (2) hormonal changes, and (3) the alternation of the neurotransmitter systems in response to passive exposure to aggression.

Since previous survey research has difficulty in controlling extraneous factors, assessing aggression objectively, and making a causal inference on the link between passive exposure to aggression and an observer's aggressiveness, the present research used an experimental approach, specifically, a rat model, so that these limitations could be minimal. A rat model enables investigators (1) to strictly deal with extraneous situational factors during their experiments, (2) to objectively and directly measure both severity/frequency of exposure to aggression, as well as aggressive behavior of observing

rats, (3) to follow up aggressiveness of rat subjects in a short time span, (4) to infer a causal relationship between exposure to aggression and aggressiveness of observing rats, (5) to easily create actual aggressive situations around an observing rat, which are more realistic than a simulated aggressive situation by using a Bobo doll, and (6) to conduct neurochemical experiments which it is difficult to run in human studies. Although it is controversial about the validity of an animal model for understanding human phenomenon, there are similarities in many aspects of behavioral responses and neurochemical activities during aggression between humans and non-humans (Nelson & Trainor, 2007; Olivier & Young, 2002; Tamashiro et al., 2005). Thus, a rat model provided valuable implications of possible behavioral/neurochemical changes that are relevant to motivational systems of aggressive behavior in humans, in response to passive exposure to aggression.

The present research consisted of four studies. Study #1 compared aggressiveness of rat subjects, called ‘observer rats,’ among six different conditions: (1) priming, (2) acute, and (3) chronic exposure to aggression and non-aggression (see Table 1). In the priming conditions, observer rats were screened their aggressiveness as soon as they were passively exposed to either aggression or non-aggression only one time. If a difference in aggressiveness was noticed between the priming groups, it would suggest that aggression of observer rats was likely to be primed by a single exposure to aggression (i.e., the priming effect of passive exposure to aggression).

In the acute exposure conditions, observer rats were screened their aggressiveness 24 hours after a single, 1-day passive exposure to aggression or non-aggression. Because there was a time lag between exposure part and screening part, the priming effect was

assumed to be eliminated in the acute exposure conditions. Rather, if the learning/socializing effects would overtake the priming effects. Thus, if there was a difference in aggressiveness between the acute exposure groups, it would suggest that passive observer rats learned aggression by only a single exposure to aggression (i.e., the learning/socializing effect of acute exposure to aggression).

Finally, observer rats in the chronic exposure conditions were screened their aggressiveness 24 hours after 23-day consecutive exposure to aggression or non-aggression. Again, there was a time lag in these conditions to reduce the potential priming effect. In addition, observer rats in the chronic exposure groups were repeatedly exposed to aggressive situations in a passive form, compared to those in the priming/acute exposure groups. It was assumed that 23 days were sufficiently chronic to influence behavior on the grounds of the empirical findings that gene expression changed as a result of chronically active involvement in aggression for 25 days among mice (Feldker, et al., 2006). Therefore, roughly speaking, 23 days seemed to be enough time to determine gene expression programming neural systems and, subsequently, changing behavioral responses resulting from passive exposure to aggression. If a difference in aggressiveness between the chronic exposure conditions were seen, it would suggest the learning/socializing effect of chronic exposure to aggression, with ruling out the potential priming effect.

Here are brief descriptions of the six different conditions in Study #1:

1. Priming exposure to aggression – Observer rats were passively exposed to an *aggressive* situation for *1 day* and screened on their aggressiveness *immediately*.

2. Priming exposure to non-aggression – Observer rats were passively exposed to a *non-aggressive* situation for *1 day* and screened on their aggressiveness *immediately*.
3. Acute exposure to aggression – Observer rats were passively exposed to an *aggressive* situation for *1 day* and screened on their aggressiveness *24 hours* later.
4. Acute exposure to non-aggression – Observer rats were passively exposed to a *non-aggressive* situation for *1 day* and screened on their aggressiveness *24 hours* later.
5. Chronic exposure to aggression – Observer rats had been passively exposed to *aggressive* situations for *23 days* and screened on their aggressiveness *24 hours* later.
6. Chronic exposure to non-aggression – Observer rats had been passively exposed to *non-aggressive* situations for *23 days* and screened on their aggressiveness *24 hours* later.

It was hypothesized that passive observer rats in the group of chronic exposure to aggression would be the most aggressive in any other groups because chronic exposure to aggression was expected to socialize the observer rats to be aggressive (Huesmann & Kirwil, 2007). This hypothesis was also based on my preliminary study which compared aggressiveness of observer rats in the six conditions: priming/acute/chronic exposure to aggression/non-aggression ($N = 36$; the method to measure aggressiveness was discussed in Chapter 2). One-way ANOVA showed that there was a significant difference in the amount of aggressive behavior (in sec) among six conditions ($F(5, 30) = 6.281, p < .01$),

and Bonferroni-typed a posteriori comparisons indicated that only the group of chronic exposure to aggression ($\bar{X} = 100.3$ sec.) showed significantly higher mean amount of aggressive behavior than any other groups ($p < .01$ for all comparisons) while no other differences were not significant ($\bar{X} = 12.0$ sec. for the group of priming exposure to non-aggression; $\bar{X} = 23.6$ sec. for the group of priming exposure to aggression; $\bar{X} = 13.2$ sec. for the group of acute exposure to non-aggression; $\bar{X} = 15.6$ sec. for the group of acute exposure to aggression; and $\bar{X} = 22.5$ sec. for the group of chronic exposure to non-aggression).

Expecting that only chronic exposure to aggression is associated with aggressiveness of observer rats (as shown by my preliminary study), Study #2 further tested another hypothesis that the observer rats in the group of chronic exposure to aggression maintained their aggressiveness in the long run. In this study, aggressiveness of observer rats in the chronic exposure conditions were assessed at two time points – 1 day and 16 days (i.e., about a half month) after their last exposure (see Table 2). It was hypothesized that their aggressiveness would not change across times, implying that the observer rats chronically exposed to aggression internalized aggressive behavior as their own behavioral repertoire through learning processes (Huesmann & Kirwil, 2007).

Study #3 analyzed the levels of serum testosterone and corticosterone of observer rats in response to passive exposure to aggression. The purpose of Study #3 was to examine if chronic passive exposure to aggression changed the levels of these hormones in relation to aggression, compared to controls (see Table 3). Blood samples, containing serum hormones, were collected from observer rats immediately after a single exposure

session (corresponding to both priming and acute exposure conditions) or chronic exposure sessions at night time. For the chronic exposure conditions, the blood samples were also collected at morning time, approximately 15 hours after exposure sessions, because hormonal levels are fluctuated in a daily cycle where the levels are peaked at evening/night time and at the bottom at morning time in nocturnal rats (Atkinson, Wood, Kershaw, Bate, & Lightman, 2006; Leal & Moreira, 1997). That is, the daily variation of hormones was assessed at two time points – night time and morning time – in the chronic exposure conditions. Because an association between aggression and the levels of corticosterone can be positive or negative, depending on types of aggression (Haller & Kruk, 2006), and because the associations between aggression and the levels of testosterone/corticosterone are sometimes weak in mammals and humans (Van Goozen, 2005), Study #3 did not specify the direction of hormonal associations with aggression. Rather, it was hypothesized that only chronic passive exposure to aggression would change the concentrations of serum testosterone/corticosterone significantly, compared to any other groups.

Finally, Study #4 examined the changes in the neurotransmitter systems, specifically dopaminergic and serotonergic systems, of observer rats in the chronic exposure conditions. Dopamine and serotonin (5-HT) were selected as the target neurotransmitters in Study #4 because they play the key roles in promoting/inhibiting aggressive behavior (e.g., Blum et al., 1996; Spont, 1992). If chronic exposure to aggression is a causal risk of aggression among observer rats, chronic exposure to aggression would develop the neural circuits that resemble the general neural mechanism

of aggression. Thus, it was expected that dopamine and 5-HT are associated with the effects of chronic passive exposure to aggression.

Specifically, Study #4 focused on the densities of dopamine D₂ receptors and 5-HT_{1B} receptors, which are known as the promising precursors of aggression (Miczek et al., 2002). According to Miczek et al. (2001), receptor densities are generally determined by gene phenotypes in response to environmental stimuli. Therefore, if chronic exposure to aggression developed aggressive dispositions of passive observer rats, it would be expected that chronic exposure to aggression alters a phenotypic expression, which, in turn, modifies the densities of dopamine D₂ receptors and 5-HT_{1B} receptors that characterizes aggressive tendencies. In this way, the densities of these receptors were considered as good markers of neurochemical responses to passive exposure to aggression.

However, the precise roles of dopamine D₂ receptors and 5-HT_{1B} receptors in regulating aggression have not been clarified (Nelson & Trainor, 2007). For example, neuropharmacological data indicate that dopamine D₂ receptor antagonists are identified as aggression inhibitor (De Almeida et al., 2005; Miczek et al., 2002). In contrast, a genetic study found that impulsivity was associated with the A1 allele genotypes, which genetically programs the reduced densities of dopamine D₂ receptors (White et al., 2008). As for 5-HT_{1B} receptors, 5-HT_{1B} receptor agonists can reduce aggression among rodents (e.g., Chiavegatto et al., 2001), whereas aggressive mice showed low serotonergic neurotransmissions and the activated 5-HT_{1A} (auto)receptors, and possibly 5-HT_{1B} autoreceptors (Caramaschi et al., 2007). Because some findings are contradictory to each other, Study #4 hypothesized that chronic exposure to aggression

led to either higher or lower densities of dopamine D₂ receptors and 5-HT_{1B} receptors in the brain of observer rats, compared to controls. Table 4 summarizes the design of Study #4.

Table 1. Summary of Experimental Design in Study #1.

		Exposure to...	
		Aggression (experimental)	Non-aggression (control)
Amount of Exposure	Priming (one time)	$n = 30$	$n = 30$
	Acute (1 day)	$n = 18$	$n = 18$
	Chronic (23 days)	$n = 30$	$n = 30$

Note. n indicates sample size for each group.

Table 2. Summary of Experimental Design in Study #2.

		23-day exposure to...	
		Aggression (experimental)	Non-aggression (control)
Time point for screening test	1 day later	$n = 30$	$n = 30$
	16 days later	$n = 30$	$n = 30$

Note. n indicates sample size for each group.

Table 3. Summary of Experimental Design in Study #3.

		Single exposure to...		Chronic exposure to...			
		Aggression (experimental)	Non-aggression (control)	Aggression (experimental)	Aggression (experimental)	Non-aggression (control)	Non-aggression (control)
		Night	Night	Night	Morning	Night	Morning
Hormonal level	Testosterone	<i>n</i> = 9	<i>n</i> = 9	<i>n</i> = 6	<i>n</i> = 30	<i>n</i> = 6	<i>n</i> = 30
	Corticosterone	<i>n</i> = 9	<i>n</i> = 9	<i>n</i> = 6	<i>n</i> = 30	<i>n</i> = 6	<i>n</i> = 30

Note. *n* indicates sample size for each group.

Table 4. Summary of Experimental Design in Study #4

		23-day exposure to...	
		Aggression (experimental)	Non-aggression (control)
Receptor densities	Dopamine D ₂ receptor	<i>n</i> = 15	<i>n</i> = 15
	5-HT _{1B} receptor	<i>n</i> = 15	<i>n</i> = 15

Note. *n* indicates sample size for each group.

CHAPTER TWO

STUDY #1: BEHAVIORAL EFFECTS OF EXPOSURE TO AGGRESSION

The purpose of Study #1 was to compare the aggression levels of observer rats in six groups: priming/acute/chronic exposure to aggression/non-aggression. It was hypothesized that observer rats in the group of chronic exposure to aggression showed the most aggressive behavior in any other groups.

Method

Participants. One hundred fifty six young male Sprague-Dawley rats ($\bar{X} = 328.7$ g with $SD = 35.3$ at the time of a screening test) were recruited as ‘observer rats.’ These rats were obtained from Charles River (Portage, Michigan) or from the breeding of Animal Care Facilities at Loyola University Chicago. All rat subjects were cared under the approval of Institutional Animal Care and Use Committee (IACUC).

Observer rats were assigned to six different conditions: priming exposure to aggression or non-aggression ($n = 30$ per group), acute exposure to aggression or non-aggression ($n = 18$ per group), or chronic exposure to aggression or non-aggression ($n = 30$ per group). There was no difference in body weights between the groups exposed to aggression (experimental group) and the groups exposed to non-aggression (control group; $t(166) = .174, p = .862$). One-way ANOVA revealed that there were differences in body weights among the six groups ($F(5, 150) = 4.16, p < .01$) such that the group of chronic exposure to non-aggression ($\bar{X} = 340.7$ g) was heavier than the groups of

priming exposure to aggression ($\bar{X} = 317.0$ g, $p < .05$) and priming exposure to non-aggression ($\bar{X} = 319.3$ g, $p < .05$) while no other differences were detected ($\bar{X} = 316.1$ g for the group of priming exposure to aggression; $\bar{X} = 323.3$ g for the groups of priming exposure to non-aggression; $\bar{X} = 337.3$ g for the group of chronic exposure to aggression). However, to my knowledge, no studies have suggested that body weights affect aggressiveness, hence these differences in body weights among the six groups should not have influenced aggressive behavior of observer rats in Study #1.

In both the priming and acute exposure conditions, observer rats were once exposed to either aggression (i.e., experimental groups) or non-aggression (i.e., control groups) for 10 min. Then, aggressive behavior of observer rats was screened immediately (for the priming conditions) or 24 hours (for acute exposure to aggression) after the single exposure. In the chronic exposure conditions, observer rats had been exposed to aggression or non-aggression for 10 min. per day for consecutive 23 days. Their screening tests took place 24 hours after the last exposure session.

My preliminary study showed that there was a difference in the amount of aggressive behavior (in sec.) between the six groups ($F(5, 30) = 6.281$, $p < .01$), and Bonferroni-typed a posteriori comparisons indicated that the group of chronic exposure to aggression ($\bar{X} = 100.3$ sec., $p < .01$ for all comparisons) displayed more aggression than any other groups ($\bar{X} = 12.0$ sec. for the group of priming exposure to non-aggression; $\bar{X} = 23.6$ sec. for the group of priming exposure to aggression; $\bar{X} = 13.2$ sec. for the group of acute exposure to non-aggression; $\bar{X} = 15.6$ sec. for the group of acute exposure to aggression; and $\bar{X} = 22.5$ sec. for the group of chronic exposure to non-aggression; the

detailed procedure was discussed later). Based on these data, power analysis estimated that seven subjects per group were needed at minimum and that 16 subjects were recommended to achieve 100% power (Hintze, 2006). Thus, the sample size in Study #1 met this recommendation.

In addition to these observer rats, aggressive dyads and non-aggressive dyads were recruited and used to manipulate an aggressive or non-aggressive situation around each observer rat. To have aggressive dyads, six 'resident rats' (with approximately 400 g or heavier) were recruited based on pre-screening tests of aggression and housed with a female rat (with approximately 250 g) for a few weeks before Study #1 started. At the time of every exposure session, the female rat was replaced with a younger male rat which weighed about 100 g less than the resident rat. This young male rat was called an 'intruder' rat because he was naïve and a potential rival for mating in the home cage (i.e., territory) of the resident rat. According to resident-intruder paradigm (Blanchard & Blanchard, 1990; Olivier & Young, 2002), the resident rats usually attack the intruder rat to protect their territory especially when the resident rats had been pre-paired with a female rat (Fish et al., 1999; 2001). Furthermore, it was important that intruder rats were approximately 100 g smaller than the resident rats so that the resident rats were likely to actively attack and defeat the intruder rats. Unless any of the six resident rats stopped showing aggression, they were repeatedly used through Study #1 (and subsequent Study #2-#4).

For the control groups, six non-aggressive dyads were formed by recruiting a big male rat (with approximately 400 g or heavier) and a small male rat (with approximately 100 g less than the other rat). They had been cohabitated together in a single cage for a

few weeks before Study #1 started. Since they shared the same space, they eventually established social hierarchy before Study #1. Hence, at the time of exposure sessions, the bigger dominant rat avoided a futile risk while the smaller subordinate rat avoided an obviously losing fight. The non-aggressive dyads were replaced about once every few weeks so that the subordinate rats did not gain more weights than their paired dominant rat and turn over the existing social hierarchy during each exposure session.

Design and Procedure. Each exposure session was administered for 10 min. under a red light illumination between 7:00 PM and 9:00 PM. For the experimental groups (i.e., exposure to aggression), a female partner for a resident rat was replaced with a naïve intruder rat. Then, a smaller plastic and transparent aquarium (22.9 × 15 × 16.5 cm) with a mesh lid was placed into the home cage (47 × 25.5 × 21.5 cm) of the resident rat. An observer rat was transferred from his own home cage (47 × 25.5 × 21.5 cm) to the smaller aquarium and stayed in the aquarium for only 10 min. per day. Compared with the body size of the observer rats, the aquarium was large enough so that the observer rats freely moved around within it. Thus, the possible restraint stress on the observer rat was minimal.

During this short exposure session, the resident rat and an intruder rat could interact with and fight each other, whereas there was no physical contact between the observer rat and the dyad (i.e., the resident rat and intruder rat). However, the observer rat could see, hear, and even smell the social interactions within the dyad. After each of 10-min. exposure session, the observer rat was immediately transferred back to his original home cage and was provided a regular care (i.e., sufficient food, water, and

largely enough clean cage) until the next exposure session or behavioral screening test.

Also, the intruder rat was replaced with the previously removed female partner in the cage of the resident rat.

The same procedure took place for the control groups (i.e., exposure to non-aggression); an observer rat in the small aquarium was introduced into the cage of the non-aggressive dyad for 10 min. under a red light illumination. Then, the observer rat was removed from the small aquarium and placed into his large home cage with a regular care until the next exposure session or screening test.

These procedures for the experimental and control conditions were run either only once (for priming and acute exposure conditions) or repeated once daily for 23 days (for chronic exposure conditions), depending on which condition an observer rat was assigned. For both experimental and control groups, all exposure sessions were recorded by a video camera.

After the exposure session(s), each observer rat was screened on his aggression level by pairing him with another naïve male rat (i.e., an ‘opponent’ rat) within a new cage under a red light illumination between 7:00 PM to 9:00 PM. For the priming conditions, this screening test took place immediately after an exposure session; for the acute and chronic exposure conditions, a screening test was conducted 24 hours after 1-day or 23-day exposure. The body weights of both the observer rat and opponent rat were approximately identical ($t(334) = 1.84, p = .07$), and their interaction was recorded by a video camera.

Behavioral Variables. To measure aggressiveness of observer rats, content analysis was performed by watching videotapes that recorded the screening tests of observer rats. A stop-watch was used to count up the amount of time in seconds during which each observer rat was engaged in aggressive behavior. In addition, the amount of time during which each opponent rat was engaged in defensive or submissive behavior was also counted in seconds because it could indirectly reflect the intensity of each observer rat's aggression. For instance, if an observer rat's aggressive behavior was severe enough to knock down his opponent rat, the opponent rat was supposed to overwhelmingly exhibit defensive and submissive behavior during a screening session.

Aggressive behavior and defensive/submissive behavior were identified according to Miczek's (1974) guidelines with some modifications. In my definition, aggressive behavior included attack (e.g., leaping at an opponent, biting, arching over and holding an opponent, pulling an opponent's skin), threat (e.g., pushing an opponent with his back), aggressive posture (e.g., bending over an opponent with his head and forelimbs arched over an opponent), allogrooming (e.g., aggressively grooming or nibbling an opponent's neck), mutual upright posture (e.g., standing on his hindlegs and boxing), and chasing (e.g., following an fleeing opponent). On the other hand, defensive/submissive behavior included immobile crouch posture (e.g., freezing), defensive upright posture (e.g., standing on his hindlegs and staring at an opponent), submissive-supine posture (e.g., lying flat on his back and exposing his ventral surface), and flight (e.g., quickly moving away from an opponent). Play fighting (e.g., contacting each other's snout, face, and nape of the neck) was excluded from my definition of aggression because the purpose of

play fighting is more related to social bonding and checking each other's social status (Pellis & Pellis, 1987; Pellis, Pellis, & Foroud, 2005).

Aggression scores of observer rats and defense/submission scores of opponent rats (in sec.) were cross-matched to test intra-rater reliability with Cronbach's α . The intra-rater reliability of aggression/defense/submission scores was .85. In addition, to avoid a potential subjective bias through coding, two well-trained research assistants also watched the same videotapes and scored aggression of observer rats and defense/submission of opponent rats. Cronbach's α for this inter-rater reliability of aggression scores was .93; Cronbach's α for the inter-rater reliability of defense/submission scores was .80.

Finally, to confirm whether or not observer rats in the experimental groups across the different conditions of the amounts of exposure (i.e., priming, acute, and chronic) were exposed to more amounts of aggression than those in the control groups, aggression scores of the aggressive and non-aggressive dyads during exposure sessions were also analyzed. These aggression scores were obtained in the same way as the one to score aggression levels of observer rats.

Statistical Strategy. For the manipulation check, a two-independent samples *t*-test was performed to test if the experimental groups were overall exposed to more average amount of aggression per day (in sec.) than the control groups.

Next, two sets of two-way MANOVA's were operated to compare (1) the mean amount of aggression of observer rats and (2) the mean amount of defense/submission of opponent rats. In terms of these dependent variables, the first MANOVA analyzed the

interactions of (a) the group effect (exposure to aggression vs. non-aggression) and (b) the effect of time point for screening tests (immediately vs. 24 hours after the single exposure) between the priming conditions and the acute exposure conditions. The next two-way MANOVA examined the interactions of (a) the group effect (exposure to aggression vs. non-aggression) and (b) the effect of exposure amount (1 day vs. 23 days) between the acute exposure conditions and the chronic exposure conditions.

Finally, one-way MANOVA's with a priori comparisons were performed to compare individual differences in (1) the mean amount of observer rats' aggression and (2) the mean amount of opponent rats' defense/submission among the six different conditions (i.e., priming/acute/chronic exposure to aggression/non-aggression). Note that, based on my preliminary study, it was hypothesized that only the group of chronic exposure to aggression would show a higher aggression level than any other groups. Thus, a priori comparison tested the differences between (a) the group of chronic exposure to aggression and (b) the other groups in terms of observer rats' aggression scores and opponent rats' defense/submission scores. Additional comparisons within (b) were also made by Bonferroni-typed a posteriori comparisons.

Results

Study #1 successfully manipulated the amount of aggression that observer rats were exposed to between the experimental group and the control group ($t(154) = 11.85, p < .01$). That is, all experimental groups across the different conditions of the amounts of exposure ($\bar{X} = 154.5$ seconds) were exposed to more aggressive situations per day than the control groups ($\bar{X} = 5.1$ seconds).

Table 5 indicates that, on average, the observer rats in the group of priming exposure to non-aggression ($n = 30$) showed 14.1 seconds as their aggression score ($SD = 54.3$); those in the group of priming exposure to aggression ($n = 30$) scored 11.5 seconds ($SD = 25.4$); those in the group of acute exposure to non-aggression ($n = 18$) scored 20.5 seconds ($SD = 29.5$); those in the group of acute exposure to aggression ($n = 18$) scored 21.3 seconds ($SD = 50.8$); those in the group of chronic exposure to non-aggression ($n = 30$) scored 41.4 seconds ($SD = 53.7$); and those in the group of chronic exposure to aggression ($n = 30$) scored 88.2 seconds ($SD = 63.6$). The mean defense/submission scores of the opponent rats in each group were following: 16.1 seconds ($SD = 58.4$) in the group of priming exposure to non-aggression ($n = 30$); 16.8 seconds ($SD = 40.3$) in the group of priming exposure to aggression ($n = 30$); 36.3 seconds ($SD = 57.4$) in the group of acute exposure to non-aggression ($n = 18$); 34.4 seconds ($SD = 81.1$) in the group of acute exposure to aggression ($n = 18$); 70.6 seconds ($SD = 98.3$) in the group of chronic exposure to non-aggression ($n = 30$); and 111.9 seconds ($SD = 110.8$) in the group of chronic exposure to aggression ($n = 30$).

Table 6 reveals the result of a two-way MANOVA between the priming conditions and the acute conditions. There were no significant main effects or interaction effect of group \times time point for screening tests. Therefore, regardless of whether aggressiveness of the observer rats was assessed immediately or 24 hours later, the single exposure did not differentiate aggression scores or defense/submission scores between the experimental groups and the control groups. That is, no priming effect of passive exposure to aggression or the effect of acute exposure to aggression was found among these observer rats.

However, as I expected, a two-way MANOVA between the acute exposure conditions and the chronic exposure conditions found significant main effects of group ($F(1, 92) = 4.51, p < .05$) and exposure amount ($F(1, 92) = 15.36, p < .01$; see Table 7). More importantly, there was a significant interaction for aggression scores ($F(1, 92) = 4.23, p < .05$). That is, the observer rats exposed to aggression ($\bar{X} = 63.1$ seconds) showed more aggressive than those exposed to non-aggression ($\bar{X} = 33.6$ seconds), depending on whether exposure was given once or repeatedly. For defense/submission scores, only a main effect of exposure amount was significant ($F(1, 92) = 8.05, p < .01$), suggesting that the opponent rats used in the chronic exposure conditions ($\bar{X} = 91.3$ seconds) overall showed more defensive/submissive than those used in the acute exposure conditions ($\bar{X} = 35.4$ seconds).

Figure 1 shows individual comparisons of the mean aggression scores of observer rats among the six conditions. A one-way ANOVA indicated that a significant difference in aggression scores existed ($F(5, 150) = 10.13, p < .01$), and a priori comparison supported my hypothesis that the observer rats in the group of chronic exposure to aggression exhibited significantly more aggressive than those in any other groups ($t(150) = 6.60, p < .01$). This result was also evident by comparing the mean defense/submission scores of opponent rats among the six conditions (see Figure 2). There was a significant difference in defense/submission scores ($F(5, 150) = 6.41, p < .01$), specifically showing that the opponent rats paired with the group of chronic exposure to aggression significantly showed defensive and/or submissive, compared to those in the other groups ($t(150) = 4.74, p < .01$). Furthermore, Bonferroni-typed a posteriori comparisons did not

find any differences in aggression scores and defense/submission scores among all groups but the group of chronic exposure to aggression.

Discussion

Study #1 investigated whether passive observer rats could become aggressive in response to (1) priming, (2) acute exposure, and (3) chronic exposure to aggression, compared to those exposed to non-aggression under each exposure amount. It was found that the observer rats which were chronically exposed to aggression showed more aggression than those in any other conditions (see Figure 1). On the other hand, there were no differences in aggressiveness of the observer rats among the groups of priming exposure to aggression/non-aggression, acute exposure to aggression/non-aggression, and chronic exposure to non-aggression. These results were replicated when defense/submission scores of opponent rats were compared (see Figure 2). That is, the opponent rats showed more defensive and/or submissive behaviors when they were paired with the observer rats chronically exposed to aggression, compared to those which were paired with the other observer rats.

There are several implications in the results of Study #1. First, the priming effect of exposure to aggression was not found in rats; there was no significant difference in observer's aggressiveness/opponent's defensiveness and submissiveness between the groups of priming exposure to aggression and non-aggression (see Table 6 and Figure 1 and 2). On the other hand, Bandura et al.'s (1961, 1963) experiments demonstrated that child participants showed aggression toward the Bobo doll immediately after a single, 10-min. exposure to the aggressive adult model, suggesting a potential priming (and/or imitative) effect. Other studies also found that aggression was primed by situational

aggression cues, such as violent films, in human subjects (Josephson, 1987; Leyens, Camino, Parke, & Berkowitz, 1975). This difference in the priming effect between rats and humans are interesting from the viewpoint of comparative psychology. If the priming effect was only human phenomenon, that may be derived from anatomical differences in the brain between rats and humans. For example, one fMRI study using human subjects showed significant reductions in the cortical activity during priming (e.g., Koutstaal et al., 2001), suggesting that existing knowledge is automatically accessed in the cortical regions. If the cortical structures have the significant roles in priming, a smaller proportion of the cortical regions in rats may make it difficult to produce aggressive behavior through an automatic retrieval process relating to priming.

The second implication is that passive exposure to aggression inclined observer rats to be aggressive only when exposure was provided to observer rats repeatedly; observer rats could not internalize aggression by only a single, acute exposure to aggression (see Table 7 and Figure 1 and 2). Thus, a learning/socializing process requires repeated and continuous observations of aggression, which was consistent with Huesmann and Kirwil's (2007) argument. This may be said to only rats, however, and human observers may be able to learn and internalize aggression through a one-time observation. In fact, Bandura et al.'s (1961) Bobo doll experiment showed that child participants became aggressive by a single exposure to aggression although it is unclear whether they 'learned' or 'imitated/be primed' aggressive behavior. At any rate, repeated observations of aggression exclusively results in developing aggressive tendencies among observers.

My results also indicated that passive exposure to aggression encouraged observer rats to be aggressive, rather than inhibiting their aggressive behavior. Prior to Study #1, it was alternatively possible that priming, acute, or chronic exposure to aggression was associated with the low levels of aggressiveness of observer rats if they were fearful of an aggressive situation or learned defensive and submissive patterns of intruder rats. But, the results of Study #1 suggested that the behavioral patterns of observer rats were rather similar to the resident rats' aggressive behavior; observer rats appeared to learn how to compete, defeat, and prevail over the other. In other words, exposure to aggression did not deter passive observer rats from being aggressively. Or, exposure to aggression might lead passive observer rats to be less sensitive to fear in a potential aggressive/threatening situation. It should be noted, nevertheless, that this behavioral similarity between the observer rats and the resident rats occurred only when observer rats were repeatedly exposed to aggression. Thus, one-time presentation of aggression may not be a risky factor (at least for rats), but repeated observations of aggression encourage, rather than discouraging, passive observers to behave aggressively.

Previous psychological studies have discussed the associations of aggression with aggressive schema/hostile attributional bias (Dill et al., 1997; Dodge, 1980; Dodge & Coie, 1987; Dodge & Frame, 1982; Dodge et al., 1990; Dodge & Tomlin, 1987; Graham & Hudley, 1994; Nasby et al., 1979; Slaby & Guerra, 1988; Steinberg & Dodge, 1983), normative beliefs about aggression (Guerra et al., 1995; Huesmann & Guerra, 1997), positive attitudes toward aggression (Bookwala et al., 1992; Kingery, 1998; Markowitz, 2001), and justification of aggression (Azar & Rohrbeck, 1986; Hyman, 1995). Although it is difficult to say that all of them is applicable to observer rats exposed to aggression

for 23 days (because some of them may be phenomena in only creatures with higher-order cognitive processing, such as humans), it is likely that observer rats might form positive attitudes toward aggression in response to chronic exposure to aggression. In other words, repeated observations of aggression might notice passive observer rats that aggressive behavior was an appropriate behavioral response in a social interaction. Consequently, the observer rats which had been chronically exposed to aggression might eventually accept aggressive means as an appropriate interaction (i.e., formation of positive attitudes toward aggression) and follow their attitudinal knowledge of aggression.

As the other implication of the results in Study #1, I had an impression that the observer rats exposed to aggression for 23 days attacked in more hyperactive manners than those in the other groups. Figure 3 presents two representative behavioral patterns of observer rats. Diagram A shows the representative behavioral records of an observer rat exposed to aggression for 23 days (in the above belt-like box) and his opponent rat (in the below box), whereas Diagram B represents the behavioral patterns of an observer rat exposed to non-aggression for 23 days (in the above box) and his opponent rat (in the below box). Within each box, the discrete lines represent offensive, defensive, submissive, and other behaviors (e.g., smelling each other, exploring environments, gentle grooming) from the above to below, respectively. In Diagram A, the observer rat (i.e., a representative of the group of 23-day exposure to aggression) exhibited aggressive behavior intermittently and frequently while his opponent rat continued to show defensive behavior. This observer rat leaped at and arched over his opponent rat for the first time. Shortly afterwards, the observer rat went away from the opponent rat and, all

of sudden, leaped at the opponent again by fits and starts. His behavioral patterns seemed to characterize hyperactive, explosive, and intermittent aggression. Moreover, he maintained aggressive behavior, regardless of whether his opponent rat showed defensive behavior or not.

In contrast, in Diagram B, the observer rat (i.e., a representative of the group of 23-day exposure to non-aggression) also showed aggression but stopped it once his opponent partner displayed defensive posture. This observer rat did not show any attacks once social hierarchy was established between his opponent rat and him.

These different behavioral patterns of aggression might imply that the observer rats chronically exposed to aggression exhibited affective and instrumental aggression (Anderson & Huesmann, 2003). In comparison, the observer rats exposed to non-aggression for 23 days might predominantly exhibit instrumental aggression. That is, the observer rats exposed to aggression for 23 days seemed to show aggression for more than establishing social hierarchy; for example, the purpose of their aggression might be to hurt their opponent rat or to fulfill pleasant feelings. However, the observer rats exposed to non-aggression seemed to use aggressive means only to determine that they were dominant. In other words, the results in Study #1 imply that chronic exposure to aggression might be remarkably associated with affective/intermittent explosive aggression.

One might criticize that exposure to aggression might be confounded with stress. That is, it was possible that stress itself due to chronic exposure to aggression resulted in the observer rat's aggressiveness, rather than learning aggression through passive observations independent of stress. For example, corticosterone (i.e., one type of stress

hormone) functions to elevate aggressive behavior (Mikics et al., 2004). If stress influences aggression, repeated presentations of stress that are unrelated to aggression (e.g., restraint, foot-shock) could also make rats even more aggressive. In fact, one study found that, although acute restraint stress reduced the frequency of aggression, the length of days in daily restraint stress (i.e., 7-day to 21-day exposure to 6-hour stress) proportionally increased aggression in male rats (Wood, Young, Reagan, & McEwen, 2003).

However, all observer rats were never restrained in Study #1, suggesting that, if stress played a role, it may be a minor one. Furthermore, it was also true that a negative association between corticosterone and aggression was found by some studies (Kariyawasam et al., 2002; McBurnett et al., 2000; Pajer et al., 2001; van Goozen et al., 1998; Vanyukov et al., 1993). Finally, stress is not always a predictor of aggression. For instance, social stress due to a personal history of defeat was associated with reduced aggressive behavior (e.g., Blanchard & Blanchard, 1990; Blanchard et al., 1993, 1995; Blanchard, Yudko, Dulloog, & Blanchard, 2001; Tamashiro et al., 2004, 2005). In female pregnant mice, daily restraint stress (i.e., 5-day exposure to 30-minute stress) does not increase or decrease the amount of time for maternal aggression while acute restraint stress reduces it (Gammie & Stevenson, 2006). Even in humans, frustration, which is a specific form of stress, leads to aggression only when anger is induced by the frustration (Berkowitz, 1978, 1989). Thus, not all types of stress facilitate aggression. Prior to Study #1, I could not predict whether observer rats were encouraged or discouraged to be aggressive. If exposure to aggression played the same role as stress due to social defeat, as in the Blanchard et al.'s (1990, 1995) model, the observer rats exposed to aggression

for 23 days would show little aggression. Nevertheless, my results suggested the opposite, namely that chronic passive exposure to aggression actually enhanced aggression.

In conclusion, chronic passive exposure to aggression predisposed passive observer rats to be aggressive maybe because of the learning/socializing effects. But, priming and acute exposure to aggression did not result in high aggressiveness of observer rats. Although it is necessary to cautiously consider whether these findings in the rat model are applicable to human behavior, our evidence was consistent with what previous survey research found (see Margolin & Gordis, 2000, for review). Therefore, I conclude that chronic passive exposure to aggression is an exclusive risk factor of developing aggressiveness of a witness.

Table 5. Mean Aggression scores (in Seconds) of Observer Rats and Mean Defense/Submission Scores (in Seconds) of Their Opponent Rats in Priming Exposure to Non-Aggression ($n = 30$), Priming Exposure to Aggression ($n = 30$), Acute Exposure to Non-Aggression ($n = 18$), Acute Exposure to Aggression ($n = 18$), Chronic Exposure to Non-Aggression ($n = 30$), and Chronic Exposure to Aggression ($n = 30$)

Condition	Aggression score	Defense/submission score
Priming exposure to...	Non-aggression	14.1 (54.3)
	Aggression	16.1 (58.4)
Acute exposure to...	Non-aggression	11.5 (25.4)
	Aggression	16.8 (40.3)
Chronic exposure to...	Non-aggression	20.5 (29.5)
	Aggression	36.3 (57.4)
	Non-aggression	21.3 (50.8)
	Aggression	34.4 (81.1)
	Non-aggression	41.4 (53.7)
	Aggression	70.6 (98.3)
		111.9 (110.8)

Note. Aggression scores were based on the amount of time (in seconds) when observer rats were engaged in aggressive behavior during a 10-min behavioral screening test. Defense/submission scores were the amount of time (in seconds) when opponent rats were engaged in defensive and submissive behaviors during a 10-min behavioral screening test. Values within parentheses indicate a standard deviation.

Table 6. Multivariate Analysis of Variance for Aggression Scores and Defense Scores between Priming Conditions and Acute Exposure Conditions ($N = 96$)

Source	Aggression score				Defense/submission score			
	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>
Between subjects								
Group (G)	1	.01	.00	.92	1	.00	.00	.96
Time point for screening tests (T)	1	.83	.01	.37	1	2.36	.03	.13
G \times T	1	.04	.00	.85	1	.01	.00	.92
S within-group error	92	(1771.64)			92	(3412.70)		

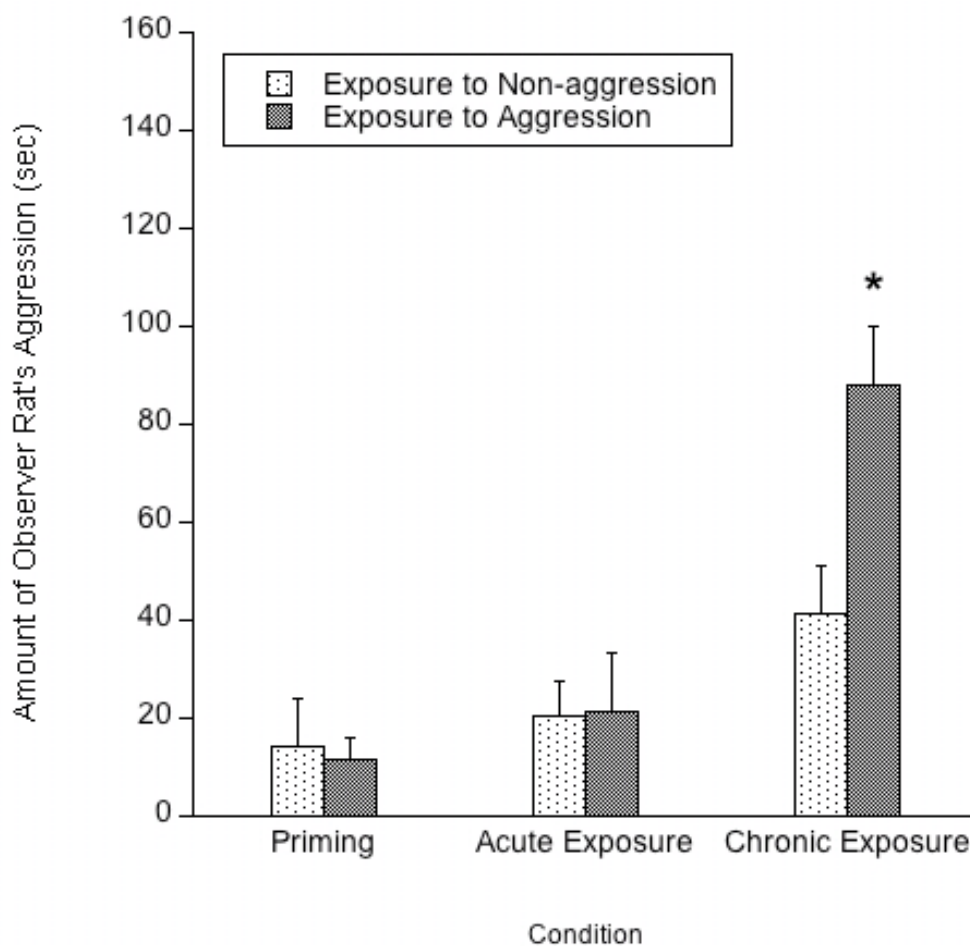
Note. Values within parentheses represent mean square errors. S = subjects.

Table 7. Multivariate Analysis of Variance for Aggression Scores and Defense Scores between Acute Exposure Conditions and Chronic Exposure Conditions ($N = 96$)

Source	Aggression score				Defense/submission score			
	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>
Between subjects								
Group (G)	1	4.51	.05	.04	1	1.00	.01	.32
Exposure amount (E)	1	15.36	.14	.00	1	8.05	.08	.01
G \times E	1	4.23	.04	.04	1	1.20	.01	.28
S within-group error	92	(2823.43)			92	(8739.56)		

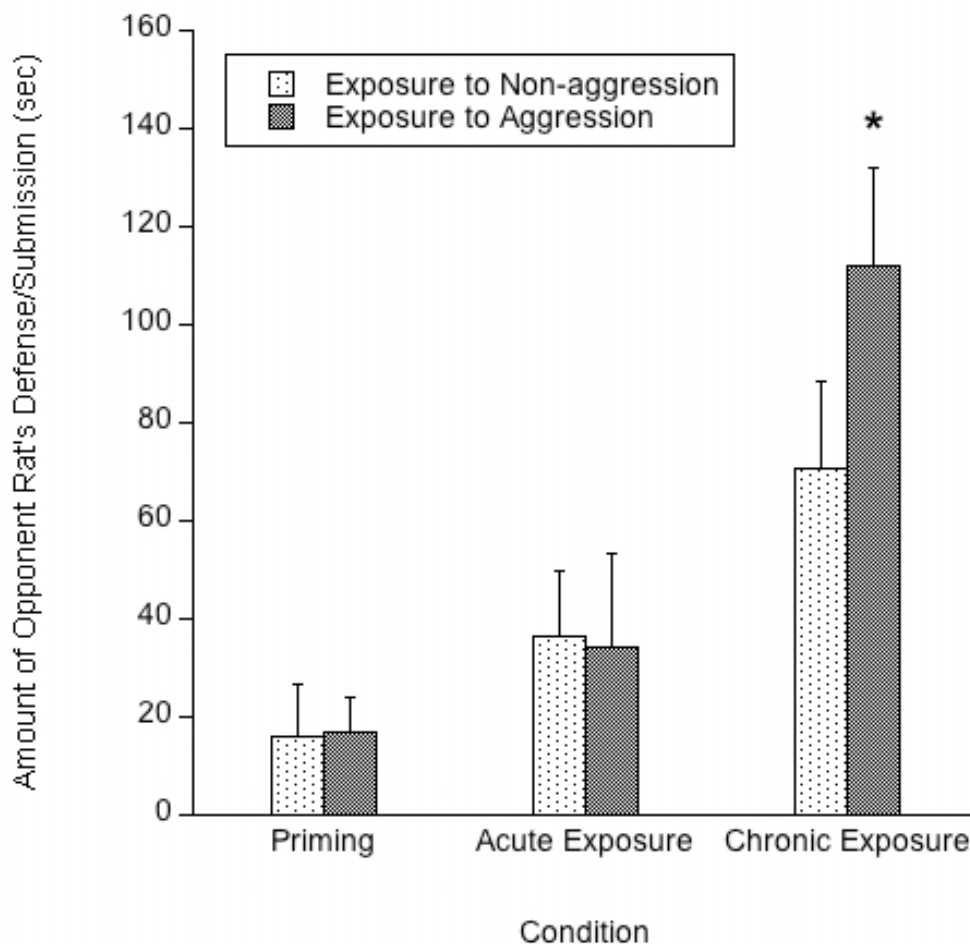
Note. Values within parentheses represent mean square errors. S = subjects.

Figure 1. Mean Aggression Scores (+SE) of Observer Rats in Priming Exposure to Non-Aggression ($n = 30$), Priming Exposure to Aggression ($n = 30$), Acute Exposure to Non-Aggression ($n = 18$), Acute Exposure to Aggression ($n = 18$), Chronic Exposure to Non-Aggression ($n = 30$), and Chronic Exposure to Aggression ($n = 30$)



Note. According to a one-way ANOVA, there was a significant difference in aggression scores among the six groups such that the group of chronic exposure to aggression scored higher than any other groups. An asterisk indicates a significant difference ($p < .01$).

Figure 2. Mean Defense/Submission Scores ($+SE$) of Observer Rats in Priming Exposure to Non-Aggression ($n = 30$), Priming Exposure to Aggression ($n = 30$), Acute Exposure to Non-Aggression ($n = 18$), Acute Exposure to Aggression ($n = 18$), Chronic Exposure to Non-Aggression ($n = 30$), and Chronic Exposure to Aggression ($n = 30$)



Note. According to a one-way ANOVA, there was a significant difference in defense/submission scores among the six groups such that the opponent rats in the group of chronic exposure to aggression scored higher than those in any other groups. An asterisk indicates a significant difference ($p < .05$).

Figure 3. Representative Behavioral Patterns of Observer Rats and Opponent Rats in the Groups of 23-day Exposure Conditions

Diagram A.

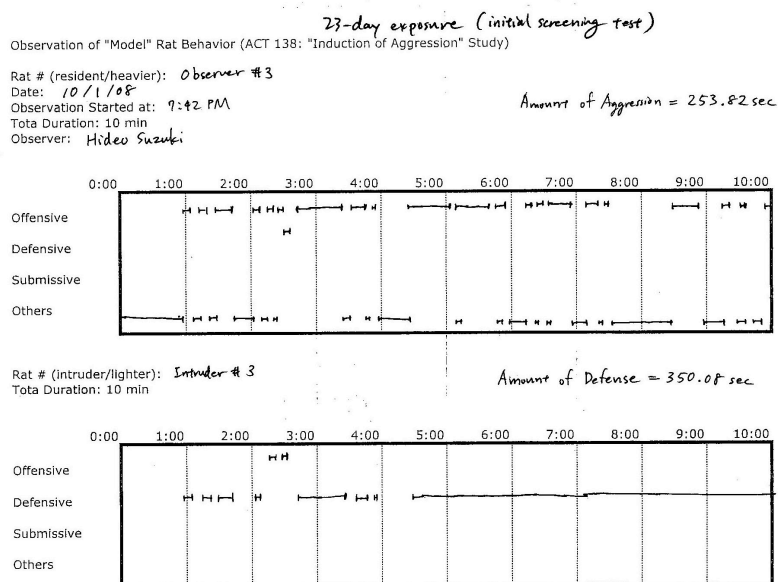
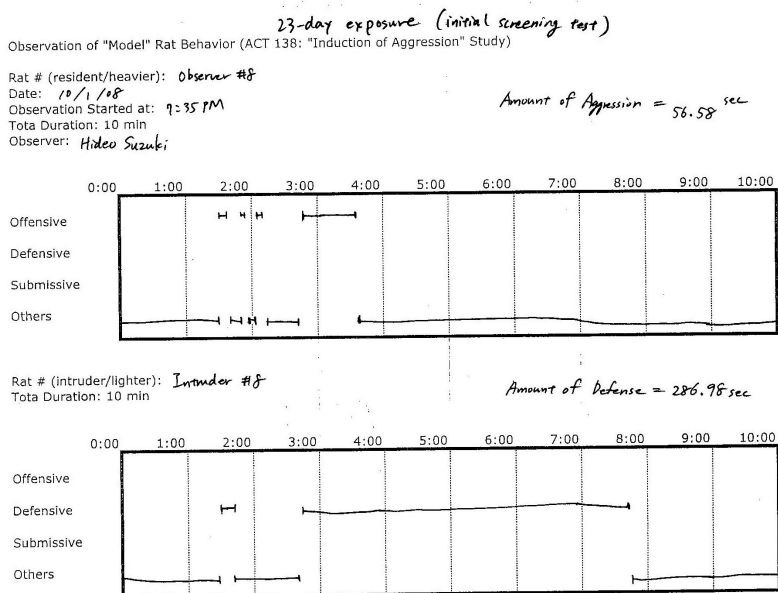


Diagram B.



Note. Diagram A describes the behavioral patterns of an observer rat (in the above box) and his opponent rat (in the below box) 1 day after 23-day exposure to aggression; diagram B describes the behavioral patterns of an observer rat (in the above box) and his opponent rat (in the below box) 1 day after 23-day exposure to non-aggression.

CHAPTER THREE

STUDY #2: FOLLOW-UP STUDY FOR BEHAVIORAL EFFECTS

In Study #1, it was found that only observer rats having chronically exposed to aggression showed more aggression, compared to those in the other conditions. The purpose of Study #2 was a behavioral follow-up study to examine whether or not the observer rats in the group of chronic exposure to aggression remained their aggressiveness even after the treatment of exposure to aggression was no longer provided. Because I could not find any significant aggressiveness among the observer rats in the priming and the acute exposure conditions, these observer rats were not used for the present follow-up study. Instead, Study #2 focused on only two groups: chronic passive exposure to aggression (as the experimental group) and chronic passive exposure to non-aggression (as the control group).

Method

Participants. Animal subjects were the observer rats which were used as the groups of 23-day exposure to aggression and non-aggression in Study #1. After they were screened on their aggressiveness in Study #1, each of them was transferred to a new regular cage (47 × 25.5 × 21.5 cm) and lived there individually. Regular care was provided to them until they were used in Study #2.

Design and Procedure. All observer rats were left alone and were not given any additional exposure sessions until the follow-up screening tests. In other words, the home cages of these observer rats were placed away from the home cages of the resident rats and non-aggressive dyads, and the observer rats were not allowed to interact with any other rats. Sixteen days counting from the last exposure session in Study #1, that is, approximately a half month of recovery from passive exposure to aggression/non-aggression, all observer rats were screened on their aggressiveness again. At that time, the observer rats weighed 390.2 g on average ($SD = 32.9$). The procedure for these screening tests was exactly the same as the one performed in Study #1 (see Chapter 2). That is, under a red light illumination between 7:00PM to 9:00PM, each observer rat was paired with another naïve male ‘opponent’ rat within a new cage. The body weights of both the observer rats and opponent rats were identical ($t(118) = .887, p = .38$), and all screening tests were again video-recorded.

Behavioral Variables. Aggressiveness of the observer rats was measured by counting the amount of time (in seconds) during which each observer rat was engaged in aggressive behavior (i.e., aggression score). In addition, defensive/submissive behavior of the opponent rats was also measured by assessing the amount of time (in seconds) during which each opponent rat was engaged in defensive/submissive behavior (i.e., defense/submission score). Aggressive behavior and defensive/submissive behavior were respectively defined in the same way as the one of what I defined them in Study #1 (see Chapter 2). Note that play fighting was excluded from the definition of aggressive

behavior because play fighting is associated to promoting a social bond or checking with social status (Pellis & Pellis, 1987; Pellis et al., 2005).

The intra-rater reliability of aggression scores of the observer rats and defense/submission scores of their opponent rats reached the acceptable level; Cronbach's α was .728. The inter-rater reliability of aggression scores between my coding and research assistants' coding was Cronbach's α of .966, and the reliability of defense/submission scores was Cronbach's α of .643. Therefore, the reliability of aggression scores was high, whereas the reliability of defense/submission scores was slightly low.

Statistical Strategy. A two-way repeated-measures MANOVA was used to test aggression scores of the observer rats and defense/submission scores of their opponent rats. By using this statistical method, Study #2 examined an interaction of group effect (exposure to aggression vs. non-aggression) \times effect of time point for a screening test (1 day vs. 16 days after exposure).

In addition, within each of the groups of exposure to aggression and non-aggression, two sets of one-way repeated-measures MANOVA's were used to evaluate the individual differences in aggression scores and defense/submission scores across the time points.

Furthermore, a one-way MANOVA was used to compare individual differences in aggression scores and defense/submission scores between the groups of exposure to aggression and non-aggression at each of the time points.

Lastly, Study #2 also examined the relationship between aggression scores on an initial screening test and on a follow-up screening test. For an exploratory purpose, it was also tested whether there was a positive regression line between these aggression scores, which might imply an individual variation in sensitivity to exposure to aggression or the effect of encoding specificity (see Results and Discussion for details). Simple regression analyses (linear and quadratic) were performed to analyze whether the initial aggression score could predict the follow-up aggression score within (1) the pooled observer rats, (2) the group of chronic exposure to aggression, and (3) the group of chronic exposure to non-aggression.

Results

Table 8 summarizes the descriptive statistics of aggression scores and defense/submission scores at the initial screening test (i.e., 1 day after 23-day exposure) and the follow-up screening test (16 days after 23-day exposure). As Study #1 showed (see Chapter 2), the initial screening test revealed the mean aggression score of 41.4 seconds ($SD = 53.7$) for the group of exposure to non-aggression and the mean aggression score of 88.2 seconds ($SD = 63.6$) for the group of exposure to aggression. The mean defense/submission scores were 70.6 seconds ($SD = 98.3$) for the group of exposure to non-aggression and 111.9 ($SD = 110.8$) for the group of exposure to aggression, respectively. At the follow-up screening test, the group of exposure to aggression showed the mean aggression score of 57.7 seconds ($SD = 72.1$) and the mean defense/submission score of 98.5 seconds ($SD = 137.6$). In contrast, the group of exposure to non-aggression showed the mean aggression score of 24.7 seconds ($SD = 42.5$) and the mean defense/submission score of 50.2 seconds ($SD = 102.1$).

Table 9 shows that, for aggression scores, there were significant main effects of group ($F(1, 58) = 9.02, p < .01$) and of time point for screening tests ($F(1, 58) = 9.95, p < .01$). However, the interaction effect was not significant. These results were also confirmed by using one-way MANOVA/one-way repeated-measures MANOVA within each group. There were significant differences in aggression scores between the groups of exposure to aggression and non-aggression 1 day after 23-day exposure ($F(1, 58) = 9.48, p < .01$) and 16 days after 23-day exposure ($F(1, 58) = 4.65, p < .05$). At the same time, the observer rats exposed to aggression significantly decreased their aggression across the time points for screening tests ($F(1, 29) = 5.06, p < .05$), and so did those exposed to non-aggression ($F(1, 29) = 7.01, p < .05$). Therefore, both groups of exposure to aggression and non-aggression decreased their aggressiveness 16 days after 23-day exposure while the significant difference in aggressiveness between the two groups maintained across 16 days (see Figure 4).

On the other hand, there were no main effects of group and of time point for screening tests, as well as interaction effect, in defense/submission scores (see Table 9 and Figure 5). Consistent with these results, one-way MANOVA/one-way repeated-measures MANOVA did not find any individual differences for defense/submission scores. Thus, both groups showed defense/submission scores at the same level across the time points for screening tests.

Furthermore, simple regression analyses revealed that there was a regression relationship between the initial aggression scores and follow-up aggression scores in the pooled observer rats (see Figure 6). Both linear ($F(1, 58) = 27.16, p < .01$) and quadratic relationships ($F(2, 57) = 13.70, p < .01$) could account for the variance in aggression

scores, and the values of r^2 were almost identical between them ($r^2 = .32$ for the linear; $r^2 = .33$ for the quadratic). Nevertheless, the predictor of the initial aggression appeared to be significant in the linear regression equation ($B = .55, t = 5.21, p < .01$), whereas the squared predictor of the initial aggression scores was not significant in the quadratic equation ($B = .36, t = 1.19, p = .24$). Thus, the linear regression was the best simple model describing the relationship between the initial aggression scores and the follow-up aggression scores in the pooled observer rats. These results suggest that, regardless of the conditions, when the observer rats showed the high levels of aggression at the initial screening tests, they also kept showing high aggressiveness at the follow-up screening tests.

More interestingly, there seemed to be differences in the values of the correlation squared between the regression models within the group of chronic exposure to aggression and within the group of chronic exposure to non-aggression. Specifically, the values of the correlation squared showed .17 for the linear regression model ($F(1, 28) = 5.54, p < .05$) and .17 for the quadratic regression model ($F(1, 27) = 2.77, p = .08$) within the group of the observer rats which had been exposed to aggression (indicated by the blue lines in Figure 6). Only the linear regression model was significant although the correlation squared was low. In contrast, for the group of the observer rats which had been exposed to non-aggression (indicated by the red lines in Figure 6), the values of the correlation squared increased to .59 for the linear model ($F(1, 28) = 40.11, p < .01$) and .75 for the quadratic model ($F(2, 27) = 39.83, p < .01$). These group differences in the regression models suggest that the relationship between the initial and follow-up aggression scores was especially stronger among the observer rats exposed to non-

aggression (see Figure 6) than those exposed to aggression. The implication for this finding was discussed in the next section.

Discussion

Study #2 aimed to test my hypothesis that the observer rats chronically exposed to aggression maintained their higher aggression level, compared to controls, across 16 days. Although the significant difference in aggressiveness between the two groups was constant at the initial and follow-up screening tests, both groups significantly decreased their aggressiveness 16 days after recovery from 23-day exposure. On the other hand, these results were not replicated when defense/submission scores of the opponent rats were used as the dependent variable. In other words, the opponent rats showed the same levels of defensive and submissive behaviors across two screening tests, regardless of whether they were paired with an observer rat which had exposed to aggression or non-aggression. Moreover, simple regression analyses showed that the initial aggression scores predicted the follow-up aggression scores, regardless of different conditions. This predictability was especially strong among the observer rats which had been exposed to non-aggression.

These results imply that a mere removal of exposure to aggression from an observer rat's environment could reduce his aggressiveness. However, the results also indicated that, as 16 days passed by, aggressiveness of the observer rat exposed to aggression was still higher than the baseline of aggressiveness shown in controls. Therefore, the results of Study #2 still demonstrated that chronic exposure to aggression had a long-term behavioral effect on an observer rat's aggressiveness, which

characterizes the learning/socializing effect of passive exposure (Huesmann & Kirwil, 2007) although the effect slowly became weakened across times.

It is still unclear how long is required to completely recover aggressiveness induced by chronic passive exposure to aggression. At least, my results of Study #2 suggested that 16-day isolation from aggressive situations was not enough to remedy the negative behavioral effect of chronic exposure to aggression. One strategy to determine the critical time point when the aggressive observer rats show only little aggression is to set up more time points to follow up their aggressiveness until their aggressiveness went back to the baseline of aggressiveness. But, this approach is difficult to control a confounding variable of 'repeated experiences of victories' (Tamashiro et al., 2005). That is, if an observer rat successfully defeats his opponent rat at the first several screening tests, his experiences of repeated victories themselves increase aggressiveness of the observer rat, independent of the behavioral effects of chronic passive exposure to aggression. Accordingly, Study #2 did not set up more than two screening tests.

Figure 6 demonstrated that there was a significant linear relationship between the initial and the follow-up aggression scores. That is, as aggressiveness of the observer rats was high on the initial screening tests, it was predicted as high aggressiveness on the follow-up screening tests (see Figure 6). This relationship was particularly high among the observer rats exposed to non-aggression. This may imply that a trait factor played a role in determining aggressiveness in the control group. For example, the inherently aggressive observer rats in this group showed high on both screening tests; otherwise, the other observer rats showed low on the both tests.

In contrast, there was a greater variability in aggression scores among the observer rats which had been exposed to aggression. This finding may imply that there were moderating variables in the relationship between the initial and follow-up aggression scores in this particular group. These variables might include (1) sensitivity to chronic exposure to aggression and (2) encoding specificity. For instance, all observer rats in the group of chronic exposure to aggression showed high aggressiveness on the initial screening test because of the learning/socializing effect of passive exposure to aggression. It might be possible, however, that only the observer rats with high sensitivity to exposure to aggression well-internalized and held aggressive behavior as their behavioral repertoire across 16 days. To the contrary, those with low sensitivity to exposure to aggression might easily cease to remember what they had perceived for 23 days and, consequently, reduced their aggressiveness 16 days later. That is, an observer rat's sensitivity to exposure to aggression might determine the term duration of the behavioral effects.

Alternatively, encoding specificity might moderate the relationship between the initial and follow-up aggression scores. According to the principle of encoding specificity, an individual tends to remember a past incident which is similar to his or her current situation. Based on previous rat experiments, encoding specificity seemed to be not only a human phenomenon but also a rat phenomenon (see Huesmann & Kirwil, 2007 for discussion). Also, it was found that encoding specificity could play a role in determining whether an observer who is exposed to television violence (i.e., a special type of passive exposure to aggression) becomes aggressive later in his/her life (e.g., Heath et al., 1986). Thus, in Study #1 and #2, if the observer rats successfully defeated

their opponent rat on the initial screening test, they might associate their current status with the status of the resident rats which they had been exposed to. Consequently, they might be more likely to be aggressive and maintain their aggressiveness across 16 days.

In contrast, if the observer rats attempted to attack their opponent rat several times on the initial screening test, but if they were defeated by him after all, they might associate their status with the status of intruder rats which had screamed, fled, and subordinated during exposure sessions. In this case, these observer rats might stop being aggressive and, instead, avoid getting contacted, threatened, and injured when they encountered their opponent rat. Or, the concept of encoding specificity can also explain a case where the observer rats showed low aggressiveness on the initial screening test but high aggressiveness on the follow-up screening test. If the observer rats started to show aggression at almost the end of the initial screening period and could defeat their opponent rat successfully (i.e., low aggression score but dominant), they might successfully project themselves to the resident rats, rather than the intruder rats. Thus, on the follow-up screening test, they started to be actively aggressive from the starting point of exposure session as if they performed as a role of the resident rats.

Finally, in Study #1, I found 'frequent, intermittent, and sudden attacks' in many cases of the observer rats exposed to aggression chronically when their aggressiveness on the initial screening tests was coded. On the follow-up screening tests, nevertheless, I obtained that such aggressive patterns were no longer found among the observer rats exposed to aggression. Figure 7 shows the two behavioral patterns of the observer rats (in the above belt-like box) and their opponent rats (in the below box) which were the same animals as those presented in Figure 3. Within Figure 7, Diagram C shows the

behavioral patterns of the observer rat/opponent rat in the group of chronic exposure to aggression, and Diagram D describes the behavioral patterns of the observer rat/opponent rat in the group of chronic exposure to non-aggression. As the statistical results in Study #2 showed, Figure 7 obviously describes that both observer rats significantly decreased their aggressiveness on the follow-up screening test. In addition, although the observer rat exposed to aggression still displayed aggressive behavior, the frequency of his aggression was reduced, and their attacks were no longer intermittent. Therefore, it is possible that the observer rats exposed to aggression exhibited less affective aggression on the follow-up screening test than on the initial screening test. Instead, these observer rats might be engaged in instrumental aggression more than affective aggression on the follow-up screening tests.

In summary, the observer rats which had been chronically exposed to aggression maintained the high levels of aggression, compared to controls. But, an isolation from exposure to aggression decreased the observer rats' aggressiveness over 16 days. Study #2 also examined the relationship between the initial and follow-up aggression scores within each group. It was found that there was a great variation between these two types of aggression scores within the group of chronic exposure to aggression, implying potential moderating variables between passive exposure to aggression and an observer's aggressiveness. These moderating variables may be the sensitivity to exposure to aggression or the observer's actual status (i.e., dominant or subordinate) on the initial screening tests through encoding specificity.

Table 8. Mean Aggression scores (in Seconds) of Observer Rats and Mean Defense/Submission Scores (in Seconds) of Their Opponent Rats 1 Day and 16 Days After Exposure to Aggression ($n = 30$) and Non-Aggression ($n = 30$)

Condition		Aggression score	Defense/submission score
1 day after exposure	Non-aggression	41.4 (53.7)	70.6 (98.3)
	Aggression	88.2 (63.6)	111.9 (110.8)
16 days after exposure	Non-aggression	24.7 (42.5)	50.2 (102.1)
	Aggression	57.7 (72.1)	98.5 (137.6)

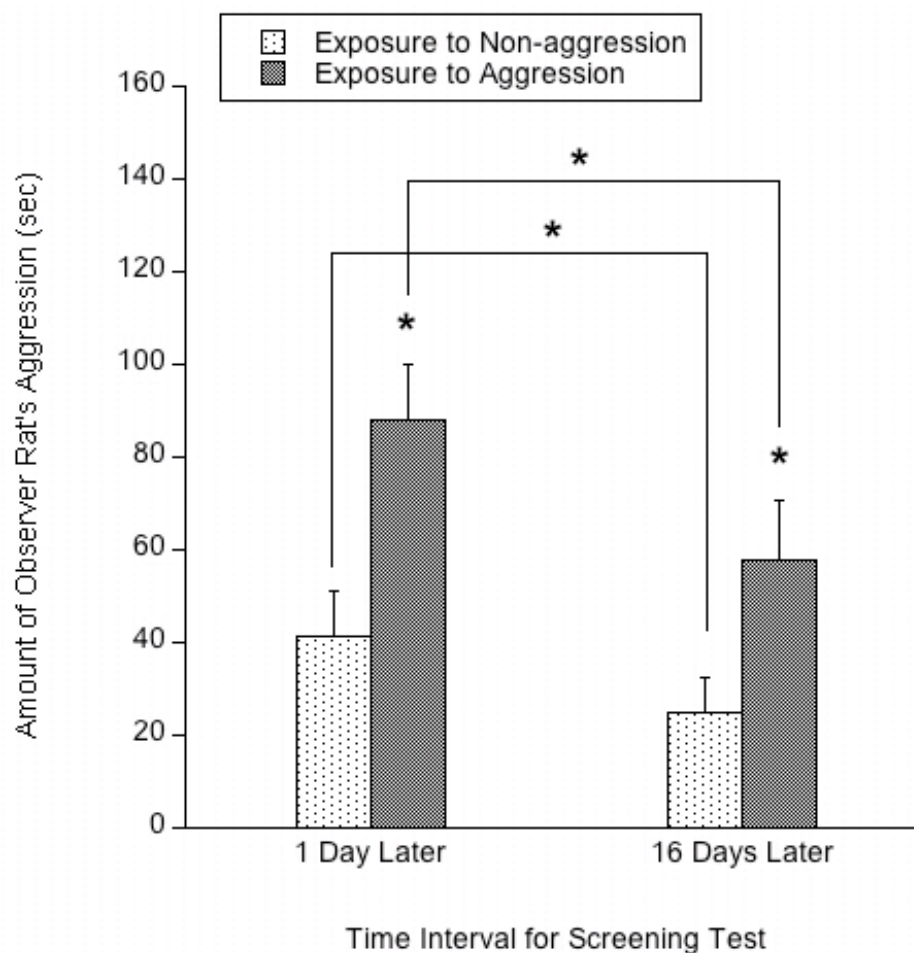
Note. Aggression scores were based on the amount of time (in seconds) when observer rats were engaged in aggressive behavior during a 10-min behavioral screening test. Defense/submission scores were the amount of time (in seconds) when opponent rats were engaged in defensive and submissive behaviors during a 10-min behavioral screening test. Values within parentheses indicate a standard deviation.

Table 9. Repeated-Measures Multivariate Analysis of Variance for Aggression Scores and Defense Scores 1 Day and 16 Days After Exposure ($N = 60$)

Source	Aggression score				Defense/submission score			
	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>
Between subjects								
Group (G)	1	9.02	.14	.00	1	3.15	.05	.08
Within subjects								
Time point for screening tests (T)	1	9.95	.15	.00	1	1.32	.02	.26
G \times T	1	.86	.02	.36	1	.06	.00	.82
S within-group error	58	(1676.65)			58	(6520.07)		

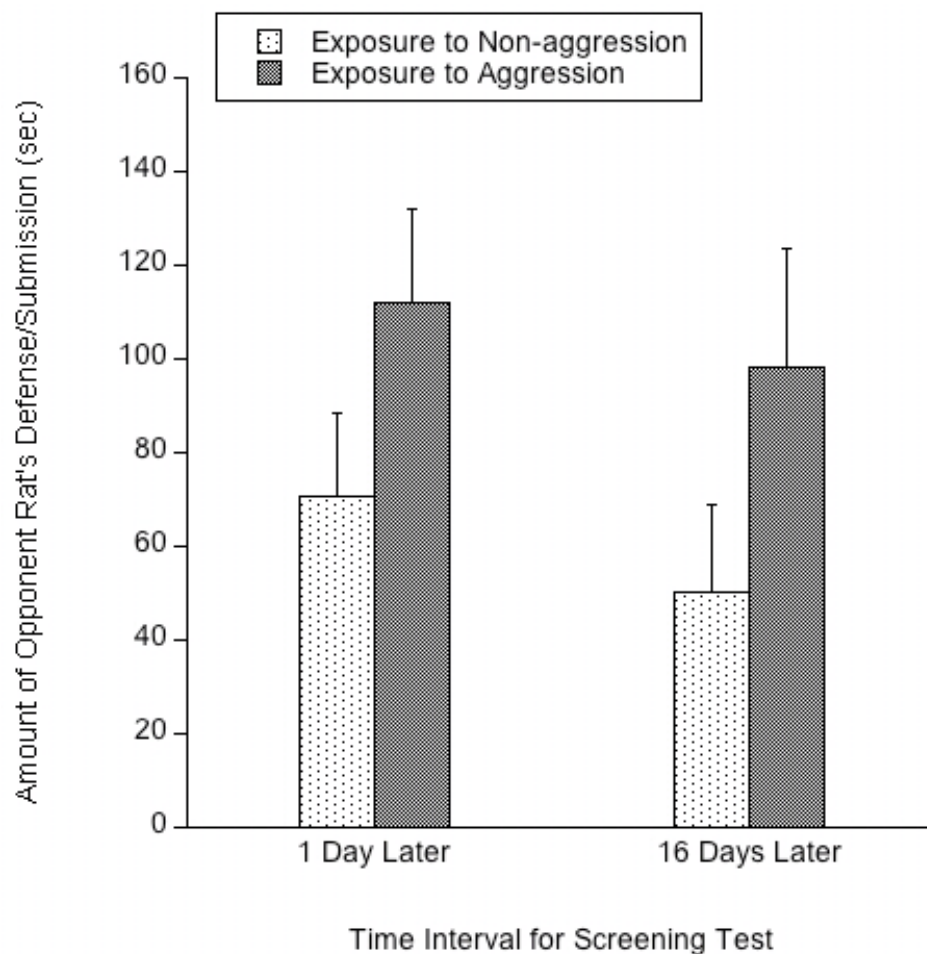
Note. Values within parentheses represent mean square errors. S = subjects.

Figure 4. Longitudinal Comparisons of Mean Aggression Levels (+SE) of Observer Rats 1 Day and 16 Days After Passive Exposure to Aggression ($n = 30$) and Non-Aggression ($n = 30$)



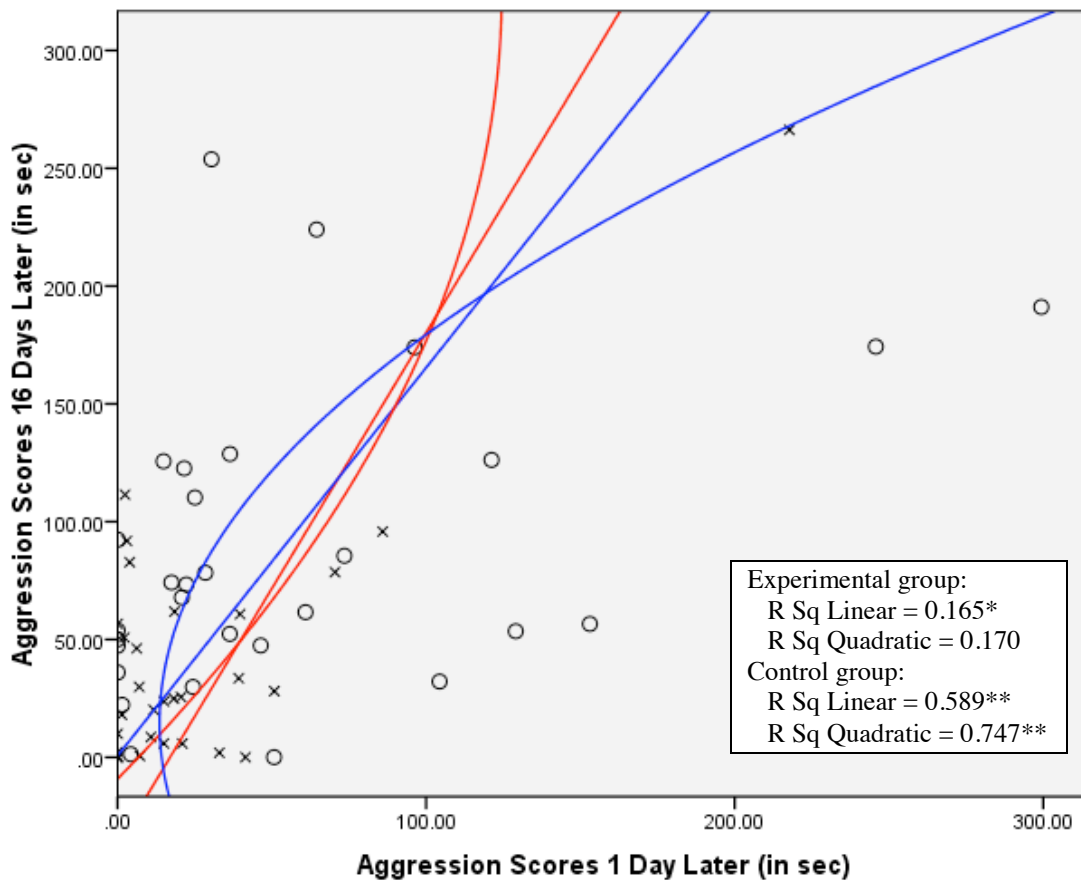
Note. While there was a significant difference between the groups across the time points for screening tests, both groups significantly decreased their aggression scores. Asterisks indicate significant differences from control or a significant difference between the initial and follow-up screening tests within each group (indicated by a line; $p < .05$).

Figure 5. Longitudinal Comparisons of Mean Defense/Submission Levels (+SE) of Opponent Rats 1 Day and 16 Days After Passive Exposure to Aggression ($n = 30$) and Non-Aggression ($n = 30$)



Note. According to a two-way repeated-measures MANOVA, there were no significant differences in defense/submission scores for any pairs, including even the difference between the groups at the initial screening test.

Figure 6. Relationship between the Initial Aggression Scores and the Follow-Up Aggression Scores ($N = 60$)



Note. Circles represent cases of the observer rats exposed to aggression chronically (experimental group); cross marks represent cases of the observer rats exposed to non-aggression chronically (control group). Blue lines indicate the relationship within the experimental group; red lines indicate the relationship within the control group. $R Sq Linear$ = the correlation squared for a linear regression; $R Sq Quadratic$ = the correlation squared for a quadratic regression. The correlation squared for the overall linear regression was .32. $*p < .05$; $**p < .01$

Figure 7. Representative Behavioral Patterns of Observer Rats and Opponent Rats (16 Days After Exposure to Aggression/Non-aggression)

Diagram C.

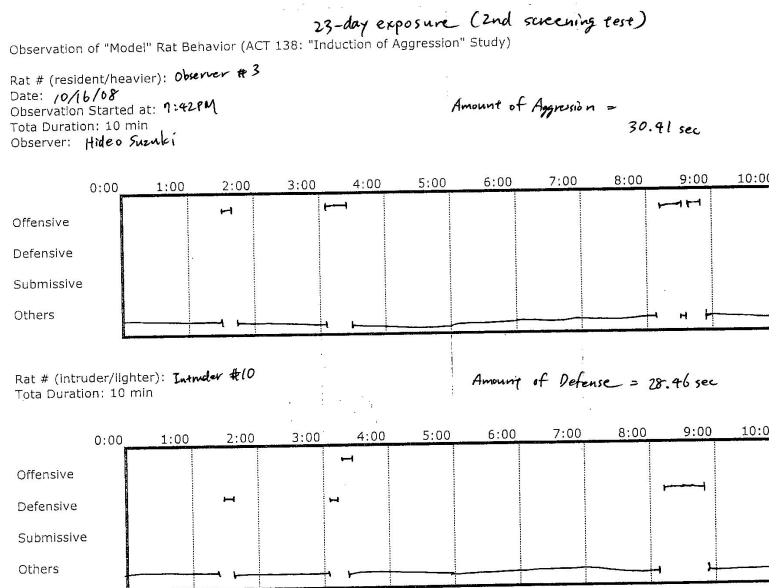
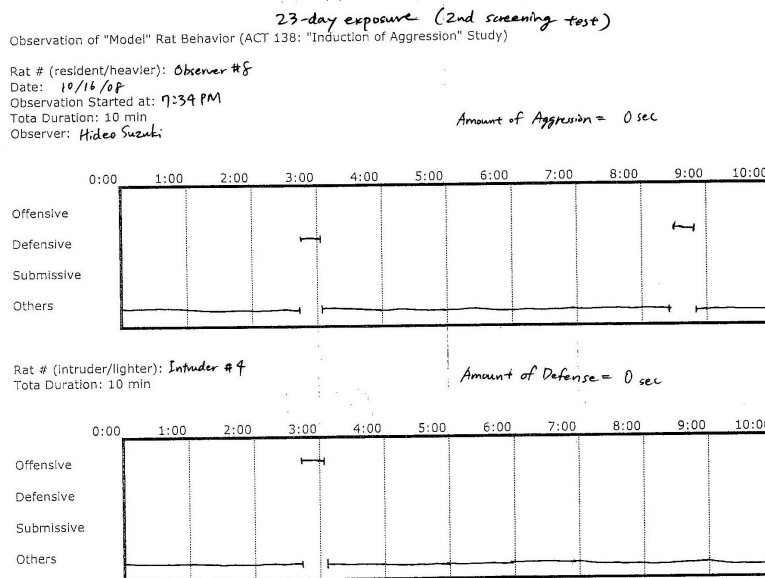


Diagram D.



Note. Diagram C describes the behavioral patterns of an observer rat (in the above box) and his opponent rat (in the below box) 16 days after 23-day exposure to aggression; diagram D describes the behavioral patterns of an observer rat (in the above box) and his opponent rat (in the below box) 16 days after 23-day exposure to non-aggression.

CHAPTER FOUR

STUDY #3: HORMONAL EFFECTS OF EXPOSURE TO AGGRESSION

In Study #1 and #2, it was found that chronic passive exposure to aggression elevated aggressiveness of observer rats. Nevertheless, it is still unclear whether or not chronic exposure to aggression influences physiological/neurobiological systems, which might, in turn, drive observer rats to behave aggressively. One of the possible physiological systems that determine aggressiveness of observer rats is the functions of hormones, especially testosterone and corticosterone. Study #3 examined the concentrations of serum testosterone and corticosterone in response to the four different conditions: single/chronic exposure to aggression/non-aggression. In addition, within the conditions of chronic exposure to aggression and non-aggression, the hormonal levels were assessed at night time (immediately after 23-day exposure sessions) and morning time (15 hours after 23-day exposure sessions).

Method

Participants. Ninety six young male Sprague-Dawley rats, which weighed 337.3 g ($SD = 33.7$) on average at the time of decapitation, were used as observer rats for Study #3. These rats were bred in Animal Care Facilities at Loyola University of Chicago. Also, they were provided a regular care until they were used to collect their blood samples containing hormones.

These observer rats were assigned to one of the following conditions: single exposure to an aggressive dyad ($n = 9$), single exposure to a non-aggressive dyad ($n = 9$), chronic exposure to aggressive dyads ($n = 39$), and chronic exposure to non-aggressive dyads ($n = 39$). For the chronic exposure conditions, the blood samples of six observer rats from each condition were collected immediately after the last exposure session at night time, and the others ($n = 33$ per group) were collected 15 hours after the last exposure session, that is, at morning time. Therefore, there were totally six different conditions in Study #3. The purpose of collecting the blood samples at two time points was to examine the group differences in the hormonal levels that can be fluctuated in a daily cycle (Atkinson, Wood, Kershaw, Bate, & Lightman, 2006; Leal & Moreira, 1997).

In the same way as Study #1, aggressive dyads and non-aggressive dyads were used to manipulate aggressive or non-aggressive situations around the observer rats. To have aggressive dyads, a male rat (i.e., resident rat) had been housed with a female rat for a few weeks before the first exposure session. Simultaneously, non-aggressive dyads, consisting of big and small rats, were also selected and allowed to live together in the same cage a few weeks before the exposure session. The resident rats/non-aggressive dyads were repeatedly used until they stopped showing aggression (for the resident rats) or started to show aggression (for the non-aggressive dyads).

Design and Procedure. When every exposure session took place under a red light illumination between 7:00PM and 9:00PM, each observer rat was transferred from his own home cage ($47 \times 25.5 \times 21.5$ cm) into a small plastic aquarium with a mesh lid ($22.9 \times 15 \times 16.5$ cm). This aquarium was then placed into a home cage ($47 \times 25.5 \times 21.5$ cm)

of either the resident-female pair or the non-aggressive dyads. For the groups of exposure to aggression, the female rat was further replaced with a naïve male rat (i.e., intruder rat) so that the resident rat and the intruder rat formed as an aggressive dyad. Note that the intruder rat was approximately 100 g smaller than the resident rat. Each exposure session took 10 min. All exposure sessions were recorded by a video camera.

After a 10-min. exposure session, the observer rats in the single exposure groups were immediately decapitated, and their blood samples were collected and stored on ice temporarily. For the chronic exposure groups, the observer rats were transferred back to their original home cages every time an exposure session ended. These procedures had been repeated for 23 days, and then the observer rats were decapitated immediately or 15 hours after the last exposure session, depending on the conditions. Again, their blood was sampled and stored on ice temporarily. After the blood samples were all collected, they were centrifuged at 2,500 rpm at 4°C for 15 min. so that serum was separated from the blood. Serum for each observer rat was sampled and stored at -12°C until it was used.

To analyze the levels of serum testosterone and corticosterone, radioimmunoassay was conducted by using the commercially available radioimmunoassay kits, Coat-A-Count Testosterone and Coat-A-Count Cortisol (Diagnostic Products Corporation, Los Angeles, CA). Radioimmunoassay procedure was instructed by the kits.

Radioimmunoassay was performed in the following ways. First of all, the serum samples were thawed and gently mixed at room temperature. Serum of each observer rat was transferred into antibody-coated tubes in duplicate for testing each hormone. In addition, the same amount of total counts, nonspecific binding, and several calibrators

(from 0-1,600 ng/dL for testosterone; 0-2,000 ng/mL for corticosterone) were prepared in tubes (total counts and nonspecific binding in uncoated tubes; calibrators in coated tubes) in duplicate. Then, radioactive ^{125}I Total Testosterone (for testing testosterone) or ^{125}I Rat Corticosterone (for testing corticosterone) was added to every tube. After mixing each tube, all tubes were incubated either for 3 hours at 37°C (for testing testosterone) or for 2 hours at room temperature (for testing corticosterone). After incubation was done, the solution contained in each tube was removed thoroughly, and the tube was counted for 1 min. in a gamma counter. Based on a logit-log calibration curve, which was drawn from the data of (1) radioactive counts and (2) the already-known concentrations of the calibrators, the concentrations of serum testosterone and corticosterone were calculated.

Statistical Strategy. An independent-samples *t*-test was used to check if the groups of exposure to aggression were overall exposed to more average amount of aggressive incidents per day than the groups of exposure to non-aggression.

Two sets of two-way MANOVA's were used to test two dependent variables: the concentrations of serum testosterone and corticosterone. The first two-way MANOVA examined the interaction effect of group (exposure to aggression vs. non-aggression) \times exposure amount (single vs. chronic) between the groups of single and chronic exposure to aggression/non-aggression immediately after the last exposure session. The second MANOVA analyzed the interaction of group (chronic exposure to aggression vs. non-aggression) \times the time of collection (immediately vs. 15 hours after exposure sessions) between the groups of chronic exposure to aggression/non-aggression immediately and 15 hours after 23-day exposure.

Finally, two sets of one-way MANOVA's with Bonferroni-typed a posteriori comparisons were performed to compare the mean concentrations of serum testosterone and corticosterone among all six conditions.

Results

According to the results of *t*-test, the manipulation was successful; on average, the observer rats which were assigned to exposure to aggression ($\bar{X} = 143.7$ seconds) were exposed to aggression more than those which were assigned to exposure to non-aggression ($\bar{X} = 1.3$ seconds; $t(94) = 7.70, p < .01$).

Table 10 summarizes the descriptive statistics of the concentrations of serum testosterone and corticosterone across the groups. The mean levels of serum testosterone were 202.17 ng/dL ($SD = 125.65$) for the group of single exposure to non-aggression, 156.40 ng/dL ($SD = 81.76$) for the group of single exposure to aggression, 368.89 ng/dL ($SD = 189.94$) for the group of chronic exposure to non-aggression at night time (immediately), 372.72 ng/dL ($SD = 254.91$) for the group of chronic exposure to aggression at night time (immediately), 460.82 ng/dL ($SD = 279.35$) for the group of chronic exposure to non-aggression at morning time (15 hours later), and 390.66 ng/dL ($SD = 281.04$) for the group of chronic exposure to aggression at morning time (15 hours later).

For the concentrations of serum corticosterone, the mean levels were the following: 450.41 ng/mL ($SD = 59.64$) for the single exposure to non-aggression, 500.83 ng/mL ($SD = 80.54$) for the single exposure to aggression, 370.85 ng/mL ($SD = 55.76$) for the chronic exposure to non-aggression at night time (immediately), 415.97 ng/mL ($SD = 98.79$) for the chronic exposure to aggression at night time (immediately), 26.30

ng/mL ($SD = 15.45$) for the chronic exposure to non-aggression at morning time (15 hours later), and 35.29 ng/mL ($SD = 50.13$) for the chronic exposure to aggression at morning time (15 hours later).

According to a two-way MANOVA for the interaction of group \times exposure amount (see Table 11), there were only the main effects of exposure amount on the levels of both testosterone and corticosterone ($F(1,26) = 10.02, p < .01$ for testosterone; $F(1,26) = 8.74, p < .01$ for corticosterone). Thus, the concentration of serum testosterone was higher in the chronic exposure groups ($\bar{X} = 370.81$ ng/dL) than the single exposure groups ($\bar{X} = 179.29$ ng/dL). The opposite pattern was shown in the concentration of serum corticosterone; it was the single exposure groups ($\bar{X} = 475.62$ ng/mL) which showed higher levels of corticosterone than the chronic exposure groups ($\bar{X} = 393.41$ ng/mL).

When the interaction of group \times time of collection was tested (see Table 12), MANOVA yielded a significant main effect of time of collection for only the concentration of serum corticosterone. That is, both chronic exposure groups showed higher levels of corticosterone at night time (immediately; $\bar{X} = 393.41$ ng/mL) than at morning time (15 hours later; $\bar{X} = 30.80$ ng/mL). This significant main effect simply resulted from a daily cycle of corticosterone levels.

In testing individual comparisons among mean levels of serum testosterone and corticosterone ($F(5,90) = 2.99, p < .05$ for testosterone; $F(5,90) = 275.89, p < .01$), the significant differences were found between (1) the groups of single exposure to aggression and chronic exposure to non-aggression at morning time in the levels of testosterone, (2) the groups of single exposure to aggression and each of chronic exposure

conditions in the levels of corticosterone, and (3) the groups of single exposure to non-aggression and each of chronic exposure conditions at morning time in the levels of corticosterone (all Bonferroni-typed p 's < .05; see Figure 8 & 9).

Discussion

Study #3 analyzed the hormonal responses, especially testosterone and corticosterone, to single or chronic exposure to aggression/non-aggression. For the single exposure conditions, the hormonal levels were tested at night time (i.e., immediately after the exposure session); for the chronic exposure conditions, the concentrations of the hormones were assayed at night time (i.e., immediately after the last exposure) and at morning time (i.e., 15 hours after the last exposure). It was found that, immediately after exposure(s), the groups of single exposure showed lower levels of testosterone but higher levels of corticosterone than the groups of chronic exposure. In addition, the observer rats in chronic exposure groups significantly decreased their corticosterone levels at morning time, compared to night time. On the other hand, no differences in the levels of testosterone and corticosterone were found between the groups of exposure to aggression and non-aggression, regardless of the exposure amount and the time of collection.

Table 11 and 12 showed that there was no difference in the levels of testosterone and corticosterone between the groups of the chronic exposure to aggression and non-aggression, in spite of time of collection (also see Figure 8). On the other hand, Study #1 and #2 demonstrated that there was a significant difference in aggressiveness of observer rats between the groups of chronic exposure to aggression and non-aggression. Taken together, the behavioral difference in aggressiveness of observer rats in the chronic exposure groups did not seem to result from the hormonal difference in testosterone or

corticosterone. In other words, testosterone and corticosterone did not mediate the link between chronic passive exposure to aggression and aggressiveness of observer rats.

On the other hand, the differences in the hormonal levels between the single exposure conditions and the chronic exposure conditions were obtained (see Table 11 and Figure 8). In previous studies, aggression is associated with the high levels of testosterone (for a review, see Van Goozen, 2005). Consistent with this, Study #1 found the main effect of exposure amount, and Study #3 showed that the chronic exposure groups exhibited higher levels of testosterone than the single exposure groups. Thus, for some reasons (e.g., frustration that was related to repeated physical separations between the observer rats and the aggressive/non-aggressive dyads for 23 days), chronic exposure conditions accelerated the concentrations of serum testosterone, which might result in the difference in aggressiveness of the observer rats between the chronic exposure conditions and the single (acute) exposure conditions. Interestingly, the chronic exposure groups maintained their higher levels of testosterone even 15 hours (at morning time) after 23-day exposure, compared to those of the single exposure groups immediately after the exposure (at night time). Given the fact that the levels of testosterone usually decrease from night time to morning time, (Leal & Moreira, 1997), it is noteworthy that the levels of testosterone among the chronic exposure groups did not seem to be fluctuated in a daily cycle.

In addition, the levels of testosterone among the chronic exposure groups were higher than the baseline of testosterone levels (indicated by a red dotted line in Figure 8). According to Leal and Moreira (1997), the basal levels of plasma testosterone was 70.7 ± 10.9 ng/dL at morning time (8:00AM) and 243 ± 42.2 ng/dL at night time (4:00PM). In

comparisons, the chronic exposure groups showed 370.8 ng/dL at night time and 425.75 ng/dL at morning time. Based on Study #3, it was unclear why the chronic exposure groups had higher levels of testosterone than the basal levels.

For the concentrations of serum corticosterone, the chronic exposure groups showed lower levels of corticosterone than the single exposure groups when their blood samples were collected immediately after exposure session(s) (see Table 11 and Figure 9). Some previous findings have shown that aggression is negatively associated with the levels of corticosterone (for a review, see Van Goozen, 2005). Based on the results in Study #1 that there was the main effect of exposure amount in aggression scores, it may be able to be interpreted that the chronic exposure groups behaved more aggressively with low fear than the single exposure groups.

However, although the chronic exposure groups showed lower corticosterone levels than the single exposure groups at night time, all of the four groups showed higher levels of corticosterone than the baseline levels (indicated by a red dotted line in Figure 9). Atkinson et al. (2006) reported that the average levels of blood corticosterone were almost no detectable concentration at morning time and approximately 40 ng/mL at night time. These high levels of corticosterone might lead the observer rats in the chronic exposure groups to display excessive affective aggression (Haller & Kruk, 2006). This implication is somewhat consistent with the results in Study #1 that the observer rats exposed to aggression showed intermittent aggression, characterizing one type of affective aggression. But, the observer rats exposed to non-aggression did not show such intermittent aggression, thus it is still unknown what an additional factor might contribute to differentiating affective aggression of the group of 23-day exposure to aggression from

non-affective aggression (probably instrumental aggression of the group of 23-day exposure to non-aggression).

Finally, to the contrary to testosterone, the chronic exposure conditions significantly lowered their levels of corticosterone at morning time, compared to night time (see Table 12 and Figure 9).

One limitation in Study #3 was that some groups, especially the chronic exposure conditions of which the blood samples were collected at night time, had a small sub-sample size. Consequently, the standard deviations in these conditions were relatively large, and the small sub-sample sizes in the present study could lower statistical power. For the future research, it is necessary to increase sample sizes to have equal group sizes across different conditions.

In summary, Study #3 examined the concentrations of two hormones – testosterone and corticosterone – in response to single/chronic exposure to aggression/non-aggression. It was found that the chronic exposure groups generally displayed the higher levels of testosterone and lower levels of corticosterone than the single exposure groups. However, there were no differences in these hormonal levels between the groups of exposure to aggression and non-aggression, regardless of different exposure amounts and times of blood collection. Therefore, the difference in aggressiveness of the observer rats between the groups of chronic exposure to aggression and non-aggression, obtained by Study #1, was not associated with the levels of serum testosterone and corticosterone.

Table 10. Mean Concentrations of Serum Testosterone (ng/dL) and Corticosterone (ng/mL) across Conditions

Condition		Testosterone	Corticosterone	
Single exposure to...	Non-aggression	Immediately	202.2 (125.6)	450.4 (59.6)
	Aggression	Immediately	156.4 (81.8)	500.8 (80.5)
Chronic exposure to...		Immediately	368.9 (189.9)	370.8 (55.8)
	Non-aggression	15 hours later	460.8 (279.4)	26.3 (15.5)
		Immediately	372.7 (254.9)	416.0 (98.8)
	Aggression	15 hours later	390.7 (281.0)	35.3 (50.1)

Note. Values within parentheses indicate a standard deviation.

Table 11. Multivariate Analysis of Variance for the Concentrations of Serum Testosterone and Corticosterone between Single and Chronic Exposure Conditions ($N = 30$)

Source	Testosterone				Corticosterone			
	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>
Between subjects								
Group (G)	1	.12	.01	.73	1	2.95	.10	.10
Exposure amount (E)	1	10.02	.28	.00	1	8.74	.25	.01
G \times E	1	.17	.01	.69	1	.01	.00	.93
S within-group error	26	(26348.68)			26	(5565.04)		

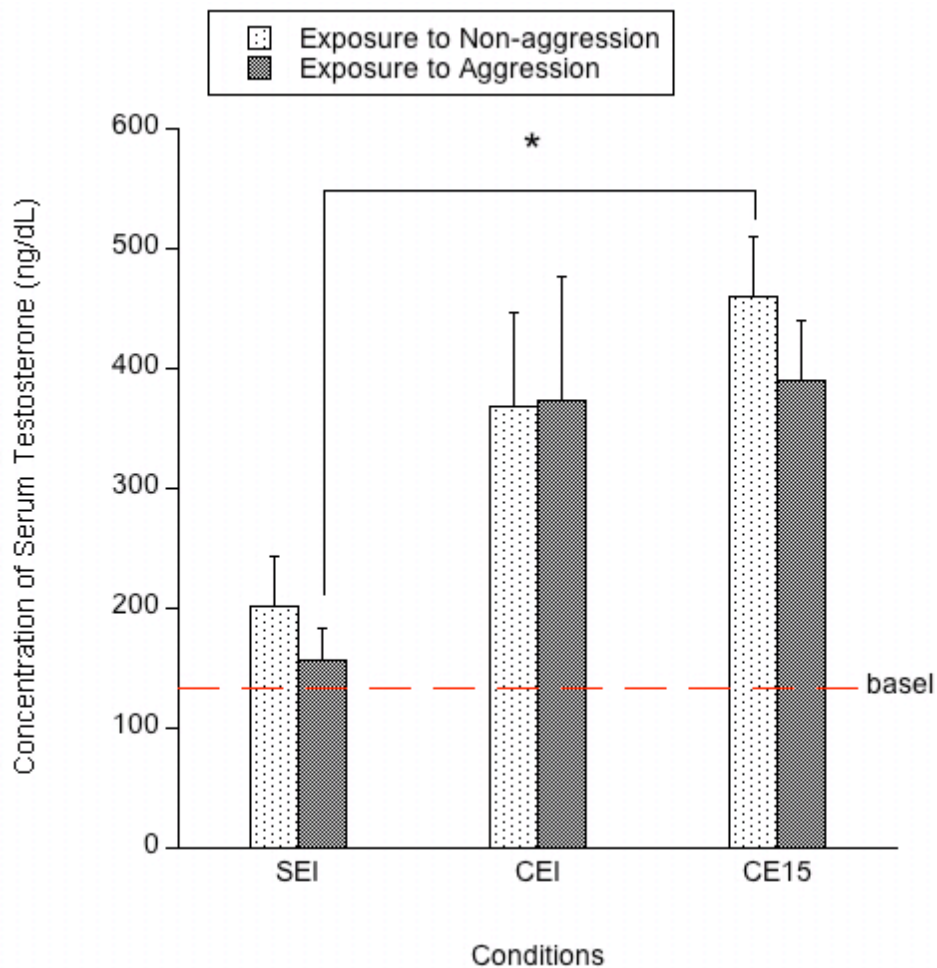
Note. Values within parentheses represent mean square errors. S = subjects.

Table 12. Multivariate Analysis of Variance for the Concentrations of Serum
 Testosterone and Corticosterone between Night Time and Morning Time ($N = 78$)

Source	Testosterone				Corticosterone			
	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>
Between subjects								
Group (G)	1	.149	.00	.70	1	3.61	.05	.06
Time of Collection (T)	1	.410	.01	.52	1	648.24	.90	.00
G \times T	1	.19	.00	.67	1	1.61	.02	.21
S within-group error	74	(74730.66)			74	(2059.60)		

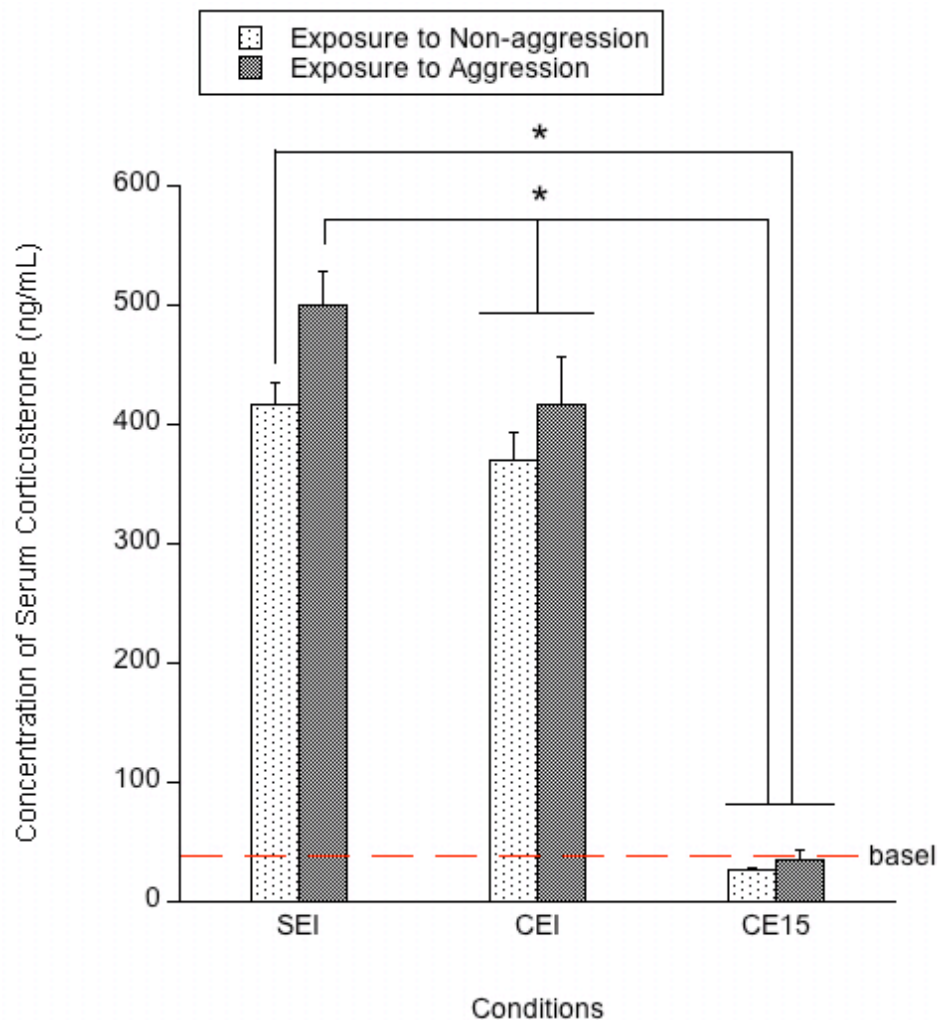
Note. Values within parentheses represent mean square errors. S = subjects.

Figure 8. Mean Comparisons in the Concentrations of Serum Testosterone among Single/Chronic Exposure to Aggression/Non-Aggression ($N = 96$)



Note. SEI = Single exposure conditions with testosterone collected immediately after a one-time exposure session; CEI = Chronic exposure conditions with testosterone collected immediately after the last exposure; CE15 = Chronic exposure conditions with testosterone collected 15 hours after the last exposure session. Red dotted line indicates the basal level of testosterone at night time.

Figure 9. Mean Comparisons in the Concentrations of Serum Corticosterone among Single/Chronic Exposure to Aggression/Non-Aggression ($N = 96$)



Note. SEI = Single exposure conditions with corticosterone collected immediately after a one-time exposure session; CEI = Chronic exposure conditions with corticosterone collected immediately after the last exposure; CE15 = Chronic exposure conditions with corticosterone collected 15 hours after the last exposure session. Red dotted line indicates the basel level of corticosterone at night time.

CHAPTER FIVE

STUDY #4: RECEPTOR DENSITIES IN RESPONSE TO EXPOSURE TO AGGRESSION

Although Study #3 could not find any hormonal responses to chronic exposure to aggression, the neurotransmitter systems are the other candidates of neurochemical pathways mediating the relationship between passive exposure to aggression and observer rats' aggressiveness. Previous neurobiological studies on aggression have focused on dopamine D₂ receptors and 5-HT_{1B} receptors. Thus, Study #4 examined their receptor densities in relation to chronic passive exposure to aggression. It was hypothesized that chronic passive exposure to aggression changed the densities of dopamine D₂ receptors and 5-HT_{1B} receptors.

Method

Participants. The subjects in Study #4 were young male Sprague-Dawley rats which were obtained from some of the observer rats used in the chronic exposure conditions and decapitated 15 hours after 23-day exposure in Study #3 ($N = 30$). The mean of their body weights was 305.7g at the time of decapitation. A half of those rats was assigned to 23-day exposure to aggressive dyads ($n = 15$), and the other remaining rats were exposed to non-aggressive dyads for 23 days ($n = 15$).

Design and Procedure. At the time of decapitating the observer rats and collecting their blood samples (15 hours after the last day of 23-day exposure sessions) in Study #3, their brains were also removed rapidly from their heads, frozen on powdered dry ice, and stored at -70°C until they were used for analyses.

Specifically, Study #4 analyzed the densities of dopamine D_2 receptors in the nucleus accumbens and its surrounding areas of the rat brain and the densities of $5\text{-HT}_{1\text{B}}$ receptors in the hypothalamus and its surrounding areas. The nucleus accumbens is known as the brain region where dopamine D_2 receptors are widely diffused (Di Matteo et al., 2008). In addition, dopaminergic neurons in the nucleus accumbens are involved in a neural pathway that is linked to aggression (Blum et al., 1996). From these reasons, Study #4 targeted the nucleus accumbens and, for interests, the other neighboring regions at the anterior brain for testing the densities of dopamine D_2 receptors.

For the densities of $5\text{-HT}_{1\text{B}}$ receptors, the hypothalamus was selected as the target region. The serotonergic systems, including $5\text{-HT}_{1\text{B}}$ receptors, are especially concentrated in the hypothalamus, as well as hippocampus and amygdala (Di Matteo et al., 2008). Blum et al. (1996) suggest that, in a neural mechanism of aggressive behavior, aggression is first initiated by serotonergic neurotransmissions in the hypothalamus. Moreover, these areas are also known as the important areas being involved in aggression (Nelson & Trainor, 2007; Spont, 1992). Thus, it is reasonable to examine the densities of $5\text{-HT}_{1\text{B}}$ receptors in the hypothalamus, hippocampus, and amygdala at the posterior part of the brain.

At the time of sectioning the brain samples, interaural sections of $20\ \mu\text{m}$ thickness were cut on a cryostat at -15°C and thaw-mounted onto gelatin-coated slides. Because

the target areas of the brain in Study #4 were the nucleus accumbens (located at the anterior part of the rat brain) and hypothalamus (located at the posterior part of the rat brain), the sections were taken from areas between 2.52 mm and 1.56 mm prior to bregma for the anterior sections and between 1.72 mm and 2.68 mm posterior to bregma for the posterior sections. Twelve slides (four sections per slide) were collected for each of the anterior and posterior parts of every observer rat, and they were stored at -70°C until they are used.

After the brain sections were collected, autoradiography was performed. The procedure for dopamine D_2 receptor binding autoradiography was adopted from Aragona et al.'s (2006) study, and the procedure for 5-HT_{1B} receptor binding autoradiography was obtained by referring to Svenningsson et al.'s (2005) study. First, only four best slides out of 12 obtained from each brain part were selected (i.e., four best slides including 16 anterior sections and four best slides including 16 posterior sections). In addition, two extra slides from each set of the anterior and posterior sections were selected for nonspecific binding. In the selection process, the slides were cross-matched so that each of the anterior and posterior parts of the brain were compatible and could be evaluated between the observer rats exposed to aggression and non-aggression.

The anterior sections (for dopamine D_2 receptor binding) were rinsed twice with 50 mM of Tris-HCl (with pH 7.4) for 10 min. The posterior sections (for 5-HT_{1B} receptor binding) were not rinsed at this point. Then, both anterior and posterior sections were incubated in a buffer solution at room temperature for 90 min. (for the anterior sections) or 120 min. (for the posterior sections). The buffer solution for the anterior sections contained 50 mM of Tris-HCl (pH 7.4), 120 mM of NaCl, 5 mM of KCl, 2 mM

of CaCl₂, 1 mM of MgCl₂, 100 pM of [¹²⁵I]2'-iodospiperone (i.e., a radioactive ligand for dopamine D₂ receptor binding), and 50 nM of ketanserin. For nonspecific binding, 100 μM of SCH23390 and 100 μM of raclopride were added into the above buffer solution. For the posterior sections, the buffer solution contained 170 mM of Tris-HCl (pH 7.4), 150 mM of NaCl, 50 pM of [¹²⁵I]cyanopindolol (i.e., a radioactive ligand for 5-HT_{1B} receptor binding), 100 nM of 8-OH-DPAT, and 30 μM of isoproterenol. For nonspecific binding, 100 μM of 5-HT were added to this buffer solution.

After the incubation, each of dopamine D₂ and 5-HT_{1B} receptor binding sites was radio-labeled with the selective ligand. Then, the sections were rinsed in cold 50mM of Tris-HCl (pH 7.4) three times for 10 min. per wash (for the anterior sections) or cold binding buffer solution two times for 5 min. per wash (for the posterior sections). Afterwards, the sections were quickly dipped in ice-cold double-distilled H₂O for 5 sec.. (for the posterior sections, this process of dipping was performed at 4°C) and dried under a stream of cool air. Once all sections were completely dried, the slides were put in cassettes and exposed to BioMax MR film (Kodak) and left under a dark area for 8 hours (for the anterior sections) or 88 hours (for the posterior sections).

After the X-ray film exposure, all films were developed in Kodak D-19 developer for 5 min., placed in a water bath for 1 min., fixed for 10 min. in Kodak Rapid Fixer, and then placed in a water bath again for 10 min. with hardener. When these films were dried, computer-assisted densitometry was used to analyze autoradiograms. Relative densities were measured by using a 10-point calibration scale based on light intensity. The darker areas of the brain indicate the higher densities of the target receptors (see Figure 11 for the dopamine D₂ receptor binding autoradiograms and Figure 13 for the 5-

HT_{1B} receptor binding autodiagrams). Only four to six best sections were selected and scanned, and the final density value was computed by (1) subtracting the local background intensity from the selected area and (2) averaging all density values from the selected area. In these image analyses, Study #4 examined the densities of dopamine D₂ receptors in the (1) dorsolateral caudate putamen, (2) dorsomedial caudate putamen, (3) the core of the nucleus accumbens, (4) the shells of the nucleus accumbens, (5) cingulate area 1, (6) cingulate area 2, (7) primary motor cortex, and (8) secondary motor cortex of both right and left hemispheres of the anterior sections. Moreover, the densities of 5-HT_{1B} receptors were examined by analyzing the (1) lacunosum-molecular layer of the hippocampus, (2) hypothalamus, and (3) anterior basolateral amygdala.

Statistical Strategy. The two sets of two-way repeated-measures MANOVA's were performed to test the interaction effect of group (exposure to aggression vs. non-aggression) × hemisphere (right hemisphere vs. left hemisphere). The first MANOVA analyzed the mean density values of dopamine D₂ receptors in eight different regions: the dorsolateral caudate putamen, the dorsomedial caudate putamen, the core of the nucleus accumbens, the shells of the nucleus accumbens, the cingulate area 1, the cingulate area 2, the primary motor cortex, and the secondary motor cortex. The second MANOVA tested the mean density values of 5-HT_{1B} receptors in three different brain regions: the lacunosum-molecular layer of the hippocampus, the hypothalamus, and the anterior basolateral amygdala.

Moreover, the several tests of one-way ANOVA's analyzed the individual comparisons in the mean density values of each of dopamine D₂ receptors and 5-HT_{1B}

receptor between the groups of chronic exposure to aggression and non-aggression across the different brain regions of each hemisphere.

Results

The mean density values of dopamine D₂ receptors in different brain regions were summarized in Table 13. The mean density values of 5-HT_{1B} receptors across the brain regions were described in Table 14. Overall, D₂ receptors were especially concentrated in the dorsolateral caudate putamen (overall $\bar{X} = 2.50$ on the right hemisphere; overall $\bar{X} = 3.68$ on the left hemisphere), the dorsomedial caudate putamen (overall $\bar{X} = 2.22$ on the right hemisphere; overall $\bar{X} = 1.73$ on the left hemisphere), the core of the nucleus accumbens (overall $\bar{X} = 1.65$ on the right hemisphere; overall $\bar{X} = 1.56$ on the left hemisphere), and the shells of the nucleus accumbens (overall $\bar{X} = 1.68$ on the right hemisphere; overall $\bar{X} = 1.66$ on the left hemisphere). On the other hand, 5-HT_{1B} receptors were widely located in the hypothalamus (overall $\bar{X} = 1.94$ on the right hemisphere; overall $\bar{X} = 1.91$ on the left hemisphere) and the anterior basolateral amygdala (overall $\bar{X} = 1.79$ on the right hemisphere; overall $\bar{X} = 1.53$ on the left hemisphere).

For testing the mean density values of dopamine D₂ receptors, the main effects of group (exposure to aggression vs. non-aggression) were detected in the following brain areas: the shells of the nucleus accumbens ($F(1, 26) = 4.94, p < .05$), the primary motor cortex ($F(1, 26) = 5.12, p < .05$), and the secondary motor cortex ($F(1, 26) = 8.18, p < .05$; see Figure 15). Furthermore, the main effects of hemisphere (right hemisphere vs. left hemisphere) were found in the dorsolateral caudate putamen ($F(1, 26) = 254.68, p$

< .01), the dorsomedial caudate putamen ($F(1, 26) = 188.24, p < .01$), the core of the nucleus accumbens ($F(1, 26) = 9.66, p < .05$), the cingulate area 1 ($F(1, 26) = 5.17, p < .05$), and the cingulate area 2 ($F(1, 26) = 6.81, p < .05$). However, none of the brain areas at the anterior part showed a significant interaction effect.

Table 16 indicates the results of testing interaction effects in the mean density values of 5-HT_{1B} receptors. There were the main effect of group in the anterior basolateral amygdala ($F(1, 26) = 10.94, p < .01$) and the main effects of hemisphere in the lacunosum molecular layer of the hippocampus and the anterior basolateral amygdala. But, there was no interaction effect of group \times hemisphere.

Table 17 shows individual difference in the mean density values of dopamine D₂ receptors between the groups of chronic exposure to aggression and non-aggression across the different brain regions (also see Figure 10). The group differences in the densities of dopamine of D₂ receptors were found in the following brain regions: the shells of the nucleus accumbens on both hemispheres ($F(1, 28) = 7.95, p < .01$ for the right hemisphere; $F(1, 28) = 4.77, p < .05$ for the left hemisphere), (2) primary motor cortex in the right hemisphere ($F(1, 27) = 5.84, p < .05$), (3) secondary motor cortex in both hemispheres ($F(1, 28) = 8.46, p < .01$ for the right hemisphere; $F(1, 27) = 5.03, p < .05$ for the left hemisphere), and (4) cingulate area 1 in the left hemisphere ($F(1, 28) = 4.30, p < .05$). In all of these brain areas, the observer rats which had been exposed to aggression showed lower densities of dopamine D₂ receptors than those which had been exposed to non-aggression (for the mean density values, see Table 13). Figure 11 shows a visual comparison in D₂ receptor densities between the two groups.

For the densities of 5-HT_{1B} receptors, the observer rats exposed to aggression showed higher densities in the anterior basolateral amygdala on both hemispheres than controls ($F(1, 26) = 19.05, p < .01$ for the right hemisphere; $F(1, 26) = 5.14, p < .05$ for the left hemisphere; see Table 16 and Figure 12). But, the lacunosum molecular layer of the hippocampus and the hypothalamus did not reveal a significant difference in the mean density value between the two groups. Figure 13 presents the posterior brain sections of two observer rats indicating the difference in the densities of 5-HT_{1B} receptors between the groups.

Discussion

The purpose of Study #4 was to investigate the differences in the densities of two receptors – dopamine D₂ receptors and 5-HT_{1B} receptors – between the observer rats which had been chronically exposed to aggression and non-aggression. Autoradiography of these receptor bindings designated that the group of chronic exposure to aggression showed lower densities of dopamine D₂ receptors in the shells of the nucleus accumbens, the primary motor cortex (The results of the two-way MANOVA revealed a significant main effects of group in the primary motor cortex across both hemispheres although the results of the one-way ANOVA indicated that the significant group difference existed only in the right hemisphere), and the secondary motor cortex on both hemispheres. The cingulate area 1 also showed the difference in the density value between the groups although this difference appeared in only the left hemisphere. Moreover, the higher densities of 5-HT_{1B} receptors were found in the anterior basolateral amygdala among the observer rats exposed to aggression, compared to controls.

First of all, Study #4 demonstrated that the observer rats exposed to aggression for 23 days showed significantly lower densities of dopamine D₂ receptors in the shells of the nucleus accumbens. Combined this with the results in Study #1 and #2, chronic exposure to aggression is linked to (1) high aggressiveness of observer rats and (2) the low densities of dopamine D₂ receptors in the brain of observer rats. This finding was consistent with White et al.'s (2008) genetic study that the A1 allele, which programs to reduce the densities of dopamine D₂ receptors, was associated with aggression. If these dopamine D₂ receptors have the low densities postsynaptically, it could lead to the low dopaminergic neurotransmissions. Thus, based on Blum et al.'s (1996) hypothesis of reward deficiency syndrome, the observer rats exposed to aggression might experience the low dopaminergic neurotransmissions and suffer from what is called 'reward deficiency syndrome,' where they might have unpleasant feelings and need to restore the sufficient amounts of dopamine in the nucleus accumbens. According to Van Erp and Miczek (2000), aggression temporarily promotes the release of dopamine in the shells of the nucleus accumbens. Hence, when the observer rats chronically exposed to aggression encountered their opponent rat, and when they had the low densities of (postsynaptic) dopamine D₂ receptors that led to low dopaminergic neurotransmissions in the shells of the nucleus accumbens, they would tend to choose aggressive responses toward him to recover or enhance dopaminergic neurotransmissions.

It was also interesting that the primary and secondary motor cortices showed significant group differences in the densities of dopamine D₂ receptors. If the low densities of dopamine D₂ receptors in these motor cortices also contributed to aggressiveness of the observer rats which had been exposed to aggression, Spont's

(1992) hypothesis of signal-to-noise ratio would theoretically explain this association between aggression and dopamine D₂ receptors. In Spont's hypothesis, the high activity of dopaminergic neurotransmissions increases noise acting on redundant motivational/motor systems, including aggressive behavior. Stated differently, however, the low densities of (postsynaptic) dopamine D₂ receptors in the motor areas might also increase noise; the low densities of these receptors may decrease their capability to detect dopamine neurotransmitters from presynaptic neurons, thus the leftover dopamine neurotransmitters may 'spill out' and excite the other parts of postsynaptic neurons in the motor cortices that may be involved in regulating aggressive acts.

As discussed in Chapter 1, previous neuropharmacological studies provided the contradictory evidence that the D₂ receptor antagonists, which function to deactivate dopamine D₂ receptors, generally reduce aggression (for a review, see De Almeida et al., 2005). This evidence claims against my suggestion that aggression results from the low densities of dopamine D₂ receptors. As one possible explanation about these contradictory findings, one study suggests that some D₂ receptor antagonists (e.g., sulpiride) act on dopamine D₂ autoreceptors, which inhibit the release of dopamine (Schmitz, Lee, Schmauss, Gonon, & Sulzer, 2001). The effect of these D₂ antagonists weakens the inhibitory role of dopamine D₂ autoreceptors and allows the overflow of dopaminergic neurotransmissions. This overflow of dopamine is probably less likely to motivate an individual to behave aggressively because reward is not deficient. Thus, it is possible that some D₂ receptor antagonists reduce aggression via the low levels of dopaminergic neurotransmissions if these agents act on dopamine D₂ autoreceptors, rather than dopamine D₂ postsynaptic receptors. Based on previous pharmacological

findings and my results, aggression probably results from two ways: (1) the activation or high densities of dopamine D₂ autoreceptors and (2) the deactivation or low densities of postsynaptic dopamine D₂ receptors. In both cases, deficiency in dopamine may occur in the brain (especially the shells of the nucleus accumbens), and this neurochemical state of reward deficiency syndrome may lead to aggressive behavior.

Alternatively, the pharmacological effects of the D₂ receptor agents depend on the dose of the agents and the specific behavioral history of animals (Miczek et al., 2002). For example, Miczek (1974) found that both low and high doses of amphetamine, which influences dopaminergic systems, reduced aggression, but medium dose of it facilitated aggression. Tidey and Miczek (1992) found that the D₂ receptor agonist quinpirole decreased aggressive behavior among mice in morphine withdrawal, but the other D₂ receptor agonists increased aggression (De Almeida et al., 2005). Accordingly, it is possible that chronic passive exposure to aggression is a special behavioral experience that uniquely influences a neural pathway of 'learned' aggression, rather than a neuropharmacological pathway of 'general' aggression. In addition, the low densities of dopamine D₂ receptors might be involved in the special neural pathway of learned aggression, but not the neuropharmacological one. In other words, the low densities of dopamine D₂ receptors contribute to aggression only when an individual has experiences of chronic exposure to aggression. The neural pathway of learned aggression may uniquely involve acquisition process, conscious retrieval process, and/or knowledge structure relating to attitudes/favorability toward aggression, whereas these cognitive components may not be influential parts of the neuropharmacological pathway of general aggression.

Study #4 also found the high densities of 5-HT_{1B} receptors in the anterior basolateral amygdala among the observer rats exposed to aggression chronically. The amygdala is known as the storage of affective information (Sagvolden, Aase, Johansen, & Russell, 2005), and the electrical stimulation of amygdala increases aggression in Syrian golden hamsters (Potegal, Hebert, DeCoster, & Meyerhoff, 1996). Thus, the amygdala is critically involved in aggression, thus the association between aggression and the high densities of 5-HT_{1B} receptors in the anterior basolateral amygdala might correspond to the amygdala-related aggression.

Nevertheless, previous studies have indicated that aggression is enhanced by the low levels of 5-HT (for a review, see Anderson & Huesmann, 2003; Nelson & Trainor, 2007; Pihl & Benkelfat, 2005), inhibiting 5-HT reuptake (Ferris et al., 1997), or the deficiency in the gene encoding 5-HT_{1B} receptors (Saudou et al., 1994). On the other hand, the 5-HT_{1B} receptor agonists, which serve as activating 5-HT_{1B} receptors, inhibit aggression (e.g., Chiavegatto et al., 2001; Cleare & Bond, 2000; also see Miczek et al., 2002). These findings may counterargue my findings that aggression is associated with the high densities of 5-HT_{1B} receptors. Nevertheless, recent studies have demonstrated that the antiaggressive effects of 5-HT_{1B} agonists are site-specific, synergistic, and affected by the hormonal environment (Cologer-Clifford, Simon, Lu, & Smoluk, 1997; Cologer-Clifford, Simon, Richter, Smoluk, & Lu, 1998; Simon, Cologer-Clifford, Lu, McKenna, & Hu, 1998). So, for instance, some 5-HT_{1B} agents (both agonists and antagonists) may not act on 5-HT_{1B} receptors in the anterior basolateral amygdala while these receptors may play a key role in regulating aggression acquired by chronic exposure to aggression.

In addition, it is recently suggested that the upregulation of the densities of 5-HT_{1B} inhibitory autoreceptors contributes to aggressive behavior (Miczek et al., 2007). That is, when 5-HT_{1B} autoreceptors are excessively activated, the release of 5-HT is reduced, which is causally linked to aggressive behavior. That is, aggression probably results from (1) the activation (or high densities) of 5-HT_{1B} inhibitory autoreceptors or (2) the deactivation (or low densities) of 5-HT_{1B} postsynaptic receptors. Although it is unknown whether 5-HT_{1B} autoreceptors are distributed in the amygdala, it is possible that Study #4 found 5-HT_{1B} autoreceptors in the anterior basolateral amygdala.

In conclusion, Study #4 analyzed the neurotransmitter changes of dopamine D₂ receptors and 5-HT_{1B} receptors in response to chronic exposure to aggression and non-aggression. Compared to controls, the observer rats having been chronically exposed to aggression showed lower densities of dopamine D₂ receptors in the shells of the nucleus accumbens, the primary/secondary motor cortex, and cingulate cortex (in only the left hemisphere) and higher densities of 5-HT_{1B} receptors in the anterior basolateral amygdala. These may imply that chronic exposure to aggression reduces the densities of dopamine D₂ receptors but increases the densities of 5-HT_{1B} receptors, which are all associated with developing aggressive tendencies among the passive observer rats exposed to aggression.

Table 13. Descriptive Statistics of the Relative Densities of Dopamine D₂ Receptors (*N* = 30)

Brain regions	Chronic exposure to...		
	Aggression	Non-aggression	
Right Hemisphere	DLCPu	2.44 (.35)	2.60 (.38)
	DMCPu	2.17 (.22)	2.30 (.31)
	AcbC	1.61 (.20)	1.72 (.28)
	AcbSh	1.58 (.17)	1.83 (.34)
	Cingulate area 1	.44 (.13)	.51 (.12)
	Cingulate area 2	.55 (.14)	.59 (.13)
	Primary motor cortex	.36 (.12)	.46 (.10)
	Secondary motor cortex	.36 (.12)	.46 (.10)
Left Hemisphere	DLCPu	3.62 (.49)	3.75 (.53)
	DMCPu	1.66 (.17)	1.79 (.29)
	AcbC	1.50 (.25)	1.61 (.36)
	AcbSh	1.56 (.23)	1.79 (.39)
	Cingulate area 1	.39 (.13)	.49 (.11)
	Cingulate area 2	.51 (.15)	.57 (.11)
	Primary motor cortex	.38 (.17)	.46 (.15)
	Secondary motor cortex	.41 (.12)	.53 (.15)

Note. DLCPu = dorsolateral caudate putamen; DMCPu = dorsomedial caudate putamen;

AcbC = the core of the nucleus accumbens; AcbSh = the shells of the nucleus accumbens.

The values within parenthesis indicate standard deviation.

Table 14. Descriptive Statistics of the Relative Densities of 5-HT_{1B} Receptors ($N = 30$)

Brain regions		Chronic exposure to...	
		Aggression	Non-aggression
Right Hemisphere	LMol	.54 (.25)	.70 (.36)
	Hypothalamus	1.94 (.47)	1.99 (.42)
	Basolateral amygdala	2.01 (.33)	1.57 (.23)
Left Hemisphere	LMol	.39 (.16)	.40 (.22)
	Hypothalamus	1.91 (.39)	1.97 (.31)
	Basolateral amygdala	1.72 (.51)	1.36 (.41)

Note. LMol = lacunosum molecular layer of the hippocampus. The values within parenthesis indicate standard deviation.

Table 15. Two-Way Multivariate Analysis of Variance for the Mean Density Values of Dopamine D₂ Receptors (*N* = 30)

Source	DLCPu				DMCPu				AcbC			
	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>
Between subjects												
Group (G)	1	.92	.03	.35	1	2.20	.08	.15	1	1.22	.05	.28
Within subjects												
Hemisphere (H)	1	254.68**	.91	.00	1	188.24**	.88	.00	1	9.66*	.27	.01
G × H	1	.02	.00	.89	1	.01	.00	.95	1	.00	.00	.97
S within-group error	26	(.07)			26	(.02)			26	(.02)		

Note. DLCPu = dorsolateral caudate putamen; DMCPu = dorsomedial caudate putamen;

AcbC = the core of the nucleus accumbens. Values within parentheses represent mean

square errors. S = subjects. **p* < .05; ***p* < .01.

(Continued)

Table 15 (Continued)

	AcbSh				Cingulate area 1				Cingulate area 2			
Source	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>
Between subjects												
Group (G)	1	4.94*	.16	.04	1	3.61	.07	.12	1	1.05	.04	.32
Within subjects												
Hemisphere (H)	1	.34	.01	.57	1	5.17*	.17	.03	1	6.81*	.21	.02
G × H	1	.05	.00	.82	1	.47	.02	.50	1	.31	.01	.58
S within-group error	26	(.02)			26	(.00)			26	(.00)		

Note. AcbSh = the shells of the nucleus accumbens. Values within parentheses represent mean square errors. S = subjects. * $p < .05$; ** $p < .01$.

Table 15 (Continued)

	Primary motor cortex				Secondary motor cortex			
Source	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>
Between subjects								
Group (G)	1	5.12*	.17	.03	1	8.18*	.24	.01
Within subjects								
Hemisphere (H)	1	.17	.01	.68	1	2.51	.09	.13
G × H	1	.05	.00	.83	1	.34	.01	.57
S within-group error	26	(.01)			26	(.01)		

Note. Values within parentheses represent mean square errors. S = subjects.

* $p < .05$; ** $p < .01$.

Table 16. Two-Way Multivariate Analysis of Variance for the Mean Density Values of 5-HT_{1B} Receptors ($N = 30$)

	LMol				Hypothalamus				Basolateral amygdala			
Source	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>	<i>df</i>	<i>F</i>	η	<i>p</i>
Between subjects												
Group (G)	1	1.14	.05	.30	1	.13	.01	.72	1	10.94**	.31	.00
Within subjects												
Hemisphere (H)	1	11.83**	.33	.00	1	.15	.01	.70	1	7.46*	.24	.01
G × H	1	1.35	.05	.26	1	.01	.00	.94	1	.19	.01	.67
S within-group error	26	(.05)			26	(.04)			26	(.11)		

Note. LMol = lacunosum molecular layer of the hippocampus. Values within parentheses represent mean square errors. S = subjects. * $p < .05$; ** $p < .01$.

Table 17. Individual Comparisons of the Mean Density Values of Dopamine D₂Receptors ($N = 30$)

	Brain regions	<i>df</i>	<i>F</i>	<i>p</i>
Between subjects				
Right Hemisphere	DLCPu	1, 28	2.30	.14
	DMCPu	1, 28	2.62	.12
	AcbC	1, 28	2.91	.10
	AcbSh	1, 28	7.95**	.01
	Cingulate Area 1	1, 28	2.72	.11
	Cingulate Area 2	1, 27	1.51	.23
	Primary motor cortex	1, 27	5.84*	.02
	Secondary motor cortex	1, 28	8.46**	.01
Left Hemisphere	DLCPu	1, 28	.66	.42
	DMCPu	1, 28	1.96	.17
	AcbC	1, 28	.75	.39
	AcbSh	1, 28	4.77*	.04
	Cingulate Area 1	1, 28	4.30*	.047
	Cingulate Area 2	1, 28	2.02	.17
	Primary motor cortex	1, 27	1.62	.21
	Secondary motor cortex	1, 27	5.03*	.03

Note. DLCPu = dorsolateral caudate putamen; DMCPu = dorsomedial caudate putamen;

AcbC = the core of the nucleus accumbens; AcbSh = the shells of the nucleus accumbens.

* $p < .05$; ** $p < .01$.

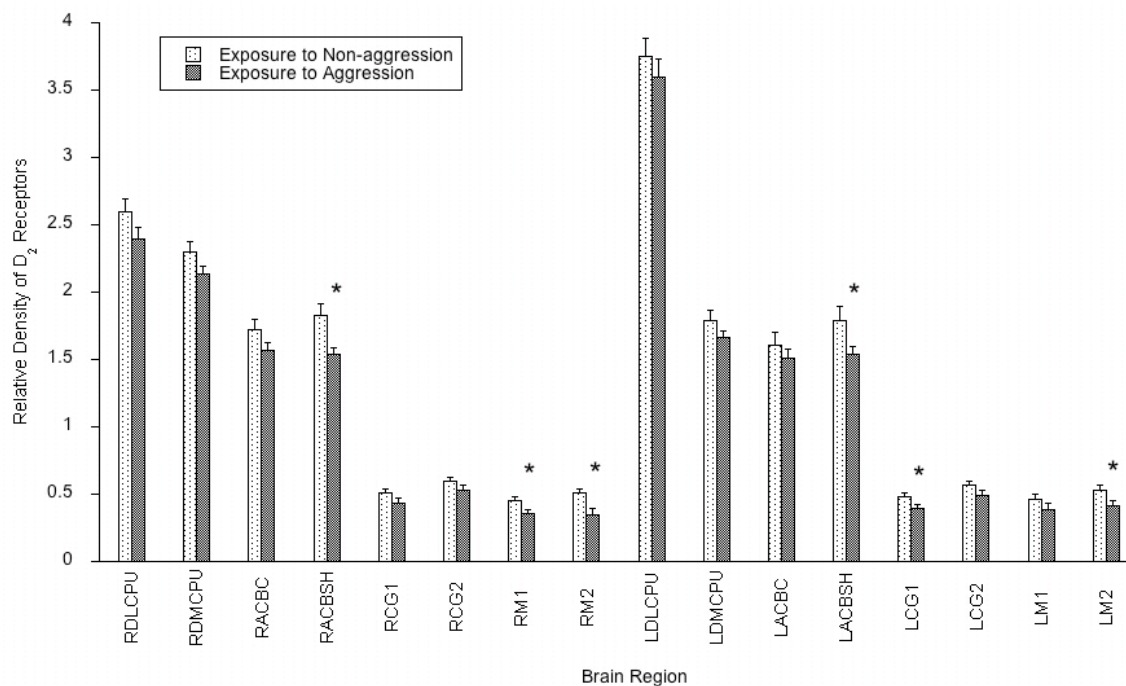
Table 18. Individual Comparisons of the Mean Density Values of 5-HT_{1B} Receptors

(N = 30)

Brain regions		<i>df</i>	<i>F</i>	<i>p</i>
Between subjects				
Right Hemisphere	LMol	1, 26	1.64	.21
	Hypothalamus	1, 26	.05	.83
	Basolateral Amygdala	1, 26	19.05**	.00
Left Hemisphere	LMol	1, 24	.01	.92
	Hypothalamus	1, 26	.14	.71
	Basolateral Amygdala	1, 26	5.14*	.03

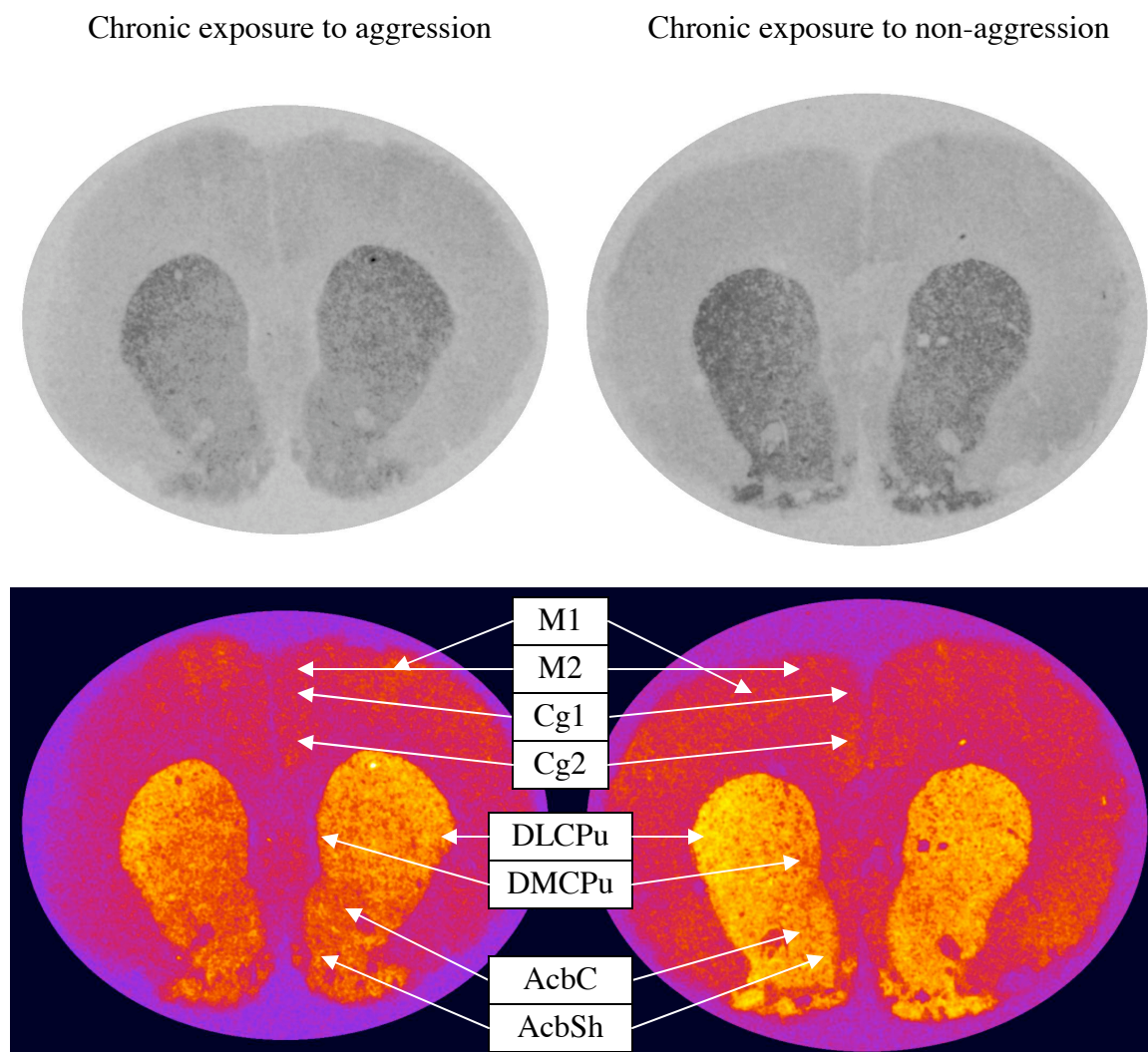
Note. LMol = lacunosum molecular layer of the hippocampus. * $p < .05$; ** $p < .01$.

Figure 10. Mean Differences in the Densities of Dopamine D₂ Receptors in the Anterior Regions of the Rat Brain (*N* = 30)



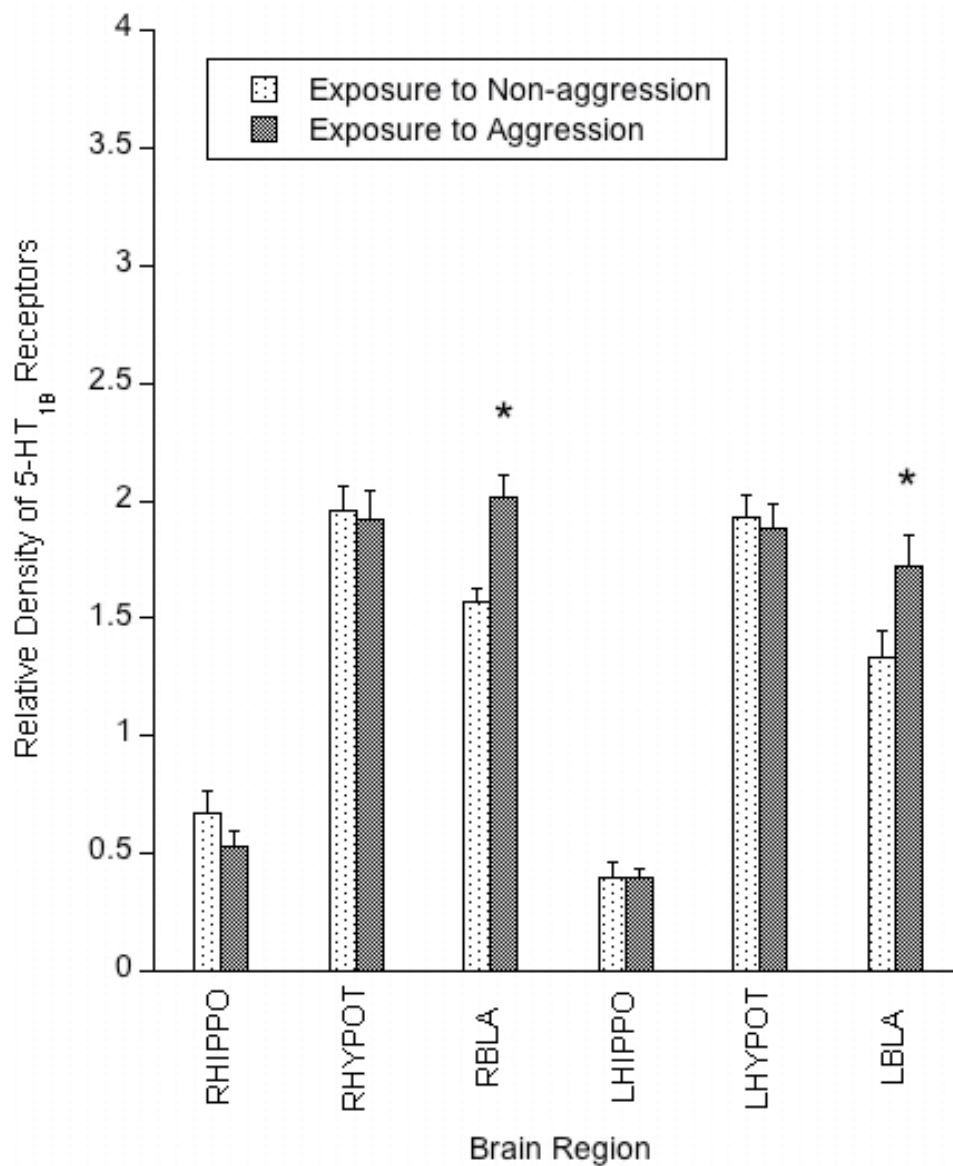
Note. Asterisk indicates a significant difference in the densities of dopamine D₂ receptors between observer rats exposed to aggression and non-aggression chronically.

Figure 11. Selected Rat Brain Sections at the Anterior Regions



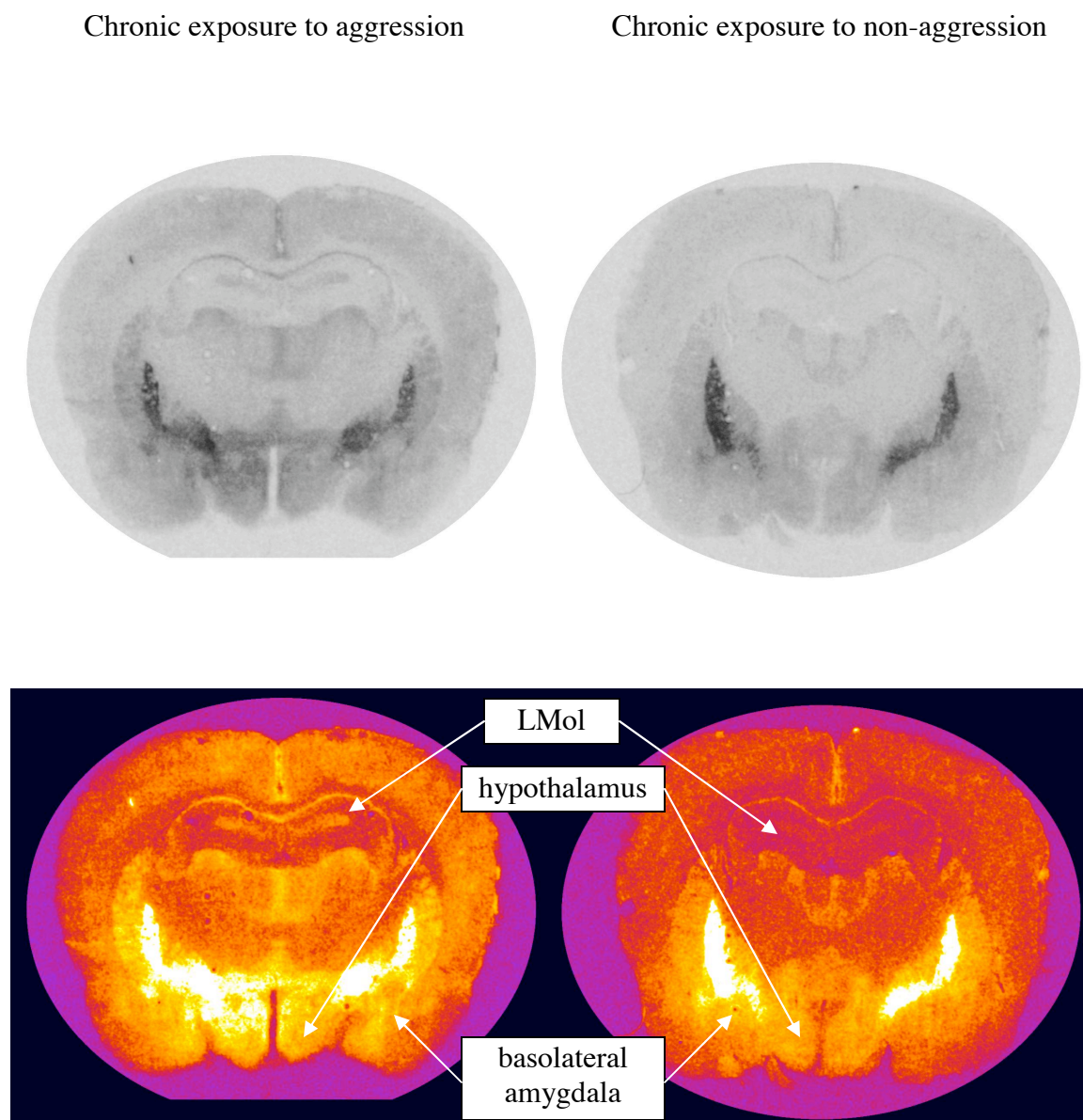
Note. DLCpu = dorsolateral caudate putamen; DMCPu = dorsomedial caudate putamen; AcbC = core of the nucleus accumbens; AcbSh = shells of the nucleus accumbens; Cg1 = cingulate area 1; Cg2 = cingulate area 2; M1 = primary motor cortex; M2 = secondary motor cortex. The two images in the above are the original ones; the below images are color-added images. Higher densities of dopamine D₂ receptors are indicated by darker areas (for the original images) or yellowish areas (for the colored images).

Figure 12. Mean Differences in the Densities of 5-HT_{1B} Receptors in the Posterior Regions of the Rat Brain



Note. Asterisk indicates a significant difference in the densities of 5-HT_{1B} receptors between observer rats exposed to aggression and non-aggression chronically.

Figure 13. Selected Rat Brain Sections at the Posterior Regions



Note. LMol = lacunosum molecular layer of hippocampus. The two images in the above are the original ones; the below images are color-added images. Higher densities of 5-HT_{1B} receptors are indicated by darker areas (for the original images) or yellowish areas (for the colored images).

CHAPTER SIX

GENERAL DISCUSSION

The major goals of the present research project were to investigate the behavioral and neurochemical effects of passive exposure to aggression on observer rats. To achieve these goals, this research consisted of the four parts. In Study #1, it was examined whether or not chronic passive exposure to aggression escalated aggressiveness of observer rats, compared to the effect of chronic exposure to non-aggression, as well as the effects of priming and acute exposure to aggression/non-aggression. As expected, only the observer rats which had been exposed to aggressive dyads for 23 days exhibited significant amounts of aggression than any other groups while there were no differences in aggressiveness of observer rats among the other remaining conditions. These results imply that passive observer rats learned aggression from the repeated occurrences of aggressive situations. On the other hand, the priming effect of acute exposure to aggression did not occur in a population of rats.

Study #2 further conducted the behavioral analyses that the observer rats given chronic exposure to aggression/non-aggression were tracked and screened on their aggressiveness 16 days after the isolation from aggressive or non-aggressive dyads. Although the observer rats in both groups (i.e., chronic exposure to aggression and non-aggression) significantly decreased their aggressiveness after the recovery of chronic exposure, the group difference in aggressiveness still maintained. That is, the observer

rats chronically exposed to aggression were still highly aggressive, compared to controls. These results confirmed that the observer rats actually socialized themselves to be aggressive through a learning process.

Study #2 also suggested that there seemed to be moderating variables that reinforced or attenuated the behavioral effects of chronic passive exposure to aggression. These moderating variables might be (1) the sensitivity to exposure to aggression and (2) the actual experience of victory/defeat combined with the role of encoding specificity. If observer rats are highly sensitive to exposure to aggression, or if they have defeated some rats and projected themselves into the aggressive resident rats, chronic exposure to aggression may be more strongly linked to aggressiveness of the observer rats.

Based on the findings that the expected behavioral effects of chronic passive exposure to aggression were successfully simulated in a rat model, Study #3 and #4 investigated the physiological and neurochemical changes of passive observer rats exposed to aggression, which was expected to indicate similar biological mechanisms in human beings. Study #3 analyzed the hormonal levels, specifically the concentrations of serum testosterone and corticosterone, in relation to single and chronic exposure to aggression. Interestingly, the chronic exposure groups showed higher levels of testosterone (regardless of a daily circadian rhythm) and lower levels of corticosterone than the single exposure groups. Thus, the high levels of testosterone and the low levels of corticosterone (which may indicate low fear) might together lead all observer rats in the chronic exposure conditions to be more aggressive than those in the single exposure conditions. In fact, Study #1 indicated this main effect of exposure amount (acute vs. chronic exposure) in aggressiveness of observer rats.

However, although Study #1 also found that there was a significant difference in observer rats' aggressiveness between the groups of chronic exposure to aggression and non-aggression, any differences in the levels of testosterone and corticosterone were not obtained between the two groups. That is, the high levels of aggressiveness of the observer rats chronically exposed to aggression did not result from the exclusive hormonal effects of testosterone and corticosterone. Moreover, it should be noted that, although the levels of corticosterone were lower among the chronic exposure groups than the single exposure groups, they were still much higher than the baseline. Thus, chronic exposure to aggression, as well as non-aggression, elevated the levels of corticosterone although this elevation is less remarkable, compared to the single exposure groups.

Finally, Study #4 examined another possibility that chronic passive exposure to aggression altered the neurotransmitter systems, specifically the densities of dopamine D₂ receptors and 5-HT_{1B} receptors, which might develop aggressive tendencies among observer rats. Interestingly, the observer rats chronically exposed to aggression showed lower densities of dopamine D₂ receptors in the shells of the nucleus accumbens, the primary/secondary motor cortices, and the cingulate cortex (in only the left hemisphere) than those chronically exposed to non-aggression. Conversely, higher densities of 5-HT_{1B} receptors were detected in the anterior basolateral amygdala among the observer rats exposed to aggression, compared to controls. These results suggest that chronic exposure to aggression seemed to lower the densities of dopamine D₂ receptors and to upregulate the densities of 5-HT_{1B} receptors in the specific brain regions.

Based on Blum et al.'s (1996) hypothesis of reward deficiency syndrome, it can be hypothesized that chronic passive exposure to aggression might reduce the densities of

dopamine D₂ (postsynaptic) receptors in the shells of the nucleus accumbens. Consequently, deficiency in dopaminergic neurotransmissions in the nucleus accumbens (i.e., reward deficiency syndrome) might occur and motivate passive observer rats to restore it. Because aggressive behavior temporarily increases the extracellular concentrations of dopamine in the shells of the nucleus accumbens (Van Erp & Miczek, 2000), the passive observer rats were engaged in aggressive behavior when they encountered their opponent rat. However, this effect of aggression on dopaminergic systems might fade out quickly, and the observer rats might suffer from reward deficiency syndrome repeatedly. Therefore, these observer rats behaved aggressively again on the follow-up screening tests. In other words, the passive observer rats maintained their aggressiveness probably until they recover the densities of dopamine D₂ receptors. This neurochemical recovery may take more than 16 days based on my finding that the observer rats still showed the high levels of aggression 16 days later.

Simultaneously, based on Miczek's (2007) hypothesis, chronic passive exposure to aggression might build up more densities of 5-HT_{1B} autoreceptors in the anterior basolateral amygdala. These autoreceptors function to inhibit the release of 5-HT presynaptically. Therefore, the high densities of 5-HT_{1B} autoreceptors could result in low serotonergic neurotransmissions, which are associated with aggression (for a review, see Anderson & Huesmann, 2003; Nelson & Trainor, 2007; Pihl & Benkelfat, 2005).

These changes in serotonergic and dopaminergic systems, in response to chronic passive exposure to aggression, probably play an important role in controlling aggressive behavior for observers. Because the observer rats in the chronic exposure groups also showed the high levels of testosterone and the moderately high levels of corticosterone

(lower than those of the single exposure groups but higher than the baseline), it might be also possible that the combination of these hormonal and neurotransmitter changes together contribute to aggressive behavior. That is, aggression learned through repeated observations of aggressive situations might result from the combination of (1) the high testosterone levels *and/or* (2) the moderately high corticosterone levels *with* (3) the low densities of dopamine D₂ receptors *and/or* (4) the high densities of 5-HT_{1B} (auto)receptors in the limbic systems.

For example, chronic passive exposure to aggression influenced the changes in the neurotransmitter systems (i.e., the low densities of dopamine D₂ receptors and the high densities of 5-HT_{1B} receptors), which might biologically prepare the observer rats for aggressiveness. Then, the high levels of corticosterone (compared to the baseline) might lead the observer rats to be engaged in affective aggression because hyperarousal (i.e., high glucocorticoid levels) is associated with intermittent, explosive, and affective aggression (Haller and Kruk, 2006). In fact, Study #1 actually found that the behavioral patterns of these observer rats seemed to be intermittent, frequent, and hyperactive aggression, compared to those of the observer rats exposed to non-aggression. Therefore, the interaction of the hormonal and neurotransmitter systems determine the degree of aggressiveness and types of aggressive behavior.

In addition, the possible effects of hormone-neurotransmitter interaction might play a key role in moderating the association between chronic exposure to aggression and an observer's aggressiveness, found in Study #2. For instance, among the observer rats exposed to aggression for 23 days, those with the high levels of testosterone but the normal densities of dopamine D₂ receptors might result in high aggressiveness on only

the initial screening test because of the temporary hormonal effect on aggression. Alternatively, because it was found that the serotonergic systems lost their role in regulating aggression in glucocorticoid-deficient rats (Haller, Toth, & Halasz, 2005; also see Kim & Haller, 2007), the observer rats with inherently low corticosterone levels (e.g., dysfunctions of the HPA axis) might remove the potential functions of 5-HT_{1B} receptors in the anterior basolateral amygdala. As a result, these observer rats would show more or less aggression on the initial and/or follow-up screening tests, regardless of the high densities of 5-HT_{1B} receptors. If these are possible cases, only the observer rats which meet all conditions in the hormonal/neurotransmitter systems (i.e., high testosterone, moderately high corticosterone, low densities of dopamine D₂ receptors, and high densities of 5-HT_{1B} receptors) might maintain their aggressiveness across 16 days.

Psychological Implications of the Present Research

All results reported in the present research can provide significant psychological implications concerning the behavioral effects of chronic passive exposure to aggression among human witness. Olivier and Young (2002) argue that the animal models, including rat models, meet predictive and face validity for examining the behavioral effects, as well as pharmacological and neurochemical ones, on human aggression (also see Nelson & Trainor, 2007; Tamashiro et al., 2005). Therefore, my findings that chronic exposure to aggression was associated with long-lasting aggressiveness of passive observers were also presumed to describe about human phenomenon of aggression.

For example, chronic exposure to family violence may facilitate aggression among family members who even passively observe it. According to Tolan et al. (2006), family violence is the most prevalent form of violence in the United States, compared to

the other forms of violence between acquaintances or strangers. Family violence includes not only illegal forms of violence (e.g., spouse abuse, child abuse, elder abuse) but also normative forms of violence (e.g., physical punishment, violence between siblings). A child who chronically and passively observes such family violence may show aggressive and bullying behavior in his/her schools. Through chronic violence between parents, the child witness may legitimate violence against intimate partners such that physical abuse is the acceptable means in family relationships (Heise, 1998; Widom, 2000). Some studies suggest mediating variables in the association between chronic exposure to family violence and aggressiveness of (child) witnesses, such as partner choice, relationship skills, and overall aggression level (Capaldi & Gorman-Smith, 2003; Tolan et al., 2006). Thus, an impulsive child witness with poor relationship skills is highly likely to socialize themselves to be aggressive through chronic exposure to family violence. Depending on his/her partner's personality, the child witness may be engaged in family violence later in lifetime. In this way, family violence may be transmitted across generations.

Not only family violence but also community violence can be a risky contextual factor of developing aggressiveness of observers. Survey research reported that living in community with high crime rates is associated with child abuse (Chalk & King, 1998; Williamson et al., 1991; Tolan et al., 2006). Guerra, Attar, and Weissberg (1997) identify the combination of chronic stress and violent environment as a critical factor that contributes to the risk of youth violence. This combined factor often exists in inner-city communities and has an impact on the effectiveness of preventive interventions. That is, preventive interventions for violent youths are less effective if they are exposed to

community violence and chronic stress. This suggestion is evidently implied by the results in Study #1 and #3; aggressive behavior was obtained among the rats which had been chronically exposed to aggression (i.e., violent environment), and these rats showed higher levels of corticosterone than the baseline. Also, the results in Study #2 suggest that the observer rats chronically exposed to aggression decreased their aggressiveness when they had been isolated from exposure to aggression for 16 days. This implies that, as Guerra et al.'s (1997) suggest, the removal of violent environment is somewhat effective in reducing aggressiveness of observers. The administration of preventive interventions under such non-violent environments may additionally promote the positive effect of recovery from exposure to aggression on reducing aggressiveness of observers, as indicated by Guerra et al.

The present study may also provide a possible explanation about the effect of mass media violence on aggression. For instance, Berkowitz and Macaulay (1971) found that, in the 40 U.S. cities, there was a significant rise in aggressive assaults and robberies (involving the use of threat) following two heavily published murder stories, the John F. Kennedy assassination and the murders by Speck and Whitman. Furthermore, other studies found that championship prizefights induced aggressive behavior among those who are exposed to them (Berkowitz & Rawlings, 1963; Berkowitz & Geen, 1966, 1967). Consistent with it, Phillips (1983) further found the increase in the U.S. daily homicides on the third and fourth day after the prizefights (also see Phillips, 1986; Phillip & Bollen, 1985). These findings might be associated with the priming effects of a single exposure to aggression, rather than the learning/socializing effects of chronic exposure to aggression, but passive exposure to aggression obviously influences aggressiveness of

human witnesses via mass media. Thus, as the results in Study #1 indicated, chronic exposure to violent TV programs (i.e., heavily watching TV) could have the potential to increase aggressiveness of TV viewers (Heath et al., 1986).

However, the relationship between TV violence and aggressiveness of TV viewers is moderated/mediated by many factors. These factors include actual parental and marital abuse (Heath et al., 1986) and the way to describe violent scenes on TV (i.e., real, exciting, uncriticized, justified, and rewarded violent scenes are associated with aggression of TV viewers; see Phillips, 1986). Some studies also suggest an effect of the media-portrayed victim, where the type of a victim in the murder is similar to the type of a boxer knocked down in the prizefight (Berkowitz & Rawlings, 1963; Berkowitz & Geen, 1966, 1967). According this effect, if a young white boxer is beaten in the prizefight, the number of young white male victims, but not young black male victims, in the murders significantly increased.

Therefore, previous studies have demonstrated that passive exposure to family, community, and media violence is associated with aggressiveness of observers. This association was simulated in the rat model used in the present research when passive exposure to aggression was provided chronically. Because the present research also found that chronic passive exposure to aggression influenced the neurotransmitter systems (and some changes in the hormonal systems, compared to the baseline), it is possible that the observers exposed to family, community, and/or media violence may experience the neurochemical changes in the neurotransmitter systems, namely the low densities of dopamine D₂ receptors in the shells of the nucleus accumbens/motor cortices/cingulate cortex and the high densities of 5-HT_{1B} receptors in the anterior

basolateral amygdala. These changes in the neurotransmitter systems biologically motivate observers to behave in an aggressive manner.

The present research also implies an effective prevention for those who become aggressive as a result of chronic exposure to aggression. The results in Study #2 imply that mere removal from aggressive situations can decrease aggressiveness of passive observers. This implication is consistent with Onyskiw and Hayduk's (2001) suggestion that preventing children from witnessing aggression mitigates learning aggressive behavior. Nevertheless, they also argue that it is also important for children to provide high maternal responsiveness (e.g., frequent communication between parents and their child, such as talk and play) and early intervention (i.e., before preschool). Therefore, the preventive intervention should focus on not only psychological and/or pharmacological remedies for violent children (or children with externalizing problems) but also a comprehensive, family-centered approach. That is, because the parenting behaviors of mothers and other family members influence children's behaviors, the assessments of both parents' and children's behavior problems are an important preventive strategy. This strategy also helps with restricting the opportunities to observe family violence for children. It will be more effective if the preventive intervention removes not only family violence but also bullying and other violent forms in school settings. For example, the FAST Track (Families and Schools Together) program provides a good example of a preventive intervention where both families and schools are involved in making short-term changes and setting long-term prevention goals together (Reppucci, Woolard, & Fried, 1999).

Neurobiological Implications of the Present Research

The present research found that chronic passive exposure to aggression influenced the densities of dopamine D₂ receptors and 5-HT_{1B} receptors. If these changes in the neurotransmitter systems resemble the neural structure in the brain of aggressive rats in general, the brain of the observer rats exposed to the aggressive dyads might ‘mirror’ the neuropsychological experiences of the aggressive dyads. In fact, it has been reported that there are neural systems, called *mirror neurons*, which are activated by both acting a certain behavior and perceiving that behavior (Rizzolatti & Craighero, 2004). Therefore, although the observer rats ‘passively’ perceived aggressive actions of the resident rats, these observer rats might neurochemically experience as if they actively performed aggressive actions. As a consequence, the densities of dopamine D₂ receptors and 5-HT_{1B} receptors in the observer rats’ brain might resemble the ones in the brain of the resident rats which repeatedly performed aggressive behavior.

Interestingly, mirror neurons are widely located in the primary and secondary motor cortices, especially premotor cortex (Ferrari, Gallese, Rizzolatti, & Fogassi, 2003). Study #4 actually found the low densities of dopamine D₂ receptors in these motor cortices among the observer rats exposed to aggression chronically. Thus, the findings in Study #4 might provide supportive evidence for the functions of mirror neurons among the observer rats exposed to aggression.

The present research also described the potential biology-environment interaction in relation to aggression. Passive exposure to aggression might first change some gene phenotypes programming dopamine D₂ receptors and 5-HT_{1B} receptors (e.g., Miczek et al., 2001). These phenotypes might, then, command lowering the densities of dopamine

D₂ receptors and upregulating the densities of 5-HT_{1B} receptors, which are the neurochemical implications of aggressive tendencies among the observer rats. In response to these neurochemical changes, the observer rats exposed to aggression were motivated to be engaged in aggressive behavior. From evolutionary perspectives, this behavioral outcome might be an adaptive behavior such that aggressive behavior is necessary for survival in chronic aggressive situations. In addition, it is also possible that the changes in the densities of dopamine D₂ receptors and 5-HT_{1B} receptors result from the functions of the biological adaptive control systems (e.g., behavioral phenotypes) in response chronic exposure to aggression.

Limitations and Future Research

One limitation might be that, while the present research successfully manipulated aggressive situations around the observer rats in the experimental groups (i.e., exposure to aggression), it sometimes failed to create perfect non-aggressive situations around those in the control groups (i.e., exposure to non-aggression). As I reported, the differences in the average amounts of exposure to aggression were significant between the experimental group and the control group ($t(154) = 11.85, p < .01$, in Study #1; $t(94) = 7.70, p < .01$, in Study #3). On average, the experimental groups were exposed to aggressive situations in 154.5 seconds (in Study #1) and 143.7 seconds (in Study #3) per day, compared to controls which were exposed to aggression only in 5.1 seconds (in Study #1) and 1.3 seconds (in Study #3). The observer rats in Study #2 and #4 were also exposed to the same amounts of aggression, depending on either the experimental group or the control group, because they were recruited from those used in Study #1 and #3 respectively. However, some observer rats in the group of 23-day exposure to non-

aggression observed aggressive incidents in more than 60 min. Of course, when this case was detected, the aggressive non-aggressive dyad was immediately replaced with a new non-aggressive dyad. Thus, no controls were chronically exposed to high amounts of aggression. But, a few highly aggressive incidents around the controls might directly or indirectly influence some results presented by this research.

Likewise, some observer rats in the group of 23-day exposure to aggression observed no aggressive incidents at all on a few days during exposure periods. Again, any resident rats which stopped showing aggression were immediately replaced with another aggressive resident rat, thus there were no cases where the experimental groups did not see aggression at all in a whole set of exposure sessions. Nevertheless, it may be necessary to improve my resident-intruder model (Olivier & Young, 2002; Tamashiro et al., 2005) to perfectly manipulate aggressive or non-aggressive situations between the experimental and control groups.

Another limitation is that, when all aggression scores and defense/submission scores were coded by my research assistants and me, we were not blind in identifying which condition a given observer rat was assigned to. In addition, we know what a given observer rat in each condition was expected to behave. Thus, regardless of assessing intra-rater and inter-rater reliabilities, our coding strategy might bias aggression scores and defense/submission scores. Especially when we detected a play fight, this behavioral pattern might be interpreted in our favorable ways; for example, we might tend to code it as aggressive behavior when we analyzed the groups of exposure to aggression; or, we might code ambiguous aggressive behavior as a play fight when we analyzed the groups of exposure to non-aggression. However, it was difficult to eliminate this potential bias

because it was necessary to keep tracking which observer rat belonged to a specific condition.

To improve my content analyses in the present research, there are two ways to deal with the above potential bias. One strategy is to recruit other research assistants who are blind of my research hypotheses. The other tactic is to code not only aggression scores but also scores of play fights and ambiguous aggression. Then, 'unbiased' aggression scores are computed by subtracting the scores of play fights/ambiguous aggression from total aggression scores.

The other limitation could be that the blood and brain samples in Study #3 and #4 were collected 15 hours after the last day of 23-day exposure sessions. Because both the hormonal levels and receptor bindings can be changed in a timely manner, the data collected 15 hours later might have a problem of the ceiling effects. For example, no difference in the levels of corticosterone was found between the groups of 23-day exposure to aggression and non-aggression when their blood samples were collected 15 hours (at morning time) after exposure sessions. However, this might not be surprising simply because a daily circadian rhythm reduced the levels of corticosterone to almost no concentrations (i.e., ceiling) for both the experimental and control groups. Therefore, the present research also collected the blood samples from both the experimental and control groups immediately after 23-day (and 1-day) exposure sessions. However, these groups have small sample size (e.g., six observer rats from each of 23-day exposure to aggression and non-aggression), so statistical power was low to detect any differences in the levels of corticosterone among the groups. This problem also happened when the levels of testosterone were tested although both groups of 23-day exposure to aggression

and non-aggression maintained the high levels of testosterone immediately and 15 hours after exposure sessions.

Similarly, receptor bindings (i.e., the densities of dopamine D₂ receptors and 5-HT_{1B} receptors) are also time-sensitive. In spite of this fact, Study #4 found some significant differences in the densities of these receptors between the groups of 23-day exposure to aggression and non-aggression. If the brains of the observer rats were collected immediately after 23-day exposure sessions, significant differences in the densities of the receptors might be detected in more areas, such as the hippocampus and the hypothalamus, which are involved in the neural mechanisms of aggression (Ase, Reader, Hen, Riad, & Descarries, 2001; Ferris et al., 1997).

For future research, it is necessary to collect the blood and brain samples from the observer rats immediately after 23-day exposure periods. In addition, it is also interesting to collect the blood and brain samples from the single exposure groups. To clarify the link between the neurochemical change and aggressiveness of observer rats, future research needs to screen aggressiveness of observer rats and to collect the blood/brain samples from the observer rats immediately after exposure session(s). This way will enable investigators to find out a more comprehensive view of the mechanisms of passive exposure to aggression.

The present research examined the behaviors and neurochemical changes of only young adult male observer rats. There may be sex and/or age differences in the sensitivity to chronic passive exposure to aggression. In addition, Study #2 suggested a possibility of moderating variables (i.e., sensitivity to exposure and actual experience of victory/defeat) in the relationship between exposure to aggression and observers'

aggressiveness. The analysis of these additional variables will widen the mechanisms of passive exposure to aggression.

Finally, based on previous studies, Di Matteo et al. (2008) argue that 5-HT_{1B} receptors in the ventral tegmental area play an important role in modulating dopaminergic neurotransmissions in the nucleus accumbens. Because Study #4 indicated that the functions of dopaminergic systems in the nucleus accumbens were associated with aggression learned by passive exposure to aggression, there may be significant group differences in the densities of 5-HT_{1B} receptors within the ventral tegmental area, which seem to have a neural link to dopaminergic neurons in the nucleus accumbens.

Conclusion

The present research suggests that chronic exposure to aggression results in (1) high aggressiveness of passive observers, (2) a long-lasting predisposition of aggressiveness, (3) the low densities of dopamine D₂ receptors in the shells of the nucleus accumbens, primary/secondary motor cortices, and the cingulate cortex, and (4) the high densities of 5-HT_{1B} receptors in the anterior basolateral amygdala. These findings notice both behavioral and neurochemical risks of chronic passive exposure to aggression, such as exposure to family violence, community violence, and mass media violence.

APPENDIX A:
SOME EPISODES PRIOR TO THE PRESENT RESEARCH

My dissertation topic, the effects of passive exposure to aggression on observers, was originally inspired by my own master's thesis on juvenile delinquency. In my master's thesis, I found that frequent exposure to (1) peer's alcohol use or (2) peer's delinquent behavior are the most strongest factor to determine whether an observer was engaged in delinquent behavior two years later. Then, through conversations with another student, I decided to study the effects of passive exposure to aggression by using an animal model.

However, as I stated previously, no studies have investigated the psychological/biological effects of passive exposure to aggression by using an animal model. There were three big issues until I developed my animal model of passive exposure to aggression. The first issue was how to physically separate an observer rat from an aggressive or non-aggressive dyad. For the first attempt, I initially placed a transparent plastic bookstand and buried the bottom part of it with bedding. Then, an observer rat was placed in one side of the bookstand, and an aggressive/non-aggressive dyad was placed in the other side. This tactic was problematic, however, because an aggressive/non-aggressive dyad sometimes pushes the bookstand down.

One day, I went to a pet shop to find any goods which could play a role of fence between an observer rat and an aggressive/non-aggressive dyad. I found a small transparent plastic aquarium, and I thought that it would be able to separate an observer rat from an aggressive/non-aggressive dyad by placing the observer rat into the aquarium. Through several preliminary studies, there were no cases where an aggressive/non-

aggressive dyad forcefully contacted an observer rat by opening a lid, breaking an aquarium, etc. Thus, I decided to use a small plastic aquarium as a barrier.

The second issue was how to induce aggression within an aggressive dyad. The present research used the resident-intruder model (Olivier & Young, 2002; Tamashiro et al., 2005) to create aggressive situations around the observer rats in the experimental groups (see Chapter #1 for details about the resident-intruder model). In addition to the resident-intruder model, there are more animal paradigms to study aggression (Olivier & Young, 2002): isolation-induced offensive behavior (used for mice), offensive behavior after electrical stimulation of the brain (used for rats), maternal offensive behavior (used for mice/rats), offensive playfighting (used for juvenile rats) and other miscellaneous models (e.g., predatory aggression such as mouse killing or locust killing). Because the resident-intruder model was typically used in rat studies, and because the model is relatively cheaper than the other models in terms of costs, I chose the resident-intruder model for the present research. In addition, I thought that aggressive behavior of the resident rat would be more intensive if the resident rat was pre-isolated (In fact, a model of isolation-induced offensive behavior is also used in animal studies). However, my impression was that the pre-isolated resident rats showed or did not show aggression whimsically.

Afterwards, I knew that some studies paired the resident rat with a female partner before their aggression experiments (Fish et al., 1999; 2001). Thus, I decided to use pre-isolated, pre-paired resident rats to induce aggression, and this model was the most successful to induce aggression of the resident rats with high probability. In my

subsequent preliminary studies, I also found that the resident rats seemed to need sufficient amounts of time (e.g., about two weeks) to induce pairing-induced aggressive behavior; the resident rat which was paired with a female partner in less than two weeks showed aggression whimsically.

The resident rats which had repeatedly been paired with many intruder rats and several female partners became even more aggressive. One day, however, I accidentally placed an intruder rat which had 50 g less than a resident rat. Although, as usual, the resident rat initially attacked the intruder rat, the resident rat was eventually defeated and did stop showing aggression later. Since this accident, I decided to have intruder rats with 100 g less than a body weight of the resident rat so that the resident rat can win with 100 % and gain repeated experiences of victories.

The third issue was how long an observer rat needed to be exposed to aggression so that he would show aggression. That is, I was wondering how many days might be needed to change the behavioral outcomes of observer rats. My initial pilot studies provided observer rats with 7-day exposure to aggression and non-aggression. At this time, exposure was not given every day; rather, it was given every other day. As a result, there were no differences between the groups of exposure to aggression and non-aggression in terms of the (1) starting time of attacking, (2) amount of time for aggressive behavior in sec., (3) frequency of aggressive behavior in percentage, (4) the number of aggressive actions, (5) amount of time for an opponent rat's defensive or submissive behavior, and (6) frequency of an opponent rat's defensive or submissive behavior.

Thus, I needed to re-consider about the amount of exposure to aggression.

Accidentally, I found one study (Feldker et al., 2006) that the authors found the changes in gene expression when rats had been actively involved in aggressive situations for 25 days. Based on this study, I attempted to expose passive observer rats to aggression for 25 days. Although I could not actually run 25-day exposure to aggression/non-aggression because of my family emergency, I could still find significant differences in the (1) amount of time for observer rats' aggression and (2) amount of time for an opponent rats' defense and submission between the groups of 23-day exposure to aggression and non-aggression. Therefore, I decided to keep collecting the behavioral data, as well as neurochemical data, from observer rats which were exposed to aggression and non-aggression for 23 days (and 1-day exposure to aggression and non-aggression as additional conditions) until a sufficient sample size was obtained.

Therefore, it had been a long time (about two years) to just find out how to test the behavioral effects of priming/single/chronic exposure to aggression/non-aggression in the present research. Thanks to all of my Dissertation Committee members, especially Dr. Louis R. Lucas, I could develop my animal model of passive exposure to aggression and achieve the results presented in this paper.

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VITA

The author, Hideo Suzuki, was born and grew up in Japan until his graduation from a high school. Afterwards, he went to California State University, Long Beach and then transferred to Vanderbilt University. At Vanderbilt University, he received a Bachelor of Arts in Sociology and Psychology, with his academic achievement recognized by Psi Chi Honor Society in Psychology. He continued to study psychology at Loyola University Chicago and received a Master of Arts in Applied Social Psychology with being honored by Phi Beta Delta, the Honor Society for International Scholars.

In 2005, Dr. Suzuki was enrolled in a doctoral program in applied social psychology at Loyola University Chicago. He had been involved in Dr. Edwards' laboratory of attitudes, Dr. Heath's Cross-cultural Studies of Media and Social Conflict (CSMSC), Dr. Han's Han Applied Laboratory of Neurocognition (HALON), and Dr. Lucas' laboratory of neurobiology. Through these research experiences, he made several poster/oral presentations and a publication on psychology and neuroscience. Dr. Suzuki eventually became interested in the interdisciplinary study of social psychology and neuroscience. He particularly focuses on a neural mechanism underlying aggression in response to social situations, such as passive exposure to aggression, which was investigated by his dissertation research. He also experiences teaching statistics, cognitive psychology, and social psychology at Loyola University Chicago for two years.

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The dissertation is therefore accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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