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Dual Process Models and Serotonergic Functioning:  
Impulse and Self-Control

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The concept of motivation is used to convey the sense of being impelled toward action of one sort or another. Once evoked, the action sometimes occurs immediately and freely. In other cases, several motivations are in play at the same time, yielding competition among incompatible actions. In this latter case, the result is typically that one action occurs and the other (or others) will be suppressed. The restraint of one or more action tendency implies the regulation of one motivation, generally by the overriding influence of another motivation. Human life is filled with such regulatory events.

Psychologists have recognized this proliferation of regulatory events with a (smaller) proliferation of terms. Some use the term *self-regulation* used to refer to such phenomena (Vohs & Baumeister, 2011). Sometimes, though, the term *self-regulation* is used to refer to a broader set of phenomena: the carrying out of an intended behavior by monitoring its consequences, to keep it on the desired path (Carver & Scheier, 1998). The latter usage does not convey any implication of a countervailing motivation. Another term for regulatory events that do involve competing motivations is *self-control*, which explicitly means restraining or suppressing one action tendency in favor of another one. In this chapter we focus on this class of events, and tend to use the label *self-control*.

In contrast to self-control is *impulsiveness*: the occurrence of some action without regard to some of its potential consequences. Impulsiveness is a another concept that can be difficult to pin down, however (Barratt, 1985; Block, 2002; Carver, 2005; Dickman, 1990; Eisenberg, 2002; Nigg, 2000; Solanto et al., 2001; Stanford & Barratt, 1992; White et al., 1994; Whiteside & Lynam, 2001, 2003). Impulsiveness can take many forms, including jumping quickly to acquire a potential incentive, being easily distracted from one's current path by other opportunities that arise while the current pursuit is ongoing, and reacting quickly to the occurrence of an emotion.

We emphasize in this chapter an aspect of impulsiveness that is implied by most

definitions of it, but is probably less salient than other properties. Specifically, we emphasize the idea that impulses are reactive: relatively immediate responses to some stimulus in disregard of other considerations. Under this view, the impulsive act need not entail approach—indeed, need not entail overt movement. A person can impulsively flee from a stimulus, if it evokes fear. A person can impulsively (reactively) remain passive when action might seem called for. The key, under this view, is that the action property that emerges represents a reactive, automatic association to the stimulus.

This chapter begins with description of a regulatory puzzle in personality psychology, which quite unexpectedly led to different puzzles in neurobiology and genetics, and now has turned to issues in clinical psychology. The focus of the chapter is on issues of impulsive reactivity versus constraint, or deliberative control of action. We begin by describing two accounts of a basis for this dimension of variability in personality. We then turn to evidence that this dimension of variability reflects (in part) variations in serotonergic function. More specifically, we suggest that certain serotonergically innervated brain regions help moderate the effects of underlying systems for approach and avoidance. Then we turn to the possibility that this view may help in thinking about how deficits in serotonergic function could be involved in a broad set of social and emotional problems, ranging from antisocial behavior to depression. The chapter closes with some further consideration of these problems.

### **Impulse and Constraint**

The field of personality is characterized by great conceptual diversity. Textbook authors often deal with the diversity by describing a range of theoretical views as alternative perspectives on personality and its functions (e.g., Carver & Scheier, 2012). Sometimes textbook authors also try to synthesize across theoretical boundaries, pointing to themes that seem to rise to the surface in one theory after another. Often enough, it turns out, similar themes are addressed by different

theories but are handled differently by them.

One such theme is the tension in life between impulsiveness and constraint. At least since the time of Freud, this issue has been important to personality theories, whether framed in terms of delay of gratification, planfulness, socialization, or id versus ego. As noted earlier, the concept of impulsiveness is used in diverse ways. However, the core of the issue as it emerges within personality psychology is relatively straightforward. People often face situations in which they can immediately follow an impulse or desire, or they can overrule that impulse and evaluate more fully before acting.

It is important to keep in mind that both impulse and constraint have valuable characteristics in the appropriate contexts (Block & Block, 1980). When it is manifested as spontaneity, impulsiveness brings a sense of vigor and freedom to the human experience (e.g., Dickman, 1990; Hansen & Breivik, 2001). There are also occasions in which survival literally may depend on impulsive action—when a threat or an opportunity must be reacted to quickly.

However, impulses can also create problems. Impulsiveness can yield physical danger (e.g., impulsively chasing a ball into the street without looking for traffic). Impulses can interfere with attainment of longer-term goals (e.g., spending for today rather than saving for the future). Impulses can lead to violation of social norms (Cooper, Wood, Orcutt, & Albino, 2003; Lynam, 1996) and thereby to interpersonal conflict and even legal problems. Potential adverse effects of impulsiveness including marital instability (Kelly & Conley, 1987), employment problems (Hogan & Holland, 2003), and disruption of health-maintaining behaviors (Bogg & Roberts, 2004; Hampson, Andrews, Barckley, Lichtenstein, & Lee, 2000; Hampson, Severson, Burns, Slovic, & Fisher, 2001; Skinner, Hampson, Fife-Schaw, 2002). Being able to control impulsive reactivity thus is crucial to successful self-management (Vohs & Baumeister, 2011).

What forces determine the balance between impulse and constraint? What prevents

impulses from always having free rein? Different theorists have posed different answers to these questions (for broader review see Carver, 2005).

### **Approach and Avoidance**

One answer stems from the general view that incentives draw behavior toward them and threats inhibit or even reverse those actions (e.g., Cloninger, 1987; Davidson, 1984, 1998; Fowles, 1993; Gray, 1994a, 1994b; Lang, 1995). The incentive system is often called a behavioral approach system (BAS; Gray, 1972, 1982, 1994a) or an activation or facilitation system (Depue & Collins, 1999; Fowles, 1980, 1987). When engaged by incentive cues, it yields approach and positive affect (Gray, 1994a, 1994b), including eagerness and desire. The threat system is often called a withdrawal system (e.g., Davidson, 1992, 1998) and was earlier called a behavioral inhibition system (BIS; Gray, 1972, 1982, 1994a), though the latter term has different connotations today (Gray & McNaughton, 2000; McNaughton & J. A. Gray, 2000). When activated by threat cues, this system causes ongoing approach to be inhibited and may lead to behavioral withdrawal (Fowles, 1993; Gray, 1994a). It also underlies emotions such as anxiety or fear (Carver & White, 1994; Davidson, 1992; Gray, 1982).

It can be argued that nothing more is needed to account for variability in impulsiveness than these basic approach and avoidance processes. The stronger the tendency to approach cues of incentives, the greater is the likelihood of impulsive approach. Indeed, Gray (1994a) chose *impulsivity* as his label for the personality dimension deriving from sensitivity of the approach system. In the presence of threat cues, however, the threat system becomes active, stifling ongoing approach. One might think of this stifling of approach as representing regulation of the approach motive by the avoidance motive. On the other hand, one might also think of a very reactive threat system as being impulsive in itself, yielding reactive avoidance that is not down-regulated by the approach system.

Approach competing with avoidance is one starting point in thinking about impulse and constraint. There are a number of reasons, however, for suspecting that the competition between approach and avoidance is not the entire story. One reason is that in comprehensive trait models of personality, both the trait that reflects approach and positive emotions and the trait that reflects avoidance and negative emotions are distinct from the trait that reflects constraint (Clark & Watson, 1999; Depue & Collins, 1999; Zelenski & Larsen, 1999). That is, threat sensitivity and constraint are separate dimensions.

Another reason for believing that approach and avoidance are not the entire story is that it is relatively easy to point to situations in which constraint seems to be unrelated to anxiety. An example is delay of gratification: foregoing a small reward now in order to obtain a larger one later (Mischel, 1974). Constraint in that situation does not seem to be based on avoidance of any threat, but rather about using time and planning to create more desirable overall outcomes.

### **Dual Process Models**

A different view derives from the idea that people process information in two somewhat distinct ways simultaneously, one more primitive than the other. The two processing modes appear to use different aspects of available information (Rudman, Phelan, & Heppen, 2007). There is also evidence that the two modes learn in different ways, and that the two patterns of learning create parallel and potentially competing paths to action, which require continuous arbitration (Daw, Niv, & Dayan, 2005). The more primitive mode operates largely outside consciousness. The other is the familiar symbolic processor of the rational mind.

By now this idea and variations on it have been taken up as a useful conceptual framework in many areas of psychology (Barrett, Tugade, & Engle, 2004; Kahneman, 2011; MacDonald, 2008). The literature of personality psychology contains several dual-process models, including what may be the earliest one in contemporary psychology: Epstein's (1973,

1985, 1990, 1994) cognitive-experiential self theory. This theory proposed that humans experience reality via a somewhat slow symbolic processor (the rational mind) and also an associative and intuitive processor that functions automatically and quickly. Epstein argued that both systems are always at work and that they jointly determine behavior. Metcalfe and Mischel (1999), drawing on decades of work on delay of gratification, proposed a similar model. They proposed that the relative strength of two systems determines whether one is able to restrain oneself: a “hot” system (emotional, impulsive, reflexive, and connectionist) and a “cool” system (strategic, flexible, slower, and unemotional). How a person responds to a situation with competing pressures depends on which system presently dominates.

The dual process idea has also been widely used in social psychology (Chaiken & Trope, 1999). The essence of such a view existed for decades in the literature of persuasion, but it has long since expanded beyond those bounds. Perhaps the most widely known dual process view in social psychology at present is Strack and Deutsch’s (2004) reflexive-impulsive model (see Hofmann, Friese, & Strack, 2009). But the ideas have proliferated far more widely.

The dual process idea also has an important presence in developmental psychology. For example, Rothbart and her colleagues (e.g., Rothbart, Ahadi, & Evans, 2000; Rothbart, Ahadi, Hershey, & Fisher, 2001; Rothbart & Bates, 1998; Rothbart, Ellis, Rueda, & Posner, 2003; Rothbart & Posner, 1985) have argued for the existence of basic temperament systems for approach and avoidance, and a third temperament termed effortful control (see also Kochanska & Knaack, 2003; MacDonald, 2008; Marcovitch & Zelazo, 2009; Nigg, 2000, 2003, 2006). Before the emergence of effortful control, behavior is a resultant of the influences of approach and avoidance temperaments (Figure 1). Greater sensitivity of the approach temperament makes impulsive action more likely; greater sensitivity of the avoidance temperament makes reflexive restraint more likely.

Effortful control emerges later in development than approach and avoidance temperaments. The label “effortful” conveys the sense that this is an executive, planful activity, entailing the use of cognitive resources to deter the tendency to react impulsively. Effortful control is said to rely on certain prefrontal brain areas (e.g., Eisenberg et al., 2004; Kochanska & Knaack, 2003; Nigg, 2001, 2003; Rothbart & Bates, 1998), and evidence from neuroimaging studies of both adults and children supports that argument (e.g., Durston, Thomas, Worden, Yang, & Casey, 2002; Durston, Thomas, Yang, Ulug, Zimmerman, & Casey, 2002).

Effortful control is superordinate to approach and avoidance temperaments (e.g., Ahadi & Rothbart, 1994; see also Clark, 2005). Its emergence permits control over reactive behavior: suppressing tendencies that are triggered by the approach or avoidance temperament, when doing so is situationally appropriate. If effortful control capacity is available, the grabbing of incentives that arises from a sensitive approach system can be restrained (Kochanska & Knaack, 2003; Murray & Kochanska, 2002). This child (or adult) can delay gratification.

Importantly, this child (or adult) can also do other things. It can override a reflexive tendency toward avoidance, in situations where the avoidance temperament is more active than the approach temperament. Thus, this person can remain in a difficult social situation rather than flee from it. If the approach temperament is weak, effortful control can override a reflexive tendency toward *inaction*. It can make you go to the gym when you don't really want to.

Thus, exerting effortful control can move a person toward either restraint or action, depending on what reflexive response is being overcome. As suggested early in the chapter, this casts a somewhat unusual light on the concept of impulsivity. In this view, what is impulsive is what is *reactive*, whether its outward display is of action or inaction.

This dual process model of influences on action seems to address issues that are not well handled by the viewpoint that considered only approach and avoidance. In this model, behavior



is restrained sometimes because anxiety is stronger than desire (thus creating a kind of reflexive restraint) and behavior is restrained sometimes because the reflective mode is acting to optimize longer-term outcomes.

Characterizations of the two processing modes by various writers are not identical, but they share many elements. The more primitive mode is typically described by such terms as impulsive, reflexive, reactive, implicit, heuristic, and associative. It is said to be responsive to situational cues of the moment, schematic associations, and especially to strong emotions. Its strengths are its quickness and its low demand on processing resources. It spontaneously creates action when its schemas are sufficiently activated. It thus can act even with little available information and high time urgency. The other mode is typically described by such terms as reflective, explicit, strategic, deliberative, and logical. Its strength is its ability to take into account circumstances that go beyond the immediate present. This mode requires substantial processing resources and thus loses efficiency when cognitive capacity is limited. This is the general viewpoint on self-regulation that we will assume as we continue.

### **Serotonergic Function**

We now turn to a different topic. A number of people have begun to consider the possible roles played by different neurotransmitter systems in the management of behavior, and thus in the variations that emerge among people's personalities. One neurotransmitter system that has been the subject of much investigation is the serotonergic system. In this section we consider a potential role for serotonergic function in impulse and constraint.

Serotonin has been studied for some time, in both humans and other animals (for greater detail see Manuck, Kaplan, & Lotrich, 2006). The processes by which it operates are not fully understood (Hensler, 2006; Lesch & Canli, 2006). It can be misleading to think only in terms of level of serotonin per se, because a good deal more is involved (e.g., Neumeister et al., 2006). On

the other hand, some manipulations do influence the level of serotonin available during a definable window of time. An example is acute tryptophan depletion. Tryptophan, an amino acid that is a precursor to serotonin, can be depleted by administering a drink (or capsules) containing high levels of other amino acids but no tryptophan. Several hours later, behavioral effects of artificially lowered serotonin can be studied.

Another methodological strategy is to relate behavior to genetic polymorphisms that have independently been linked to serotonergic function (Manuck et al., 2006). Most of this research has examined the gene that codes the serotonin transporter. Transcriptional activity of this gene is believed to be influenced by (or at least associated with) a repetitive sequence in a region called 5-HTTLPR, which has a short version and a long version (i.e., has more repetitions). A variety of indirect evidence links this polymorphism to variation in serotonergic function (reviewed in Carver, Johnson, & Joormann, 2008). It is now widely believed that the short allele is a marker of low serotonergic function (e.g., Canli & Lesch, 2007). This genetic paradigm is used to test what kinds of characteristics (behavioral, affective, cognitive, or personality) differ between persons with the short allele and those with the long allele.

The sections that follow provide a flavor of some of the research that has been done using these methods and others. We will argue that this research tends to suggest that the serotonergic system functions to decrease reactivity and to increase constraint.

### **Correlates of Serotonergic Markers in the Laboratory**

Some of the evidence comes from laboratory studies, in which tryptophan depletion appears to impair constraint over automatic emotional responses. As an example, consider a task in which specific cues are rewarded, and for which the response thus becomes habitual. Then the rules change and this response is no longer rewarded. Tryptophan depletion impairs the ability to inhibit those responses after the rule changes (Cools, Blackwell et al., 2005; Park et al., 1994;

Rogers et al., 2003). Tryptophan depletion has also led persons to report more sadness during exposure to uncontrollable stress (aversive noise), whereas the effect was only minor when the noise was controllable (Richell, Deakin, & Anderson, 2005). These types of studies suggest that the serotonin system can help inhibit responses to both rewarding and aversive stimuli.

Several studies have examined effects of tryptophan depletion on aggression. An important conceptual point was made in a study by Cleare and Bond (1995). Participants were pre-assessed as being either high or low in aggression. Those high in aggressive tendencies became more aggressive, hostile, and quarrelsome after tryptophan depletion, but there was no effect for those low in aggressive tendencies. Similar results were reported by Finn, Young, Pihl, & Ervin (1998). This pattern suggests that effects of low serotonergic function on aggression are less about aggression per se and more about the release of existing habitual tendencies to be aggressive (see also Manuck et al., 2006; Spooon, 1992). A later study (Bjork et al., 2000) further reinforced this point: tryptophan depletion in this case led to greater aggressive response to provocation among men high in aggressiveness but had an opposite effect among those low in aggressiveness.

### **Correlates of Serotonergic Markers with Personality**

Another set of studies has examined relationships of serotonergic function to personality self-reports, using several procedures to assess serotonergic function. Some of this work focused on qualities pertaining to aggression and impulsiveness; others examined a broader range of qualities. Hostility as a trait has been related to low serotonergic function in nonclinical samples (Cleare & Bond, 1997; Depue, 1995; Netter, Hennig, & Rohrmann, 1999). Depue (1995) related low serotonergic function as well to the Control-impulsivity facet scale from the Constraint factor of the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1985), the Aggression facet of the MPQ's Negative emotionality factor (but not other facets), two sensation seeking

subscales, and several indices of impulsiveness.

There is also a substantial literature on the serotonin polymorphism and personality as assessed by broad-ranging self-report inventories. These studies permit investigation of diverse possible associations, if facets as well as factors are examined. This work began with several large-scale studies with thorough examination of the data. Lesch et al. (1996) found that the short allele (linked to low serotonergic function) related positively to neuroticism (by NEO-PI-R) and inversely to agreeableness. In facet analyses, the neuroticism facets most closely linked to the short allele were Angry hostility, Depression, and Impulsiveness. Greenberg et al. (2000) also related the short allele to both neuroticism and agreeableness, with an additional weaker association for conscientiousness. Analysis of neuroticism facets again revealed the strongest relations for Angry hostility and Depression.

Many other studies have since been done, and even several meta-analyses (for review see Carver et al., 2008). Importantly, however, the meta-analyses have all focused on neuroticism, as has most of the developing literature. The consistent association with agreeableness has generally been disregarded.

### **Correlates of Serotonergic Markers with Impulsive Disorders**

A good deal of research has also examined serotonergic function in adults with clinical conditions reflecting impulsive aggression (for more extensive review see Manuck et al., 2006). Lower serotonergic function has long been linked to history of fighting and assault (Coccaro, Kavoussi, Cooper, & Hauger, 1997), domestic violence (George et al., 2001), and impulsive aggression more generally (Coccaro, Kavoussi, Hauger, Cooper, & Ferris, 1998; Cleare and Bond, 1997).

Genetic evidence also connects serotonergic function to violent and antisocial behavior. For example, Dolan, Anderson, and Deakin (2001) linked low serotonergic function to higher

impulsivity and higher aggression in male aggressive offenders. Interestingly, both impulsivity and aggression also related to higher anxiety in this sample. This argues against a path in which impulsive aggression is a product of low anxiety.

### **Characterizing the Pattern**

The pattern of these findings (and others) appears consistent with the view that serotonergic pathways are involved in impulse control (Depue, 1995; Depue & Collins, 1999; Depue & Spont, 1986; Manuck, Flory, Muldoon, & Ferrell, 2003; Soubrié, 1986; Spont, 1992; Zuckerman, 2005), particularly impulses that reflect strong emotions. On the other side, high serotonergic function appears to relate to consideration of the future consequences of one's behavior (promoting conscientiousness) and to positive social connection (promoting agreeableness).

We have characterized this pattern in terms of the dual process viewpoint described in the preceding section of the chapter (Carver et al., 2008). We said there that the basic, reactive mode of functioning is impulsive and is highly responsive to strong emotions. The reflective mode is planful and less reactive to immediate emotional cues. Joining these descriptions with findings described in this section, we suggest that serotonergic function may shift the balance of influence between these two modes of functioning. That is, it appears that lower serotonergic function may increase the influence of the reactive system or decrease the influence of the reflective system.

### **Depression and Serotonergic Function**

We now turn to depression. As mentioned in the previous section, depression as a facet scale of neuroticism has been linked repeatedly to the serotonin transporter gene, with the short allele being associated with higher depression scores. There is also an accumulation of evidence from other studies linking serotonergic function to more clinically meaningful depression (for review see Carver et al., 2008). Early studies looked for direct links from the polymorphisms to

depression vulnerability. More recent work has focused on gene by environment interactions.

Caspi et al. (2003) first reported that the serotonin transporter polymorphism interacted with early maltreatment to predict depression diagnosis by early adulthood: negative life events had an adverse effect on those carrying at least one short allele, but not among those with two long alleles. A number of other studies followed, and by now there have been several meta-analyses of this literature (Risch, et al., 2009; Uher & McGuffin, 2008, 2010). The outcomes of the meta-analyses have varied as a function of selection criteria. However, Uher and McGuffin (2010) found that the serotonin transporter polymorphism interacted with early maltreatment to predict vulnerability to depression in each of the 11 studies that used objective or interview measures of maltreatment (see also Caspi, Hariri, Holmes, Uher, & Moffitt, 2010).

### **Impulsivity and Depression**

Previous sections described studies linking low serotonergic function to impulse expression, particularly impulsive reactions to emotional cues. Studies were also described linking low serotonergic function to behavioral problems in which a salient feature was poor control over impulsive action. The idea that high reactivity to emotions underlies impulsive violence, sensation seeking, and externalizing problems such as substance abuse is both intuitive and supported by a great deal of data (Cyders, Flory, Rainer, & Smith, 2009; Dick et al., 2010; Whiteside & Lynam, 2003).

Now we are saying that low serotonergic function also relates to vulnerability to depression. This assertion may seem paradoxical in light of the others. Depression is not generally associated with impulsive overt action. It is more often associated with lethargy, an absence of behavioral engagement (Sobin & Sackeim, 1997). What accounts for this very substantial difference in presentation?

In addressing this question we return to the dual-process models, and also to our working

definition of impulsiveness. Dual process models suggest that the reactive mode acts impulsively (reflexively) and is highly responsive to emotions. But these are “operating characteristics” of that mode of function. How the operating characteristics are manifested overtly depends on what emotions the person is experiencing and what reactive action impulse thereby is being triggered.

In most cases, emotions call for outward action of some sort. Eagerness promotes approach. Fear promotes avoidance. But sadness is different. Sadness—the affective core of depression—is a *deactivating* emotion (Frijda, 1986). It calls for passivity, for giving up of effort (Frijda, 1986). A general over-responsiveness to emotions, if applied to sadness, would promote behaviors that sadness ordinarily triggers. The behavior that is triggered by sadness is *inaction*. Thus, many aspects of depressed behavior reflect passivity and apparent difficulty in initiating action.

Paradoxically, then, the same functional property (behavioral reactivity to emotion) that can help release bursts of violence or acting out may also help create essentially the opposite profile of behavior in response to a different emotion. This leaves two issues. First, something other than low serotonergic function must distinguish between these divergent cases. People who are sensation seekers and people who are vulnerable to depression must differ systematically from each other in some way other than low serotonergic function. How do they differ? Second, the case that depression should be viewed as similar to overtly impulsive, externalizing sorts of behavior seems somewhat circumstantial. It depends entirely on a set of findings concerning correlates of the serotonergic system. Is there any further evidence that this argument is tenable? These questions are considered, in order, in the next sections.

### **Further Influences: Approach and Dopamine**

The idea that low serotonergic function and the resulting deficits in effortful control have divergent effects in different groups of people requires hypothesizing an interaction of some sort

(see Depue & Lenzenweger, 2005). That is, it is not just low serotonergic function that yields a phenotypic manifestation. Something else must differ between the groups.

What other variable might interact with serotonin function to yield such a divergence with respect to sensation seeking versus depression? A plausible candidate is the sensitivity, or engagement, of the incentive approach system. When poor executive oversight is combined with moderately high incentive sensitivity (a reactive approach system), the result is overt approach-related impulsiveness. When poor executive oversight is combined with low incentive sensitivity (a nonreactive approach system), the result is impulsive inaction: lack of effort toward potential rewards. In both cases, the effects of variation in level of basic incentive sensitivity (high and low, respectively) are amplified by the absence of effortful override (Figure 1, earlier).

In the case of depression vulnerability, a lack of incentive sensitivity means that the person is not strongly motivated to approach potentially rewarding contexts. A relative deficit in effortful control amplifies this problem, such that the person has greater difficulty overcoming this lack of motivations. This combination thus should yield apathy, passivity, and fatigue, which characterize many cases of depression.

There are several sources of evidence that depression is associated with a blunted approach system. For example, EEG laterality has been used as a way to measure activity of the approach system. Several studies suggest that behavioral and personality measures of approach motivation correlate with higher activation in left than right anterior cortical areas (e.g., Coan & Allen, 2003; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). Previously depressed (Henriques & Davidson, 1990) and clinically depressed persons (Henriques & Davidson, 1991) have been found to have lower activation in left anterior cortical areas than non-depressed persons, with no difference in right anterior activation.

Behavioral research also suggests that depression relates to blunted incentive sensitivity.



For example, depressed persons have been found to be less responsive to reward than non-depressed persons (Henriques, Glowacki, & Davidson, 1994; Henriques & Davidson, 2000). Other evidence relates self-reports of low incentive sensitivity to depression (Campbell-Sills, Liverant, & Brown, 2004; Pinto-Meza, Caseras, Soler, Puigdemont, Perez, & Torrubia, 2006). Indeed, three separate studies have found that self-reports of low incentive sensitivity predicted worse course of depression over time (Campbell-Sills et al., 2004; Kasch, Rottenberg, Arnow, & Gotlib, 2002; McFarland, Shankman, Tenke, Bruder, & Klein, 2006).

Blunted approach motivation may also be reflected in low dopaminergic function. Dopaminergic pathways are believed to be critical in the engagement of goal-directed effort (Farrar, Pereira, Velasco, Hockemeyer, Müller, & Salamone, 2007; Salamone, Correa, Farrar, & Mingote, 2007; Salamone, Correa, Mingote, & Weber, 2005; Salamone, Correa, Mingote, Weber, & Farrar, 2006). A weakly functioning dopaminergic system yields less “wanting” for appetitive outcomes (Berridge, 2007) and less engagement of effort in pursuit of them (Salamone et al., 2005, 2006, 2007). A recent review reported a range of evidence for deficits in the function of dopamine among depressed persons, drawing from pharmacological studies, genetic studies, and dopamine challenge studies (Dunlop & Nemeroff, 2007).

### **Depression and Impulsive Reactivity to Emotion**

Is there is any direct evidence linking depression to over-reactivity to emotions? Evidence comes from at least three studies. Two of them (Ekinici, Albayrak, & Caykoylu, 2011; Peluso et al., 2007) linked a particular measure of impulsiveness to diagnosis of major depressive disorder (MDD). In both of these studies, persons diagnosed with depression reported greater motor impulsivity on the Barratt Impulsiveness Scale (BIS; Barratt, 1965) than controls; in one of them (Ekinici et al., 2011), a similar difference emerged for BIS attentional impulsivity. The measure used in those two studies is a relatively general one, and its item content makes it

difficult to attribute the impulsiveness assessed to emotional versus non-emotional sources.

We have recently collected data of our own to explore this idea more explicitly (Carver, Johnson, & Joormann, in press). We examined a sample of college students, using a variety of questionnaires bearing on impulsiveness versus control, some preexisting and others developed explicitly for our study. A subsample also completed a diagnostic interview for lifetime episode of major depressive disorder (MDD). The question of interest at present is whether the self-reports differentiated those who had positive diagnoses from the others.

Of the self-report scales administered, some were chosen to pertain to reflexive reactivity to emotions. Some focused on reactivity to negative emotions. Associations of these measures with lifetime MDD would be consistent with the widely held view that depression vulnerability is related to an enhanced experience of negativity (Bylsma, Taylor-Clift, & Rottenberg, 2011; Kendler, Neale et al., 1993). However, the measures we used focused not on the frequency of occurrence of negative emotions but on the tendency to respond relatively reflexively and automatically to them, either cognitively (e.g., by drawing further conclusions) or behaviorally.

It is important, though, that the dual-process view suggests that what is involved here is not just a propensity toward negativity. In holding that the reflexive system is highly reactive to emotions, this viewpoint does not distinguish among emotional valences. The reflexive system is simply held to be highly reactive to emotions. In applying this idea to depression vulnerability, the implication would seem to be that people who are vulnerable to depression should have a general reactivity to emotion of diverse sorts, not just negative emotions. To test this reasoning, we included one scale that addressed impulsive behavioral reactions to emotions “in general,” and another scale that assessed impulsive reactions to *positive* emotions in particular (the Positive Urgency Measure; Cyders et al., 2007).

Our focus, then, was on aspects of impulsivity that imply a reflexive response to

emotions. However, we also included measures to test the specificity of this reasoning, that is, measures that pertain to better versus worse self-control without involvement of emotions. A measure of comorbid alcohol problems was also included, to test whether any associations of lifetime MDD with reactivity to emotions would actually be attributable to this commonly comorbid externalizing syndrome.

The impulse-related questionnaires used in this project were distilled to 3 underlying factors (Carver, Johnson, Joormann, Kim, & Nam, 2011). Factor 1 (Pervasive Influence of Feelings) reflects a broad tendency for emotions to reflexively shape the person's orientation to the world: having one's worldview affected by temporary feelings, generalizing from negative events to the overall sense of self-worth, and reacting to sadness and fatigue with inaction. Factor 2 (Follow-Through) centers on the tendency to complete tasks versus being distracted and letting things go. This factor has no obvious involvement of reacting to emotion. Factor 3 (Feelings Trigger Action) centers on impulsive behavioral reactivity to emotions, including positive emotions. Factor scores for each participant were created from that factor analysis by the regression method, and the factor scores were the outcomes of interest.

Regression analyses (Carver et al., in press) confirmed that persons diagnosed with MDD lifetime had higher scores on factors 1 and 3 than did persons with negative diagnoses. There was no difference between groups on factor 2. Importantly, these differences between groups were robust to several kinds of analyses controlling for effects of current depressive symptoms and externalizing symptoms.

Results thus support the idea that lifetime MDD is related to elevated reactivity to emotions. This result is unsurprising with respect to Factor 1, because Factor 1 reflects in part reactions to negative emotions and to fatigue, along with overtones of passivity and automatic coloring of one's view of the world from (mostly negative) events. Less intuitive, but far less

ambiguous in supporting the dual-process viewpoint, is the finding that the lifetime MDD group also endorsed a more general impulsive reactivity to emotions—including positive emotions—to a greater degree than did the control group. This suggests that a contribution to depression vulnerability is made by an over-responsiveness to emotions in general, rather than only by a specific responsiveness to sadness or negativity. It is worth emphasizing that a link between history of MDD and reactivity to positive emotion would be very hard to predict from a viewpoint other than the dual-process viewpoint with which we entered the study.

### **Serotonergic Polymorphism and Impulsive Reactivity to Emotion**

This project has yielded another outcome that is also quite relevant to the overall argument being made here (Carver et al., 2011). Blood was also drawn from the participants, and assays were conducted for the serotonin transporter polymorphism. The three factors pertaining to impulsivity that were described in the preceding section were then related to the serotonin transporter polymorphism. Both of the factors that reflected impulsive reactions to emotions—the very factors that distinguished persons with a history of MDD from controls—were related to the polymorphism in the expected way. That is, carriers of the short allele (in interaction with reports of early childhood adversity) had higher levels of emotion-triggered impulsiveness. The factor that did not convey any implication of reactivity to emotions did not display this pattern.

### **Toward Transdiagnostic Vulnerability?**

Our focus on depression in this section of the chapter reflects our interest in that disorder, but it also reflects the highly counterintuitive nature of the idea that vulnerability to depression would be associated with reactivity to emotions. Previous findings have related reactivity to positive emotions to a range of externalizing problems, including vandalism, risky sexual behavior, and gambling, and drug use (Cyders et al., 2007; Zapolski, Cyders, & Smith, 2009), but there is less evidence regarding its role in internalizing problems. Across how broad a

spectrum of disorder is reactivity to emotion—even positive emotion—a contributor?

The three factors described here have also been studied in one other psychopathology-related context (Johnson, Carver, Mulé, & Joormann, in press). That study examined correlates of manic temperament, measured by the Hypomanic Personality Scale. This scale was found to be correlated significantly with Feelings Trigger Action, but not to the other two factors. Thus, reports of an over-responsiveness to positive emotions and emotions in general appears to relate to mania vulnerability as well as to depression.

The possibility that the broad spectrum of psychopathologies may be characterized by a more limited number of features that are actually trans-diagnostic has been raised in a number of places in recent years (e.g., Harvey, Watkins, Mansell, & Shafran, 2004; Johnson-Laird et al., 2006). It is worth asking whether an impulsive overreactivity to emotions may be one such trans-diagnostic feature (see an argument made by Johnson-Laird et al., 2006, about the role of emotional over-responsiveness in psychopathology). We are presently pursuing this question further.

Indeed, a broad question for the future is whether other interactions should also be explored more fully (Depue & Lenzenweger, 2005; Nigg, 2006). For example, it has been argued that overt expression of a vulnerability to anxiety disorders may also depend on poor executive control (Lonigan et al., 2004). Consistent with this idea, serotonin has been implicated in the development of anxiety disorders (Leonardo & Hen, 2006). These findings suggest an interactive combination of a highly sensitive threat system and low serotonergic functioning.

Many observers have noted that the attempt to link any given neurotransmitter to the operation of a single behavioral system is likely to be a great oversimplification. Nonetheless, it does not seem too far an extrapolation from the evidence to suggest that low serotonergic

function promotes a stronger manifestation of whatever tendencies the person has at the reflexive or implicit level of functioning (for similar conclusions see Depue, 1995; Nigg, 2006; Spont, 1992). In an incentive-sensitive person, low serotonergic function amplifies the pursuit of incentives. In an incentive-*ins*sensitive person, low serotonergic function exaggerates the lack of effortful engagement. In a threat-sensitive person, low serotonergic function may enhance vigilance to threat.

The specific cases of depression, externalizing disorders, and anxiety disorders are only three possibilities, reflecting interactions of a serotonergic system with two other systems. A more complete understanding of the role of serotonin in behavior will require a more elaborated understanding of how serotonergic function interacts with effects of other neurotransmitters. The idea that diverse disorders follow from diverse combinations of system sensitivities (Depue & Lenzenweger, 2005; Fowles & Dindo, 2009; Lenzenweger & Willett, 2007) is very intriguing, and seems worthy of much more examination.

### **Dual Process Models**

Recent years have seen an explosion of interest in neurobiological processes underlying behavior. Psychologists are now routinely collecting genetic data and they are very often, if not quite routinely, collecting imaging data to indicate what areas of the brain are especially active in varying experimental conditions. The involvement of different neurotransmitters—such as serotonin—in psychological phenomena is also an active area of exploration.

We have argued that it is useful to conceptualize certain functions of the serotonergic system in terms of dual process models of self-regulation. Viewed through this lens, the evidence suggests that serotonergic function can be linked to the trait dimension of impulsivity versus constraint in the personality literature, effortful control processes in the cognitive and

developmental literatures, and (not addressed here, but discussed in Carver et al., 2008) executive control over the amygdala and other subcortical areas in the neurobiological literature. This dual process picture helps organize what is known about the experience of depression, and it may also be useful in suggesting new areas of investigation.

The serotonergic system is a biological system. Yet ideas and evidence from literatures that are psychological in nature appear to foster a deeper understanding of the role of this system. It is increasingly said today that biological concepts and knowledge form constraints within which psychological theory must fit. We would hold, however, that the path of influence goes both ways, that interpretation of neurobiological evidence also benefits from considering the findings through the lens of psychological principles.

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*Figure 1.* Three temperamental influences on behavior. A. A reactive system for approaching rewards and a reactive system for avoiding threats or punishment compete for ascendance; in the absence of effortful control, the resultant of that competition is expressed in behavior. B. The engagement of an effortful control system permits the resultant arising from the competition of the reactive systems to be overridden, thus dampening the role of the reactive systems in determining behavior. From Carver, Johnson, & Joormann (2008), adapted from various statements by Rothbart, Eisenberg, and others.

