


### 13 Action Control and Its Failure in Clinical Depression: A Neurocognitive Theory

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Clinical depression is a mind/brain disorder of major public interest. It is a serious disorder that affects within a lifetime more than 15 percent of the population in western societies (e.g., Comer 2001). A central feature of this disorder is a strongly reduced level of activity (e.g., American Psychiatric Association 2000) that is characterized by reduced approach actions toward stimuli normally perceived as rewarding and an increased avoidance behavior toward stimuli perceived as threatening (e.g., Lewinsohn, Antonuccio, Steimetz, and Teri, 1984; Jacobson, Martell, and Dimidjian 2001; Yasch, Rotteberg, Asmow, and Gotlib 2002). In other words, the selection and execution of actions is fundamentally disturbed in clinical depression. If misregulation of action control is a central feature of depression, then an adequate explanation of how actions are normally selected, prepared, initiated, and terminated should be a decisive step in understanding this disorder. Following this line of reasoning in the first part of the chapter, a neurocognitive framework for action control is outlined along thirteen main hypotheses. "Neurocognitive framework" here simply means that, first, basic functions such as motor, perceptual, memory, or emotional processes are conceptualized as information processing by neurons in various, partly overlapping neural networks of the primate brain, and, secondly, that not only are behavioral data from normal human subjects taken into consideration but also data from human functional imaging, electroencephalograph (EEG) and single-cell recording (in monkeys), and neuropsychological lesion studies are considered (e.g., Kosslyn and Koenig 1992; Gazzaniga, Ivry, and Mangun 2002).

Basically, the action control framework presented here attempts to specify how goals compete, how a winner is selected, how this winner controls the preparation, initiation, and termination of motor actions, how the successful or erroneous outcome of an action is monitored and evaluated with respect to the intended goal, and how this evaluation modifies long-term memory contents. Furthermore, volitional processes that allow, in case of competing action tendencies, shielding of the current goal-directed action as well as executive processes for the execution of nonroutine activities will be introduced as central elements of action control. Finally, the role of a
processing mode called "state analysis" and of chronic stress in changing the neurocognitive processes and structures of action control in the primate brain will be sketched.

Based on this goal-based action control theory, the second section of the chapter will offer steps toward a neurocognitive theory of clinical depression along ten main hypotheses. The basic idea put forward here is that depression arises as a chronic stress response due to repeated failures in pursuing important goals. As a consequence of this chronic failure, two main types of changes in the action controlling brain are assumed to occur, that is, short-term state-dependent changes (e.g., depressive mood) and long-term changes (e.g., generalized negative probability estimates of action outcomes), which should cause inactivity, reduced approach behavior, and increased avoidance behavior as well as deficient executive control and volitional processes.

13.1 Goal-based Action Control

Action control can be conceptualized at many different levels (e.g., from single neurons to social attitudes) and by various theoretical perspectives (e.g., from connectionist networks to the ecological approach). The "neurocognitive action control framework" advocated here uses four main research traditions from psychology and cognitive neuroscience as starting points. These four sources will be described below before the action control theory itself will be presented in the form of thirteen hypotheses.

The first source for conceptualizing action control originates from a research tradition that can be called the "functional perspective on information processing" (e.g., Allport 1980; Heuer and Sanders 1987; Neumann 1987; Prinz 1987; Schneider 1995; Hommel, Müsseler, Aschersleben, and Prinz 2001). It assumes that basic neurocognitive operations such as perceptual, motor, memory, or attentional processes have evolved in order to allow efficient action control. In other words, an adequate conceptualization of these basic neurocognitive processes requires understanding their functions in the control of actions. Work in our own lab applied this functional perspective on information processing to the experimental investigation of visual attentional processes. My colleagues and I asked what role visual attention processes might have in controlling simple actions such as saccadic eye movements (e.g., Deubel and Schneider 1996) or hand movements (e.g., Schiegl, Deubel, and Schneider 2003).

One basic conclusion from this line of experimental research is that "selection-for-perception" processes and "selection-for-action" processes are coupled by one common attentional control mechanism (for recent overviews, see Schneider and Deubel 2002; Deubel and Schneider, in press).

The second source refers to the idea that goal states in the sense of reference states (e.g., Miller, Galanter, and Pribram 1960) or action outcome states (action effects; e.g.,

Duncan 1986; Prinz 1987; Hommel 1993) control the selection of motor actions. One variant of this goal-based action control approach is grounded within the functional view on information processing mentioned above, that is, the work by Prinz, Hommel, and colleagues (e.g., Prinz 1987, 1997; Hommel 1993; Hommel et al. 2001). Its most recent version, the "Theory of Event Coding" (TEC; Hommel et al. 2001), starts with the assumption that actions are controlled by their "effects" ("events"). Decisively, the "perceived event codes" and the "to-be-produced-event codes" (action effects) are claimed to be represented within a common representation medium. This "common coding" assumption (e.g., Prinz 1997) predicts an overlap of perceptual and action planning processes—a prediction that has been confirmed by a substantial number of experimental studies (see Hommel et al. 2001 for an overview).

Given this functional perspective on control, a guideline question is how effect- or goal-based processes are mediating the efficient control of actions in the human brain. Outside the functional view on information processing, this idea of goal-based control of motor actions has up to now not attracted a great deal of systematic research in cognitive neuroscience. The situation is beginning to change, and an increasing number of experimental and neural network studies have recently addressed the question how goal states are represented within different parts of the primate brain (e.g., prefrontal cortex; PFC) and how they may contribute to the control of action selection (e.g., Braver and Cohen 2000; Matsumoto and Tanaka 2004; Ramnani and Owen 2004; Schultz 2004). For simple actions such as saccadic eye movements, experimental and computational neurocognitive studies have now specified in considerable detail how the goal-based selection and execution of such actions may be carried out by neural computations in interacting cortical and subcortical areas (see, e.g., Glimcher 2003; Brown, Bullock, and Grossberg 2004).

The third source of the action control framework suggested here refers to the German tradition of human motivation and volition research that distinguishes action selection from action realization (for overviews, see, e.g., Ach 1935; Kühl 1984; Heckhausen 1991). A first central assumption of this kind of approach is that two main factors for determining action selection are the value of an action, on the one hand—often expressed in terms of "incentives" or "emotional values"—and, on the other hand, the estimated probability that these actions lead to the intended goal state (see, e.g., Atkinson 1957; Weiner 1980; Heckhausen 1991). A second assumption is that action selection processes should be distinguished from action realization processes (for overviews, see, e.g., Ach, 1935; Kühl 1984; Heckhausen 1991). "Action selection" refers to motivational processes that choose one action tendency among several other tendencies. "Action realization" refers to volitional processes that allow initiation and continuation of a motor action in case of strongly competing action tendencies. Up to now, theories of motivation and volition in this tradition have mainly relied on the social science research approach, that is, for example, on verbal report data (e.g.,

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Kuhl 1984; Carver and Scheier 1990; Heckhausen 1991; Grawe 2004; see also Cohen and Gollwitzer, this volume). Imaging, neuropsychological, and neurophysiological data, as well as the language of neural information processing, did not play a prominent role in this kind of approach—see, however, for instance, Goschke (2003), for a recent attempt in this direction, and, see, for instance, Adolphs (2003) for a brain science shift in social psychology in general.

Finally, the fourth source for conceptualizing action control draws on recent ideas and experimental work on “executive control” processes and their potential subdivision within the prefrontal cortex and other parts of the primate brain. A diverse set of functions have been investigated under the labels “executive control” or “cognitive control,” including processes such as “planning in problem solving,” “shifting the task set (attention),” “suppressing a prepotent response,” and “error correction” (e.g., Norman and Shallice 1986; Miller and Cohen 2001; Monsell 1996). A central common feature of these executive functions is the idea that they are required in order to prepare and execute “nonroutine” actions (e.g., Norman and Shallice 1986), that is, actions that cannot be carried out by combining current environmental information with motor information retrieved from long-term memory (e.g., stored schemas of sensorimotor skills). Based on neuropsychological, single-cell, and imaging data, the prefrontal cortex has been suggested as a central site for implementing these executive control functions (for overviews, see, e.g., Passingham 1993, Fuster 1997; Schneider, Owen, and Duncan 2000). A lively debate is still going on concerning whether and which control functions may be carried out by different parts of the PFC (e.g., Duncan and Owen 2000; Grafman and Krueger, this volume; Owen, this volume; Burgess et al., this volume). Despite this debate, a theoretical framework on control—the “biased competition” approach—that is shared by many researchers (e.g., Norman and Shallice 1986; Desimone and Duncan 1995; Miller and Cohen 2001) assumes that executive PFC control processes modulate the competition of sensory-driven lower-order brain processes located in parts of the brain posterior to PFC.

The neurocognitive theory of goal-based action control proposed here not only relies on these four theoretical sources but also attempts to combine them in a new way. For instance, the functional view of information processing—the “cognitive side” of action control—will be linked with the social science research tradition on motivation and volition—the dynamic side of action control. Furthermore, the biased competition hypothesis from the executive control literature will be used to specify the common coding assumption of perceptual and motor processes. Moreover, beyond combining elements from these four sources, a few new suggestions about action control will be made, for instance, how mood states may arise due to long-term monitoring of action control outcomes and how these mood states may influence the current selection and execution of motor actions. The overall action control theory will be presented in the following sections in the form of thirteen hypotheses (13.1.1 to 13.1.14). These hypotheses are by far not meant as a complete theory of action control—the selection of questions and the issues addressed by the theory are heavily influenced by the aim to develop an action control account of clinical depression. This account will be specified in the second part of the chapter.

13.1.1 Actions Are Controlled by Goal States, and Goal States Refer to Representations of Action Outcomes

The most fundamental assumption of the action control framework proposed here is that actions are controlled by goal states (e.g., Miller, Galanter, and Pribram 1960; Powers 1973; Anderson 1983; Duncan 1986; Carver and Scheier 1990; Hommel et al. 2001; Grawe 2004; Matsumoto and Tanaka 2004). Here, the term “goal state” refers to a state or an event that has not yet occurred. More precisely, the content of a goal state represents the outcome (or effect) of an action (see, e.g., Duncan 1986; Prinz 1987; Hommel 1993; Grawe 2004). This implies that each action is carried out in order to achieve a certain effect or outcome—an outcome that is extracted by the perceptual system and stored as a goal state in reference to the action.

Goal states can refer not just to end states of actions but also to the results of ongoing actions (e.g., parts of the melody when playing piano) or the outcomes of complex events (for complex events and their potential structure, see Grafman and Krueger, this volume). In cases of events, a dynamic variable is the reference value (goal state).

13.1.2 Goal States Are Intrinsically Linked to Motor Action Representations in the Form of “Goal-Action Episodes” (Common Coding Assumption)

What is the relationship between goal states and motor action representations? It is suggested here that they are always and intrinsically linked in the form of a “goal-action episode.” In other words, it is claimed that representations of goals and actions do not exist in isolation but always as goal-action units, named here goal-action episodes. Every goal state should be connected to a corresponding (motor) action representation.1 Many goal states can be realized by different motor actions. For instance, in football a “goal” can be shot by kicking the ball either via foot or via head. Importantly, it is suggested here that each combination of a goal state (e.g., shooting a “goal”) with a motor action (e.g., foot or head kicking) is assumed to form an individual goal-action episode.

Even for highly overlearned and compatible actions such as reading, in which simple and direct links between stimuli (e.g., words) and motor actions (verbal responses) exist, individual actions or responses are assumed to be connected to the corresponding goal states, that is, representations of action outcomes. That is, in case of reading aloud, the verbal response to a word should be always connected to its intended sound pattern. This intrinsic link between “perceptual” goal states, on the one hand, and “motor” action processes, on the other hand, implements a version of the “common coding” approach to perception and action (e.g., Prinz 1987; Hommel
et al. 2001) mentioned before as the second theoretical source. A new feature for this common coding approach is the assumption that goal-action episodes are selection units of competition for action control—see the next hypothesis, 13.1.3.

Goal-action episodes differ in respect to several dimensions (see also Graffman and Krueger, this volume). One dimension refers to the temporal domain of the goal states (e.g., Fuster 1997, 2001). In simple forms of goal pursuit, "goal states" refer to environmental stimuli that are always present during the pursuit (e.g., seeing, grasping, and eating a candy located directly in front of the actor). In more complex temporal forms, the goal stimuli are not all the time physically present and have to be maintained in working memory (e.g., imaging a special candy located somewhere in a drawer at home). Following Fuster (1997), pursuing temporally more distant goal states should require involvement of PFC areas. A second dimension of goal states refers to the flexibility of the goal-action connection of episodes. Either the connection can be well-learned and stored in long-term memory (LTM) or, alternatively, the episode may take the form of a temporary connection (e.g., short-term memory) between the goal state and the representation of an action—"mental simulation" (and not real execution) of a possible action for goal state achievement may be used in this case for setting up the temporary connection. Such flexible connections allow the linking of new but not yet learned couplings of goal states and motor action processes in an ad hoc manner (e.g., for implementing instructions).

A third dimension of goal-action episodes refers to their content domain and complexity. Goal states can refer to very simple action outcomes such as the landing position of a saccadic eye movement or the results of complex social actions—see Graffman and Krueger (this volume) for a neuropsychologically based attempt to structure goal domains of different contents and complexities with respect to different prefrontal lobes areas. Analogous to this, motor action processes can have different levels of complexities, ranging from simple motor patterns for saccade generation to more complex motor action patterns involved in complex skills such as playing football (see, e.g., Rosenbaum 1991; Arbib 1990; Krakauer and Ghazhi 2000; Graffman and Krueger, this volume). For simple actions just a few motor brain areas are recruited, while more complex actions presuppose the activation of a wide-ranging network of motor areas (e.g., Krakauer and Ghazhi 2000). Importantly, the "action" part of an episode refers here only to medium and high levels of motor representations and not to low-level representations (e.g., muscle-specific motor patterns).

13.1.3 The Units That Compete for Action Control Are "Goal-Action Episodes," and Only One "Goal-Action Episode" at a Time Controls the Execution of an Action

As mentioned above, the neurocognitive theory of action control suggested here relies on the biased competition approach to the control of attention and executive functions within the brain (e.g., Phaf, van der Heijden, and Hudson 1990; Desimone and Duncan 1995; Miller and Cohen 2001). In line with this approach, it is assumed that activated "goal-action episodes" compete for controlling the current action. Neither representations of goals in isolation nor of actions in isolation should compete but only units of individual goal-action episodes.

Competition is assumed to be regulated by the activation value (e.g., Cohen, Dunbar, and McClelland 1990; Phaf, van der Heijden, and Hudson 1990) of an episode—for factors that determine this value, see hypothesis 13.1.5. The winner of the competition is always one episode—see hypothesis 13.1.4. Competition between goal-action episodes is probably organized in a domain-specific way (see, e.g., Phaf, van der Heijden, and Hudson 1990). In line with Graffman and colleagues (e.g., Graffman and Krueger, this volume), it is assumed that complex goal-action episodes—which may be identical to Graffman and Krueger’s (this volume) "structured event complex" (SEC) sequences—and also less complex "episodes" containing parts of SECs—are stored in prefrontal cortex. These sequences should also compete as goal-action episodes.

Why is competition between episodes and the determination of one winner for controlling the execution of action necessary? The functional view of information processing suggested the following answer: Given that humans (and other primates) have a large repertoire of stored goals and corresponding actions, and given that internal (e.g., body-related) and external environmental stimuli often activate several goals simultaneously, it is important to prevent behavioral chaos—chaos that may, for instance, emerge from the simultaneous execution of incompatible actions. The behavioral chaos can be prevented by allowing only one goal-action episode at a time (e.g., Neumann 1987) or "one list of goals" (Duncan 1986) at a time—to control the execution of overt action (see, also, Norman and Shallice 1986). Here, this episode is called the "goal-action episode in charge." This capacity limitation of one episode in charge at a time refers only to the currently highest level of control. At lower levels of the goal-action episode hierarchy, several "subgoal-action episodes" might be simultaneously activated.

13.1.4 The Competition between Goal-Action Episodes Is Regulated on the Basis of Their Activation Values that, in Turn, Are Determined by Stored or Anticipated Emotional States, Importance, Urgency, and Action-Outcome Probability Estimates

External and internal stimuli usually activate several goals simultaneously that are stored in LTM. Which goal-action episode controls the current action and gets the status of the episode in charge? It is assumed that the episode with the highest activation value wins the competition. The selection principle is suggested to rely on the winner-takes-all principle of many connectionist models (e.g., Phaf, van der Heijden, and Hudson 1990; Cohen, Dunbar, and McClelland 1990). Given this winner-takes-all principle for competition resolution, the question arises of how the activation
values of goal-action episodes are determined. In line with research on human motivation (see, e.g., Weiner 1980; Heckhausen 1991; Damasio 1996) it is assumed that four not necessarily exclusive variables determine the episode activation value, namely, emotional states stored in relationship to past attempts of the action to pursue the goal, importance of the goal-action episode, urgency of the goal-action episode, and the estimated probability that the action will be able to achieve the goal state; this list of factors is not meant to be complete.

First, in line with classical work on the psychology (e.g., Atkinson 1957; for an overview, see Weiner 1980) and neuroscience of motivation (e.g., Damasio 1996; Rolls 2000) it is assumed that competition and selection among goal-action episodes depends on stored emotional states associated in the past with successful or failed goal pursuit. "Emotions" may refer to the evaluation of the action outcome itself—for example, emotions of a pleasant or unpleasant kind. Furthermore, emotions may emerge as a consequence of successful or failed goal pursuit, that is, on the basis of the comparison between expected goal state (retrieved from memory or anticipated via mental simulation) and actual action outcome. Different kinds of emotions are probably computed in different brain areas such as amygdala or orbitofrontal cortex (e.g., LeDoux 2000; Rolls 2000).

Consistent with the dimension approach to emotion (for an overview, see, e.g., Gross 1995), it is assumed that emotions can be categorized as either of the positive (pleasant) or negative (unpleasant) type. It is assumed that these two types of emotions determine which kind of goal—desired or undesired—is generated (see also Gray 1990; Davidson et al. 2002) and which kind of episode is emerging. If in connection with an episode primarily positive emotions are stored, the goal state should be a desired state and the episode will be an approach episode. If in connection with an episode primarily negative emotions are stored, the goal state should be an undesired state and the episode will be an avoidance or withdrawal episode—a possible consequence may be a withdrawal action away from the goal object (e.g., Gray 1990; Davidson et al. 2002). A further function of emotion—besides tagging a goal state of an episode as a desired or undesired state—may be to influence the episode competition value via the strength of emotions. The strength of a conscious emotion (feeling) may be directly related to the competition value. The stronger a feeling evoked by a previous pursuit of a goal episode is, the higher the current competition value of this episode should be (see also Damasio 1996; Rolls 2000). Besides relying on stored information (LTM) about emotions associated with past goal pursuit, the mental simulation of a new action, more precisely, the mental simulation of the execution of an action not performed before, should also be able to anticipate possible emotions associated with successful or failing goal pursuit and should therefore be able to bias episode competition.

Second, it is assumed that goal competition depends on urgency and on importance of the goal state (see, e.g., Lewin 1926; Heckhausen and Kuhl 1985). Some goals such as the central representation of physiological needs (e.g., hunger, thirst, sex) have different degrees of urgency depending on the currently computed state of deprivation. The stronger the deprivation, the higher will be the urgency value of the corresponding goal. Another important factor in determining the urgency of an avoidance action is the expected occurrence of an undesired state. The closer the time of occurrence is, the more urgent should be the goal and the more urgent should be the initiation of an avoidance action.

"Importance" refers to a further class of factors determining the goal competition value. It is reasonable to assume that goals related to fundamental needs (e.g., hunger) should have a relatively high importance value. Social psychologists have long been claiming that goals in humans related to central aspects of the "self" (e.g., self-esteem; e.g., Crocker and Park 2004; Grawe 2004) have a high priority in action control. It is not claimed here that the variables of importance and urgency are completely separate from emotional processes—both variables may be connected to certain affective states. Currently, however, at least to my knowledge, no specific and empirically tested hypotheses about the relationship between importance, urgency, and emotions exist.

Third, a further factor that determines the competition value of an episode refers to action-outcome probability estimates (e.g., Weiner 1980; Heckhausen 1991). Probability estimates can be based on records of actual action executions of the past stored in LTM. They can also be derived from anticipation, that is, mental simulation, of the action execution. At least two kinds of probability estimates in relationship to goals and actions have to be distinguished (see Heckhausen 1991 for further options). First, the estimate can refer to the probability that a certain action is able to achieve the goal state—indeed independent of a specific actor. Second, the estimate can refer to the availability of the required action to the actor—it reflects the action competence of the actor—similar to Bandura's (1977) concept of self-efficacy. The first probability to achieve goal X by action Y may be high—given knowledge of the actor about this relationship—but the second probability that the actor himself or herself is able to produce the action—either by LTM retrieval or by mental simulation—may be quite low.

How importance and probability estimates may be represented in the different areas of the primate brain has been investigated for "visual-saccadic decision making"—see, for example, Glimcher (2003) and Schultz (2004). Parietal and frontal areas at the cortical level, as well as parts of the basal ganglia (BSG; e.g., striatum) at the subcortical level, seem to be involved in this kind of computation.

13.1.5 When the Context for Initiation Is Perceived, a Go Signal Will Be Issued for Action Execution

Not every goal-action episode that wins the competition immediately causes the execution of corresponding motor processes and, therefore, an overt action. Humans are
able to delay actions that have been completely prepared. Whether the motor action process of an episode is executed or not is a matter of context. Human action is characterized by a high degree of context dependence (e.g., Miller and Cohen 2001). Context can refer to spatial, temporal, and more complex, semantically defined conditions (see also Grafman and Keueger, this volume). It is assumed that these context representations are part of goal-action episodes. This context dependency of human action and cognition is sometimes computationally modeled as control by “If condition X, then action Y” linkages, also called “conditional operations” (e.g., Newell and Simon 1972; Anderson 1983; Meyer and Kieras 1997). In our terminology, this means “If context X, then action Y.” Such linkages have been found to be coded at the level of single neurons within the PFC (see, e.g., for an overview, Miller and Cohen 2001).

How might the action execution be controlled? A go signal controlled by the BSG is a very plausible suggestion given current evidence (e.g., Schulz 1999; DeLong 2000). Once the context for initiation of a motor action process is detected, inhibition by BSG should be removed, that is, a go signal for the execution the goal-action episode, more precisely, for executing the motor action part of the episode, should be given.

How does the ability of human “prospective memory” (e.g., Burgess et al. 2003; Burgess et al., this volume) fit into this conception? For instance, think about Lewin’s (1926) famous example of prospective memory “in action,” namely, the intended action to put a recently written letter in a mailbox when such a mailbox is encountered on one’s way home. Here, the current action (e.g., walking home) has to be briefly interrupted for performing the context-adequate action stored in prospective memory, namely, the action of putting a letter into a mailbox. In order to explain how processing works in such prospective memory examples, it is assumed that the perceived context information not only could lead to a go signal of a prepared and inhibited episode but may also deliver input to the goal-action episode competition system that may cause the replacement of the current episode by the context-defined episode. Structures involved in setting up and storing context for prospective action are probably the hippocampus (HC; e.g., Jefferery 2004) and maybe PFC area 10 (see Burgess et al., this volume, but also Raminani and Owen 2004; Miller and Cohen 2001).

### 13.1.6 Persistence and Intensity of Action Execution as well as the Probability of a Goal-Action Episode Shift Depend on the Episode Competition Value

Persistence and intensity of motor action execution are important parameters in action control (e.g., Heckhausen 1991). If an action does not lead to immediate success, a decision has to be made in terms of persistence, that is, how often and how long attempts to achieve the goal via this action should be executed. It is assumed here that the goal-action episode competition value determines persistence (see also Heckhausen 1991). The higher the competition value, the stronger the degree of persistence should be. Furthermore, the intensity of a motor action process execution should also depend on the competition value. The higher the competition value, the higher the intensity should be. Finally, the competition value should be related to the probability of a goal-action episode shift, that is, a change of the episode in charge (see, e.g., Goschke 2003). The lower the competition value of the current goal (e.g., due to low importance or urgency), the higher goal shift probability should be—see, for example, Monsell (1996) for an overview on the experimental literature on goal (task) shifting factors.

### 13.1.7 Executive Control Processes Are Called in Case of Action Failure, That Is, When the Action Did Not Produce the Intended Goal State and When the Probability Estimate for Being Successful by Repeating the Action Is Low

Sometimes, routine actions stored in LTM are not sufficient to achieve the intended goal state. As stated in the introduction to this section, executive control processes should be called in this case of nonroutin action (e.g., Norman and Shallice 1986). More precisely, it is suggested here that executive processes are called when the result of a simulated or actually executed motor action process is “negative,” that is, the action of the winning episode was not able to achieve the goal state and the discrepancy between goal state and action outcome continues to exist. A second necessary condition for the call should be a low probability estimate that a repeated execution of the currently selected and failing action will be successful (see also Botvinick, Carter, Brauer, Barch, and Cohen 2001 for a different concept of calling executive operations). If the probability is sufficiently high, persistence of the already selected action should occur. A further condition for triggering executive processes might be given when the rate of discrepancy reduction between current state and goal state (e.g., Miller, Galanter, and Pribram 1960) is too slow (e.g., Carver and Scheier 1990).

Which brain areas may be important for calling executive processes? Executive processes themselves are probably stored within the (lateral) PFC (see, e.g., Duncan and Owen 2000). The detection of the need to use executive processes—discrepancy detection—and the call of these processes is assumed to be carried out by the anterior cingulate cortex (ACC); for imaging, EEG, and other sources of empirical evidence, see, for example, Botvinick, et al. (2001), Paus (2001), and Holroyd and Coles (2002). For instance, the error-related negativity (ERN) in EEG recordings observed after action errors can be localized within the ACC (see, e.g., Holroyd and Coles 2002). ERN may not just reflect action failures (errors) but may also arise when executive operations are called, that is, when the actual or simulated action application was not successful and the probability estimate for a successful, repeated application of the same action is lower; for a different view on executive control and the role of the ACC, see Botvinick et al. (2001).
13.1.8 If the Goal–Action Episode in Charge Is in Danger of Being Replaced by Competitors, Then Volitional Processes Located within the ACC Accomplish Shielding of the Episode in Charge

The term “volition” is sometimes used in the broad sense of the word, equating volition with the control of “consciously-based” action (see, e.g., Grafman and Krueger, this volume). Here, the term should be used in the narrow sense (e.g., Ach 1935; Kuhl 1984; Heckhausen 1991), namely, that the currently pursued action (goal–action episode in charge) is in danger of being replaced by competing actions (episodes) that may be internally or externally cued and that shielding processes are required for maintaining the current goal pursuit (see also Prinz, Dennett, and Sebanz, this volume). For instance, writing a paper during a beautiful summer day may be threatened by a friend asking for an external event (e.g., for company to go to an ice cream. Alternatively, an internal event such as a feeling of hunger might compete with writing a paper. Within the framework of the action control theory suggested here, these situations can be characterized as follows: The current goal–action episode competes with other externally or internally triggered episodes that have a higher current competition value. Without additional processes, the episode in charge would be replaced by another competing episode. Extra volitional processes are needed that temporarily support the current episode in charge with additional activation so that its overall activation is higher than the activation of the competitors.

This idea presupposes that the danger of replacement of the current episode in charge is detected and that this detection causes a call of volitional shielding processes (see also, e.g., Ach 1935; Kuhl 1984). The shielding process may work by supplying extra phasic activation to the goal in danger. This phasic activation should then increase the goal–action episode competition to a value sufficient for staying in charge. One way to implement this extra activation supply would be to instantiate a further temporary goal of high activation value that might correspond to the feeling of “will” and that sends activation to the episode in danger. A loop between this volitional goal episode and the episode in danger may be a possible mechanism for maintaining the increased phasic activation.

An experimental example of such a volitional situation is the Stroop task, in which the strong prepotent response tendency of word reading has to be suppressed and the weaker response tendency of color naming has to be executed for correct performance. Without the experimental instruction, subjects would read the word, but the temporary goal of color naming prevents this and allows articulation of the color name. Viewed this way, the executive process of inhibiting a prepotent response (e.g., Miller and Cohen 2001) would be a volitional process (see below). Volitionally supported goal–action episodes should not be irreplaceable—otherwise, important environmental changes (e.g., a dangerous event) would not be able to replace the shielded goal (e.g., Goschke 2003). An experimental technique that measures the volitional capabilities of subjects is the “goal neglect” paradigm developed by Duncan and colleagues (e.g., Duncan et al. 1996). The task requires shifting occasionally from the currently executed main task to a less-often-performed secondary task. The call for the task shifting is given by an environmental signal. For correct performance, the task shifting set (including the signal) has to be maintained in prospective memory. Subjects with PFC damage or with a low G factor show “goal neglect,” that is, they have problems in carrying out the task shift (see Duncan et al. 1996). Within the action control theory suggested here, goal neglect should arise due to volitional problems in maintaining the temporary supported goal episode of the secondary task.

Given the anatomical connections of the ACC with other action-control-related cortical and subcortical structures, and imaging data that ACC is activated in Stroop and other volitional tasks (for an overview, see e.g., Miller and Cohen 2001; Paus 2001), and findings that large ACC lesions can lead to akinetic mutism—a disorder of volitional action initiation (see, e.g., Bush, Luu, and Posner 2000; Paus 2001; Holroyd and Coles 2002)—this brain structure seems to be a reasonable candidate for calling and executing volitional control. Volitional processes are usually perceived as “effortful” and the same holds for most executive processes that are needed in case of nonroutine actions and should also be mediated by the ACC (see section 13.1.7). As stated above, volitional processes may refer to a subset of executive control processes (e.g., inhibition of prepotent response) that are called when the current goal–action episode is in danger of being replaced. This assumption fits with the last hypothesis in section 13.1.7 saying that the ACC should be involved in calling executive control processes. Thus, when effortful processing in the sense of executive or volitional processing is required for action control, the ACC should be the structure for calling and executing this type of processing.

13.1.9 Simulated or Real Outcomes of Actions Lead to the Generation of Emotional States, to Modifications of the LTM Connections between Goal–Action Episodes and Emotional States, and to Computations of Probability Estimates of Action–Outcome (Goal) Relationships

“Goal states” refers to the intended outcomes of an action associated with the goal–action episode. When the outcome of an action is perceived—due to either an actually executed or an anticipated (simulated) action—then a comparison between the goal state, (the intended action outcome) and actual action result should be made (e.g., Miller, Galanter, and Pribram 1960; Carver and Scheier 1990). Three kinds of events are important for action control as an outcome of this comparison process, namely, the generation of emotional states, LTM modifications of connections between goal–action episodes and emotional states, and the computation of action–outcome (goal) probability estimates.
First, depending on the result of this comparison, and influenced by context processing (e.g., attributions of action result; e.g., Weiner 1980; Heckhausen 1991), emotional states, more precisely, feelings (the conscious part of emotions), should arise. In case of success, that is, when a desired goal state is realized, positive feelings of pleasure or happiness may arise. When an undesired state is removed or avoided, positive feelings of relief should arise (e.g., Weiner 1980; Heckhausen 1991; Rolls 2000). The intensity of the feeling may be modulated by the importance and urgency of the goal pursuit. In case of a failure to achieve the goal state, negative emotions should arise—for example, anger or shame depending on context and further cognitive inference processes (see, e.g., Weiner 1980; Heckhausen 1991). A brain structure for computing emotional states based on the comparison between the intended (goal) state and the actual action outcome may be the ACC. As stated above in sections 13.1.7 and 13.1.8, the ACC is possibly involved in detecting discrepancies for calling executive and volitional processes, so it seems reasonable to suggest that the ACC may also be involved in detecting this emotion-generating discrepancy. The emotional subdivision of the ACC (e.g., Bush, Luu, and Posner 2000) may be the place for generating the comparison-based emotions that may actually be stored somewhere else in the cortex (e.g., amygdala or other limbic structures).

Second, the comparison between intended and actual action outcome should cause modifications of long-term connections between the goal-action episode and emotions. In general, connections between emotions and an episode should be adjusted according to the action result and its comparison-based emotional state. Connections of episodes to emotional states stored as results of previous actions may be weakened or strengthened depending on the congruence between expected and actual (or simulated) outcome states; connections to emotional states that arise for the first time as a result of an action may be stored as new connections between emotion and episode.

Third, probability estimates of action-outcome (action-goal state) relationships should be modified as a result of goal pursuit. If the action was successful, then an increase in the probability estimate that the action will lead to the goal state (intended outcome) should occur. In the case of failure, a decrease in the estimate should occur. Instead of modifying explicit representations of probability estimates, the connection between the goal state and action representations could be modified as an implicit representation of probability estimates. For instance, an action failure could lead to a weakening of the goal-action connection within the episode.

Given the suggestion that the ACC computes the discrepancies for issuing comparison-based emotional states, it seems reasonable to assume that the ACC may initiate—in combination with the BSG—these LTM modifications of connections between goal states, action representations, and emotional states (e.g., Holroyd and Coles 2002) and may also initiate the probability estimate modifications.

13.1.10 “Metamonitoring” of Action Outcomes Generates Bias Signals for Episode Competition in the Form of “Probability Estimates of Action–Outcome Relationships” and Mood States

For efficient action control, it is necessary not just to monitor and store the outcomes of individual actions but also to metamonitor the results of the same action across repeated executions over a longer time scale. That metamonitoring plays a central role in action control has already been suggested by Carver and Scheier (1990, 1999), but they meant something different by this concept. They suggested that a low-level comparison process should compute the discrepancy between the goal state and the current action result, while a metacomparison process should detect the “speed” of discrepancy reduction during action execution. Here it is agreed with Carver and Scheier (1990, 1999) that such a metamonitoring process exists, but its function is conceptualized differently. It should not check the rate of discrepancy reduction for each individual action execution, but it should check the results of a repeated pursuit of the same goal-action episode at a longer time scale, that is, involving the stored results of several instances of pursuing the same goal.

Moreover, it is suggested here that metamonitoring leads to two major consequences. First, probability estimates of the action-outcome relationships (see section 13.1.9)—in the case of explicit representations called “beliefs”—should be produced or modified, and, second, “moods” should be generated. First, these beliefs should refer to the previously mentioned explicit (probably propositional) representations of probability estimates that an action leads to the achievement of the goal state of the episode. A major function of beliefs or implicit probability estimates is to bias episode competition. For instance, in the case of one goal and several possible actions for realizing the goal, competition between the corresponding episodes should occur. A high probability of an action X for successful goal pursuit should increase the chance of the corresponding episode’s being selected, while a low probability of another action Y should decrease it (see Glithofer 2003 for evidence of such processes in parietal cortex in saccadic decision making).

Second, high-level monitoring should generate not just beliefs, but also moods—affective states with longer durations than emotions that are characterized by brief durations (e.g., Gross 1999). Along the suggestions about comparison-based emotions of section 13.1.9, it is assumed that successful pursuit of the same goal-action episode on several occasions (over time), that is, successful and repeated discrepancy reduction between the goal state and the current state (Miller et al. 1960) by the same action, should lead to a positive, happy, optimistic mood. Repeated failure to reduce discrepancies should lead to an angry, hostile, or depressive mood dependent on attribution processes (see, e.g., Weiner 1980). A major function of mood is to bias the episode selection process. It is assumed here that mood states strengthen—in line with the congruency principle—emotions of the same type (positive or negative emotion).
during the episode competition and weaken emotions of the opposite type. Consequently, episodes containing mainly emotions of the same type as the current mood will receive an activation increase of their competition value, and episodes containing mainly emotions of the opposite type will receive a reduction of the competition value. For instance, in the case of a depressive mood, those avoidance episodes associated with negative emotions should get a higher competition weight as compared to approach episodes with positive emotional values. Therefore, depressive mood should increase the chance of withdrawal or avoidance behavior controlled by episodes with negative emotional tags, and it should decrease the chance of approach behavior controlled by episodes with positive emotional tags.

Goal pursuit might be monitored not only regarding a certain goal but also with respect to a domain of goals (e.g., academic achievements). The results of such a domain-wide metamonitoring process may be estimations of "own abilities," that is, "self-related beliefs" (see, e.g., Heckhausen 1991). Verbally specified self-estimations such as "I am a loser" may reflect the result of a generalized metamonitoring process that refers to several important domains of life (e.g., personal relationships, professional career, faith, etc.). These generalized monitoring results should lead to generalized probability estimates.

13.1.12 A "Stress Response" Elicited by "Goal Pursuit in Danger" Leads to Increased Cortisol Output

A stressor is viewed here as a stimulus or event that activates a goal-action episode (in charge) that is characterized by high importance, high urgency, and low probability of successful goal pursuit. In other words, a stressful event activates an episode whose realization is in danger—the calling condition for executive and possibly volitional processes. Due to the action-outcome-related probability estimation, normal (routine) processing is expected to be insufficient for achieving the goal state. Extra effortful processing—either executive or volitional processes—should be mobilized in this case. In line with substantial evidence (for an overview, see McEwen 2002), it is assumed here that in the case of a stressful event what is initiated is not just effortless executive and volitional processing but also an increase of cortisol emission for delivering the extra energy for coping with the event—the cortisol level increase may be directly related to the degree of effortful processing.

What "stress" adds to the issue of estimated nonobtainment of important goals is the dimension of urgency. The goal pursuit cannot be delayed. In this case, it seems reasonable to assume that the degree of stressfulness of a goal-action episode in danger may depend on the goal competition value. Following section 13.1.4, the competition value should depend on how low the probability of achieving the goal by a certain action is—uncontrollability (e.g., Heckhausen 1991; Dickerson and Kemeny 2004)—and on how urgent and important the goal achievement is. The higher the urgency and importance are, and the lower the probability is, the higher the competition value of the goal would be, the more stressful an event should be, and the more cortisol should be emitted (see Dickerson and Kemeny 2004 for a recent meta-analysis of the role of uncontrollability in cortisol emission).
13.1.13 Chronically Stressful Events and, as a Consequence, a Chronically Increased Cortisol Level Can Damage Action-Control-Related Brain Structures (e.g., HC)

Chronically stressful events, that is, the repeated experience of goal pursuit in danger, causes a prolonged emission of cortisol and probably leads to a slower than normal return of free cortisol to baseline (e.g., McEwen 2002; Dickerson and Kemeny 2004). Consequently, over time, important brain structures for action control should be subject to a chronically high cortisol level, which is toxic for these structures. For the HC, such a damaging effect of chronically increased cortisol has been empirically confirmed (see, for overviews, McEwen 2002; Davidson et al. 2002); however, damage in HC can be reversed (e.g., McEwen 2002) if stressful events and cortisol level are reduced to a consistently normal level. Besides the HC, chronically increased cortisol may damage the ACC (which contains cortisol receptors) and parts of the PFC too—areas that are important in action control and whose functions are reduced in clinical depression (e.g., Davidson et al. 2002; Kempermann and Kronenberg 2003).

13.2 Clinical Depression as a Failure of Goal-based Action Control

In this chapter, the analysis of clinical depression is restricted to the primary type, that is, depression that is not a secondary consequence of another disorder such as Parkinson’s disease or dementia, and to the unipolar type, that is, depression without mania (e.g., Cömer 2001). In line with the neurocognitive framework, selected data from empirical depression research of the last decades that refer to all levels of the mind/brain system—ranging from experimentally measured behavioral deficits in humans and animals to brain activation and structural changes in specific cortical and subcortical areas (see, for recent overviews, e.g., Drevets 2001; Davidson, Pizzagalli, Nitschke, and Putman 2002; Gotlib and Hammen 2002; Nitschke and Mackiewicz, this volume) will be considered here.

The action control theory developed in the first section of this chapter will be used as the conceptual tool for understanding the mechanisms that cause and maintain the symptoms of depression. Along these lines of reasoning, depression is viewed as a disorder of action control characterized by an increased level of inactivity and avoidance behavior as well as a decreased level of approach behavior (see, e.g., Levinsohn et al. 1984; Kasch et al. 2002). Such a point of view on depression has recently been put forward by R. J. Davidson and colleagues (e.g., Davidson and Irwin 1999; Davidson et al. 2002, 2003). Davidson’s framework for understanding depression consists of two main sources. The first source is “affective neuroscience” (e.g., LeDoux 2000; Davidson and Irwin 1999) that aims to understand brain processes and representations of emotion regulation at the neurocognitive level. Structures of the primate brain such as PFC, the amygdala, the ACC, and the HC are suggested to play a central role in emotional processing. The second source refers to Gray’s (e.g., 1990) distinct-

13.2.1 Repeated Failures in Realizing Important Goals Are Perceived via Metamonitoring and Cause State-Dependent Changes of Action Control in Terms of Depressive Mood and of Negative Probability Estimates

Several psychological theories (e.g., Klinger 1975; Kuhl and Helle 1986; Pyszczynski and Greenberg 1987; Carver and Scheier 1990) have suggested that depression arises due to (short- and long-term) consequences of not being able to realize important goals. This perceived realization failure refers to unsuccessful action execution, that is, execution that was not able to achieve the goal state.

As stated in section 13.1.10, metamonitoring processes should evaluate the repeated attempts to pursue goals. In the case of repeated failures to achieve the intended action outcome, metamonitoring should initiate, first, the generation of negative action–outcome probability estimates—in the case of explicit representations called beliefs; see also Beck (1967) and Abramson, Alloy, Hankin, Haefel, MacCoun, and Gibb (2002) for psychological theories that ascribe a central role to negative beliefs in causing and maintaining depression. As stated in previous sections, these action–outcome probability estimates can refer to the probability that the actor is able
to perform the required action. Explicit probability estimates in the form of negative beliefs should be characterized by an estimated low probability of being able to successfully pursue the goal in question. Activated negative belief representations should send bias signals to the goal-action episode competition. The perception of repeated failures in pursuing important goals by metamonitoring processes should be sufficient to trigger the activation of such beliefs or implicit probability estimates.

Second, in cases of repeatedly perceived action control failures, metamonitoring should also cause the state-dependent initiation of an extended negative depressive mood state—a central characteristic of a unipolar mood disorder such as major depression (American Psychiatric Association 2000). Following section 13.1.10, depressive mood states should have a generalized effect in action control because they concern all activated goal episodes—see the next hypothesis. Third, the currently perceived goal pursuit failure by metamonitoring should also lead to the state analysis mode of action control that is characterized in this case by “ruminations” (e.g., Nolen-Hoeksema 2000). A main function of state analysis and rumination is to allow the restructuring and disengagement of goals that seemed to be nonrealizable (e.g., Klinger 1975).

13.2.2 State-dependent Depressive Mood Causes a Reduction of Approach Actions and an Increase of Avoidance Behavior

How do activated negative beliefs, the state analysis mode, and the current depressive mood bias goal-action episode competition and selection in a short-term way? Following hypothesis 13.1.10, it is assumed that the goal-action episode selection is biased in a generalized way by negative depressive mood. More precisely, the congruency principle (section 13.1.10) predicts that negative mood should increase the value of all episodes primarily tagged with negative emotional states and should probably decrease the value of episodes primarily tagged with positive emotions. Consequently, during depressive mood, the episode value should be lower for approach goal-action episodes that are primarily tagged by positive emotional values (section 13.1.4)—these approach goals should therefore have a lower probability of winning the competition for action control compared to a state of nondepressive mood. This predicted feature of less approach behavior (see also Davidson et al. 2002) has, for instance, been confirmed for patients with major depression measured by self-reports of actions (Kasch, Rottenberg, Arnow, and Gotlib 2002). Furthermore, the increased impact of negative emotions by the depressive mood should increase the competition value of withdrawal- or avoidance-related goal-action episodes that are primarily tagged by negative emotions. Therefore, the probability of selecting avoidance episodes should be increased, and more avoidance actions should be issued. This second type of behavioral change has also been confirmed by the Kasch et al. (2002) study. Moreover, a substantial reduction of this increased avoidance behavior of depressive patients is a central goal of cognitive-behavioral psychotherapies (e.g., Jacobson, Martell, and Dimidjian 2001).

13.2.3 State-dependent Depressive Mood and Negative (Low) Probability Estimates Cause Impaired Decision Making, as well as an Increase of Calls of Executive Control and Volitional Processes

Given the reduction of approach actions and the increase of avoidance actions, decision making in the sense of determining an episode competition winner should also be hampered by depressive mood. The episode activation value difference between competing approach goal-action episodes should be less due to their lower overall values. The opposite should be true for avoidance actions. Thus, on the one hand, decisions between approach goals should be harder to achieve. This reduced goal competition value in approach goals should also lead to less persistence and more interruptions or goal shifts (see hypothesis 13.1.6). In order to avoid goal-action episode shifts, volitional shielding processes have to be called (section 13.1.8). On the other hand, decisions between an avoidance goal-action episode and competing approach goal-action episodes should be easier to achieve due to the increased competition value of the avoidance episode. Persistence for avoidance episodes should also be increased, and goal shifts should be reduced. To my knowledge, there are no empirical data on these predictions distinguishing number, persistence, and shifts of approach and avoidance actions.

As stated in section 13.1.7, executive control processes should be called in the case of actual or anticipated action failure, that is, when the real or simulated action did not produce the intended goal state and when the probability estimate for being successful by repeating the action is low. Therefore, more executive processes should be called in case of depressive mood. How well these executive processes and volitional processes may work will be discussed in sections 13.2.9 and 13.2.10.

13.2.4 Modifications in LTM due to Repeated Failures in Realizing Important Goals: I. A Decrease in the Number of Stored Approach Episodes and an Increase in the Number of Stored Avoidance Episodes

A second, sustained consequence of repeated nonattainment of important goals refers to learning, that is, to modifications of specific LTM connections between emotional states and goal-action episodes. These connections should be modified according to the principles of “operative conditioning” and “reinforcement learning” (e.g., Bower and Hilgard 1981) so that selection of failing goal episodes will be less probable in the future. More precisely, after every failed goal pursuit, connections between the episode and negative emotions should be increased and connections to positive emotions
should be decreased. For approach goal-action episodes, at some point in the 
learning history of repeated failures, these episodes may turn from desired into unde-
sired, that is, into avoidance episodes. Consequently, the number of approach episodes 
should decrease during the course of depression while the number of avoidance episodes should increase (see, e.g., Jacobson et al. 2001; Kasch et al. 2002). In other 
words, the depressive state-dependent tendency to select less approach and more 
avoidance goal-action episodes (section 13.2.2) should be materialized in the form of 
a long-term correlate.

13.2.5 Modifications in LTM due to Repeated Failures in Realizing Important Goals: 
II. Decreased Persistence and Increased Switches of Approach Episodes in Charge
Similar to short-term changes, a long-term-based reduction of persistence of action 
execution and an increase of goal-action episode shifts should occur. Given that 
number and intensity of action execution calls depends on the goal competition value 
hypothesis 13.1.6), persistence should be reduced due to chronically reduced com-
petition values of approach episodes. These reduced competition values should also 
make a replacement of the current approach episode by competing goal-action episodes—episode shift—probable to a higher degree (see hypothesis 13.2.3). For 
avoidance episodes, the opposite pattern with respect to episode shift should hold. 
Current avoidance behavior should be harder to replace by competing approach episodes.

13.2.6 Modifications in LTM due to Repeated Failures in Realizing Important Goals: 
III. A Generalized Reduction of Probability Estimates (Negative Beliefs)
A further sustained consequence of repeated nonattainment of important goals refers 
to explicitly represented probability estimates of action-outcome relationships in 
the form of beliefs. First, due to learning of repeated failure, probability estimates for 
actions to lead to intended goal states (action outcomes) should be reduced (sections 
13.1.9 and 13.2.1). Second, if several attempts to pursue the same goal by alternative 
actions—that is, several episodes containing the same goal but different actions—were 
unsuccessful, the probability that any kind of action for this goal is available for the 
actor should also be reduced. In other words, perceived “self-efficacy” (e.g., Bandura 
1977) should be diminished (see hypothesis 13.1.9). Third, if several important goals of 
one action domain (e.g., the domain of interpersonal action abilities) could not be 
realized via different actions, a generalized probability estimate, a generalized belief, 
will emerge (see hypothesis 13.1.9, as well as, e.g., Beck 1967; Abramson et al. 2002). 
For instance, if several attempts to realize the fundamental goal of experiencing inti-
macy did not work over a longer time period, then a negative belief about this social 
action domain should be generated.

13.2.7 A Depressive Episode Triggered by a Stressful Event Is Maintained by an 
Increased Frequency of the “State Analysis Mode” and by Anticipated Negative 
Goal Pursuit Outcomes
As stated in sections 13.1.12 and 13.1.13, a stress response may arise when the organ-
ism perceives high importance and urgency but a low probability of goal pursuit. The 
cortisol emission should be strongly increased in this case. If the stressful event and 
the associated goal-action episode have been subject to repeatedly failing goal real-
ization attempts in the past, the short- and long-term changes in action control (e.g., 
depressive mood, negative belief activation) specified before (sections 13.2.1–13.2.6) 
should occur. Important for clinical depression is the fact that depressive mood 
continues to exist, that is, a depressive episode emerges—despite the disappearance 
of the stressful event (or of other negative results of goal pursuit) or, in case of 
severe depression, even despite the presence of positive events. It is assumed here that 
this continuation of depressive mood beyond the stressor occurs because the state analysis mode of action control (including rumination) will be maintained or 
called with an increased frequency (compared to neutral or positive mood). The main 
reason for this maintenance or increased frequency of calls should be found in the 
long-term modifications of the action control system made by repeated failures to 
pursue important goals. A state analysis mode is initiated when actions for goal pursuit 
fail or when the failure is anticipated. This second case should be critical for the 
maintenance of depressive mood. Due to long-term modification in directions of gen-
eralized negative beliefs, that is, generalized low probability estimates (hypothesis 
13.2.6) and due to an decreased competition value of approach goal-action episodes 
hypothesis 13.2.5), the anticipation of action outcomes should suffer from generat-
ing an increased number of negative results. The anticipated negative results, in 
turn, should contribute via metamonitoring to the maintenance of the depressive mood.

13.2.8 Chronic Stress Causes HC Damage and Deficits in Context-guided Action, 
That Is, Leads to Reduced Execution of Routine Action and to Fewer 
Context-Triggered Episode In Charge Shifts
The activation of goal-action episodes with low probability for achieving important, 
urgent goals—that is, stressful events—should cause a prolonged emission of cortisol 
and probably a slower than normal return of free cortisol to baseline (see hypotheses 
13.1.12 and 13.1.13). Consequently, over time, important brain structures for action 
control such as the HC, ACC, amygdala, or parts of PFC may be damaged by stress-
induced, chronically increased cortisol. However, experimental evidence for the dam-
aging effect of cortisol exists—to my knowledge—only for the HC (see, e.g., Davidson 
et al. 2003; McEwen 2002; Kempermann and Kronenberg 2003). As stated in section:
13.1.5. The HC should be involved in prospective memory, that is, in setting up and retrieving the context conditions for action execution. Consequently, HC damage should lead to deficits in the context guidance (e.g., Davidson et al. 2002) of action execution. Context, in the sense of action triggers, may be less easily and less often retrieved. This, in turn, should lead to disturbances of context-guided daily routine actions that are characteristic for clinical depression (e.g., Jacobson et al. 2001). Consequently, the overall level of approach activity should decrease (e.g., Kasch et al. 2002). Furthermore, a switch of action in the direction of a context-induced goal-action episode—remember Lewin’s (1926) mailbox example of section 13.1.5—should occur less often, and depressives should be stuck more often within the currently pursued goal-action episode.

13.2.9 In a First Phase of the Mood Disorder, Chronic Stress Should Lead to PFC Damage (ACC Still Intact) and to a Less Severe Form of Depression Characterized by Deficits in Executive and Volitional Processes and by Low-Probability Estimates for Successful Goal Pursuit

From functional and structural brain imaging studies, it is known that in individuals with depression, there is a reduction in not only the HC activation and HC volume but also the functioning of ACC, and of parts of the PFC (for an overview, see, e.g., Drevets 2001; Davidson et al. 2002). It is assumed here (see also Davidson et al. 2002) that HC and PFC damage occurs prior to the more far-reaching ACC damage as a first response to chronic stress and as a first phase of a less severe depressive disorder. Given that depressive mood and low probability estimates should lead to an increased call of the executive and volitional processes (see hypothesis 13.2.3), and given that executive and volitional processes may be located within PFC areas (see, e.g., Norman and Shallice 1986; Schneider, Owen, and Duncan 2000; Miller and Cohen 2001), PFC damage should cause problems in the efficiency of executive as well as volitional processes in depressives. For executive processes, this efficiency reduction has been empirically supported (see, e.g., Austin, Mitchell, and Goodwin 2001; Porter, Gallagher, Thompson, and Young 2003) but—to my knowledge—no data exist on the reduction of volitional processes.

Furthermore, if executive and volitional operations are not working properly in the service of goal pursuit, feedback about this failure (e.g., stored in LTM or simulated) should further decrease the probability estimates of a successful pursuit of goals in danger, that is, further decrease the probability estimates to respond appropriately to stressful events. This, in turn, should increase the stress response, that is, increased cortisol emission and further brain damage. More inactivity and less persistence should be further consequences.

13.2.10 In a Second Phase of the Mood Disorder, Chronic Stress Should Cause ACC Damage and a More Severe Form of Depression Characterized by Executive Control and Volitional Deficits as Well as Blunting of Emotions

If, within a second phase of the response to chronic stress, not only the HC and PFC but also the ACC are damaged, then a more severe form of clinical depression should arise. Given the suggested central functions of the ACC for action control (see hypotheses 13.1.7 and 13.1.8), namely, detecting the calling conditions of executive and volitional processes and initiating processes located within the PFC, severe deficits in goal-based action control should occur. Conditions for initiating executive and volitional processes should be detected and called less often in this second phase of the depressive disorder. Consequently, in the case of nonroutine actions that require executive control and in the case of goal pursuit in danger that requires volitional control, less and less successful action execution should be seen. Mainly, highly overlearned actions that do not rely on executive and volitional processes should be carried out. This description fits with a very severe form of depression that is characterized by a very high degree of inactivity (up to the level of stupor).

Davidson et al. (2002) suggested that an ACC subtype of depression exists that is characterized by a lack of “will to change.” Within the theory suggested here, this lack of will to change should result from a strongly reduced ability of the damaged ACC to detect actual or anticipated failures in goal pursuit. If no failure is perceived, then no executive processes can be called, and consequently much fewer nonroutine actions will be executed.

Furthermore, blunting of affect in severe forms of depression (American Psychiatric Association 2000) should also be due to ACC dysfunction (Davidson et al. 2002). Discrepancies between the expected and real action outcomes should not be processed properly anymore, and therefore the resulting emotions (see section 13.1.9) should be much less intense.

13.2.11 Open Questions

The neurocognitive theory of depression developed up to now leaves many important questions without answers and is surely underspecified in many respects. For instance, the role of neuromodulators (e.g., serotonin or noradrenaline) in normal action control and the changes of these modulators in depression (see, e.g., Thase et al. 2002) and after recovery (e.g., by antidepressive medication) were not addressed here. The suggested capability of antidepressive medications, via the neuromodulatory changes they produce, to influence the repair of damaged neural structures (see, e.g., Thase et al. 2002; McEwen 2002) may be of some interest here. Furthermore, the standard diagnostic distinction between a less severe and more chronic form of depression, called “dysthymia,” and a more severe form of “major depressive disorder” (American Psychiatric Association 2000) has not been explicitly addressed here. A more detailed
explanation of some classical symptoms of both forms of clinical depression (e.g., psychomotor slowing or agitation; American Psychiatric Association 2000) is also needed.

Overall, the neurocognitive theory about action control and about its failure in clinical depression outlined here should be viewed as a set of hypotheses that attempt to jink often unconnected research domains (e.g., executive control and goal-based action control) and that attempt to fill the gap between basic experimental psychology and cognitive neuroscience, on the one hand, and empirical clinical research on depression on the other hand. Hopefully, some of these hypotheses will serve as a basis for future experimental research that will provide new insights into the neurocognitive processes and representations mediating goal-based action control and clinical depression.

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Notes

1. In case of "covert actions" such as thinking and other inference processes, covert "motor" representations are assumed to be part of the goal-action episode.

2. This dynamic, competition-based part of action control theory is missing in Grafman's SEC framework and may deliver a useful supplement.

3. The idea of a "list of goals" (Duncan 1986) controlling the current action means in the framework advocated here that these goals are represented at a lower level and linked to one higher-level goal that won the competition against other goals.

4. See also Brown, Bullock, and Grossberg 2004 for an overview about evidence and specific computational suggestions about the role of BSG in saccade control.

5. Volitional processes should only be called when the danger of replacing the current goal exists and not when a new episode containing the same goal with a different action attempts to take control.

6. As stated in section 13.1.3, each combination of the same goal with a different action builds an individual episode.

7. The distinction between the state analysis and goal realization mode has some similarity to Kuhl's (1984, 1994) distinction between "action and state orientation." However, in contrast to Kuhl's (1994) theoretically complex distinction, a much simpler definition of goal realization versus state analysis is intended, namely, that the organism either pursues one goal or analyses the results of goal pursuit with respect to the overall goal structure.

8. However, in the Kasch et al. (2002) study, increased avoidance behavior for depressive patients did not—in contrast to increased approach actions—predict the course of depression.

References


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**Plate 1**

A. Activation of posterior parietal area 39 in subjects experiencing a mismatch between the movements they perform and the visual reafference of their movements. B. Amplitude of parietal activation for different degrees of mismatch. 0°, no mismatch; 25°, 50°, increasing mismatch; Other, complete mismatch. Subjects see the effect of movements performed by another agent. Squares: normal subjects. Note the progressive increase in parietal activation. Diamonds: schizophrenic patients. Note the poor correlation between activation and degree of mismatch. rCBF, regional cerebral blood flow. Data from Farrer et al. 2004.
Plate 2
(opposite top) Functional magnetic resonance imaging study of auditory oddball target detection in 28 patients with schizophrenia and 28 healthy controls. Colored regions denote clusters of voxels in which activation was greater during processing of target stimuli than novel stimuli (cluster significance p < 0.05 corrected for multiple comparisons). The patients exhibit less activation than controls in the motivated attention system, especially in limbic/paralimbic areas (amygdala, ventral striatum, rostral anterior cingulate) despite a normal level of activity in sensorimotor cortex.

Plate 3
(opposite middle) Activations in subjects performing spontaneous actions using two fingers of the right hand. There is significantly greater activation of bilateral prefrontal, premotor supplementary motor area, and left motor cortices. The figure shows statistical parametric maps thresholded for display purposes at p < 0.05. Data reported in Hunter et al. 2003.

Plate 4
(opposite bottom) Changes in Sisyphus' brain over time on the two-finger version of the behavioral randomness task (Hunter et al. 2004). Over successive scan sessions, generation of random motor sequences elicits relatively greater activation in left prefrontal cortex (PFC), supplementary motor area (SMA), and motor cortex (left) and greater deactivation in right PFC foci (right). The figure shows statistical parametric maps thresholded for display purposes at p < 0.05.

Plate 5
(below) Notional response space for subject exhibiting stereotypical responses (blue line) and another exhibiting a wider range of potential responses (in red). A narrow Gaussian distribution implies a lack of variety in responses to a given environment. Such behavior will be more predictable. The subject who varies his or her behavior within that environment is, accordingly, less predictable.
Plate 6
Key brain regions involved in volition and depression. (A) Dorsolateral prefrontal cortex shown in blue. (B) Anterior cingulate cortex shown in yellow.

Plate 8
Regional cerebral blood flow maps from three independent positron-emission tomography studies activating an almost identical location within the midventrolateral frontal cortex. (a) Spatial span (adapted from Owen et al. 1996b), (b) digit span (adapted from Owen et al. 2000), and (c) spatial span (adapted from Owen et al. 1999). Right hemisphere only is shown.

Plate 7
Schematic drawing of the lateral surface of the macaque brain (top) and the human brain (bottom), to indicate the location of the ventrolateral frontal cortex (Areas 45, 47, 12). Adapted from Petrides and Pandya 1994. sp, sulcus principalis; ifs, inferior frontal sulcus; mfs, middle frontal sulcus; sfs, superior frontal sulcus.

Plate 9
Event-related functional magnetic resonance imaging blood-oxygenation-level-dependent signal changes when volunteers are asked to either look at (left) or remember (right) a complex visual pattern. Signal change differs significantly in the midventrolateral frontal cortex. Right hemisphere only is shown. Adapted from Dove et al. 2001.
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