Depression and Motivation

The Influence of Dysphoria and Task Characteristics on Cardiovascular Measures of Motivational Intensity

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Depression is one of the most frequent mental disorders with a strong impact on people’s daily lives. Besides a persistent negative mood, depressed individuals often experience a loss of interest or pleasure and are characterized by anhedonia. Therefore, it is not surprising that the term motivational deficit is used in association with depression. Moreover, aspects of motivation are thought to play a role when it comes to other distinctive features of depression. Instigated by the notion of motivational deficits in depression and by recent evidence for the influence of momentary moods on motivational intensity, the present research aims at investigating more closely depressed individuals’ motivation.

Specifically, the present research is concerned with the intensity aspect of motivation in terms of mobilization of energy resources for coping with mental demands. The theoretical framework is built by Brehm’s motivational intensity theory and the mood-behavior-model (MBM) by Gendolla. Motivational intensity theory predicts the intensity of effort that people will invest in dependence on task characteristics and task demand. The MBM describes how moods can influence behavior-related judgments and, by this means, interact with task characteristics in the energy mobilization process. Moreover, the present research takes advantage of Wright’s conceptualization of effort intensity in terms of individuals’ cardiovascular response.

In a series of four experiments, we compared university students who scored low versus high on self-report depression scales and who were therefore referred to as nondysphoric versus dysphoric. After the assessment of cardiovascular baseline activity, participants worked either on a memory task or on a concentration task. Studies 1 and 2 confronted participants with the respective mental task without providing a fixed performance standard, whereas studies 3 and 4 presented participants with either an easy or a difficult version of the mental task. We hypothesized that dysphoria would result in high effort mobilization for tasks with easy performance standards or when no standard was provided. In contrast, we expected that dysphoria would result in disengagement for tasks with difficult performance standards. In accordance with prior research on motivational intensity theory, participants’ cardiovascular reactivity—especially their systolic blood pressure reactivity—to the mental tasks was considered as the central dependent variable referring to effort intensity.

Confirming the main hypotheses, students with high depression scores showed higher systolic reactivity to the tasks without performance standards as well as to the tasks that involved an easy standard compared to students with low depression scores. Likewise in corroboration of our hypotheses, dysphoric participants showed lower systolic
reactivity to the tasks with a difficult standard than nondysphoric participants. Moreover, results demonstrated that students with high depression scores were in a more negative mood. However, participants’ evaluations of the tasks just before performing them showed only partly the expected pattern of higher subjective demand in dysphoria.

On the basis of well established theories, the present research questions the view that dysphoria and depression are invariably associated with a motivational deficit in terms of effort intensity. Rather, it depends on task characteristics and task demand if dysphoria leads to lower or even higher effort mobilization. With respect to application, our series of experiments presents possibilities how to elicit high or low task engagement in dysphoric individuals. Awaiting corroboration of the described effects in a clinical population, this line of research may have implications for research in clinical depression and other psychopathologies characterized by negative affectivity.
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THEORETICAL PART
1. Introduction

Depression is by far one of the most prevalent mental disorders that can dramatically affect our quality of life. Recent data from surveys in the United States and in Europe report a lifetime prevalence of about 16% and 14%, respectively, for an episode of major depressive disorder (MDD; ESEMeD/MHEDEA 2000 Investigators, 2004; Kessler, 2002; Kessler et al., 2003). Characteristic for major depression is its time of onset in early adulthood and its frequent comorbidity with anxiety disorders. About 60% of lifetime MDD patients also show some sort of anxiety disorder, which often precedes depression (Kessler et al., 2003). The prevalence rates for women are generally twice as high as those for men. According to the World Health Organization (2001), depression is considered as the leading cause of “years of life lived with disability” and the fourth leading cause of “disability-adjusted life years” as a measure for the global burden of disease (estimates for 2000). For example, patients with MDD report being unable to perform their normal daily activities on 35 days per year on average (Kessler et al., 2003). Because of this, efforts are undertaken to investigate and understand the etiology of depression, its phenomenology, its course, as well as the effects of different treatments. Moreover, researchers also underline that subthreshold forms of depression (e.g., minor depression or recurrent brief depression), which do not fulfill the number or duration of symptoms for a MDD, are of particular importance because they have strong predictive power for the further development (e.g., Angst & Merikangas, 1997; Judd, Akiskal, & Paulus, 1997).

Besides other important aspects, as for instance cognitive or emotional distinctive features in depression, an obvious deficit of depressed individuals refers to their lack of motivation. There has been research investigating for example behavioral approach and avoidance motivation, initiation of action, and responsiveness to rewards (Fowles, 1994; Heckhausen, 1991; Henriques & Davidson, 2000; Hertel, 2000; Layne, Merry, Christian, & Ginn, 1982). Even if it has been proposed by some researchers that the lowered motivation in depression is a form of adaptation that prevents the organism from pursuing a fruitless or dangerous action (Nesse, 2000), motivational deficits in depression are predominantly seen as hindering and unwelcome. Moreover, to some extent motivational factors have been shown to play a role in cognitive deficits of depressed individuals (e.g., Abramson, Alloy, & Rosoff, 1981).

On the other hand, based on one of the most prevalent symptoms of depression, namely negative affect, and on research concerning mood influences on motivational intensity (Brehm & Self, 1989; Gendolla, 2000), the assumption that depression is not necessarily associated with motivational deficits cannot be denied. The present series of experiments therefore aims to investigate effects of dysphoria—as a mild form of depres-
sion mainly characterized by negative affect (Kendall, Hollon, Beck, Hammen, & Ingram, 1987)—on one specific aspect of motivation, namely motivational intensity, that is, the magnitude of effort expenditure at a certain point in time. This research program dealing with the phenomenology of depression is chiefly based on two theories: motivational intensity theory by Jack Brehm (Brehm & Self, 1989) and the mood-behavior-model (MBM) by Guido Gendolla (Gendolla, 2000). Moreover, it relies on Rex Wright’s elaboration of the construct of effort mobilization—that is, the mobilization of energy resources to carry out instrumental behavior—in terms of cardiovascular response (R. A. Wright, 1996). In the following parts, we present the theoretical background about depression and dysphoria with a focus on motivation, about motivational intensity theory and the MBM, as well as about cardiovascular measures. Afterwards, we describe the methodology and results of four experiments. Finally, we integrate the findings of these studies and discuss them with respect to the main research question about motivational issues in depression or dysphoria and related topics.
2. Depression and Motivation

2.1 Definitions, Classification, and Theories of Depression

2.1.1 Definitions

We begin the theoretical part with some relevant definitions. The concept of human motivation can be described by several basic dimensions. Generally, one distinguishes four such dimensions, namely the initiation, the direction, the intensity, and the persistence of behavior (Geen, 1995; Vallerand & Thill, 1993). This means that human motivation can vary on those dimensions across individuals but also across situations: People may differ in the behavioral choices they make, the vigor or intensity of their actions, and the tenacity or persistence with which they pursue their goals. As we present in greater detail in the following chapters, the empirical part of the present research concentrates on the intensity aspect of motivation as described in motivational intensity theory (Brehm & Self, 1989).

Furthermore, a brief definition of the terms affect, emotion, mood, and depression is warranted. Following a common definition, we use affect as the higher-order term for subjective experiences with positive or negative valence that comprises moods and emotions (Schwarz & Clore, 1996; Scott & Ingram, 1998). Emotions are regarded as short-lived, object-related periods involving specific autonomic adjustments that prepare the organism for action (Cacioppo, Klein, Berntson, & Hatfield, 1993; Frijda, 1993; Levenson, Ekman, & Friesen, 1990). By contrast, moods are regarded as long-lasting states that are not object-related. They can be experienced without conscience of their origin and they may be the residual of an emotion (Frijda, 1993; Schwarz & Clore, 1996). Moreover, moods do not have a direct motivational function and do not directly implicate autonomic responses related to energy mobilization (Gendolla, 2000). In general, depression is regarded as a disorder that refers rather to mood than to emotion (Scott & Ingram, 1998). However, some researchers describe depression in emotion terms, for instance as a combination of sadness and disgust (see Power, 1999).

Following the sections on classification and theoretical approaches of depression, we present theories and research that deal with the relation of depression to all dimensions of human motivation. As the theoretical distinction of motivation from other concepts, especially cognition and emotion, does not always hold up in real human behavior, these sections partly touch on cognitive aspects of depression, too.

2.1.2 Classification of Depression and Dysphoria

In this section, we briefly delineate the different forms and categories of mood disorders, together with the terminology we use in the present research when we refer to
depression and dysphoria. Among the mood disorders, one distinguishes depressive ("unipolar") from bipolar disorders, which means disorders characterized only by depressive episodes versus those also involving manic or mixed episodes. The fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* by the American Psychiatric Association (DSM-IV; APA, 1994; and DSM-IV-TR; APA, 2000) distinguishes furthermore among the depressive disorders the most known major depressive disorder (MDD) from dysthymic disorder and from depressive disorders not otherwise specified. MDD is characterized by at least five symptoms over a period of at least 2 weeks, amongst them depressed mood or loss of interest or pleasure. Other symptoms of a major depressive episode can be changes in appetite or weight, sleep, and psychomotor activity; decreased energy; feelings of worthlessness or guilt; difficulties in thinking, concentration, or decision making; or recurrent thoughts of death or suicidal ideation, plans, or attempts. MDD may take the form of an isolated episode or of recurrent episodes.

In contrast, dysthymic disorder is considered to persist for at least 2 years during which depressed mood and other symptoms (not meeting the criteria for a major depressive episode) are present at least half of the time. Finally, the category of depressive disorders not otherwise specified includes, amongst other things, minor depressive disorder (characterized by fewer symptoms than MDD for a period of at least 2 weeks), recurrent brief depressive disorder (brief depressive episodes occurring at least once a month over 1 year), and premenstrual dysphoric disorder (associated with most of the menstrual cycles over 1 year). Large, community-based surveys underscore that depressive symptoms are stable in the sense that they persist over time, even if people change from one diagnostic subtype to another (Angst & Merikangas, 1997; Judd et al., 1997). These studies underline furthermore that not classified, subthreshold forms of depression as well as minor depression and recurrent brief depression are not only important from the point of view of their prevalence rates but also as strong predictors for a major depressive episode.

The term *dysphoria* does not correspond to a special diagnostic class of the DSM-IV. Rather, it is variably used in the clinical-psychopathological literature. On the one hand, dysphoria is described as an unhappy, tense, and irritated mood that also involves a component of anger and aggression (Berner, Musalek, & Walter, 1987; Musalek, Griengl, Hobl, Sachs, & Zoghliami, 2000; Starcevic, 2007). In this sense, dysphoria is even considered as a third dimension apart from depressed and elated mood. On the other hand, the term dysphoria is often used to refer to nonclinical, “analogue” samples that have elevated scores on depression scales. This use, which goes mainly back to an article by Kendall et al. (1987), is the prevalent use in recent scientific articles investigating various aspects of depression. In the empirical part of this thesis, we are using the term dysphoria in this latter sense to denominate the nonclinical participants in our
studies who reported elevated scores on depression scales. Likewise, in the theoretical part, where we report findings about depression and dysphoria, we use these terms in the same way. This means that the term depression is used for research with clinical samples and the term dysphoria for research with nonclinical samples.

### 2.1.3 Depression as a Categorical or Dimensional Variable?

Directly related to the use of the term dysphoria is a controversial issue with important impact on the present research program, namely the question whether depression and dysphoria are considered as categorical or dimensional constructs. The answer to this question strongly determines how and with which samples depression research is conducted. Exponents of the categorical perspective—which is also the perspective of the DSM-IV—argue that clinical depression, as for instance MDD, is marked by qualitatively distinct features (e.g., somatic symptoms) compared to subclinically depressed mood. According to this perspective, depression research can only reveal valid results if it compares groups of patients with healthy controls (e.g., Gotlib, 1984; Santor & Coyne, 2001).

By contrast, exponents of the dimensional approach refer to the continuum of normal and abnormal processes with “healthy” personality at the one end and psychopathology at the other end (e.g., A. M. Ruscio & Ruscio, 2002; J. Ruscio & Ruscio, 2000; Watson, Gamez, & Simms, 2005). Following this perspective, research with community or student samples (i.e., analogue samples) can reveal results as valid and interesting as research with clinical samples. Moreover, some researchers argue that especially student samples provide several advantages compared to clinical samples: the typical age of depression onset in young adulthood, the homogeneity of a student sample, the presence of relatively manifold environmental stressors, and the relative absence of other (psychopathic) pathologies in student samples (Vredenburg, Flett, & Krames, 1993).

In 1995, the *Journal of Personality and Social Psychology* published a discussion of the state of the art in depression research. This debate mainly emphasized the advantages of clinical samples (e.g., their better external validity). For assessment, multiple measures and especially structured interviews are regarded as more conclusive than self-report measures. When self-report measures are to be used, it is recommended to assess depression twice in order to insure that the scores remain stable and to exclude cases of comorbid anxiety (Kendall & Flannery-Schroeder, 1995; Tennen, Hall, & Affleck, 1995a, 1995b). Concerning the issue of comorbid anxiety, it is argued that this represents a problem only in cases when the dependent variable reacts to this confounding variable but not to depression. It is not regarded as problematic when the confounding variable acts not at all or in the same way as depression (Weary, Edwards, & Jacobson, 1995).
Recently, with the advancements in statistical modeling (i.e., the taxometric approach), researchers largely agree that the dimensional perspective is better supported by the existing empirical evidence than the categorical view—at least on the manifest measurement level (Hankin, Fraley, Lahey, & Waldman, 2005; J. Ruscio & Ruscio, 2000; Solomon, Haaga, & Arnow, 2001). Beach and Amir (2003) qualify this conclusion and note that the continuity hypothesis applies mainly for distress-related aspects but not for somatic symptoms of depression. Most researchers favoring the dimensional perspective argue for the use of the full range of depressive manifestation when investigating analogue or clinical samples (MacCallum, Zhang, Preacher, & Rucker, 2002; A. M. Ruscio & Ruscio, 2002). They argue that splitting the full rage of depression scores in two or more groups by using the median, quartiles, or meaningful cut-off scores has the disadvantage of losing information and statistical power. Nevertheless, some of the authors acknowledge that the comparison of groups is still the prevailing procedure and is appropriate in certain cases.

The present research program is guided by the assumption that psychopathology in general and depression in particular are better understood by the dimensional approach. Moreover, we think that research with analogue, subclinical samples can give important insights in the psychological mechanisms of depression. In order to underscore that we use student samples for this research and that our focus is on negative affect, we refer to the quasi-experimental groups as “dysphoric” versus “nondysphoric”, as mentioned above. Despite the recommendation to investigate the full range of depression scores, we favor the extreme-groups approach, mainly for practical reasons in testing the theoretical model, which we describe in detail in chapter 4.

### 2.1.4 Further Epidemiological Data

To complement the information given in the introduction, we briefly present in this section some further epidemiological data about depression in general. We have already mentioned that individuals suffering from depression encounter serious difficulties with respect to daily demands either at home or at work. In a large community-based survey in the United States, people who had had a depressive episode over the last year reported that they had been unable to pursue their daily activities on 35.2 days on average (Kessler et al., 2003). Moreover, it is important to note that depression is a recurrent disorder. After a first depressive episode, only 30% of the patients remain largely symptom free. The great majority, however, experience residual symptoms, relapse of symptoms, or a chronic course. Because of this, complete symptom remission as well as prevention of relapse and recurrence are major challenges in the treatment of depression (Bondolfi, 2004). Another serious problem in depression is increased mortality in general and from suicide in particular. Estimates for risk of suicide differ with respect to its com-
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putation. Recent reviews suggest that an appropriate estimate for the lifetime risk of suicide lies between 2.2% and 4.0% for affective disorder outpatients and inpatients, respectively, and 8.6% for suicidal inpatients, compared to 0.5% for the general population (Bostwick & Pankratz, 2000).

Furthermore, it is important to note that depression is often accompanied by other disorders, especially substance use disorder (24.0%), impulse control disorder (30.0%), and anxiety disorders (59.2%). Typically, comorbid anxiety disorders have an earlier age of onset, that is, they precede the onset of depression (Kessler et al., 2003). Finally, it is a well documented finding that women have, from puberty on, prevalence rates for depression that are about twice as high as those for men. None of the various potential biological, social, and psychological factors alone has proven to be the single cause for this gender difference. However, there seems to be an interplay between the fact that women encounter more stressful experiences in their lives and the fact that women tend to respond to these experiences in a more depression-facilitating way (Nolen-Hoeksema, 1987, 2001; Nolen-Hoeksema, Larson, & Grayson, 1999).

2.1.5 Theories of Depression

There are various psychological and biological approaches that deal with the development and maintenance of depression and that imply different treatments. Recent research acts on the assumption of an interplay between psychological, social, and biological factors, with different factors being more or less involved in different forms of mood disorders.

Amongst the psychological approaches, the psychoanalytical theories of depression do not receive much attention as they are not well corroborated. These psychoanalytical approaches emphasize that a depressed person’s self-esteem is dependent on other individuals and that the loss of a loved person or object leads to ambivalent feelings and—in case of unsuccessful mourning work—to self-contempt and depression (Freud, 1917). In contrast, behavioral theories like those dealing with loss of reinforcement (e.g., Ferster, 1973; Lewinsohn, 1974) focus on personal and environmental factors leading to the experience and expectation of a low rate of positive reinforcement. These factors comprise personality dispositions involving negative interpretations about the self, few social skills that could give rise to positive reinforcement, as well as an environment that offers few positive reinforcements. A low rate of positive reinforcement, in turn, is considered to favor the development and maintenance of depression. Similar to the behavioral approaches, interpersonal theories emphasize risk factors like deficient social skills, interpersonal inhibition including aversive interaction, and excessive interpersonal dependency (Joiner, 2002). Consequently, such at-risk individuals do not have a
supportive social network and satisfying marital and parental relationships. This lack of social support is considered to reduce the resources for coping with negative life events.

Amongst the psychological approaches, the cognitive and the attributional theories of depression have received by far the most attention and confirmation. As a common assumption, those approaches postulate a cognitive vulnerability for depression due to negative cognitive styles that promote negative interpretations of life events. More precisely, Beck (1967, 1976, 1987) emphasizes cognitive biases and the negative cognitive triad consisting of a negative view of the self, the world, and the future. He reasons that, in a given situation, negative cognitive schemata containing dysfunctional attitudes are activated and lead to false inferences and thinking errors. Those negative cognitions are thought to play an important role in the development and maintenance of depression and are the starting point for cognitive therapy. Similarly, the differential activation hypothesis (Teasdale, 1988) describes the vicious cycle of negative cognitive patterns, but underlines furthermore that such negative cognitions activated during a depressive episode are particularly diagnostic for the further course.

The helplessness-hopelessness theories of depression were originally based on learning experiments with animals showing that the experience of uncontrollable aversive events leads to helplessness and passiveness even in following controllable situations (Seligman, 1974). A reformulation of the original theory emphasizes the depressive attributional style that relates aversive events to internal, stable, and global causes (Abramson, Seligman, & Teasdale, 1978). The most recent version underlines furthermore a hopeless person’s conviction that aversive outcomes will occur, whereas desired outcomes will not occur, and that it is impossible to change this situation (Abramson, Metalsky, & Alloy, 1989; see also Abramson et al., 2002).

It is beyond controversy that depression has a somatic component. Amongst the biological approaches, especially genetic, neurochemical, neuroendocrinological, as well as chronobiological factors are thought to play a role. On the basis of family studies, the genetic component particularly of severe forms of depression is well documented. This genetic vulnerability is even more important in bipolar disorders than in unipolar depression and involves also a higher susceptibility to environmental stressors (Wallace, Schneider, & McGuffin, 2002). With respect to the neuroendocrinological system, there are two models involving both the hypothalamus and the pituitary gland (see Davison & Neale, 1998). The more prominent explanation emphasizes the increased activity of the hypothalamus-pituitary-adrenal axis in depression leading to high levels of cortisol. The other explanation focuses on the relation between (subclinical) thyroid hypofunction and depression. However, the most important biological approaches are based on findings about the effects of certain antidepressant drugs (see Davison & Neale, 1998). The monoaminergic hypothesis postulates that depressive symptoms are associated with
reductions in monoamine neurotransmission, particularly serotonin and noradrenalin. This hypothesis has been reformulated with a focus on postsynaptic receptors. It states that the low concentration of monoaminergic neurotransmitters leads to a high density of postsynaptic receptors and to changes in their responsiveness. This reformulation better accounts for the commonly observed delay in the effects of antidepressant drugs. Moreover, recent research on the neurobiology of depression focuses on the involvement of the hippocampus, the frontal cortex, and the mesolimbic dopamine system (Nestler & Carlezon, 2006). Finally, there are also chronobiological factors involved in depression. Among those factors are alterations in the circadian rhythm and the sleep-awake rhythm as well as the typical seasonal rhythm with higher onset rates in spring and in autumn-winter (Thase, Jindal, & Howland, 2002).

2.2 Phenomenology of Depression

2.2.1 Negative Affectivity

After this introductory information about depression, we deal in the following sections with several distinctive features concerning the phenomenology of depression that are related to our research question. Besides the other symptoms listed in the DSM-IV (American Psychiatric Association, 1994), a particularly prominent characteristic of depression and dysphoria is the general "negative affectivity", which is also termed "negative emotionality" or "neuroticism". This personality construct is phenomenologically similar to MDD and positively correlated with its affective, cognitive, and motivational symptoms but not its vegetative symptoms. Moreover, negative affectivity is considered to be a predisposition and precursor of MDD and dysphoria (Klein, Durbin, Shankman, & Santiago, 2002; Teasdale, 1988). Ingram and Siegle (2002) also emphasize that "negative affect is a variable that is critical to the definition of depression and is also thought to play a significant role in determining the psychological and social function of depressed people" (p. 96).

Moreover, as a rather broad construct, negative affectivity is phenomenologically underlying most other psychopathologies as well. Especially the anxiety disorders are characterized by negative affect. On the basis of the high comorbidity of depression and anxiety (chapter 2.1.4), Watson and colleagues proposed in their tripartite model a hierarchical structure with negative affectivity as a higher order factor that is common to depression and anxiety (especially generalized anxiety disorder) and specific factors that differentiate between the two: anhedonia and low positive affectivity in depression versus anxious hyperarousal in the anxiety disorders (Clark & Watson, 1991; Mineka, Watson, & Clark, 1998). This hypothesis, which also aimed to explain the high overlap between the two classes of disorders, proved to be empirically sound in clinical and nonclinical
samples (Brown, Chorpita, & Barlow, 1998; Watson et al., 2005; Weinstock & Whisman, 2006).

### 2.2.2 Cognitive and Neuropsychological Impairments

Another characteristic of depression pertains to the fact that depressed individuals often show impairments or deficits with respect to cognitive and neuropsychological functions. Research on those deficits has mainly focused on memory functions but also on executive functions and attention. In a meta-analysis of the memory literature, Burt, Zembar, and Niederehe (1995) find a strong association between depression and impaired memory functions, depending on the type of task (e.g., recall vs. recognition) and stage of information processing but also on patient characteristics and age. Moreover, in a quantitative review of the clinical literature, Christensen, Griffiths, MacKinnon, and Jacomb (1997) report cumulative evidence for depressed individuals’ impaired performance on a large spectrum of psychometric tests. This review suggests that not only memory functions but almost all aspects of cognitive functions are affected, especially those that involve a speed component. There is also evidence that such neuropsychological impairments recover to a certain degree, but residual deficits are likely to persist even after remission of depression (Austin, Mitchell, & Goodwin, 2001; Elliott, 1998).

With respect to memory functions, it seems that controlled and effortful, respectively, processes are more impaired than automatic processes (Hartlage, Alloy, Vazquez, & Dykman, 1993; Hertel, 1998; Jermann, Van der Linden, Adam, Ceschi, & Perroud, 2005). Furthermore, depressed individuals tend to have an overgeneral memory, particularly for episodic, autobiographical material (Healy & Williams, 1999). In the domain of attention, it has been shown that depressed or dysphoric individuals have problems with thought suppression and concentration on task-relevant aspects (Elliott, 1998; Gotlib, Roberts, & Gilboa, 1996; Hertel & Gerstle, 2003). Likewise, working memory functioning and especially its central executive component are adversely affected in depression (Van der Linden, 2007). Finally, there is evidence for the impairment of executive functions in general. In particular, depressives show performance deficits when specific problem solving strategies are required. Executive functions, in turn, are closely associated with prefrontal cortical areas—that is, those areas where depressed individuals show abnormalities and less activity (Rogers et al., 2004).

More detailed information about cognitive deficits in depression and dysphoria can be found in the reviews of the literature by Gotlib and colleagues (Gotlib et al., 1996) and Williams and colleagues (Williams, Watts, MacLeod, & Mathews, 1997). These reviews also point to the fact that similar results are shown for clinical and subclinical depression but often also for experimentally manipulated mood (Ellis & Moore, 1999).
Those findings underline the important role that moods—sometimes in interaction with dispositional affectivity—may play in the cognitive domain. Moreover, in their neuropsychological theory of positive affect, Ashby, Isen, and Turken (1999) postulate that positive affect is linked to increased dopamine levels in certain brain areas. This increase, in turn, is considered to improve performance in various cognitive domains, such as memory, problem solving, and interpersonal tasks. In a review of the literature of studies with induced and pathological affect, Ashby et al. report evidence for this assumption.

Taken together, besides other rather cognitive explications, some findings also point to affective and motivational factors being involved in cognitive impairments or deficits in depression. We discuss these factors (e.g., deficient motivation to attention control and self-regulation or deficient motivation to action initiation and effort mobilization) in more detail in chapter 2.3.

### 2.2.3 Memory, Attention, and Judgment Biases

Besides general cognitive deficits, there exists a vast body of literature with regard to cognitive biases in depression, often in comparison with anxiety (see Gotlib et al., 1996; Mineka & Gilboa, 1998; Scott & Ingram, 1998; Segal, 1996; Williams et al., 1997, for reviews). Especially the mood-congruent memory bias for negative material in depression has been studied extensively. It is a robust finding that depressed individuals better recall and recognize negative stimuli (e.g., words or faces) compared to neutral material and compared to nondepressed controls (e.g., Gilboa & Gotlib, 1997; Gotlib, Kasch et al., 2004). However, this bias is more pronounced in explicit and autobiographical memory tasks than in implicit tasks and it is more pronounced in clinical than in subclinical individuals. Whereas clinically depressed people generally show a negative bias and nondepressed people generally show a positive bias, dysphoric (i.e., subclinical) individuals are “even-handed” in the sense that they do not preferentially remember either negative or positive material (e.g., Gilboa, Roberts, & Gotlib, 1997).

In the domain of attention, anxious individuals are shown to selectively pay attention to threatening stimuli. In contrast to this and in contrast to the robust memory bias in depression, evidence for an attention bias of depressed individuals with respect to negative (especially sad) material is equivocal (see Gotlib et al., 1996; Mineka & Gilboa, 1998; Scott & Ingram, 1998; Williams et al., 1997, for reviews). Some recent studies suggest, however, that clinically depressed and dysphoric individuals do have a biased attention for negative words and faces (e.g., Gotlib, Kasch et al., 2004; Gotlib, Krasnoperova, Neubauer Yue, & Joormann, 2004; Koster, De Raedt, Goeleven, Franck, & Crombez, 2005). This bias seems to result not only from maintained attention to negative material but also from impaired disengagement from negative material, which occurs at later stages of the attention process (Koster et al., 2005).
Of particular importance for the present research program is evidence showing that depression and dysphoria can bias evaluations and judgments. Reviews of the literature by Scott and Ingram (1998), Williams et al. (1997), Mineka and Gilboa (1998), and MacLeod (1999) report findings showing biased evaluative judgments with respect to varying material in depression and induced negative affect. Depressed and negative affect individuals give more negative and pessimistic judgments concerning, for instance, pleasure of imagined activities, satisfaction with their own performance, standards they set for themselves, or statements they make about themselves. Furthermore, negatively biased attributions and higher subjective probability for negative outcomes have been observed as well as more pessimistic and negative judgments of contingency. This means that depressed individuals do not tend to overestimate contingency and control like non-depressed individuals. These findings have led to the notion that depressed individuals are in fact more realistic than nondepressed individuals. Like in memory biases, dysphoric individuals seem to be even-handed in the sense that they are less characterized by the presence of a negative self-evaluation bias but rather characterized by the absence of the “normal” positive self-evaluation bias compared to nondysphorics (e.g., Gilboa et al., 1997).

Findings like those reported above fit well in prominent cognitive theories of depression, notably the negative cognitive triad posited by Beck (1967) but also the attributional approach with negatively biased internal, global, and stable attributions (Abramson et al., 1989; Abramson et al., 1978). However, these biased evaluations and judgments are not limited to the domain of depression. Similar results for the influence of negative affect on risk estimates in anxious individuals have been shown for instance by Gasper and Clore (1998).

2.2.4 Emotion Context Insensitivity

Related to cognitive biases in depression is a recent hypothesis about “emotion context insensitivity”, that is, the inability to express and remember positive and especially negative emotions. Rottenberg and colleagues (e.g., Rottenberg, Gross, & Gotlib, 2005; Rottenberg, Joormann, Brozovich, & Gotlib, 2005) hypothesize that depressed individuals are insensitive to the emotional context of a situation and do not respond to emotional stimuli in the same way as nondepressed individuals. Research demonstrates that depression indeed goes along with less sensitivity not only for positive material but also for negative stimuli (Rottenberg, Gross et al., 2005). Moreover, the authors show that this emotion context insensitivity is a predictor for worse depression outcomes 1 year later (Rottenberg, Joormann et al., 2005).
In the previous sections, we have presented definitions, classifications, and theories of depression as well as affective and cognitive distinctive features of depressive phenomenology. The review of those distinctive features demonstrates that there exists a large body of research investigating cognition in depression. As we outline in the following chapter 2.3, motivation may play a role in these cognitive impairments and deficits. Given this role of motivational states, we aim to investigate in our research program whether and under which conditions motivational deficits with respect to effort intensity exist in situations where dysphoric and nondysphoric individuals have to mobilize resources in order to cope with cognitive demands.

2.3 Motivation and Cognition in Depression

As previously stated, cognition may be influenced by affective and motivational factors, and to some extent motivation may play a role in the manifestation of neuropsychological and cognitive deficits in depression. Even if motivation does not account for all sorts of cognitive deficiencies, its role is not negligible. For example, a series of experiments highlights the motivational implications of negative feedback. Elliott and colleagues (Elliott, Sahakian, Herrod, Robbins, & Paykel, 1997; Elliott, Sahakian, McKay, & Herrod, 1996) report that clinically depressed individuals who failed on a first task are more likely to show impairments on a subsequent task. In contrast, control groups of other patients and healthy individuals are less affected by negative feedback.

A controversial issue refers to the question whether cognitive impairments in depression or negative mood mainly emerge for difficult in comparison to easy tasks. Inferring effort expenditure from performance outcomes, some authors ascribe cognitive impairments to differences in the expenditure of cognitive effort (Ellis, Thomas, & Rodriguez, 1984; see also Williams et al., 1997). If tasks requiring more effort are indeed more affected in depression than tasks requiring less effort, this can be an indicator for a motivational or self-regulation deficit. However, other authors underline that the hypothesis stating that effortful processes are generally more affected in depression than automatic processes still awaits corroboration (Austin et al., 2001; Christensen et al., 1997; Elliott, 1998).

There are very few studies that directly address motivational deficits in depression. Moreover, some of this research is not conducted under the label of motivation. However, it can easily be interpreted in this sense, especially when it deals with action initiation or self-regulation issues. In the following sections, we present empirical findings that illustrate motivational influences on cognition and performance outcomes in depression and dysphoria.
2.3.1 Control Expectancies

To begin with, there is some evidence for the fact that diminished control expectancies of depressed individuals are influenced by motivational factors. On the basis of hopelessness theories of depression, Abramson et al. (1981) investigated depressed individuals’ generation of complex hypotheses and generalized expectancies in terms of less perceived control about outcomes. The authors report that dysphoric undergraduates underestimate their control possibilities when underlying complex hypotheses have to be self-generated but not when those hypotheses are generated for them. Thus, the authors conclude that a motivational and not a cognitive-associative deficit is at the origin of pessimistic generalized control expectancies.

Ford and Neale (1985) come to a similar conclusion on the basis of an experiment with undergraduates who underwent a learned helplessness induction. In this study, helpless participants did not underestimate control. On the contrary, they had higher and more accurate control expectancies. The authors confirm the conclusion by Abramson et al. but additionally offer an alternative interpretation, namely that a helplessness induction might lead to higher motivation in terms of reactance as described by Wortman and Brehm (1975). According to this theory, reactance is the first reaction to the restriction of freedom, followed by resignation when efforts to reinstall control fail.

2.3.2 Initiation and Attention Control

The initiation of action as one of the basic dimensions of human motivation is a prominent example of motivational deficits in depression. Most practitioners agree that depressed individuals have problems in commencing and initiating actions. Likewise, research supports the notion that deficient action initiation may account to some extent for various impairments in depression (Gotlib et al., 1996). In a series of studies, Hertel and colleagues (e.g., Hertel, 2000; Hertel & Gerstle, 2003) show to which extent initiation is deficient in depression and how this might be overcome by special task features. In their cognitive-initiative account, the authors conclude that memory impairments in depression are mainly caused by a lack of spontaneously developed initiative, and that diminished cognitive capacity does not play an important role. This conclusion is based on findings showing that depressed individuals have problems in focusing their attention on task-relevant features and that performance can be augmented by hints that help with attention control. Furthermore, a lack of initiative is more prominent in intentional remembering and forgetting and less important in implicit memory (Hertel, 2000; Hertel & Gerstle, 2003). This fits in the previously mentioned observation that in depression explicit memory tasks are more affected than implicit tasks and strengthens the conclusions by Hertel.
Nitschke, Heller, Etienne, and Miller (2004) confirm Hertel’s findings with respect to electrocortical activity. The authors demonstrate associations between stronger activity in right frontal areas and better memory performance when depressed participants actually listen to a sad receipt but not when they prepare to listen to that receipt. In this way, Nitschke et al. show that their findings are consistent with left frontal hypoactivity in depression because initiative is regarded as an approach-related action associated with left frontal activity. Furthermore, the authors demonstrate that the contrast between depressives’ worse memory performance in general and their better memory performance for negative material in particular depends on different stages of the information processing. Finally, Austin et al. (2001) make an important point with respect to initiation deficits in test situations by stating that most experimental tasks and clinical tests are highly structured. Their stimulative nature may thus mask deficits in participants’ motivation, self-monitoring, or planning, which are otherwise part of the clinical phenomenology of depression.

### 2.3.3 Rumination and Intrusion

Another important aspect of depressive phenomenology that accounts for performance deficits is depressed individuals’ proneness to ruminate—that is, to continuously focus on themselves and on their negative feelings. Several studies have demonstrated that rumination about past or current concerns or tasks impairs problem-solving abilities and performance. A series of studies by Lyubomirsky and Nolen-Hoeksema (1995) demonstrates that dysphoria together with a ruminative self-focus causes participants to provide more negative interpretations of hypothetical situations, more pessimistic causal attributions, lowered expectation of positive events, and less effective solutions to interpersonal problems. However, when dysphoric participants are distracted, their responses do not differ from those of nondysphoric participants, either ruminating or distracting.

In a similar series of experiments that focused on current problems of their participants, Lyubomirsky, Tucker, Caldwell, and Berg (1999) have found that dysphoric participants’ mood becomes more depressed after a ruminative task, whereas their mood becomes less depressed after a distractive task. Compared to dysphoric participants in the distraction condition and compared to nondysphoric participants, dysphoric participants in the rumination condition rate their problems as more severe and less solvable and express a lower solution implementation probability, even if their confidence in the solution does not differ from the other three groups. Importantly, nondysphorics’ mood and problem ratings are not affected by the rumination task, indicating that rumination is not harmful per se but particularly in combination with depressed mood.
In addition to impaired problem solving abilities, Hertel (1998) shows the impact of self-relevant tasks (i.e., tasks that have consequences for one's self-definition, self-esteem, or personal interest, see Gendolla, 2004) on memory performance in dysphoria. Results demonstrate that dysphoric individuals have worse memory performance if a self-relevant task is presented between learning and recall or if there is uncontrolled free time. By contrast, dysphoric individuals do not have worse memory performance if a self- or task-irrelevant activity distracts them from ruminating between learning and recall. Similarly, rumination is considered to be partly responsible for depressives’ overgeneral autobiographical memory and executive dysfunction because rumination, compared to distraction, occupies more cognitive resources (Healy & Williams, 1999; Watkins & Brown, 2002).

From the perspective of the degenerated-intention hypothesis, Kuhl and Helle (1986) put the emphasis on the perseveration of unrealistic intentional states in depression. The authors demonstrate that depressive participants who are experimentally induced to an unrealized intention show worse performance in a subsequent task and more intrusions of the unrealized intention, compared to participants in the condition without unrealized intention. Especially women are prone to respond to stressful life experiences with rumination—a behavior that negatively reinforces the course of depression and that is considered to play a role in women’s higher prevalence rates for depression (Nolen-Hoeksema, 1987; Nolen-Hoeksema et al., 1999).

### 2.3.4 Self- and Affect Regulation

The findings about depressive rumination directly lead to the domain of self- and affect regulation because rumination is predominantly seen in the context of self-regulation problems. There exist several theoretical conceptions that relate to depression and dysphoria and that underline the vicious cycle of depressed mood, self-focused rumination, and negative thinking. To begin with, Pyszczynski and Greenberg (e.g., Greenberg, Pyszczynski, Burling, & Tibbs, 1992; Pyszczynski & Greenberg, 1987) emphasize in their self-focusing theory that, after a negative event, depressed and dysphoric individuals are caught in a state of constant self-focus, unable to disengage. Thus, these people do not display the common self-serving bias of nondepressed individuals who tend to respond with internal attributions for successes and external attributions for failures. In a way similar to the studies cited in the previous sections (Hertel, 1998; Lyubomirsky & Nolen-Hoeksema, 1995; Lyubomirsky et al., 1999), Pyszczynski, Hamilton, Herring, and Greenberg (1989) corroborate their hypotheses by showing that the negative memory bias for self-referent events can be eliminated by distracting dysphoric participants from self-focusing. The authors conclude that distraction from self-focusing deactivates depressives’ negative self-schemas.
Similar to Pyszczynski and Greenberg’s assumptions, Carver and Scheier (1999) postulate in their self-regulation model that depressed individuals continue to be committed to a goal in spite of low expectations for attainment. Also in the domain of self-regulation theories, self-discrepancy theory by Higgins (1998) postulates individual differences in self-regulation focus: Individuals with a dispositional or situational “promotion focus” are guided by their ideal self and the attainment of their own hopes and wishes. In contrast, individuals with a “prevention focus” are guided by their ought self, the fulfillment of their obligations, and the avoidance of losses. Indeed, in a sample of undergraduates, a correlational study by Miller and Markman (2007) shows that hopelessness depression symptoms correlate positively with a prevention and negatively with a promotion focus. Moreover, in this study, promotion focus mediates the association of hopelessness depression symptoms with poorer academic motivation and poorer short-term performance.

Depressed individuals’ impaired self-regulation abilities are particularly prominent in negative affect regulation. Besides evidence for mood-congruency effects on memory, which have been described above, mood-incongruent recall for the purpose of negative mood regulation is a common phenomenon. Depressed individuals, however, have been shown to have problems regulating their negative mood by means of mood-incongruent (i.e., positive) autobiographical recall. A study by Josephson, Singer, and Salovey (1996) demonstrates that dysphoric participants are less likely to repair their current mood after a negative mood induction by retrieving positive memories. Nondysphoric participants, in contrast, successfully recall positive memories to counteract their current negative mood. Similar findings have been reported for individuals with low self-esteem (S. M. Smith & Petty, 1995) and self-focused rumination (McFarland & Buehler, 1998). Recently, Joormann and Siemer (2004) have replicated the findings for dysphoric versus nondysphoric undergraduates. They demonstrate furthermore that, in contrast to distraction, ruminative self-focus is an important factor that is responsible for deficient negative mood regulation in depression.

In the preceding sections, we have focused on selected phenomena in depression and dysphoria that originally refer to cognitive issues but can be inspected from a motivational point of view as well. This inspection demonstrates that sometimes motivation and cognition are not easily separated and that depressed or dysphoric individuals differ from control participants in important aspects. In the following chapters 2.4 and 2.5, in contrast, we directly address motivational issues and findings with reference to depression and dysphoria. These findings ultimately lead us to our research question concerning the presence or absence of motivational deficits in terms of effort intensity in dysphoria.
2.4 Motivational Systems in Depression

2.4.1 Basic Motivational Orientations

A prominent line of research supported by a vast body of empirical evidence is the postulate of different motivational systems with neurobiological and neurophysiological correlates. Gray (1982) proposed three motivational systems with different cortical and subcortical bases that determine behavior and emotion. The behavioral approach system (BAS)—also termed behavioral facilitation system or behavioral activation system by other researchers—reacts to signs of reward with the goal of approaching. In contrast, the behavioral inhibition system (BIS)—also termed withdrawal system—reacts to signs of punishment with the goal of avoiding. Furthermore, Gray postulated a fight and flight system responding to direct aversive stimulation. On this basis, Fowles (1994) formulated a motivational theory of psychopathology and connected the motivational systems and their underlying physiological mechanisms to anxiety, depression, psychopathy, schizophrenia, and substance abuse. With respect to depression, Fowles postulated an interruption of the appetitive motivation (i.e., a weak BAS) together with a disinhibition of the BIS.

In analogy to the BIS/BAS approach, Strauman (2002) uses the terms put forward by Higgins (1998, see chapter 2.3.4) to refer to depression as a self-regulation dysfunction characterized by a hypoactivation of the promotion system in combination with a hyperactivation of the prevention system. Strauman furthermore underlines the role of the self and successive self-regulation failures in the promotion system for understanding the vulnerability for depression. Following the long-standing notion referring to loss of reinforcer effectiveness (e.g., Costello, 1972), Strauman describes depression as a loss of the motivation to respond to rewarding stimuli, which manifests itself on the neural, cognitive, and behavioral level.

2.4.2 Evidence From Self-Report Data

In the last 2 decades, a huge body of studies using self-report, behavioral, and neurophysiological measures has supported the differential activation of the BIS and BAS in depression. With respect to self-report measures—most often using the BIS/BAS scales by Carver and White (1994)—evidence is converging on the fact that depressed individuals consistently report higher BIS and lower BAS activation (but see also Johnson, Turner, & Iwata, 2003). Especially reduced approach motivation seems to be a robust marker of depression that persists after remission of depressive symptoms and has predictive value for depression status some months later (Kasch, Rottenberg, Arnow, & Gotlib, 2002). In contrast, increased inhibition motivation does not seem to persist after remission (Pinto-Meza et al., 2006).
In an earlier series of studies on the basis of “expectancy x value” theories of motivation, Layne and colleagues conclude that depressed individuals are characterized by a motivational deficit (Layne et al., 1982; Layne, Walters, & Merry, 1983). In their experiments that involved a forced-choice paradigm the authors demonstrate that dysphoric undergraduates attribute lower value to rewards and have slightly lower expectancy for obtaining rewards. Following the multiplicative association of expectancy and value, this results in reduced reward (i.e., approach) motivation in dysphoria compared to nondysphoric individuals. Results with respect to punishment revealed that dysphoric individuals tend to evaluate punishment as less aversive and partly have higher expectancy of being punished. Together, this results in lower or equal punishment (i.e., avoidance) motivation in dysphoria compared to nondysphoric individuals. The authors conclude that depressed individuals do not care about any outcome, either positive or negative, a fact that is described by the term anhedonia.

Research on the basis of the Pleasant Event Schedule (MacPhillamy & Lewinsohn, 1982) points in the same direction. This scale involves the evaluation of a list of events referring to “obtained pleasure”, “activity level”, and “potential for reinforcement”. In a study with undergraduate participants who are induced into a positive, neutral, or negative mood, Carson and Adams (1980) report less positive ratings after the negative mood induction and more positive ratings after the positive mood induction. However, participants’ depression scores do not have an effect on the ratings on the Pleasant Event Schedule. MacPhillamy and Lewinsohn (1974) compare a group of clinically depressed patients to a group of other psychiatric patients and to a control group. The depressed group shows more negative ratings on all three subscales in comparison with both other groups, suggesting that anhedonia and insensitivity to rewarding activities are specific for depression. These two studies imply that depression and negative mood are associated with less obtained and anticipated pleasure concerning a variety of activities and thus with less approach motivation.

### 2.4.3 Evidence From Behavioral Data

The notion that the two basic motivational systems are differently pronounced in depression is supported on the behavioral level, too. A recent study investigated approach and avoidance goals and plans (Dickson & MacLeod, 2004). Two other studies dealt with responsiveness to reward and punishment (Henriques & Davidson, 2000). Participants in the study by Dickson and MacLeod had to generate approach and avoidance goals and plans. Results reveal that adolescents with high depression scores generate fewer approach goals and plans and more avoidance plans (but not more avoidance goals). Furthermore, depressed participants are less specific when describing their goals and plans, compared to the control group.
Similarly, in two experiments, Henriques and colleagues (Henriques & Davidson, 2000; Henriques, Glowacki, & Davidson, 1994) demonstrate that subclinical as well as clinical depression leads to reduced responsiveness to rewards. The authors analyzed participants’ response bias in a signal detection task under three within-subjects conditions: neutral, reward, and punishment. Although depressed and nondepressed individuals’ performance does not differ with respect to the neutral condition, depressed participants fail to adopt a more liberal response bias in the reward condition, in contrast to nondepressed individuals. Provided that task instructions are equally well understood in both groups, this means that depressed individuals are not motivated to earn money by simply changing their responses to the reward trials. Results under punishment conditions are ambiguous and not consistent across the two studies. Further analyses suggest that concurrent anxiety might account for this inconsistency insofar as anxious individuals are sensitive to punishment signs, whereas purely depressed individuals are not motivated by monetary punishment. In summary, Henriques and colleagues successfully presented behavioral indices for depressives’ deficits in approach motivation that are in line with neurophysiological findings indicating a hypoactivation of the brain reward system.

2.4.4 Evidence From Neurophysiological Data

Differences in cerebral activation between depressed and nondepressed individuals but also between people with transient negative versus positive affect have been shown by numerous studies. Reviewing the existent evidence, Davidson and colleagues (Davidson & Henriques, 2000; Davidson, Pizzagalli, Nitschke, & Putnam, 2002) summarize depressed individuals’ abnormalities in the prefrontal cortex, the anterior cingulate cortex, the hippocampus, and the amygdale. Especially differences in the prefrontal cortical areas, which are associated with the representation and maintenance of goals as well as with approach and withdrawal behavior, corroborate findings with respect to the two hypothesized motivational systems: Mainly on the basis of electroencephalography, but also on cerebral metabolism and blood flow, a substantial number of studies confirms that depression is related to a relative hypoactivation in left frontal areas, compared to the activation pattern found under normal conditions (Davidson & Henriques, 2000; Davidson, Pizzagalli, Nitschke et al., 2002; see also Gotlib, Ranganath, & Rosenfeld, 1998; Harmon-Jones et al., 2002; Tomarken & Keener, 1998; Tremblay, Naranjo, Cardenas, Herrmann, & Busto, 2002). The findings are also in line with the model put forward by Davidson (1992) that relates right frontal activation to negatively valenced, withdrawal-related emotion and left frontal activation to positively valenced, approach-related emotion.
This evidence is complemented by neurobiological studies on subcortical structures that are involved in the dopamine reward circuit and therefore in the regulation of reward-related behavior. Accordingly, symptoms of depression such as impaired motivation and reduced reward responsiveness are associated with alterations in mesolimbic reward areas (Nestler & Carlezon, 2006, see also chapter 2.1.5). In summary, data from self-report, behavioral, and neurophysiological studies strongly suggest that—as the term anhedonia implies—depression is indeed characterized by reduced approach behavior and partially also by increased avoidance behavior.

### 2.5 Standards and Perfectionism in Depression

To conclude the chapter on motivational issues in depression, an interesting question pertains to the setting of (performance) standards. This question is related especially to the persistence and intensity dimensions of motivation and effort mobilization. As clinical observations often report a pronounced perfectionism in depression, the assumption that depressed individuals try even harder and thus show the opposite of a motivational deficit suggests itself.

On the basis of research about high performance goals (e.g., Ahrens, 1987) and low self-efficacy expectations (e.g., Kanfer & Zeiss, 1983) in depression and dysphoria, Cervone, Kopp, Schaumann, and Scott (1994) hypothesize that depression and negative affect would be characterized by a negative discrepancy between self-set standards and corresponding expectations to meet them. Experiments with subclinical and clinical samples involving self-report measures reveal that depressed and dysphoric participants hold higher performance standards than nondepressed and nondysphoric participants. Self-efficacy expectations, in contrast, do not differ between the groups. Taken together, these findings demonstrate a negative discrepancy between standards and expectations in depression (Cervone et al., 1994; Scott & Cervone, 2002; Tillema, Cervone, & Scott, 2001).

Moreover, one of the studies (Tillema et al., 2001) confirms that the results are due to an affect-as-information mechanism (Schwarz & Clore, 1983, 1996). In their study, Tillema et al. applied the “mood discounting” procedure introduced by Schwarz and Clore. This means that momentary mood is made salient to the participant, so that its influence on an outcome measure is diminished. Cervone, Scott, Tillema, and colleagues conclude furthermore that an undervaluation of potential outcomes leads to anticipated dissatisfaction. This, in turn, is considered to be responsible for higher performance standards in depression and negative mood (Cervone et al., 1994; Scott & Cervone, 2002). Insofar, their considerations are consistent with other findings regarding undervaluation of rewards, which we have reported in chapter 2.4.2.
Dealing with standard setting and perfectionism has also led to the question whether these variables have trait or even predictive character in depression. In this context, Flett, Hewitt, Blankstein, and Gray (1998) underline the importance of perfectionistic cognitions, which explain variance in depression beyond trait perfectionism and which even have predictive value for depression, anxiety, and negative affectivity in general. The authors also emphasize that perfectionistic thinking may be interpreted as a preoccupation with one’s feebleness including rumination about not meeting one’s perfectionistic standards. On the other hand, Carver and colleagues (Carver, 1998; Carver & Ganellen, 1983) report findings that corroborate neither the connection of depression with high standards nor the predictive power of standards for high depression scores at a later point in time. According to these studies, it is rather the tendency to overgeneralize that represents a strong correlate and predictor of depression and a cognitive vulnerability factor (see cognitive theories of depression in chapter 2.1.5).

In the preceding first two chapters of the theoretical part, we have presented various findings concerning depression, motivation, and its relation. The great majority of the reviewed research literature suggests that depressed and dysphoric individuals have impairments or deficits with respect to motivation and self-regulation. However, as foreshadowed in the introduction, a series of experiments by Gendolla and colleagues (e.g., Gendolla & Krüskens, 2001a, 2002c) investigating the integration of the MBM (Gendolla, 2000) and motivational intensity theory (Brehm & Self, 1989) has led us to hypothesize that depression and dysphoria are not necessarily associated with motivational deficits in terms of effort mobilization. Therefore, in the following two chapters of the theoretical part, we describe motivational intensity theory, the concept of effort mobilization, and the use of cardiovascular measures as an operationalization for the mobilization of energy resources. Subsequently, we present the MBM and its related research in more detail.
3. Effort Mobilization: Cardiovascular Measures and Motivational Intensity

The main dependent variable of our research program is effort intensity, which can be defined as the mobilization of energy resources at a certain point in time in order to carry out behavior (Gendolla & Wright, in press). As we operationalize effort intensity by a person’s cardiovascular response (see R. A. Wright, 1996), we present in this chapter some background about the cardiovascular system, its control by the autonomic nervous system, as well as the resulting cardiovascular parameters. Then, we report research on effort mobilization in the framework of motivational intensity theory (Brehm & Self, 1989). In order to underline the importance of cardiovascular parameters for the present research, we begin the chapter with a section on cardiovascular activity in depression.

3.1 Depression and Cardiovascular Activity

To date, a certain number of studies have already addressed the issue of cardiovascular activity and reactivity in depression. This research was mainly instigated by findings suggesting a relation between subclinical and clinical depression on the one hand and the development and worsening of cardiovascular diseases, as for instance coronary heart disease, on the other hand (e.g., Frasure-Smith & Lespérance, 2005b; Rugulies, 2002). The prevailing view states that a dysregulation of autonomic cardiovascular balance in depression (i.e., stronger sympathetic and weaker parasympathetic activity) leads to stronger cardiovascular reactivity and ultimately to higher vulnerability for cardiovascular diseases (Carney, Freeland, & Veith, 2005; Kibler & Ma, 2004; Light, Kothandapani, & Allen, 1998). Thus, strong cardiovascular reactivity is regarded as a mediator in patients suffering from cardiovascular disease and depression. But the evidence for a causal relationship between depression and higher vulnerability for cardiovascular diseases is ambiguous and third factors are thought to play a causal role as well (Carney et al., 2005; Frasure-Smith & Lespérance, 2005a). Suls and Bunde (2005) emphasize that it is rather negative affectivity in general—as a component of depression but also of anxiety, hostility, and other psychopathological states—that plays a mediating role in the development of coronary heart disease (see also Jorgensen, Johnson, Kolodziej, & Schreer, 1996; Kubzansky, Davidson, & Rozanski, 2005).

Despite the evidence for a relation between depression and cardiovascular disease, the findings concerning cardiovascular response to behavioral challenges in depression are mixed. A study with subclinically depressed women by Light et al. (1998) found evidence for depressives’ enhanced cardiovascular activity under baseline conditions and enhanced cardiovascular reactivity under physically demanding challenges (i.e., stand-
ing) and mentally demanding challenges (i.e., speech task). The authors report higher systolic blood pressure (SBP) and diastolic blood pressure (DBP), shorter pre-ejection period (PEP), and lower heart rate variability (HRV) for depressed participants in all experimental periods. Furthermore, they found higher cardiac output, heart rate (HR), and plasma noradrenalin, but only for the speech task. These results suggest that depressed individuals are characterized by a generally enhanced cardiovascular activity, even under resting conditions. A meta-analysis on depression and cardiovascular reactivity by Kibler and Ma (2004), however, revealed only weak SBP and DBP effects as well as moderate HR effects over the 11 studies included. The authors conclude that depression may be more strongly associated with cardiac activation than with vascular activation.

Recently, Carroll, Phillips, Hunt, and Der (2007) reported data from a large community-based sample that—against the initial hypotheses—show weak negative associations between depression and SBP and HR reactivity. However, from our point of view, most of the cited studies failed to acknowledge the differential impact of task characteristics as specified for instance in motivational intensity theory and its derivations (Brehm & Self, 1989; R. A. Wright & Kirby, 2001). For example, in the Carroll et al. study, cardiovascular measures from two different difficulty levels were merged to one index of cardiovascular reactivity. We think that the mixed findings concerning cardiovascular reactivity to behavioral challenges might be partly due to the fact that potential effects were neutralized because different task characteristics had not been considered.

In this context, we should also note that there is recent evidence for differences in HRV and respiratory sinus arrhythmia (RSA) between depressed and nondepressed individuals. Nevertheless, this evidence is mixed. From a theoretical point of view, a high RSA is thought to be adaptive because an active and rapid withdrawal of the “vagal brake” is important for an organism to face rapidly changing environmental demands (Porges, 1995, cited in Rottenberg, Salomon, Gross, & Gotlib, 2005). The level and reactivity of RSA are thus important parameters of human self-regulation, and a high RSA is thought to be a buffer against psychopathology. Indeed, in most of the studies, depression has been shown to be associated with low HRV (Carney et al., 2005; Frasure-Smith & Lespérance, 2005a) and less vagal withdrawal in behavioral challenges (Rottenberg, Clift, Bolden, & Salomon, 2007). But the reverse pattern has been found as well (Rottenberg, Wilhelm, Gross, & Gotlib, 2002).

### 3.2 The Cardiovascular System

In the following sections, we deal in more detail with the cardiovascular system, its parameters, and its autonomic control. The main function of the cardiovascular system is to maintain blood pressure, to ensure the blood flow through the tissues of the
body, and to supply oxygen to the various body organs. The cardiovascular system does so by reacting to the rapidly changing metabolic requirements and at the same time by trying to maintain a state of metabolic homoeostasis. In case of physical demand, blood flow in the skeletal muscles—but also in the heart and the skin—increases, whereas blood flow in other organs diminishes. Only the blood flow in the brain is relatively unaffected by these rapid changes that occur in association with various behavioral and psychological states (see Papillo & Shapiro, 1990).

The cardiovascular system is functionally divided into two divisions. The pulmonary circulation pumps oxygen-deficient blood in the lungs and returns oxygen-rich blood to the heart. In contrast, the systemic circulation pumps oxygen-rich blood to the body organs and returns oxygen-deficient blood to the heart. The heart itself consists of a left and a right pump, each consisting of two chambers—the atrium and the ventricle. The vasculature serves for the distribution, exchange, and collection of the pumped blood (see Papillo & Shapiro, 1990; Stern, Ray, & Quigley, 2001).

The sinoatrial node located in the wall of the right atrium is responsible for the initiation of the rhythmic contraction of the heart and is the origin of electrophysiological depolarization. A heart cycle normally begins with the depolarization of the atria. Subsequently, the electrical activity spreads over the atrioventricular node and causes the ventricles to depolarize, while the atria relax (i.e., repolarize). Finally, all chambers are repolarized during rest period. Consequently, the main states of a heart cycle comprise the atrial systole during which blood flows from the atria to the ventricles, the ventricular systole during which ventricular pressure increases until the pressure becomes greater than in the aorta and causes the valves to open, and, finally, the ventricular diastole during which aortic and pulmonary valves are closed and the atrioventricular valves are not opened yet (see Brownley, Hurwitz, & Schneiderman, 2000).

### 3.3 Sympathetic and Parasympathetic Influences

The functioning of the cardiovascular system is influenced and hierarchically controlled by various factors, including local control mechanisms on the cellular, metabolic, hormonal, and receptor level but also regulation by autonomic mechanisms and by subcortical and cortical areas. Especially the sympathetic and parasympathetic branches of the autonomic nervous system exert a control function on the heart and the vasculature. The sympathetic nervous system (SNS) is always active in homeostasis but becomes more active during periods of stress. In general, SNS activation increases the activity of the target organs. The parasympathetic nervous system (PNS), in contrast, supports feeding, energy storage, and reproduction and exerts inhibiting influences. For example, the “normal” HR of approximately 105-110 beats per minute (bpm) under resting conditions is restricted by vagal (i.e., parasympathetic) influences to approximately 70 bpm.
The PNS also exerts inhibiting influence on the SNS. Because of the opposing effects of the SNS and PNS, their joint activation allows for highly graded reciprocal regulation of organic functioning (see Brownley et al., 2000; Lovallo, 2005; Stern et al., 2001).

With respect to the cardiovascular system, sympathetic activation generally causes activity: SNS activation can lead to HR increases but only to the extent that its influence is not masked (i.e., inhibited) by PNS activation. Moreover, SNS activation directly enhances the force of the contractility of the heart, that is, the force with which blood is ejected from the (left) ventricle into the aorta. Such increases in HR and heart contractility are mediated by beta-adrenergic sympathetic activation. Furthermore, the SNS influences the constriction of some blood vessels and the dilation of other blood vessels, depending on which adrenergic postsynaptic receptor is involved (see Brownley et al., 2000; Levick, 2003; Lovallo, 2005).

### 3.4 Cardiovascular Parameters

The measurement of various parameters of the cardiovascular system can give an insight in the momentary state of the organism and allows inferring the activity of the autonomic nervous system. The following list of cardiac and vascular parameters is not exhaustive but presents parameters that are theoretically or empirically important for the present research (Levick, 2003; Papillo & Shapiro, 1990):

- Total peripheral resistance (TPR) refers to the level of resistance to the blood flow caused by all blood vessels in the systemic circulation;
- Diastolic blood pressure (DBP) is the minimum pressure in the aorta during the diastolic phase, recorded in millimeters of mercury (mmHg; normally approximately 80 mmHg);
- Systolic blood pressure (SBP) is the maximum pressure in the aorta during ventricular ejection, recorded in mmHg (normally approximately 120 mmHg);
- Mean arterial pressure (MAP) is the mean pressure in the vasculature throughout a cardiac cycle, recorded in mmHg and approximated as $\text{MAP} = \frac{1}{3} (\text{SBP} - \text{DBP}) + \text{DBP}$;
- Pre-ejection period (PEP) corresponds to the time beginning with the onset of ventricular depolarization and ending with the opening of the semilunar valves;
- Heart rate (HR) is typically measured in beats per minute (bpm; at rest between 60 and 80 bpm);
- And, finally, respiratory sinus arrhythmia (RSA) refers to the variability in HR associated with breathing, especially the high frequency band of the HRV spectrum (between 0.15 and 0.50 Hz).

In accordance with the sympathetic and parasympathetic influences on the heart and the vasculature, these parameters are differently influenced by the activation of the
one or the other branch of the autonomic nervous system. As sympathetic activation can have vasodilative as well as vasoconstrictive effects in different vascular beds in dependence on the receptor type involved, TPR is only unsystematically linked to sympathetic discharge. Consequently, DBP, which is mainly determined by TPR, is not systematically influenced by SNS activity. Hence it can—but does not necessarily have to—mirror SNS activity. In sum, DBP is not regarded as a good indicator of sympathetic activation. By contrast, SBP is only marginally influenced by TPR but chiefly determined by myocardial contractility (i.e., the force with which the heart contracts). As myocardial contractility is mainly influenced by innervations of the sympathetic branch of the autonomic nervous system, SNS activation directly enhances contractile force. Therefore, SBP can be considered as an indicator of sympathetic discharge. An even more direct indicator of contractile force is PEP, which is highly correlated with more direct measures of ventricular contractility and especially sensitive to beta-adrenergic influences on the heart. \(^1\) As already outlined above, HR is expected to respond to sympathetic activation only to the extent that its influence is not masked by parasympathetic activity. RSA, in contrast, is widely accepted as an indicator of vagal (i.e., parasympathetic) autonomic control because projections of the vagal nerve contribute to RSA level and RSA fluctuation (see Levick, 2003; Papillo & Shapiro, 1990).

As it is obvious from this brief description, the interplay of the different cardiac and vascular parameters is complex, and autonomic activity does not always influence all of them in the same manner. Furthermore, other non-autonomic factors may influence cardiovascular activity as well. Therefore, it has to be pointed out that such other factors as well as the specific situational context have to be considered when conclusions about specific physical or psychological states of the organism, as for instance arousal or effort, are drawn from cardiovascular parameters (see R. A. Wright, 1996).

### 3.5 Active Coping and Effort Mobilization

In psychophysiology, the cardiovascular system has received much attention as a system that responds to many psychological and behavioral processes and therefore may be an indicator of different states of the organism. Psychological states usually investigated in this domain are attentional functions, arousal, stress, the social context, and so forth (Papillo & Shapiro, 1990). An important contribution to the understanding of individuals’ response to behavioral demands has been made by the work of Paul Obrist (1976, 1981). He based his analyses on the observation that, under rest as well as under performance conditions, the mobilization of the cardiovascular system is proportional to the demands for blood supply to the organs. Moreover, he put forward the notion of the

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\(^1\) However, PEP may also be influenced by pre- and afterload, that is, ventricular filling and aortic diastolic pressure, respectively (see e.g., Obrist, Light, James, & Strogatz, 1987).
“cardiac-somatic uncoupling”. This means that the cardiovascular system reacts proportionately not only to the demand of a physical task but also to the engagement in a mental task, even though in the latter case metabolic demand is relatively low. Furthermore, Obrist introduced the classification of tasks in dependence on the fact if they call for active versus passive coping. Active coping tasks are situations where the organism anticipates that some action or effort investment will result in successful coping, which means that the individual has control over behavioral outcomes. By contrast, passive coping tasks are situations where any action seems futile and therefore the situation and its consequences have to be undergone without the possibility to change anything by effort investment (see also Light, 1987; Papillo & Shapiro, 1990).

Most important, Obrist and his colleagues (Obrist, 1976, 1981) provide evidence for the assumption that active and passive coping tasks elicit different responses on the cardiovascular level. Active coping involves stronger beta-adrenergic sympathetic stimulation and leads to more pronounced myocardial responses. It resembles the pattern elicited by a flight reaction. Consequently, indicators of beta-adrenergic sympathetic activation, as for instance SBP, HR, and PEP, are more affected in an active coping situation. By contrast, passive coping involves less beta-adrenergic sympathetic stimulation but stronger vascular responses. This means that stronger increases in TPR are present as well as increases in DBP that equal or even exceed those of SBP changes (see also Light, 1987; Obrist et al., 1987; Papillo & Shapiro, 1990).

Evidence for the implications of the active/passive coping concept stems from a body of research investigating task engagement under different performance conditions. This research domain builds on the notion that active coping corresponds to effort mobilization and that sympathetically mediated cardiovascular reactivity is enhanced in response to behavioral challenges—in anticipation (i.e., preparation) of a task as well as during the respective task. Such experiments usually comprise tasks within a social context or not, with or without performance-contingent consequences, and with different levels of performance standards. These studies confirm that beta-adrenergic activity is heightened in active coping, as indicated by increases in SBP, HR, and cardiac output, as well as reductions in TPR (e.g., T. W. Smith, Ruiz, & Uchino, 2000). Sometimes, even DBP increases have been shown to accompany the active coping response pattern (e.g., T. W. Smith, Allred, Morrison, & Carlson, 1989). Moreover, when performance standards are impossible to meet, reduced reactivity of PEP, SBP, and pulse transit time has been demonstrated (e.g., Light & Obrist, 1983).

### 3.6 Motivational Intensity Theory

Even more evidence for sympathetic response to active coping tasks comes from the work by Rex Wright and colleagues. Wright (see R. A. Wright, 1996) has integrated
Obrist’s active coping approach and motivational intensity theory by Jack Brehm (Brehm & Self, 1989). Amongst other things, motivational intensity theory builds on the difficulty law of motivation by Ach (1935) stating that individuals avoid wasting energy and therefore mobilize resources proportionately to perceived task demand (see also Kukla, 1972). In contrast to other approaches postulating a proportional relationship between motivation and factors like success importance, individual needs, or self-involvement, motivational intensity theory distinguishes between the actual intensity of motivation at a given point in time (i.e., momentary effort mobilization) and the so-called potential motivation that is determined by classical variables like individuals’ needs, instrumentality of success, or performance-contingent incentives. According to motivational intensity theory, potential motivation influences task engagement and effort mobilization only indirectly via its interaction with task difficulty.

Another important classification in motivational intensity theory concerns the distinction between tasks with and without fixed performance standards. Under situations of fixed standards, motivational intensity is thought to rise proportionately with increasing task difficulty. This holds true until perceived task difficulty exceeds the level of potential motivation or until a task exceeds the person’s abilities. Under these conditions, motivational intensity is thought to be low, either because success is not possible or because effort mobilization is not justified by the value of success. This means that people disengage and withhold effort under these two conditions. The relation between task difficulty and motivational intensity is depicted in chapter 4.4.2, Figure 3, Panel A. In contrast, when no performance standard is given and people are free to determine their extent of engagement, individuals are thought to strive for the highest justified performance level. Consequently, motivational intensity should directly vary with factors influencing potential motivation, as for instance success importance, until a maximum is reached.

As outlined before, R. A. Wright and colleagues have provided a vast body of evidence supporting the predictions of motivational intensity theory, not only with respect to self-report indices of task engagement but especially with respect to cardiovascular indices of effort mobilization. According to expectations on the basis of the physiological mechanisms described by the active coping approach, R. A. Wright and colleagues find SBP and HR reactivity, and seldom DBP reactivity, to correspond to the theoretical predictions of motivational intensity theory: Under the condition of fixed performance standards, effort mobilization (i.e., cardiovascular reactivity) is low for easy tasks and rises with difficulty; but it is low for extremely difficult tasks due to disengagement (e.g., R. A. Wright, 1984; R. A. Wright, Brehm, Crutcher, Evans, & Jones, 1990). Moreover, there is evidence for the interaction of potential motivation and fixed task difficulty. This means that high instrumentality, high incentive value, or strong needs can lead to high effort investment even if a task is relatively difficult (e.g., Storey, Wright, & Williams, 1996; R.
A. Wright & Gregorich, 1989; R. A. Wright, Shaw, & Jones, 1990). Concerning tasks without fixed performance standards, effort mobilization directly depends on factors influencing potential motivation. This means that the maximum level of justified effort is mobilized (e.g., R. A. Wright, Killebrew, & Pimpalapure, 2002). For an overview over this research program and the empirical evidence see Wright (R. A. Wright, 1996, 1998; R. A. Wright & Kirby, 2001).

### 3.7 The Influence of Subjective Task Difficulty

Among the work of R. A. Wright and colleagues, one specification of the predictions of motivational intensity theory is of particular importance for the present research program. In a series of studies, R. A. Wright and colleagues investigated effort mobilization in samples with low versus high perceived task-relevant ability (see R. A. Wright, 1998, for an overview). The rationale for their predictions is that individuals with high perceived abilities should appraise a given task as easier and therefore mobilize less energy than individuals with low perceived abilities. Consequently, those latter individuals should withhold energy mobilization at a lower level of objective difficulty than high ability individuals because of subjectively too high task demand. However, at a very high difficulty level, ability perception should not make a difference because both groups are supposed to withhold effort. The resulting pattern of predictions corresponds to the one presented for the influence of mood in the following chapter (see chapter 4.4.2, Figure 3, Panel B).

In a series of studies either manipulating perceived ability by means of performance feedback or comparing natural differences in perceived ability, R. A. Wright and colleagues corroborate their theoretical predictions. Specifically, they find that low ability participants show higher SBP (and sometimes HR) reactivity than high ability participants for easy but not for difficult tasks (e.g., R. A. Wright & Dismukes, 1995; R. A. Wright, Wadley, Pharr, & Butler, 1994). Furthermore, cardiovascular reactivity is low for both groups under extremely difficult conditions (e.g., R. A. Wright, Murray, Storey, & Williams, 1997). Finally, a study by Annis, Wright, and Williams (2001) confirms the interaction effect of task difficulty and ability perception for PEP and stroke volume but surprisingly not for SBP.

Building on the ability assumptions, R. A. Wright and colleagues have extended their reasoning to individual differences in trait optimism and to the depletion of performance resources. The authors reason that trait optimism has an influence on ability perception and therefore on resource mobilization. Two experiments confirm the expected crossover interaction pattern, that is, higher SBP response for pessimists at low difficulty levels but for optimists at high difficulty levels (Kirby, Vaga, Penacerrada, & Wright, 2003, cited in R. A. Wright & Franklin, 2004).
A second line of ongoing research assumes that the depletion of performance resources influences ability appraisals in a related but not in a completely different domain. Depletion is experimentally manipulated by asking participants to perform a physical or mental task that is either easy (no depletion) or difficult (depletion). Afterwards, participants work on a second task that either taps into a similar or a different domain. Results confirm that depletion does not influence effort mobilization in a different domain. However, in a similar domain, depleted individuals show higher SBP reactivity in the second task due to lower ability perception (R. A. Wright & Penacerrada, 2002). Moreover, if task difficulty of the second task is manipulated as well, it interacts with task difficulty of the first task: Individuals who first perform a difficult task and who are therefore depleted show higher cardiovascular reactivity if the second task is easy but not if the second task is difficult (R. A. Wright, Martin, & Bland, 2003).

Most relevant for the present research program, Gendolla has integrated the work of Brehm and R. A. Wright with considerations about mood influences on behavior—the mood-behavior-model (MBM; Gendolla, 2000). In a series of experiments that are presented in greater detail in the following chapter 4, Gendolla and colleagues corroborated their assumption that mood influences behavior-related evaluations and judgments, which leads to the same pattern of effort mobilization as has been shown for ability perception (see Richter, Gendolla, & Krüsken, 2006, for an overview). The extensions of motivational intensity theory presented in this section are of great importance for the present research on depression effects because depression is not only characterized by a persistent negative mood but also by lower self-esteem together with lower evaluations of one’s own abilities (e.g., Power, 1999). Therefore, the findings about the influence of manipulated transient moods by Gendolla and colleagues build the basis for our core hypothesis, namely that depression and dysphoria are not necessarily associated with a motivational deficit in resource mobilization but that it depends on task context and task difficulty whether depression leads to low or high effort mobilization.
4. The Mood-Behavior-Model and Related Research

**Mood, Motivation, and Performance: An Integrative Theory, Research, and Applications**

An integrative theory about mood and motivation—the mood-behavior-model (MBM; Gendolla, 2000)—is presented and the results of related studies on mood and motivational intensity are discussed. A series of experiments with implications for motivation in organizational and educational settings assessed motivational intensity as cardiovascular reactivity in the context of task performance. The results support the theoretical predictions and demonstrate that (1) moods do not per se have an effort mobilizing function, (2) moods are pragmatically used as information for demand appraisals, (3) mood congruent demand appraisals determine effort-related cardiovascular response, (4) performance-contingent incentive determines the level of maximally justified effort, (5) effort is linked to achievement. The findings specify and extend other approaches to the role of affect in motivation and have direct implications for clinical psychology, work psychology, and health psychology.

Individuals’ affective experiences are a central variable in the person system mediating between external stimuli and people’s behavioral reactions (Mischel & Shoda, 1995). This applies, of course, also to organizational and educational settings: On the one hand, work settings directly influence the moods and emotions people experience (Kelly & Barsade, 2001). On the other hand, moods and emotions have multiple effects on performance and organizational behavior (see Baron, 1993; Forgas & George, 2001; George & Brief, 1992; Isen & Baron, 1991, for reviews). Furthermore, moods’ motivational effects can play significant roles in psychological problems and the development of physical disease (Gendolla & Brinkmann, 2005; Gendolla & Richter, 2005b).

In the last decade, an abundance of research has identified manifold influences of affective states on motivation and task performance. Although most of this research was not conducted in applied settings, there are numerous insights of direct relevance for work motivation. In an attempt to contribute to this understanding, the present chapter focuses on one special type of affective experience—*mood states*—and on one special aspect of motivation—*motivational intensity*, which corresponds to *effort* mobilization or

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task *engagement*. After presenting an integrative theoretical framework about mood and motivation, we will discuss research on mood and motivational intensity. Finally, we will outline implications for applied issues, such as work motivation, clinical psychology, and health psychology.

### 4.1 Affect and Motivation

Emotions have the motivational function to guide behavior. Specific emotions, such as anger, fear, or interest control goal priorities and involve action tendencies, such as anger-aggression, fear-flight, or interest-exploration. Thus, emotions facilitate a fast and effective adaptation of the organism to the environment (Frijda, 1986; Scherer, 2001; C. A. Smith & Lazarus, 1990). Emotions' motivational function becomes, for instance, visible in their effects on autonomic nervous system activity (see Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000, for a review). Autonomic adjustments as a component of specific emotions reflect the fast and short-lived mobilization of body resources to prepare the organism for action.

The motivational implications of moods are less clear than those of emotions. Moods are defined as relatively long lasting affective states that are experienced without concurrent awareness of their elicitors. That is, moods are "feeling states themselves" (Clore, Schwarz, & Conway, 1994; Frijda, 1993; Wyer, Clore, & Isbell, 1999). Whereas individuals typically know the incidents and stimuli that elicit specific and short-lived emotions (e.g., happy or sad about...), they are usually not aware of the origins of their moods (e.g., feeling good or bad) (e.g., Wilson, Laser, & Stone, 1982). Given that moods have been conceptualized as always-present frames of mind (Morris, 1989), it appears likely that they should also influence motivation and performance. A recent integrative theory about the role of mood in the motivation process aims to explain how this takes place.

### 4.2 The Mood-Behavior-Model (MBM)

According to the mood-behavior-model (MBM; Gendolla, 2000), transient mood states can have highly context dependent, though predictable, effects on behavior (Richter et al., 2006). Unlike specific emotions, moods per se do neither have stable effects on goal priorities nor on the intensity or persistence of behavior—the three aspects of behavior that are determined in the motivation process (Geen, 1995). Moods can, however, influence motivation and behavior through two processes—directive and informational mood impacts. The strength of each process depends on variables that refer to the context in which moods are experienced, as summarized in five basic postulates presented in Figure 1.
Theoretical Part: Mood-Behavior-Model

The Mood-Behavior-Model (MBM)

(1) Moods have—in contrast to emotions—no stable or specific motivational implications or function. Moods are experienced without simultaneous awareness of their causes. Consequently, moods do not urge the organism to act in a specific way toward objects or incidents that elicited the moods.

(2) Moods influence behavior by their informational and directive impacts. The informational impact refers to congruency effects on behavior-related judgments and appraisals. The directive mood impact refers to the pursuit of a hedonic motive, and thus behavioral preferences are affected.

(3) The informational and directive mood impacts can influence behavior independently. Either of these impacts is sufficient to mediate mood states to behavior, but both can occur simultaneously as well. In addition, both mood impacts can be so weak that neither has a significant influence.

(4) The strength of the informational mood impact is a function of the effective informational weight of mood and the extent of mood-primed associations. Mood is particularly diagnostic for evaluative judgments, and mood-primed associations are especially likely to be activated in specific mood states, when moods are residuals of specific emotions.

(5) The strength of the directive mood impact is jointly determined by the strength of the hedonic motive and the magnitude of behaviors’ instrumentality for motive satisfaction. The strength of the hedonic motive is, in turn, determined by mood intensity, mood salience, and situational context. The magnitude of behaviors’ instrumentality for motive satisfaction is determined by the hedonic tone of behavior itself, the hedonic tone of behavioral outcomes, and mood valence.

Figure 1
The five basic postulates of the mood-behavior-model (MBM; Gendolla, 2000)

4.2.1 Directive Mood Impact

The directive mood impact influences the direction of behavior in compliance with a hedonic motive and turns out in preferences for affect regulative behaviors. However, if and how strongly the directive mood impact works depends on context variables: The MBM predicts that the strength of a person’s hedonic motive—that is, the need to feel good—increases with both mood intensity and mood salience and that it decreases with situational circumstances that prohibit pleasant feelings (like visiting a funeral). Only if the hedonic motive is strong, a person will prefer behaviors that are instrumental for hedonic affect regulation in that they promise positive feelings as a result of pleasant
associations of a behavior itself (i.e., intrinsic pleasure) or pleasant consequences of behaviors in terms of hedonic reward (i.e., extrinsic pleasure).

The directive mood impact on behavior is particularly visible in decision-making—a major aspect of human behavior and organizational work. Besides having an influence on judgments of expectancy and value in the decision process (Isen, 2000), moods can also influence behavioral decisions because these are often oriented to their hedonic consequences (e.g., Damasio, 1994; Loewenstein, 1996). Referring to the role of mood valence (i.e., feeling good or bad) in the latter process, the actual state of research is not conclusive. On the one hand, there is evidence for the assumption that negative mood is particularly powerful in instigating affect regulative behaviors in terms of mood repair (e.g., Schaller & Cialdini, 1990). On the other hand, positive mood is a major trigger of affect regulation in terms of mood maintenance (e.g., Wegener & Petty, 1994). This equivocal evidence suggests that mood valence cannot be the critical variable that strengthens a person’s hedonic motive. Rather, the MBM postulates that mood intensity is the crucial variable that strengthens a person’s hedonic motive and thus triggers the interest in mood repair or mood maintenance. Thus, both intense negative and positive moods lead to preferences for behaviors that promise positive affective experiences (e.g., Handley & Lassiter, 2002). Consequently, the MBM predicts that especially people in an intense mood—either negative or positive—who are aware of their current feeling state (e.g., McFarland & Buehler, 1998) are highly interested in hedonic affect regulation in terms of mood repair and mood maintenance when the experience of well-being is not forbidden in the person’s actual context.

An example for the directive mood impact on behavior may be a manager who needs to make some investment decisions at the stock market. If this person is in an intense negative or positive mood, the MBM suggests that he or she will experience a strong hedonic motive and thus prefer stock options that promise fast and high gains and thus positive affective experiences. In a less intense mood, the manager should experience a weaker hedonic motive. Thus, he or she should be less interested in stock market operations that seem to promise fast and high gain and consequently positive affect. Likewise, a personnel manager in an intense good or bad mood who has to choose among applicants for opened positions may orient his decisions on applicants’ charm and the pleasantness of their physical appearance rather than on their mere qualification. However, the MBM also predicts that the strength of this manager’s hedonic motive would be significantly lower if it is clearly inappropriate to feel good in the organizational setting. Under this condition, decisions should be less influenced by concerns of hedonic affect regulation (see Parrott, 1993).
4.2.2 Informational Mood Impact

The informational mood impact influences behavior-related judgments and appraisals and takes effect on the persistence and the intensity of behavior. Informational mood impact refers to the answers people find when they ask themselves implicit questions once they are confronted with a demand. Examples are “Can I cope with this demand?”, “Have I already achieved enough?”, “How much effort do I have to mobilize?”, or “How difficult is the task?”. The MBM posits that moods can influence these appraisals in terms of mood congruency effects (e.g., Erez & Isen, 2002; Kavanagh & Bower, 1985; Lane, Whyte, Terry, & Nevill, 2005; J. C. Wright & Mischel, 1982). The result is that people are more optimistic in a positive mood than in negative mood (e.g., Efklides & Petkaki, 2005).

Imagine a person who comes to work in a bad mood. Imagine further this person has a desk full of work, which he or she needs to complete as soon as possible. According to the MBM, the person’s mood will not have much visible impact on behavioral decisions, like preferences for pleasant activities. Sufficient commitment provided, there are actually not many choice options regarding what to do today. Consequently, the directive mood impact is rather weak. But the MBM posits that the employee’s mood state will have a systematic influence on his or her performance. At least, the mood state will influence work persistence in that it can determine the point in the work process at which the employee will believe that enough is done. According to studies by Martin, Ward, Achee, and Wyer (1993), this point is reached later in a bad mood than in a good mood, because negative mood can signal a lack of approach to the working goal. And the mood state will influence how vigorously the work is done—as will be shown in more detail below. The MBM suggests that these mood effects on persistence and engagement occur because people will use their momentary mood state as information for evaluating if enough has been accomplished or how much engagement is necessary to complete the work.

In more detailed terms, the MBM posits that informational mood impact on persistence and effort mobilization occurs because people use their mood as a piece of information and integrate it with all other available information into an evaluative judgment (e.g., Abele & Gendolla, 1999; Abele & Petzold, 1994). In the present context, evaluative behavior-related judgments are critical. The result is that people who face a challenge judge task difficulty, subjective ability, required effort, and the likelihood of success lower in a negative mood than in a positive mood, resulting in lower subjective demand in a positive mood than in a negative mood (e.g., Gendolla, Abele, & Krüsken, 2001). The MBM also considers that moods may activate associations, like remembering pleasant events in a good mood and unpleasant events in a bad mood. These associations can also contribute to mood congruency effects because people are expected to use...
all available information for making judgments. However, such “mood as priming” effects are only predicted for mood states that are residuals of specific emotions (Niedenthal, Halberstadt, & Setterlund, 1997). But concerning evaluative judgments, like demand appraisals, the effective weight of mood as a piece of diagnostic information is higher than that of remembered events. We agree with Clore et al. (1994), that mood congruency effects on *evaluative judgments* are the most reliable and best replicated finding in affect-cognition research, because the valence of an experienced mood provides direct information for answering an evaluative question.³

It is of note that the MBM differs from other accounts of mood-cognition linkages like, for example, the affect-infusion-model (AIM; Forgas, 1995). The AIM posits that mood is either used as diagnostic information (heuristic processing) or makes memory associations accessible (substantial processing). By contrast, the MBM does not make processing style assumptions and predicts that mood is always more or less informative for evaluative judgments and that it can activate knowledge when mood is the residual of a specific emotion. As we have shown in the studies to be discussed below, the extent of engagement in information processing (processing style, respectively) is an *effect* rather than a *determinant* of informational mood impact. Other differences between the MBM and leading approaches concerning the role of mood in information processing and behavior are discussed elsewhere (Gendolla, 2000).

### 4.3 Mood and Motivational Intensity

According to the MBM, moods can influence motivational intensity through the following process: Moods provide information for demand appraisals. Consequently, the extent of subjective demand is higher in a negative mood than in a positive mood when people are confronted with a challenge. Moods can then systematically influence effort intensity, because subjective demand directly determines engagement as long as it is possible and worthwhile to perform a task (Brehm & Self, 1989). However, given that moods themselves are not conceptualized as motivational states, it is of note that these mood effects should not occur before moods are experienced in a context that directly calls for effort. Thus, moods should only influence the mobilization of effort when people are confronted with a challenge and make the necessary appraisals of its extent of demand. In this context, people can use their moods as information for demand appraisals, which in turn determine the level of engagement. Furthermore, the MBM predicts that moods are only one piece of information people use for appraising task demand. When more information about the extent of demand is provided—for example, in terms

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³ It is of course also possible that moods activate memories for emotional events that in turn elicit mood states. But even in this case, the resulting mood state rather than the memory itself is used as a piece of information for an evaluative judgment.
of a given performance standard—people use and integrate it simultaneously into the final judgment.

The deeper reason for this “pragmatic” use of all available diagnostic information to evaluate the level of subjective demand is the effort conservation principle (Ach, 1935; Hull, 1943; Tolman, 1932). That is, people try to avoid wasting resources. Therefore, they try to mobilize only as much effort as necessary for goal attainment (Brehm & Self, 1989) and try to use all available diagnostic information for demand appraisals.

4.3.1 Measuring Engagement

The psychological literature is full of attempts to measure motivational intensity or effort mobilization. Examples are self-reports, persistence, or achievement. Unfortunately, each of these measures is ambiguous and thus problematic as motivational intensity index (see Gendolla, 2004, for a discussion). A pioneering research program of psychophysiologist Paul Obrist has offered an alternative way to assess motivational intensity in terms of cardiovascular reactivity in the performance context (e.g., Obrist, 1981). Accordingly, the changes in the activity of the heart and the vasculature in the performance context can reliably reflect task engagement. It is of note that these cardiovascular changes are independent of metabolic demand and thus apply to both physical and cognitive tasks. Among the possible indices of cardiovascular arousal, one index—systolic blood pressure (SBP; i.e., the pressure in the vasculature on the peak of a pulse wave)—is especially responsive to the extent of task demand (see Gendolla, 2004; Gendolla & Wright, 2005; Obrist, 1981; R. A. Wright, 1996; R. A. Wright & Franklin, 2004; R. A. Wright & Kirby, 2001, for overviews). Other indices, like diastolic blood pressure (DBP; e.g., Storey et al., 1996) and heart rate (HR; e.g., Gellatly & Meyer, 1992) are less sensitive.4

4.4 Empirical Evidence

A number of experimental studies in our laboratory have investigated the impact of mood states on motivational intensity quantified as cardiovascular response. Specifically, these studies examined engagement under three performance conditions that are

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4 SBP is the pressure in the vasculature on the peak of a pulse wave. It depends on the contractility of the heart muscle, which is systematically related to beta-adrenergic sympathetic discharge to the heart and the flow resistance in the vasculature (Brownley et al., 2000). DBP describes the pressure between two pulse waves. It depends on vascular resistance, which is unsystematically related to sympathetic arousal—sympathetic arousal leads to dilatation in some vascular beds and to vasoconstriction in others. HR is independently affected by both sympathetic and parasympathetic arousal and should only respond to effort mobilization when the actual sympathetic impact is stronger (Berntson, Cacioppo, & Quigley, 1993; Obrist, 1981). Consequently, SBP is the most reliable measure of effort mobilization among these cardiovascular indices. SBP, DBP, and HR can, however, respond simultaneously during task performance (e.g., Bongard & Hodapp, 1997; Gendolla & Richter, 2005a; Lovallo et al., 1985; T. W. Smith et al., 1989).
of direct relevance for work motivation: These are performance settings without clear performance standards, performance settings with a clear performance standard, and settings in which reward is contingent versus non-contingent upon performance.

Typically, an experiment in this program of research consisted of three parts: (1) A habituation period to assess participants’ cardiovascular baseline values, (2) a mood manipulation in which positive versus negative moods were induced, and (3) a task performance period with one of the three performance settings mentioned above. In the performance period, participants worked on a cognitive task. That is, performance was mentally rather than metabolically demanding and thus reflected the mobilization of mental effort (see Hockey, 1997). Cardiovascular indices were assessed during habituation, the mood inductions, and task performance. According to the psychophysiological understanding about the determination of cardiovascular reactivity during task performance, our primary effort-related measure was SBP reactivity relative to the baseline values assessed during habituation (see Obrist, 1981; R. A. Wright, 1996).

4.4.1 Tasks Without Performance Standard

The MBM posits for tasks that are performed without a clear performance standard that people will use their moods as task-relevant information to evaluate the extent of demand and that the magnitude of subjective demand will in turn determine engagement because the demand-effort relationship is proportional (Ach, 1935; Brehm & Self, 1989). Given that moods usually produce congruency effects when people use them as diagnostic information for evaluative judgments like demand appraisals, the predicted result is higher engagement in a negative mood than in a positive mood—as long as the diagnostic value of mood is not called into question.

Gendolla and Krüsken (2002a) tested these predictions in an experiment. After habituation and assessment of cardiovascular baseline values, participants watched depressing versus elating video excerpts to induce positive versus negative moods. After completion of filler questions and a mood manipulation check, participants received instructions for a learning task. The task was to correctly memorize in 5 min as many items of a list of 20 letter series (e.g., Q P T Z) as they could and to write them down afterwards (“do-your-best”). In addition to mood, the diagnostic value of mood as task-relevant information was manipulated. Therefore, half the participants read a short note under the task instructions that provided a cue for the mood manipulation: In the “cue” conditions participants read that previous research would suggest that the video excerpts they had seen may have long lasting effects on people’s feeling states. This hint to the possibility that the moods may have been manipulated was expected to reduce the diagnostic weight participants assigned to their feelings as task-relevant information. Consequently, no significant mood effect on task engagement was anticipated in the cue condi-
tion. The other half of the participants performed in a “no-cue” condition. Here, nothing was mentioned with regard to the previously presented video excerpts. Thus, participants in this condition were expected to use their moods as diagnostic information for their demand appraisals and to mobilize more effort in a negative mood than in a positive mood.

The results were as expected. Mood had no effects on the cardiovascular responses assessed during the mood inductions, although the mood inductions were highly effective according to the manipulation check. However, in all experimental conditions, the cardiovascular values remained on the baseline levels during the film presentations, supporting the MBM notion that moods do not per se mobilize effort. But the reactivity of SBP during performance on the learning task—that is, when mood was experienced in a setting that directly called for effort mobilization—exactly described the predicted pattern. As depicted in Figure 2, SBP increased significantly higher in a negative mood than in a positive mood when no cue for the mood manipulation was provided. In further support of the predictions, this mood effect diminished in the cue-condition.

![Figure 2](image)

**Figure 2**

Achievement effects corresponded to those of systolic reactivity during task performance. As presented in Table 1, participants in the no-cue condition memorized significantly more items in a negative mood than in a positive mood. By contrast, no reliable mood effect emerged in the cue condition. Thus, achievement effects resembled those of effort intensity and the relatively high engagement in the negative-mood/no-cue condition had the merit of better performance. This association between effort and achieve-
ment was also visible in a significant, positive correlation between SBP reactivity during task performance and the total number of remembered letter series.

Table 1
Mean Number of Recalled Letter Series in Study 1 by Gendolla and Krüsken (2002a).

<table>
<thead>
<tr>
<th></th>
<th>Negative Mood</th>
<th>Positive Mood</th>
</tr>
</thead>
<tbody>
<tr>
<td>No-cue</td>
<td>8.65</td>
<td>6.71</td>
</tr>
<tr>
<td>Cue</td>
<td>6.65</td>
<td>8.12</td>
</tr>
</tbody>
</table>

Note: n = 17 in each cell.

A follow-up experiment administered a letter cancellation task for the performance period and replicated the effort-related findings of the first study. Additionally, the follow-up study involved a second mood manipulation check after task performance and found that the cue manipulation had no effect on mood intensity—mood remained stable in all experimental conditions. This is an important finding because it makes an alternative explanation implausible, according to which the mood zero-effect in the cue-condition emerged because the cue manipulation neutralized participants’ moods. Furthermore, the follow-up experiment found a mood congruency effect on ratings of subjective task difficulty in the no-cue condition but not in the cue condition. This finding further demonstrates that mood influenced task engagement because it had a congruency effect on demand appraisals.

In summary, the studies by Gendolla and Krüsken (2002a) have demonstrated that moods per se have no effects on effort mobilization but that they lead to higher engagement in a negative mood than in a positive mood when people use their moods as diagnostic information for demand appraisals. The results of further studies on the effects of mood under performance conditions without performance standards even allow generalizing these findings. Experiments that used different mood induction techniques (music, autobiographical recollection, video excerpts) and different types of mental demand revealed identical results: In line with the predictions, effort-related SBP reactivity was stronger in a negative mood than in a positive mood during performance on a letter cancellation task (Gendolla et al., 2001; Gendolla & Krüsken, 2002c, Study 2) or a verbal creativity task that was additionally manipulated in terms of task valence (Gendolla & Krüsken, 2001b). Thus, the informational mood impact on task engagement as conceptualized in the MBM is a robust effect.
4.4.2 Tasks With Performance Standards

In order to predict mood effects on motivational intensity when a performance standard is provided, it is necessary to note that the MBM posits that people use their moods as task-relevant information in a pragmatic way. As outlined earlier, this means that people use all available diagnostic information to evaluate the level of task demand that in turn determines effort intensity due to the conversation of resources principle. Thus, mood is one piece of diagnostic information that is integrated, according to Anderson’s (1981) averaging rule, into a judgment together with all other available information (Abele & Gendolla, 1999; Abele, Gendolla, & Petzold, 1998; Abele & Petzold, 1994). It follows that people in a positive or negative mood who are confronted with a task and receive information about a performance standard should consider both of the available diagnostic pieces of information—their mood state and the performance standard—to appraise the extent of demand. The resulting actual engagement is predictable through an application of the MBM reasoning on informational mood impact to Brehm’s motivational intensity theory (e.g., Brehm & Self, 1989), as illustrated in Figure 3.

Panel A of Figure 3 shows the general relationship between task difficulty and effort intensity. Effort is mobilized proportionally to the magnitude of subjective task difficulty up to the level of maximally justified effort. Once this point is accomplished, no more effort will be mobilized. The same happens on task difficulty levels that clearly exceed a person’s ability, making success impossible. Panel B of Figure 3 shows the informational impact of mood in this process: (1) Individuals in a negative mood will

![Figure 3](image-url)
mobilize more effort than individuals in a positive mood when the task is easy. This is because subjective task demand is higher in a negative mood than in a positive mood. (2) If a task is difficult, individuals in a negative mood will mobilize only little effort because they already perceive task demand as too high for them, resulting in disengagement. Individuals in a positive mood will, by contrast, mobilize high effort because they perceive task demand as high but not yet too high. (3) If a task is extremely difficult so that success is obviously impossible, mood will play no significant role and only little effort reflecting disengagement will be mobilized. This is predicted because the diagnostic weight of the information that task difficulty is extreme is so high that the effective weight of mood as a piece of diagnostic information becomes very low, according to the mood-and-information-integration perspective applied to the MBM.

An experiment by Gendolla and Krüsken (2001a) tested these predictions. After habituation and assessment of cardiovascular baseline values, we induced positive versus negative moods by exposing participants to either elating or depressing music. After a short distraction task and assessment of the mood manipulation check, participants received instructions for a letter cancellation task—the so-called “d2 task” (Brickenkamp, 1981). The task materials consisted of rows with random sequences of the letters d, p, and q. Placed above or below each letter were one, two, or no apostrophes. Participants received instructions to mark all d’s that carried two apostrophes (e.g., d”, d,,). To get an impression of the task, participants performed one practice line of 47 letters. Thereafter, all participants received the feedback that they had needed 25 s to complete the line (which was about the actual performance time). For the upcoming 5-min performance period, half of the participants were then informed that they would have to complete each line within 20 s (difficult condition), whereas the other half was told that they would have 30 s per line (easy condition). That is, participants in the difficult condition had to work 20% faster than during practice while those in the easy condition could work 20% slower.

The results showed no mood effects on cardiovascular reactivity during the mood inductions, although the mood manipulation was highly effective according to the manipulation check. However, participants remained on their cardiovascular baseline levels and showed even slight decreases in systolic reactivity, lending further support to the MBM notion that moods do not per se mobilize effort. Effects on SBP and DBP reactivity during task performance are depicted in Figure 4.
As anticipated, cardiovascular reactivity was significantly stronger in a negative mood than in a positive mood when the task was easy. Conversely, reactivity was significantly stronger in a positive mood than in a negative mood when the task was difficult. That is, mood state and performance standard had the anticipated joint effect on effort-related cardiovascular response.

Achievement was quantified as the number of correctly marked target symbols. As presented in Table 2, mood had no impact on this measure in the easy condition. Presumably, the performance standard was so low that modest engagement was sufficient for success. However, in the difficult condition, where the standard was high and where relatively intense effort was thus necessary for performing well, the achievement pattern corresponded to the pattern of effort-related cardiovascular response: Participants in the positive mood condition, where relatively high effort was mobilized, marked significantly more target letters correctly than participants in the negative mood condition, where only low effort was mobilized.

Participants in another experiment by Gendolla and Krüsken (2002c, Study 1) were first induced into positive versus negative moods through autobiographical recollection of happy versus sad life events and then performed a learning task. A number of letter series had to be memorized in 5 min. Specifically, participants tried to memorize four series in an easy condition, seven series in a difficult condition, and 20 series in an extremely difficult, actually impossible condition. The results exactly showed the pattern of SBP reactivity predicted in Figure 3: Systolic reactivity was stronger in a negative mood than in a positive mood when the task was easy, whereas it was stronger in a
positive mood than in a negative mood when the task was difficult. Most relevant, mood had no significant impact in the extremely difficult condition—SBP reactivity was low in both mood states. This extends the findings of the above-discussed experiment by Gendolla and Krüsken (2001a) because it demonstrates that mood loses, as predicted, its impact on motivational intensity when a performance standard provides the clear information that success is impossible. Furthermore, achievement was positively correlated with SBP reactivity in this study and systolic reactivity did not differ between the conditions during the effective mood inductions.

Table 2
Mean Number of Correctly Marked Target Letters in the Experiment by Gendolla and Krüsken (2001a).

<table>
<thead>
<tr>
<th></th>
<th>Negative Mood</th>
<th>Positive Mood</th>
</tr>
</thead>
<tbody>
<tr>
<td>Easy</td>
<td>456.43</td>
<td>454.07</td>
</tr>
<tr>
<td>Difficult</td>
<td>490.64</td>
<td>529.50</td>
</tr>
</tbody>
</table>

Note: \( n = 14 \) in each cell.

4.4.3 Effects of Performance-Contingent Reward

So far, the experimental evidence suggests that people use their moods as task-relevant information to evaluate the extent of subjective demand that determines the intensity of effort and that the use of moods as information follows pragmatic principles in that people consider mood and other diagnostic information, like performance standards, simultaneously. However, it remained open whether performance-contingent reward has an impact on this process. This is an important question because other researchers have claimed that performance-contingent reward directly determines the intensity of motivation (e.g., Eisenberger, 1992; Fowles, 1983). Given that reward has not been manipulated in the studies from our laboratory discussed so far it might be possible that the informational mood impact on effort mobilization only works when reward is relatively unclear or non-contingent upon success, but that reward directly determines engagement when it is clear and performance-contingent. Two recent experiments by Gendolla and Krüsken (2002b) addressed these questions. Again, predictions were derived from an application of the MBM reasoning on informational mood impact to Brehm’s motivational intensity theory (e.g., Brehm & Self, 1989).

Brehm’s theory distinguishes the level of maximally justified effort, defined as the level of potential motivation, from actual effort, which is motivational intensity. The level of potential motivation is determined by the importance of success: Relatively important
outcomes justify more engagement than relatively unimportant outcomes. Up to the level of potential motivation, motivational intensity is proportional to the extent of subjective demand in accordance with the difficulty law of motivation (Ach, 1935). That is, following the principle of resource conservation, the organism does not mobilize more effort than necessary and disengages if success necessitates more engagement than justified by performance-contingent reward. Thus, performance-contingent reward for accomplishing a goal (only) indirectly influences motivational intensity by setting the level of maximally justified effort.

What are the predictions for performance conditions where reward is contingent versus non-contingent upon success? In terms of Brehm’s theory, these two performance conditions differ only with regard to the level of maximally justified effort: An attractive performance-contingent reward makes success relatively important. Thus, performance-contingent reward justifies more effort than non-contingent reward. However, a person’s actual engagement depends, up to the level of potential motivation, on the level of subjective demand, which is systematically influenced by the informational mood impact on demand appraisals. Consequently, effort should not differ between contingent and non-contingent conditions when a task is easy or when a difficult task is performed in a positive mood because the extent of subjective demand is identical in both contingency conditions. But for succeeding on a difficult task in a negative mood, task engagement should be highly increased when success promises valuable consequences. The reason for this effect is that the very high effort that is perceived as necessary in this condition then becomes justified. Consequently, performance-contingent incentive can eliminate the above-presented motivational deficit of people in a negative mood who face a difficult task.

An experiment by Gendolla and Krüsken (2002b, Study 1) tested these predictions. Cardiovascular assessments were made during habituation, during the mood inductions through exposure to depressing versus elating video excerpts, and while participants performed an easy or difficult memory task. The task was to memorize either four (easy) or seven (difficult) series of four randomized letters in 5 min. Reward contingency was manipulated as follows: Half the participants (non-contingent reward) were told that a relaxation period with exposure to pleasant music would follow the performance period, irrespective of achievement. The other half (reward contingent) was told that the relaxation period would follow the performance period only if they succeeded on the memory task. Results are depicted in Figure 5.
When reward was non-contingent upon success, SBP responses were in a pattern consistent with the view that negative mood participants tried harder than positive mood participants under easy conditions but withheld effort under difficult conditions. As anticipated, the same pattern occurred in all but one condition when reward was contingent upon performance: The exception was that the motivational deficit of participants in the negative-mood/difficult condition disappeared, as predicted, when reward was contingent upon success. The subjectively necessary very high effort in this condition was only mobilized when it was justified by performance-contingent reward. Furthermore, achievement on the memory task was positively correlated with SBP reactivity during task performance, and demand appraisals assessed prior to performance showed that mood and performance standard had the anticipated additive effect on the extent of subjectively experienced demand. A follow-up experiment that promised non-contingent exposure to unpleasant materials versus performance-contingent exposure to pleasant materials replicated these findings. Given that the performance contingent incentive in these studies allowed hedonic affect regulation, the findings also support the MBM notion that informational and directive mood impact can occur simultaneously. Effort intensity was determined by the informational mood impact during the attempt to regulate the actual affective state by means of instrumental behavior that lead to hedonically pleasant consequences of success.
4.5 Conclusions and Implications

After outlining the predictions of the MBM (Gendolla, 2000) for the role of mood in motivation, the here discussed studies have focused on mood effects on motivational intensity. As predicted by the MBM, the empirical evidence demonstrates that moods can influence task engagement and performance through their informational impact. With reference to the question whether moods have stable motivational implications—which has been suggested by other researchers (e.g., Morris, 1992; Schwarz, 1990)—it is of note that none of the present studies has found mood effects on cardiovascular reactivity before task performance, although the mood states were successfully manipulated according to the manipulation checks and although mood had the predicted effects during task performance. That is, mood had no effects on effort mobilization until it was experienced in a setting that directly called for the mobilization of resources and in which it would be used as task-relevant information.

4.5.1 Basic Findings and Relations to Other Approaches

As conceptualized in the MBM as informational impact, participants used their moods as diagnostic information for demand appraisals, which in turn determined task engagement (Ach, 1935; Brehm & Self, 1989). When participants performed tasks without performance standards, mood was used as the only source of information for demand appraisals (e.g., Gendolla et al., 2001; Gendolla & Krüsken, 2001b, 2002c, Study 2). Consequently, more effort was mobilized in a negative mood than in a positive mood. As predicted, this effect diminished when the diagnostic value of mood as task-relevant information was called into question (Gendolla & Krüsken, 2002a). It is of note that this latter finding makes it implausible that the reported mood effects occurred because mood influenced mental capacity (e.g., Ellis & Ashbrook, 1988), which could have made cognitive tasks objectively more difficult (low capacity) in a negative mood than in a positive mood (high capacity). Likewise, the present findings are hard to explain with a mood-as-priming process (Bower, 1991; Forgas, 1995) in which mood congruency effects occur because moods activate related concepts in memory. According to both approaches, the present findings are only explicable by a mood decay effect of the cue manipulation—but this effect did not occur.

As further predicted by the MBM, participants pragmatically used two available sources of information—mood and performance standard—to evaluate the extent of demand when they faced tasks with a fixed performance standard. Engagement was then proportional to the magnitude of experienced difficulty, resulting in low effort when an easy task was performed in a positive mood (low subjective difficulty) and when a difficult task was performed in a negative mood (disengagement). Engagement was high when an easy task was performed in a negative mood and when a difficulty task was
performed in a positive mood (high subjective difficulty in both cases). When task difficulty was extremely high, so that success was clearly impossible, mood had no significant impact and effort was low in general (disengagement; Gendolla & Krüsken, 2001a, 2002c, Study 1). It is of note that these findings contradict the view that moods can only be used as information according to an “all-or-nothing” principle as Schwarz and Clore (1988) have proposed. Obviously, our participants considered their moods and other diagnostic information simultaneously.

Most interestingly, the motivational deficit of people facing a difficult task in a negative mood disappeared when reward for successful performance was contingent upon performance (Gendolla & Krüsken, 2002b). However, rather than determining effort intensity directly, as suggested by some researchers (e.g., Eisenberger, 1992; Fowles, 1983), performance-contingent reward had this effect by increasing only the magnitude of maximally justified effort. These findings fit Brehm’s motivational intensity theory (Brehm & Self, 1989) and contribute to the whole body of evidence for its applicability to effort mobilization in various settings (see Gendolla, 2004; Gendolla & Wright, 2005; R. A. Wright, 1996, 1998; R. A. Wright & Franklin, 2004; R. A. Wright & Kirby, 2001, for reviews).

It is noteworthy that also other researchers have considered informational effects of affective states on cardiovascular response. Blascovich and Berry-Mendes (2000) have posited that so called “affective cues” (e.g., sounds, smells, objects etc.) can determine whether tasks are appraised as “challenge” or “threat.” Challenge is then claimed to coincide with a strong cardiac response, whereas threat should coincide with a moderate cardiac and a strong vascular response. Beside numerous other problems of this framework and research that has already been discussed elsewhere (see R. A. Wright & Kirby, 2003), it is unclear how challenge and threat appraisals affect DBP, SBP, and mean arterial pressure (MAP) since these indices are determined differently (Brownley et al., 2000; Obrist, 1981). Furthermore, Blascovich and Berry-Mendes make no predictions on the impact of manipulated performance standards and thus cannot precisely predict the magnitude of cardiovascular response when people perform tasks of different difficulty levels. Also, neither the role of mood states nor the impact of performance-contingent consequences is considered in their approach. Thus, it is unclear how Blascovich and Berry-Mendes’ analysis could refer to the present findings.

4.5.2 Implications for Work Motivation in Organizational Settings

The here discussed studies on mood and effort mobilization investigated university student samples rather than employees and administered standardized cognitive tasks rather than real life work challenges. Nevertheless, the findings have clear and direct implications for work motivation and performance. In work settings, people have to cope
with demands that are related to memory, attention, and verbal creativity, like the participants in the present experiments. Likewise, organizational performance conditions either provide clear performance standards or they do not (Locke & Latham, 1990), and reward in work settings is either contingent or non-contingent upon performance. Furthermore, studies by Krüsken (2002) have found that naturally occurring mood states have the same effects on the intensity of motivation as experimentally manipulated mood states. This suggests that the present findings apply to organizational and educational settings because the basic organizational performance conditions correspond to those of our laboratory research.

The major suggestion of the present research is that organizations should take care about their employees’ moods. Moods are not only a key variable for work climate (e.g., George & Brief, 1992) but also for work motivation and performance as the here presented studies suggest (see also Grawitch & Munz, 2005). Likewise, it is necessary to care about work climate and work conditions because they will have consequences on workers’ mood states. Mood states can in turn systematically influence work motivation and thus performance and efficiency. However, it should also be considered that in the present studies the achievement gain through high effort was not very high, although significant. One has to keep in mind that the relationship between effort and achievement is complex and that it depends on several moderator variables such as inter-individual ability differences and the type of task (see Eysenck, 1982). Effort refers to the mobilization of resources in order to carry out instrumental behavior, whereas achievement (only) describes the outcome of behavior—which depends on more factors than only engagement. Consequently, associations between effort and achievement can but do not need to occur (Gendolla, 2004).

However, in the studies discussed above, engagement explained about 4-9% of the variance in achievement. This may be an important share that can decide about success or failure. But in the light of the potential health costs of chronically high work motivation, it might be worth considering that conditions, which elicit moderate effort intensity, could also be sufficient for maintaining good performance.

### 4.5.3 Implications for Ergonomics and Mental Workload

A central variable of interest in ergonomics is “mental workload.” Conceptualized as a multidimensional construct, it is assumed to reflect an individual’s level of attentional and mental engagement in a task (Wickens, 1984). In the last decades a vast bulk of techniques, among them behavioral, subjective, and physiological measures (Baldwin, 2003; O’Donnell & Eggemeier, 1986; Wierwille & Eggemeier, 1993), has been developed and applied for the assessment of mental workload. If one considers only the mental effort component of the mental workload construct, implications of our integrative
approach are obvious. So far, most applied research in the area of ergonomics assessing mental effort has been primarily based on the rather simple assumption that mental effort will increase proportionally with the complexity or the difficulty of the task (e.g., Veltman & Gaillard, 1998). Correspondingly, mental effort is often used as indicator of task difficulty.

The presented studies from our laboratory and other empirical evidence based on motivational intensity theory suggest that this view might be oversimplified. First, according to motivational intensity theory (Brehm & Self, 1989) and the supporting empirical evidence (R. A. Wright & Kirby, 2001), mental effort is not only determined by task difficulty but by the importance of success as well. Correspondingly, success importance determines the maximal level of mental effort one is willing to invest in the task. That is, mental effort indicators of mental workload might be biased by participants’ potential motivation to work hard. A measure indicating low mental workload might actually imply an easy task or a task for which the necessary engagement was not justified by the participant’s potential motivation. Furthermore, applied research often does not consider the influence of affective states. According to the here discussed MBM, moods can have an influence on mental effort via mood-congruency effects on appraisals of task difficulty. Again, mental effort measures of mental workload can be ambiguous. For instance, a mental effort measure indicating low mental workload might result from an indeed easy task, from a difficult task that was appraised as easy by an individual in a positive mood, or from a moderately difficult task that was appraised as too difficult by an individual in a negative mood.

In summary, the integrative approach developed in this chapter offers some insights in problems involved in the assessment of mental workload by means of mental effort and the use of mental effort as indicator of task complexity and difficulty. Since not only the willing to invest energy in the task has an influence on these measures, but affective states as well, the assessment of mental workload via measures of mental effort should always take the affective and motivational state of the individual into consideration.

4.5.4 Implications for Health Psychology: The Development of Cardiovascular Disease

There is clear and replicated evidence that the extent of cardiovascular reactivity during effortful coping with demands predicts the risk of hypertension and cardiovascular disease (see Blascovich & Katkin, 1993). The stronger the cardiovascular system reacts to a mental demand, the higher is the likeability of developing hypertension and a cardiovascular disease. A longitudinal study by Light, Dolan, Davis, and Sherwood (1992), for example, found that especially the magnitude of SBP reactivity during performance on
a mental demand predicted baseline blood pressure and the magnitude of cardiovascular reactivity to stressors 15 years later. The stronger the SBP responses were during earlier effortful coping, the higher was the probability of developing hypertension. Furthermore, there is clear evidence that personality characteristics that are related to negative affective experiences are correlated with the vulnerability to cardiovascular disease (Rugulies, 2002; Steptoe, Cropley, & Joekes, 2000). Examples are hostility (T. W. Smith, 1992) and neuroticism (Byrne, 1992). According to an analysis by Gendolla and Richter (2005b), the here discussed laboratory findings from our laboratory may contribute to an explanation of how this can happen. Accordingly, people who experience negative affect evaluate the extent of subjective demand higher when they face mental challenges. Consequently, they mobilize more effort than people who experience positive affect as long as they believe that it is possible and worthwhile to attain a goal. This is particularly important for work settings in which people are chronically confronted with mental demands. If people who chronically experience negative affect also chronically face such demands, they are especially vulnerable for the development of cardiovascular disease. This suggests that work settings that promote high cardiovascular reactivity over a long work period—like working without a clear performance standard in a negative mood—are harmful and provide a health risk (Gendolla & Richter, 2005b).

From this perspective, work and educational settings should facilitate the experience of positive rather than negative moods during work. This is relatively easy to be realized by pleasant illumination, pleasant odors, ergonomically well structured work places (see Gendolla, 2000), and any variables that are known for long as being related to high work satisfaction (see Locke, 1976). Positive mood during work may prevent the development of cardiovascular disease. This is especially relevant because the achievement deficit due to mobilizing only moderate effort for routine tasks seems to be relatively small. Positive mood is, however, not beneficial when people have to attain high performance standards. Consequently, both performance standards and mood have to be considered in order to prevent health risks, qualifying other approaches that have proposed that positive affect has mainly or even only health benefits (e.g., Fredrickson, 2001; Ryff & Singer, 1998; Salovey, Rothman, Detweiler, & Steward, 2000).

Nevertheless, negative mood during work seems to bear multiple risks: The cardiovascular system reacts strongly during performance on easy tasks and tasks without performance standards. When reward is contingent upon performance, which is frequently the case in work settings, very strong reactivity is facilitated. Creating work settings that facilitate effective but non-harmful performance is thus a major challenge for effective leadership, ergonomics, and organizational behavior.
4.5.5 Implications for Clinical Psychology: Mood, Depression, and Motivation

As outlined above, an important aspect for organizations and for the construction of work settings is the mental health of their employees. In this context, it is important to note that not only diagnosed clinical pathologies but also minor subsyndromal symptoms may have an impact on employees’ work, the working atmosphere, and organizational structures. In this section, we highlight implications of our integrative approach for depressed mood and depression, respectively. However, there is also evidence for the implications of the MBM with regard to other individual difference variables like, for example, dispositional anxiety or low self-esteem. Research on depression is particularly important because depression is considered by the World Health Organization as the most burdensome disease in the world in terms of total disability-adjusted life years among people in the middle years of life (Kessler, 2002). The prevalence rates for depression are high and still rising, not only for clinically diagnosed depression (e.g., major depressive disorder) but also for subsyndromal depressive symptoms (e.g., minor depression), which means that many students’ and employees’ working life may be affected at some time and in some way by a depressive episode.

An integrative approach of mood and motivation regarding depression and dysphoria lends itself because a persistent negative mood is considered as one of the two core symptoms of major depressive disorder (DSM-IV, American Psychiatric Association, 1994). Thus, it allows the investigation of the role of naturally occurring negative mood. In addition, research among clinically depressed individuals as well as subclinical populations supports the notion that depression is associated with emotional, cognitive, functional, and motivational deficits (e.g., Heckhausen, 1991). The motivational deficit—defined as an inability to initiate actions, to mobilize efforts, and to persist on these actions—has been demonstrated in research from different perspectives (see for example Abramson et al., 1981; Fowles, 1993; Henriques & Davidson, 2000; Layne et al., 1982; Tomarken & Keener, 1998). Thus, considering that moods can influence behavior, as for instance action initiation and effort mobilization, our integrative approach may help explaining and investigating depressed individuals’ motivational deficit.

Evidence for an informational impact of naturally occurring mood stems, for example, from studies by Lyubomirsky et al. (1999). Dysphoric individuals rated the likelihood of solving a personal problem and implementing their solution significantly lower than nondysphorics. The authors concluded that dysphoric rumination might interfere with efforts to take steps to problem solving. In terms of the MBM, this means that dysphoric individuals have a motivational deficit due to their more negative evaluation of the actual situation on the basis of their negative mood. Evidence for the conceptualization of a directive mood impact of naturally occurring mood is provided by a body of
Theoretical Part: Mood-Behavior-Model

research showing that depression and other negative affect dispositions coincide with a weak directive mood impact in terms of deficits in self-regulation of affective experiences. As Josephson et al. (1996) reported, depressed individuals are not able to self-regulate their negative affect by activating positive memories ("mood-incongruent recall"). Moreover, Gilboa and Gotlib (1997) showed that previously dysphoric individuals better remembered negative stimuli and stayed longer in negative affect after a negative mood induction than never-dysphoric individuals. Further evidence for individual differences in self-regulation stems from research showing that depressives are unable to withdraw from a "depressive self-focusing style" (Pyszczynski & Greenberg, 1987): Depressives tended to reflect on themselves after failure but not after success, whereas nondepressives showed the inverse pattern.

Moreover, in the extensive literature about depressives’ undervaluation of rewards, there is evidence for both informational and directive mood impact. Costello (1972) suggested that depression decreases the effectiveness of reward insofar as depressed individuals showed a lack of responsiveness to pleasurable stimuli. Henriques and colleagues have explained this with decreased activity in the left anterior cortex, which is considered to produce an underactivation of the reward-based system (e.g., Henriques & Davidson, 2000). Moreover, Tillema et al. (2001) found that a dysphoric individuals’ actual negative mood state elicited the undervaluation of a perceived outcome. These findings regarding the undervaluation of rewards by depressives show on the one hand that a depressed individuals’ actual mood state may influence the evaluation of a perceived outcome, which is explicable in terms of an informational mood impact on self-regulation. On the other hand, this leads to low perceived instrumentality of potential affect-regulative acts and thus reduces the strength of directive mood impact. This interpretation is consistent with the MBM postulate that informational and directive mood impacts can occur simultaneously.

In support of our approach, recent research from our laboratory has investigated and shown the influence of vulnerability for depression on cardiovascular reactivity (Brinkmann & Gendolla, 2005). Students with high depression scores were compared to a control group while being confronted with a cognitive task without fixed performance standard. The results showed that depressed students—who were at the time of the experiment also in a more negative mood—showed stronger SBP reactivity than the control group. Thus, depressed individuals mobilized more mental effort, indicating higher motivational intensity at the time of task performance. Interestingly, the high effort of depressives’ was accompanied by poorer performance. That is, depressives’ mobilized relatively high effort without the merits of better performance.

Besides this evidence for the relations between depression and motivation and the implications for the MBM, there has been a body of recent research showing that depres-
sion is among the risk factors for development and worsening of coronary heart disease (Frasure-Smith & Lespérance, 2005a; Rugulies, 2002; Wulsin & Singal, 2003). One of the most prominent explications states that dysregulation of the autonomic nervous system may explain why depressed patients are at increased risk for coronary heart disease. The altered autonomic nervous system activity found in depressed patients is seen as an indicator of increased sympathetic nervous system activity (Carney et al., 2005). As outlined above, a stable disposition to stronger cardiovascular reactivity can be considered as a risk factor for cardiovascular disease. Moreover, there is evidence that also other negative psychological states like hopelessness, pessimism, rumination, anxiety, and anger are linked in varying degrees to cardiovascular diseases (Kubzansky et al., 2005). One may speculate whether, among other factors, also affective states play an important role in the linkage between depression and coronary heart disease.
EXPERIMENTAL PART
1. Overview Over the Studies and Hypotheses

As outlined in the first chapters of the theoretical part, evidence about motivational deficits in depression and dysphoria is existent but not always conclusive. Moreover, motivation is a broad concept, so that the notion of motivational deficits in depression has to be qualified by taking into account the different dimensions of motivation, namely the initiation, direction, persistence, and intensity of behavior (Geen, 1995; Vallerand & Thill, 1993). Some of the research dealing with motivational issues in depression takes this into account and focuses on one specific aspect—as for instance the cognitive-initiative account by Hertel (2000) that refers to the initiation of actions. Clinical observations of depressives’ deficient motivation are mainly concerned with the initiation of actions, too.

Motivational intensity theory (Brehm & Self, 1989) and its related research offer a different approach for the investigation of motivation: They allow formulating and testing hypotheses about the intensity of resources mobilized by individuals who are engaged in instrumental challenges. Moreover, the integration of the MBM (Gendolla, 2000) and motivational intensity theory offers clear predictions about mood’s influence on the mobilization of resources. As outlined in chapter 4 of the theoretical part, a series of experiments has tested hypotheses with respect to experimentally manipulated, transient mood states. These studies have shown convincing evidence for the postulated process of an informational mood impact on effort mobilization.

On the basis of the assumption that one of the core symptoms of depression and dysphoria is a persistent negative mood (DSM-IV, American Psychiatric Association, 1994; Mineka et al., 1998; Scott & Ingram, 1998), the link between dysphoria and effort mobilization via an informational mood impact seems plausible. Therefore, we designed four quasi-experiments to test the following hypotheses:

1) Dysphoria is characterized by a negative mood that persists even if the trait measure of dysphoria precedes the state measure of momentary mood by some weeks.

2) There is evidence that dysphoric individuals’ cardiovascular activity is enhanced under resting conditions (Light et al., 1998). On the other hand, the MBM (Gendolla, 2000) predicts that cardiovascular baseline activity should not differ with regard to mood. As we build our assumptions on the MBM, we expect cardiovascular baseline activity not to differ in our young and relatively homogeneous samples.

3) The MBM predicts furthermore that dysphoria is associated with a more pessimistic evaluation of task demand. Therefore, we hypothesize that, in contrast to baseline activity, dysphoric individuals show higher effort mobilization under conditions of unfixed task difficulty compared to nondysphoric individuals.
4) Under conditions of fixed task difficulty, dysphoria is also associated with a more pessimistic evaluation of task demand. However, these appraisals should interact with the objective difficulty of the given task. For the comparison of an easy and a moderately difficult task, we expect a crossover interaction pattern with high effort in the easy-dysphoric and the difficult-nondysphoric groups. In contrast, participants in the easy-nondysphoric group should mobilize less effort and participants in the difficult-dysphoric group should disengage because of subjectively too high task demand.

5) In a design with fixed task difficulty, the extent of subjective task demand depends on both, objective task difficulty and dysphoria. Therefore, subjective demand appraisals should linearly increase from the easy-nondysphoric to the difficult-nondysphoric and easy-dysphoric groups. They should be the highest for the difficult-dysphoric group.

6) Cognitive performance is likely to be impaired in dysphoria (e.g., Burt et al., 1995; Christensen et al., 1997). However, previous research has found moderately positive correlations between performance and effort (see Gendolla et al., 2001; Gendolla & Krüsken, 2002a, 2002b, 2002c), suggesting a more differentiated pattern of performance in dependence on effort and other factors. Moreover, the mental tasks used in this research program are chosen rather because of simplicity and minimal physical demand than because of their sensitivity to detect cognitive impairments. Therefore, we do not formulate specific hypotheses with respect to performance effects.

In the theoretical part, the conceptualization of depression as a dimensional versus a categorical construct has been discussed (chapter 2.1.3). Depending on this conceptualization, the selection of students with high depression scores as analogues for patients with clinical depression is controversial (see Kendall & Flannery-Schroeder, 1995; Tennen et al., 1995a; Weary et al., 1995). However, recent findings favor the dimensional perspective (Hankin et al., 2005; J. Ruscio & Ruscio, 2000; Solomon et al., 2001). Moreover, depending on the respective research focus, student participants with subclinical symptoms can provide a valuable contribution to the understanding of depression (Vredenburg et al., 1993). As our research program mainly focuses on the influence of negative affect—and not on the influence of somatic symptoms—we chose samples of undergraduate students for our studies. Then, we selected those students with depression scores located in the lower and upper third or quartile of the distributions. We refer to these participants as either “dysphoric” or “nondysphoric” to give consideration to the subclinical character and to avoid any confusion with clinical depression.

Each of the four studies tested Hypotheses 1 and 2 as basic conditions for the subsequent assumptions. Studies 1 and 2 were conceptually identical and tested Hypothesis 3 with respect to two different kinds of tasks without fixed performance standard. Studies 3 and 4 were conceptually identical and tested Hypothesis 4 with respect to
two different kinds of tasks on two fixed performance levels—easy and moderately difficult. Moreover, the latter two studies assessed participants’ subjective demand appraisals to test Hypothesis 5. Performance was analyzed exploratively in each of the studies.

As in the studies by R. A. Wright and colleagues (see R. A. Wright, 1996, 1998; R. A. Wright & Kirby, 2001, for reviews) and by Gendolla and colleagues (see Gendolla & Brinkmann, 2005; Richter et al., 2006, for reviews), we operationalized motivational intensity—our primary dependent variable—as the change in cardiovascular activity from a baseline level to task performance. Our physiological devices allowed measuring SBP, DBP, and HR. According to Obrist’s (1981) active coping approach and its integration with motivational intensity theory (Brehm & Self, 1989) by R. A. Wright (1996), beta-adrenergic sympathetic influences on the myocardium are proportional to effort or task engagement. Therefore, we expected that especially SBP reactivity would display our hypotheses concerning resource mobilization.
2. Studies 1 and 2

Dysphoria and Mobilization of Mental Effort: Effects on Cardiovascular Reactivity\(^5\)

Two studies examined the influence of dysphoria on motivational intensity in a student sample. Participants worked on a memory task (Study 1) or a mental concentration task (Study 2) without fixed performance standard ("do your best"). Based on their scores on the Center for Epidemiologic Studies – Depression Scale (Radloff, 1977), dysphoric and nondysphoric students were compared with regard to their effort-related cardiovascular reactivity during task performance. As predicted on the basis of the mood-behavior-model (Gendolla, 2000) and motivational intensity theory (Brehm & Self, 1989), dysphoric participants showed stronger cardiovascular reactivity while working on the cognitive tasks than nondysphoric participants. In Study 1, nondysphoric participants performed better on the memory task than dysphoric participants. Theoretical implications are discussed.

People suffering from depression or dysphoria are said not only to experience long lasting negative mood and to have emotional, functional, and cognitive deficits but also to lack motivation (Heckhausen, 1991). A motivational deficit in depression has been considered under different perspectives—amongst others with respect to behavioral approach and inhibition systems (Gray, 1982), self-regulation (e.g., Strauman, 2002), motivational influences on cognitive deficits (e.g., Hertel, 2000), and mood-congruent negative cognitions (e.g., Scott & Ingram, 1998).

Our research addresses the question whether dysphoria and depression are associated with motivational deficits with regard to the intensity of motivation. Specifically, we are interested in the mobilization of mental effort during performance of instrumental tasks as an indicator of the intensity of motivation at a certain point in time. Based on the notion that dysphoria is largely characterized by a depressed mood, we posit that moods play an important role in determining how dysphoric individuals react to behavioral challenges when they need to self-regulate the mobilization of resources for performance. Based on the research reported above, the mood-behavior-model (MBM; Gendolla, 2000), and motivational intensity theory (Brehm & Self, 1989), we develop and test predictions for the impact of dysphoria on motivational intensity. Effort mobilization

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is quantified as the reactivity of the cardiovascular system, especially the reactivity of systolic blood pressure (Gendolla & Brinkmann, 2005; R. A. Wright, 1996).

### 2.1 Dysphoria, Motivation, and Self-Regulation

Before describing theory and hypotheses in detail, we highlight some findings in depression and dysphoria research—stemming from clinical patient as well as subclinical community or student samples—which address motivational and self-regulation processes. First, there is evidence based on behavioral, self-report, and neurophysiological measures that depression is associated with weak approach motivation, as indicated by a relative hypoactivation of the left frontal cortex and lower responsiveness to rewards (e.g., Davidson, Pizzagalli, & Nitschke, 2002; Fowles, 1994; Gotlib et al., 1998; Henriques & Davidson, 2000; Kasch et al., 2002; Tomarken & Keener, 1998). Findings with respect to a hyperactivation of behavioral inhibition in depression or dysphoria are existent but less consistent (e.g., Dickson & MacLeod, 2004; Kasch et al., 2002; Pinto-Meza et al., 2006).

In light of this evidence, Strauman (2002) postulated an incorporation of the self into the brain-behavior system in order to better describe and understand human approach and avoidance motivation. Accordingly, depressive individuals suffer from impairments of self-regulation resulting in affective and motivational deficits. The importance of self-regulation has also been stressed by others who point to the fact that the cognitive deficits commonly observed in depressive individuals and patients (see Burt et al., 1995; Rogers et al., 2004, for reviews) might partially be caused by motivational deficits. There is, for instance, evidence that aspects of self-regulation problems, such as rumination (Hertel, 1998; Watkins & Brown, 2002), unrealistic intentions (Kuhl & Helle, 1986), and deficits in attention control and initiative (Hertel, 2000), play an important role in the association between depression or dysphoria and cognitive deficits. Likewise, Abramson et al. (1981) concluded that depressed individuals suffer from a motivational rather than from a cognitive-associative deficit.

Furthermore, studies on the mood-congruent memory bias in depression and dysphoria point at reduced self- and affect-regulation abilities. Josephson et al. (1996) found that depressed individuals were not able to self-regulate their negative affect by activating positive memories (mood-incongruent recall). Likewise, Gilboa and Gotlib (1997) showed that previously dysphoric individuals better recalled negative stimuli and stayed longer in a negative mood after a negative mood induction than did never-dysphoric individuals. Greenberg and Pyszczynski emphasized the role of the depressive self-focusing style as a mediator for attributions after success or failure that leads to a self-serving bias in nondepressed but not in depressed individuals (e.g., Greenberg et al., 1992; Pyszczynski & Greenberg, 1987).
Finally, there is evidence that depression and dysphoria can—besides other negative cognitive biases—influence judgments and evaluations (e.g., Scott & Ingram, 1998). Lyubomirsky et al. (1999) reported that dysphoric individuals rated the probability of solving a personal problem lower than did nondysphoric individuals. The authors concluded that dysphoric rumination depletes energy and motivation and interferes with efforts to problem solving.

2.2 Mood and Effort Mobilization

A number of recent studies on the effects of transient mood states on the mobilization of mental effort have supported the predictions of the MBM (Gendolla, 2000). This model posits that moods per se do not have motivational implications but that they influence behavior in the context of behavioral challenges, such as instrumental tasks. One of the two processes proposed to influence behavior is the informational mood impact, which states that moods influence the intensity and persistence of behavior. This impact presumes that moods may influence behavior via mood-congruency effects on behavior-related judgments and evaluations. Accordingly, people in a negative mood tend to appraise a given task as more difficult and their own capacities as lower than do people in a positive mood. Gendolla and colleagues have found clear support for these assumptions (see Gendolla & Brinkmann, 2005; Richter et al., 2006, for reviews).

The authors manipulated their participants’ mood and subsequently asked them to do their best (i.e., to self-regulate resources for performance) on a mental task for which no performance standard was provided. By this means, they could show that participants in a negative mood actually mobilized more effort than participants in a positive mood as indicated by stronger systolic blood pressure reactivity of negative mood participants (e.g., Gendolla et al., 2001; Gendolla & Krüsken, 2002c, Experiment 2). The replicated finding from studies administrating different mood induction procedures and different types of cognitive demands was that cardiovascular activity did not differ between positive and negative mood groups before and after the mood induction. It was only at the time of task performance that negative mood led to stronger cardiovascular reactivity than positive mood. This effect did not occur because of different importance attributed to the task but because of subjectively higher task demand in a negative mood (e.g., Gendolla & Krüsken, 2002b, 2002c). Similar results have been found by R. A. Wright and colleagues in samples with high versus low perceived ability (e.g., R. A. Wright & Dill, 1993; R. A. Wright et al., 1997). The mood effect on difficulty appraisals

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6 Motivational intensity theory distinguishes between tasks with and without fixed performance standards, that is, with or without a certain performance level to attain. Tasks without fixed performance standards are labeled “unfixed” difficulty tasks; the difficulty level can be determined by the participants themselves who are simply asked to do their best (Brehm & Self, 1989).
and systolic blood pressure reactivity during performance diminished when the significant value of mood as information was reduced (Gendolla & Krüsken, 2002a).

These studies also support the notion that the intensity of motivation can be reliably operationalized by cardiovascular reactivity as proposed by R. A. Wright’s integration of motivational intensity theory (Brehm & Self, 1989) and Obrist’s (1981) active coping approach (see R. A. Wright, 1996; R. A. Wright & Kirby, 2001, for reviews). The active coping approach builds on evidence that beta-adrenergic influences exerted by the sympathetic nervous system on the cardiovascular system are proportional to effort and task engagement (Obrist, 1981). Therefore, assessing the reactivity to a behavioral challenge of cardiovascular indices reflecting beta-adrenergic sympathetic activation provides an effective means of operationalizing task engagement. This line of research has mainly focused on systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR; R. A. Wright, 1996; R. A. Wright & Kirby, 2001), even though other more direct measures as for instance pre-ejection period are also conceivable.

However, especially SBP has been proven to be a reliable index of effort mobilization, which makes sense with respect to sympathetic and parasympathetic influences on the heart: SBP mainly varies with the contractility strength of the heart muscle. Myocardial contractility is known to directly increase with sympathetic discharge, which is related to activation. Consequently, an increase in SBP activity is regarded as a reliable indicator of increased task engagement. In contrast, DBP is predominantly determined by the overall flow resistance of the blood vessels in the body. Total peripheral resistance, however, is not systematically influenced by sympathetic activation, and therefore DBP reactivity constitutes a less reliable index of effort mobilization. Finally, HR is independently determined by both sympathetic (i.e., HR accelerating) and parasympathetic (i.e., HR decelerating) influences. An increase in HR can therefore represent both sympathetic activation and parasympathetic withdrawal. Consequently, HR can be considered an indicator of sympathetic activation to the extent that its influence is not masked by parasympathetic activity. In summary, SBP is a reliable and sensitive indicator of effort mobilization, even though increases in sympathetic activation may also result in simultaneous elevations of SBP, DBP, and HR (see Brownley et al., 2000; Obrist, 1981; Papillo & Shapiro, 1990; T. W. Smith et al., 1989, for more details).

### 2.3 The Present Studies

The present studies tested the central hypothesis that dysphoric individuals would show stronger cardiovascular reactivity—especially SBP—while performing a cognitive task without fixed performance standard than nondysphoric individuals. The main advantage of operationalizing motivational intensity as cardiovascular reactivity is that it is less susceptible to social presentation biases than self-report measures and less influ-
enced by individual capacity and performance strategies than performance measures. Therefore, no directed hypotheses were formulated with regard to performance on the cognitive tasks. On the one hand, performance has been shown to be positively related to effort mobilization (e.g., Gendolla et al., 2001; Gendolla & Krüskens, 2002a, 2002b, 2002c). But on the other hand, there is evidence that depressed and dysphoric individuals perform worse in cognitive tasks than normal controls (e.g., Burt et al., 1995; Rogers et al., 2004). Thus, in the present context of dysphoria effects on motivational intensity, performance was assessed exploratively.

We chose student samples with extreme scores on a self-report depression scale for our studies. We did not expect that students with high scores were suffering from manifest clinical depression but assumed that they would be dysphoric, show subclinical symptoms, and have higher vulnerability for depression. Therefore, we also assessed participants’ current mood state. Moreover, there are good reasons to choose a student sample for depression research because of many environmental stressors, high prevalence of depressive symptoms, and lower probability of other (psycho-)pathologies and concurrent treatments in such a sample, as well as the homogeneity of the group (Vredenburg et al., 1993). Results concerning gender differences in student samples with respect to depressive symptoms are mixed: The usual gender differences between women and men in both clinical and subclinical populations are not consistently found in student samples (Nolen-Hoeksema, 1987, 2001). Thus, we decided to include only women in our sample with the objective of reducing variability in group assignment due to gender biases in the self-report of depressive symptoms. Finally, we chose cognitive tasks that demanded a minimum of motor movement from the participants to ensure that cardiovascular changes could be attributed to the mobilization of mental resources rather than to metabolic movement effects.

2.4 Study 1

This study compared two groups of university students (dysphoric vs. nondysphoric) with regard to their cardiovascular reactivity during performance of a memory task. We expected performance-related cardiovascular reactivity—especially SBP—to be more pronounced in dysphoric than in nondysphoric participants.

2.4.1 Method

Participants

One-hundred forty-seven students in an introductory psychology course participated in questionnaire sessions in exchange for course credit. Out of this sample, 35 women (mean age 23 years) who scored in the lower or the upper quartile of the Center for Epidemiologic Studies – Depression Scale (CES-D; Radloff, 1977) were randomly
selected for participation in an ostensibly unrelated experiment via a personal code that
guaranteed anonymity. Seventeen participants were situated in the upper quartile of the
CES-D ($M = 33.47$, $SE = 2.15$) and therefore referred to as dysphoric. Eighteen partici-
pants were located in the lower quartile of the CES-D ($M = 6.61$, $SE = 0.66$) and there-
fore referred to as nondysphoric.

**Physiological Apparatus**

Cardiovascular measures were obtained noninvasively with a computer-aided,
multichannel monitor (Par Electronics Physioport III) that uses oscillometry to determine
SBP (millimeters of mercury [mmHg]), DBP (millimeters of mercury [mmHg]), and HR
(beats per minute [bpm]). A blood pressure cuff (Boso) was placed over the brachial
arteria above the elbow of participants’ nondominant arm and inflated automatically
every 2 min during 2 measurement periods—habituation (baseline) and task per-
formance. Each single measure took less than 1 min. Obtained values were stored on a
computer disk so that both experimenter and participants were ignorant of all physiologi-
cal values measured during the experiment.

**Self-Report Data**

In order to survey depressive symptomatology, the French version of the CES-D
(Fuhrer & Rouillon, 1989) was administered in the mentioned questionnaire sessions
about 2 weeks prior to the experiment. The CES-D is a short self-report scale especially
developed for use in community samples and consists of 20 items asking for frequency of
depressive symptom experience on a scale from 0 (never, very seldom) to 3 (frequently,
always). Radloff (1977) proposed a cut-off score of 16 to distinguish depressed from
nondepressed individuals, whereas Fuhrer and Rouillon (1989) favored cut-off scores of
17 and 23 for men and women, respectively. The CES-D has proven its excellent validity
in depression research and is reported to have better scale discriminability than the Beck
Depression Inventory (see Santor, Zuroff, Ramsay, Cervantes, & Palacios, 1995). It also
showed high internal consistency ($\alpha = .96$).\(^7\)

At the beginning of the experimental session, we administered the positive (i.e.,
“happy”, “joyful”, “contented”, “cheerful”) and negative (i.e., “sad”, “frustrated”,
“depressed”, “dissatisfied”) hedonic tone scales of the UWIST mood adjective checklist
(Matthews, Jones, & Chamberlain, 1990) in order to assess participants’ momentary
mood state. Participants indicated the extent to which each adjective reflected their
momentary feeling state on 7-point scales ranging from 1 (not at all) to 7 (very much).

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\(^7\) We also included the BDI-II (Beck, Steer, & Brown, 1996) in the initial questionnaire session. Participants’ scores on the BDI-II were highly correlated with their CES-D scores, $r(35) = .96$, $p < .001$. Group assignment did not change when considering the BDI-II instead of the CES-D for the selection process.
Procedure

The study was conducted with the help of the psychological software E-Prime (version 1.1, Psychology Software Tools) by an experimenter who was hired and unaware of both the hypotheses and participants’ depression status. The experimental session took about 30 min and was described as an investigation of physiological activity during periods of relaxation and demand. Participants, who attended the session individually, were greeted and seated in front of a computer. After signed consent was obtained, the experimenter attached the blood pressure cuff and gave instructions for computer use. Then she left the room and monitored the experiment from an outside control room. Computer-generated beeps informed her about the beginning of each experimental period without the need for seeing the participants and their entries on the computer. The experiment started with general instructions and assessment of biographical data and participants’ momentary mood state with the UWIST scale.

For the following habituation period, the experimenter reentered the room, handed a magazine to the participant, and informed her that she was allowed to read for a period of about 9 min while cardiovascular measures would be taken. Then the experimenter left the room and started the blood pressure monitor. After assessment of the five cardiovascular baseline values, the experimenter reentered the room, took the magazine, and instructed the participant to continue working on the computer.

The experiment continued with instructions concerning the memory task. Participants were instructed to memorize within 5 min as many letter series (e.g., EPQZ) as possible out of a list of 15 senseless letter series and to note the recalled series afterwards. That means that we simply asked them to do their best instead of providing them with a fixed performance standard (i.e., a fixed number of letter series to memorize). A preview of the list of letter series was then presented for 1 s on the computer screen to give participants an impression of the task. Then the 5 min performance period started. Meanwhile, the 15 letter series were simultaneously presented on the computer screen, and 3 cardiovascular measures were taken 15 s, 135 s, and 255 s after task onset. After having noted the letter series they could recall, participants learned that the experiment was over. The experimenter reentered the room, removed the blood pressure cuff, and thanked the participant. Finally, participants were carefully debriefed, interviewed with regard to suspicion, and received their course credit.

2.4.2 Results

Self-Reported Mood

We calculated mood scores by summing the scores of the positive ($\alpha = .93$) and the negative ($\alpha = .88$) hedonic tone adjectives of the UWIST scale. Additionally, we also formed a global mood score by summing the positive and the reverse-scored negative
adjectives of the UWIST scale (α = .88). Results revealed highly significant differences between the two dysphoria groups with regard to their momentary mood state at the beginning of the experiment: Mood scores of dysphoric participants on the positive adjectives \((M = 16.29, SE = 1.11)\) were significantly lower than those of nondysphoric participants \((M = 20.72, SE = 1.17)\), \(t(33) = 2.73, p < .01\). The reverse was found for the mood scores on the negative adjectives (dysphoric \(M = 11.12, SE = 1.31\) vs. nondysphoric \(M = 4.67, SE = 0.29\)), \(t(17.57) = 4.79, p < .001\). Finally, dysphoric participants \((M = 37.18, SE = 1.95)\) had lower scores on the global mood score than nondysphoric participants \((M = 48.06, SE = 1.25)\), \(t(33) = 4.75, p < .001\). Accordingly, as expected, dysphoric students were in a more negative and a less positive mood when arriving at the laboratory.

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Means and Standard Errors of Cardiovascular Baselines in Study 1</th>
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<tbody>
<tr>
<td>Cardiovascular baselines</td>
<td>Dysphoric</td>
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<tr>
<td></td>
<td>(M)</td>
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<tr>
<td>SBP</td>
<td>112.82</td>
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<tr>
<td>DBP</td>
<td>74.35</td>
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<tr>
<td>HR</td>
<td>76.79</td>
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Note. \(N = 17\) (dysphoric) and \(N = 18\) (nondysphoric). SBP and DBP are indicated in millimeters of mercury (mmHg), HR is indicated in beats per minute (bpm).

Cardiovascular Baselines

Cardiovascular baseline scores for SBP, DBP, and HR were created by averaging the last two values obtained during habituation (αs = .91 for SBP baseline, .79 for DBP baseline, and .96 for HR baseline).\(^{9}\) Means and standard errors are presented in Table 3. According to independent-samples \(t\)-tests, there were no significant differences between dysphoric and nondysphoric participants with regard to their cardiovascular baseline measures; SBP and HR: \(ts < 1, ps > .50\); DBP: \(t(33) = 1.60, p > .13\).

\(^{8}\) Degrees of freedom are adjusted because of inequality of variances.

\(^{9}\) The reason for this is that repeated measures ANOVAs revealed a decline of cardiovascular values over the first measures, whereas the last two measures remained stable (\(ps > .30\)). This decline is a common finding and due to habituation to the experimental setting and the fact being seated for a while.
**Cardiovascular Reactivity**

We calculated change (delta) scores for each participant by subtracting the baseline values from the arithmetic means obtained during task performance (see Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). A preliminary analysis revealed that DBP and HR delta scores were not correlated with the respective baseline values, $r_s < .21, ps > .23$. In contrast, SBP delta scores were significantly correlated with systolic baseline values, $r(35) = -.38, p < .03$. Therefore, we adjusted systolic change scores with respect to SBP baseline values in order to prevent carry-over effects and biases due to the law of initial values (see Llabre et al., 1991).

**SBP.** We first submitted the baseline-adjusted reactivity scores (delta) to an independent-samples $t$-test. Results revealed that albeit dysphoric participants ($M = 5.92, SE = 1.49$) showed stronger SBP reactivity than nondysphoric participants ($M = 3.64, SE = 1.07$), the difference did not reach significance, $t(33) = 1.25, p = .11$. Subsequently, we analyzed the single reactivity scores for each measurement time (delta 1, delta 2, and delta 3). This procedure is reasonable because of the above mentioned self-regulation difficulties in depression that might implicate difficulties in maintaining self-regulation (and effort mobilization) over a certain period. Therefore, we submitted the single baseline-adjusted reactivity scores to a 2 (dysphoria) x 3 (time) mixed model ANOVA to test whether systolic reactivity changed over time. This analysis revealed no main effect of time, $F(2, 64) = 1.64, p = .20$, but a significant dysphoria x time interaction, $F(2, 64) = 3.69, p = .03$, indicating that dysphorics’ reactivity declined over time while nondysphorics’ reactivity remained relatively stable (see Figure 6). To further highlight this effect, we compared dysphoric and nondysphoric individuals with regard to each single baseline-adjusted reactivity score. As depicted in Figure 6, dysphorics generally showed stronger SBP reactivity than nondysphorics. However, only the difference on the first reactivity measure delta 1 was reliable (dysphoric $M = 7.31, SE = 1.64$ vs. nondysphoric $M = 3.59, SE = 1.15$), $t(33) = 1.88, p = .03$.

**DBP and HR.** There were no differences between dysphoric and nondysphoric participants with regard to their DBP and HR overall reactivity scores (delta), as indicated by independent-samples $t$-tests, $ts < 1, ps > .17$. We then calculated single reactivity scores for DBP and HR as we did for SBP and submitted them to 2 (dysphoria) x 3 (time) mixed model ANOVAs. Results revealed neither main effects of time nor dysphoria x time interactions, $Fs < 2.15, ps > .13$. Therefore, we did not further compare the DBP and HR single reactivity scores.

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10 Given our directed a priori hypothesis we conducted one-tailed $t$-tests for comparisons between dysphoric and nondysphoric groups with regard to their cardiovascular reactivity.
Experimental Part: Studies 1 and 2

Figure 6
Cell means and standard errors of systolic blood pressure reactivity for measurement times 1, 2, and 3 in Study 1. Cell means of systolic reactivity scores are baseline-adjusted.

Task Performance
In order to investigate participants’ task performance, we analyzed the total number of recalled letter series, the number of correctly recalled letter series, and the ratio of correctly to totally recalled letter series with independent-samples t-tests. Data of 3 participants were excluded because they differed more than two standard deviations from the respective group mean, so that there were 16 participants per group. With regard to the number of correctly recalled letter series (nondysphoric $M = 6.56$, $SE = 0.56$ vs. dysphoric $M = 4.63$, $SE = 0.42$), $t(30) = 2.79$, $p < .01$, as well as the ratio of correctly to totally recalled letter series (nondysphoric $M = 0.82$, $SE = 0.04$ vs. dysphoric $M = 0.68$, $SE = 0.05$), $t(30) = 2.16$, $p < .04$, nondysphoric students significantly outperformed dysphoric students. Results regarding the total number of recalled letter series also revealed that nondysphoric students tended to show better performance than dysphoric students (nondysphoric $M = 7.94$, $SE = 0.45$ vs. dysphoric $M = 6.81$, $SE = 0.37$), $t(30) = 1.93$, $p = .06$. The three performance indices were not substantially correlated with the baseline-adjusted SBP reactivity scores (delta, delta 1, 2, and 3), $-.28 < r < -.05$, $ps > .12$. 
2.4.3 Discussion

The present results confirmed our central prediction that dysphoric participants would show stronger systolic reactivity when faced with a mental challenge without fixed performance standard than nondysphoric participants. As expected and in accordance with the MBM (Gendolla, 2000) statement that mood states per se do not have motivational implications, we found no differences in cardiovascular baseline measures between dysphoric and nondysphoric participants. As outlined earlier, systolic reactivity was our primary index for measuring effort intensity because SBP, which largely depends on myocardial contractility, is systematically influenced by sympathetic discharge to the heart (Brownley et al., 2000; Obrist, 1981; R. A. Wright, 1996).

Results actually revealed that dysphoric participants showed stronger SBP reactivity—reflecting the mobilization of more mental effort—than nondysphoric participants. Nevertheless, this difference proved to be significant only for the first of the three measures taken during task performance. For the other two measures as well as for the overall reactivity index results pointed into the same direction. In addition, there was a decline of SBP reactivity over time in the dysphoric group. One could conceive different explanations for this finding. For instance, given self-regulation difficulties in depression, it could be that our dysphoric participants mobilized much effort in the beginning but failed to maintain this level of effort over the 5-min period. As Tillema et al. (2001) pointed out, depressed individuals tend to set higher performance standards than nondepressed individuals. Possibly, participants in the dysphoric group had very high standards and reduced effort mobilization as they realized that they could not meet them.

Interestingly, performance quantified as the number of totally and correctly recalled letter series as well as the ratio between them described the opposite pattern of SBP reactivity during task performance. According to our results, nondysphoric participants clearly outperformed dysphoric participants. This is in accordance with results showing that depressed and dysphoric individuals tend to have cognitive deficits (e.g., Burt et al., 1995; Rogers et al., 2004). Finally, this study revealed highly significant differences between the dysphoric and nondysphoric groups with regard to their momentary mood state. As anticipated, participants who were classified as dysphoric on the basis of the CES-D 2 weeks prior to the experimental session reported being in a more negative mood than nondysphorics at the beginning of the experiment.

2.5 Study 2

This study was designed to replicate and extend the findings of Study 1. First, we replaced the memory task by a mental concentration task in order to address different cognitive processes and thus to facilitate generalization of our findings. In addition, following recommendations by Ingram and Siegle (2002) how to enhance the meaning of
findings from research with subclinical populations, we assessed self-reported dysphoria twice in order to ensure that the scores were stable. Only participants whose CES-D scores were located in the lower or upper third of the distribution at both the first (questionnaire session) and the second (experimental session) measurement time were retained for analyses. Moreover, we assessed participants’ momentary mood state not before but after task performance. This was supposed to prevent participants from relying too much on their momentary mood state by being forced to think about it, and thus to show that our findings are independent of being concerned with one’s momentary mood state. With some exceptions, we used the same materials and methods as in Study 1. In order to have an even better temporal resolution, cardiovascular values were taken every min.

2.5.1 Method

Participants
One-hundred seventy-one students in an introductory psychology course participated in questionnaire sessions in exchange for course credit. Out of this sample, we randomly selected women who scored in the lower or upper third of the CES-D for participation in an ostensibly unrelated experiment. As mentioned above, only data of participants whose scores remained within the limits set by the initial distribution (i.e., CES-D score ≤ 10 and ≥ 20, respectively) after the second assessment time were included in the analyses. Thus, data of 25 women (mean age 21 years) were retained and divided into 2 groups. Fourteen participants were referred to as dysphoric (CES-D [t1]: M = 26.93, SE = 0.96; CES-D [t2]: M = 25.43, SE = 1.08). Eleven participants were referred to as nondysphoric (CES-D [t1]: M = 5.64, SE = 0.97; CES-D [t2]: M = 4.73, SE = 0.99). The CES-D scales showed high internal consistency (αs = .93 at t1 and .94 at t2).11

Procedure
This study was conducted by means of the psychological software Inquisit (version 2.0, Millisecond Software) by an experimenter who was hired and unaware of both the hypotheses and participants’ depression status. The procedure was identical to Study 1 with the exception of the type of cognitive task, the moment of mood assessment, and the second questionnaire. We administered a computerized task that was adapted from the “d2 mental concentration test” (Brickenkamp, 1981). Participants learned that different symbols would appear on the computer screen and that they had to decide for each stimulus whether it was the letter d accompanied by exactly two apostrophes, or not (i.e., the letter d with more or less than two apostrophes or the letter p with one, two,

11 As in Study 1, scores of the BDI-II and the CES-D were highly correlated at both assessment times, \( r_s > .83, p_s < .001 \).
three, or four apostrophes) by pressing a “yes” or a “no” key. They were advised to work as quickly but also as precisely as possible. That means that we did not provide them with a fixed performance standard (e.g., a fixed presentation time of each stimulus) but simply asked them to do their best. It has also to be acknowledged that for this task participants had to move, that is, to press a button, in comparison with the quiet sitting in Study 1. Movement artifacts, however, were avoided by attaching the blood pressure cuff to the non-dominant arm. Before participants started the 5-min performance period, they performed 8 test trials to familiarize with the task. Then they worked for 5 min on this task while 5 cardiovascular measures were taken, starting 15 sec after task onset. Subsequently, participants’ momentary mood state was assessed with the UWIST scale. Finally, participants completed an ostensibly unrelated questionnaire (CES-D, BDI-II), were carefully debriefed, probed for suspicion, and received their course credit.

### 2.5.2 Results

#### Self-Reported Mood

As in Study 1, we calculated three scores based on the positive and negative hedonic tone adjectives of the UWIST scale (αs = .95 for the positive, .87 for the negative, and .93 for the global score). Results revealed that there were significant differences between dysphoric and nondysphoric participants with regard to their momentary mood state immediately after having worked on the cognitive task: Dysphorics \((M = 16.57, \ SE = 1.08)\) had lower positive scores than nondysphorics \((M = 21.09, \ SE = 1.16)\), \(t(23) = 2.83, p < .01\). Furthermore, dysphorics \((M = 12.21, \ SE = 1.31)\) showed higher negative scores than nondysphorics \((M = 6.18, \ SE = 0.69)\), \(t(19.18) = 4.07, p < .001\). Finally, they also differed significantly on the global sum score (dysphoric \(M = 36.36, \ SE = 2.29\) vs. nondysphoric \(M = 46.91, \ SE = 1.53\)), \(t(21.60) = 3.83, p < .001\).

#### Cardiovascular Baselines

SBP and DBP baseline scores were created by averaging the last three of the eight values obtained during habituation period (αs = .97 for SBP baselines and .96 for DBP baselines).\(^{12}\) HR baselines were determined by the last of the eight measures.\(^{13}\) Means and standard errors are presented in Table 4. According to independent-samples \(t\)-tests, there were no significant differences between the dysphoric and nondysphoric groups with regard to cardiovascular baseline measures \((ts < 1.33, ps > .20)\).

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\(^{12}\) The reason for this is that repeated measures ANOVAs revealed a decline of cardiovascular values over the first measures, whereas the last three measures remained stable \((ps > .16)\).

\(^{13}\) The last three measures of HR baselines were stable \((p > .50)\) but the internal consistency was insufficient \((\alpha = .44)\). Therefore, we used only the last of the eight measures as HR baseline. In addition, because of problems with the measurement equipment, there were some missing HR data, so that HR analyses are based on the data of 19 participants.
Table 4
Means and Standard Errors of Cardiovascular Baselines in Study 2

<table>
<thead>
<tr>
<th>Cardiovascular baselines</th>
<th>Dysphoric</th>
<th>Nondysphoric</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SE</td>
</tr>
<tr>
<td>SBP</td>
<td>100.73</td>
<td>2.12</td>
</tr>
<tr>
<td>DBP</td>
<td>64.33</td>
<td>1.47</td>
</tr>
<tr>
<td>HR</td>
<td>73.18</td>
<td>0.52</td>
</tr>
</tbody>
</table>

Note. N = 14 (dysphoric) and N = 11 (nondysphoric). SBP and DBP are indicated in millimeters of mercury (mmHg), HR is indicated in beats per minute (bpm).

Cardiovascular Reactivity

As in Study 1, we calculated for each participant both an overall change score (delta) and single change scores for each cardiovascular measure taken during task performance (delta 1, delta 2, delta 3, delta 4, and delta 5) (see Llabre et al., 1991). The latter scores were calculated for the sake of comparability of results between Studies 1 and 2. There were no significant correlations between SBP and DBP reactivity scores and their respective baseline scores, -.18 < r < -.15, ps > .40. In contrast, HR baseline scores were significantly correlated with HR reactivity scores, r(18) = -.58, p < .01. Therefore, we adjusted HR change scores with respect to HR baseline scores (see Llabre et al., 1991).

SBP. We first compared dysphoric and nondysphoric individuals with regard to their overall reactivity score (delta). As expected and depicted in the left panel of Figure 7, dysphoric participants (M = 8.72, SE = 2.00) showed stronger systolic reactivity than nondysphoric participants (M = 4.20, SE = 1.39), t(23) = 1.76, p < .05.10 We then submitted the single reactivity scores to a 2 (dysphoria) x 5 (time) mixed model ANOVA. In contrast to Study 1, there was no significant change over time neither for the total of the participants nor for either group, Fs < 1.89, ps > .11. Therefore, we did not further compare the single reactivity scores.
Experimental Part: Studies 1 and 2

Figure 7
Cell means and standard errors of overall systolic blood pressure reactivity (left panel) and heart rate reactivity (right panel) in Study 2. Cell means of heart rate reactivity scores are baseline-adjusted.

**DBP.** Comparison of the overall reactivity scores (delta) revealed that dysphoric participants ($M = 6.19, SE = 1.11$) tended to have stronger DBP reactivity than nondysphoric participants ($M = 3.70, SE = 0.97$), $t(23) = 1.63, p < .06$.\(^{10}\) We then submitted the single reactivity scores to a 2 (dysphoria) x 5 (time) mixed model ANOVA. It revealed a significant decline in DBP reactivity over time in general, $F(4, 92) = 3.98, p < .01$, but there was no dysphoria x time interaction, $F(4, 92) = 1.38, p > .24$. Hence we did not further compare the single reactivity scores.

**HR.** We first submitted the baseline-adjusted reactivity scores (delta) to an independent-samples $t$-test. Results revealed that dysphoric participants ($M = 1.22, SE = 0.74$) showed stronger HR reactivity than nondysphoric participants ($M = -0.56, SE = 0.42$), $t(17) = 1.90, p < .04$\(^{10}\) (see right panel of Figure 7). Next, we submitted the single baseline-adjusted reactivity scores to a 2 (dysphoria) x 5 (time) mixed model ANOVA. There was neither an effect for the time factor nor a dysphoria x time interaction, $Fs < 1.62, ps > .18$. Therefore, we did not further compare the single reactivity scores.

**Task Performance**

We registered the following indices of participants’ task performance: total number of stimuli a participant had performed, number of stimuli a participant had correctly identified, and the ratio between those indices. Moreover, we assessed the average response latency (in ms). Data of 2 participants were excluded form the analyses
because they differed more than two standard deviations from the respective group means. We then submitted the indices to independent-samples t-tests. Results revealed that there were no reliable differences, ts < 1, ps > .40 (average number of stimuli performed: $M = 371.65$, $SE = 8.18$; average number of correctly identified stimuli: $M = 364.04$, $SE = 7.94$; average ratio of correctly to totally performed stimuli: $M = 0.98$, $SE = 0.003$; average response latency: $M = 779.37$, $SE = 19.16$). The four performance indices were not correlated with SBP reactivity (delta), $-.02 < r < .11$, ps > .50.

### 2.5.3 Discussion

The present study replicated and extended the findings of Study 1. Again, the dysphoria groups did not differ with regard to their cardiovascular baseline values. But when they were confronted with a cognitive challenge that called for self-regulation, dysphoric participants showed significantly stronger systolic reactivity as well as marginally stronger diastolic reactivity than nondysphoric participants. Moreover, even if HR reactivity was generally rather low, dysphoric students showed significantly stronger HR reactivity than nondysphoric students. These findings hold true for the overall reactivity scores.

Unlike Study 1, there was no decline in systolic reactivity during performance in Study 2, neither for dysphoric nor for nondysphoric participants. One may argue that differences in task characteristics were responsible for dysphorics’ decline of SBP reactivity during performance of the memory task in Study 1 as opposed to the mental concentration task in the present study. The d2 task continuously presented new stimuli on the screen, asking for a reaction and thus creating a new situation for each trial. Consequently, it might have been easier to maintain self-regulation during work on the d2 task because this task was more strongly controlled by the “outside”—that is, the computer program that successively presented stimuli. In contrast, the screen with the 15 letter series in the memory task of Study 1 did not change over the 5-min performance period and thus the situation rested more or less the same.

With regard to task performance, the differences between both dysphoria groups in the present study were not reliable. As for the pattern of cardiovascular reactivity, we think that task characteristics were responsible for the differences in performance results between Studies 1 and 2. As we argued above, the memory task in Study 1 required more self-regulation skills than the concentration task in Study 2. In consideration of research stating that depressed and dysphoric individuals have self-regulation deficits (e.g., Strauman, 2002; Tomarken & Keener, 1998) and need specific task characteristics in order to control their directed attention (e.g., Hertel, 2000), it is reasonable to assume that the d2 task in the present study provided dysphoric participants with the required hints so that they performed on the same level as nondysphoric participants.
Finally, the mood assessment showed, in accordance with the results of Study 1, that individuals with low and high self-reported dysphoria differed highly significantly with regard to their momentary feeling state, even if the CES-D and the UWIST scales were assessed with a time lag of several weeks. Moreover, because mood was assessed after task performance at the end of the experimental session, the results of Study 2 demonstrate that the effects of dysphoria on cardiovascular reactivity and performance are not due to a high salience of one’s own feelings that might have been elicited by prior mood assessment.

2.6 General discussion

The main goal of our two studies was to test our prediction based on the MBM (Gendolla, 2000) that dysphoric individuals would show stronger effort-related cardiovascular reactivity than nondysphoric individuals when faced with a cognitive task without fixed performance standard. The results largely support our predictions. As expected, both studies found highly significant differences between dysphoric and nondysphoric participants’ momentary mood states—regardless if mood had been assessed before or after completion of the mental task. Undoubtedly, even students selected via a self-report depression scale several weeks before the experimental session differed clearly with regard to their naturally occurring momentary mood.

More important, Study 1 provided first evidence for our central hypothesis insofar as the dysphoric group showed significantly stronger systolic reactivity at the beginning of the memory task, demonstrating that dysphoric participants actually mobilized more mental effort. Study 2 confirmed our hypothesis for the overall reactivity scores not only for SBP but also for HR and marginally for DBP. Even if among these indices SBP is the most reliable and sensitive effort-related cardiovascular reactivity parameter, there is evidence that SBP, DBP, and HR can also respond simultaneously in active coping (Brownley et al., 2000; Obrist, 1981; Papillo & Shapiro, 1990; T. W. Smith et al., 1989). Accordingly, dysphoric students mobilized more mental effort during the mental concentration task than did nondysphoric students. The fact that this impact of dysphoria on cardiovascular reactivity holds true for two different types of cognitive tasks—a memory and a concentration task—makes us confident that the findings can be generalized.

The present results question the notion that depressed and dysphoric individuals have a motivational deficit with respect to intensity of motivation. According to our findings, motivational intensity is higher in dysphoric than in nondysphoric individuals for tasks without fixed performance standard. Correspondingly, our results replicate and extend the findings by Gendolla and colleagues (see Gendolla & Brinkmann, 2005, for a review), in that not only experimentally manipulated, transient mood states but also longer lasting affective dispositions that are related to the experience of certain moods
can have an impact on effort mobilization. Most important, this effect is not attributable to individual differences regarding cardiovascular baseline values because both dysphoria groups did not differ from one another on these measures.

Furthermore, the results of the present studies are compatible with research in depression and cardiovascular disease. Accordingly, depression and other negative affect dispositions are among the risk factors for the development and worsening of cardiovascular disease (e.g., Frasure-Smith & Lespérance, 2005a; Rugulies, 2002; Suls & Bunde, 2005). Our studies demonstrate that dysphoric individuals tend to mobilize more resources in terms of SBP reactivity when they are asked to do their best. A tendency to strong cardiovascular reactivity is in turn associated with and considered as a risk factor for the development of hypertension and cardiovascular disease (e.g., Kibler & Ma, 2004; Light et al., 1992). One may argue that, among other factors, also affective states play an important role in the linkage between depression and coronary heart disease—for instance because of stronger sympathetic activation induced by subjectively higher task demand in a (dispositionally) more negative mood (see Gendolla & Richter, 2005b).

Interestingly, the stronger resource mobilization of dysphorics was not related to performance gains. In Study 1, nondysphoric participants clearly performed better than dysphoric participants. In Study 2, where cardiovascular reactivity differences between both dysphoria groups were more obvious, no performance differences emerged. It seems that the cognitive deficit found in clinically and subclinically depressed individuals (e.g., Burt et al., 1995; Rogers et al., 2004) partly appears also in our dysphoric sample. In contrast to Gendolla and colleagues (e.g., Gendolla et al., 2001; Gendolla & Krüsken, 2002a, 2002b, 2002c) who found positive associations between SBP reactivity and performance when mood was experimentally manipulated, the dysphoric participants in our studies did not profit from mobilizing more resources. One can think of several interpretations for this finding.

On the one hand, given the mentioned cognitive deficits in depression and dysphoria, it may be that these individuals generally have to mobilize more resources in order to reach the same results than normal controls (see Hockey, 1997; R. A. Wright, 1998). In this light, the dysphoric participants in Study 2 successfully compensated their slight disadvantage by mobilizing more effort, which resulted in a performance that was comparable with those of nondysphorics. However, when dysphoric participants did not maintain effort—as it seems to be the case in Study 1—they attained worse outcomes. On the other hand, given that pronounced cognitive impairments are unlikely to occur in a student sample, the worse performance of dysphoric participants in Study 1 may also reflect their difficulties in self-regulation and attention control (e.g., Hertel, 2000; Kuhl & Helle, 1986; Strauman, 2002). This explication seems plausible when one considers the different task characteristics of the cognitive tasks used in these studies and fits well the
above mentioned evidence for motivational deficits being partly responsible for cognitive deficits. One could argue that cognitive deficits especially emerge in connection with a motivational deficit—what may explain the performance differences in Study 1 that did not emerge in Study 2.

The relatively weak association between performance and SBP reactivity in both studies is not surprising. There is evidence that in some circumstances effort may increase performance; previous research regarding the informational mood impact on effort mobilization, for instance, has found positive associations between performance and SBP reactivity (e.g., Gendolla et al., 2001; Gendolla & Krüsken, 2002a, 2002b, 2002c). Nevertheless, it would be incorrect to equate the two because performance on a task is an outcome determined by effort, ability, and strategy use, and different tasks are differentially influenced by these factors (Locke & Latham, 1990).

Finally, some open questions and shortcomings concerning the present research should be mentioned. First, it is an important issue to investigate the mechanisms that are responsible for the stronger cardiovascular reactivity of dysphoric individuals in self-regulation. According to the MBM (Gendolla, 2000), behavior-related judgments and evaluations should mediate between depressed mood and effort mobilization. As outlined above, dysphoria has been shown to have an effect on subjective evaluations and ratings (e.g., Scott & Ingram, 1998). Alternatively, dysphoria might have influenced people’s performance standards on this unfixed difficulty task, which led to stronger effort mobilization (see e.g., Sherry, Hewitt, Flett, & Harvey, 2003; Tillema et al., 2001, for research on perfectionistic standards in depression). Even if we are confident—based on prior research on experimentally manipulated mood (Gendolla & Krüsken, 2002b, 2002c)—that dysphoria affected the subjective difficulty of the task, future research should address these possible mediations between depressed mood and effort mobilization. Second, the issue regarding the here reported disconnection between effort and performance needs to be further addressed. Finally, one shortcoming of our studies is their relatively small sample size and their limitation to women. Future research may benefit from extending the studies to a broader population recruited from the community with more severe depressive symptomatology.
3. Studies 3 and 4

Does Depression Interfere With Effort Mobilization? Effects of Dysphoria and Task Difficulty on Cardiovascular Response

On the basis of predictions of the mood-behavior-model (Gendolla, 2000) and motivational intensity theory (Brehm & Self, 1989) two studies critically tested the common assumption that dysphoria is associated with a motivational deficit. Dysphoric and nondysphoric undergraduates performed a cognitive task that was either easy or difficult. Effort intensity (i.e., resource mobilization) was assessed as performance-related cardiovascular reactivity. In support of our predictions and in contrast to the popular view of a general motivational deficit, both studies found a crossover interaction between dysphoria and task difficulty: In the difficult condition, nondysphoric participants indeed showed stronger systolic blood pressure reactivity than dysphorics. But in the easy condition, dysphoric participants showed stronger systolic reactivity than nondysphorics. The findings are discussed with respect to motivational deficits in depression and possible underlying mechanisms.

Dysphoria and depression are said to affect motivation (e.g., Heckhausen, 1991)—a lack in motivation is even a diagnostic criterion of major depressive disorder as defined in the DSM-IV (American Psychiatric Association, 1994). Thus, it is not surprising that researchers have already investigated various aspects of the depression-motivation link—amongst others effects on behavioral approach and inhibition (Gray, 1982), self-regulation (e.g., Strauman, 2002), and action-relevant information processing (e.g., Hertel, 2000; Scott & Ingram, 1998). However, little is known about dysphoria effects on effort intensity—the mobilization of resources for attaining goals.

On the basis of the assumption that a central characteristic of dysphoria is a persisting negative mood (DSM-IV; American Psychiatric Association, 1994; see also Mineka et al., 1998) and on predictions of the mood-behavior-model (MBM; Gendolla, 2000) and motivational intensity theory (Brehm & Self, 1989), we conducted research that has shown that dysphoria directly influences resource mobilization in terms of cardiovascular reactivity for cognitive tasks. But in contrast to the popular view that dysphoria is associated with a general motivational deficit, dysphoric individuals mobilized more effort than...
Experimental Part: Studies 3 and 4

nondysphorics under “do-your-best” instructions (Brinkmann & Gendolla, 2007). This suggests that there is in fact no general motivational deficit but that dysphorics rather mobilize more effort than nondysphorics.

However, given that those studies used tasks without fixed performance standards, it remained open if this effect was a general one or if it was restricted to the specific performance condition asking to “do one’s best.” To come to clearer conclusions, we investigated dysphoric and nondysphoric individuals’ resource mobilization during the performance of tasks with fixed difficulty levels. On the basis of the MBM (Gendolla, 2000) and previous research on mood effects on effort mobilization (see Gendolla & Brinkmann, 2005; Richter et al., 2006, for reviews), we guided our studies by the idea that dysphoric individuals would mobilize less effort only in the context of difficult tasks but that dysphorics would mobilize even more effort than nondysphorics in the context of easy tasks. As in our previous studies, effort mobilization was quantified as the reactivity of the cardiovascular system during task performance (Gendolla & Brinkmann, 2005; R. A. Wright, 1996).

3.1 Dysphoria, Self-Regulation, and Motivation

Several researchers have stressed that self-regulation and motivation play important roles in the development, manifestation, and change of depression and dysphoria. Amongst others, a number of the identified cognitive deficits of depressed individuals (see e.g., Burt et al., 1995; Rogers et al., 2004, for reviews) might in fact be caused by motivational deficits (e.g., Abramson et al., 1981). Likewise, there is evidence that aspects of self-regulation problems, such as rumination (Hertel, 2000; Watkins & Brown, 2002), maintenance of unrealistic intentions (Kuhl & Helle, 1986), and deficits in attention control (Hertel, 2000; Hertel & Gerstle, 2003), play an important role in the relation of depression or dysphoria and cognitive malfunctioning. In addition, the motivational implications of negative feedback have a detrimental effect on subsequent performance. This effect has been shown to be especially pronounced in depression, compared to control as well as other clinical groups (Elliott et al., 1997).

With respect to the concept of approach and avoidance motivation, Strauman (2002) emphasized that depression is a disorder of self-regulation, which leads to affective and motivational deficits. In further support of their diminished approach motivation, depressed individuals have been shown to be less responsive to rewards (e.g., Henriques & Davidson, 2000) and have been characterized by a relative hypoactivation of the left prefrontal cortex—an area involved in approach-related behaviors (e.g., Davidson, Pizzagalli, Nitschke et al., 2002). Thus, it seems to be clear that dysphoria and depression are associated with impairments of a number of motivational and self-regulation functions. Whether depression is also linked to impairments in the mobilization of energy
resources is less clear. Carroll et al. (2007) found weak negative associations between depression and cardiovascular reactivity. However, from our perspective, a shortcoming of this study is the fact that task characteristics were not considered.

### 3.2 Effort Intensity and Cardiovascular Reactivity

Motivational intensity theory (Brehm & Self, 1989) has elaborated the “difficulty law of motivation” (Ach, 1935). Motivational intensity theory states that individuals mobilize resources proportionately to experienced task difficulty as long as success is possible and worthwhile. Consequently, fewer resources should be mobilized for an easy task than for a difficult task when the maximally justified effort is high. Disengagement occurs when task difficulty exceeds a person’s ability or when the necessary effort is not justified by the value of success.

R. A. Wright (1996) has integrated motivational intensity theory with Obrist’s (1981) active coping approach to cardiovascular arousal. Research instigated on the basis of this perspective has consistently found that resource mobilization can be reliably quantified by cardiovascular reactivity—changes in the activity of the cardiovascular system—in the context of task performance (see R. A. Wright & Kirby, 2001, for a review). Most significantly, performance-related changes in systolic blood pressure (SBP) follow the effort pattern predicted by motivational intensity theory, taking into consideration varying degrees of task difficulty and value of success (e.g., Bongard, 1995; Gendolla & Krüsken, 2002b; Light et al., 1992; Richter & Gendolla, 2006; T. W. Smith, Nealey, Kircher, & Limon, 1997). Evidence for diastolic blood pressure (DBP) and heart rate (HR) also exists but is less consistent (e.g., Storey et al., 1996). The high sensitivity of SBP to respond to resource mobilization is not surprising because SBP is systematically influenced by the sympathetic nervous system via myocardial contractility. By contrast, DBP mainly depends on flow resistance in the vasculature, which is only unsystematically influenced by sympathetic discharge. HR is influenced by both the sympathetic and the parasympathetic branches of the autonomic nervous system and should thus respond to resource mobilization only to the extent that sympathetic influences dominate (see Brownley et al., 2000; Obrist, 1981; Papillo & Shapiro, 1990).

### 3.3 Dysphoria, Mood, and Effort Intensity

To come to clear predictions about dysphoria effects on effort intensity, we elaborate on the fact that an enduring negative mood is one of the core characteristics of depression (DSM-IV; American Psychiatric Association, 1994; Mineka et al., 1998) and that both mood states and depression can influence judgments in a mood congruent way (see Scott & Ingram, 1998; Wyer et al., 1999, for reviews). The result is that more negative and pessimistic judgments are made in a negative mood or by depressed individuals.
and more positive and optimistic judgments are made in a positive mood or by non-depressed individuals.

According to the MBM (Gendolla, 2000), such mood congruency effects also occur on task-related appraisals during task performance, such as judgments of demand, difficulty, or ability. This results in higher perceived task demand in a negative mood than in a positive mood (e.g., Cunningham, 1988; Kavanagh & Bower, 1985; J. C. Wright & Mischel, 1982) and influences resource mobilization in compliance with the difficulty law of motivation (Ach, 1935). With reference to the effects of transient mood states, there is clear, replicated evidence that effort-related cardiovascular reactivity is stronger in a negative mood than in a positive mood when participants are asked to do their best and have to self-regulate the necessary resources (e.g., Gendolla et al., 2001; Gendolla & Krüsken, 2002c; Silvestrini & Gendolla, 2007).

Demand appraisals assessed immediately before performance—the best possible approximation for demand appraisals during performance—have been shown to mediate the effect of mood on cardiovascular response (Gendolla & Krüsken, 2002c). This supports the idea of an informational mood impact (i.e., that momentary mood functions as information for perceived task demand). Furthermore, the mood effects on both demand appraisals and cardiovascular reactivity disappear when mood’s informative value is called into question (Gendolla & Krüsken, 2002a).

When individuals face challenges with fixed performance standards, mood effects on effort-related cardiovascular response are moderated by task difficulty. This happens because people avoid wasting energy and consequently consider all available task-relevant information simultaneously in a pragmatic way when they make demand appraisals. Consequently, for an easy task, a negative mood leads to higher effort (relatively high subjective demand) than a positive mood (relatively low demand). By contrast, for a difficult task, a positive mood leads to higher effort (high, but still possible demand), but a negative mood leads to lower effort (disengagement because of too high demand; Gendolla & Krüsken, 2002b). Research within the same paradigm has shown, furthermore, that ability perception (R. A. Wright & Dill, 1993; R. A. Wright et al., 1997) and dispositional optimism (Kirby et al., 2003, cited in R. A. Wright & Franklin, 2004)—another dispositional variable that is associated with mood differences (Scheier & Carver, 1992)—combine with task difficulty to determine effort-related cardiovascular reactivity. Whether depression and dysphoria have the same effects is an open question.

From the perspective that depression and dysphoria are, among other symptoms, characterized by a persistent negative mood, we posit that dysphoria influences the mobilization of resources via an informational mood impact, as specified in the MBM (Gendolla, 2000). Consequently, we do not assume that dysphoria is associated with a general deficit in effort intensity. Rather, compared to nondysphorics, dysphoric individu-
als should mobilize even more resources for an easy task but fewer resources for a difficult task. This effect should occur because of higher subjective demand during performance of an easy task and the experience of too high demand during performance of a difficult task in dysphoria.

### 3.4 The Present Studies

In two quasi-experimental studies, we confronted dysphoric and nondysphoric individuals with tasks of fixed low versus high difficulty levels. If dysphoria is associated with a general motivational deficit, then dysphorics should mobilize fewer resources than nondysphorics on both difficulty levels. However, if our reasoning that the motivational effects of dysphoria are the result of an informational mood impact is correct, then task difficulty should moderate dysphoria effects on effort intensity. This should occur because the level of subjectively experienced demand during task performance depends on the joint effect of the two accessible types of information—mood and performance standard (e.g., Gendolla & Krüsken, 2002b). The resulting pattern of effort intensity is depicted in Figure 8.

![Figure 8](image-url)

**Figure 8**

Specifically, we predict a crossover interaction effect of dysphoria and task difficulty on effort-related cardiovascular response. In the context of an easy task, dysphoric individuals should show stronger cardiovascular reactivity, especially SBP (see above),
than nondysphoric individuals do. In the context of a difficult (but still possible) task, nondysphoric individuals should show stronger SBP reactivity than the dysphoric group. In order to be able to generalize our predicted crossover interaction pattern, we conducted two studies, each with a different type of cognitive challenge—a memory task and a mental concentration task.

3.5 Study 1: Mental Concentration Task

Participants were randomly assigned to the conditions of a 2 (dysphoric vs. nondysphoric) x 2 (task: easy vs. difficult) between-persons design, respecting the restriction of approximately equal cell distributions for dysphoric and nondysphoric participants as well as for men and women. Cardiovascular activity was assessed during a habituation period and during performance of the mental concentration task.

3.5.1 Method

Participants
One-hundred eighty-seven university students from an introductory psychology course participated in questionnaire sessions containing various personality questionnaires. By means of their scores on the Center for Epidemiologic Studies—Depression Scale (CES-D; Radloff, 1977), a self-report depression scale, we randomly selected students who scored in the lower and the upper quartile of the distributions of men and women (i.e., ≤ 6 or ≥ 20 for men and ≤ 13 or ≥ 20 for women) and invited them via an anonymous code to participate in an ostensibly unrelated experiment in exchange for course credit. The CES-D was assessed a second time in the context of the experimental session, and only students whose scores stayed within the limits set by the initial distribution were retained (see Ingram & Siegle, 2002; Weary et al., 1995). Therefore, a total of 53 participants (45 women, 8 men; average age 20 years) constituted our final sample. Twenty-six individuals (4 men and 22 women) scored in the lower quartile of the CES-D (Time 1 [t1]: \( M = 6.38, SE = 0.54 \); Time 2 [t2]: \( M = 6.31, SE = 0.73 \)) and were therefore referred to as nondysphoric. The remaining 27 students (4 men and 23 women) scored in the upper quartile of the CES-D (t1: \( M = 30.41, SE = 1.27 \); t2: \( M = 30.00, SE = 1.62 \)) and were therefore referred to as dysphoric.

Physiological Apparatus
Blood pressure measures were obtained noninvasively with a computer-aided monitor (Par Electronics Physioport III-S, Berlin, Germany) that uses oscillometry to determine SBP (in millimeters of mercury) and DBP (in millimeters of mercury). A blood pressure cuff (Boso, Bosch + Sohn, Jungingen, Germany) was placed over the brachial arteria above the elbow of participants’ nondominant arm and inflated automatically in 1-
min intervals during two measurement periods—habituation (baseline) and task performance.

Inter-beat intervals (IBIs) were assessed noninvasively using an electrocardiogram (ECG; Psylab System, Contact Precision Instruments, London, England) that continuously monitored the electrical activity of the heart muscle. Two Ag/AgCl 16 mm electrodes (Red Dot, 3M, Neuss, Germany) were placed on each participant: (a) one on the musculature between the neck and the distal end of the right collarbone, and (b) one on the left lateral abdomen below the lower rib cage (Lead II). Data were filtered using a 10 Hz high pass and a 40 Hz low pass filter and sampled at 500 Hz. The ECG signal was transferred to an internal R-wave detector (Psylab System, Contact Precision Instruments, London, England) that calculated IBIs.

The beginning and end of the two measurement periods (habituation, task performance) were synchronized via Transistor-Transistor Logic signals sent by the experimental software (Inquisit 2.0, Millisecond Software, Seattle, WA) through a parallel port. The ECG signal was assessed continuously; blood pressure measures were taken in 1-min intervals starting 15 s after the beginning of each measurement period. All obtained values were stored on computer disk so that both experimenter and participants were ignorant of all physiological values measured over the course of the experiment.

**Self-Report Data**

Dysphoria was assessed with two short self-report scales that have proven their applicability in depression research. They were administered about 4 weeks prior to the experiment and a second time directly after the experimental session. The French version of the CES-D (Fuhrer & Rouillon, 1989) consists of 20 items asking for frequency of depressive symptom experience on scales from 0 (never, very seldom) to 3 (frequently, always) and has been developed for community samples. In addition to the CES-D, we administered the French version of the revised Beck Depression Inventory (BDI-II; Beck et al., 1996)—a 21-item scale initially developed for clinical samples but frequently used in student samples as well. It asks for intensity of depressive experience during the past 2 weeks on scales from 0 (lowest intensity) to 3 (highest intensity). At both assessment times, the CES-D and the BDI-II showed high internal consistency (αs > .92) and were highly correlated with one another, rs(53) > .84, ps < .001.

At the beginning of the experimental session, the positive (i.e., “happy”, “joyful”, “contented”, “cheerful”) and the negative (i.e., “sad”, “frustrated”, depressed”, “dissatisfied”) hedonic tone scales of the UWIST mood adjective checklist (Matthews et al., 1990) assessed participants’ momentary mood. Participants indicated the extent to which each adjective reflected their momentary feeling state on 7-point likert scales ranging from 1 (not at all) to 7 (very much).
**Procedure**

The experimental session, which took about 30 min, was computerized using a personal computer and experimental software (Inquisit 2.0) that presented all instructions and stimuli. Participants attended individually. They were greeted by the experimenter—who was hired and unaware of both the hypotheses and participants’ dysphoria groups—and took a seat in front of a computer monitor. After participants had read introductory information and given signed consent, the experimenter attached the blood pressure cuff and the electrodes, left the room, and monitored the experiment from an outside control room. Participants first answered biographical questions and indicated their momentary feeling state on the UWIST scale. This was followed by a 9-min habituation period to determine cardiovascular baseline values while participants could read an old issue of a geographic magazine. After completion of the habituation period, the experimenter reentered the room, took the magazine, and asked participants to continue with the concentration task.

Instructions explained that participants would work on a concentration task for 5 min. Different stimuli would appear on the screen, preceded by a fixation cross (presented for 1,000 ms), and followed by a mask. As soon as the stimulus was erased by the mask, participants had to indicate within a 4,000-ms response time window whether the presented stimulus had been the letter “d” accompanied by exactly two apostrophes by pressing a “yes” or a “no” key on the keyboard. Distraction stimuli were the letter “d” with one, three, or four apostrophes and the letter “p” with one, two, three, or four apostrophes. Following each answer, participants received feedback before the new trial began. Task difficulty of this adapted version of the Brickenkamp (1981) d2 mental concentration task was manipulated by the presentation time of the stimuli. In the easy condition, stimuli were presented for 1,000 ms, but in the difficult condition, stimuli were presented for 160 ms—long enough to perceive the stimuli but short enough to make it difficult, albeit not impossible, to detect the difference, as indicated by pretests.

Task instructions were followed by 40 training trials to familiarize participants with the task and to give them an impression of task difficulty. Before starting the 5-min performance period, participants appraised task demand (i.e., perceived task difficulty, probability of success, and perceived own capacity) on 7-point scales ranging from 1 (very easy, not probable, and low capacity, respectively) to 7 (very difficult, very probable, and high capacity, respectively). Then the performance period—including the assessment of cardiovascular activity—started.

After the 5 min of task performance, the experimenter reentered the room, removed the blood pressure cuff and the electrodes, and informed participants that the experimental session was over but that she would like to ask them to complete another short paper-pencil questionnaire (CES-D, BDI-II), ostensibly for an unrelated question-
Experimental Part: Studies 3 and 4

3.5.2 Results

Self-Reported Mood

We calculated two mood scores by summing the scores of the positive hedonic tone adjectives of the UWIST scale (α = .96) and of the negative hedonic tone adjectives (α = .84). Two (dysphoria) x 2 (task difficulty) between-persons analyses of variance (ANOVAs) revealed only the anticipated highly significant dysphoria main effects, $F_s > 21.54, ps < .001, .304 < \eta^2 < .330$, in absence of any other main or interaction effects, $F_s < 2.06, ps > .15$. As expected, dysphoric participants had higher scores on the negative items (dysphoric $M = 11.78, SE = 0.87$ vs. nondysphoric $M = 6.19, SE = 0.70$) and lower scores on the positive items (dysphoric $M = 14.93, SE = 0.87$ vs. nondysphoric $M = 20.85, SE = 0.95$) compared to nondysphoric participants. Thus, dysphoric students were in a more negative mood during the experimental session.15

Demand Appraisals

A preliminary analysis revealed that the ratings of task difficulty, capacity, and success probability were highly intercorrelated. Therefore, we created a perceived task demand index by summing the ratings of task difficulty, reverse-coded own capacity, and reverse-coded success probability so that a higher score reflected higher perceived task demand (α = .84). Participants in the difficult-dysphoric group should appraise task demand as highest and participants in the easy-nondysphoric group should appraise demand as lowest, with the easy-dysphoric and the difficult-nondysphoric groups in between. We subjected perceived task demand to a 2 (dysphoria) x 2 (task difficulty) ANOVA. However, there were neither the expected main effects nor an interaction, $F_s < 1.03, ps > .31$. Cell means and standard errors were as follows: difficult-dysphoric ($M = 9.79, SE = 0.74$), easy-dysphoric ($M = 8.31, SE = 0.92$), difficult-nondysphoric ($M = 9.07, SE = 0.61$), and easy-nondysphoric ($M = 9.36, SE = 1.29$).

Cardiovascular Baselines

For the sake of comparability between the blood pressure measures and the continuously assessed IBIs, we integrated the ECG data over 1-min intervals. Then we calculated cardiovascular baseline scores for SBP, DBP, and HR by averaging the last four of the eight measures obtained during habituation (as were .91 for SBP, .94 for DBP, and

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15 We also formed a global mood score of positive and reverse-coded negative items (α = .91) that revealed lower scores for dysphoric participants ($M = 35.26, SE = 1.36$) than for nondysphoric participants ($M = 46.65, SE = 1.42$), $F(1, 49) = 33.57, p < .001, \eta^2 = .404$. The global score was correlated with the positive and negative scores, $r(53) = .90$ and $-.87, ps < .001$. The two subscales were also correlated, $r(53) = -.57, p < .001$. 
.99 for HR).\textsuperscript{16} Means and standard errors are depicted in Table 5. According to 2 (dysphoria) x 2 (task difficulty) ANOVAs,\textsuperscript{17} there were no differences between the experimental conditions for SBP and DBP baseline values $F_s < 1.14$, $p_s > .29$. An ANOVA for HR baseline values revealed a marginally reliable main effect of dysphoria, $F(1, 44) = 3.57$, $p = .07$, $\eta^2 = .072$, that was considered in an analysis of covariance (ANCOVA), which is presented below.

**Table 5**

**Means and Standard Errors of Cardiovascular Baselines in Study 1**

<table>
<thead>
<tr>
<th>Condition</th>
<th>$M$</th>
<th>$SE$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SBP</td>
<td>DBP</td>
</tr>
<tr>
<td>Easy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nondysphoric</td>
<td>105.55</td>
<td>66.66</td>
</tr>
<tr>
<td>Dysphoric</td>
<td>106.72</td>
<td>69.78</td>
</tr>
<tr>
<td>Difficult</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nondysphoric</td>
<td>103.57</td>
<td>67.85</td>
</tr>
<tr>
<td>Dysphoric</td>
<td>106.39</td>
<td>68.32</td>
</tr>
</tbody>
</table>

*Note.* Systolic blood pressure (SBP) and diastolic blood pressure (DBP) are shown in millimeters of mercury. Heart rate (HR) is shown in beats per minute.

**Cardiovascular Reactivity**

We created cardiovascular reactivity (delta) scores for each participant by subtracting the baseline values from the arithmetic means obtained during task performance (see Llabre et al., 1991). Preliminary 2 (dysphoria) x 2 (task difficulty) ANCOVAs that tested for associations between the reactivity scores and the respective baseline values found no significant covariations, $F_s < 0.71$, $p_s > .40$. Consequently, we analyzed the raw reactivity scores without baseline-correction.

\textsuperscript{16} For the sake of easier readability, we recoded the IBIs so that we will from now on refer to HR in beats per minute. The reason for averaging the last four baseline measures is that repeated measures ANOVAs revealed a decline of cardiovascular values over the first measures, but the last four measures remained stable ($p_s > .30$). HR data of some participants could not be analyzed because of too many movement artifacts. Therefore, analyses of HR data are based on 47 and 48 participants, respectively.

\textsuperscript{17} Due to the small number of men in each condition, it was not possible to look at gender as a separate factor in this study.
SBP. Cell means are depicted in Figure 9. In confirmation of our predictions, a 2 (dysphoria) x 2 (task difficulty) ANOVA revealed that the only significant effect was the expected crossover interaction between dysphoria and difficulty, $F(1, 49) = 5.37, p < .03, \eta^2 = .094$, in absence of reliable main effects of task difficulty, $F(1, 49) = 2.54, p > .12$, and dysphoria, $F(1, 49) = 1.00, p > .32$. Additional cell contrasts revealed a significant difference between the strong reactivity of dysphoric participants ($M = 5.73, SE = 1.25$) and the weak reactivity of nondysphoric participants ($M = 1.51, SE = 1.25$) in the easy condition, $t(49) = 2.24, p < .02$ (because of our clear theory-based directional predictions, focused contrasts are reported one-tailed). In the difficult condition, reactivity of dysphoric participants ($M = 4.81, SE = 1.33$) tended to be slightly lower than that of nondysphoric participants ($M = 6.49, SE = 1.19$), $t(49) = 0.98, p = .17$ (one-tailed).

**Figure 9**
Cell means and standard errors of systolic blood pressure (SBP) reactivity in Study 1.

**DBP and HR.** Means and standard errors are presented in Table 6. According to 2 (dysphoria) x 2 (task difficulty) ANOVAs, there were no significant main or interaction effects for either reactivity measure, $Fs < 1.07, ps > .30$, indicating that there were no reliable differences in DBP and HR reactivity between the four experimental groups.
Table 6
Means and Standard Errors of DBP and HR Reactivity in Study 1

<table>
<thead>
<tr>
<th>Condition</th>
<th>DBP M</th>
<th>HR M</th>
<th>DBP SE</th>
<th>HR SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Easy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nondysphoric</td>
<td>3.09</td>
<td>1.57</td>
<td>0.84</td>
<td>1.24</td>
</tr>
<tr>
<td>Dysphoric</td>
<td>4.19</td>
<td>0.88</td>
<td>1.63</td>
<td>1.29</td>
</tr>
<tr>
<td>Difficult</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nondysphoric</td>
<td>4.56</td>
<td>1.88</td>
<td>1.06</td>
<td>1.33</td>
</tr>
<tr>
<td>Dysphoric</td>
<td>4.73</td>
<td>0.05</td>
<td>0.89</td>
<td>0.89</td>
</tr>
</tbody>
</table>

Note. Diastolic blood pressure (DBP) is displayed as changes in millimeters of mercury. Heart rate (HR) is displayed as changes in beats per minute.

**Task Performance**

Raw performance indices like the total number of stimuli performed or the number of correctly and incorrectly identified stimuli were of limited interest because these indices were mainly determined by the greater number of stimuli performed in the difficult condition due to shorter presentation times, $F_s > 13.98$, $p < .001$, $\eta^2 < .869$. We therefore calculated the ratios of correctly identified, incorrectly identified, and omitted stimuli to the total number of stimuli performed. Furthermore, we registered the reaction times for the number of correctly and incorrectly identified stimuli as well as for the total number of stimuli performed.

Reaction times were normally distributed. Data of 1 participant were excluded from all analyses because the response latencies differed more than three standard deviations from the respective group mean. Two (dysphoria) x 2 (task difficulty) ANOVAs revealed task difficulty main effects, $F_s > 7.67$, $p < .01$, $\eta^2 < .259$, with respect to the following: ratio of incorrectly identified stimuli (easy $M = 0.01$, $SE = 0.01$ vs. difficult $M = 0.04$, $SE = 0.01$), reaction times of correctly identified stimuli (easy $M = 332.66$ ms, $SE = 29.07$ vs. difficult $M = 462.12$ ms, $SE = 14.56$), and total reaction times (easy $M = 334.56$ ms, $SE = 29.11$ vs. difficult $M = 461.94$ ms, $SE = 14.90$), in absence of other main or interaction effects, $F_s < 0.84$, $p > .36$. There were neither main nor interaction effects for the ratios of correctly identified and omitted stimuli, $F_s < 1.53$, $p > .22$. In summary, these results show better performance of participants in the easy task condition according to some indices, whereas dysphoria did not have a significant influ-
ence. Finally, we examined the relation of performance indices and SBP reactivity. Correlation coefficients ranged from $r = -0.24$ to $r = 0.22$, $p > 0.22$, indicating a weak tendency to better performance and faster reaction times (negative relation) associated with more effort.

### 3.5.3 Mediation Analysis

The results of cardiovascular reactivity display the predicted crossover interaction of dysphoria and task difficulty. In order to investigate the underlying mechanism of this effect, we examined the data with regard to mediation by momentary mood and perceived task demand as reported by Gendolla and Krüsken (2002c). Contrary to our expectation, positive and negative mood were not correlated with perceived task demand, $-0.10 < r(53) < -0.07$, $p > 0.48$. Then, we examined (separately for each task condition) the association of mood and perceived task demand with SBP reactivity. The correlations were generally weak, but the relation of negative mood to greater SBP change in the easy condition approached significance, $r(24) = 0.38$, $p < 0.10$ (two-tailed). As one would expect from those weak associations, mediation analyses performed separately for each task condition did not reveal evidence for mood and demand appraisals as mediators for dysphoria effects on SBP reactivity.

### 3.5.4 Discussion

The main result of this study was the predicted crossover interaction effect between dysphoria and task difficulty on SBP reactivity in absence of any significant main effects. This finding clearly shows that there was no general motivational deficit of the dysphoric participants with regard to resource mobilization. Rather, dysphorics mobilized even more resources than nondysphorics when they performed an easy version of the task.

We should nevertheless note that additionally performed focused cell comparisons found that the lower SBP reactivity of dysphoric participants in the difficult condition was not significantly different from the higher reactivity of nondysphorics. This may be attributed to a suboptimal difficulty manipulation. The fact that participants’ demand appraisals were below the scale’s midpoint (i.e., 11) also suggests that the difficult task was indeed not difficult enough for all dysphoric participants to withhold effort.

As outlined in the introduction, we reasoned that dysphoria would be associated with negative mood and related to a more pessimistic evaluation of task demand. Dysphoric and nondysphoric participants—as selected 4 weeks prior to the experimental session—indeed differed with regard to their naturally occurring mood when they performed the mental concentration task. In contrast, the task demand index assessed prior to
performance did not corroborate our assumption. Likewise, mood was not associated with perceived task demand and the proposed mediating mechanism not supported. The finding that DBP and HR reactivity were not influenced by dysphoria or task difficulty can be explained by the physiological mechanisms determining DBP and HR activity. As noted earlier, DBP and HR are less systematically influenced by the sympathetic nervous system than SBP. Therefore, DBP and HR can, but do not necessarily have to, show the same pattern as SBP (Brownley et al., 2000; Papillo & Shapiro, 1990). Finally, the analysis of task performance revealed better performance of participants in the easy task condition for some indices (smaller ratio of incorrectly identified stimuli as well as faster reaction times). Besides these rather trivial difficulty effects, no significant influence of dysphoria became evident.

3.6 Study 2: Memory Task

This study aimed to replicate and extend the findings of Study 1. In order to generalize dysphoria effects on effort intensity, we administered a memory task instead of the concentration task. Except for this, the same materials and procedure were used as in Study 1. In addition, we recruited more men for this study to have them better represented than in the previous study. Participants were again randomly assigned to the conditions of a 2 (dysphoric vs. nondysphoric) x 2 (task: easy vs. difficult) between-persons design respecting the restriction of equal cell distributions for dysphorics and nondysphorics as well as for men and women.

3.6.1 Method

Participants

Participants were selected out of a sample of 398 university students with various majors who had been recruited via announcements in introductory classes that were followed by short questionnaire sessions to assess dysphoria by means of the CES-D. Students scoring in the upper or lower quartile of the distributions of men and women, respectively, (i.e., ≤ 7 or ≥ 16 for men and ≤ 10 or ≥ 18 for women) were invited to participate in an ostensibly unrelated experiment via an anonymous code. For their participation in the experimental session, respondents received 10 Swiss francs (about $8). As in Study 1, only participants whose scores remained within the limits set by the initial distribution were retained for analyses. Consequently, a total of 64 participants (41 women, 23 men; average age 26 years) constituted our final sample. Thirty-two students scored in the lower quartile of the CES-D (t1: $M = 5.04, SE = 0.59$; t2: $M = 6.09, SE = 0.45$) and were therefore referred to as nondysphoric. The remaining 32 students scored in the upper quartile of the CES-D (t1: $M = 24.67, SE = 2.39$; t2: $M = 26.09, SE = 1.29$) and were therefore referred to as dysphoric. The distribution of men and women was
balanced in the four cells (11 women and 5 men in the difficult-nondysphoric cell, 10 women and 6 men in the other three cells).

**Procedure**

The experimental session was again computerized and accompanied by a hired experimenter who was unaware of both hypotheses and participants’ dysphoria status. Physiological apparatus and self-report measures (CES-D, BDI-II, and UWIST) were identical to Study 1. Both depression scales again showed high internal consistency (α > .93) and were highly correlated with one another, \( r(64) = .89, p < .001 \). Participants attended the experimental session individually, were greeted, and were seated in front of a computer monitor. After having provided signed consent, the blood pressure cuff and the electrodes were attached, and participants started reading the instructions and providing the answers to the biographical questions and the UWIST scale.

After an 8-min habituation period during which cardiovascular measures were taken in the same way as in the previous study, participants learned that they would perform a memory task for the next 5 min. They were asked to memorize a list of senseless letter strings, each string consisting of four letters (e.g., WPQA, CLTW) and to recall the entire list afterwards. The list comprised four strings in the *easy condition* and nine strings in the *difficult condition*. The exact number of presented strings had been pre-tested to adjust the difficulty level. After participants had learned about the forthcoming task, they were presented the list they would have to memorize for 2 s. This was supposed to give them an impression of task difficulty. Before starting to memorize, participants answered the same subjective demand questions as in Study 1 (i.e., perceived difficulty, probability, and capacity) on the same 7-point scales. Then the 5-min performance period started during which cardiovascular measures were taken. Subsequently, the experimenter reentered the room and gave participants a sheet of paper and a pencil so that they could note the letter strings they recalled. Afterwards, the experimenter removed the blood pressure cuff and the electrodes and asked participants to complete an ostensibly unrelated questionnaire (CES-D, BDI-II). Finally, participants were thanked, carefully debriefed, and given their payment.

**3.6.2 Results**

**Self-Reported Mood**

We again calculated a positive (α = .95) and a negative (α = .89) mood sum score and submitted them to 2 (dysphoria) × 2 (task difficulty) ANOVAs. The only significant results were the expected dysphoria main effects, \( F_s > 12.25, p_s < .001 \), .168 < \( \eta^2 < .222 \), in absence of other main or interaction effects, \( F_s < 1.06, p_s > .30 \). Accordingly, dysphoric participants had higher negative scores (dysphoric \( M = 11.19, SE = 1.09 \) vs. nondysphoric \( M = 6.22, SE = 0.46 \)) and lower positive scores (dysphoric \( M = 15.91, SE \)
= 0.84 vs. nondysphoric $M = 20.31, SE = 0.92$) compared to nondysphoric participants, indicating that dysphoric students were in a more negative mood.\textsuperscript{18}

**Demand Appraisals**

As in Study 1, we calculated a perceived task demand index ($\alpha = .80$) so that higher scores reflected higher perceived task demand. We then subjected the index to a 2 (dysphoria) x 2 (task difficulty) ANOVA. This revealed a significant dysphoria main effect, $F(1, 60) = 6.70, p < .02, \eta^2 = .098$, indicating that dysphoric participants appraised the task as more demanding than nondysphoric participants (dysphoric $M = 14.78, SE = 0.57$ vs. nondysphoric $M = 12.69, SE = 0.57$). Neither the task main effect nor the interaction proved to be reliable, $Fs < 0.94, ps > .33$. Nevertheless, cell means were in the hypothesized order: difficult-dysphoric ($M = 14.81, SE = 0.78$), easy-dysphoric ($M = 14.75, SE = 0.86$), difficult-nondysphoric ($M = 13.44, SE = 0.90$), and easy-nondysphoric ($M = 11.94, SE = 0.68$).

**Cardiovascular Baselines**

Again, we integrated the ECG data over 1-min intervals and calculated cardiovascular baseline scores for SBP, DBP, and HR by averaging the last three of the seven measures obtained during habituation (as were .95 for SBP, .94 for DBP, and .99 for HR).\textsuperscript{19} Means and standard errors are presented in Table 7. Preliminary 2 (dysphoria) x 2 (task difficulty) x 2 (gender) ANOVAs of the baseline values revealed a significant gender main effect for SBP, $F(1, 56) = 21.21, p < .001, \eta^2 = .258$, due to men having higher SBP baseline values ($M = 111.25, SE = 1.94$) than women ($M = 99.10, SE = 1.63$)—a common finding (Wolf et al., 1997). On the DBP baseline values, there was a significant task difficulty main effect, $F(1, 56) = 5.63, p < .03, \eta^2 = .077$, which was further qualified by a significant difficulty x gender interaction, $F(1, 56) = 4.50, p < .04, \eta^2 = .061$. Finally, there was a dysphoria main effect on the HR baseline values, $F(1, 51) = 4.17, p < .05, \eta^2 = .062$, indicating higher HR baseline values for nondysphoric ($M = 78.87, SE = 2.35$) than for dysphoric ($M = 70.87, SE = 2.32$) participants. These findings were considered in ANCOVAs of the cardiovascular reactivity scores, which are presented below.

\textsuperscript{18} The global mood score ($\alpha = .92$) revealed lower scores for dysphoric ($M = 36.72, SE = 1.77$) than for nondysphoric participants ($M = 46.09, SE = 1.15$), $F(1, 60) = 19.48, p < .001, \eta^2 = .242$. The global score was correlated with the positive and negative scores, $r(64) = .90$ and -.89, $ps < .001$. The two subscales were also correlated, $r(53) = -.60, p < .001$.

\textsuperscript{19} As in Study 1, we recoded the IBIs so that we refer to HR in beats per minute. The reason for averaging the last three baseline measures is that repeated measures ANOVAs revealed a decline of SBP and DBP values over the first measures, but the last four (SBP) and three (SBP and DBP) measures remained stable ($ps > .23$). There was no decline of HR values ($ps > .25$), but for the sake of comparability with SBP and DBP, we also averaged the last three baseline measures. Due to movement artifacts, there were HR data of only 59 participants available.
### Table 7
Means and Standard Errors of Cardiovascular Baselines in Study 2

<table>
<thead>
<tr>
<th>Condition</th>
<th>Nondysphoric</th>
<th>Dysphoric</th>
<th>Nondysphoric</th>
<th>Dysphoric</th>
</tr>
</thead>
<tbody>
<tr>
<td>Easy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>102.90</td>
<td>102.93</td>
<td>105.30</td>
<td>102.75</td>
</tr>
<tr>
<td>DBP</td>
<td>63.84</td>
<td>65.52</td>
<td>70.63</td>
<td>65.85</td>
</tr>
<tr>
<td>HR</td>
<td>76.47</td>
<td>73.81</td>
<td>81.27</td>
<td>67.73</td>
</tr>
<tr>
<td>SE SBP</td>
<td>2.64</td>
<td>2.66</td>
<td>2.69</td>
<td>3.68</td>
</tr>
<tr>
<td>SE DBP</td>
<td>1.46</td>
<td>1.59</td>
<td>1.78</td>
<td>2.79</td>
</tr>
<tr>
<td>SE HR</td>
<td>2.49</td>
<td>3.38</td>
<td>3.99</td>
<td>3.07</td>
</tr>
</tbody>
</table>

Note. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) are shown in millimeters of mercury. Heart rate (HR) is shown in beats per minute.

**Cardiovascular Reactivity**

We calculated cardiovascular reactivity (delta) scores in the same way as in Study 1. Preliminary 2 (dysphoria) x 2 (task difficulty) ANCOVAs on these reactivity scores with the respective baseline values as covariates revealed significant covariations for all cardiovascular indices, $F_s > 4.22$, $p_s < .05$, $< .061 < \eta^2 < .109$, $-.34 < r < -.22$. Because of this and because of the above mentioned differences in cardiovascular baseline values, we adjusted the SBP, DBP, and HR reactivity scores with regard to their respective baseline values in order to prevent carry-over effects and biases due to the law of initial values (see Llabre et al., 1991). Preliminary 2 (dysphoria) x 2 (task difficulty) x 2 (gender) ANOVAs of the baseline-adjusted SBP, DBP, and HR reactivity scores did not reveal any main or interaction effects for gender, $F_s < 2.58$, $p_s > .11$. Therefore, we did not consider gender in the following analyses.

**SBP.** A 2 (dysphoria) x 2 (task difficulty) ANOVA of the baseline-adjusted reactivity scores revealed only the predicted significant interaction effect, $F(1, 60) = 5.17$, $p < .03$, $\eta^2 = .077$, in absence of significant main effects of task difficulty, $F(1, 60) = 2.06$, $p > .15$, or dysphoria, $F(1, 60) = 0.06$, $p > .50$. As depicted in Figure 10, SBP reactivity clearly reflected the predicted crossover interaction pattern. Additionally, we conducted planned cell contrasts that confirmed a significant difference between nondysphoric individuals ($M = 8.58$, $SE = 1.62$) and dysphoric individuals ($M = 5.06$, $SE = 1.14$) in the difficult condition, $t(60) = 1.77$, $p < .05$ (one-tailed). In the easy condition, the reactivity...
of dysphoric participants ($M = 6.24, SE = 1.37$) tended to be higher than that of non-dysphoric participants ($M = 3.37, SE = 1.45$), $t(60) = 1.44, p < .08$ (one-tailed).

**Figure 10**
Cell means and standard errors of baseline-adjusted systolic blood pressure (SBP) reactivity in Study 2.

**DBP and HR.** Means and standard errors are presented in Table 8. A 2 (dysphoria) x 2 (task difficulty) ANOVA of the baseline-adjusted DBP reactivity scores revealed no significant effects, $F_{s}(1, 60) < 1.69, ps > .19$. A 2 (dysphoria) x 2 (task difficulty) ANOVA of the baseline-adjusted HR reactivity scores revealed a significant task difficulty main effect, $F(1, 55) = 5.79, p < .03, \eta^2 = .091$, due to higher reactivity in the difficult condition ($M = 6.24, SE = 0.98$) than in the easy condition ($M = 3.31, SE = 0.68$). There was neither a main effect of dysphoria, $F(1, 55) = 0.01, p > .50$, nor an interaction effect, $F(1, 55) = 2.57, p > .11$. 
Table 8
Means and Standard Errors of Baseline-Adjusted DBP and Baseline-Adjusted HR Reactivity in Study 2

<table>
<thead>
<tr>
<th>Condition</th>
<th>DBP</th>
<th>HR</th>
<th>DBP</th>
<th>HR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SE</td>
<td>M</td>
<td>SE</td>
</tr>
<tr>
<td>Easy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nondysphoric</td>
<td>5.12</td>
<td>1.01</td>
<td>4.26</td>
<td>0.83</td>
</tr>
<tr>
<td>Dysphoric</td>
<td>2.59</td>
<td>0.98</td>
<td>2.47</td>
<td>1.02</td>
</tr>
<tr>
<td>Difficult</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nondysphoric</td>
<td>5.03</td>
<td>0.93</td>
<td>5.12</td>
<td>1.43</td>
</tr>
<tr>
<td>Dysphoric</td>
<td>4.84</td>
<td>1.24</td>
<td>7.19</td>
<td>1.34</td>
</tr>
</tbody>
</table>

Note. Diastolic blood pressure (DBP) is displayed as changes in millimeters of mercury. Heart rate (HR) is displayed as changes in beats per minute.

**Task Performance**

Raw task performance indices of the memory task (e.g., the number of correctly or overall recalled letter strings) were of limited interest because they were mainly restricted by task difficulty and thus only showed difficulty main effects, $F$s(1, 60) > 16.34, $p$ < .001, .213 < $\eta^2$ < .642. Therefore, the primary performance indices were the percentages (a) of overall recalled to presented strings and (b) of correctly recalled to presented strings. Two (dysphoria) x 2 (task difficulty) ANOVAs of these difficulty-adjusted indices also revealed significant task difficulty main effects, $F$s > 16.20, $p$ < .001, .211 < $\eta^2$ < .282, in absence of any other effects, $F$s < 0.56, $p$ > .46. On average, participants in the easy condition overall recalled 100% of the presented strings, whereas participants in the difficult condition overall recalled 82% of the presented strings. Compared to the number of presented strings in each condition, participants in the easy condition correctly recalled 88%, whereas participants in the difficult condition correctly recalled 61%. Finally, correlations of performance indices and SBP reactivity ranged from $r$ = .06 to $r$ = .33, $p$ > .06, indicating again a tendency to better performance associated with more effort.

**3.6.3 Mediation Analysis**

As in Study 1, we examined the associations of momentary mood, perceived task demand, and SBP reactivity. In contrast to Study 1 and according to expectations, positive mood correlated negatively with perceived task demand, $r$(64) = -.25, $p$ < .05 (two-
tailed), and negative mood tended to correlate positively with perceived task demand, \( r(64) = .24, p < .06 \) (two-tailed). However, the indirect effect (see Sobel, 1982) of dysphoria on perceived task demand via positive and negative mood was not reliable, \( z < 1.02, ps > .30 \). Negative mood and perceived task demand tended to be associated with SBP reactivity in both task conditions, in the appropriate direction, though only one of the four correlations approached significance (greater demand appraisal with less SBP change in the difficult condition). Mediation analyses performed separately for each task condition revealed no evidence for the mediation of dysphoria effects on SBP reactivity by mood and demand appraisals.

### 3.6.4 Discussion

The findings of Study 2 conceptually replicate and complement those of Study 1. According to the self-report measures, participants who were assigned to the dysphoric and nondysphoric groups on the basis of their depression scores 3 weeks prior to the experimental session differed clearly with regard to their naturally occurring mood during the experiment. As it is the most relevant result of this study, the predicted crossover interaction pattern of dysphoria and task difficulty on SBP reactivity again became evident in absence of any significant main effects. In the difficult condition, systolic reactivity was stronger for nondysphoric than for dysphoric individuals, but in the easy condition, the pattern was reversed. Here, reactivity tended to be stronger for dysphorics than for nondysphorics. As in Study 1, the effects on DBP and HR reactivity did not correspond to those of SBP, which is, however, not surprising given their less systematic linkage to sympathetic arousal (Brownley et al., 2000; Papillo & Shapiro, 1990).

Unlike Study 1, results on the perceived task demand index assessed prior to performance confirmed that dysphoric participants evaluated task demand higher than nondysphoric participants. Moreover, participants in the difficult-dysphoric group appraised the task the most demanding and participants in the easy-nondysphoric group appraised it the least demanding. This displays the expected order, even though the task difficulty main effect was not reliable. However, the mediation of dysphoria effects on effort mobilization via perceived task demand as reported by Gendolla and Krüsken (2002c) was not corroborated. Finally, as in the previous study, participants’ task performance was not influenced by dysphoria but only by task difficulty, with participants in the easy condition performing better than participants in the difficult condition.

### 3.7 Meta-Analysis of SBP Reactivity in Studies 1 and 2

Both experiments found the predicted crossover interaction effect of dysphoria and task difficulty on SBP reactivity in absence of significant main effects. However, as reported above, additional cell comparisons revealed a significant difference between the
easy-dysphoric and easy-nondysphoric cells in Study 1 but no significant difference between the difficult-nondysphoric and difficult-dysphoric cells. In Study 2, dysphoria had a significant effect in the difficult condition but only a marginally significant effect in the easy condition. Therefore, we additionally tested our predictions with an analysis that combines the two studies using the “Stouffer” or “adding z-method” described by Rosenthal (1978). It has to be acknowledged, however, that the two cognitive tasks were not identical, and their difficulty levels were not exactly corresponding.

The adding z-method consists of calculating cumulative z scores by (a) converting the one-tailed $p$ level of each cell comparison to its associated $z$ score, (b) summing the $z$ scores, and (c) dividing the sum by the square root of the number of studies being combined. The results confirmed the predicted cell differences: In the easy condition, dysphoric participants had higher SBP reactivity than nondysphoric participants, $z = 2.57, p < .01$ (one-tailed). In the difficult condition, nondysphoric participants showed higher SBP reactivity than dysphoric participants, $z = 1.88, p < .04$ (one-tailed). Together with the replicated significant crossover interaction in absence of significant main effects, this indicates that dysphoria only leads to less effort mobilization when a task is difficult. But dysphoria results in the mobilization of high effort when a task is easy.

### 3.8 General Discussion

According to the present results, dysphoria does not lead to a general deficit in resource mobilization. Rather, it depends on the task context whether dysphoria reduces or even boosts effort intensity. As indicated by the two studies and additionally confirmed by the combined analysis, SBP reactivity—our central dependent variable referring to resource mobilization—displayed the predicted crossover interaction effect of dysphoria and task difficulty in absence of significant main effects. When task difficulty was high, nondysphorics showed higher performance-related SBP reactivity than dysphorics, which is in accordance with the idea of a motivational deficit in dysphoria. But when task difficulty was low, dysphoric participants showed higher SBP reactivity than nondysphorics, which is the very opposite of a motivational deficit. Accordingly, dysphoria can increase effort intensity when task difficulty is low, as shown in the present studies, or unfixed (i.e., individuals are asked to “do their best” instead of attaining a defined performance standard), as has been shown in previous studies (Brinkmann & Gendolla, 2007).

Regarding our research sample, it is of note that our studies involved groups of dysphoric students with CES-D scores from 16 up to 51, averaging at 30 (Study 1) and 25 (Study 2), respectively. This is beyond the usual cut-off score of 16 (Radloff, 1977) and thus makes us confident that these samples represent cases of dysphoria with—at

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20 Other methods summarized by Rosenthal (1978) like the “method of adding probabilities”, the “method of adding ts”, or the “method of adding logs” revealed virtually the same results.
least—subclinical symptoms of depression. Of course, future research may benefit from including clinical measures (e.g., structured clinical interviews) and clinical samples.

The finding that dysphoria can boost effort intensity in the context of easy and unfixed challenges confirmed our predictions derived from the notion that a central characteristic of dysphoria and depression is an enduring negative mood (DSM-IV; American Psychiatric Association, 1994) and from theorizing about the role of mood states in resource mobilization in terms of the MBM (Gendolla, 2000). In support of this reasoning, both studies found that dysphoric and nondysphoric individuals differed significantly with respect to their momentary mood state—dysphorics’ mood was much more negative than nondysphorics’ mood.

With respect to perceived task demand, Study 2 found the expected dysphoria main effect—that was paralleled by the correlation of mood and perceived task demand—as well as an increase in demand appraisals from the easy-nondysphoric to the difficult-dysphoric condition, even though the task difficulty main effect was not reliable. One plausible reason for the different results in Study 1 is that those participants had the opportunity to practice on the concentration task before making their judgments, but participants in Study 2 did not. Therefore, individuals in Study 2 had to rely much more on other information than task difficulty—for instance their momentary mood state. This explanation is in accordance with the MBM-postulate that the effective weight of moods in evaluations and judgments depends on the amount of other accessible, diagnostic information (Abele et al., 1998; Abele & Petzold, 1994; Gendolla, 2000). Moreover, given that appraisals are considered to be explicit as well as implicit (Kappas, 2006), the absence of an effect on the three self-report questions in Study 1 does not exclude the possibility that implicit difficulty appraisals influenced the mobilization of resources. In this context, it has to be pointed out that experienced task demand during rather than before performance is thought to be the critical variable, which is, however, difficult to assess.

According to the MBM logic, mood states should mediate dysphoria effects on demand appraisals and effort mobilization, and demand appraisals should mediate dysphoria effects on effort mobilization. The present studies did not find evidence for this assumption reported by Gendolla and Krüsken (2002c): In Study 1, demand appraisals did not form the expected pattern. In Study 2, demand appraisals and negative mood—but not positive mood—tended to show the expected associations with SBP reactivity. These correlations were, however, weak, and the indirect effects were not reliable.

Thus, at this time, we cannot tell whether an informational impact of momentary mood was the mechanism underlying the dysphoria effects on resource mobilization. On the one hand, our experimental design with a maximum of 16 participants per cell and restricted variance within the conditions does not provide adequate statistical power for
examining a causal chain with three to four variables. Therefore, it might be that the weak evidence of Study 2 comes out more clearly in a design with more statistical power. In favor of this assumption is evidence concerning not only mood congruency effects on evaluations and judgments in depression and dysphoria (Scott & Ingram, 1998) but also mood effects on perceived task demand and effort mobilization in studies within the same experimental design as the present ones (Gendolla & Krüsken, 2002b, 2002c). On the other hand, it might be that naturally occurring dysphoria differs from manipulated transient mood states in some respects: Even though dysphoria has been shown to have similar effects on effort mobilization, the principal mechanism might be something other than via momentary mood and demand appraisals—or at least the consciously experienced and verbalized parts of them. Future researchers interested in dysphoria and depression effects on cardiovascular reactivity should investigate the underlying mechanism, maybe using a stronger or even more obvious manipulation of objective task difficulty in order to achieve a stronger impact on demand appraisals. Future researchers might furthermore manipulate more than two levels of task difficulty to examine a broader range of the predictions depicted in Figure 8.

A finding of the present studies that also deserves discussion is that task performance was affected only by task difficulty—which is a rather trivial effect—whereas dysphoria had no significant impact. Furthermore, effort was weakly or not related to performance in both studies and both difficulty conditions. Previous research, on the one hand, has shown positive associations between performance and SBP reactivity and has thus provided evidence that, in some circumstances, effort may increase performance (Gendolla et al., 2001; Gendolla & Krüsken, 2002a, 2002b, 2002c). On the other hand, the vast body of research reporting cognitive deficits in clinical and subclinical depression, including impairments in the domains of attention, memory, and executive functions (see Burt et al., 1995; Rogers et al., 2004, for reviews), suggests diminished performance of dysphoric participants, especially in the difficult condition where they mobilized less effort.

We attribute the absence of such performance effects in the present studies to the fact that participants were university students who are unlikely to suffer from massive cognitive deficits. Moreover, as we did not focus on cognitive deficits in dysphoria, we selected tasks that met our criteria of simplicity and minimal physical demand rather than tests specially designed to reveal cognitive impairments. At this point, it is also of note that performance and effort are complex constructs and that it would be incorrect to equate the two; performance on a task is an outcome that is determined by effort, ability, and strategy use, and different tasks are differentially influenced by these factors (Locke & Latham, 1990). Furthermore, task performance only reflects the effectiveness of behavior but not its efficiency, which is the relationship between outcome and invested
effort (see Eysenck, Derakshan, Santos, & Calvo, 2007). The absence of dysphoria effects on performance in our studies might thus represent a case of successful effort mobilization (see Hockey, 1997).

In summary, the present research demonstrates that dysphoria influences resource mobilization but does so with dependence on task characteristics. In accordance with R. A. Wright (1996), we regard resource or effort mobilization as a direct indicator of one specific aspect of motivation, namely the intensity of motivation at a given point in time (Brehm & Self, 1989). This means that sympathetically mediated cardiovascular reactivity displays the mobilization of resources to actively cope with demands. In this sense, we conclude from our studies that dysphoria and depression are not necessarily associated with a general motivational deficit. We have shown that dysphoria can even lead to increased effort intensity—and thus to enhanced motivational intensity—given the specific task characteristics of an easy task or a task without fixed performance standards as shown in the studies by Brinkmann and Gendolla (2007).

To be clear, our studies were not concerned with a problem specifically prominent in depression, namely the initiation of actions. However, under the condition that behavior has been instigated, our research demonstrates that measures of effort mobilization may complement and possibly also explain performance measures. There are instances when dysphoric individuals are willing to mobilize much effort and others when they withhold effort, which might also have an impact on performance. Moreover, this sheds light on a pattern of task engagement that may be maladaptive: High engagement at a point that does not call for much effort but disengagement at a point where it would not be necessary to withhold effort. On the other hand, this implies that reduced daily demands (i.e., easy tasks but also do-your-best tasks) can lead to high task engagement—an experience from which dysphoric individuals ultimately may benefit in terms of experienced efficacy.
DISCUSSION
1. Summary of the Results

In this part, we first summarize the results, which have been described in more detail in the preceding experimental part, with reference to the six hypotheses formulated before. Afterwards, we discuss these results and integrate them with respect to the main research question concerning motivational issues in depression and dysphoria. We conclude with an outlook on clinical and health-related implications of our results. In the last section, we delineate limitations of the present and suggestions for future research.

1.1 Mood

Our research question was chiefly grounded on the assumption that the dysphoric students would be in a more negative mood than the nondysphoric students. All four studies clearly confirmed this first hypothesis—on the global mood score as well as on the separately analyzed positive and negative scales of the UWIST mood adjective checklist (Matthews et al., 1990). It should be noted that several weeks passed between the trait measure of depressive experiences by means of the CES-D and the assessment of momentary mood state at the beginning of the respective experiment. This corroborates the persisting nature of depressed mood. In Study 2, we assessed mood after the cognitive task. This confirmed that cardiovascular effects were not influenced by prior measurement that might elicit heightened salience of momentary mood.

1.2 Demand Appraisals

According to the logic of the MBM (Gendolla, 2000), mood has an informational impact on task-related appraisals of difficulty, demand, ability, and so forth. Such demand appraisals during task performance are considered to mediate mood effects on effort mobilization—that is, on cardiovascular response. Studies 1 and 2 did not assess these variables. In Studies 3 and 4, task demand appraisals were measured after task instructions but prior to task performance by means of self-reports that were merged to a perceived task demand index. In those studies, we reasoned that both available sources of information (i.e., mood state and task difficulty) would have a joint effect on perceived task demand. As pointed out in Hypothesis 5, we expected task demand to be the highest in the difficult-dysphoric group and the lowest in the easy-nondysphoric group with the two other groups in between. Study 3 did not reveal any differences in demand appraisals between the four experimental groups. Study 4, in contrast, showed a significant main effect of dysphoria, confirming that dysphoric individuals perceived the task as more difficult and themselves as less capable. Even though the task difficulty main effect was not reliable, cell means were in the expected order. However, mediation
analyses did not corroborate statistical mediation of dysphoria effects on SBP reactivity via perceived task demand.

As discussed in chapter 3.8 of the experimental part, differences in the possibility to exercise oneself in the task before making the judgments are most probably responsible for the null effect in Study 3. Especially with respect to the nonsignificant mediation analyses, we have to point at the fact that the number of participants in each cell was rather small and adapted to reveal the more prominent effects on cardiovascular reactivity. This means that our studies presumably lacked sufficient statistical power to detect the more subtle effects on self-report measures. In addition, it has to be underlined that self-report questions are subject to certain problems including the tendency to answer them in a social desirable and self-defensive manner (e.g., Pyszczynski & Greenberg, 1983; Rhodewalt & Fairfield, 1991). In summary, the present series of studies can provide only preliminary evidence for the influence of dysphoria on ability perception.

1.3 Cardiovascular Baseline Measures

As outlined in the theoretical part (chapter 3.1), there are few conclusive results with respect to differences in cardiovascular baseline activity in depression or dysphoria compared to nondepressed or nondysphoric individuals. Most of the studies and theorizing focuses on cardiovascular reactivity to various challenges (see e.g., Carroll et al., 2007; Kibler & Ma, 2004). However, Light et al. (1998) report evidence for dysphoric individuals’ enhanced cardiovascular activity under resting conditions.

In contrast to this latter study, but in accordance with Hypothesis 2, dysphoric participants in our four studies did not have higher SBP or DBP baseline values than nondysphoric participants. Study 4 found that nondysphoric participants had higher HR baseline values than dysphoric participants. But as this result only emerged in one of four studies, it should not be over-interpreted or generalized. Study 4—the only one that included enough men to analyze gender differences—revealed higher SBP baseline values for men than for women as well as an interaction effect of gender and task difficulty for DBP baseline values. Gender differences in cardiovascular measures are, however, a common finding (Carroll et al., 2007; Wolf et al., 1997).

Taken together, the present four studies indicate that there are no differences in cardiovascular baseline activity between dysphoric and nondysphoric individuals—at least in subclinical and relatively young samples like ours. This finding is important in light of the differences in cardiovascular response to various behavioral challenges that we discuss in the following.
1.4 Systolic Blood Pressure Reactivity

SBP response to the cognitive tasks corroborated our predictions in all four studies. Specifically, confirming Hypothesis 3, Studies 1 and 2 revealed stronger SBP reactivity of dysphoric participants compared to nondysphoric participants. In those studies, no performance standards were given and participants were asked to do their best, that is, to memorize as many letter strings as possible or to work as precisely and quickly as possible on the d2 task. It has to be acknowledged, however, that in Study 1 this effect emerged only on the first of three reactivity measures. We interpreted this in terms of self-regulation difficulties in dysphoria. Presumably, dysphoric participants failed to maintain effort mobilization over the 5-min performance period and disengaged. This interpretation seems reasonable given the differences in task characteristics between the d2 task that continuously re-attracts attention to each new stimulus and the more static memory task with a long list of letter strings that does not change over the 5-min performance period.

In Studies 3 and 4, participants were confronted with an easy or a moderately difficult performance standard. Confirming Hypothesis 4, these studies revealed the expected crossover interaction pattern. This means that dysphoric participants had stronger SBP reactivity than nondysphoric participants under easy task conditions but lower SBP reactivity under difficult task conditions. Single comparisons revealed reliable differences between the cells, except for the difficult condition in Study 3 and a marginal effect in the easy condition in Study 4. We suspected that the difficult task condition of Study 3 had not been difficult enough for dysphoric participants to completely withhold effort as intended. Therefore, we provided a more difficult standard in Study 4—even though it was about a different type of task. An interesting result of these two studies is that the crossover interaction pattern is mainly caused by the nondysphoric participants who had significantly higher SBP reactivity in the difficult than in the easy task conditions. However, in both studies, SBP reactivity of dysphoric participants did not differ between the easy and the difficult task conditions. We discuss this finding in more detail in the following (see chapter 2.3).

The fact that SBP reactivity very well reflected our hypotheses about effort mobilization is not surprising. As outlined in the theoretical part, SBP reactivity mainly depends on myocardial contractility and TPR. TPR is not systematically linked to the activation of the SNS but myocardial contractility is determined by sympathetic discharge to the heart. Therefore, SBP reactivity represents an indicator of beta-adrenergically mediated SNS activity (see Brownley et al., 2000; Levick, 2003; Papillo & Shapiro, 1990). Sympathetic discharge to the heart, in turn, is considered to correspond to experienced task demand in active coping (Obrist, 1981). Therefore, we regard SBP reactivity as an indicator of
effort mobilization and thus as an indicator of the intensity of motivation at a given point in time (see Brehm & Self, 1989; R. A. Wright, 1996).

### 1.5 Diastolic Blood Pressure and Heart Rate Reactivity

The pattern of DBP and HR reactivity did not consistently correspond to the pattern of SBP reactivity and thus adds to the mixed evidence of those cardiovascular measures with respect to effort mobilization (see chapters 3.4 and 3.5 in the theoretical part). HR is influenced by both the sympathetic and the parasympathetic branches of the autonomic nervous system and can reflect SNS activation only to the extent that this influence is not masked by the inhibitory effect of the PNS. Studies 1 and 3 did not show any HR differences. In contrast, Study 2 revealed stronger HR reactivity for dysphoric participants; and Study 4 revealed stronger HR reactivity in the difficult task condition independent of dysphoria. HR reactivity in Study 2 mirrored SBP reactivity so that it seems plausible to conclude that HR reactivity reflected effort mobilization in that study. In Study 4, the higher HR reactivity in the difficult condition mirrored to some extent the nonsignificant trend toward higher SBP reactivity in the difficult condition. Even though HR failed to show an interaction effect and thus did not perfectly mirror SBP reactivity, we interpret the HR pattern in terms of effort mobilization: Participants in the difficult condition mobilized more effort than in the easy condition, qualified by the influence of dysphoria.

DBP mainly depends on the resistance in the vasculature, that is, TPR. Because SNS activation leads to vasoconstriction in some and vasodilation in other blood vessels, TPR—and thus DBP—are only unsystematically influenced by sympathetic activation. In accordance with that, only Study 2 revealed a nonsignificant trend toward higher DBP reactivity of dysphoric participants. As this trend mirrors SBP reactivity, it represents one of the rather rare cases when DBP reactivity may be interpreted in terms of effort mobilization. On the other hand, given the null effects on DBP reactivity in the other three studies, this trend should not be over-interpreted.

### 1.6 Performance

In our four studies, we applied two different kinds of cognitive tasks in order to generalize the findings of cardiovascular reactivity across domains. Specifically, Studies 1 and 4 employed a memory task consisting of memorizing a list of senseless letter strings. Studies 2 and 3, in contrast, employed a modified version of the d2 task by Brickenkamp (1981) that calls for attention and concentration on the presentation of different stimuli. Therefore, as discussed above, both tasks differed with respect to required self-regulation abilities and the possibility to exercise oneself in the task before performing. However, both tasks had been selected with the aim to present simple tasks that are not
physically demanding. This allows attributing effort mobilization to mental and not to physical demand.

With respect to previous studies about mood influences on cardiovascular reactivity within the same paradigm (Gendolla et al., 2001; Gendolla & Krüsken, 2002a, 2002b, 2002c), one could expect a positive relation between task performance and effort mobilization. On the other hand, in light of the vast body of research showing cognitive deficits in depression and dysphoria (see chapter 2.2.2 in the theoretical part), one could expect better performance of nondysphoric participants. Consequently, we had not formulated a priori hypotheses about performance results in our studies.

Results uncovered an effect of dysphoria on task performance only in Study 1. Here, in accordance with memory deficits in depression, dysphoric participants performed worse than nondysphorics. Because our memory task mainly asked for controlled processes in the memorization and free recall of senseless letter strings, this result fits well in the evidence that especially controlled processes are impaired in depression and dysphoria, whereas automatic processes are often unaffected (Hartlage et al., 1993; Hertel, 1998; Jermann et al., 2005). In contrast, Study 2 did not find performance differences; and Studies 3 and 4 only revealed difficulty main effects inasmuch as participants in the easy task conditions performed better than participants in the difficult task conditions. When interpreting these effects, it has to be considered that the tasks had not been selected for being particularly sensitive to dysphoria effects on cognitive performance.

In general, correlations between performance indices and SBP reactivity did not indicate substantial associations. Nevertheless, correlation coefficients in Study 1 indicated a trend toward a negative association between performance and effort, as one would expect from the SBP and performance results (i.e., higher SBP reactivity but worse performance in dysphoria) reported above. In contrast, correlation coefficients in Study 4 showed a trend toward a positive association between performance and effort in the difficult condition. This fits in the finding that in the difficult condition nondysphoric participants mobilized more effort than dysphoric participants. Therefore, both studies using memory tasks indicate worse performance of dysphoric participants—despite high effort mobilization in Study 1. This worse performance of dysphoric participants corresponds to the general evidence for memory deficits in depression and dysphoria, especially for controlled processes (e.g., Burt et al., 1995; Hartlage et al., 1993; Jermann et al., 2005). However, it is of note that the correlations were not substantial and that the other two studies using the concentration task did not find associations between effort and performance.
2. Discussion

The main conclusion that we draw form the results summarized on the preceding pages is a confirmation of our central hypothesis: Dysphoria is not necessarily associated with a motivational deficit. Rather, it depends on task characteristics whether dysphoria leads to decreased or increased effort mobilization. This conclusion is chiefly based on R. A. Wright's (1996) integration linking sympathetically mediated cardiovascular activity to motivational intensity. In the following sections, we critically discuss our main conclusion and the other findings summarized above and integrate them in a broader framework of related research. Specifically, we discuss to what extent naturally occurring dysphoria can be compared to experimentally induced mood and which mechanisms may be responsible for mood and dysphoria effects on cardiovascular reactivity. Furthermore, we examine more closely dysphoric participants’ pattern of cardiovascular reactivity. We discuss the possible impact of dysphoria on maximally justified effort and, finally, the relation of effort mobilization to performance outcomes.

2.1 Experimentally Induced Mood, Naturally Occurring Dysphoria, and Clinical Depression

The present research program builds on the assumption that depression and dysphoria are, amongst other symptoms, characterized by a persistent negative mood (DSM-IV, American Psychiatric Association, 1994; Mineka et al., 1998; Scott & Ingram, 1998) and on previous research about the influence of manipulated transient mood states on cardiovascular reactivity (e.g., Gendolla & Krüsken, 2001a, 2002c). Our studies have provided convincing evidence for the stability of negative mood in our dysphoric samples. Moreover, the present results about cardiovascular reactivity parallel the results by Gendolla and Krüsken. This makes us confident that the effects of naturally occurring dysphoria on effort mobilization are comparable to the effects of transient mood states on effort mobilization.

In their discussion about the magnitude of the influence of natural versus manipulated mood, Scott and Ingram (1998) emphasize that manipulated mood states tend to be stronger and therefore to have a stronger influence on dependent measures. They argue: “When affect-induction studies produce findings that parallel the cognitive features of depression, this type of evidence strengthens the case for the role of affective states in producing cognitive features in depression” (p. 203). We think that their reasoning can be extended with respect to physiological processes and measures. From this perspective, the present findings concerning the effects of (supposedly weaker) naturally occurring mood strengthen the assumption advanced in the theoretical part, namely that
the persistent negative mood of dysphoric or depressed individuals plays an important role in the cardiovascular and motivational features of depression.

Nevertheless, the debate about the generalizability of findings from subclinical to clinical samples and about analogue versus clinical depression should not be neglected at this point. As discussed in the theoretical part, researchers do not agree whether subclinical depression should be regarded as analogue to clinical depression (see chapter 2.1.3). Moreover—provided that one adopts the dimensional perspective—there is a discrepancy between scientific practice that often compares extreme groups and methodological considerations in favor of investigating the whole spectrum of depression scores. Our approach using extreme groups of nondysphoric and analogue dysphoric students bears the risk of underestimating and overestimating the effects at the same time: On the one hand, as some reviews about cognitive deficits in depression conclude, dysphoric individuals’ results often lay in between the control group and the clinical depression group (Mineka & Gilboa, 1998). Transferred to the psychophysiological domain, this suggests that our cardiovascular findings may be even more pronounced in a group of clinically depressed patients. On the other hand, the extreme group approach admittedly enhances the probability of finding differences that do not come out so clearly when investigating the whole spectrum of depression scores (MacCallum et al., 2002). Therefore, at this point, we have to underline that the parallelism of our results to those obtained by Gendolla and Krüsken is encouraging but that the generalizability ultimately has to be proven in clinical samples.

2.2 Mediation of Dysphoria Effects on Cardiovascular Reactivity

In this place, it is also important to discuss possible mechanisms that are responsible for dysphoria effects on effort mobilization. According to the MBM logic (Gendolla, 2000), moods, together with other factors as for instance performance standards, function as diagnostic information for task-related demand appraisals during performance. This perceived task demand, in turn, is considered to influence effort mobilization, that is, cardiovascular response. Consistent with this notion, studies by Gendolla and Krüsken have demonstrated subjective task demand to vary in dependence on the induced mood state (Gendolla & Krüsken, 2002b) and to statistically mediate the relation of mood and SBP reactivity (Gendolla & Krüsken, 2002c).

Studies 1 and 2 of our research program did not assess task-related demand appraisals and cannot give an answer to this question. Studies 3 and 4 addressed this issue by assessing perceived task demand immediately prior to task performance by means of self-reports. We regard this as the best approximation for demand appraisals during performance that cannot be assessed without interrupting the ongoing task. Only
Study 4 revealed demand appraisals to differ in dependence on dysphoria. Additionally conducted mediation analyses, however, did not find evidence for the proposed indirect effect of mood and demand appraisals. As discussed in more detail in chapter 3.8 of the empirical part, we think that a lack in statistical power necessary for this type of analyses is responsible for the null effects.

A methodological consideration that has not been discussed so far pertains to the advantages of the experimental approach in contrast to the mediational approach. A recent article by Spencer, Zanna, and Fong (2005) argues for the stepwise exploration of a hypothesized causal chain by means of several experiments—each investigating one specific link. According to the authors, this approach should be preferred in cases when the mediating variable can be manipulated easily. With respect to our research program, one of the hypothesized mediating variables—mood—has been manipulated and proven its influence on effort mobilization in previous research (see Gendolla & Brinkmann, 2005; Richter et al., 2006, as well as chapter 4 of the theoretical part for reviews). The other hypothesized mediating variable—demand appraisals—has been manipulated indirectly in previous research by manipulating objective task difficulty and/or subjective ability perception (see e.g., R. A. Wright, 1998; R. A. Wright & Franklin, 2004, for reviews). This means that the mood-demand appraisals link and the demand appraisals-cardiovascular reactivity link have already been corroborated. As discussed in the experimental part, the present research adds to this evidence the link of dysphoria to negative mood and higher subjective task demand. Consequently, even if statistical mediation did not corroborate our hypothesis, we are confident that the hypothesized causal chain with mood and task demand as mediating variables withstands in light of previous evidence of studies within the same experimental paradigm as the present research.

### 2.3 Modulation of Effort Mobilization in Dysphoria

An interesting finding from Studies 3 and 4 that has not been discussed so far is the fact that the crossover interaction of dysphoria and task difficulty was mainly due to differential effort mobilization in the nondysphoric group: Nondysphoric participants consistently mobilized more effort in the difficult than in the easy conditions. In contrast, dysphoric participants’ SBP reactivity did not differ between the easy and the difficult conditions and was different from zero in the difficult condition. At least two explanations seem plausible.

On the one hand, in both studies, participants in the difficult conditions had higher SBP reactivity than participants in the easy conditions, even if the difficulty main effect was not significant. This suggests that the difficult conditions caused participants to mobilize slightly more effort. But the significant interactions and especially the significant
Discussion: Discussion of the Results

1.18

Single comparison in Study 4 confirm the lower SBP reactivity of dysphoric students in the difficult conditions. This shows that dysphoric participants indeed disengaged to some degree—even though not completely.

On the other hand, one could argue that nondysphoric participants effectively regulated (i.e., adapted) their effort level to the demand of the respective task condition: less effort in the easy and more effort in the difficult condition. Dysphoric participants, in contrast, did not show this modulation of effort intensity in dependence on task demand. One could reason that this represents a case of impaired self-regulation insofar as dysphoric individuals seem not to adapt their effort mobilization but to mobilize a certain level of resources independent of the task at hand. Even if this interpretation is not in accordance with our general reasoning and the assumption about the comparability of dispositional and transient mood states, it offers an interesting perspective for understanding motivational issues in depression. One might argue that depression is characterized by a motivational deficit in terms of a deficient motivation and/or ability to regulate task engagement and energy resources appropriately.

In order to test both interpretations against each other, future research should manipulate objective task difficulty at more than two levels, including extremely difficult, almost impossible levels. Our assumption about the parallelism of dispositional and transient mood states is strengthened, if dysphoric and nondysphoric participants clearly disengage from an extremely difficult task (see studies that included an extremely difficult condition, e.g., Gendolla & Krüsken, 2002c; R. A. Wright et al., 1997; R. A. Wright et al., 1994). However, if dysphoric individuals’ resource mobilization level does not differ across a wide range of difficulty levels and does not correspond to nondysphorics’ disengagement from an extremely difficult task, the alternative interpretation about dysphorics’ inability to appropriately regulate and modulate effort mobilization should be advanced.

2.4 Depression and Perfectionism: The Role of Potential Motivation

Another issue not discussed so far is the relation of depression and dysphoria to the level of potential motivation, that is, the level of maximally justified effort. As reported in the theoretical part, several authors, in accordance with clinical observations, suggest that depressed individuals are perfectionistic and set too high performance standards that exceed their level of perceived self-efficacy (Cervone et al., 1994; Flett et al., 1998; Scott & Cervone, 2002; Tillema et al., 2001). This means that, theoretically, depression and dysphoria might interfere with potential motivation insofar as the level of maximally justified effort might be elevated in depression. In the present studies, we did not manipulate the classical factors that determine potential motivation, that is, our par-
participants’ needs or the incentive and instrumentality of successful task performance. We thus assumed that the level of maximally justified effort was identical in all conditions. However, an alternative explanation for the results of Studies 1 and 2 might be that dysphoria led to the striving for a higher performance standard in the do-your-best tasks and therefore to higher effort mobilization compared to the nondysphoric participants. As we did not include any measure of standard setting (e.g., a self-report check), we cannot rule out this interpretation.

However, there are several aspects that argue against an influence of dysphoria on standard setting in our studies. First of all, it has to be pointed out that the studies by Scott and Cervone (2002) and Tillema et al. (2001) are not exactly comparable to our research insofar as their studies assessed participants’ self-reported standards. It is questionable whether such verbalized standards are equivalent to the more implicit performance standards that actually influence effort mobilization. In addition, the studies by Scott and Cervone and Tillema et al. mainly involved standards in the academic domain. This is a domain where high ego-involvement—justifying high effort (see Gendolla & Richter, 2005a, 2006)—can be expected. However, this is not necessarily the case in our cognitive tasks that were presented without direct relation to participants’ academic career.

Moreover, Carver and colleagues (Carver, 1998; Carver & Ganellen, 1983) neither found evidence for the assumption that depression is associated with high standards nor that high standards are a predictor for depression. Furthermore, previous studies about mood influences on effort mobilization did not show effects of mood states on reported importance of success (Gendolla & Krüsken, 2001a, 2002c). Given the parallelism of our dysphoria studies with the mood studies, we are confident that there were no influences on success importance in our studies.

Finally, we have addressed this question separately in another study in our laboratory (Brinkmann, Gendolla, & Grotta, 2007). In this computerized experiment, university students worked on several short cognitive tasks similar to the tasks used in the present research: a memory task, a concentration task, a mental rotation task, and a creativity task. After instructions but before beginning the respective task, participants could choose among ten increasing, but not further specified, difficulty levels. Dysphoria was assessed by means of the CES-D and the BDI-II several weeks before as well as directly after the experimental session. Results revealed no association of the depression scores and the chosen difficulty levels. In summary, several aspects argue against dysphoria effects on the level of maximally justified effort in the present studies, even though the possibility cannot completely be ruled out.
2.5 Performance and its Relation to Effort Mobilization

In the theoretical part, performance impairments in depression and dysphoria have been presented. Taken together, the present studies did not invariably find evidence for cognitive deficits in our dysphoric samples. Rather, the effect of objective task difficulty dominated performance outcomes. In this section, we focus on the relation of performance and effort mobilization in general and under conditions of dysphoria and depression in particular.

First of all, as we have underlined in the respective discussion sections in the empirical part (chapters 2.6 and 3.8), performance and effort mobilization cannot be equated. Nevertheless, performance measures have been used and are still used to infer a person’s motivation in the respective task (e.g., Atkinson, 1957; Kukla, 1972). However, there is ample evidence that characteristics of the person and of the task influence the association between performance and effort. It is obvious that performance represents the outcome of energy mobilization and that not only effort but also personal abilities and strategy use contribute to this outcome (Heckhausen, 1991; Locke & Latham, 1990). Likewise, Eysenck et al. (2007) emphasize the distinction between the effectiveness of behavior that is reflected by performance outcome and the efficiency of behavior that is reflected by the relation of outcome to invested effort. As outlined by Hockey (1997), effort mobilization can be used in a way to compensate for lower task-related abilities or for high stress and workload with the goal of protecting performance (see also Kukla, 1974).

This notion of compensatory effort mobilization is intriguing, particularly with regard to depression and dysphoria. Under the condition that depressed individuals have cognitive deficits, it seems plausible to assume that effort mobilization aims at compensating for these impairments by means of increased task engagement. In this sense, the effect of dysphoria on SBP reactivity in the present studies might have been caused by ability differences with respect to the memory and concentration tasks.

However, there are several arguments suggesting that effort mobilization in our studies did not aim at compensating for cognitive deficits. First of all, compensatory effort mobilization implies that depressed individuals do not have a motivational deficit concerning specific tasks with specific characteristics. It implies rather that they are generally more motivated in terms of task engagement than nondepressed control participants and thus that we would find a dysphoria main effect. This assumption is corroborated neither by clinical observation nor by our data that show lower SBP reactivity of dysphoric participants in the difficult task conditions. Moreover, as we have argued above, it is unlikely that our dysphoric university students had strong cognitive impairments that they had to compensate for.
Nevertheless, we do not exclude that resource mobilization leads to better performance. On the contrary, it is very likely that, to some extent, higher resource investment contributes to better performance. Yet we think that effort mobilization in our studies did not primarily aim at compensating for cognitive deficits but was influenced by participants’ momentary mood and their task-related demand appraisals as discussed above.
3. Outlook

This last chapter concludes the present thesis with several considerations that place our research in a broader context and delineate connections to clinical psychopathology and other health-related issues. Finally, in the last section, we state some methodological and theoretical limitations of the present research program and provide suggestions for future research.

3.1 Effort Mobilization in Clinically Depressed and Other Patients

We have already delineated the potential generalizability of our results to clinical depression. Research on characteristics of depressed mood, dysphoria, and clinical depression suggests that results can indeed be transferred from subclinical to clinical samples and vice versa in many cases (e.g., cognitive biases, motivational systems). However, it is also possible that motivational features in a clinical population differ from motivation in subclinical individuals. Clinically depressed patients are most probably characterized not only by negative affect and distress but also by somatic and vegetative symptoms. Changes in appetite or sleep habits may have an additional impact on motivation. One clinical observation is of particular importance with respect to motivation: the loss of interest or pleasure in almost all activities appreciated before (DSM-IV; American Psychiatric Association, 1994). As our research is not concerned with the initiation aspect of motivation, our findings do not tell us about how to overcome this deficit.

However, our results tell us about effort mobilization once an action has been instigated. According to our findings, easy tasks and tasks without predefined performance standards cause dysphoric individuals to mobilize more energy. This may ultimately lead to the experience of success because effort mobilization is one of the factors that may positively influence performance outcomes. As mentioned by Austin et al. (2001), situations that are highly structured tend to mask motivational deficits. One can therefore imagine confronting depressed patients with clearly structured tasks with easy or unfixed difficulty. Such tasks should cause them to mobilize energy and by this means strengthen their self-esteem and self-confidence.

The potential generalizability of our findings is, however, not limited to depression. As mentioned repeatedly, there is a high degree of comorbidity of depression and other psychopathologies, especially anxiety disorders (Kessler et al., 2003). Moreover, there are approaches as for instance the tripartite model (Clark & Watson, 1991; Mineka et al., 1998) that regard negative affectivity as a general characteristic of almost all psychopathologies. Likewise, Hokanson, Rubert, Welker, Hollander, and Hedeen (1989) report correlational results demonstrating that various psychosocial characteristics are
similar in depression and other psychopathologies involving negative affective states. As our theoretical reasoning is mainly based on the influence of negative mood on effort mobilization, one can imagine that similar results emerge for other negative affective states, for example when comparing groups with low versus high anxiety. Of course, compared to depression, other psychopathologies are not to the same extent characterized by a motivational deficit in terms of action initiation and loss of interest or pleasure. However, the basic mechanism proposed by the MBM (Gendolla, 2000), namely that mood-congruent evaluations of task demand influence resource mobilization, should apply to other clinical groups as well. Under the assumption that most clinical and subclinical psychiatric individuals are in a more or less stable negative mood, these people should evaluate daily demands as more demanding and therefore show higher cardiovascular reactivity for certain types of tasks but lower reactivity for others, depending on task characteristics and difficulty.

3.2 Negative Affectivity and Physical Health

In a related domain, Suls and Bunde (2005) support the assumption that depression effects are comparable to the effects of other psychopathologies characterized by negative affectivity. As outlined in chapter 3.1 of the theoretical part, a large body of recent studies suggests that subclinical and clinical depression is associated with greater risk for the development and worsening of cardiovascular diseases (e.g., Frasure-Smith & Lеспérance, 2005a; Rugulies, 2002; Wulsin & Singal, 2003). Suls and Bunde qualify these results by showing that negative affectivity as a component of various psychopathological states underlies the relationship of depression and coronary heart disease.

However, regardless of whether one refers to depression or to negative affectivity in general, it is obvious that the causal chain supposed to be responsible for its association with cardiovascular diseases should be closely examined. The findings of the present research offer one piece of information for this causal chain.21 Several authors emphasize that a heightened cardiovascular response to daily demands and stressors can cause essential hypertension, which in turn is regarded as a mediator and precursor of the development of cardiovascular diseases (Blascovich & Katkin, 1993; see also Gendolla & Richter, 2005b; Light, 1987; Light et al., 1992). Some studies have indeed demonstrated (slightly) enhanced cardiovascular reactivity of depressed individuals (Kibler & Ma, 2004; Light et al., 1998). Our research corroborates this for easy tasks and for tasks without performance standards but goes beyond simply comparing depressed and nondepressed groups. As demonstrated by our results, but also by the studies by Gendolla and col-

21 We are, of course, aware of other biological third variables that have been proposed to partly account for the relation of depression and cardiovascular diseases (Frasure-Smith & Lеспérance, 2005a).
laborators and R. A. Wright and collaborators (see Gendolla & Brinkmann, 2005; Richter et al., 2006; R. A. Wright & Kirby, 2001, for reviews), cardiovascular reactivity depends on the characteristics of the various tasks and on the context within which these tasks are presented. According to the present results, one can expect a connection of depression (and negative affectivity in general) to hypertension and cardiovascular disease in dependence on the types of daily demands that those people generally face and on the extent of task engagement that they habitually show.

The notion that negative affect (as a transient state as well as an affective disposition) has detrimental effects on people’s health applies not only to cardiovascular problems but to a broad range of physical and mental health problems. As summarized for instance by Salovey et al. (2000), negative mood is associated with a weaker immune system and a higher vulnerability for various diseases. Moreover, negative mood (together with high self-focus) leads to increased symptom perception and decreased well-being (see also Gendolla, Abele, Andrei, Spurk, & Richter, 2005). In contrast, positive mood is associated with more coping resources, more information seeking and planning with regard to health behavior, decreased symptom perception, and lower vulnerability for diseases. Finally, the reciprocal influence of positive mood and social support represents a buffer against health problems (Salovey et al., 2000). Taken together, their affective disposition makes depressed individuals more vulnerable for health-related problems. This applies not only to the domain of cardiovascular problems but to a broad range of physical and mental diseases. It is not difficult to imagine the resulting vicious cycle, in which the negative effects of negative affect foster depression and depressed mood—and vice versa.

3.3 Limitations of the Present Studies and Suggestions for Future Research

Finally, we delineate some important limitations of the present research and at the same time provide suggestions for future research. The first point pertains to the broad question of sample selection. As already mentioned, authors that adopt the continuity perspective of psychopathological phenomena in general and depression in particular—as we do as well—recommend using the whole range of depression scores in order to meet the implications of psychopathology as a continuum (MacCallum et al., 2002; A. M. Ruscio & Ruscio, 2002). Research within the framework of motivational intensity theory (Brehm & Self, 1989) raises some problems with respect to such a correlational approach—especially when the nonlinear relationship for disengagement is to be investigated. For this reason, we decided to compare different groups of participants. Nevertheless, future studies might consider assessing the whole range of depres-
sion scores. In this case, nonlinear tests are required in order to test at which point of the depression continuum disengagement from a difficult task occurs.

Another recommendation for data collection pertains to the use of additional measures, as for instance a structured clinical interview, to assure that participants classified as dysphoric or depressed meet the respective criteria and to screen out other psychopathologies, if desired. This suggestion mainly concerns the comorbidity of depression and anxiety (Kessler et al., 2003; Mineka et al., 1998). As discussed in the preceding sections, it is interesting to investigate whether the results of the present research are restricted to depression and dysphoria or whether they also apply to other states of negative affectivity like for instance anxiety.

A second point for improvement concerns further implications of motivational intensity theory and the MBM (Gendolla, 2000). In particular, it is interesting to extend the difficulty spectrum to a broader range of performance standards including extremely difficult tasks. This would allow testing conclusively the hypothesized saw tooth curve depicted in Figure 3 (chapter 4.4.2 of the theoretical part) and seeing how depressed and dysphoric individuals regulate their task engagement, even at impossible levels of task difficulty (see discussion in chapter 2.3 of this part). Moreover, from the discussion in chapter 2.4 it turned out that future research should not only hold constant but also measure the importance of success, that is, the level of potential motivation. This would allow controlling for confounding influences.

Furthermore, as already discussed as well, assessment of experienced task demand and confirmation of the mediating role of those judgments is a crucial point for the integration of the MBM and motivational intensity theory. This means that sensitive operationalizations for the assessment of subjective task demand before—and ideally during—performance have to be found. It could be that more subtle, implicit measures are more appropriate than explicit self-report questions. Moreover, direct manipulation of demand appraisals is desirable (see the proposition of Spencer et al., 2005, to directly manipulate the potential mediating variable). Possible lines for operationalizing this suggestion pertain to (mood) discounting, priming procedures, and bogus information about actual task difficulty.

A final note relates to the cardiovascular measures used in the present studies. From the description of the cardiovascular system and possible indicators of sympathetic and parasympathetic activation in chapter 3 of the theoretical part it turned out that SBP is a good and reliable but not the most direct indicator of beta-adrenergic sympathetic discharge to the heart. Therefore, depending on available facilities, it is desirable to include impedance cardiography in order to measure PEP, which is a more direct index of myocardial contractility and of beta-adrenergic sympathetic activation. A disadvantage of impedance cardiography and related measures is, however, the more obtrusive place-
ment of electrodes that might more strongly influence the experimental situation than the more common blood pressure cuff.
FRENCH SUMMARY
L’introduction

La dépression est un trouble fréquent qui peut avoir des effets importants sur la qualité de vie. Selon des données récentes aux Etats-Unis comme en Europe, les taux de prévalence sur la vie s’élèvent à environ 16% et 14% pour un épisode de dépression majeure (ESEMeD/MHEDEA 2000 Investigators, 2004; Kessler, 2002; Kessler et al., 2003). D’après l’organisation mondiale de la santé (2001), la dépression est considérée comme la cause la plus importante concernant les « years of life lived with disability ». À cause de ce grand impact de la dépression sur la vie et la société, de plus en plus d’efforts sont entrepris afin d’investiguer et de comprendre l’étiologie de la dépression, sa phénoménologie, sa course ainsi que les effets de différents traitements.


La dépression et la motivation

La première partie théorique se réfère aux sujets de la classification et de la phénoménologie de la dépression ainsi qu’aux particularités motivationnelles et cognitives des personnes dépressives. Pour commencer, il est important de noter que l’intensité de la motivation n’est qu’une dimension du concept de la motivation qui englobe en plus les dimensions de l’initiation, de la direction et de la persistance d’un comportement (Geen, 1995; Vallerand & Thill, 1993). La classification de la dépression fait généralement réfé-
rence à celle du « Diagnostic and Statistical Manual of Mental Disorders » (American Psychiatric Association, 1994, 2000). Elle distingue les troubles dépressifs (« unipolaires ») des troubles bipolaires. Parmi les formes du trouble dépressif elle distingue le trouble dépressif majeur de la dysthymie et du trouble dépressif non spécifié. Un épisode dépressif majeur est caractérisé principalement par une humeur dépressive et une perte d’intérêt ou de plaisir ainsi que par d’autres symptômes psychiques et somatiques. De grandes enquêtes ont révélé que les symptômes dépressifs sont relativement stables, c’est-à-dire qu’ils persistent au cours du temps, malgré le fait que les personnes passent d’une catégorie diagnostique à une autre (Angst & Merikangas, 1997; Judd et al., 1997). Cela souligne l’importance qu’il faut accorder aux formes de dépression sous-syndromiques, comme par exemple la dysphorie.

Le terme dysphorie ne correspond pas à une catégorie diagnostique et son usage est controversé. Dans notre recherche nous nous référions au terme dysphorie pour faire référence à des échantillons « analogues », non-cliniques (Kendall et al., 1987). Directement lié à cette controverse est celle de la dépression comme une variable catégorielle ou dimensionnelle. Selon le point de vue catégoriel, la dépression clinique est qualitativement différente de l’humeur dépressive sous-clinique. Cela implique la recherche comparant des groupes cliniques avec des groupes de contrôle (Gotlib, 1984; Santor & Coyne, 2001). Par contre, la perspective dimensionnelle souligne le continuum des processus normaux et anormaux et l’importance de la recherche avec le spectre entier, c’est-à-dire avec des personnes cliniques comme avec des personnes tout-venants comme par exemple des étudiants (A. M. Ruscio & Ruscio, 2002; J. Ruscio & Ruscio, 2000; Vredenburg et al., 1993; Watson et al., 2005). Cette dernière perspective étant bien confirmée par des recherches récentes (Hankin et al., 2005; Solomon et al., 2001), nous adoptons ce point de vue dans notre recherche qui se base sur des échantillons d’étudiants, appelés par la suite des participants « dysphoriques » versus « nondysphoriques ».

Concernant la phénoménologie de la dépression en lien avec notre question de recherche, il est avant tout à noter qu’une des caractéristiques les plus importantes de la dépression et de la dysphorie est une affectivité négative générale et persistante qui est largement comparable au concept de névrotisme. Cette affectivité négative faisant partie de la phénoménologie d’autres psychopathologies comme par exemple des troubles anxieux, il a été postulé qu’elle soit un facteur principal sous-jacent à beaucoup de psychopathologies (Clark & Watson, 1991; Mineka et al., 1998). Une particularité plus spécifique de la dépression et de la dysphorie se réfère aux déficits cognitifs et neuropsychologiques. Les personnes souffrant d’une dépression clinique mais aussi de symptômes sous-cliniques sont souvent entravées dans leurs fonctions mnésiques, surtout en ce qui concerne les processus contrôlés. Des atteintes des fonctions attention-
nelles et de la mémoire à courte terme sont en lien direct avec cela ainsi que des fonctions exécutives en général impliquant avant tout des problèmes à se concentrer et à s’empêcher de ruminer (Burt et al., 1995; Elliott, 1998; Hartlage et al., 1993; Hertel, 1998; Hertel & Gerstle, 2003; Van der Linden, 2007).

Au-delà des déficits cognitifs en général, les personnes dépressives ou dysphoriques sont caractérisées par des biais cognitifs négatifs. Il est bien documenté que ces personnes reconnaissent et se souviennent préférentiellement des stimuli négatifs liés à la tristesse comparés aux stimuli neutres (Gilboa & Gotlib, 1997; Gotlib, Kasch et al., 2004). Un biais similaire se trouve aussi concernant l’attention qui est portée préférentiellement au matériel négatif (Gotlib, Krasnoperova et al., 2004; Koster et al., 2005). Finalement, et d’une importance particulière pour notre recherche, la dépression et l’affect négatif sont susceptibles de biaiser les évaluations et jugements d’une manière négative. Cela concerne entre autres les jugements concernant le plaisir d’une activité imaginée, ses propres capacités, la satisfaction avec sa propre performance ainsi que les standards visés (MacLeod, 1999; Mineka & Gilboa, 1998; Scott & Ingram, 1998; Williams et al., 1997).

A part des déficits du genre plutôt cognitif, on peut constater que certaines de ces altérations cognitives sont en lien étroit et influencées par des processus motivationnels. Entre autres, le manque d’initiation spontanée des stratégies visant à contrôler l’attention et à se concentrer sur une tâche est responsable pour les déficits mnésiques des personnes dépressives, surtout concernant des tâches mnésiques nécessitant des processus contrôlés (Hertel, 2000; Hertel & Gerstle, 2003). En plus, la rumination sur soi-même et son affect négatif ainsi que l’intrusion des pensées négatives sont des particularités dépressives qui, d’un côté, ont un grand impact sur le fonctionnement cognitif, mais qui, de l’autre côté, dépendent surtout des capacités de la régulation de soi, c’est-à-dire des fonctions motivationnelles. Ainsi, il a été démontré que le fait d’empêcher les personnes dépressives de ruminer réside en des interprétations, des attributions, des descriptions de soi et des estimations moins négatives, similaires à celles des personnes nondépressives (Lyubomirsky & Nolen-Hoeksema, 1995; Lyubomirsky et al., 1999; Pyszczynski et al., 1989). Dans le domaine de la régulation d’affect, il a été démontré en plus que les personnes dépressives ne se servent pas du rappel des souvenirs positifs afin de réguler leur affect négatif comme le font les personnes nondépressives (Josephson et al., 1996).

Un aspect important du concept de motivation, qui présente des particularités en cas de dépression, est la conception des deux orientations motivationnelles de base. D’après Gray (1982), on distingue le système d’approche comportemental (SAC) réagissant à des signes de récompense du système d’inhibition comportemental (SIC) réagissant à des signes de punition. Transféré au domaine de la psychopathologie par Fowles (Fowles, 1994), il a été démontré par le biais de questionnaires, de mesures comporte-
mentales et de mesures (neuro-) physiologiques que la dépression est associée à une faible activation du SAC ainsi qu’à une désinhibition du SIC. Ainsi, les personnes dépressives et d’humeur dépressive reportent moins de plaisir lors de différentes activités (MacPhailamy & Lewinsohn, 1974), sous-estiment la valeur de récompenses et s’y attendent moins (Layne et al., 1982). De plus, Henriques et collaborateurs ont montré que des personnes dépressives et d’humeur dépressive sont moins motivées à gagner une récompense ou à éviter une punition (Henriques & Davidson, 2000; Henriques et al., 1994). Sur le niveau cérébral, la dépression est caractérisée par une sous-activation relative des régions préfrontales gauches, régions liées aux comportements d’approche ainsi qu’à l’affect positif (Davidson, Pizzagalli, Nitschke et al., 2002; Gotlib et al., 1998; Harmon-Jones et al., 2002; Tomarken & Keener, 1998). Finalement, et également en lien avec la sous-estimation de résultats potentiels, il a été postulé que des personnes dépressives sont souvent très perfectionnistes et visent des standards de performance élevés, malgré une plus basse perception de leur efficacité de soi (Cervone et al., 1994; Scott & Cervone, 2002; Tillema et al., 2001).

La revue de la littérature sur la dépression, la motivation et la cognition suggère que des personnes dépressives manquent de motivation et sont caractérisées par des déficits motivationnels et de la régulation de soi. Toutefois, selon la théorie de l’intensité de la motivation (Brehm & Self, 1989) et l’impact d’une humeur négative sur la mobilisation de l’effort (Gendolla, 2000), nous faisons l’hypothèse selon laquelle des personnes dépressives et dysphoriques ne mobiliseraient pas toujours moins d’effort. Par la suite, le cadre théorique pour cette hypothèse ainsi que l’opérationnalisation de la mobilisation de l’effort par des mesures cardiovasculaires sont présentés.

**La mobilisation de l’effort**

Avant de décrire plus en détail les mesures cardiovasculaires, il est à noter que les liens entre la dépression et la (rég-) activité cardiovasculaire ont été sujet d’investigation auparavant. Cette recherche a été instiguée avant tout par le fait que la dépression est liée au développement et à l’aggravation des maladies cardiovasculaires (Frasure-Smith & Lespérance, 2005b). Il a été postulé qu’une dysrégulation de la balance des systèmes nerveux sympathiques et parasympathiques mènerait à une forte réactivité cardiovasculaire qui finalement prédisposerait à une vulnérabilité pour des maladies cardiovasculaires (Carney et al., 2005; Kibler & Ma, 2004; Light et al., 1998). Néanmoins, des études par rapport à l’activité et à la réactivité cardiovasculaire chez des personnes dépressives obtiennent des résultats mixtes, témoignant d’une activité et d’une réactivité cardiovasculaire plus élevée pour quelques études (Light et al., 1998), mais pas pour d’autres (Carroll et al., 2007; Kibler & Ma, 2004).
La tâche principale du système cardiovasculaire est celle de maintenir la pression artérielle et d’assurer l’écoulement du sang et l’alimentation des organes avec de l’oxygène. Le fonctionnement du système cardiovasculaire est influencé et contrôlé par plusieurs facteurs, entre autres par la régulation du système nerveux autonome. Une activation de la branche sympathique du système nerveux autonome entraîne généralement une augmentation de la fréquence cardiaque (FC) et de la contractilité du cœur. De plus, le débit sympathique cause la constriction et la dilatation des vaisseaux, dépendant du récepteur impliqué. Par contre, une activité parasymphathique peut inhiber la FC (Brownley et al., 2000; Lovallo, 2005; Stern et al., 2001). Mesurer les paramètres du système cardiovasculaire peut donc donner une vue sur l’état actuel de l’organisme et inférer l’activité autonome. Parmi les paramètres cardiovasculaires les plus communs, la pression artérielle systolique (PAS) est considérée comme une mesure sensible et fiable vis-à-vis d’une influence du système nerveux sympathique parce qu’un déchargement sympathique beta-adrénergique conditionne directement la force de la contractilité myocardique, qui avec la résistance totale des vaisseaux détermine la PAS. Comme mentionné plus haut, la FC n’est pas seulement déterminée par l’activation sympathique, mais aussi par l’activation parasymphathique. En conséquence, elle peut reproduire l’influence sympathique seulement dans la mesure où celle-ci n’est pas masquée par l’inhibition parasymphathique. La pression artérielle diastolique (PAD), qui est déterminée avant tout par la résistance totale des vaisseaux, ne représente pas toujours un bon indicateur pour l’influence sympathique parce que la résistance totale des vaisseaux ne varie pas systématiquement lors des décharges sympathiques (Brownley et al., 2000; Levick, 2003; Papillo & Shapiro, 1990).

La recherche en psychophysiology s’est intéressée au système cardiovasculaire parce que celui-ci répond à beaucoup de processus psychologiques (Papillo & Shapiro, 1990). Une contribution importante pour la compréhension des réponses individuelles aux exigences comportementales a été apportée par Obrist (1976, 1981). Il montre que la mobilisation du système cardiovasculaire est proportionnelle non seulement lors d’une tâche physique, mais aussi lors d’une tâche mentale. En outre, Obrist démontre que des tâches mentales du type « active coping »—c’est-à-dire des situations où l’individu a du contrôle sur les résultats de ses actions—suscitent une réponse du système nerveux sympathique beta-adrénergique. Ainsi, il est possible de quantifier l’engagement pour une tâche mentale par la réactivité du système cardiovasculaire et plus spécifiquement par des paramètres influencés par la stimulation beta-adrénergique du cœur, comme par exemple la PAS ou la FC (Light, 1987; Obrist et al., 1987; Papillo & Shapiro, 1990). Des recherches sur l’engagement pour une tâche sous différentes conditions de performance ont confirmé ces implications, se basant sur l’hypothèse que « l’active coping » correspond à la mobilisation de l’effort et que la réactivité cardiovasculaire (et surtout la réacti-
vité de la PAS) est élevée lors des exigences comportementales, en anticipation d’une tâche ainsi que pendant une tâche (T. W. Smith et al., 1989; T. W. Smith et al., 2000).

De plus nombreuses évidences encore proviennent des travaux de R. A. Wright (1996) qui a intégré l’approche d’Obrist et la théorie de l’intensité de la motivation (Brehm & Self, 1989). Cette théorie suppose que les individus évitent de gaspiller de l’énergie et mobilisent donc leur énergie proportionnellement à l’impression subjective des exigences d’une tâche. De plus, la théorie distingue entre l’intensité de la motivation à un moment donné et la « motivation potentielle » qui est déterminée par des facteurs classiques comme les besoins d’une personne, les traits (« incentive values ») d’une tâche et l’instrumentalité d’une tâche. Ces facteurs constituent le maximum qu’une personne est prête à investir pour une tâche donnée et ne déterminent l’intensité de la motivation que d’une façon indirecte. En outre, la théorie de l’intensité de la motivation distingue entre des tâches à difficulté fixée, c’est-à-dire des tâches pour lesquelles le niveau de performance est défini et des tâches à difficulté non fixée, c’est-à-dire des tâches pour lesquelles l’individu peut choisir le niveau de performance lui-même (avec pour consigne de faire de son mieux). La théorie suppose que pour les tâches à difficulté fixée, l’intensité de la motivation (l’intensité de l’effort) augmente proportionnellement avec la difficulté de cette tâche jusqu’au point où la mobilisation de l’effort ne semble plus possible ni justifiée. Autrement dit, on ne mobilise pas le maximum d’effort potentiellement possible, mais autant d’effort que nécessaire pour avoir du succès (voir chapitre 4.4.2, Figure 3, Panel A). Par contre, pour les tâches à difficulté non fixée l’intensité de la motivation est censée varier directement avec la magnitude de la motivation potentielle (Brehm & Self, 1989).

R. A. Wright et collaborateurs ont effectué des études confirmant les prédictions de la théorie de l’intensité de la motivation concernant la réactivité cardiovasculaire (surtout de la PAS) juste avant et pendant l’exécution d’une tâche : Pour une tâche à difficulté fixée, la mobilisation de l’effort augmente proportionnellement avec sa difficulté, jusqu’au point où la tâche semble trop difficile ou l’effort ne semble plus justifié (R. A. Wright, 1984; R. A. Wright, Brehm et al., 1990). R. A. Wright et collaborateurs ont également montré des interactions entre le niveau de difficulté et les besoins, les traits et l’instrumentalité. Ainsi, ils ont démontré que la motivation potentielle (qui elle-même n’avait pas d’effet sur la réactivité cardiovasculaire) modérait la relation entre la difficulté et l’effort (Storey et al., 1996; R. A. Wright & Gregorich, 1989; R. A. Wright, Shaw et al., 1990). En ce qui concerne des tâches à difficulté non fixée, des études ont confirmé la supposition que l’organisme cherche à atteindre le niveau de performance le plus élevé justifié et que la mobilisation de l’effort dépend directement des facteurs influençant la motivation potentielle (R. A. Wright et al., 2002; voir R. A. Wright, 1996, 1998; R. A. Wright & Kirby, 2001, pour une revue).
Une spécification de la théorie de l’intensité de la motivation particulièrement importante pour notre recherche consiste en l’influence de la capacité perçue face à la tâche. Wright et collaborateurs ont fait l’hypothèse selon laquelle les personnes avec des capacités subjectives faibles estiment la difficulté d’une tâche plus haute et mobilisent plus d’effort comparées aux personnes avec des capacités subjectives fortes. Cela implique aussi que les personnes avec des capacités faibles abandonnent à un niveau de difficulté objective plus bas (voir chapitre 4.4.2, Figure 3, Panel B, pour le patron similaire par rapport à l’influence de l’humeur). Les auteurs ont corroboré leurs hypothèses concernant la réactivité de la PAS (R. A. Wright & Dismukes, 1995; R. A. Wright et al., 1997; R. A. Wright et al., 1994). Wright et collaborateurs ont également élargi leurs hypothèses quant à l’influence de l’optimisme dispositionnel (Kirby et al., 2003, cité dans R. A. Wright & Franklin, 2004) et de la déplétion des ressources (R. A. Wright et al., 2003; R. A. Wright & Penacerrada, 2002), en se basant sur le même raisonnement, c’est-à-dire que le pessimisme et la déplétion entraînent la perception des ses propres capacités comme étant plus faibles.

Le mood-behavior-model


L’influence directive, se réfère à l’initiation et à la direction du comportement en commun avec la poursuite d’un motif hédonique. L’influence directive est censée avoir un effet sur les préférences comportementales et les intérêts dans les situations de choix. La force de l’influence directive est déterminée conjointement par la force du motif hédonique d’une personne et par l’instrumentalité d’un comportement pour satisfaire ce motif. Le MBM suggère ainsi que la force de l’influence directive dépend plutôt des variables contextuelles que de la valence de l’humeur elle-même, dans la mesure où le motif hédonique est censé être déterminé par l’intensité de l’humeur, par la saillance de l’humeur ainsi que par le contexte situationnel. Autrement dit, le modèle suggère non seulement qu’une humeur négative peut déclencher un comportement visant à réguler l’affect actuel négatif, mais aussi qu’une humeur positive peut entraîner l’individu à avoir un comportement visant à maintenir l’affect actuel positif.

L’influence informationnelle se réfère aux effets de congruence à l’humeur sur les jugements et les évaluations et prétend que l’humeur est une forme d’information qui est
intégrée à d’autres informations au moment de faire un jugement par rapport à un comportement. Ainsi, le modèle prédit qu’une évaluation sera plus positive voire plus optimiste dans le cas d’une humeur positive et plus négative voire plus pessimiste dans le cas d’une humeur négative. Le poids de l’influence informationnelle est censé être fonction du poids effectif informationnel de l’humour et de l’étendue des associations amorcées par l’humour. L’influence informationnelle de l’humour est censée avoir un effet sur l’intensité et la persistance du comportement et prédit que l’humour peut influencer la mobilisation de l’effort pour atteindre un but via des évaluations et des jugements congruents à l’humour.


Au-delà des effets de l’humeur sur la mobilisation de l’effort, Gendolla et collaborateurs ont également investigué l’interaction entre la motivation potentielle et la difficulté de la tâche. Brehm et Self (Brehm & Self, 1989) soulignent que l’effort maximalement justifié, c’est-à-dire la motivation potentielle, est déterminée par l’importance du succès : un rendement plus important justifie plus d’engagement qu’un rendement moins important. Dans une expérience de Gendolla et Krüsken (2002b) la contingence d’une récompense a été manipulée par la promesse d’une période de relaxation, indépendamment de la performance (condition non contingente) ou à la condition que les sujets réussissent la tâche (condition contingente). Les résultats montrent le patron
d’interaction prédit : si la récompense est indépendante de la performance, les personnes dans une humeur négative mobilisent plus d’effort pour la tâche facile, mais moins d’effort pour la tâche difficile. Par contre, si la récompense est dépendante de la performance, les personnes d’humeur négative mobilisent plus d’effort que les personnes d’humeur positive pour la tâche facile, mais également pour la tâche difficile. Ces résultats montrent qu’il est possible d’augmenter la motivation potentielle et d’inciter les personnes d’une humeur négative à surmonter leur déficit motivationnel par rapport à une tâche difficile.

**Les hypothèses**

Sur la base de la notion d’un déficit motivationnel et de la recherche montrant l’influence de l’humeur sur l’intensité de la motivation, notre recherche vise à investiguer les hypothèses suivantes :

1) Les personnes dysphoriques sont caractérisées par une humeur négative.
2) La ligne de base cardiovasculaire ne diffère pas entre les participants dysphoriques et nondysphoriques.
3) Pour une tâche sans standard de performance, les participants dysphoriques mobilisent plus d’effort que les participants nondysphoriques.
4) Pour une tâche avec difficulté fixée, un patron d’interaction entre la dysphorie et la difficulté de la tâche se produit, avec les participants dysphoriques mobilisant plus d’effort pour une tâche facile mais moins d’effort pour une tâche difficile que les participants nondysphoriques.
5) Pour une tâche à difficulté fixée, la demande subjective de la tâche dépend de la dysphorie et de la difficulté de la tâche, avec une progression de la difficulté perçue de la condition facile-nondysphorique à la condition difficile-dysphorique.
6) Aucune hypothèse directionnelle n’est formulée quant à la performance aux tâches mentales.

Nous avons conçu une série de quatre études testant chaque fois les Hypothèses 1 et 2 comme conditions fondamentales pour les prédicitions suivantes. Les Etudes 1 et 2 sont des réplications conceptuelles investiguant l’Hypothèse 3 pour deux tâches différentes sans standard de performance. Les Etudes 3 et 4 sont également des réplications conceptuelles investiguant les Hypothèses 4 et 5. Dans chaque étude, la performance est analysée d’une manière exploratoire. Suivant la perspective dimensionnelle de la dépression, nous avons recruté des étudiants et étudiantes après un examen de dépistage quant à la dépression, réalisé quelques semaines avant l’expérience. Favorisant l’approche des groupes extrêmes, nous avons ensuite sélectionné des personnes avec des scores bas (nondysphoriques) ou élevés (dysphoriques) pour participer à nos études. La mobilisation de l’effort—notre variable dépendante centrale—a été opérationnalisée

Etudes 1 et 2

Les deux premières études ont été conçues afin de tester l’hypothèse selon laquelle les étudiantes dysphoriques mobiliseraient plus d’effort pour une tâche mentale sans standard de performance que les étudiantes nondysphoriques. Pour cela, les deux études ($N = 35$ et $N = 25$) comparent des groupes d’étudiantes avec des scores sur la « Center for Epidemiologic Studies – Depression Scale » (Radloff, 1977; version française de Fuhrer & Rouillon, 1989) situés dans le quartile inférieur et le quartile supérieur. Lors de la deuxième étude, les participantes étaient priées de remplir une deuxième fois le questionnaire à la fin de l’expérience. Seules les personnes dont le deuxième score confirmait l’appartenance au groupe de départ étaient retenues pour les analyses.

Le déroulement était presque identique pour les deux études. Les mesures cardiovasculaires étaient récoltées d’une façon intermittente, non invasive et obtenues au moyen d’un moniteur multicanal informatisé (PAR Physiopart III, Par Electronics, Berlin, Allemagne) qui utilise l’oscillométrie pour déterminer la pression artérielle systolique (PAS, millimètres de mercure), la pression artérielle diastolique (PAD, millimètres de mercure) ainsi que la fréquence cardiaque (FC, battements par minute). Un brassard était fixé sur le bras non dominant des participantes et gonflé automatiquement dans des intervalles fixes (chaque 2 minutes en Etude 1 et chaque minute en Etude 2) pendant les deux phases de mesure : la phase d’habituation de 8 minutes et la phase de performance de 5 minutes. Afin de vérifier si l’humeur varierait en fonction du groupement, l’humeur actuelle était relevée à l’aide d’une version traduite de l’échelle « UWIST mood adjective checklist » (Matthews et al., 1990). Dans l’Etude 1, l’humeur actuelle était relevée au début de l’expérience ; dans l’Etude 2, l’humeur était relevée après avoir travaillé sur la tâche afin de montrer que la saillance de l’humeur n’influencerait pas la réactivité cardiovasculaire au cours de la tâche.

Les deux études différaient par rapport à la tâche mentale : une tâche mnésique dans l’Etude 1 et une tâche de concentration dans l’Etude 2. La tâche mnésique demandait de mémoriser des séries de lettres du type « non-sens ». Chaque série consistait en quatre lettres choisies au hasard (p. ex. IWKS). Afin de présenter une tâche sans standard prédéfini, la liste consistait en 15 séries de lettres avec l’instruction de mémoriser autant de séries que possibles. Après le temps de mémorisation, les séries rappelées étaient à noter. La tâche de concentration demandait de discriminer la lettre « d » accompagnée de deux barres de la lettre « d » accompagnée de moins ou plus de barres ainsi que de la lettre « p » accompagnée également de un, deux, trois ou quatre barres.
Les participantes avaient à indiquer, si la lettre présentée constituait le stimulus-cible en appuyant sur une des deux touches. Afin de présenter une tâche sans standard, le temps de présentation n’était pas fixé. Les participantes recevaient l’instruction de travailler aussi précisément et aussi rapidement que possible.

Par rapport à l’humeur actuelle, les résultats montraient que les participantes dysphoriques étaient d’une humeur plus négative et moins positive comparées aux participantes nondysphoriques (ps < .01), indépendamment du fait que l’humeur était relevée au début de l’expérience (Etude 1) ou après la tâche (Etude 2). La ligne de base cardiovasculaire était calculée comme moyenne des deux (Etude 1) ou trois (Etude 2) dernières mesures pendant la phase au repos, puisque pendant cette phase d’habituation les mesures cardiovasculaires diminuent normalement. Seules les dernières mesures, qui ne différaient plus (ps > .16), étaient alors prises en compte pour établir la ligne de base. Confirmant l’hypothèse, la ligne de base de la PAS, de la PAD et de la FC des deux études ne différait pas selon les groupes dysphoriques et nondysphoriques (ps > .13). Pour obtenir la réactivité cardiovasculaire, des scores de changement étaient calculés (Llabre et al., 1991) en soustrayant la ligne de base de la moyenne des mesures obtenues pendant la phase à l’effort. Des analyses préliminaires montraient que la réactivité de la PAS corrélait négativement avec la ligne de base de la PAS (Etude 1) et que la réactivité de la FC corrélait négativement avec la ligne de base de la FC (Etude 2). Afin d’éviter des effets du type « carry-over » et des biais liés à la loi des valeurs initiales (Llabre et al., 1991), nous avons ajusté la réactivité de la PAS (Etude 1) et de la FC (Etude 2) par rapport à la ligne de base. Les autres scores de réactivité n’étaient pas ajustés.

Pour l’Etude 1, l’analyse de la réactivité de la PAS montrait que les participantes dysphoriques avaient une réponse plus importante (M = 5.92, SE = 1.49) que les participantes nondysphoriques (M = 3.64, SE = 1.07). Pourtant, cette différence n’était pas significative (p = .11, unidirectionnel). Regardant de plus près les scores de changements pour chaque point de mesure pendant la phase à l’effort, il s’est avéré que les participantes dysphoriques avaient une réactivité plus prononcée surtout au début de la tâche (M = 7.31, SE = 1.64) comparées aux participantes nondysphoriques (M = 3.59, SE = 1.15), t = 1.88, p = .03 (unidirectionnel ; voir chapitre 2.4.2, Figure 6). Il n’y avait pas de différences entre les deux groupes quant à la réactivité de la PAD et de la FC dans l’Etude 1 (ps > .17, unidirectionnel). L’Etude 2 confirmait la réactivité de la PAS plus importante dans le groupe dysphorique (M = 8.72, SE = 2.00), comparé au groupe nondysphorique (M = 4.20, SE = 1.39), t = 1.76, p < .05 (unidirectionnel ; voir chapitre 2.5.2, Figure 7). L’analyse de la réactivité de la FC révélait également une réponse plus prononcée dans le groupe dysphorique (p < .04, unidirectionnel). En plus, la réactivité de la PAD montrait tendanciellement le même patron (p < .06, unidirectionnel). Concernant la performance aux tâches mentales, l’Etude 1 indiquait une meilleure performance
mnésique des participantes nondysphoriques sur tous les indices de performance (ps < .06), tandis que pour la tâche de concentration dans l’Etude 2 aucune différence n’a été observée (ps > .40).

**Etudes 3 et 4**

Ces deux études ont été conçues afin de tester les prédictions de la théorie de l’intensité de la motivation (Brehm & Self, 1989) concernant l’influence de la dysphorie sur la mobilisation de l’effort si la difficulté d’une tâche mentale est fixée, c’est-à-dire si des standards de performance sont définis. Les deux études sont des réplications conceptuelles avec un design 2 (dysphorique vs. nondysphorique) x 2 (tâche : facile vs. difficile). Comme pour les deux premières études, des étudiants dysphoriques et nondysphoriques ont été choisis selon leur score sur l’échelle CES-D et testés encore une fois à la fin de l’expérience (N = 45 femmes et 8 hommes et N = 41 femmes et 23 hommes, respectivement).

Les mesures de la pression artérielle étaient récoltées de la même façon que dans les deux premières études de manière intermittente avec le Par Physioport. La FC était récoltée de manière continue à l’aide d’un électrocardiogramme (Psylab System, Contact Precision Instruments, London, Angleterre). Ces valeurs continues étaient converties en une valeur moyenne par minute afin d’avoir les mêmes intervalles que pour la pression artérielle. L’humeur actuelle était relevée au début de l’expérience à l’aide de l’échelle UWIST. Après avoir reçu les instructions pour la tâche mais avant d’exécuter celle-ci, les participants étaient priés d’indiquer comment ils évaluerait la difficulté de la tâche, leurs propres capacités ainsi que leur probabilité de succès. Comme tâche mentale, la tâche de concentration « d2 » était utilisée dans l’Etude 3. Elle était similaire à celle de l’Etude 2, avec l’exception qu’un standard de performance était défini en limitant le temps de présentation à 1000 ms (condition facile) ou à 160 ms (condition difficile). Dans l’Etude 4, la tâche mnésique consistant en des séries de lettres du type « non-sens » était utilisée de la même manière que dans l’Etude 1, avec l’exception qu’un standard de performance était défini en demandant aux participants de mémoriser la liste entière qui consistait en quatre séries (condition facile) ou en neuf séries (condition difficile).

Quant à l’humeur actuelle au début de l’expérience, les deux études confirmaient que les participants dysphoriques étaient d’une humeur moins positive et plus négative comparés aux participants nondysphoriques (ps < .01). L’analyse de la demande subjective de la tâche (composée des trois questions d’auto-évaluation) n’indiquait pas de différences entre les quatre conditions pour l’Etude 3 (ps > .31). Par contre, la demande subjective montrait le patron attendu pour l’Etude 4. La tâche était perçue comme plus difficile dans la condition difficile-dysphorique et comme moins difficile dans la condition...
facile-nondysphorique. Néanmoins, uniquement l’effet simple de la dysphorie était fiable ($p < .02$), mais pas celui de la difficulté. La ligne de base pour la PAS, la PAD et la FC était établie en moyennant les valeurs des quatre (Etude 3) et trois (Etude 4) dernières mesures récoltées pendant la phase d’habituation. Pour l’Etude 3, il n’y avait pas de différences entre les quatre conditions concernant la ligne de base de la PAS et de la PAD ($p > .29$), à l’exception d’un effet tendanciel pour la FC, indiquant des valeurs plus élevées des personnes dysphoriques ($p = .07$). L’Etude 4, par contre, révélait une différence entre les hommes et les femmes concernant la ligne de base de la PAS, une interaction entre la difficulté et le genre concernant la ligne de base de la PAD et un effet simple de la dysphorie concernant la ligne de base de la FC ($p < .05$). Pour l’Etude 3, la ligne de base ne covariait pas avec les scores de changements ($p > .40$) et, en conséquence, la réactivité n’était pas ajustée. Par contre, la ligne de base dans l’Etude 4 covariait avec les scores de changement ($p < .05$) et, par conséquent, la réactivité était ajustée par rapport à la ligne de base.

Pour l’Etude 3, la réactivité de la PAS corroborait le patron prédit, c’est-à-dire une interaction entre la dysphorie et la difficulté de la tâche ($p < .03$ ; voir chapitre 3.5.2, Figure 9). Des analyses détaillées révélaient que les participants dysphoriques ($M = 5.73$, $SE = 1.25$) avaient une réactivité plus prononcée dans la condition facile que les participants nondysphoriques ($M = 1.51$, $SE = 1.25$), $t = 2.24$, $p < .02$ (unidirectionnel). Contrairement aux prédictions, la différence dans la condition difficile n’était pas significative ($p = .17$, unidirectionnel). Pour l’Etude 4, le même patron d’interaction quant à la réactivité de la PAS s’est avéré ($p < .03$ ; voir chapitre 3.6.2, Figure 10), avec une différence tendancielle entre les participants dysphoriques ($M = 6.24$, $SE = 1.37$) et les participants nondysphoriques ($M = 3.37$, $SE = 1.45$) dans la condition facile, $t = 1.44$, $p < .08$ (unidirectionnel), ainsi qu’une différence significative dans la condition difficile entre les participants dysphoriques ($M = 5.06$, $SE = 1.14$) et les participants nondysphoriques ($M = 8.58$, $SE = 1.62$), $t = 1.77$, $p < .05$ (unidirectionnel). Concernant la réactivité de la PAD et de la FC, il n’y avait pas d’effets en Etude 3 ($p > .30$). Dans l’Etude 4, la PAD ne différait pas selon les conditions ($p > .19$) ; par contre, la réactivité de la FC était plus élevée dans la condition difficile comparée à la condition facile ($p < .03$). Finalement, la performance aux tâches mentales indiquait pour les deux études que les participants dans les conditions faciles avaient de meilleures performances que ceux dans les conditions difficiles ($p < .01$), même si les indices de performance étaient ajustés par rapport au nombre de stimuli « d2 » ou au nombre de séries de lettres.

**Discussion**

Pour résumer, nos quatre études ont corroboré l’hypothèse selon laquelle des étudiants avec des scores de dépression élevés seraient de plus mauvaise humeur, même si...
l’expérience avait lieu quelques semaines après l’examen de dépistage de la dépression. L’effet de congruence à l’humeur sur la demande subjective de la tâche s’est avéré uniquement dans l’Etude 4, dans laquelle les participants dysphoriques percevaient la tâche mnésique comme plus difficile et leur capacité comme moins élevée que les participants nondysphoriques. Avec l’exception de la FC dans l’Etude 4, nos expériences n’ont pas trouvé de différences entre les participants dysphoriques et nondysphoriques concernant la ligne de base cardiovasculaire, contrairement à, par exemple, l’étude de Light et al. (1998). Nos hypothèses principales concernant la mobilisation de l’effort ont été confirmées par la réactivité de la PAS. Les deux premières études ont montré que les participantes dysphoriques avaient une réactivité plus importante pour les tâches sans standard de performance comparées aux participantes nondysphoriques. Les deux études suivantes ont corroboré le patron d’interaction entre la dysphorie et la difficulté de la tâche. Dans ces études, les participantes dysphoriques avaient une réactivité plus importante dans les conditions faciles, mais une réactivité moins prononcée dans les conditions difficiles, comparés aux participants nondysphoriques. A l’exception de la FC dans les Etudes 2 et 4, le patron de réactivité de la PAD et de la FC ne correspondait pas au patron de la PAS (voir Brownley et al., 2000; Levick, 2003; Papillo & Shapiro, 1990). Quant à la performance aux tâches mentales, uniquement l’Etude 1 révélait une différence entre les participantes dysphoriques et nondysphoriques concernant la tâche mnésique, tandis que les autres études n’indiquaient ni déficits cognitifs des participants dysphoriques ni associations entre la mobilisation de l’effort et la performance cognitive.

Notre série d’expériences a confirmé notre hypothèse centrale selon laquelle la dysphorie n’est pas nécessairement associée à un déficit motivationnel, mais que cela dépend plutôt des caractéristiques de la tâche ainsi que de sa difficulté si la dysphorie entraîne une mobilisation de l’effort plus ou moins élevée. Nos prédictions étaient principalement fondées sur le fait que la dysphorie et la dépression sont avant tout caractérisées par une humeur négative persistante (American Psychiatric Association, 1994; Mineka et al., 1998; Scott & Ingram, 1998) et sur la recherche qui montre l’influence de l’humeur sur la mobilisation de l’effort. La similarité de nos résultats quant à la dysphorie et des résultats de Gendolla et collaborateurs quant à l’humeur manipulée (p. ex. Gendolla & Krüsken, 2001a, 2002c) indique que ce patron de mobilisation de l’effort serait également comparable à celui qu’on trouverait auprès d’une population clinique. Néanmoins, cette hypothèse reste à être confirmée sur un échantillon clinique. De même, à l’avenir, il serait intéressant d’aborder la question de savoir si un patron similaire se trouve également auprès d’un échantillon anxieux ou auprès d’autres psychopathologies caractérisées par une humeur négative persistante (voire une affectivité négative) ainsi que par des évaluations pessimistes congruentes à l’humeur. Notre raisonnement concernant l’influence de la dysphorie sur la mobilisation de l’effort est fondé sur les
effets de congruence à l’humeur sur la perception de la demande d’une tâche qui en revanche détermine la mobilisation de l’effort. Même si l’Etude 4 ne corrobore que partiellement cette influence et même si la médiation statistique ne s’est pas avérée significative, la médiation de ce processus par l’humeur et par la demande subjective est très probable vu les études antérieures, manipulant non seulement l’humeur, mais aussi la perception subjective (R. A. Wright, 1998; R. A. Wright & Franklin, 2004, pour des revues).

Une question intéressante à aborder est le résultat des Etudes 3 et 4 montrant que la réactivité de la PAS des participants dysphoriques ne diffère pas en fonction de la difficulté de la tâche. Il semble donc que ces personnes n’ajustent pas leur effort en fonction des exigences objectives, ce qui peut être un indice pour une régulation de soi altérée. Il reste désormais à investiguer à l’aide d’expériences manipulant un spectre plus large de difficultés possibles et impossibles si les personnes dysphoriques montrent un clair désengagement à un moment donné ou non. Pour finir, il est important de discuter de l’absence de corrélations entre la mobilisation de l’effort et la performance dans trois de nos quatre études. Dans ce cas, il est avant tout à souligner que la performance est le résultat d’une action et que non seulement l’effort, mais aussi d’autres facteurs comme des capacités personnelles et des stratégies contribuent à ce résultat (Eysenck et al., 2007; Locke & Latham, 1990). Cela soulève la question de savoir si la mobilisation de l’effort a été utilisée par nos participants dysphoriques comme moyen pour compenser un déficit cognitif éventuel (Hockey, 1997). Cependant, nos deux dernières études montrent des performances comparables entre des personnes dysphoriques et nondysphoriques, malgré une mobilisation de l’effort moins importante des participants dysphoriques dans la condition difficile. En outre, notre échantillon d’étudiants n’est pas un échantillon très susceptible de souffrir de déficits cognitifs, même parmi les étudiants avec des scores de dépression élevés.

Pour conclure, nous traitons quelques limites de notre recherche et montrons en même temps des suggestions pour la recherche future. Quant à notre échantillon, il est important de noter que nous n’avons pas investigué tout le spectre du continuum dépressif, mais uniquement des étudiants avec des scores bas ou élevés. Pour l’avenir, il est recommandable de tenir compte de la perspective dimensionnelle en investiguant le spectre entier du continuum dépressif. Une deuxième suggestion pour la recherche à venir serait de présenter aux participants également des tâches très difficiles voire impossibles afin de pouvoir tirer des conclusions plus univoques concernant le désengagement du groupe dysphorique. Concernant les autres implications de l’intégration du MBM (Gendolla, 2000) avec la théorie de l’intensité de la motivation (Brehm & Self, 1989), il est crucial de montrer le processus médiateur proposé, notamment quant à la demande subjective de la tâche. A cette fin, une manipulation intéressante serait par
exemple la procédure de « mood discounting » (voir Schwarz & Clore, 1996). Finalement, le concept de la mobilisation de l’effort ou de l’engagement pour une tâche implique l’activation du système nerveux sympathique et plus spécifiquement des récepteurs beta adrénergiques du cœur (Obrist, 1981). Sachant que la contractilité du cœur est un indicateur plus direct pour cette influence sympathique beta-adrénergique que la PAS, la récolte des mesures comme par exemple de la période de pré-éjection (« pre-ejection period ») du cœur est à prendre en considération afin d’améliorer la fiabilité des conclusions.
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