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General introduction

1 RECURRENCE OF DEPRESSION

One of the greatest problems with major depressive disorder is that it is a highly recurrent disease. About 50% of patients recovered from an episode of major depression will suffer another episode once in their life (Belsher and Costello, 1988). The picture is even worse for patients who have been depressed already a number of times, as risk of recurrence seems to increase progressively with each successive episode (Bauer et al., 2002; Kessing et al., 2004). On the other hand, recurrence rates progressively decrease as the duration of recovery increases (Belsher and Costello, 1988; Solomon et al., 2000). These observations point to the relevance of identifying mechanisms involved in recurrence of depression, as these may be targeted upon to help remitted patients to remain well.

Despite a widespread agreement that complex mental illnesses, such as depression, are heterogeneous in their etiology and course (Bebbington, 1987; Costello et al., 2002), theory and research aimed at identifying risk factors for (recurrence of) depression are predominantly monodimensional in nature. Factors from single fields of research are usually studied in isolation, and attempts to integrate findings are rare. Studies that simultaneously investigate factors belonging to different fields are needed for a multifactorial account of depression (Costello et al., 2002). The study described in this thesis was designed in this way.

The research fields focused upon in the present study are: personality, social cognition, interpersonal behavior, stress physiology, and interpersonal stress. The study centers around an intriguing concept from the field of personality, neuroticism. Neuroticism is a personality construct that is very consistently related to depression (and several other kinds of distress). At the same time, it is rather unclear what this construct actually is and how it confers risk of depression. In this thesis, we investigate how neuroticism is linked to recurrence of depression by examining potentially intermediating factors from the 4 other fields of depression research. We study these factors in patients whose depression is in remission.

The specific factors under investigation were chosen from an interpersonal perspective. This was done because the social realm is inextricably bound up with depression (Joiner and Coyne, 1999; Rhodes and Lakey, 1999), and presumably particularly so when the disease has taken a recurrent course (Coyne, 1999). We expect that interpersonal factors also play an important

role in the mechanisms linking neuroticism to recurrence of depression.

In the following section, we introduce the 5 research fields of our interest and the factors we selected from these fields to investigate. In section 3, we review what is known about the interrelationships between factors from these different fields. On the basis of this literature, we subsequently formulate our hypotheses on how the factors under investigation are related to recurrence of depression and how their combined action may explain the risk associated with neuroticism (section 4).

2 FIVE FIELDS OF INVESTIGATION

The 5 fields that are focused upon in this thesis are all widely studied in the context of depression. We will outline how these fields are implicated in depression theory and research, and introduce the potential risk factors of recurrence that were investigated in the present study.

2.1 Personality

Personality refers to an individual's characteristic patterns of thinking, feeling, and behaving (Klein, 2002). Most contemporary models of personality distinguish a small number of higher-order personality dimensions (e.g., the "Big Five", McCrae and Costa, 1999), each of which is subdivided into a larger number of lower-order traits or "facets". Neuroticism is one of the higher-order personality dimensions included in all major models of personality. Neuroticism has been defined as "a stable, heritable, and highly general trait dimension with a multiplicity of aspects ranging from mood to behavior (...) the core of which is a temperamental sensitivity to negative stimuli" (Clark et al., 1994), or as "a broad dimension of individual differences in the tendency to experience negative, distressing emotions and to possess associated behavioral and cognitive traits" (Costa and McCrae, 1987). These widely used definitions of neuroticism nicely illustrate the indeterminate character of the concept. It comprises an assemblage of aspects as divers as emotional instability, irritability, stress reactivity, fearfulness, the tendency to worry, to be moody, to feel guilty and lonely. These aspects are usually assessed by means of a range of items on a questionnaire, which are taken together to make up the "neuroticism score". This has led to uncertainty as to what neuroticism actually is and how much explanatory value the concept has (e.g., Claridge and Davis, 2001; Ormel et al., 2004c).

Despite the indistinctness of the concept (or probably: *due to* its indistinctness), a great variety of clinical phenomena, both somatic and psychiatric, has been found to be associated with neuroticism (Claridge and Davis, 2001; Neeleman et al., 2002; Neeleman et al., 2004). Depressive disorder is one of the diseases showing very consistent associations with neuroticism. Levels of neuroticism are higher in depressed individuals compared to healthy controls and population samples, and high neuroticism is related to more severe episodes and to a poorer course (Enns and Cox, 1997; Klein, et al., 2002). It is clear that neuroticism has a strong state-dependent component; neuroticism levels decrease as depressive symptoms abate (Klein, et al., 2002). A number of studies, however, have reported that neuroticism scores are still higher in remitted individuals compared to control or population samples (Barnett and Gotlib, 1988; Klein, et al., 2002). Results are not entirely consistent in this respect, but this may have to do with sample selection effects (Barnett and Gotlib, 1988; Klein, et al., 2002). There are more reasons, however, to assume that neuroticism is not only a concomitant of the depressed state, but also confers risk for the development of depressive episodes. First, premorbidly assessed neuroticism is predictive of the onset of depression (Enns and Cox, 1997; Klein, et al., 2002; Ormel et al., 2004b). Secondly, neuroticism measured in remission is predictive of subsequent relapse and recurrence (Marks et al., 1992; Surtees and Wainwright, 1996; Mulder, 2002).

To conclude, neuroticism is a very reliable, but rather non-specific correlate and predictor of (recurrence of) depression. In this thesis we try to give this broad personality construct some hands and feet by concurrently investigating factors from other fields of depression research, which may figure as a link between neuroticism and depression.

2.2 Social cognition

Cognitive research in depression has been influenced greatly by the theory of Aaron Beck (Beck, 1967; Beck et al., 1985). Central to this theory is the concept of the cognitive *schema*. Cognitive schemas are stable underlying structures that guide the perception, integration, interpretation, and retrieval of information. The schemas of particular concern to Beck's theory are those containing negative information about the self and the self in relation to others. These negative schemas are postulated to be developed early in life in response to certain situations and to be later activated by (certain types of) stressful life events. Once activated, these schemas bring about systematic

biases and distortions in the processing of information. Specifically, these biases and distortions are thought to lead to overly pessimistic views of the self, the future, and the world (the "negative cognitive triad"). These, in turn, lead to sadness and the other symptoms of depression.

Since the emergence of Beck's theory (and other important cognitive theories of depression, e.g., Higgins, 1987; Teasdale, 1988; Abramson et al., 1989), a mass of empirical research has shown that depressives do indeed have cognitive distortions and negative biases (Mathews and MacLeod, 1994; Weary and Edwards, 1994; Williams et al., 1997; Alloy et al., 1999; Mineka and Gilboa, 1998). Much of this research, however, suggests that these dysfunctional cognitions are secondary to the depressed mood, disappearing when the depression is over (Barnett and Gotlib, 1988; Persons and Miranda, 1992). Yet, there is also some evidence of the presence of cognitive distortions and biases before onset of the depression or after recovery (Alloy et al., 1999; Austin et al., 2001; Rude et al., 2002). Moreover, it is not sure whether the negative findings in remitted samples refute the proposed *causal* role of cognition in depression. Dysfunctional cognitions may be inaccessible to measurement outside of the depressive episode because they might need to be primed by negative mood or activated by stressful experiences (Segal and Ingram, 1994; Miranda and Gross, 1997; Just et al, 2001).

The field of *social* cognition concerns the cognitive processing of *social* information. The present study focuses on the perception part of social cognition, in particular the perception of emotions from facial and vocal expressions. Emotional expressions play an important role in human communication (Burgoon, 1985; Darwin, 1998), and the accurate decoding of emotional cues is quite essential to social functioning. Misidentification of emotional expressions may lead to interpersonal problems, and thus contribute to the experience of interpersonal stress and the erosion of social supportive resources. Moreover, a negatively biased emotion perception may promote pessimistic views of the self and (one's relationships with) others. In these ways, deficits in emotion perception may increase the risk of depression (Hammen, 1992; Gotlib et al., 2004).

Empirical studies on emotion perception of depressives most often concern the decoding of facial expressions. A few studies are done on emotions expressed in tone of voice. Both lines of research show evidence of perceptual deficits in depressed patients. Depressives are found to be *impaired* in emotion recognition; i.e. they make more errors than controls in the identification

of emotional expressions (Feinberg et al., 1986; Zuroff and Colussy, 1986; Murphy and Cutting, 1990; Persad and Polivy, 1993; Mikhailova et al., 1996; Emerson et al. 1999; Leppanen et al., 2004; Luck and Dowrick, 2004; Surguladze et al., 2004) (but see also some negative findings: Walker et al., 1984; Archer et al., 1992; Gaebel and Wolwer, 1992; Ridout et al., 2003). Depressives are also found to have a *negative bias* in emotion perception, i.e. they show increased attention to and enhanced recall of negative expressions vs. other expressions (Gilboa-Schechtman et al., 2002; Ridout et al., 2003; Gotlib et al., 2004), and they interpret emotional expressions in a more negative (or less positive) way than controls do (Gur et al., 1992; Hale et al., 1998; Leppanen et al., 2004; Luck and Dowrick, 2004; Surguladze et al., 2004). Such a negative perceptual bias has been shown to be related to a poor course of the depression (Bouhuys et al., 1999a; Geerts and Bouhuys, 1998). A few studies with assessments done both in depression and in remission showed that perceptual deficits tend to diminish when patients recover (Mikhailova et al., 1996; Bouhuys et al., 1999b; Levkovitz et al., 2003). One of these studies also related emotion perception of remitted patients to subsequent relapse (Bouhuys et al., 1999b). This study found that higher levels of negative emotions perceived in facial expressions were associated with a higher risk of relapse.

2.3 Interpersonal behavior

In response to Beck's theory, which seeks the origins of depression exclusively "inside the head" of the individual, theories have emerged that stress the importance of interpersonal interactions in depression. The best known of these approaches is James Coyne's interactional theory of depression (Coyne, 1976). This theory holds that depression-prone persons, because of their behavior, elicit negative reactions from others that serve to exacerbate depressive symptomatology. Specifically, it proposes that the depression-prone person seeks reassurance from others when feeling distressed, insecure, or worthless. Other people may provide reassurance, but the depression-prone person doubts its genuineness, for example because he or she thinks that they act from pity or obligation, or because the verbal messages do not correspond with the nonverbal ones. This leads the depression-prone person again to seek reassurance. A repetitive pattern is established, in which increasing demands for reassurance are made. As a result, others become annoyed and irritated, or even distressed themselves. This increases the likelihood that they will reject

and avoid the depression-prone person, which adds to the deterioration of this person's self-concept and the disruption of his or her social network, leading to exacerbation or maintenance of depressive symptoms.

Although Coyne's theory as formulated in his 1976 article was in important respects vague and underdeveloped (Coyne, 1999), it has provoked a lot of empirical work on the interpersonal context of depression. This research has confirmed that depressed individuals elicit rejection from others (at least in the long term), and that they often experience dissatisfactory or disrupted relationships (Marcus and Nardone, 1992; Segrin, 1998). There is also evidence for excessive reassurance seeking in depressed patients, and a few studies show that this interaction style indeed predicts longitudinal increases in depressive symptoms (Joiner, 2000). The literature further points to various other problems in depressives' interpersonal behavior, such as showing overly dependent or clinging behavior, talking about self-focused or negative issues, and having poor conflict-resolution skills (Gotlib and Hammen, 1992; Segrin, 1998; Joiner, 2002).

The interpersonal behavior of depressives is also unfavorable in several of its *nonverbal* aspects (Segrin, 1998; Segrin, 2000; Joiner, 2002). Nonverbal cues are very powerful elements of human communication (Burgoon, 1985; Depaulo and Friedman, 1998), notwithstanding the fact that people are often not consciously aware of them. Empirical evidence substantiates that the greater part of the meaning in face-to-face interchanges is communicated nonverbally (Burgoon, 1985). Research also shows that people tend to place greater reliance on nonverbal than on verbal cues, especially when nonverbal signals conflict with the verbal message (Burgoon, 1985). Given the importance of nonverbal signals in interpersonal communication, the observation that depressed patients show deviant nonverbal behavior is quite relevant. Depressives often exhibit a behavioral pattern of low interpersonal involvement, unresponsiveness, and negativity. They tend to speak slowly, with little volume and long pauses, gaze little at their interaction partners, gesture and head-nod infrequently, and exhibit sad facial expressions (Segrin, 1998; Segrin, 2000). Many of these behaviors are correlated with aversion and rejection from others (Gotlib and Hammen, 1992; Segrin and Abramson, 1994). Whether such behaviors are also *causal* to the depression is not clear. The longitudinal studies done to examine this have yielded mixed results thus far (Bouhuys et al., 1991; Bouhuys and Van den Hoofdakker, 1993; Troisi et al., 1989; Segrin, 2000; Joiner, 2002).

Most studies on the interpersonal behavior of depressed or depression-

prone individuals are one-sided; they focus on either the behavior of these individuals or that of their interaction partners. The strong point of Coyne's interactional theory has been that it paid attention to the *interplay* between the behaviors of the people concerned. Particularly relevant in this regard is the observation that interaction partners usually adjust their behaviors to each other. They automatically adopt each other's postures, movements, facial expressions, speech patterns, rhythms, and mannerisms (Bernieri and Rosenthal, 1991; Burgoon et al., 1993; Cappella, 1996; Lakin et al., 2003). Such behavioral matching and synchronization processes are a characteristic feature of everyday interactions and can be observed already at a very young age (Condon and Sander, 1974; Isabella et al., 1989). They are thought to be a fundamental element of human communication, serving to promote harmonious relationships, as a kind of "social glue" (Kendon, 1970; Chartrand and Bargh, 1999). This thought is corroborated by evidence that interactions characterized by a high degree of behavioral symmetry and synchrony are experienced as pleasant, supportive, and rewarding (Tickle-Degnen and Rosenthal, 1990; Bernieri and Rosenthal, 1991; Cappella, 1997), and that interaction partners who are "in sync" or "in tune" with one another report high feelings of attraction and affiliation (Cappella and Palmer, 1990). Two studies of our own research group showed that mutual adjustment of nonverbal behavior is also related to a favorable course of depression. The degree to which depressed patients and their experimental conversation partners adjusted their levels of nonverbal involvement behavior to each other was predictive of the subsequent improvement of the patients' depression (Geerts et al., 1996, 2000).

The above reviewed findings and ideas prompted us to investigate whether mutual adjustment of nonverbal behavior also plays a role in recurrence of depression. A lack of nonverbal "match" during social interactions presumably increases the likelihood that interactions become dissatisfactory and stressful, and that interaction partners eventually withdraw. Interpersonal stress and shrinkage of the social network, in turn, increase the risk of recurrence of depression (Paykel, 1994).

2.4 Stress physiology

The term "stress" is generally used in two ways: to identify events or circumstances that are perceived as being adverse ("stressors") or to describe the state induced by such events or circumstances (the "stress reaction"). This section deals with the latter.

The physiological system that plays a key role in the stress reaction is the hypothalamic-pituitary-adrenal (HPA) axis. During a perceived physical or psychological threat, a cascade of hormones is released from this axis. First, corticotropin releasing factor (CRF) is released from the hypothalamus. CRF subsequently triggers the release of adrenocorticotropin hormone (ACTH) from the pituitary gland into the bloodstream. Finally, ACTH stimulates the release of corticosteroids (cortisol) from the adrenal cortex. The "stress hormone" cortisol brings about a variety of physiological, cognitive, and behavioral changes that are critical for successful adaptation to the stressor (Sapolsky et al., 2000; Erickson et al., 2003). It also serves to down-regulate the stress response, inhibiting further release of CRF and ACTH. This negative feedback mechanism prevents the system from overshooting (Sapolsky et al., 2000).

Whereas activation of the HPA axis is an essential part of a normal response to stress, prolonged or excessive HPA-axis activation is almost always deleterious (McEwen, 2000; Wolkowitz et al., 2001). Hyperactivity of the HPA axis is also related to psychopathology. Elevated cortisol levels and impaired feedback inhibition of the stress system are the most widely replicated biological abnormalities in major depression (Thase and Howland, 1995; Wolkowitz et al., 2001). This is not to say that HPA-axis dysregulations are unique to depression or that they can be found invariably in depressives (e.g., Thase et al., 2002; Strickland et al., 2002). But the association between depression and hypercortisolism is evident enough to have reached the status of textbook truism (Cowen, 2002).

Many authors consider HPA-axis hyperactivity to be a state effect of the depression, as it usually resolves upon remission (Thase and Howland, 1995). A number of studies, however, show that HPA-axis hyperactivity may persist after apparent clinical recovery and that the individuals concerned are at a higher risk of subsequent relapse and recurrence (Gurguis et al., 1990; Ribeiro et al., 1993; Zobel et al., 1999; Holsboer, 2001; Zobel et al., 2001). Besides, there are other indications that dysregulations in HPA-axis functioning may also be causal to the development of depressive episodes. Three lines of evidence are relevant in this respect. First, substantial evidence from a variety of studies suggests that having been exposed to stress early in life represents a major risk for the development of depression (Heim and Nemeroff, 2001). Secondly, animal research has convincingly demonstrated that early stress (either pre- or postnatal) and deprived caregiving conditions lead to perma-

nent changes in the stress system, resulting in a long-lasting hyperactivity and sensitization of the HPA axis, thus increasing susceptibility to stress later in life (Weinstock, 1997; Kaufman et al., 2000). A number of clinical studies have extended these findings to humans (Heim and Nemeroff, 2001; Meyer et al., 2001). Thirdly, it is well established that the onset of depressive disorder is often precipitated by stressful life events or chronic difficulties (see below). Together, these lines of evidence suggest that adverse experience during early development may lead to hypersensitivity of the stress system, predisposing these individuals to the development of depression (Post and Weiss, 1998; McEwen, 2000; Meyer et al., 2001).

2.5 Interpersonal stress

The importance of interpersonal stress in the etiology of depression has come to the fore a number of times already in the above sections. "Interpersonal stress" does not refer to a clearly defined research field, but the concept plays a key role in several interpersonal approaches of depression (see Hammen, 1999). It figures throughout this thesis as a factor of special relevance to our understanding of recurrence of depression. Our measure of interpersonal stress is the occurrence of stressful life events during the follow-up of our study, in particular life events in which interpersonal interactions play a role (e.g., divorce).

Stressful life events (traditionally defined as "objective experiences that are sufficiently disruptive or threatening as to require a substantial readjustment on the part of the individual" (Cronkite and Moos, 1995)) have long been implicated in depression. The work of George Brown has been very influential to this research. Brown showed that severe life events, often involving loss and disappointments in close relationships, had a crucial role in the precipitation of depression, and he also pointed to the relevance of the social context in which stressful life events occur (Brown and Harris, 1978; Brown, 1993). By now, there is a large body of literature showing that exposure to stressful life events is associated with the subsequent onset of depressive episodes (Brown and Harris, 1989; Kessler, 1997; Paykel, 2003). Because of the consistency of this association, the common notion is that stressful life events trigger or provoke depression.

Stressful life events have also been found to be predictive of recurrence of depression (Post, 1992; Mazure, 1998; Monroe and Harkness, 2005). Recurrent episodes, however, show less strong associations with stressful life events than

first onsets of depression do. This finding can be explained in two ways: 1. major life stress loses its *potential* to trigger depression (a progressive insensitivity to stress develops with recurrences of the disease, and alternative etiological mechanisms become increasingly important); 2. major life stress loses its *opportunity* to trigger depression (a progressive sensitivity to stress develops with recurrences of the disease, as a result of which increasingly minor stressors can provoke already a depressive episode) (see Monroe and Harkness, 2005). The latter explanation nicely fits in with stress-sensitization models of depression (e.g., Post and Weiss, 1998). The design of most studies, however, is unfit to distinguish between the two explanations (Monroe and Harkness, 2005; but see Brilman and Ormel, 2001; Ormel et al., 2001).

Although many depressed people have experienced a stressful life event before the onset of their depressive episode, only a minority of the people exposed to stressful life events become depressed. This observation has led to the supposition that there are individual differences in stress reactivity. Predispositional and contextual factors may modify the effects of a stressor, enhancing or diminishing its impact. Such thinking is the core of diathesis-stress models of depression, which propose that stress activates a diathesis (vulnerability), transforming a predisposition into the presence of psychopathology (Monroe and Simons, 1991). The negative cognitive schemas of Beck are a good example of such a diathesis, and neuroticism is also frequently conceived of in this way. Stress-buffering models of depression are the positive counterparts of diathesis-stress models. They assume that certain resilience factors protect the individual against depression by buffering the effects of stress. One important resilience factor is social support (Cohen and Wills, 1985; Coyne and Downey, 1991; Rhodes and Lakey, 1999).

3 INTERRELATIONSHIPS BETWEEN THE DIFFERENT RESEARCH FIELDS

Background idea of this thesis is that factors from different fields of depression research do not figure as isolated risk factors for depression but are related to each other. Such relationships can be causal, one factor leading to the other. In such a case, the one factor may mediate the effect of the other factor, the latter acting as a more proximate cause of depression (mediation). Factors can also act as modulators of one another, enhancing or reducing each other's effect (moderation). Another possibility is that factors are related to

each other because of a conceptual overlap, i.e. that they (partly) measure the same thing. Finally, factors can covary without further being related to each other, for example when they share a common cause.

The empirical literature provides some evidence for interrelationships between factors from the fields of our interest. In this section we review what is known. Where available, we also address relevant theories of depression that incorporate interfield relationships. The presented material serves as a basis for the multifactorial model of recurrence of depression that we will propose in section 4.2.

3.1 Personality ↔ social cognition

As becomes clear from influential definitions of neuroticism like the one of Costa and McCrae (see above), part of neuroticism reflects cognitive traits. Most researchers expect neuroticism to be related to the cognitive processing of emotional rather than neutral information (Martin, 1985). This is because neuroticism is generally conceived of as influencing a person's responsiveness to emotional stimuli. Indeed, several studies suggest that high neuroticism favors the cognitive processing of negative emotional information (Martin, 1985; Rusting, 1998). Mainly studies using memory tasks have found such relationships (Martin, 1985). Individuals high on neuroticism show enhanced recall of negative information, especially when the information is self-referent. Studies on the relationship between neuroticism and other aspects of cognitive processing, like attention, interpretation, or speed of perception, show less consistent results (Rusting, 1998). There is some evidence that individuals high on neuroticism show increased attention to negative or threatening information, that they are more likely to interpret stimuli in a negative way, and that they are faster to respond to negative stimuli (Rusting, 1998; Geerts and Bouhuys, 1998). Not all studies found such relationships, however, and in studies that did, it was often necessary to prime subjects prior to the cognitive assessment by means of stress- or mood induction (Mathews and MacLeod, 1994; Eysenck, 2000).

To summarize, part of neuroticism is conceived of as the tendency to process emotionally relevant information in a negative way. Empirical evidence is generally supportive of this conception, although the most convincing evidence comes from priming studies and studies using memory tasks.

3.2 Personality ↔ interpersonal behavior

Although behavior is often implicated in definitions of neuroticism and other personality traits (e.g., Costa and McCrae, 1987; Clark et al. 1994), personality research does not provide many data on what individuals actually do (Funder, 2001). This may have to do with the lack of consensus concerning what behaviors to measure and the time-consuming nature of behavioral observation studies. A number of studies have been done on the relationship between neuroticism and emotional expressiveness. These generally report a negative correlation, neurotics being less able to accurately express emotions (Riggio and Riggio, 2002). One study reports that neuroticism is related to gaze aversion (Campbell and Rushton, 1978). One study, in which a range of nonverbal behaviors was measured, found that neuroticism was related to an unfriendly expression and a soft and unpleasant voice (Borkenau and Liebler, 1995). Two other studies measuring a range of nonverbal behaviors did not find any significant correlations between neuroticism and behavior (Berry and Sherman-Hansen, 2000; Spain et al., 2000). In a study of our own research group, in which mutual adjustment of nonverbal involvement behavior was measured, no associations with neuroticism were found either (Geerts et al., 2000).

In sum, neuroticism is generally supposed to be reflected in (interpersonal) behavior, but evidence for this supposition is not abundant and results vary with the kind of behavior measured.

3.3 Personality ↔ stress physiology

Both high neuroticism and HPA-axis hyperactivity are considered as reflecting increased reactivity to stress. Therefore, one would expect a positive association between these two measures. Some studies indeed found such an association. High neuroticism has been related to high basal cortisol levels (Bridges and Jones, 1968) as well as to an increased cortisol response after a challenge (Houtman and Bakker, 1991; Kirschbaum et al., 1995; Zobel et al. 2004). One study, however, found a *de*creased cortisol response after a challenge in neurotic individuals (McCleery and Goodwin, 2001). In two other studies, neuroticism was not related to cortisol levels at all, neither at baseline nor after a challenge (Roy, 1996; Schommer et al., 1999). Another study found neuroticism to be related to plasma cortisol but not to urinary cortisol (Miller et al., 1999). Thus, studies on the association between neuroticism and HPA-axis hyperactivity have yielded mixed results so far.

3.4 Personality ↔ interpersonal stress

Whereas stress reactivity is considered a main element of neuroticism, neuroticism is probably also related to stress *exposure*. A number of studies found that individuals high on neuroticism are more likely to experience stressful life events than individuals low on neuroticism (Fergusson and Horwood, 1987; Headey and Wearing, 1989; Ormel and Wohlfarth, 1991; Poulton and Andrews, 1992; Magnus et al., 1993; Van Os et al., 2001; Kendler et al., 2003). Some other studies, however, failed to find such an association (Zautra et al., 1991; Ormel et al., 2001; Neeleman et al., 2003; Saudino et al., 1997 (elderly); Oldehinkel et al., 2003 (elderly)). The type of events that is investigated also makes a difference: "person-dependent" events (events that individuals may have brought upon themselves) seem to be more strongly associated with neuroticism than "person-independent" events (events that are "bad luck" or happen due to the actions of others) (Ormel, 1983; Ormel and Wohlfarth, 1991; Poulton and Andrews, 1992). Especially dependent events that involve interpersonal relationships are associated with neuroticism (Bolger and Schilling, 1991; Gunthert et al., 1999; Ormel et al., 2004c). It is not clear *why* high-neuroticism individuals seem to be more event prone. Many authors suggest that the reason must be found in the way neurotics behave or select their environment, but there is very little explicit research done to investigate this.

A number of studies report a modulating effect of neuroticism on stressful life events; individuals high on neuroticism seem to be more sensitive to the depressogenic effects of stressful events (Ormel et al., 1989; Bolger and Schilling, 1991; Gunthert et al., 1999; Van Os and Jones, 1999; Ormel et al., 2001; Kendler et al., 2004). These studies are in line with the common notion that neuroticism acts as a vulnerability factor in the etiology of depression; it is supposed to render the individual at risk of becoming depressed especially following adversity.

To conclude, evidence that neuroticism leads to a higher exposure to stressful life events is suggestive though not entirely consistent. Neuroticism probably (also) reflects a higher sensitivity to the effects of stressful events.

3.5 Stress physiology ↔ social cognition

The widespread use of synthetic analogues of cortisol in the treatment of inflammatory diseases (e.g., prednisone) has lead to the clinical observation that corticosteroids have also important (side-)effects on cognition. Empirical

studies have confirmed this observation. Elevated levels of corticosteroids generally are detrimental to cognitive functioning, especially when exposure is sustained (Lupien and McEwen, 1997; Sapolsky et al., 2000; Wolkowitz et al., 2001; Lupien et al., 2005). Mainly arousal, attention, and memory function are affected by persistent hypercortisolism.

In the short term, elevated cortisol levels serve to increase alertness and to focus attention, which is clearly adaptive under stressful circumstances (Wolkowitz et al., 2001; Erickson et al., 2003). Acute cortisol elevations also modulate the processing of emotional stimuli (Sapolsky et al., 2000; Lupien et al., 2005). In particular, the selective processing of threatening stimuli is enhanced (Rosen and Schulkin, 1998; Erickson et al., 2003). The relationship is reciprocal; the perception of threat also enhances cortisol excretion (as, for example, in the initiation of the stress response). Usually, feedback mechanisms serve to downregulate this system, preventing it from overshooting. Prolonged or repeated hypercortisolism, however, may induce long-lasting changes in this "fear circuit", so that an increased sensitivity to fearful or other negative stimuli may persist even while cortisol concentrations return to normal levels (Post and Weiss, 1998; Erickson et al., 2003; Bouhuys et al., 2005). In such a hyperexcitable fear circuit, elevated cortisol and threat perception have an excessive and prolonged effect upon each other, which is thought to play an important role in many forms of psychopathology (Rosen and Schulkin, 1998). Thus, with regard to the cognitive processing of emotional stimuli, cortisol and cognition mutually influence each other. In pathological cases these reciprocal effects become exaggerated and longer lasting.

3.6 Stress physiology ↔ interpersonal behavior

HPA-axis hyperactivity has been associated with a typical behavioral response to interpersonal stress characterized by submissiveness, inhibition, and social avoidance (Henry, 1982; Weinstock, 1997). Animal studies, for example, have shown that high cortisol levels are related to low social status and subordinate behavior in hierarchically organized species (Sapolsky, 1999; Abbott et al., 2003). Studies of humans have shown that high cortisol is related to shyness, social withdrawal, and fearful and internalizing behavior (Kagan et al., 1988; Granger et al., 1996; Legendre and Trudel, 1996; Gunnar et al., 1997; Schmidt et al., 1997; Fernald and Grantham-McGregor, 1998; Cacioppo et al., 2000; Goldsmith and Lemery, 2000; Smider et al., 2002). It is not clear which of the

two comes first, behavioral inhibition or hypercortisolism. The relationship is probably a complex one, allowing reciprocal influences, which may be modulated by early or severe stress (Henry, 1982; Kagan et al., 1988; Gunnar, 1994; Weinstock, 1997).

Typically, most of the above studies were done in children, and in all studies global qualitative measures were used to assess interpersonal behavior. We found 2 studies with quantitative measures of discrete nonverbal behaviors (Sgoifo et al., 2003; Makatsori et al., 2004), one of which confirms the idea that inhibitory behavior is related to higher HPA-axis activation, while the other seems to do not.

To summarize, high cortisol is related to inhibitory or fearful behavior, but the causal direction of this relationship is unclear. Studies relating cortisol to quantitative measures of nonverbal interpersonal behavior are uncommon.

3.7 Stress physiology ↔ interpersonal stress

The relationship between cortisol and interpersonal stress is a rather trivial one, since cortisol is conceptualized as a "stress hormone". It is released under stressful circumstances, thus likely also after interpersonal stress. The inverse relationship is not trivial; HPA-axis (hyper-)activity may contribute to the occurrence of interpersonal stress, or modulate its effects. There is a dearth of empirical studies in this respect. We found 3 studies that related HPA-axis hyperactivity to the subsequent occurrence of stressful life events, one of which indeed found such an association (Goodyer et al., 2001), the other two reporting a negative result (Charles et al., 1989; Harris et al., 2000). We did not find studies explicitly investigating modulatory effects of HPA-axis hyperactivity on stressful life events. Indirect evidence, however, suggests that such effects are likely. Individuals with a hyperactive HPA axis show an exaggerated, inappropriate, or prolonged response to stressful stimuli (Glue et al., 1993), and there is also evidence that these individuals exhibit less efficient coping strategies (Weinstock, 1997). So, the *impact* of a stressful life event presumably varies with the sensitivity of the physiological stress system.

3.8 Social cognition ↔ interpersonal stress

The main cognitive models of depression are diathesis-stress models; they postulate that cognitive factors act as a diathesis that increases risk of depression especially in combination with stressful events (cf. Abramson et al., 2002). Stressful events serve as triggers that activate negative cognitive

schemas (Beck, 1967) or negative attributional styles (Abramson et al., 1989), leading to the onset or exacerbation of depressive symptoms (Sweeney et al., 1986; Alloy et al., 1999). Cognitive factors may also influence the way in which stressful events are perceived or interpreted. For example, a cognitive bias toward threatening signals may make that stressors are more readily attended to or more easily interpreted as aversive (Gotlib and Hammen, 1992; Mineka and Gilboa, 1998). Further, the impact of a stressful event may vary with the individual's coping style (Cronkite and Moos, 1995; Nolen-Hoeksema, 2002). Avoidant and ruminative coping styles, for example, seem to increase the likelihood that stressful events become depressogenic, while active coping styles seem to reduce this likelihood. Differences in cognitive make-up may also make that different *types* of stressful events have a differential impact. Stressors that involve interpersonal relationships, for example, are supposed to be particularly depressogenic for individuals who are predominantly focused on interpersonal relationships (which are often women) (Champion and Power, 1995; Hammen, 1999; Nolen-Hoeksema, 2002). Individuals who are focused on other fields (e.g., achievement) are thought to be more sensitive to other types of stressors (e.g., work or financial stress).

The above theories and ideas suggest that (social) cognitions may interact with (interpersonal) stress, amplifying or reducing its effects, and there is some empirical evidence for all of them. A number of studies specifically investigated the interaction between stressful life events and the cognitive processing of emotional information. These studies showed that a processing bias toward threatening information predicts a stronger response to stressful life events (MacLeod and Hagan, 1992; Van den Hout et al., 1995; MacLeod et al., 2002; Pury, 2002; Beevers and Carver, 2003; Munafo and Stevenson, 2003).

The experience of stress may also affect emotion perception. There is evidence that stress has the potential to impair accurate decoding of emotional information (e.g., Gard et al., 1982; Keeley-Dyreson et al., 1991), and to enhance the selective processing of threat stimuli (Mogg et al., 1990; Mogg et al., 1994). These findings are consonant with those regarding the influence of elevated cortisol levels on cognition (see section 3.5), so cortisol is probably the more proximate factor here. We do not discuss this option further, as our design is unfit to test it; our stress measure (stressful life events) was assessed *after* our measure of emotion perception (cf. Box 1 at the end of this chapter). That is not to say that this option is not relevant (see also our remarks in section 4.2).

Some authors have raised the possibility that individuals may *generate* interpersonal stress as a result of their dysfunctional social cognitions (e.g., Hammen, 1999; Simons et al., 1993). For example, individuals who are not able to accurately decode the other's emotional expressions are probably less able to react appropriately to the other's signals, which may lead to interpersonal problems. Indirect evidence for this idea comes from a study that showed that individuals with low emotion decoding ability are experienced by others as less warm and sympathetic (Funder and Harris, 1986). Presumably, however, dysfunctional interpersonal behavior is an intermediating variable here; deficits in emotion perception likely first lead to inappropriate interpersonal behavior, which subsequently may lead to interpersonal problems (Gotlib and Hammen, 1992; Hammen, 1999, see below).

To conclude, several theories and empirical studies suggest that social cognitions can act as moderators of the effects of stressful life events, increasing or decreasing their impact. Social cognitions may also contribute to the occurrence of stressful life events, but presumably only indirectly, via behavior.

3.9 Interpersonal behavior ↔ interpersonal stress

The notion that some individuals may generate stressful life events as a result of their own behavior has received more and more attention in recent decades (e.g., Ormel, 1980; Ormel and Wohlfarth, 1991; Hammen, 1991; Potthoff et al., 1995). Hammen and colleagues, for example, showed in a number of longitudinal studies that depressed women were more likely than other groups to experience events of the dependent type (as opposed to events that occur independently of the individual's behavior) (Hammen, 1999). Especially dependent events that involved interpersonal relationships were elevated in these women. Importantly, these stress-generation patterns also occurred outside of periods of depression, were related to poor social problem-solving skills, and precipitated further depression.

The stress-generation theme is also an implicit element of the theories of Coyne and successors (cf. section 2.3). These propose that depressed and depression-prone individuals, because of their behavior, generate an array of interpersonal and other problems, which lead to onset, maintenance, or recurrence of depression (e.g., Coyne, 1976; Coyne et al., 1991; Segrin and Abramson, 1994; Coyne, 1999; Joiner, 2000; Joiner, 2002). The evidence for these ideas is suggestive, but mainly correlational in nature. Thus, depression,

dysfunctional interpersonal behavior, and interpersonal problems clearly covary, but evidence that dysfunctional behavior is also *antecedent* to interpersonal problems and subsequent depression is sparse (Marcus and Nardone, 1992; Segrin, 1998; Segrin, 2000; Joiner, 2000; Ormel et al., 2004a).

A few studies have prospectively linked interpersonal behavior to the occurrence of stressful life events (Potthoff et al., 1995; Davila et al., 1995; Segrin, 2001; Shahar et al., 2004). All but one (Segrin, 2001) found that dysfunctional interpersonal behavior or poor social skills indeed contribute to the occurrence of stressful life events. Interpersonal behavior was assessed by means of interviews or self-report questionnaires in these studies. There are no studies that relate observational measures of interpersonal behavior to the subsequent occurrence of stressful life events.

The opposite may also hold; the occurrence of stressful events likely also influences interpersonal behavior (e.g., Slane et al, 1980; Burgoon, 1985; Lehman et al., 1987; Pagano et al., 2004). We will not review the research on this topic, because the reverse relationship is more directly relevant to our case (stressful events being measured *after* the behavioral assessments; see Box 1).

In the tradition of diathesis-stress models of depression, some authors have proposed that dysfunctional interpersonal behavior might represent a diathesis for depression that increases risk only in combination with stressful events (e.g., Segrin and Flora, 2000). The reasoning behind this model is that people with good social skills can arrange sufficient social support, which buffers the effects of stressful events. Two studies (Frye and Goodman, 2000; Segrin and Flora, 2000), using self-report measures of social skills, provide some evidence for this suggestion. Another study, however, failed to find such a modulating effect of interpersonal behavior on stressful life events (Shahar et al., 2004). No studies with observational measures of interpersonal behavior are done on this topic.

In conclusion, although no studies with observational measures of non-verbal behavior exist that relate dysfunctional interpersonal behavior to the subsequent occurrence of stressful life events, such a relationship is plausible given existing theories and related empirical evidence.

3.10 Social cognition ↔ interpersonal behavior

Social cognition researchers for a long time focused on social cognition to the neglect of interpersonal behavior (Fiske, 1992). In recent decades, however, it

has become increasingly acknowledged that "social cognition is for social doing" (Fiske, 1992; Ostrom, 1994; Bandura, 2001). Concurrently, theories have begun to emerge that try to integrate cognitive and behavioral accounts of depression (e.g., Lewinsohn et al., 1985; Gotlib, 1992; Gotlib and Hammen, 1992; Patterson, 1995). In these theories also, cognitions are supposed to influence behavior. Activation of negative cognitive schemas, for example, is thought to lead to enhanced display of dysfunctional interpersonal behaviors (Lewinsohn et al., 1985; Gotlib and Hammen, 1992). Interpersonal behavior may also feed back on cognition according to these theories, but via the interpersonal consequences of that behavior (e.g., dysfunctional interpersonal behavior may induce aversion and rejection in others, the perception of which may reactivate negative cognitive schemas, *et cetera*; Lewinsohn et al., 1985; Gotlib and Hammen, 1992).

Empirical studies that relate social cognition to interpersonal behavior have become more frequent in recent decades as well, and the common notion in these studies also is that cognitions influence behavior. Focusing specifically on the relationship between the decoding of emotional expressions and interpersonal behavior, we found a number of studies done in children and schizophrenic patients (Penn et al., 1996; Most and Greenbank, 2000; Schultz et al., 2000; Hooker and Park, 2002). These studies report that lower decoding ability is related to poorer social functioning. Interpersonal behavior was assessed globally in these studies, by means of qualitative ratings of classroom or ward behavior by teachers or psychiatric nurses. Studies relating social cognition to quantitative measures of nonverbal behavior are rare. We found 3 studies. Dovidio et al. (2002) measured implicit racial prejudices using a cognitive priming task and related these to nonverbal and verbal friendliness behavior during interracial interactions. A cognitive racial bias appeared to go together with a similar bias in nonverbal friendliness, but not in verbal friendliness. Holland et al. (2004) investigated whether cognitive conceptualizations of the self (independent vs. interdependent self-concepts) influence interpersonal proximity in a waiting room. They found that a more independent self-concept was related to greater spatial distance between interactants. Geerts and Bouhuys (1998) investigated whether depressives' perception of negative emotions was related to the degree of mutual adjustment of nonverbal involvement behavior during a conversation, but found no such relationship.

To summarize, both theory and empirical evidence suggest that social

cognitions influence interpersonal behavior. Evidence based on studies using quantitative measures of nonverbal behavior is limited.

4 HYPOTHESES

On the basis of the literature reviewed above we have formulated the hypotheses that will be tested in the remainder of this thesis. We first present our hypotheses regarding the raw (univariate) relationships of the various factors to recurrence of depression, which are rather straightforward. Our hypotheses regarding the multivariate prediction of recurrence including factors from all 5 research fields are more explorative in nature.

4.1 Univariate prediction of recurrence

We expect that recurrence of depression can be predicted from:
1. Personality: high neuroticism
2. Social cognition: impaired emotion perception or a negative perceptual bias
3. Interpersonal behavior: poor nonverbal communication (lack of adjustment of nonverbal involvement behavior)
4. Stress physiology: HPA-axis hyperactivity (high cortisol levels)
5. Interpersonal stress: stressful life events (of the interpersonal type)

4.2 Interplay of putative determinants in the prediction of recurrence

Below, we do a proposal for a model on how the factors from the 5 different fields jointly may explain risk of recurrence of depression. The question of what neuroticism actually is and how it confers risk of depression is a leading issue here (see section 1). We are, however, also interested in the interplay as such of the various other factors in the prediction of recurrence. In our model, we show how neuroticism may be linked to recurrence by specifying intermediating and modulating effects of the factors from the other 4 fields. The literature regarding interfield relationships may not be always fully applicable to the present case (e.g., evidence often concerns healthy or depressed samples instead of remitted samples, interpersonal behavior is rarely assessed by direct observations of nonverbal behavior), it did give some direction to our thinking. Our proposal is based on what is most likely given this literature. In case there is plenty evidence from studies focusing on the specific factors of our interest, we relied on this evidence. In case there is not, we fell back on broader evidence and relevant theories.

Our model has certain constraints related to the study design (see Box 1). Stressful life events were assessed during the follow-up of our study, thus *after* the baseline measurements in which the other variables were assessed. Therefore, considering interpersonal stress or stressful life events as an *antecedent* of (changes in) the other variables is not suitable here. That means, we do not deny that stressful life events may have effects on the other variables, but we refrain from implementing such potential effects in our model as they are not testable within our design. For the same reason, we do not consider feedback loops in our model, although these are likely and should be included in a more complex model of depression which also includes long-term effects (cf. Gotlib and Hammen, 1992; Joiner, 2002).

The starting point of our model is neuroticism, this personality dimension so consistently associated with all kinds of distress and adversity while nobody knows for sure why. We expect that this elusive concept can be substantialized by measuring interpersonal behavior, social cognition, and stress physiology (see Figure 1). Specifically, we expect that high neuroticism becomes manifest in poor nonverbal communication, negative emotion perception, and HPA-axis hyperactivity (1). We suppose that each of these factors explains part of the risk of recurrence associated with neuroticism. Together, they may also explain why neurotic individuals are more prone to experience stressful life events, which further explains their increased risk of recurrence: We expect that HPA-axis hyperactivity and negative emotion perception mutually reinforce each other (2). Further, we expect that negative emotion perception leads to problems in nonverbal communication (3), and that problems in nonverbal communication contribute to the occurrence of stressful life events of the interpersonal type (4), which subsequently trigger depression. We also expect that some factors modulate the effect of other factors. Specifically, we expect that the effect of stressful interpersonal events is amplified by HPA-axis hyperactivity and negative emotion perception (5).

OUTLINE OF THE THESIS

Chapter 2 describes a pilot study on the prediction of depression relapse on the basis of interpersonal behavior. This study concerns a different sample than the one investigated in the rest of the thesis, and had a slightly different design. The sample consisted of inpatients who were just discharged from the

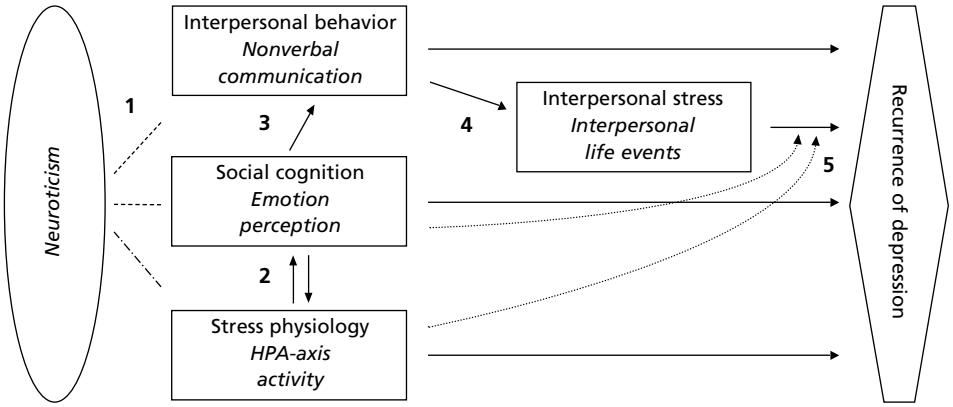


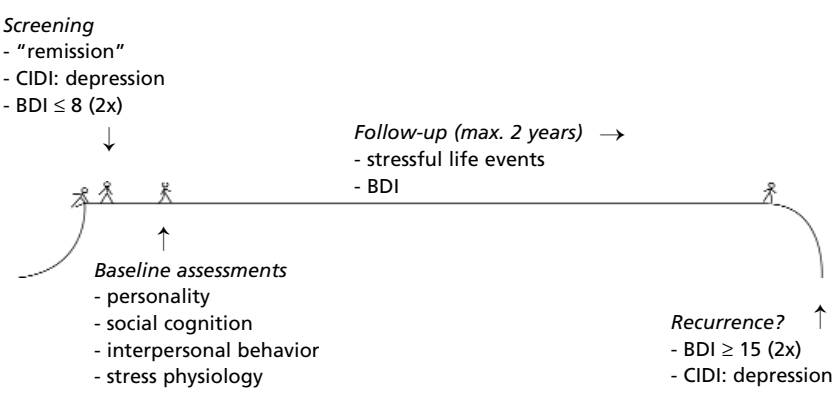
Figure 1 A multifactorial model of recurrence of depression. **1**. The personality dimension of neuroticism is reflected in poor nonverbal communication, negative emotion perception, and HPA-axis hyperactivity, each of which increases risk of depression. **2**. HPA-axis hyperactivity and negative emotion perception mutually reinforce each other. **3**. Negative emotion perception leads to poor nonverbal communication. **4**. Poor nonverbal communication contributes to the occurrence of stressful life events of the interpersonal type (which trigger depression). **5**. The effect of interpersonal events is amplified by negative emotion perception and HPA-axis hyperactivity.

hospital (n = 51). The follow-up was 6 months.

The Chapters 3 through 6 report on studies of a sample of remitted outpatients (n = 104), who were followed 2 years (see Box 1). *Chapter 3* investigates whether participants with a *history* of recurrent depression differ from participants with a single previous episode, as regards personality, social cognition, and stress physiology. *Chapter 4* deals with future recurrence, in particular with the question of whether it can be predicted from social cognition and stress physiology. *Chapter 5* describes a study on the prediction of recurrence from interpersonal behavior and personality. *Chapter 6* is also on the prediction of recurrence from interpersonal behavior, but now the question of whether stressful interpersonal events play an intermediating role in this relationship is investigated. In *Chapter 7* the findings are integrated and evaluated. This is done on the basis of the model that was presented above. In *Chapter 8* some implications for future research and clinical practice are discussed.

Box 1 Global study design

The study described in this thesis had a prospective longitudinal design with a 2-year follow-up. The determinants of interest were assessed at baseline, when the depression was in remission, and used to predict subsequent recurrence (see the figure below). Participants were outpatients recruited from mental health care centers in the northern part of the Netherlands, suffering from major depressive disorder or dysthymic disorder. They were asked to participate in the study if their psychiatrist considered them remitted from a depressive episode. We confirmed the psychiatrist's diagnosis by means of the Composite International Diagnostic Interview (CIDI, lifetime version; World Health Organization, 1997), a structured interview with good reliability and validity (Andrews and Peters, 1998). Remission was established by means of the Beck Depression Inventory (Beck and Steer, 1987), a widely used self-report depression scale with good psychometric properties (Beck et al., 1988). Participants were considered remitted in case the BDI score was 8 or less for 2 consecutive times, with a 4-week interval in between (following the consensus definition of Frank et al., 1991). Remitted participants (n = 104) underwent a series of baseline measurements on potential predictors of recurrence. These included self-report questionnaires to assess personality, computer tasks to assess social cognition (emotion perception), and videotaped interviews to assess interpersonal behavior (nonverbal communication). Stress physiology was assessed by measuring 24-h cortisol levels from urine samples that were collected the day after the baseline session. Stressful life events, which serve as a measure of (interpersonal) stress, were assessed over the whole length of the 2-year follow-up. Every 4 weeks during the follow-up, participants completed the BDI. They were suspected to have a recurrent episode if their BDI score was 15 or more for 2 consecutive times (cf. Frank et al., 1991). Recurrence was further established by means of the CIDI (12-months version).



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