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Classification and functional approach of emotional disorders: Psychomotor symptoms and personality pathology

Classificatie en functionele benadering van emotionele stoornissen: psychomotorische symptomen en persoonlijkheidspathologie

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Classification and functional approach of affective-spectrum disorders: Psychomotor symptoms and personality pathology
Classificatie en functionele benadering van stemmingsstoornissen: Psychomotorische symptomen en persoonlijkheidspathologie

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Minding the Gap

"I suppose it's tempting, when the only tool you have is a hammer, to treat everything as if it were a nail."[1]

Abraham Maslow-

[1] *Note. (2021). Mind the gap sign on of Moorgate underground station platform of London Underground, UK. (graphic). Retrieved 4/2/2022 from https://assets.adobe.com/libraries/urn:aaid:sc:EU:6b365e10-18c0-4455-8e00-b50824f9fb1d/a34240fd-beed-4a9d-8c1c-7a010527a3c6*

Table of Contents

General Introduction

Preface

This thesis is first and foremost the product of my work as a clinical psychologist, working in a psychiatric ward responsible for the treatment of patients with affective spectrum disorders, i.e. patients with (persistent) depression and bipolar disorder. This thesis is the natural outcome of my clinical interest in this patient group. Out of the many characteristics that typify these patients, two stood particularly out for me. The first was the psychomotor retardation (PR) that is so typical in this patient group. The second was the purported role of severity of personality disorders (also referred to as personality pathology) as a predictor of onset, course, and treatment response in these disorders.

Even if the importance of these two characteristics is recognized for the creation of an effective clinical treatment plan, it is highly unfortunate that they are too often considered as belonging to different or even separate clinical perspectives, symptom/syndrome-based versus process-based approaches, between which the best choice has to be made (e.g., Billieux et al., 2014).

Obviously, the two approaches are different. The symptom-based disorder-specific approach certainly has its merits, but also recognized limitations, that can partly be overcome by a complementary process-based approach (see Bentall, 2003 for a critical discussion). Still, the symptom-based perspective remains a necessary precondition for a process-based approach in treatment of psychiatric disorders, by certifying that symptoms signaling diseased biological states, are properly observed, recognized, and measured in a reliable way. 'Adequate and faithful distinctions in the phenomenal or experiential realm are therefore a fundamental prerequisite for classification, treatment, and research.' (Parnas, Sass, & Zahavi, 2013). In the medical field, it would not be justified, for instance, to work with an 'individually experienced' temperature of the body. In the symptom-based perspective, I opted for the thorough and meticulous study of PR within a well-controlled context. More specifically, I studied the PR-symptom dimensions, i.e., the initiation time, the motor time, and the re-inspection time of drawing tasks in an elderly population and in particular in depressed elderly patients who were medication free. In elderly patients, the role of PR is more prominent than in younger patients, certainly in comparison to the expression of mood complaints, but is frequently explained as a 'normal' sign of biological aging, lacking motivation by grief over different types of loss, or as caused by medication. I, however, deliberately chose for PR because this symptom optimally serves the purpose to prove that the critical appraisal of symptoms should not exclusively rely on the reporting of the patient. It is indeed a well-known phenomenon that the extremely psychomotor

retarded psychotic depressive patient reports that there is nothing wrong, that he is only too lazy and not cooperating because he is a bad person.

The process-based approach, which is the result of criticism on the limitations of the classification approach or disorder specific approach, "supports the development and validation of individualized and transdiagnostic treatments targeting specific psychological factors underlying symptoms and problematic behaviors (which are identified through individual case conceptualization) and criticizes the adoption of standardized treatments targeting discrete syndromes (Dudley et al., 2011)" (Billieux et al.,2015, p3).

To introduce the process-based approach more concretely I will start from an exemplary individual case. Such an introduction is necessary as this approach can only be realized with a person-centered method comprising Phenomenological unfolding (explanation of the patients' field of experience), Hermeneutic analysis (the explanation of the patients' position-taking toward their experience) and Dynamic analysis (the explanation of the life history in which experiences and position-taking are embedded), i.e., a PHD-method (Messas et al., 2018). Adopting this method and following the process-based approach, I did research on the level of personality functioning, starting from the subjects' representations of their own intersubjective matrix, thereby following the theory of Blatt (2008) and colleagues.

The overall purpose of my contribution is to open new perspectives in the approach to psychopathology by bringing together two – apparently – very different approaches from different fields. Linking a symptom/syndrome-based approach starting from descriptive classification and a process-based approach considering subjective representation will reveal that the two research exercises are not contradictory, but consistent and complementary. The symptom-driven approach starting from descriptive classification categories ends up in the study of associations with clinical utility and the functional classification approach starting with a description of functional processes from a contextualized and subjective point of view ends up identifying observable dimensions and signs in these processes. The two-pronged approach in this thesis ultimately endeavors to reveal the potential of reciprocal corroboration and strengthening of both research and clinical practice.

General Outline

This thesis will follow two common and prototypical clinical paths into the study of affective-spectrum disorders and depression in particular, with each path introduced by a prototypical case.

In PART I (chapter 1) we follow the first, symptom descriptive path of the study by investigating one, exemplary symptom of depression in elderly, namely psychomotor retardation (PR) and we will show how a well-controlled observation of a symptom in all its critical dimensions in relationship to other symptoms may lead to the identification and understanding of functional pathological processes that are treatment relevant.

We will start with the necessary state of the art of the study of PR in depressed elderly (1.1). Studies on major depression in elderly are scarce, especially in non-medicated patients and patients with monotherapy. As PR is also intrinsically linked to aging, to medication use and to all kinds of physical comorbidities, the symptom is relatively complex in an elderly population. Therefore, we will study different dimensions of the symptom. Motor functioning can be slower, by different possible dimensions of PR, the initiation time, the executive motor time, and the reinspection time.

Then, we will compare PR in 20 medication-free depressed elderly and 20 matched controls, and study associations with cognitive functioning and mood symptoms (1.2). We will check whether depressive slowing is more extreme than normal slowing in elderly and whether depressive slowing is related to specific aspects or dimensions of slowing.

Next, we discuss the follow-up of this group of depressive elderly and matched controls after a treatment with 5-20mg of escitalopram (1.3), a first choice Selective Serotonin Reuptake Inhibitor (SSRI) in the treatment of depression in elderly. We will study how psychomotor impairment decreases with treatment relative to other symptoms of mood and cognition at week 2, 6 and 12 after baseline measurement.

In PART II (chapter 2) we take the second, functional or process-related path and we start from the treatment-relevant concept of severity of personality disorder, working backwards to end up in defining features, signals or symptoms that give indications for psychotherapy, especially in emotional disorders, affective-spectrum disorders (e.g., major depression, bipolar depression, …) as well as emotion regulation disorders (e.g., descriptive borderline personality disorder)

The introduction (2.1) is devoted to the assessment of personality disorders. We will indicate how problems with the categorical definition of personality disorders created the need of a new, hybrid dimensional approach to personality disorders, as shown in section III of DSM-5 (American Psychiatric Association, 2013), where apart from traits, the definition of a personality disorder itself is at stake. For this thesis, not the dimensionality is the final endpoint, but the associated processes to which the dimensions refer. Even if much attention has been given to a better delineation of different personality disorders and to the subtyping of personality disorders, more important for treatment are severity criteria and the differentiation from normality on the one hand and the differentiation from or interaction with other psychopathology such as affective-spectrum disorders on the

other hand. As severity of personality disorders in DSM-5 section III (American Psychiatric Association, 2013) is defined as the level of personality dysfunction, consisting of the interacting dimensions of self and relatedness, we will focus our research on personality functioning as the interaction of the two constituent dimensions. Yet, the hybrid system with traits and level of personality functioning has not been retained as the official approach to personality disorders because more research is needed into the personality functioning model.

Therefore, we perform a study on the validity of the object-relational model of personality functioning (2.2) by means of the Differentiation and Relatedness Scale (DRS), an instrument to assess the level of personality functioning with ratings of representations of self and significant others. In this study, we specifically focus on the validity of this instrument in a normal population of young adults to find out whether normal students can be differentiated based on their level of personality functioning, and whether a similar linear relationship can be found with symptoms as in a population of severe personality disorders. Associations with gender and relational functioning will be considered.

The DRS is also applied in a population with less severe personality pathology, namely an inpatient population with general psychopathology, especially affective-spectrum disorders (2.3). Here we want to find out whether a similar linear relationship exists between symptoms and personality functioning as in the population with severe personality disorders. As we also want to differentiate effects of personality pathology and clinical distress, we investigate this relationship with the Inventory of Personality Organization (IPO), a well-known and validated instrument for the assessment of severity of personality disorders, and we look at associations with symptoms of depression, dissociation, and clinical distress. We also look at associations with coping and relational functioning in this population. Here too, we control for possible gender differences.

We then study the structure of personality disorders (2.4.) by comparing the model fit of different models, with special attention for a possible general p-factor of psychopathology. We thereto compare the model fit of a model with only one p-factor subsequently with a model with correlating personality disorder cluster factors, with a hierarchical model and with a bifactor model with the p-factor and the specific cluster factors, to learn more about the best approach to personality disorders and to evaluate the possibility of transdiagnostic approaches to personality disorders.

Finally, in the general discussion (chapter 3), we discuss the main findings of the two research parts (3.1., 3.2.), indicate avenues of their integration (3.3.) and suggest directions for future research. Also, of key-importance in this research, we try to derive some practical advice for clinicians dealing with patients with depression and affective-spectrum disorders in general, and we discuss a different approach to personality disorders and formulate possibilities to ‘bridge the gap’ between the descriptive symptomatic approach and the

functional approach of chronic affective-spectrum disorders (3.4.). We finally provide helpful appendices and a short summary.

Chapter 1

Part I: Cognitive and psychomotor retardation in late-life depression[2]

[2] *Note. (n.d.). traffic sign for paying attention for elderly people (graphic). Retrieved 4/2/2022 from https://assets.adobe.com/libraries/urn:aaid:sc:EU:931bf7c9-e698-452e-81a8-a328ac2cc6e1/fe35e9a2-3f37-4f03-a24b-114cc1ba548c*

Lily

Lily (age 72) was brought to the hospital on a stretcher. She had suddenly become very anxious and agitated at home and reacted hardly in interaction. Her sons then organized an admission to a hospital ward for her and her husband. Her husband was suffering from Alzheimer disease, and she had been taking care of him for years. After the admission, she showed strikingly changing moods. At times, she was confused and agitated with a fear of a deadly virus that 'would kill us all'; other moments of the day, she appeared alert or sedated. However, reality testing was not always intact. Sometimes she believed that her husband was dead, and even that she herself was dead. Brain imaging showed no alarming signs or important abnormalities, however.

When I met her with her sons, she was more stable but significantly slowed down. Her children had noticed a steady but significant impairment in functioning over the last year. They also had noticed that she had become more indecisive and was more soliciting the children; she really needed their proximity in case something would go wrong. She got worse in problem solving and ruminated a lot, could not let worries go anymore. She needed being comforted and would crash or freeze. A little later, she also stopped going out to social activities, and started isolating herself from other befriended people. Everything was getting more difficult and cumbersome for her. However, until 6 weeks before the admission, she still drove her car, performed household tasks, went to the groceries store together with her demented husband and still cooked some dinner for herself. Then, she started to lose weight extremely; she stopped cooking and could not think clearly enough anymore to make the shopping list. That was the first time she needed residential care, although she had known two depressive episodes before in her life, each time with a remission of twenty years between the episodes. During those depressions, she had been lying on the coach all day. Her husband had then taken leave of absence to take care of her. She had been continuing a sustaining pharmacological treatment for years and more recently, she had contacted a psychologist.

The last five years she had been taking care of her husband. For a long time, she had not had a decent night rest because her husband was wandering around at night before he finally got a sleeping pill. The last two years he could not talk anymore and the last couple of months, he was unable to stand up. It was only then that she accepted help from a

home care nurse. Two weeks after her admission in hospital, her husband died in a nursing home. In her psychomotor retarded depressive state, she was not able to cry and feel the sadness over the loss. That only came later. The moment she could cry for him and feel the loss, she was already getting out of depression.

The two had always had a romantic relationship, apart from the normal struggles of a relationship. She had lost her father from a stroke when she was only 12 years old. That was for her a terrible loss, as her father meant everything to her. At that age, as an only child, he was also her playmate. The sudden death had been traumatic. She had not been allowed to go to the funeral due to circumstances then. Because his death occurred right after the war, her mother had no time to spend to come to terms with the loss together with her daughter, as all energy was needed to make ends meet. However, this phase did not last a long time; her mother quickly remarried, also and foremost because she needed support. However, her stepfather, a widower with whom she had a reasonably warm relationship, also had one son and so the two grew up together. The moment he had to leave the house to fulfill his duty in the army, she missed him enormously, the two then realized what they meant for each other, and they started a relationship and later a family. It was a happy family with two great sons and, later, with great grandchildren. She had never experienced significant problems in the relationships with her husband or with the children. Also, being a teacher, she had always lived a socially fulfilling life, with pleasant hobbies such as guiding cultural visits.

1.1 Introduction: Psychomotor retardation in depressed elderly

Psychomotor retardation (PR) is an objective slowing of physical movements and emotional reactions or agitation as a symptom of depression that involves cognitive and motor impairments. Clinical descriptions of PR usually comprise disturbances in speech, facial expression, fine motor behavior, gross locomotor activity, or ideation (Bennabi, Vandel, Paraxanthis, Pozzo, & Haffen, 2013), but also eye movements (Mahlberg, Steinacher, Mackert, & Flechtner, 2001), postures or self-touching (Bennabi et. al., 2013). PR is referred to by Parker as a trunc of non-interactiveness with two branches, a retardation, and an agitation branch (Parker et al., 1993) (Figure 1).

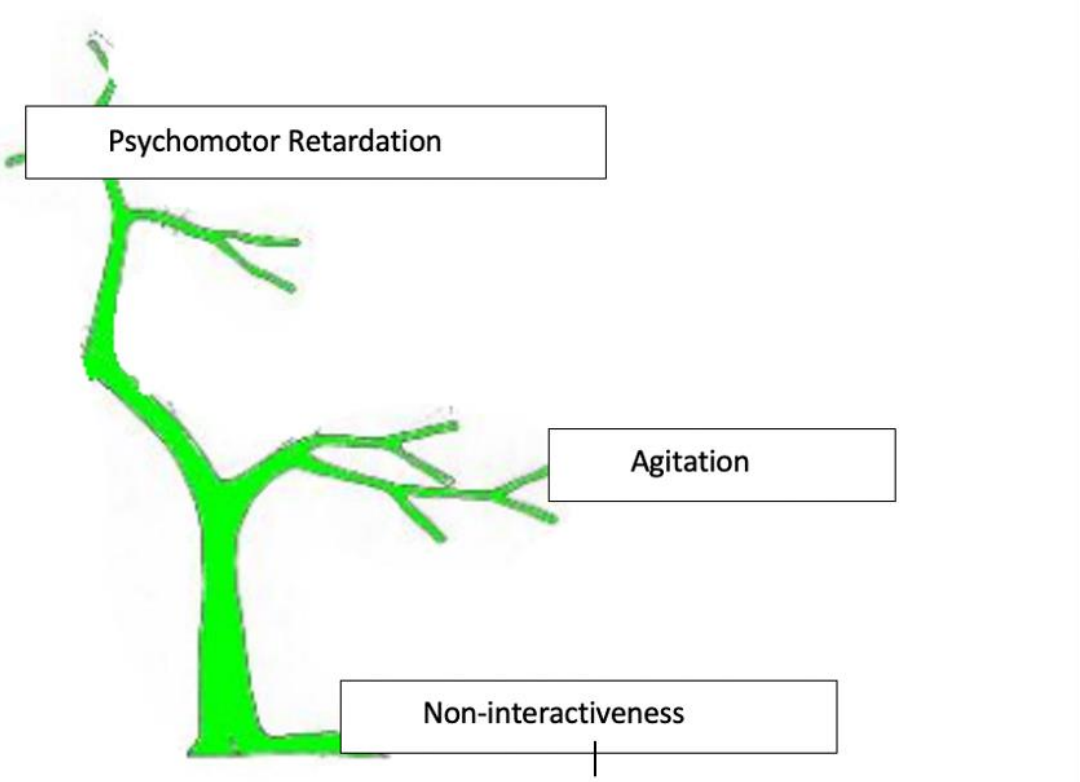

Figure 1 Psychomotor retardation and agitation as a major and minor branch respectively of a common trunc Non-interactiveness (Parker et. al., 1993).

1.1.1 The renewed interest in psychomotor retardation

PR was already described in antiquity by Hippocrates and later by Darwin. Kraepelin described how it was considered even more prominent than depressed mood as a symptom of depression and how it affected speech, thought, and behavior (Buyukdura, Mcclintock, & Croarkin, 2011). PR was recognized as an important feature of melancholia from the 17th until the beginning of the 20th century, but the symptoms of PR subsequently lost their status as core features of depression (Schrijvers et al., 2009), as is still notable in DSM-5 (Kendler, 2016). However, since the end of the previous century, many authors again acknowledge the central significance of PR for depression (e.g., Buyukdura et al., 2011; Bennabi et al., 2013; Schrijvers, Hulstijn & Sabbe, 2008). After the first review in 2008 by Schrijvers, Hulstijn & Sabbe, two systematic reviews also acknowledged PR as a diagnostic, pathophysiological and therapeutic tool in depression: one by Buyukdura, Mc Clintock and Croarkin in 2011 and one by Bennabi et al. in 2013. We refer to these papers for an overview of publications, measures, and diagnostic tools. PR is now also proposed to be incorporated as a transdiagnostic dimension in the Research Domain Criteria (RDOC), a new classification system in the search for transdiagnostic factors linked to specific brain circuits, (Bernard & Mittal, 2015; Peralta & Cuesta, 2017), because different aspects of psychomotor functioning are associated with different brain circuits across psychiatric diagnoses (Bernard & Mittal, 2015). Indeed, PR is not exclusively related to depression. In studies on treatment response and remission in Major Depressive

Disorder, Vrieze and colleagues (Vrieze et al., 2013) found PR to be a distinct factor apart from negative affect and anhedonia but failed to find a relationship of PR with treatment outcome, in contrast with the other two factors that were clearly related to treatment.

1.1.2 Prognostic and pathophysiological significance of psychomotor retardation

PR is helpful in the determination of differences between subtypes, between unipolar and bipolar depression, between major depression and the melancholic or psychotic subtype. It has, moreover, prognostic, and pathophysiological significance (Schrijvers et al., 2009). It is prognostic for treatment resistance, for differential effects of psychopharmaca, and it can be affected by or show overlap with chronic illnesses. For instance, Calugi and colleagues (Calugi et al., 2011) found that depressed patients with lifetime PR were more likely to have a longer duration of illness, likeliness of suicide attempts, an earlier age of onset, more depressive symptoms, and higher indicators of bipolarity than non-retarded depressed patients. However, PR in bipolar disorder differs from PR in unipolar depression. Bipolar PR is more related to motor control while PR in unipolar major depression is more associated with cortical regions of premotor programming. Thus, different neural impairments converge in phenotypically similar manifestations (Cantisani et al., 2016).

1.1.3 Psychomotor retardation as a distinctive criterion of depression in elderly

Brain aging is not uniform (Laks & Engelhardt, 2010) and the role of PR in depressed elderly is more prominent than in younger depressed patients. Brodaty et al. (Brodaty, Luscombe, & Parker, 1997) concluded that there were robust phenomenological differences in PR between elderly and younger patients, and even if research on the subject is still scarce, PR is obviously more prevalent in late-life depression (e.g., Butters et al., 2004). Interaction of depression and aging may also result in a more pronounced PR in the elderly (Pier, Hulstijn, & Sabbe, 2004). Depression causes prominent functional psychomotor limitations in the the elderly, between 58% and 82% more than in the elderly without depression, depending on the motor performance test used. The association between depression and functional psychomotor disability is stronger for walking and chair stand tests, for instance, and weaker for handgrip strength (Santos, Fernandes, Reis, Cocha, 2012). In addition, poor test performance on diverse cognitive tests involving psychomotor ability, such as memory tests, figure classification, clock drawing and block design tests in 85-olds, was mainly associated with PR (Pállsson, Johansson, Berg & Skoog, 2000). While PR in younger patients is mainly related to severity of depression, PR in elderly also appears in atypical clinical presentations of depression such as subsyndromal depression (Judd, Rapaport, Paulus, & Brown, 1994) or the depression-dysexecutive syndrome (Alexopoulos GS, Kiosses DN, Klimstra S, Kalayam B, & Bruce ML., 2002). That depression-dysexecutive syndrome is a typical presentation of late-life depression with reduced fluency, impaired visual naming, PR, loss of interest in activities, and paranoia, but it is a rather mild vegetative syndrome as

defined by Alexopoulos (2005). In the present clinical presentation of depression, PR is assumed a manifestation of impaired executive functioning due to fronto-limbic and fronto-striatal abnormalities. These are responsible for executive dysfunction as well as for mood dysregulation. More recently, however, in a meta-analytic study on the depression-dysexecutive syndrome hypothesis, the hypothesis that vascular damage in pre-frontal circuits results in depression with executive dysfunction and that this executive dysfunction is prognostic for treatment resistance was called into question, because medication response depended on only one executive skill but also on four non-executive skills (Mc Lennan & Matthias, 2010). PR, on the contrary, is an independent predictor of relapse in major depression in elderly depressed, without occurrence of stressors or physical illnesses (Kivelä, Viramo, & Pahkala, 2000). In summary, PR is one of the distinctive criteria of depression in elderly. It can moreover be specified that in patients with late-life depression no differences in PR could be noticed between early-onset and late-onset depressive patients (Hegeman, Kok, Vander Mast, & Giltay 2012).

1.1.4 The role of psychomotor retardation in the conceptualization of depression in elderly: five hypotheses

Currently, there are five predominant hypotheses concerning the more prominent PR-symptoms in elderly depressed. They are: 1) the vascular depression hypothesis, 2) the depression-aging interaction hypothesis, 3) the inflammation hypothesis, 4) the degenerative hypothesis and 5) the bipolar hypothesis. Because these hypotheses are important for the conceptualization of PR in geriatric depression, we related some of our research questions (2.2. and 2.3.) to the background of these five hypotheses. In our two studies on PR, we had the unique opportunity to observe PR related to depression in elderly, sorting out effects of depression, aging, deterioration and medication. A sophisticated empirical observation of behavioral effects with the use of different neuropsychological performance tasks, apart from self-descriptive questionnaires and rating scales, enabled us to test some of the predictions related to the five hypotheses on the role of PR in geriatric depression. The results concerning these predictions may prove significant in the discussion about the conceptualization of depression in elderly. The controlled study of PR in relation to depression in elderly served as an 'in vitro' study in this thesis.

In the present study, we wanted to explore the nature of PR specific for depression in elderly by a refined behavioral analysis in stringently controlled conditions and by evaluating the effect of treatment with SSRI as monotherapy. After a long search, we were able to collect a group of depressed elderly, who were psychotropic-medication-free at baseline. Their medication-free status was needed to explore the confounding nature of depressive PR in elderly, and to compare it to PR in healthy elderly. It is important to stress that this population is exceptional. Elderly depressed patients are usually prone to polypharmacy and diverging medication may have different and differentially interacting effects. Since the study population is exceptional, the findings based on this population will

be hard to apply to all elderly depressed patients. However, it serves a useful aim, that of better understanding processes of physiopathology. In that respect, it illustrates the importance of prioritizing specific methodological strategies depending on the research goal. Clearly, to evaluate the effect of a medication on PR, a baseline with patients on medication would have been very confounding, as also the effect of the medication in elderly is more marked than in a younger population.

At the outset, this study was designed as a multi-center study with seven participating psychogeriatric hospitals in Belgium, but it soon turned out that because of comorbidity and multi-pharmacology not a single inpatient could be included. Subsequently, in care homes, most of the participants registered for inclusion had to be excluded too after or during the first assessment because of strongly invalidating disability. The study ended up with a collection of almost exclusively outpatients, referred by their general practitioner or their psychiatrist. The eventual selection of the population showed less degeneration and disability. But even from the eventual selection, four patients had to be excluded because of the sudden appearance of physical or somatic disease after initial testing. The research priority, however, was to distillate the neuropsychological process of depression in elderly with the least possible confounding factors. Thus, even if this selection makes the study somewhat artificial, comparable to an in vitro test or a laboratory result, it serves a complementary purpose to vast naturalistic studies or large population research with, in turn, assessments that are more restricted. Like in other studies comprising broader assessments of depressed elderly, the comparably restricted sample number in our study allows a more extensive in-depth analysis.

Given the generally recognized significance of PR in the classification, assessment, and treatment of elderly depressed patients, it was indicated to relate the etiopathogenesis of depressive mood and PR in our specific population to the five predominant hypotheses mentioned before. The possibility of controlling for a number of confounding factors in our research method was bound to yield new insights. The two studies we performed on PR in our specific population of elderly will be reported on in the following two chapters. In the ensuing discussion of these results, we intend to elaborate on the differentiation of PR caused by age and by depression and on the processes underlying the symptom of PR in elderly depressed patients. Eventually, the discussion of the results may lead us to implications for the treatment of patients with or without PR, considering confounding factors.

PSYCHOMOTOR RETARDATION IN ELDERLY UNTREATED DEPRESSED PATIENTS

[3]

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[3] *Note. (n.d.). Despairing senior man (graphic). Retrieved 4/02/2022 from https://assets.adobe.com/libraries/urn:aaid:sc:EU:931bf7c9-e698-452e-81a8-a328ac2cc6e1/b468a3fb-2fc4-4401-bdf3-06fa118b973c*

1.2 Psychomotor retardation in elderly untreated depressed patients.

1.2.1 Abstract

Background: Psychomotor retardation (PR) is one of the core features in depression according to DSM-5 (American Psychiatric Association, 2013), but also aging causes cognitive and psychomotor slowing. This is the first study investigating PR against the background of cognitive functioning and the concomitant effect of depression and aging in a geriatric population ruling out confounding effects of psychotropic medication.
Methods: A group of 28 non-demented late-life depressed elderly is compared to a matched control group of 20 healthy elderly by whom clinical depression measures as well as cognitive measures of processing speed, executive function and memory were assessed and set out against clinical ratings and objective experimental measures of PR with computerized fine motor skill-tests. Statistical analysis consisted of a General Linear Method (GLM) multivariate analysis of variance to compare the psychomotor and cognitive outcomes of the two groups.
Results: Patients performed worse on all clinical, cognitive and PR measures. Both groups showed an effect of cognitive load, but the influence was significantly larger for patients than for healthy elderly except for the initiation time. Comparison with a younger depressive group and a younger control group indicated interaction effects of depression and aging.
Limitations: Only a relative limited sample size was obtained due to the restrictive inclusion criteria.
Conclusion: With a medication free sample of patients, older than 65, an additive effect of depression and aging on cognition and PR in geriatric patients was found. Moreover, this effect was also independent of demand of effort (by varying the cognitive load) and thus not a motivational slowing effect of depression.

Keywords: Depression, Elderly, Psychomotor, Cognition, Drawing Tasks, Neuropsychological assessment, Medication free

1.2.2 Introduction

Apart from a depressed mood and lack of interest, psychomotor symptoms are also core features of a major depressive episode (DSM-IV-TR, American Psychiatric Association, 2000). Recently, a 3-factor model of depression was found, representing negative affect, anhedonia, and psychomotor change (Vrieze et al., 2013). The psychomotor change symptom cluster has an important clinical, diagnostic, pathophysiological and therapeutic significance in the clinical and scientific approach of Major Depressive Disorder (MDD) (Sobin and Sackheim, 1997; Schrijvers et al., 2008). Psychomotor retardation (PR) has repeatedly been denoted as an important marker of the melancholia subtype of depression

(Parker, 2000; Schrijvers, 2008), and as a predictor for treatment response to several types of antidepressant treatment (Schrijvers et al., 2008). Since it is the only factor of depression that does not correlate with severity of depression and since it is not predictive for clinical outcome, psychomotor functioning is thought to be a dimension defining a separate typology (Vrieze et al., 2013), though not exclusively the melancholic subtype of depression. PR has been found to be present in other subtypes of depression too (Benazzi, 2002; Gupta, 2009; Niculescu and Akiskal, 2001; Schrijvers et al., 2009; Smith et al.,1995; Widlöcher, 1983 in Buyukdura, 2011). Yet not only the presence of PR is important. Even the type of slowing and the cognitive share in the PR are thought to be differentiating between subtypes of depression (Caligiuri et al., 2000). These reasons indicate the importance of investigating psychomotor functioning in depression against the background of cognitive functioning.

Specifically, in elderly depression, PR appears to be a predominant symptom of late life depression, an organic subtype of geriatric depression with vascular damage of frontal-subcortical circuits and a depressive-executive dysfunction syndrome (Bella et al., 2010; Alexopoulos et al., 1996), but also in other atypical depression presentations such as subsyndromal depression (Judd et al., 1994). As aging itself already causes a substantial psychomotor slowing in healthy volunteers (Cerella, 1993; Pier, 2004; Seidler et al., 2010), elderly depressed patients could be expected to show a more pronounced form of PR than healthy elderly did. Pier and colleagues hypothesize an additive effect of aging and depression on the psychomotor performance, admittedly based on a sample of eleven medicated patients. Bonin- Guillaume et al. (2007) too found an additive PR effect in sixteen patients. The retardation showed to be an addition of two different types of slowing. There was a general slowing in aging, affecting all stages of information processing, and a more specific slowing in depression, affecting the decisional stage and the neuromotor stage but not affecting the sensori-motor stage. It should be noted that they did only investigate the reaction time and not the motor time as a measure of psychomotor speed (Bonin-Guillaume et al., 2007). However, it should be remembered that the included patients in both studies were all using psychotropic medication, i.e., antidepressants (selective serotonin re-uptake inhibitors and tricyclic antidepressants) as well as anxiolytics and that confounding medication effects were observed (Pier et al., 2004). Indeed, the use of medication (and often polydrug use) is very common in elder age groups, but since these patient groups are also more sensitive to all kinds of adverse medication induced side-effects, the sorting out of the specific effect of depression, age and medication is particularly difficult, especially as the medication profiles of the subjects in previous studies may be extremely variegated. Studies on PR in elderly depression are still scarce and show partial results because most of these have only measured PR based on cognitive reaction times without distinguishing and separating out motor slowing (Tarbuck and Paykel, 1995; Bonin-Guillaume, 2007; Hart and Kwentus, 1987; Nebes et al., 1998). The two studies also investigating the motor time include medicated patients (Pier et al., 2004; Beats et al., 1996). All in all, differentiated research of psychomotor symptoms in geriatric depression is very limited and only exists in medicated clinical cohorts, so that evidence is still missing

for the usefulness of these types of symptoms as a diagnostic tool for this subgroup of depressed patients.

PR not only deters motor processes, but also cognitive functioning. Indeed, 'the term not only encompasses the output of muscle contractions, but also the wider involvement of perceptual processes and cognitive-control mechanisms, underlining that motor control involves more than an adjustment of muscle contractions' (Schrijvers et al., 2008, p14). Furthermore, several cognitive subprocesses contribute to the psychomotor processing. Studies on neuropsychological functioning in late life depression generally mention processing speed and executive function as the main impairments in MDD in the elderly (Dybedall et al., 2013). PR and executive functioning are not correlated (Baudic et al., 2007), but reduced processing speed is suggested to explain deficits in higher order cognitive function (Butters et al., 2004; Sheline et al., 2006; Nebes et al., 2006). However, Sexton et al. (2012) found that executive deficits could not be fully explained by general impairments in processing speed. Controlling for processing speed, Dybedall et al. (2013) still found impaired executive function in elderly depressed compared to healthy controls. Considering that both processing speed and executive functioning are the cognitive hallmarks of depression, we endeavor to study them separately as the background of the psychomotor measures in our study. Since executive function and PR are not correlated, it would be interesting to figure out whether depression severity without interfering medication effects, has a different impact on cognitive and psychomotor functioning.

The current study aims to measure cognitive and psychomotor functioning in a sample of unmedicated depressed elderly, applying objective psychomotor and cognitive assessment methods. In accordance with previous studies (Rosenberg et al., 2011; Tarbuck and Paykel, 1995; Pier et al., 2004), it is hypothesized that unmedicated elderly depressed patients will perform worse both on the cognitive and psychomotor tasks. Different cognitive and psychomotor measures will be applied to shed a light on different cognitive factors that may influence PR, most importantly processing speed, but also inhibition and interference resistance, cognitive flexibility, and memory. With the objective measures of PR, the cognitive reaction time, i.e., the initiation time of a movement and the reinspection time, the time needed to verify the stimuli, will be separated from the motor time, i.e., the real movement time. Finally, the effect of cognitive load in PR will be tested by experimentally varying the complexity of the stimuli of the copying task to investigate the interaction of cognition and motor functioning in PR.

1.2.3 Material and methods

1.2.3.1 *Study population*

Twenty-eight non demented (Mini Mental State Examination Score > 24) elderly (age >60) in- and outpatients with unipolar single episode or recurrent major depressive disorder (MDD), meeting DSM-IV-TR criteria (American Psychiatric Association, 1994), were

compared to 20 healthy controls, matched for age, gender, education and vascular risks (diabetes, hypertension, smoking, obesity, hyperlipidemia). Patients with a MMSE score under 24, the consensus cut off score for probable dementia (Tombaugh & McIntyre 1992; Anstey et al., 2010), were excluded. Depression was identified using the DSM-IV-TR criteria and the severity of depression was assessed by means of the Geriatric Depression Scale (GDS). A minimum score of 11 on the GDS was required for inclusion. Patients taking medication with important psychotropic impact such as psychopharmacological treatments, but also antihistamines and anticholinergics for instance were excluded. For every type of disallowed or concomitant medication, the drug free period before testing was specified. For most antidepressants, a washout period of one week prior to baseline was applied, except for fluoxetine (5 weeks), fluvoxamine (2 weeks), monoamine oxidase inhibitors (2 weeks). Any anxiolytics (including benzodiazepines) were disallowed within the last week prior to testing as well as hypnotics, except Zolpidem, Zopiclone or Zaleplon. Patients suffering from any medical condition (e.g., Parkinson's disease, dementia, psychotic disorders, mental retardation, substance- or alcohol abuse, organic mental disorders due to a general medical condition as defined in the DSM-IV-TR) that might affect fine motor or cognitive processes were excluded, as well as patients with personality disorders that might compromise the study. All patients were native Dutch speakers and had given their informed consent after the study was fully explained to them. The study was carried out consistent with the latest version of the Helsinki Declaration and was approved by the medical ethics committee of the participating hospitals.

1.2.3.2 *Assessments and tasks*

All participants performed an extensive cognitive and psychomotor test battery (see below). All testing, for patients as well as healthy controls, took place in the afternoon. Depression severity was assessed using the Geriatric Depression Scale (GDS, 30 items) (Yesavage and Brink, 1982) and the State and Trait Anxiety Inventory (STAI 1, STAI2) (Spielberger et al., 1983) informed about the degree of subjective anxiety symptoms. Both tests were also applied to the controls. The 15-item Salpêtrière Retardation Rating Scale (SRRS) (Widlöcher, 1983) was administered to assess the subjective, rated level of PR. To obtain a differentiated image of the participants' psychomotor and cognitive abilities, all of them performed cognitive tasks measuring attention, information processing, memory, and executive function. Finally, objective psychomotor tests were administered, with varying levels of cognitive impact. For the objective psychomotor assessment participants carried out drawing tasks. Subjects were asked to copy figures from a computer screen with use of a special pressure-sensitive pen and a digitizer (Maarse et al., 1988). A full description of the set up as shown in Figure 1 can be found in Pier et al. (2004). In a first task patients had to draw a line in one of four directions (horizontal, vertical or one of the two diagonals) as quickly as possible. In the second task, they had to copy figures consisting of four line-segments with varying complexity, some were well known letters, other were familiar figures and the third kind were less-familiar patterns. As soon as participants started drawing, the figure disappeared from the screen. However, there was the possibility (which was not encouraged) to reinspect the figure by retouching the starting

spot. Initiation time, the time between the presentation of the stimulus and the start of the first drawing movement, was measured. Also, the motor time, the time from the start of the first drawing movement to the end of the last drawing movement, was calculated. In the second task the reinspection time, the time from retouching the spot to resuming starting the drawing was also determined. Time to reinspect was not included in the motor time.

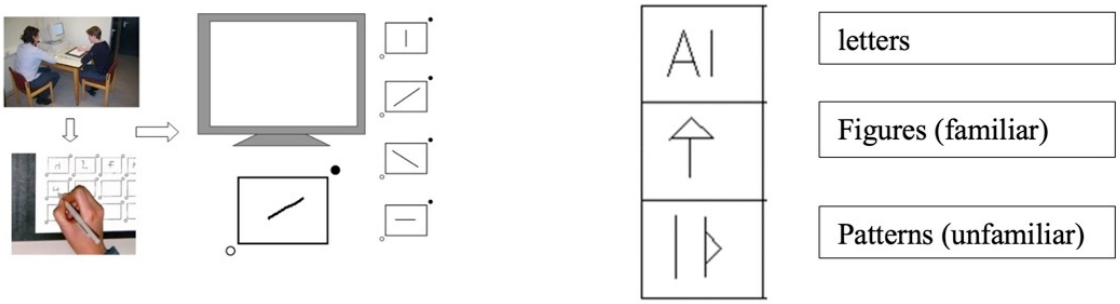


Figure 1. Set up of the line and complex figure copying task with pressure-sensitive pen and digitizer

All subjects completed the Standard Symbol Digit Substitution Test (SDST) (Mc Leod et al., 1982; Wechsler, 1981; Wechsler, 1997). The same recording techniques were used as with the copying tasks. This made it possible to differentiate between a cognitive and a psychomotor component apart from the general measure of information processing speed. The subjects had to substitute symbols by digits during a period of 90 seconds, using a key consisting of nine symbol-digit pairs. The following variables were analyzed: raw scores, i.e., the number of correct answers, matching time, representing mean pen-up time and pause time between two successive digits (comparable to the initiation time in the copying tasks), and writing time, representing the time needed to write a digit (comparable to motor time).

In the Wisconsin card-sorting task, which is primarily intended to measure cognitive flexibility, an executive function, four key cards were presented with geometric figures that vary according to three perceptual dimensions (color, form, or number). The subjects had to discover the correct sorting principle by trial and error. After each choice, they got feedback (right or wrong). Once the participant made a correct choice, this sorting principle had to be maintained across changing stimulus conditions while ignoring the other –now irrelevant- stimulus dimensions. After ten consecutive correct matches, the classification principle changed without warning. As the WCST is not timed, sorting continued until all cards were sorted or a maximum of six correct sorting criteria had been reached. Indices of the participants' performance were the number of categories completed (Barcelo, F. & Knight, R. T., 2002; Bardenhagen, F.J. & Bowden, S.C., 1998; Greve, K. W., Bianchini, K. J., Hartley, S. M. & Adams, D. 1999, Greve, K.W. et al., 2002). However, since the patients did not even complete one category, executive functions such as switching were not measured,

so the measures of perseverative and non-perseverative errors were not meaningful in this case.

The Stroop color-word test (Stroop, 1935; Mc Leod, 1991) is a cognitive test that requires participants to firstly read the names of colours printed in black ink (trial 1), then name printed colors (trial 2) as quickly as possible without making errors and then naming the color of a word in which it is printed. The test measures the individual's ability to suppress task-irrelevant responses (i.e., the tendency to read the color name rather than name the color) and ability to maintain attention and concentration (Dodrill, C.B., 1978). The Stroop interference score was calculated as the time taken to name colors in trial 3 minus the time taken to name color names in trial 2. A higher Stroop interference score was interpreted as the degree of interference afforded by suppressing the habit of reading words to name colors; thus, higher scores reflect poorer performance. (Dodrill, C.B., 1978).

In the 15-words task (Kalverboer & Deelman, 1964; Saan & Deelman, 1986), subjects were presented five times a list of fifteen words which they had to reproduce. After an interval of twenty minutes, the experimenter asked to reproduce the memorized words once more. Afterwards they had to recognize in a list of thirty words, which were the words they had studied.

1.2.3.3 *Statistical analysis*

Statistical analysis of the data was performed using SPSS 17.00. and consisted of a General Linear Method (GLM) multivariate analysis of variance to compare the psychomotor and cognitive outcomes of the two groups. Cohen's d was calculated for all measures to make comparison of effect sizes possible. To measure the effect of cognitive load in the figure copying tasks, a GLM Repeated Measures analysis of variance with Group (MDD, Controls) as between-subjects factor and Complexity (simple, complex) as within-subjects factor was performed. In addition, bivariate Pearson correlations were computed between severity of depression and the other clinical, cognitive and psychomotor measures.

1.2.4 Results

1.2.4.1 *Demographic and clinical variables*

As can be seen in Table 1, there were no significant differences between groups on demographical variables. Patients were significantly more depressive, more anxious (as well state as trait anxiety) and showed more PR (SSRS) and cognitive impairment (MMSE). Severity of depression correlated with none of the cognitive and psychomotor measures, only with the other clinical measures of state anxiety (r GDS-STAI I=0.006) and slightly with the clinical rating of retardation (r GDS-SRRS=0.047).

	Patients (N=28)	Controls (N=20)	F	p
Age	74.71 (7.56)	71.95 (5.14)	2.01	.163
Male/Female	4/24	5/15	X^2=.879	.348
MMSE	25.52 (3.80)	28.30 (1.38)	9.73	.003
GDS	17.58 (4.46)	4.15 (2.50)	145.83	<.0001
STAI 1	51.93 (11.38)	34.50 (7.83)	34.98	<.0001
STAI 2	51.00 (10.25)	34.45 (7.65)	36.81	<.0001
SRRS	16.44 (8.74)	2.30 (1.92)	50.16	<.0001

Table 1 Demographic and clinical variables of patients and controls. Standard deviations are shown in parentheses.

1.2.4.2 *Cognitive and psychomotor performance*

Patients performed significantly worse than controls on all cognitive measures. For an overview, see Table 2. The largest effects are found for Number of correct filled in items on the SDST, the matching time of SDST, the Wisconsin number of categories completed and the total recall of the verbal memory test. The measures of the perseverative errors and non-perseverative errors had to be left out because they proved meaningless, as patients could not complete one category. The verbal memory scores confirm the impaired learning capacity. As can be seen in the table, the Stroop tasks too almost reached significance on the 0.01 level, but the significance was lowered by the difference in variance between patients and healthy controls, with a larger variance in the patient scores, except for the WCST presumably be explained by a floor effect, as patients did not even manage to learn one category. The difference in SDST total correct, the measure of processing speed, reveals that a general retardation of processing speed is a central feature of depression in the elderly. Still on the SDST, both the matching and the writing time were significantly higher in patients, indicating cognitive as well as psychomotor slowing on this task. As for performance on the copying tasks, patients' initiation time was found to be impaired on the LCT, but not on the FCT, whereas movement time was significantly higher in patients than in controls on both the LCT and the FCT. Analysis reveals a more significant difference between the healthy and the depressive elderly on the movement time compared to the initiation time. Finally, patients reinspected significantly longer than controls on the FCT.

	Patient	Control	*F*	*p*
Neuropsychological tests				
SDST Number correct	43.63 (9.38)	27.52 (13.46)	19.41	<.0001
SDST_MatchingTime	3.42 (2.90)	1.47 (0.45)	8.44	.006
SDST_WritingTime	1.17 (1.08)	0.66 (0.13)	4.23	.047
Stroop Card 1	63.43 (24.10)	47.32 (11.21)	7.19	.011
Stroop interference	111.43 (110.54)	46.11 (21.42)	37.23	.016
WCST N categories completed	0.65 (0.83)	2.00 (1.12)	19.16	<.0001
Verbal Memory Total	26.71 (11.91)	36.32 (7.77)	9.55	.003
Verbal Memory Recall	4.59 (3.24)	6.63 (3.06)	4.63	.037
Verbal Memory Recognition	22.72 (4.21)	25.72 (2.61)	7.15	.011
Psychomotor tasks				
CL_ Initiation Time (s)	1.46 (1.00)	0.97 (0.17)	4.49	.040
CL_ Movement Time (s)	0.73 (0.38)	0.47 (0.17)	7.78	.008
CC_Initiation Time (s)	2.98 (1.03)	2.60 (0.85)	1.67	.203
CC_Reinspection Time (s)	0.41 (0.66)	0.10 (0.19)	3.99	.053
CC_Movement Time (s)	3.94 (2.36)	2.38 (1.15)	7.03	.011

Table 2 Mean performance levels of patients and controls on cognitive measures. Standard deviations are shown in parentheses

As shown in Figure 2, increasing figure complexity in the FCT for increased cognitive load, resulted in a significantly increased initiation time (F=10.38, p=.0002) and execution time (F=10.721, p=.0002) with both patients and controls and in a significantly longer reinspection time (F=3.89, p=.0.029). However, the increased cognitive load affected patients' psychomotor performance more than that of controls, except for the initiation time (IT: F=1.27, p=0.267, ns; MT: F=10.721, p=0.0002**; Reinspection: F=4.98, p=0.031*). Patients as well as healthy controls initiated the drawing movements immediately, but the patients faltered while drawing and had more need to refresh the stimuli.

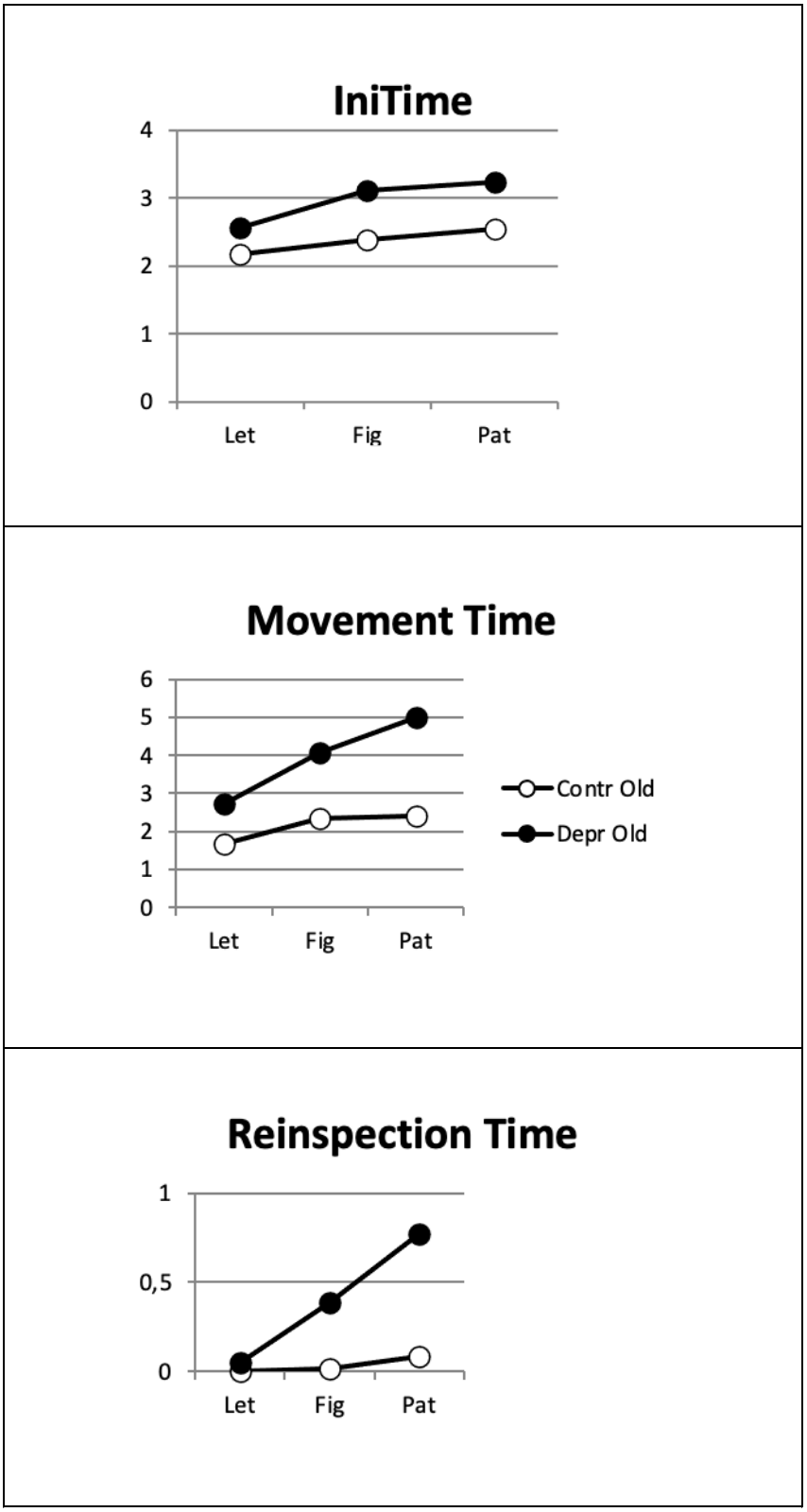


Figure 2 Differences in initiation time, movement time and reinspection time as a function of complexity between depressive patients and healthy controls.

1.2.5 Discussion

In this study, we investigated psychomotor and cognitive performance as an effect of depression in an elderly medication-free depressed sample, with both objective motor and cognitive measures. To find out the impact of a cognitive factor in PR, we experimentally varied the amount of cognitive load in psychomotor functioning. Because Tarbuck and Paykel (1995), in an unmedicated sample, assumed that retardation due to age is associated with timed tasks only and that PR due to depression is associated with the complexity of the task, we chose to use a not-timed psychomotor task to see whether the difference still showed. The geriatric depressed patients (as a group) were found to be significantly slower on almost all psychomotor measures, as reflected in high SRRS scores as well as in inferior outcomes on most of the copying tasks, compared to the outcomes recorded for the matched healthy controls. In general, this is in line with previous studies in depressed samples that applied the same assessment methods, in elderly (Pier et al., 2004) and in younger patients (Sabbe et al., 1996; Sabbe et al., 1999; Schrijvers et al., 2009; Destoop et al., 2010). However, the sampling in this medication free population shows peculiarities of slowing that, moreover, provide valuable insights into the very specific interaction of cognitive and psychomotor slowing in the convergence of depression and aging.

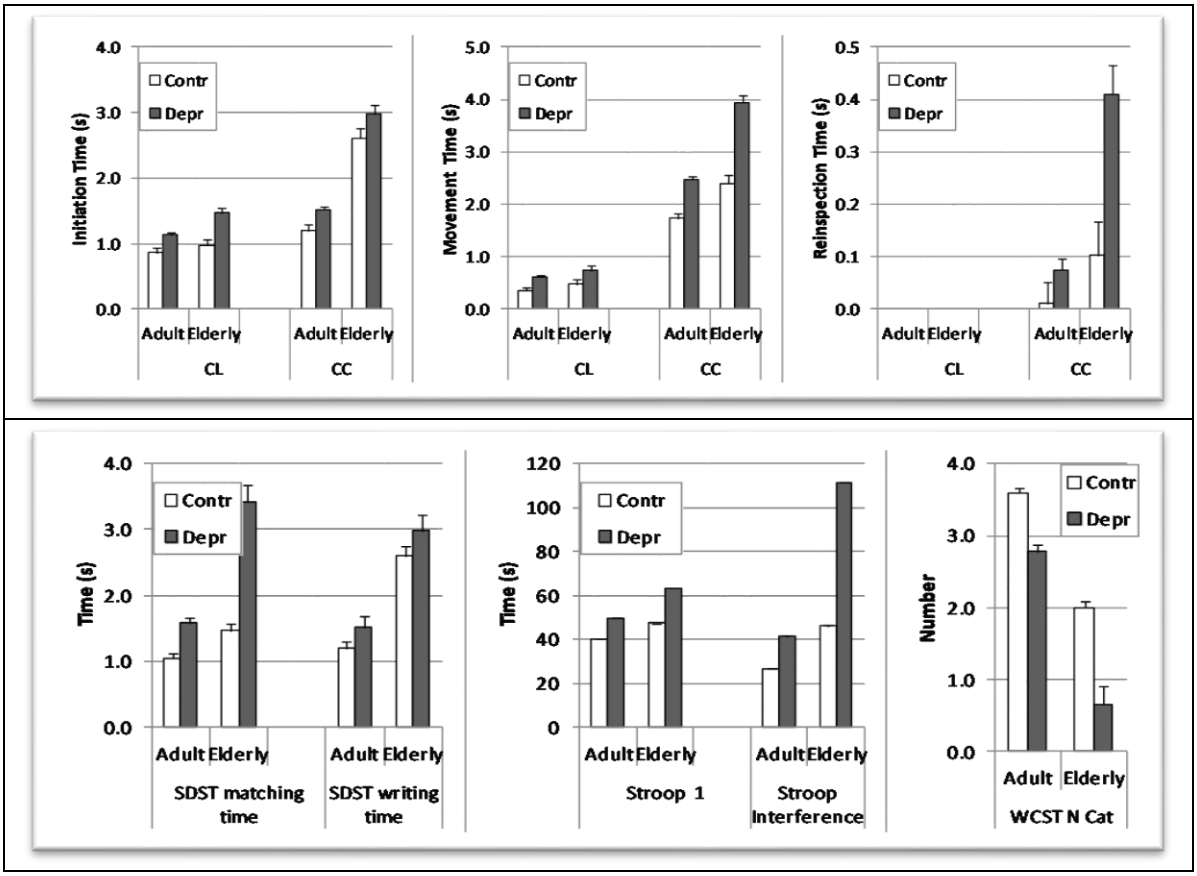
When varying the complexity of figures to copy and thus varying the cognitive load, strikingly the motor time shows the most significant interaction effects of group (depressive elderly versus healthy elderly) and complexity, the reinspection time is less significant, the initiation time not at all. Patients start copying immediately, irrespective of the complexity of the task. Nevertheless, in cognitive more difficult motor tasks, the movements of the depressed elderly become slower or more hesitating, with some more reinspection. Apparently, various cognitive and motor processes are involved in figure copying. Initiation times are assumed to chiefly reflect the cognitive processes and encompass the attention for and the perception of the stimulus figure, as well as the storage of the representation in working memory, but also the programming and planning of the first drawing movement and the activation of motor programs that initiate the muscle to start drawing (Schrijvers et. al., 2009). Clearly, figure copying is different from the separate cognitive measures in standard cognitive testing. Even the SDST tends to reflect higher-order cognitive, memory-related functions more than it does psychomotor speed (Morrens et al., 2007 in Schrijvers et al., 2009). The bigger higher-order executive cognitive load of searching for a number in the legend code, memorizing the found digit and subsequently performing the initiation and planning of writing the digit in the SDST and the relative easiness of writing a well-known automatized digit compared to an unknown pattern, may also explain the difference in effect size of the matching time of SDST (Cohen's d= 0,94) and the initiation time of the figure copying (Cohen's d CL initiation=0,65; Cohen's d CC initiation =0,39). Furthermore, patients performed worse

than controls on all cognitive measures in the standard cognitive tasks. It must be remembered, however, that above all, the cognitive executive aspects show interaction effects of aging and depression, except for the WCST. The lack of interaction effect on the WCST is clearly a result of the lack of measured executive function due to the patients' inability to learn even one category. Measuring adaptation and perseveration thus became impossible.

Hence, the difference in slowing because of increasing cognitive load may be explained as an effect of cognitive aspects in psychomotor functioning. Presumably, the cognitive component of PR is different in nature and has more motor circuitry involvement than that measured by the standard cognitive tasks.

Our results suggest that PR observed in the patient group was caused by both a cognitive and a motor factor, as, respectively, most matching times and writing times were higher in patients. To further explain the possible cognitive effect, we compared the current results post hoc to the ones obtained in a similar study in an adult population of depressed medicated patients and in healthy controls (18-60 year). This way, we could also gain some insight into possible interaction effects of age and depression, and we could determine whether there was a link with cognitive functioning. In Figure 3, we have presented the results of this post hoc comparison. Since adult medicated patients appear even less retarded than elderly depressive unmedicated patients do, these results only corroborate the hypothesis of a depression aging effect. The overall comparison in Figure 3 reveals a clear effect of depression in all ages, both, for the cognitive measures (F SDST matching time=36.40, p<0.001; F SDST writing time=22.36, p<0.001; F stroop card 1=25.58, p<0.001; F Stroop interference=31.24, p<0.001; F WCST N categories completed = 10.54, p=0.001) and for the psychomotor measures (F CL initiation time=24.29, p<0.001; F CL movement time = 13.83, p<0.001; F CC initiation time=8.54, p=0.004; F CC reinspection time = 14.71, p<0.001; F CC movement time = 25.35, p<0.001). An aging effect is equally obvious, also in both, in cognitive measures (F SDST matching time = 29.96, p<0.001; F writing time= 45.32 p<0.001; F Stroop card 1=16.21, p<0.001; F Stroop interference = 39.19, p<0.001; F WCST N categories completed = 31.21, p<.001) and in psychomotor measures (F CL initiation time= 8.55, p=0.004; F CL movement time = 3.22, p=0.074; F CC initiation time = 144.70, p<0.001; CC reinspection time = 19.37, p<0.001; CC movement time = 22.02, p<0.001). A calculation of possible real interaction effects in this general linear model test indicates that only the matching time and the writing time of the SDST and the Stroop interference show interaction effects (F SDST matching time = 11.80, p=0,001; F SDST writing time = 9.50, p=0,002; F Stroop card 1= 1.57, p=0.211; F Stroop interference = 12.65, p<0,001; F WCST = 0.63, p=0.429). In the psychomotor measures, only the reinspection time shows a slightly significant interaction effect (F CL initiation time = 2.10, p=0.149; F CL movement time = 0.001, p=0.979; F CC initiation time = 0.04, p=0.837; F CC reinspection time = 6.35, p=0.12; F CC movement time = 3.09, p=0.80). However, this effect was not reflected in the results. The significance was diminished by the much larger variance on the reinspection

times of the complex figure-copying task in the elder population. Indeed, there is an overall increase of variance in the elderly, especially in psychomotor tasks where motor and cognitive aspects coincide (SDST matching, writing time, complex figure reinspection). Overlooking the overall results leads to the assumption that the interaction of depression and aging reveals itself in executive functioning and in the interaction of cognitive and psychomotor functioning'. The main comparison of the Cohen's d effect sizes in the elderly and adult group shows that the effect of depression is always bigger in elderly. The relatively small difference between the effect sizes of the adults and the elderly, however, is explained by the large variance in older groups, which limits the found intergroup effects. Surprisingly, the effect sizes of initiation time of the copying tasks show the reverse direction; it is bigger in adults. Evidently, these results need to be confirmed by direct comparative research.



Note. Because of limited competence of the population, with the elderly the copying task consisted of just four lines, whereas with the adults a task with eight lines was used. To make the results comparable, recalculations were made for the adult scores based on the mean time for four lines. Separate times for each line were available.

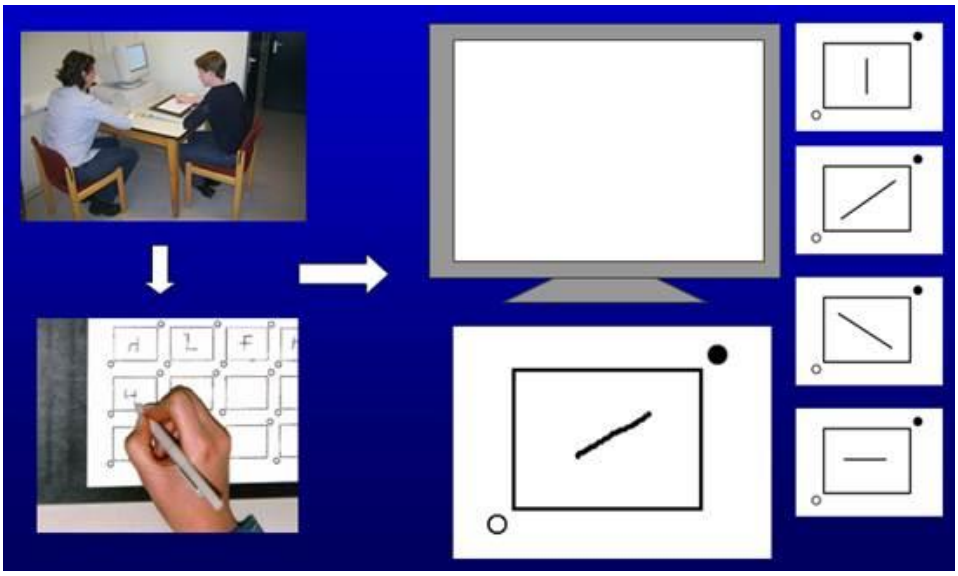
Figure 3 Comparison of psychomotor and cognitive measures between healthy and depressed elderly against the background of previous research with the same tasks in adults.

The present study not only confirms the results of a similar study by Pier et al. (2004), it also provides a valuable contribution, as it overcomes some of the restrictions of the earlier study. Whereas the study by Pier et al. was a small sample study (n=11) in which patients

were taking medication that could have impacted the results, the present study is unique in that it involves only patients that are free of psychotropics. The importance of the latter condition is apparent from the fact that in the Pier et al. study (2004) correlations were found between the use of antidepressants and anxiolytics on the one hand, and several psychomotor outcomes on the other. With our larger medication free sample, we succeeded in replicating the results of Pier et al. (2004), corroborating their preliminary results concerning the presence of PR in elderly depressed patients, independent of medication status. Apart from that, the present study revealed an interesting difference between medicated and unmedicated patients. In comparison to the control groups (healthy aged, younger depressed), the pattern of interaction between the degree of slowing and the cognitive complexity of the task in the unmedicated sample seemed to be the reverse. In the unmedicated sample, PR was proportionately more visible in more complex tasks (copying more complex figures, less familiar figures) than in copying simple lines. In the medicated sample, on the contrary, the PR was more obvious in comparison with the other groups in the simple copying task than in the more complex tasks (Pier et al., 2006:24). This result is in line with the suggestion by Caligiuri et al. (2000) that retardation caused by medication is predominantly neuromotor retardation, i.e., abnormal velocity, as opposed to the psychomotor slowing in depression, in which the cognitive factor is more important. Benzodiazepines, opioids, anticholinergics, but also tricyclic antidepressants (Moore, A.R., O'Keeffe, S.T., 1999) often elicit modest or more pronounced psychomotor or cognitive impairments (Robles Bayon, A. & Gude Sampedro, F., 2012). These findings support the diagnostic relevance of the quality of slowing in major depression, in aging and in a broad range of psychopathological disorders.

Notwithstanding the relatively small sample size, the reported effects were robust. The very restrictive inclusion criteria determining the sample size were introduced because of the high comorbidity of depression and the considerable use of medication in the elderly and because of the numerous possible cognitive – and psychomotor – side-effects of somatic and degenerative diseases. To avoid such confounding cognitive effects a selection of elderly depressive patients that can hardly be seen as representative for the 'natural' population imposed itself. On the other hand, this strict selection afforded a unique opportunity to rule out possible medication and comorbidity effects and to obtain an unbiased view on the differential PR effects of depression in the elderly.

COGNITIVE AND PSYCHOMOTOR EFFECTS OF THREE MONTHS OF ESCITALOPRAM TREATMENT IN ELDERLY PATIENTS WITH MAJOR DEPRESSIVE DISORDER



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1.3 Cognitive and psychomotor effects of three months of escitalopram treatment in elderly patients with major depressive disorder.

1.3.1 Abstract

Background: Although psychomotor retardation (PR) and cognitive dysfunction are essential symptoms of elderly depressed patients, the differential effect of treatment with an SSRI in the elderly on these symptoms hardly has any attention in studies with objective experimental measures. Since effects appear relatively slower in elderly, this study evaluates the effect on cognitive and psychomotor functioning as compared to mood, on four points during a twelve week follow up of monotreatment with escitalopram.
Methods: 28 non-demented elderly unipolar depressive patients on 5 to 20 mg escitalopram were compared to 20 matched healthy elderly. All participants underwent a test battery containing clinical depression measures, cognitive measures of processing speed, executive function and memory, clinical ratings of PR, and objective computerized fine motor skill-tests at the start and after 2, 6 and 12 weeks. Statistical analysis consisted of a General Linear Model (GLM) repeated measures multivariate analysis of variance of per protocol analysis to compare the psychomotor and cognitive outcomes of the two groups.
Results: Although, apart from the significant mood effect, no interaction effects were found for the psychomotor and cognitive tasks, the means in general show a trend of differential effects in cognitive and psychomotor functions, with smaller effects and delayed timeframes and with presence of subgroups compared to mood effects.
Limitation: Longer follow-up is necessary to evaluate differential long-term effects.
Conclusion: In elderly, moderate effects of Selective Serotonin Reuptake Inhibitors treatment on mood precede slow or limited effects on cognition and PR.

Keywords: Major depression, Elderly, Psychomotor retardation, Cognitive, Escitalopram, Functional burden

Highlights:

Psychomotor retardation is a core symptom in geriatric depression.
Three months of treatment with escitalopram 5-20mg is beneficial for mood.
It appears lacking psychomotor or cognitive effects.
There was a trend of differential effects in psychomotor and cognitive function.
Moderate mood effects of Selective Serotonin Reuptake Inhibitors precede presumed slow or limited effects on PR.

1.3.2 Introduction

Selective Serotonin Reuptake Inhibitors (SSRIs), and especially escitalopram and sertraline appear to be the first-choice antidepressant pharmacological treatment for Major Depressive Disorder (MDD) (Cipriani et al., 2009), given their favorable balance between benefits (Cipriani et al., 2009; Kok, Nolen & Heeren, 2012), tolerability (Kasper, De Swart & Friis Andersen, 2005; Mao et al., 2008; Gorwood, 2007; Bose, Li & Gandhi, 2008), and acquisition cost.

Psychomotor symptoms have clinical relevance, and they are indicative of melancholic depression with or without psychotic features and could be relevant in the choice of antidepressants (Schrijvers, Hulstijn & Sabbe, 2008). In psychomotor functioning, three domains are generally distinguished: fine versus gross motor functioning, and speech functioning (Bennabi et al., 2013; Buyukdura et al., 2011; Schrijvers, Hulstijn & Sabbe, 2008; Sobin & Sackeim, 1997).

Despite the importance of the psychomotor symptom cluster and the widespread use of SSRIs in the treatment of MDD, only few studies have investigated the impact of SSRIs on Psychomotor Retardation (PR). Some of these studies applied subjective observer-rated methods such as the retardation item of the Hamilton Depression Rating Scale (HDRS) (Hamilton, 1960) and the Salpêtrière Retardation Rating Scale (SRRS) (Widlöcher, 1983), whereas very few used an objective measurement method, a battery of figure copying tasks with the use of a pressure-sensitive pen and a digitizer. The latter technique results in objective and real-time recordings of perceptual motor activity and enables to distinguish between the cognitive and motor processes involved in a writing movement. Hegerl et al. (2005) and Mergl et al. (2004) reported an increase in velocity of rapid hand movements after treatment with [reboxetine and] citalopram, applying such a computerized test battery during a 4-week treatment. Sabbe and colleagues (1996) treated depressed inpatients, for whom other psychotropic medication was restricted to the absolute minimum, during six weeks with fluoxetine 20 mg and observed an overall cognitive but no motor improvement on a battery of digitized writing tasks. Using the same drawing tasks, Schrijvers et al. (2009) compared the psychomotor performance of 22 MDD inpatients to a control group of 19 healthy subjects to evaluate for 6 weeks the effect of treatment with 50mg sertraline, while ruling out effects of other psychotropic medication. They found decreased cognitive and motor times in patients for copying simple lines or figures, but no decrease in motor times for drawing more complex figures, with a higher cognitive load for motor planning.

Depression presents differently in elderly, with less mood complaints and more somatic, psychomotor, and cognitive symptoms (Alexopoulos et al., 2002). Moreover, depression may be secondary to a different medical condition or drug, entailing more risk of drug-drug and drug-disease interaction and adverse effects of medication. In addition, aging itself causes decline in psychomotor and cognitive functioning (Alexopoulos et al., 2002). PR is a

particularly relevant symptom cluster, given its direct relationship with loss of activity and functioning in daily life (Santos et al., 2012), reduced self-care, and higher risk of falling (Chen, Peronto & Edwards, 2012). It would even be bi-directionally associated as a risk factor for and as a result of depression. Moreover, PR is more distinct in elderly (Parker et. al., 2000, 2001), and characteristic for the dysexecutive syndrome (Lockwood, 2002). Finally, PR predicts poor treatment response and chronicity of geriatric depression (Kalayam et al., 1999).

SSRIs are efficacious, but elicit a delayed response in depression in elderly, compared to younger patients (Kok, Nolen & Heeren, 2012; Topiwala et al., 2012). In the very old, SSRIs are more effective than placebo, but only in severe depression. Important differences in results were found with ranges of 18 to 82% for placebo and 16 to 80% for citalopram (Roose et al., 2004). Finally, SSRI reduces the relapse rate significantly (Gorwood et al., 2007), known to be higher in elderly patients (Mitchell & Subramanian, 2005).

Non-responders to SSRIs appear to be a subgroup with standard cognitive impairments (Culang et al., 2009). Citalopram-treated patients with deficient response inhibition show an even worse response than placebo-treated patients. With intact response inhibition, on the contrary, results are the reverse (Sneed et al., 2010).

This study will investigate the differential effects of escitalopram on cognitive and psychomotor measures in elderly patients and compare them to mood effects, without interfering effects of other psychotropic medication. Since effects of SSRIs in elderly are slower, the timeframes of the various symptoms were also compared. Drawing on previous research, we hypothesize, apart from a decrease in depressive symptoms, a decrease of motor time in simple motor tasks (Hegerl, 2005; Mergl, 2004), an improvement of all cognitive measures and of cognitive initiation times (Sabbe, 1996), but no improvement of motor times in complex motor times involving more motor planning (Schrijvers, 2009). Further, we explore the possibility of the existence of subgroups in elderly patients, based on processing speed.

1.3.3 Materials and methods

For a full description of the study population, inclusion and exclusion criteria, assessments, tasks, and baseline results, see the baseline report of this investigation in chapter 1.2. (Beheydt et al., 2015).

Twenty-eight non-demented (Mini Mental State Examination (MMSE) Score > 24) elderly (age >60) medication-free in- and outpatients with unipolar single episode or recurrent MDD (score on Geriatric Depression Scale (GDS)>11; Yesavage & Brink, 1982) were compared to 20 healthy controls, matched for age, gender, education and vascular risks.

All participants were administered a questionnaire about health, medication, wellbeing status and educational level. Next, the MMSE (Kok & Verhey, 2002) and GDS were administered. After inclusion, the cognitive and psychomotor functioning of this group were compared to those of the healthy elderly at four time points (T) after the start of treatment with escitalopram 5-20 mg: at baseline and at week two, six and twelve. All assessments took place in the afternoon.

Clinical depression severity was assessed using the GDS (30 items) (Yesavage and Brink, 1982), whereas the State and Trait Anxiety Inventory (STAI 1, STAI2) (Spielberger et al., 1983) informed about the degree of anxiety symptoms. The 15-item Salpêtrière Retardation Rating Scale (SRRS) (Widlöcher, 1983) was administered to assess the clinical level of PR.

For the objective psychomotor assessment, participants were asked to copy lines (CL) or figures (CF) from a computer screen with the use of a special pressure-sensitive pen and a digitizer (Maarse et al., 1988). The initiation time (IT), the time between the presentation of the stimulus and the start of the first drawing movement, and the motor time (MT), the time from the start of the first drawing movement to the end of the last drawing movement, were calculated. In the second task, the reinspection time (REIN T), the time from retouching the starting spot to resuming starting the drawing, was also determined. Reinspection time was not included in the motor time. For the Symbol Digit Substitution Test (SDST) (Mc Leod et al., 1982), the same recording techniques were used as with the copying tasks. The following variables were analyzed: the number of correct answers (SDST NCORR), the matching time, i.e., initiation time (SDST IT), and the writing time, i.e., motor time (SDST MT).

Cognitive functioning was assessed using the computerized Wisconsin Card Sorting Test (WCST; Barceló, F. & Knight, R.T., 2002; Greve, et al., 2002). Indices used were the number of correct answers (WCST NCORR) and the number of categories (WCST CAT) completed. Additionally, from the Stroop color-word test (Mc Leod, 1991) the variables reading speed (Stroop1) and interference (Stroop INT) were analyzed. From the 15-words verbal memory test (Saan & Deelman, 1986), only the number of correct recalls in the fifth trial (15W TOT) was recorded (Verbal Memory Total). The delayed recall was scored as 15 W RECALL. For the Verbal Memory Recognition too, only correct recognitions (15W RECOG) were scored.

Statistical analysis of the data was performed using SPSS 17.00. and consisted of a General Linear Model (GLM) repeated measures per protocol analysis to compare the psychomotor and cognitive outcomes of the two groups on all assessment moments, with Time as within-subjects factor and Group as between-subjects factor (Field, 2009). When sphericity could not be assumed, the Greenhouse Geisser correction was used to reduce Type 1 errors. Effect sizes were calculated with partial $\eta 2$. Per protocol analysis was chosen because of the known high variance between and within patients, which makes estimations

inappropriate. However, to rule out completer's bias, missing data were imputed by a Last Observation Carried Forward (LOCF), because drop out patients never got better afterwards, and the risk of Type 1 errors was non-existent (Supplement 1). The LOCF was only used to check the reliability of the data found in the completers group (see Supplement 2 for a significance and effect size summary). Subsequently, an exploratory analysis tested for differences between patients with high (<28) and low level (≥28) processing speed, using the median as a (central tendency) cut off score between the groups.

1.3.4 Results

After screening 41 patients for severe comorbidity, dementia, and cardiovascular contra-indications, 28 patients were included. Subsequently, 11 patients and 1 control fell out because of unexpected medical or functional adverse events, leaving 17 patients and 20 controls in the end (Supplement Figure S1). Drop out patients only differed in gender, with more female dropouts.

There were no significant differences between groups on demographic variables. Patients were significantly more depressed ($F(1, 34) = 112.58$; $p<0,001$), more anxious ($F(1, 34) = 25.32$; $p<0.001$) and showed more PR (SSRS) ($F(1, 34) = 33.77$; $p<0.001$) and cognitive impairment (MMSE) ($F(1, 34) =7.48$; $p=0.001$) at baseline assessment (Table 1).

	N	T1 mean (sd)	T2 mean (sd)	T3 mean (sd)	T4 mean (sd)
GDS patient	16	17.69 (4.69)	16.44 (4.43)	12.25 (5.41)	11.63 (6.03)
GDS control	18	4.33 (2.56)	4.33 (2.35)	3.94 (2.56)	3.56 (2.62)
STAI 1 patient	16	50.94 (11.29)	50.31 (8.72)	44.81 (9.85)	44.06 (11.68)
STAI 1 control	19	34.84 (7.89)	32.16 (6.25)	33.53 (6.74)	31.53 (6.81)
SRRS patient	17	15.13 (8.79)	15.19 (7.26)	13.00 (10.03)	10.88 (9.54)
SRRS control		/	/	/	/
SDST patient	13	29.15 (11.68)	30.92 (11.54)	31.85 (12.50)	36.08 (14.68)
SDST control	17	43.35 (9.25)	45.53 (9.33)	46.18 (8.86)	47.71 (10.32)
Stroop 1 patient	14	55.86 (11.07)	58.21 (11.92)	56.57 (12.57)	57.14 (11.43)
Stroop 1 control	18	47.00 (11.45)	47.33 (9.20)	46.00 (7.07)	47.00 (7.83)
Stroop INT patient	13	99.85 (98.55)	74.23 (53.19)	71.31 (53.67)	69.77 (66.98)
Stroop INT control	18	45.83 (22.09)	41.06 (24.94)	39.11 (23.45)	34.67 (17.52)
WCST CAT patient	11	0.45 (0.82)	0.64 (1.03)	0.91 (1.2)	0.91 (1.58)
WCST CAT control	15	2.07 (1.03)	1.47 (0.83)	1.80 (0.86)	1.93 (1.10)
WCST NCORR patient	10	30.40 (12.42)	27.50 (9.89)	28.00 (6.90)	37.70 (10.48)
WCST NCORR control	15	38.67 (9.36)	38.60 (9.09)	40.07 (6.41)	40.60 (7.94)
15 W TOT patient	16	7.81 (2.88)	9.56 (3.35)	8.62 (3.44)	9.75 (3.13)
15 W TOT control	18	9.5 (2.62)	10.72 (2.76)	10.11 (2.89)	10.28 (2.68)
15 W RECALL patient	16	5.25 (3.51)	7.19 (4.28)	6.88 (4.32)	6.81 (3.71)
15 W RECALL control	18	6.61 (3.15)	7.17 (2.62)	8.44 (2.66)	7.56 (2.83)
15 W RECOG patient	14	23.14 (4.26)	24.86 (5.02)	24.79 (4.17)	25.93 (3.45)
15 W RECOG control	17	25.59 (2.62)	26.71 (2.85)	26.00 (2.94)	26.29 (2.76)
CL IT patient	13	1.28 (0.28)	1.15 (0.19)	1.06 (0.22)	1.07 (0.22)
CL IT control	19	0.97 (0.17)	0.93 (0.17)	0.86 (0.14)	0.86 (0.15)
CL MT patient	13	0.69 (0.26)	0.60 (0.22)	0.56 (0.23)	0.54 (0.20)
CL MT control	19	0.49 (0.17)	0.40 (0.13)	0.36 (0.11)	0.36 (0.11)
FC IT patient	12	2.72 (0.50)	2.79 (0.42)	2.68 (0.73)	2.48 (0.58)
FC IT control	19	2.50 (0.78)	2.30 (0.37)	2.25 (0.31)	2.21 (0.35)
FC MT patient	13	3.19 (1.56)	2.65 (1.17)	2.70 (1.81)	2.75 (1.80)
FC MT control	19	2.08 (0.71)	1.89 (0.52)	1.83 (0.47)	1.86 (0.46)
FC ReinT patient	13	0.23 (0.56)	0.18 (0.32)	0.21 (0.61)	0.29 (0.80)

	N	T1 mean (sd)	T2 mean (sd)	T3 mean (sd)	T4 mean (sd)
FC ReinT control	19	0.01 (0.04)	0.06 (0.11)	0.02 (0.01)	0.02 (0.05)
SDST IT patient	9	2.50 (1.70)	2.10 (0.94)	1.92 (1.04)	1.89 (0.94)
SDST IT control	17	1.48 (0.46)	1.37 (0.44)	1.33 (0.37)	1.21 (0.44)
SDST MT patient	9	0.77 (0.22)	0.79 (0.20)	0.81 (0.25)	0.77 (0.18)
SDST MT control	17	0.66 (0.13)	0.64 (0.11)	0.66 (0.16)	0.73 (0.19)

Table 1 Means (and standard deviations) of cognitive and psychomotor assessment scores at baseline and at weeks 2, 6 and 12 suggest differentiated time frames and staging of change.

Following treatment with escitalopram, patients showed a significant response, but no remission (GDS>11). Also, the anxiety scale of patients decreased significantly. The SRRS, showed no significant time effect in patients, likely due to the high variance (1,876<SEM<2.899). Interaction effects were restricted to depression and anxiety, whereas both group effects and learning effects were found for motor times, processing speed and memory. Cognitive measures and cognitive initiation times, on the other hand, only showed differences between groups (see Table S2 [Supplement]). In the cognitive and psychomotor variables, no significant interaction effect of time and group was found. However, all variables, except the memory tests and the SDSTIT, showed significant group differences, favoring the control group. Anxiety, processing speed, memory tasks and psychomotor measures showed a positive evolution over time for both groups, probably due to learning effects. These effects were not found in SSRS, SDSTIT, Stroop and WCST measures. Although no significant interaction effect was found (Figure 1), scrutinizing the means (Table S3 [Supplement]) suggests more subtle and delayed effects for psychomotor and cognitive variables. Yet, after sorting the patients in a high (H) and a low group (L) of processing speed, the high group did not differ significantly from the control group in cognitive and psychomotor variables, except in WCST NCORR ($F(1, 20) = 15.55$, $p=0.001$), whereas the low group did, except in memory measures (Figure 1).

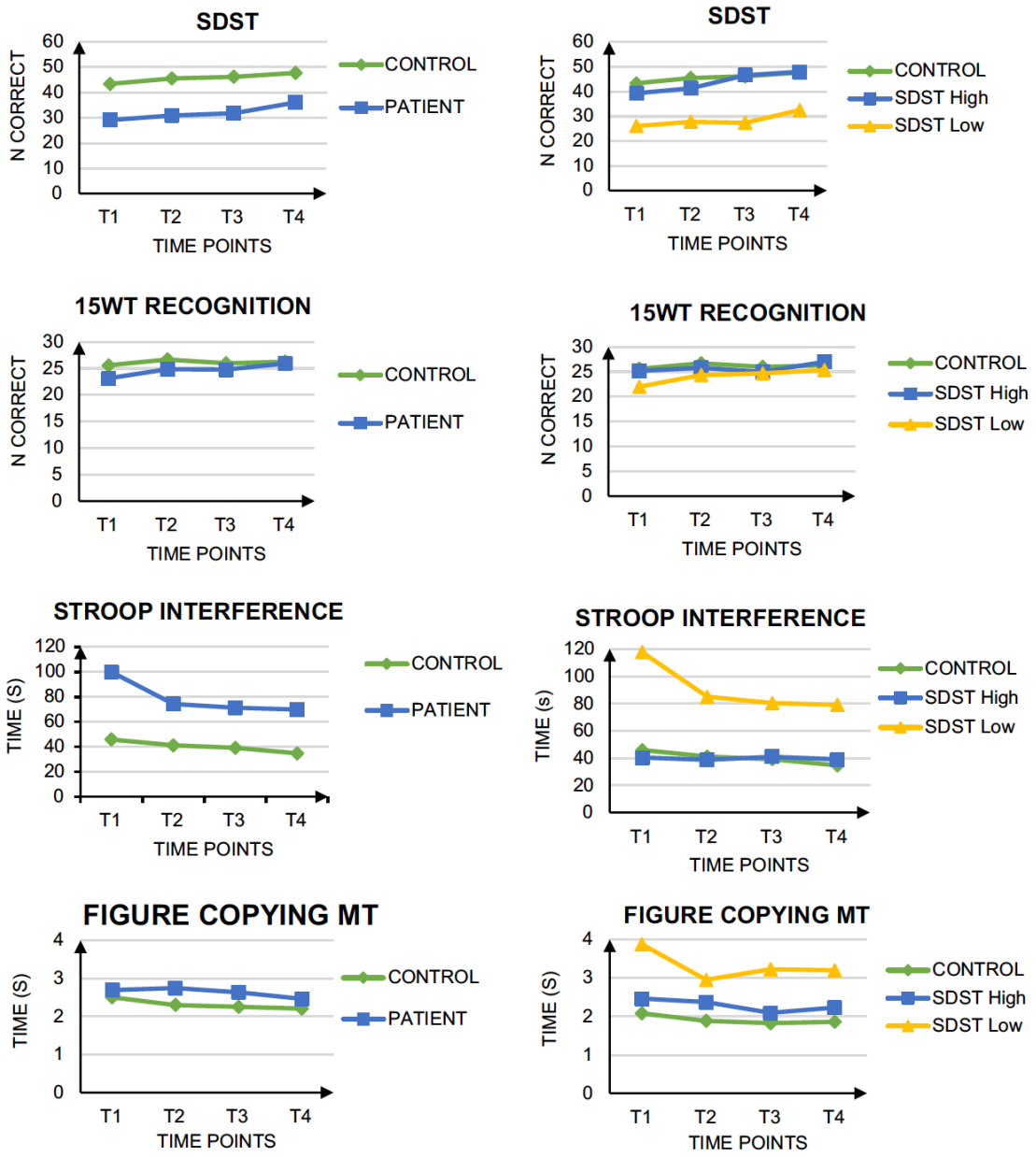

Figure 1 Limited interaction effects were found but also subgroups of high and low score (separated by the median) on SDST in patients

1.3.5 Discussion

The results of this study can be summarized in four points. The main conclusion is that, while a treatment of 12 weeks with escitalopram improves mood to a moderate level, cognitive and fine motor functioning change much less over a same period.

Further, detailed analyses indicated different timeframes and staging's of change, which suggest slowed and delayed change. Even mood symptoms did not reach remission (GDS>11). Scrutinizing the means showed that interaction effects do exist, attenuated,

however, by the typical large variance of populations of higher age. Given the slower and delayed change in symptoms, longer follow up research in elderly would seem indicated.

Also, two subtypes of patients emerged, differing on cognitive and psychomotor functioning. Although this could not be statistically established because of the limited sample, explorative analyses indicated that patients with high scores on SDST did not differ from controls on cognitive and psychomotor measures, though exhibiting the same level of depression as the low score group. The subtype of so-called late life depression and/or the subtype with a dysexecutive syndrome are known to show more lasting psychomotor and cognitive, especially executive, symptoms. Executive dysfunction happens to be the symptom that predicts bad prognosis in treatment with escitalopram. In our baseline study (Beheydt et al., 2015); it became clear that executive dysfunction is typical for depression, and slow processing speed for aging. In our present study, we found that following treatment with escitalopram, some executive functions (Stroop INT) improve, but only in the low-level processing speed group. As in comparison to the control group, high-level processing speed patients were hardly impaired from the start, a ceiling effect prevented improvement (Figure 1). The additive effect of aging and depression on cognitive and PR seems to be an aging –perhaps comorbidity- effect of disturbed processing speed.

Finally, even if an important limitation of the study is the large number of excluded patients and dropouts, studying depressed elderly remains necessary, given the observed specific functional impairing effect of depression in such a population. Rigidly eliminating possible effects of other psychotropic medication, showed, moreover, that elderly depression entails long lasting motor impairment along with specific cognitive defects, particularly in processing speed and executive function. Marked differences in response between core symptoms seems to be peculiarly age related, as, in younger patients with severe PR, mood symptoms generally improve right after psychomotor symptoms and not long before. The interaction effect of physical health, aging and depression, therefore, demands increased attention. However, in geriatric depression, permanent mild cognitive impairment, and PR because of comorbidity should always be taken into account. The differentiated assessment of core symptoms to evaluate effectivity of antidepressants in elderly patients clearly appears necessary.

Evidently, another limitation of the study is the choice of a control group of untreated healthy volunteers, matched for age. The scientifically spoken logical choice to evaluate a specific treatment would have been a group with the same features except the treatment, i.e., a group of depressed elderly without treatment. However, given the invalidating effect of – even mild - depression on elderly, it would not have been ethical not to treat the depression for 12 weeks. Because of important effects and frequently adverse side effects of other types of pharmacological treatment (e.g., tricyclic antidepressive medication), comparing with other types of treatment was beyond the focus of the study, which is the effect of SSRI treatment on psychomotor symptoms of depression. A comparison with

patients matched only for age served as a normative steady standard baseline with respect to depression in the population of the same age, whereby it was assumed that they would not change, and that, therefore, their analysis of within subjects was not relevant. However, using this control group was important in that it offered the possibility of controlling for age effects, which was part of the central question. A comparison with the same treatment in a younger depressive group, though interesting in itself, lay beyond our research goal.

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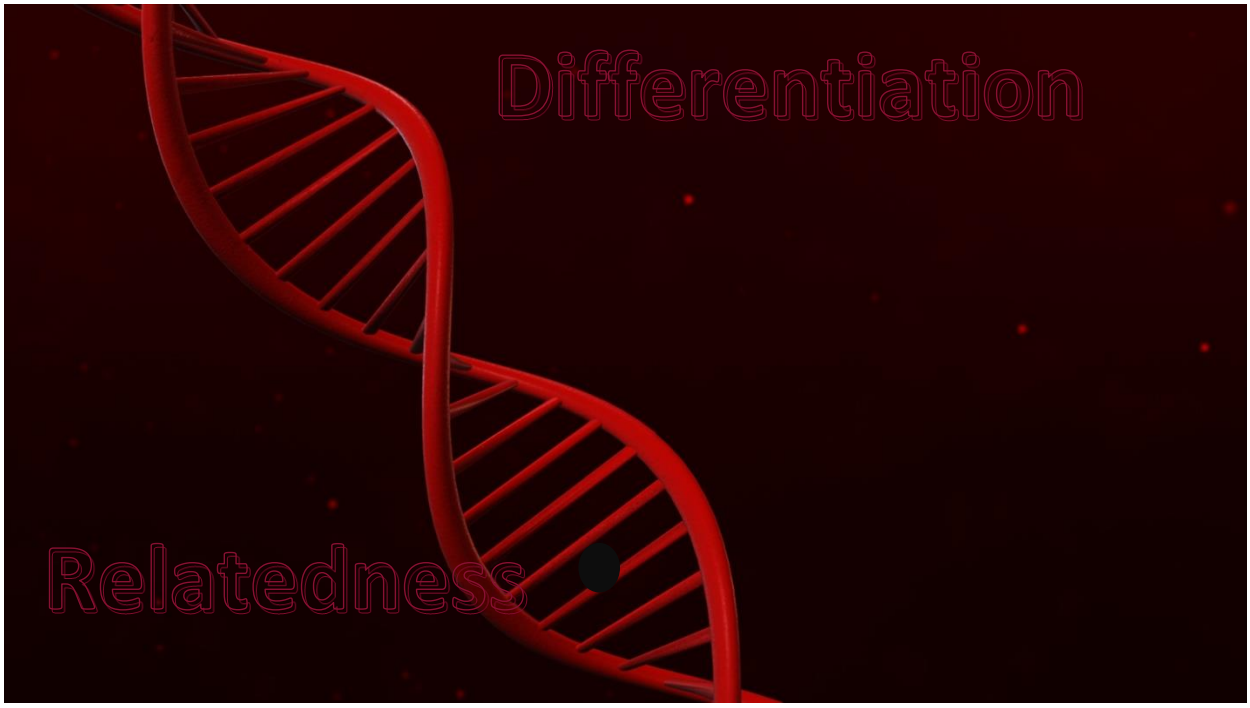
Chapter 2

Part Two: The level of Personality Functioning



[4]

[4] The dialectics of differentiation and relatedness as DNA of personality and personality. Adapted from (2021) DNA structure on abstract red background 3d illustration (graphic). Retrieved 4/2/2022 from https://assets.adobe.com/libraries/urn:aaid:sc:EU:931bf7c9-e698-452e-81a8-a328ac2cc6e1/90f4d1cf-1e27-4caf-8fd3-1a80d4be5b9f

Jimmy

Jimmy was 21 when I first met him. Having been drunk in the weekend, he had severely attacked his brother in a furious assault when they were going out together. Yet, in general, he is fond of his brother. Further, he was absolutely determined to commit suicide within a fortnight, during the summer break, before a new working year would start for him as one of the leaders for the little boys in the local youth organization. The suicide would have to take place at that moment, because he could never forsake his responsibility for the boys once the working year would start again. Over the last two years, he had experienced a sense of aimlessness for which he had already sought guidance or treatment two times.

He had already experienced two depressive episodes. During these episodes, he experienced intense oppressiveness and tightness, as if there was someone inside him who had to get out urgently. It then helped temporarily to write. He also had had suicide thoughts for which he had needed a residential stay in a hospital because of the severity. During that stay, the thoughts became more urgent at the moments he was forbidden to retreat in his room. He could not bear stimuli. The feelings of distress really started after high school, although there had already been some problems before, because school felt as a place where 'dreams were murdered'. He started a study to become a master of cultural studies. Though he was interested, the study was for him too much associated with the narrowmindedness of the 'petit bourgeoisie' he experienced at home and found pretentious, and he 'just did not like being at university'. Still, he switched to sociology in a more liberal university, which exuded less the catholic atmosphere of his family. Quickly enough, he noticed that he did not manage to study, he could not concentrate and especially living in 'student digs' was an ordeal for him. So, he switched again, this time to a study to become a teacher in graduate school and he went back living at home with his mother and father. In fact, he liked that study, but at that moment, Jimmy observed 'he was already too far gone', meaning he was really depressed. Either he felt he showed too little empathy with others, or he had to drink to be able to show empathy. That is when he started drinking systematically for every social event. He always got along well with his friends of the youth organization with whom he had grown up, but he never talked about what was going wrong. At the moment of intake, he felt he was no longer able to take the role of a leader, because he was afraid of the pressure and felt he could not handle the

responsibility anymore. Moreover, he had become afraid of his own impulsivity and the possibility of a rage attack.

Jimmy had already been in two relationships, one of six months, and one of three months. But, in both he suddenly got caught by a moment of taking distance and feeling distance, which he could not understand himself, and, worse, could not explain to his girlfriend. That is why he then decided to break up the relationship. This experience made him thereafter explore whether he was perhaps gay, but he concluded that he felt absolutely not attracted to men. His failure was only due to the feeling that everything was oppressing, to his fear of responsibility and to the feeling that society was too demanding for him. Many times, he condemned himself because he thought he was 'only making excuses'.

Jimmy was born two weeks too late because of breech presentation. As a young child, he stuttered, but in elementary school he was a good pupil and in high school he always came out with more than 70%, studying Latin and human sciences, without really making an effort. Yet, he had a slight developmental motor delay; he was late to start talking and sitting up independently and he was late in swimming and biking. He was not so interested in typical technical 'boys' things' and preferred reading, but only non-fiction. As a child, he played a lot with Lego castles and had a rich imagination. He always had many problems with emotions. During puberty, he tried many music styles, cheerful as well as dark ones, looking for his identity, but he 'only loved dead musicians'. He knew overactive episodes but that never caused problems, as he could spend his excess energy in playing Ice Hockey. He had to stop playing because of a knee problem. Then he started to run and between 15 and 17 years old, he ran 30 kilometers two times a week, he said. However, three years ago, he stopped doing sports. He never had attention problems or never experienced restlessness. He was growing despondent because the antidepressants did not help. He thought he just did not like living and, therefore, behaved self-destructive by misuse of alcohol, smoking, doing nothing all day, and then started to mull it all over until it really got destructive. Jimmy felt bad, particularly in the morning, and he isolated himself. He was also worried, especially about his lack of empathy, although friends did not recognize that problem. They said that, in his usual state, he was friendly, intelligent, a little moody and somewhat malicious and averse to hierarchy.

2.1 Introduction: The level of personality functioning

2.1.1 The level of personality functioning: theory driven

According to DSM-5 (American Psychiatric Association, 2013), a personality disorder (PD) is an enduring pattern of inner experience that deviates markedly from the expectations of the individual's culture. This pattern is manifested in two (or more) of the following areas: cognition, affectivity, interpersonal functioning, or impulse control (DSM-5; American Psychiatric Association, 2013). In the prevailing diagnostic system of DSM-5 II, however, neither a measure of PD nor a measure for severity of PD is available. Because PDs as diagnosed with DSM-5 do not seem to demand pharmacological or very specific psychotherapeutic interventions, assessment of PDs is often omitted in psychiatric diagnosis or restricted to 'personality diagnosis Not Otherwise Specified, NOS' (Kirk, 1994). However, while, indeed, the deviation from expectancies cannot justify the need for treatment, the corresponding problems in functioning, especially in personal achievements and interpersonal functioning do.

In contrast to DSM-5, the psychoanalytic tradition proposed a theory-driven approach to classify patients in three levels of functioning following the theory of Kernberg[5] on the structure and organization of personality functioning (Kernberg & Caligor, 2005). Three levels of personality organization (PO) can be discerned, based on combinations of specific problems in ego functions (Table 1). The psychotic personality organization (PPO) is characterized by impaired reality testing (e.g. delusions, not checking or doubting beliefs), marked identity diffusion (lacking a coherent and stable positive sense of self), and the use of primitive defenses like splitting (in all-good or all-bad others) or projection (projecting unaccepted or intolerable feelings in the other). In the borderline personality organization (BPO), reality testing is relatively intact, but identity is diffuse and defense mechanisms are primitive. In the neurotic personality organization (NPO), finally, none of the three features indicative of disturbed object relations is manifest. In this theoretical thinking with a gradational manifestation of deficits, three levels of personality functioning emerge. Though this theory inspired clinical and psychotherapeutic thinking, it never played a prominent role in the development of evidence-based psychiatry and psychotherapy. The model itself was not empirically supported until the publication of the Inventory of

[5] This theory is a combination of psychoanalytical theory on object relations and ego-psychology. The first is a model on deficits in personality development and attachment by interaction with early caregivers (Klein, 2002), the second is an intrapsychic model based on conflicts between impulses and defenses in function of adaptation. According to Kernberg (1976), the structure of personality is an invisible enduring and relatively stable construct based on the internalized object relations, a representation of the self in relation to others. This invisible structure can be derived from the surface structure of the personality organization, a combination of ego-functions (identity integration, defense mechanisms and reality testing) that are indicative for the underlying structure.

Personality Organization (IPO; Lenzenweger, Clarkin, Kernberg & Foelsch, 2001), a self-report measure, in 2001. The publication of the IPO elicited numerous studies on the structure of personality organization, but findings suggest that structural personality pathology may not be fully captured by self-report (Eurelings-Bontekoe, Luyten, Remijsen, & Koelen, 2010). In addition, although on theoretical grounds, the dynamic personality organization is considered a proxy of the stable but invisible personality structure (Kernberg & Caligor, 2005); the relationship between the two cannot be validated easily. The present study is intended as a further step in the empirical validation process of the levels model of personality functioning.

Problem	Psychotic personality organization (PPO)	Borderline personality organization (BPO)	Neurotic personality organization (NPO)
Reality testing	impaired	relatively intact	intact
Identity integration	markedly diffuse	diffuse	intact
Use of defenses	primitive	primitive	intact

Table 1 Kernberg's types of personality organization as levels of personality functioning (Kernberg & Calligor, 2005)

2.1.2 DSM-III and further: descriptive and consensus-driven

With the waning of psychoanalytic theory on classification, DSM-III provided a shift toward a descriptive approach to personality pathology (and psychiatric disorders more generally). Thereto, DSM-III (American Psychiatric Association, 1980), IV (American Psychiatric Association, 1994) and 5 (American Psychiatric Association, 2013) were set up with categories of PDs defined by clinical prevalent combinations of observable personality traits, together with thresholds of the number of features necessary for diagnosis. In DSM-IV-TR and DSM-5, there are 12 PD diagnoses of which 2 in the appendix (depressive and passive-aggressive PD) and 10 placed in three clusters defined by a common behavior style. Cluster A with bizarre or eccentric behavior includes the paranoid, the schizoid and the schizotypal PD, cluster B, the cluster with dramatic, emotional, or erratic behavior includes the antisocial, the borderline, the histrionic and the narcissistic PD and cluster C, standing for anxious and fearful behavior comprises the dependent, the avoidant and the obsessive-compulsive PD (American Psychiatric Association, 2013). In these descriptions of observable features, consisting of symptoms, behavior, subjective experiences and affective states, each feature is weighed equally.

DSM-IV-TR PDs	CLUSTER A	CLUSTER B	CLUSTER C	Not otherwise specified
	Paranoid PD Schizotypal PD Schizoid PD	Borderline PD Narcissistic PD Histrionic PD Antisocial PD	Avoidant PD Dependent PD Obsessive-Compulsive PD	Depressive PD Passive-Aggressive PD
DSM-5 III PDs Only evidence-based	Schizotypal PD	Borderline PD Narcissistic PD Antisocial PD	Avoidant PD Obsessive-compulsive PD	

Table 2 Ruling out personality disorders with lack of evidence in DSM-5 III (APA, 2013).

It soon became apparent that this approach was fraught with problems. I would write: These included, among others, arbitrary thresholds (Krueger, 2013), extensive co-occurrence of PDs, heterogeneity among patients receiving the same diagnosis, temporal instability of PD diagnoses occurring at rates incompatible with the definition of a PD as 'lasting over time' in contrast to state or symptom disorders that were seen as temporary, arbitrary diagnostic thresholds in polythetic criterion sets with little or no empirical basis, limited validity and clinical utility, and poor convergent validity (Morey, Skodol & Oldham, 2014).

The categorical model of PDs in DSM-IV (American Psychiatric Association, 1994) was flawed and therefore often clinically unusable. Patients could meet the criteria for borderline disorder in 256 possible combinations with 5 out of 9 symptoms for diagnosing borderline PD (Galatzer-Levy & Bryant, 2013). In the case of PDs, a lot of patients have fallen into the category of 'not otherwise specified' (PDNOS; American Psychiatric Association, 2013) because of missing some criteria of a specific disorder or because they corresponded to a PD not recognized by DSM (masochistic PD or depressive PD). Subsyndromal PDs were not identified, and more severe personality pathology was more likely to correspond with multiple PDs, up to five or more (Skodol, 2014). This lack of reliability and validity of the categorical model of PDs in DSM-5 led to clinical impracticability, which was compensated in subsequent multicenter studies of DSM-IV by adding to almost half of the PDs the requirement that symptoms "cause clinically significant distress or impairment in social, occupational, or other important areas of functioning" and by the reassurance that "there is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries dividing it from other mental disorders or from no mental disorder" (American Psychiatric Association, 1994 and 2000). The addition of the latter two statements was symptomatic for the weaknesses of the diagnostic system and was an attempt to cover up two major problems, the extensive comorbidity, and the lack of differentiation from normality. Although multicenter research

improved the reliability of the content of the criteria, the validity remained problematic. Defining PDs had become harder and severity estimation was trusted again to the appraisal of the clinician without a decision algorithm. Thus, the reliability of the diagnosis was jeopardized.

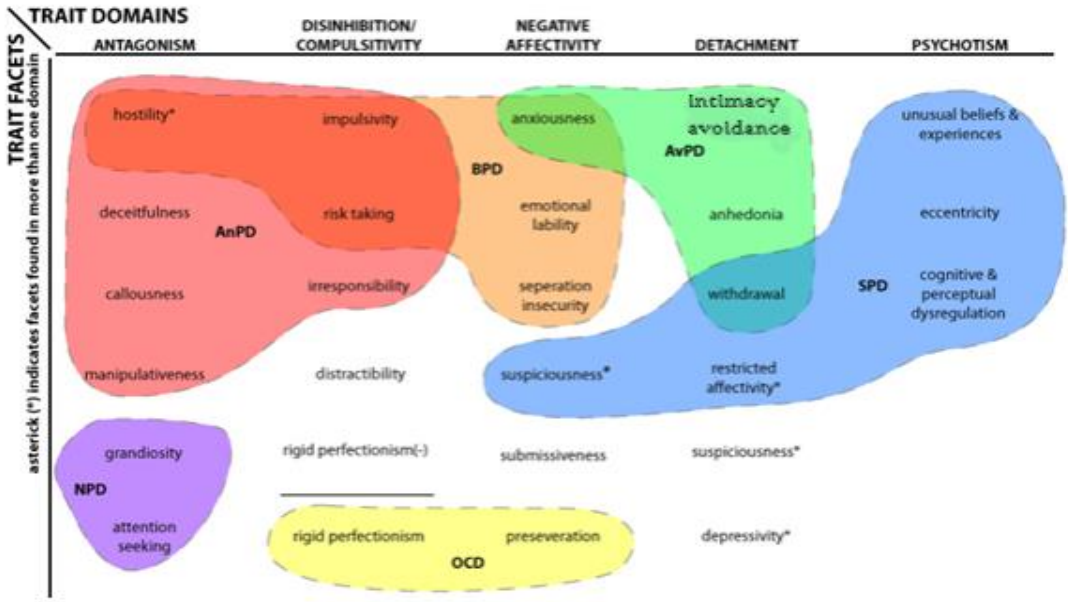


Figure 1. The overlap of traits between categorical personality disorders (PD). AnPD = antisocial PD, BPD = borderline PD, AvPD = avoidant PD, SPD = schizotypal PD, NPD = narcissistic PD, OCD = obsessive Compulsive PD. [6]

Reliability is a signal-to-noise balance, and there are two sources of noise in diagnosis, inconsistency of expression of diagnostic criteria and application of criteria by the clinician. Both are intolerably high in DSM-IV-TR (American Psychiatric Association, 2000). The ambiguous profiles are intrinsic to the determination system, which is, moreover, so complicated that it is virtually impossible to apply it accurately (e.g., Kraemer, Kupfer,

[6] *Note: Facets organized within trait domains. DSM 5 Personality Disorder Map. Adapted from 'Combinations in the Alternative DSM-5 Model for Personality Disorders, In SciFI Central: Sociopathy and Personality Types, by doctor Scifi (2014), retrieved february 4, 2022, from http://scificentralsociopathy.blogspot.com/2014/09/combinations-in-alternative-dsm-5-model.html*

Clarke, Narrow, & Regier, 2012). Therefore, the conditions for reliability are violated. And without reliability, no validity.

2.1.3 Rebooting the approach to personality pathology

Faced with the above-summarized problems, a Task Force was formed to develop a new approach to personality pathology. That resulted in a hybrid system, combining traits and level of personality functioning, which, remarkably, was only included in the DSM-5 in section III for further research. In the meantime, the 'old' approach of DSM-IV remained largely in place.

2.1.3.1 *The alternative DSM-5 proposal, section III*

In section III of DSM-5, an alternative hybrid categorical-dimensional multiple level model is proposed in which the 'severity' Criterion A, presenting impairments in self (identity and self-direction) and interpersonal functioning (empathy or intimacy), is separated from the 'style' Criterion B, presenting one or more out of 5 pathological trait domains (negative affectivity, detachment, antagonism, disinhibition, and psychoticism) or 25 trait facets (e.g., eccentricity). To assess criterion A, an index is used of overall severity of personality impairment in self-definition and in interpersonal relatedness. To assess criterion B, a dimensional model of pathological personality traits is presented to replace the diagnostic criteria of DSM-IV (American Psychiatric Association, 1994). The trait domains, similar to the Personality Psychology Five (Psy5) (American Psychiatric Association, 2013, p773) are perceived as the maladaptive counterpart traits of the Big Five personality traits (extraversion, neuroticism, openness to experience, agreeableness, and conscientiousness), also known as the Five Factor Model (FFM) of normal personality. The five maladaptive traits are detachment, negative affectivity, psychoticism, antagonism, and disinhibition. It is subsequently specified that only six of the ten original PDs be retained: schizotypal, antisocial, borderline, narcissistic, avoidant, and obsessive-compulsive.

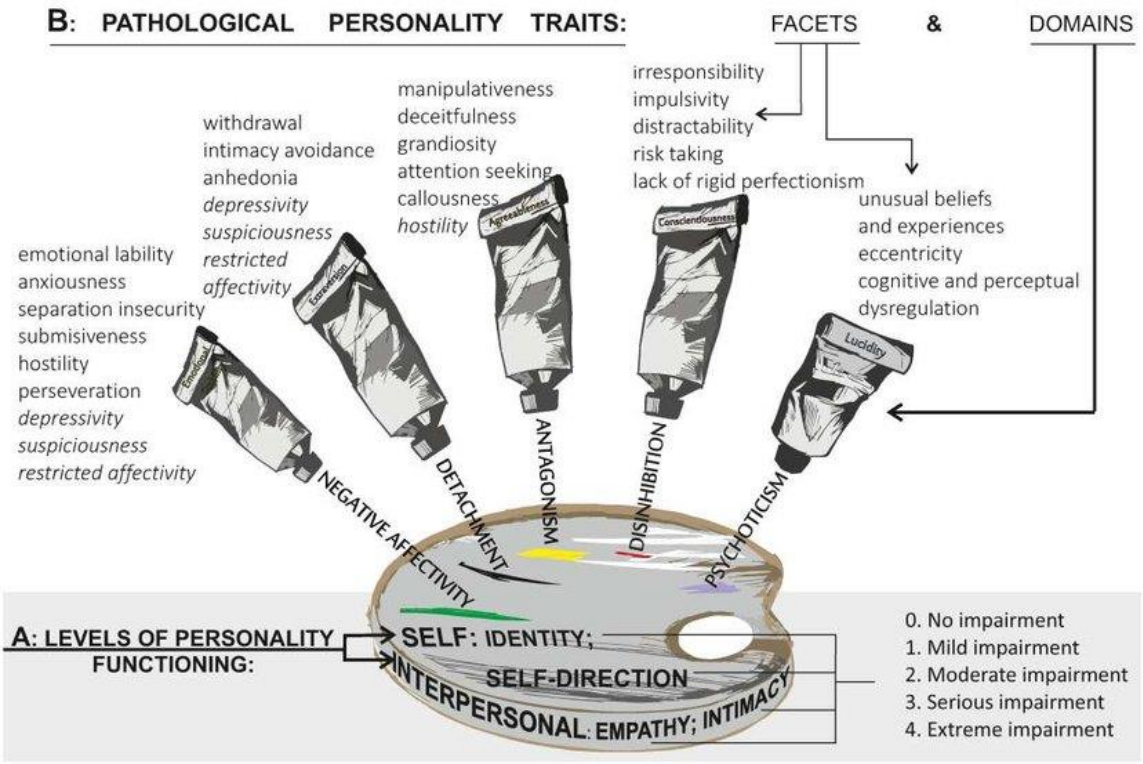


Figure 2 How dimensions of traits and of personality functioning concur in the Alternative Model of Personality Disorders (AMPD) in DSM-5 section III (American Psychiatric Association, 2013)

[7]

2.1.3.2 *Dimensional trait-modeling of Personality Disorders in DSM-5 III, criterion B*

Dimensional models of personality pathology are believed to better fit clinical use and empirical data (Skodol, 2014). There is yet considerable evidence that most common forms of general psychopathology can be divided in internalizing and externalizing pathology clusters. A third, meta-cluster, is characterized by bizarre, eccentric, or unusual behaviors and cognitions. These vast dimensional domains correspond well to and correlate with the five pathological trait domains in the alternative model of PDs (Negative Affectivity, Detachment, Antagonism, Disinhibition, and Psychoticism). Antagonism/low Agreeableness and Disinhibition/low Conscientiousness are related to externalizing behaviors, whereas Negative Affectivity/Neuroticism is most significantly related to internalizing symptoms (Sleep, Hyatt, Lamkin, Maples-Keller, & Miller, 2018). In the hierarchical factor analysis of the FFM, detachment was moreover related to externalizing

[7] *Note. Reprinted from Popiel & Zawadski (2017). Diagnosis of personality disorders: Selected methods and models of assessment. Roczniki Psychologiczne, 20 (2), p243. (https://doi.org/10.18290/rpsych.2017.20.2-1en). © 2017, Towarzystwo Naukowe Katolickiego Uniwersytetu Lubelskiego.*

behavior (Wright & Simms, 2014). With the advocacy of the addition of the third meta-cluster with bizarre behavior in the structure of psychopathology (e.g., Kotov et al., 2011; Caspi, 2014), also the psychoticism dimension of the FFM was represented. Thus, patterns of co-occurrence of disorders within the broad spectrum of psychopathology were commonly found (Skodol, 2014). Higher-order personality traits such as internalizing, externalizing, or thought disorders (unusual cognitions) proved stronger predictors of mental disorder chronicity, suicide gestures, psychiatric hospitalizations and impairments in social, occupational, and leisure functioning than symptoms or states (Skodol, 2014).

The above considerations recently led to the proposal of a Hierarchical Taxonomy of Psychopathology (HiTOP) (Kotov et. al., 2017) as an alternative model to traditional nosologies. It presents psychopathological syndromes and their components/subtypes based on observed covariation of symptoms, grouping related symptoms together and thus reducing heterogeneity. It also combines co-occurring syndromes into spectra, thereby mapping out comorbidity. It, moreover, characterizes these phenomena dimensionally, thus addressing boundary problems and diagnostic instability. The HiTOP thus provides a promising avenue for criterion B. However, in this thesis we will focus on criterion A.

2.1.3.3 *Severity of personality disorder, criterion A*

Because psychopathology and disorders can be described, but not be explained by mere trait elevations, i.e. the B criterion, a second necessary dimension was included for severity of dysfunction, the A criterion of level of personality functioning (Wakefield, 2008). Trait-combinations can discriminate between different disorders, but severity is common to all PDs. Severity of impairment in personality pathology or the level of personality functioning may be the single most important aspect of personality pathology in predicting current and future functioning (Hopwood, Malone, Ansell, Sanislow, & Grilo, 2011) and consists of two domains, self and interpersonal functioning, and four facets, identity and self-direction on the one hand and empathy and intimacy on the other hand (see Bender, Morey, & Skodol, 2011).

Severity of PDs defined as impairment in self and interpersonal functioning is consistent with multiple theories of PD and their research bases, including cognitive/behavioral (Beck, 1983), interpersonal (Horrowitz et. al., 2006; Pincus,2005; Hopwood, Wright, Ansell, & Pincus, 2013), psychodynamic (Blatt, 2008; Luyten, Mayes, Fonagy, Target & Blatt, 2017), attachment (Meyer, & Pilkonis, 2005; Mikulincer, & Shaver, 2007, Chiesa, Cirasola, Williams, Nassissi, & Fonagy, 2017), self-determination theory (Ryan, & Deci, 2006), development (Mayes, Fonagy, & Target, 2007), social-cognitive (Bandura, 1989) and evolutionary theories (see Luyten, & Blatt, 2013) (Skodol, 2012; Skodol, 2014). Impairments regarding the self and interpersonal problems such as maladaptive schemas and insecure attachment have repeatedly been shown to be associated with personality pathology, impairments in psychosocial functioning more generally, and with adverse treatment alliance and outcomes (Skodol, 2014).

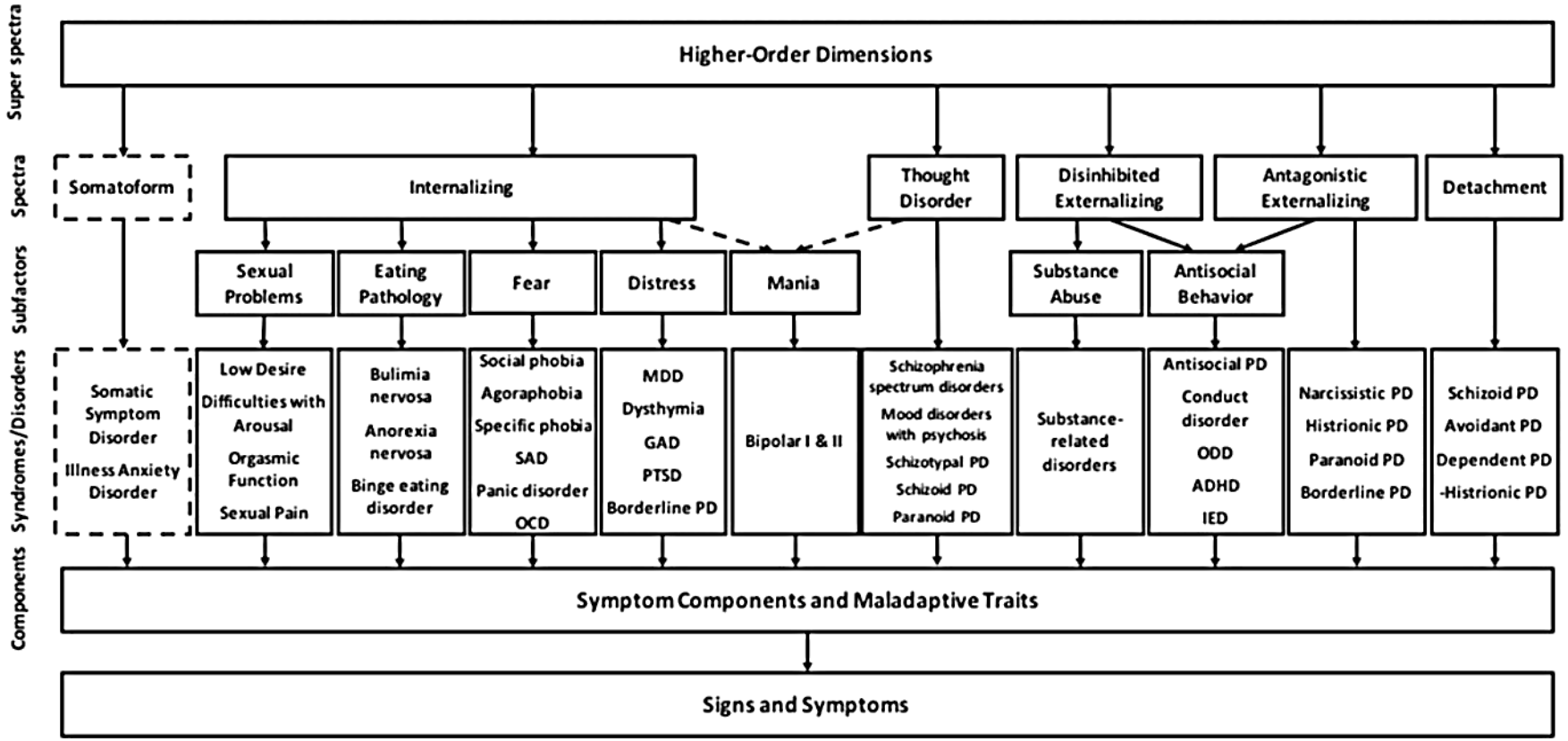


Figure 3 *Hierarchical Taxonomy of Psychopathology classification. (HITOP) (Kotov et al., 2017). ADHD, attention-deficit hyperactivity disorder; GAD, generalized anxiety disorder; IED, intermittent explosive disorder; MDD, major depressive disorder; OCD, obsessive–compulsive disorder; ODD, oppositional defiant disorder; PTSD, post-traumatic stress disorder; SAD, seasonal affective disorder.*

Note: reprinted from Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., Zimmerman, M. (2017). The Hierarchical Taxonomy of Psychopathology (HiTOP): A dimensional alternative to traditional nosologies. Journal of Abnormal Psychology, 126(4), p 462. (http://dx.doi.org/10.1037/abn0000258)

2.1.4 The theory of Blatt on the level of personality functioning

The level of personality functioning is a result of the dialectics, as in object relational theory, between differentiation of self from others (differentiation) and interaction with others (relatedness). This conceptualization refers to the views of Blatt and colleagues of differentiation and relatedness as interactive dimensions that unfold throughout personality development (Blatt, 2008; Luyten & Blatt, 2011, 2013). According to Blatt, personality develops along two fundamental parallel development lines, the relatedness line that involves the development of the capacity to establish increasingly mature and mutually satisfying relationships, and a self-definitional line that involves the development of a consolidated, realistic, essentially positive, differentiated, integrated and mature self-identity. In normal development, these two developmental processes evolve in an interactive, reciprocally balanced, mutually facilitating fashion from birth to senescence (Meehan, & Lévy, 2017). Psychopathology origins in the overemphasis of one of the two developmental lines, creating two types of psychopathologies, anaclitic pathology, with an overemphasis of relatedness, and introjective psychopathology with an overemphasis of self-definition.

In his model, Blatt integrated the structural view on PDs of Kernberg (Kernberg & Caligor, 2005) with the developmental model of Erikson (Erikson & Erikson, 1998). He integrated Kernberg's intrapersonal structural model, framing PDs along a structural and a severity dimension, with the psychosocial developmental view of Erikson (1998), establishing that an individual develops in predetermined stages, solving each time a conflict in the ego caused by social needs, resulting in a positive or negative outcome for personality development. In that process of integration, Blatt transformed the model of Erikson into a dialectical model with alternating stages of focus on interpersonal relatedness and self-definition and introducing a dynamic component in the development, with development as growth but also as possible relapse (Figure 4). Disruptive experiences and biological predispositions together with their interaction can result in exaggerated distortions in one line at the cost of the other, reflecting compensatory or defensive maneuvers against developmental disruptions. This way, personality can be seen as a permanent dynamic attempt to maintain balance between relatedness and self-definition (Luyten, & Blatt in Luyten, 2017). Research suggests that there is no one-to-one relationship between the two dimensions and descriptive PDs. Developmental psychopathology has shown that vulnerability for psychopathology is best conceptualized in terms of equifinality and multi-finality (Cicchetti & Rogosch, 1996) rather than assuming that every disorder has a relatively unique etiology. Measures of severity of PDs should hence be dynamic too, it appears.

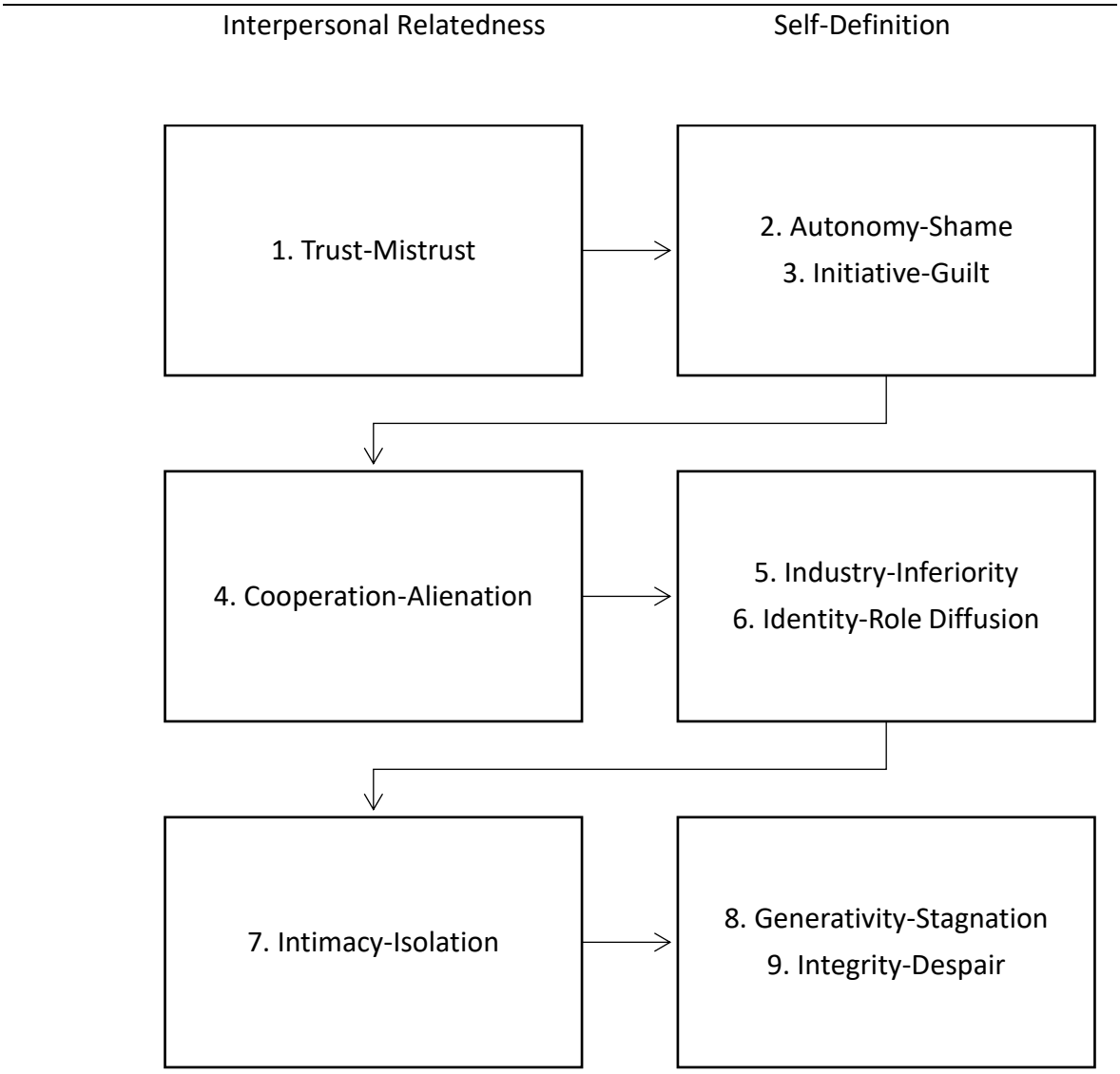


Figure 4 Dialectical development of differentiation and relatedness in Erikson's developmental model (according to Blatt, 2008)

2.1.4.1 *Measurement of the level of self and interpersonal functioning*

Starting from the trait-approach, Widiger and colleagues (Widiger, Trull, Clarkin, Sanderson, & Costa, 2002) developed a way to evaluate the severity or level of personality dysfunction using traits. Two questionnaires were subsequently developed, resulting in a score for severity of personality dysfunction and based on a so called two polarities model of personality (Luyten, & Blatt, 2013) as formulated by Livesley (2006), stating that the level of personality functioning is associated with the level of self and interpersonal functioning. The first ensuing General Assessment of Personality Disorder questionnaire developed by

Livesley contains 290 items (GAPD; Livesley, 2006) but is, unfortunately, not conceptualized with the same dimensions of self and interpersonal functioning as the two polarities model used in DSM-5 (American Psychiatric Association, 2013). Bender, Morey and Skodol (2011) developed the second Level of Personality Functioning (LPF) questionnaire with the proposed two dimensions of self and interpersonal functioning and the four related facets (identity and self-directedness and empathy and intimacy, respectively). Both scales are still in the process of validation and "[T]he only validated measure available is a clinician rating scale" (Anderson, & Sellbom, 2016). Moreover, both questionnaires share the known limitations of self-report (e.g., Ganellen, 2007), that are even more distinct in patients with PDs as they may be limited in insight and awareness (Westen, & Shedler, 2000).

Therefore, it appears appropriate to continue further research on one of the best validated measures in the field, the Differentiation and Relatedness Scale (DRS; Diamond, Blatt, Stayner, & Kaslow, 1991), a ten-level clinician rating scale of the level of personality functioning originating from the two-polarities model of Blatt, yielding one score as a result of the rating of the dialectics between self and relatedness (Table 3). However, at the same time, it is interesting to see in how far there is concurrent validity with a self-report instrument. We therefore investigated its relationship with the well-validated Inventory of Personality Organization (IPO; Lenzenweger, Clarkin, Kernberg, & Foelsch, 2001), a direct self-descriptive instrument based on the theory of Kernberg, resulting in levels of personality organization.

Level	Comments
1. Self/other boundary compromise (physically)	*Basic physical cohesion/integrity of representations is compromised*
2. Self/other boundary confusion (intellectual, affective)	*Affective/intellectual boundaries are confused, fused, or compromised*
3. Self/other mirroring	*Consolidation and stabilization of representations based on mirroring*
4. Self/other idealization or denigration	*Consolidation and stabilization of representations based on unitary, unmodulated idealization or denigration*
5. Semi-differentiation	*Tenuous, semi-differentiated consolidation of representations achieved through primitive splitting and/or rigid adherence to concrete properties to achieve a tenuous cohesion*
6. Emergent, ambivalent constancy (cohesion) and an emergent sense of relatedness	*Emergent differentiated, constant, integrated representation of self and other*
7. Consolidated, constant (stable) self and others in unilateral relationship	*Increasing tolerance for ambiguity*

8. Cohesive, individuated, empathically related self and other	*Representations of self and others as empathically interrelated*
9. Reciprocally related, integrative unfolding self and other	*Representations of self and other in reciprocal and mutually facilitating interactions*
10. Integrative, creative constructions of self and other in empathically and reciprocally attuned relationships	*Reflectively constructed, integrated representations of self and others in reciprocal and mutual relationships*

Table 3 The 10 levels of the Differentiation-Relatedness Scale (see also Huprich, Auerbach, Porcerelli, & Bupp, 2016)

2.1.5 The p-factor: a novel approach to Personality Pathology?

2.1.5.1 *Introduction*

Quite separate from the work on DSM, Caspi et al. (2014) advanced the idea that a p-factor, one general psychopathology factor, underlies the structure of personality pathology. This hypothesis has attracted considerable research attention. Yet, more research is still needed. In this section, I first describe the approach that led to the idea of a higher-order dimension of 'general psychopathology' and then discuss its results and limitations.

2.1.5.2 *The p-factor or the 'general psychopathology'-factor*

Systematic covariation between disorders and high comorbidity rates in psychopathology have recently created the presumption of one or more latent dimensions. Caspi and colleagues (2014) showed that relative to the generally accepted correlated trait-factors model of internalization, externalization and thought disorder, a one p-factor model, standing for general psychopathology vulnerability, was a reasonable model. However, the best fitting model proved a model, which allows both specific traits and a general p-factor simultaneously, not hierarchically modeled as presumed in the trait model, but in a bi-factor model. In the bi-factor model PD criteria load on a general factor whereas unique criteria of PDs load on additional factors. Many publications followed and corroborated this bi-factorial modeling of psychopathology. Thus, the p-factor is not the communality of internalization and externalization but the communality of all individual disorder-items.

Although Caspi's model (Figure 5) was based on general psychopathology, it is in line with the hybrid conceptualization of PDs in specific traits and a general psychopathology factor. Caspi et. al. (2014) showed, moreover, that higher p-scores are associated with more life impairment, greater familiarity, worse developmental histories, and more compromised early-life brain function. The model can explain why it is challenging to find causes, consequences, biomarkers, and treatments with specificity to individual disorders (Caspi et. al., 2014). The clinical relevance of the p-factor is impressive. For instance, extracting the p-factor revealed to Caspi et. al. that much of the propensity to persistent conduct disorder from adolescence to midlife was indicative of general psychopathology rather than specific to an externalizing style (Caspi et. al., 2014). Such findings have important consequences for treatment planning, such as differentiating style features from

susceptibility, or making provision for more intensive and long-term care. In the meantime, also some studies on the p-factor in personality pathology appeared, which might explain generally shared clinical experience. For instance, the 256 combinations of obtaining a borderline PD (BPD) diagnosis can partly be explained by the fact that BPD criteria load principally on the general factor, without simultaneously loading on a BPD specific factor (Sharp et. al., 2015), moreover, the comorbidity between substance disorder and mainly cluster BPDs is characterized by general (pervasive) pathology and by cluster B pathology (Jahng et. al., 2011).

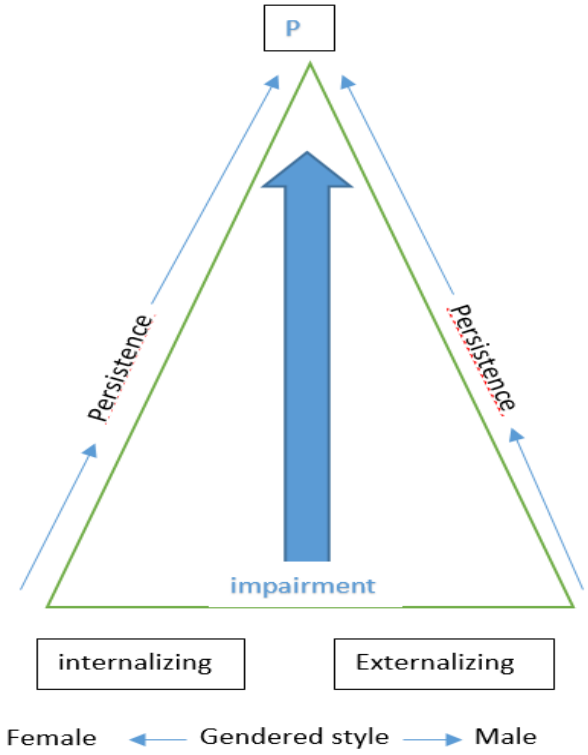


Figure 5 The general factor of psychopathology or the p-factor (Caspi, 2014)[8].

Meanwhile, there is a fast growing amount of studies accumulating evidence for a bi-factor model with a general pathology factor that is independent from specific factors and traits and that is not reducible to negative affectivity, but is a core vulnerability factor defined as 'absence of resilience to adversity' (Chiesa, Cirasola, Williams, Nassisi, & Fonagy, 2016),

[8] *Note. The P factor. Adapted from 'The p Factor: One General Psychopathology Factor in the Structure of Psychiatric Disorders?' by A. Caspi, R. Houts, D.W. Belsky, S.J. Goldman-Mellor, H. Harrington, S. Israel, M.H. Meier, S. Ramrakha, I. Shalev, R. Poulton, & T.E. Moffitt, 2014, Clinical Psychological Science, 2, 119. Copyright, 2014 by the American Psychological Association.*

independently predictive for prognosis of psychosocial functioning (Conway, Hammen, & Brennan, 2016), prognosis of treatment use, comorbidity and suicidality, and common factor for PDs and clinical pathology (Jahng et. al., 2011). The fact that current psychiatric distress was found to be the most consistent predictor of PD, capturing a large share of the variance, and that attachment variables correlate with the presence of PD alone, but have no specific association with particular PDs (Chiesa et al., 2016), suggests that development and epigenetic factors are at work in the general psychopathology factor. Moreover, the p-factor in bi-factor modeling of PDs appeared to be influenced by childhood adversities and Caspi (2014) found the p-factor in general psychopathology to be related to brain integrity, a biological epigenetic feature.

2.1.6 An empirical and theoretical stance

The differences in modeling in personality research (e.g., variable-centered, person-centered, hybrid) and the inevitable impact of subjective interpretation, yet all presented as objective because of the stringent methodology used, make it hard to discern the scientifically and clinically 'better' models. But, as Eaton and colleagues (Eaton, Krueger, Docherty, & Sponheim, 2014) pointed out, it is possible nowadays to use information-theoretic fit indexes and allow for direct comparison of non-nested models to arrive at qualitative comparative evaluation. It is thereby critical to characterize latent structures first, then develop appropriate measurement devices, and finally apply these assessment tools for clinical and research purposes (e.g., Reise & Waller, 2009).

Following that line of reasoning, we will test the structure of personality pathology, by comparing the fit of a one p-factor, a correlated factor, a hierarchical and a bi-factor model with 3 correlated factors and the p-factor at the same time. Referring to Eaton et al. (2014), we acknowledge that also in the way of proceeding, interpretative aspects are inevitable even in data-driven methodologies, particularly when no model emerges as clearly optimal over the others. However, as explained above, we will execute our empirical model within the framework of a coherent theoretical approach based on recent findings. A state-of-the-art study of the most prominent studies informed us, for instance, that all models converge on the importance of hierarchy and adding dysfunction in modeling PDs and their accompanying comorbidity.

2.1.7 Conclusion and framing the present investigation

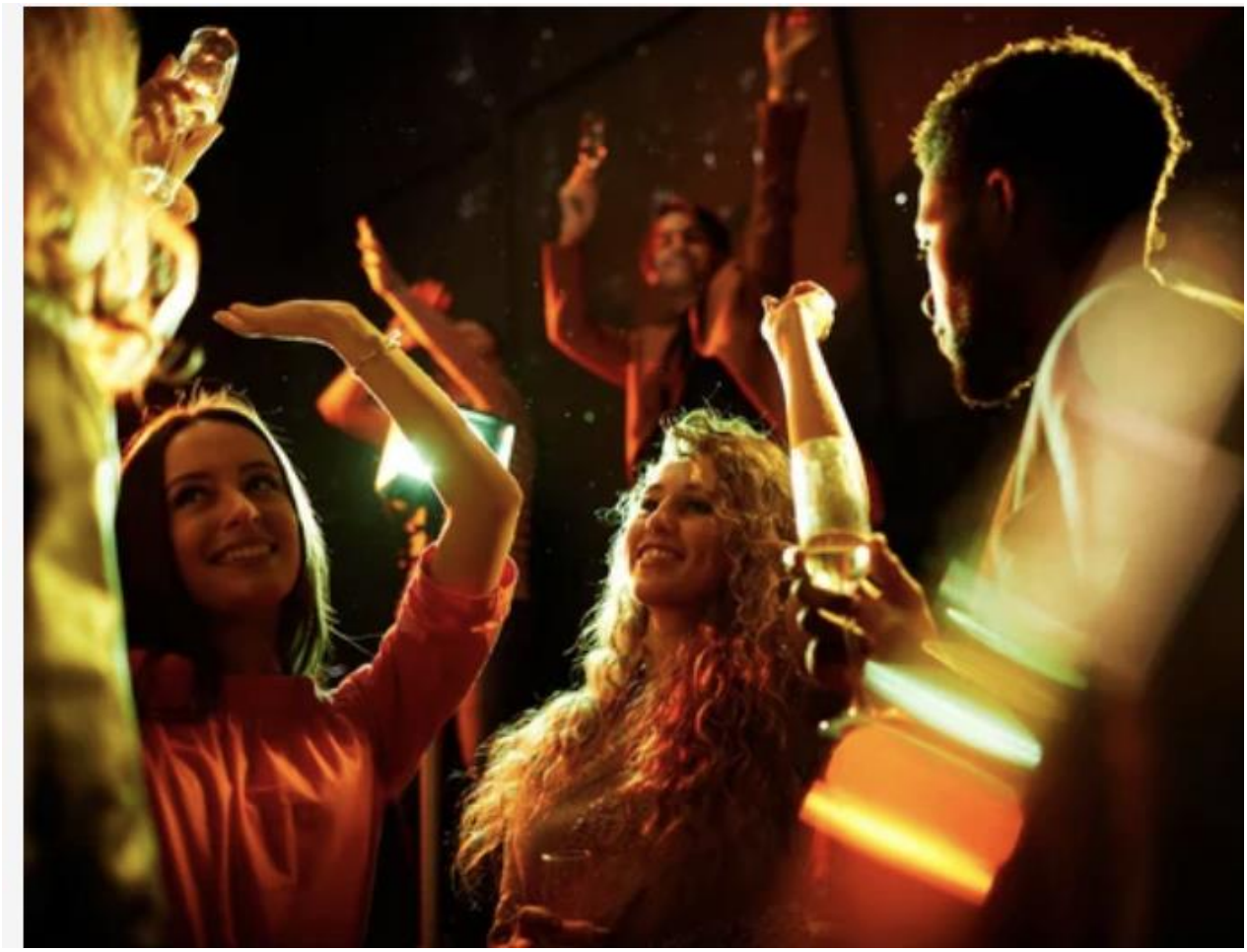
What emerges from the above overview of research on PD since the publication of DSM-5 in 2013 is a double research path. On the one hand, there is the ongoing attempt to present a reliable and valid comprehensive descriptive set of criteria for the classification system of PDs. On the other hand, there is the necessary search for an integrative model that is theoretically underpinned but makes it possible, through empirical bottom-up data driven approaches, to differentiate between core dimensions of personality pathology, general

and specific, and circumstantial influences of person (age, gender, medication, comorbidity, …) and environment.

This double research path is partly caused by the somewhat twin-track presentation of PDs in DSM-5 (American Psychiatric Association, 2013) which, while taking over from DSM-IV (American Psychiatric Association, 1994) the classification system of PDs and their diagnostic criteria, added an alternative multiple level trait model of PDs. But the double line of investigation is certainly also led by the inconveniences with the use of the DSM in clinical practice.

Finding answers for these inconveniences of clinicians in search of tailored psychotherapy for PDs by further substantiating a phenomenological guiding schema as they in general intuitively use to appreciate presenting PDs is the central focus of the following studies. In pursuing this goal, we will explore the level of personality functioning in different aspects that usually tend to be typical fuzzy areas of defining PDs. First, we will study whether the LPF can differentiate normal and abnormal personality functioning in young adults, the developmental stage of consolidation of personality and thus, emerging PDs. Secondly, we will study the level of personality functioning in a population with important clinical distress to investigate whether clinical distress can be disentangled from disordered personality functioning. Finally, we will investigate separately associations of the general factor of psychopathology and of traits with disease, distress, cognitive, and personality features, trying to make a phenomenological and narrative, but evidence-based description of what constitutes a PD diagnosis. After analyzing the nature and associations of the different aspects meticulously, we will come to an organizing lens a treating clinician can use in formulating treatment goals for patients, indicating ‘what works for whom’ in confusing cases with uncertainty whether it concerns a PD at all or maybe just a temporary struggle, whether and when a psychiatric disorder or a PD should be the central focus of treatment and finally, how to choose the focus of treatment in psychiatric patients with several descriptive PDs according to DSM-5 (American Psychiatric Association, 2013).

LEVELS OF RELATEDNESS AND SELF-DEFINITION IN YOUNG ADULTS: ASSOCIATIONS WITH PSYCHOPATHOLOGY AND INTERPERSONAL FUNCTIONING



[9]

Published as: Beheydt, L.L., Van Liefferinge, D., Lowyck, B., Schrijvers, S., Sabbe, B., & Luyten, P. (2020). Levels of relatedness and self-definition in young adults: Associations with psychopathology and interpersonal functioning. Psychoanalytic Psychology, 37(3), 232–240. https://doi.org/ 10.1037/pap0000297

[9] *Note. (2021). Cheerful pretty young ladies dancing with raised arms and talking to handsome men while flirting with them (graphic). Retrieved february 4, 2022 from https://assets.adobe.com/libraries/urn:aaid:sc:EU:931bf7c9-e698-452e-81a8-a328ac2cc6e1/5dcafc8b-f0cc-4d4a-a12a-851145a8c650*

2.2 Levels of Relatedness and Self-Definition in Young Adults: Associations with Psychopathology Features and Interpersonal Functioning

2.2.1 Abstract

Background: The Differentiation-Relatedness Scale (DRS) is a reliable and valid 10-level scale designed to rate levels of personality functioning on narrative descriptions of self and significant others. However, to date, most studies of the DRS have been done in clinical samples. Little is known about its psychometric properties in nonclinical samples. Methods: This study examined linear and potential categorical relationships of the DRS with demographic features and with indices of intrapersonal and interpersonal functioning (i.e., depressive, and dissociative symptoms, dependent and self-critical personality features, and warmth, conflict and depth of intimate relationships), in a nonclinical sample of young adults (N = 333). It also investigated the unidimensionality of the DRS in the relationships between the level of self-representation (DR-S) and representation of the mother (DR-M) and father (DR-F), and the relationship of DR-S with disruptions in the balance between differentiation and relatedness assumed to underlie low levels of DR-S. Results: Results showed little evidence for dimensional relationships between levels of DRS and indices of intrapersonal and interpersonal functioning. By contrast, a cut-off of DRS level 6 clearly differentiated young adults at risk for psychopathology from those with more adaptive levels of functioning. Limitations: Moreover, the DRS seems not to be a unidimensional scale. Conclusion: The implications of these findings for future search and the clinical use of the DRS are discussed.

The publication of DSM-5 (American Psychiatric Association, 2013) has fueled existing criticism of the nomothetic classification approach of personality disorders (PDs) that leads to arbitrary distinction from normal functioning and excessive comorbidity. In response, the proposal of an Alternative Model of Personality Disorders in Section III of DSM-5 has revived interest in the level of personality functioning (LPF; Morey, 2017), the common core that determines severity. Impairments in LPF have inevitable implications for the course of PDs and their treatment, as they relate closely to treatment utilization and the level of care required (e.g., Hutsebaut, Kamphuis, Feenstra, Weekers, & De Saeger, 2017). Since severity is both the best predictor of functional impairment of patients with PD after 10 years (Hopwood et al., 2011) and a better predictor of therapy outcome than PD classification (Bernstein, 1998), early detection of the level of impaired personality functioning appears valuable. The current assumption is that impairments in LPF consist of impairments in self and interpersonal functioning as core features of personality pathology (Morey, 2017). This assumption is consistent with a wide range of personality theories (i.e., psychodynamic, cognitive-behavioral, interpersonal, and trait approaches) and congruent with fundamental assumptions of psychoanalytic object-relations theories arguing that different forms of psychopathology involve impairments in representations of self and

others (Luyten, 2017; Huprich, Auerbach, Porcerelli, & Bupp, 2016). These representations involve cognitive-affective schemas of self in relation to others that develop in interpersonal interactions throughout the lifespan, beginning with interactions with primary caregivers. Age-appropriate frustrations of needs, beginning in infancy, would lead to the development of increasingly differentiated and integrated representations of self and others necessary to deal with new challenges and life tasks. However, when these disruptions are age-inappropriate, persistent, or severe, they are likely to disturb the capacity to accommodate such experiences; this then leads to impairments in the sense of both self and others (Luyten & Blatt, 2011).

Blatt, in his two-polarities model of personality development, described how standard personality development moves toward the emergence of a consolidated, integrated, and individual sense of self-definition and an empathically attuned, mutual relatedness with significant others (Blatt, 2008; Luyten, 2017). Psychopathology then is "an attempt to find balance, however, distorted […] in the dialectical, synergistic interaction between [the] self-definition and relatedness [lines]" (Luyten, 2017, p. 473). Differences in the development of the two lines give rise to specific types of psychopathologies: anaclitic types with a preoccupation with issues of relatedness, and introjective types with a focus on themes of self-worth, self-definition, and self-control (Blatt, 2008).

Adolescence and young adulthood form the pivotal stages in the integration of the two lines. In this second phase of individuation and separation from the parents, youngsters turn to peers for intimate relationships, bringing about a new balance that eventually leads to emerging consolidation of disparate aspects of self and relatedness in young adulthood. Hence, young adulthood is associated with the emergence of psychopathology. Most patients date the onset of their symptoms to the period following puberty (Hopwood et al., 2011), with three-quarters displaying symptoms by their mid-20s (Evans, 2009). However, associations of LPF with symptoms are not straightforward. Blatt did not differentiate between "symptom disorders" and PDs, but he mentioned despair (reflecting a sense of meaninglessness), fragmentation, and lack of purpose as the significant effects of deficient identity integration at adolescence and beyond (Blatt, 2008, p. 128). In addition, lower self-concept clarity and polarized evaluation of segmented aspects of the self can be associated with dissociation proneness in subclinical subjects, independent of childhood trauma, depression, or anxiety states (Chiu, Chang, & Hui, 2017). Even in a nonclinical population, changes in the interaction of neediness (maladaptive dependency) with self-criticism (maladaptive differentiation) predicted suicidal ideation (Campos, Holden, Baleizão, Caçador, & Fragata, 2018). Self-criticism and neediness were found to mediate the relationship between depressive symptoms and perceptions (representations) of maternal caring (Campos, Besser, & Blatt, 2010). Given the reported prevalence of PDs of 18% in college students (Hunt & Eisenberg, 2010) and the period of emerging adulthood laying the foundations for potential future parental roles and thus for the next generation

(Werbart et al., 2011), there is an urgent need for studies focused on the emergence of personality pathology in young adulthood.

2.2.2 The Differentiation-Relatedness Scale

Blatt and colleagues developed the Differentiation-Relatedness Scale (DRS; Diamond et al., 2014; Huprich et al., 2016) to measure LPF. The DRS (Table S1 [Supplement]) assesses the degree of differentiation of self and others and the maturity of relatedness in different representations of self (DR-S) and significant others such as the mother (DR-M) and father (DR-F). The representations are derived from interviews such as the Object Relations Inventory (ORI; Blatt, Wein, Chevron, & Quinlan, 1979), or open-ended one-page written descriptions (Diamond et al., 2014). These are unstructured methods, typical for the assessment of representations. Descriptions of significant others are used to assess the ability to understand both oneself and one's intersubjective matrix.

A considerable body of research has provided evidence for sufficient interrater reliability and both concurrent and discriminant validity of the DRS (Calamaras, Reviere, Gallagher, & Kaslow, 2016; Huprich et al., 2016). An overview of studies conducted with the DRS and conceptually related measures reveals essential features of the DRS (Huprich et al., 2016). First, in clinical populations, lower levels of DR-S were associated with the use of primitive defense, identity diffusion, disturbed reality testing, more clinical dysfunction (Lowyck, Luyten, Verhaest, Vandeneede, & Vermote, 2013; Harpaz-Rotem & Blatt, 2009), and suicide attempts (Kaslow et al., 1998). Increases in DR-S were associated with better clinical functioning (Harpaz-Rotem & Blatt, 2009). In long-term psychoanalytical treatment, patients showed a reduction of low-level responses of DR-S, DR-M, and DR-F, and an increase in responses reflecting object constancy, identity, and intersubjectivity (Diamond, Kaslow, Coonerty, & Blatt, 1990). Reductions in psychological symptoms were associated with increases in DR-S, DR-M, and DR-F (Harpaz-Rotem & Blatt, 2005). Treatment led to a decrease in psychiatric symptoms and a linear increase of developmental levels in DR-S, DR-M, and DR-F; changes in differentiation-relatedness also predicted global symptom severity and personality functioning (Vermote et al., 2010). Overall, these studies suggest that, in a severely disturbed clinical population, positive linear relationships exist between the different subscales (DR-S, DR-M, and DR-F), and between those scales and clinical, psychiatric, or psychological symptoms and personality functioning.

To date, no study has investigated the DRS in non-clinical populations. In a less severely disturbed population of outpatients, the DRS has shown slightly different features. In a study of young adults seeking help for mental health problems, between intake and follow-up, DR-M increased, but DR-S and DR-F did not change significantly (Lindgren, Werbart, & Philips, 2010). Following treatment, increases in representations of others assessed with the DRS were only small (Philips, Wennberg, Werbart, & Schubert, 2007). Moreover, the DRS levels did not relate to the termination of therapy or therapy outcomes (Philips, Wennberg, & Werbart, 2006).

Crucial questions arise from these findings. First, to what extent are the relationships of DRS with clinical features in clinical populations different from such relationships in nonclinical populations? Community samples may not be representative of the full range of severity of psychopathology in the population and could lead to attenuation of linear associations that might exist in the population. Secondly, the theoretical assumption of the DRS is that a rating of 7 distinguishes healthy controls from patients (Blatt, 2008; Diamond et al., 2014), and that ratings of 6 and, more notably, 7 would reflect adaptive levels of personality functioning. Empirical research has shown that levels of DR-S in patient samples typically range from 4.84 (SD = 1.29) (Dirkx & Zevalkink, 2016; Vermote, 2005) to 6.45 (SD = 1.19) (Werbart et al., 2011) depending on the nature of the sample, and that they may increase to levels between 5 (SD = 1.97) (Dirkx & Zevalkink, 2016) and 7.56 (SD = 0.51) (Werbart et al., 2011) as a result of (intensive) psychotherapy. These findings raise the questions of whether there is a threshold level of DRS for the detection of psychopathology in the otherwise dimensionally distributed personality features, and whether there is a point of "good enough" personality functioning as a goal for psychotherapy.

Further, distinct (Werbart et al., 2011) but correlated (Werbart et al., 2011; Dirkx & Zevalkink, 2016) differences in the level of DRS between representations of mother and father have been found in patients seeking psychoanalytic treatment. Changes in DR-S (but not DR-M or DR-F) have been shown to predict therapeutic outcomes (Harpaz-Rotem & Blatt, 2005). Werbart et al. (2011) also found that in a sample of young adults seeking treatment, women's level of DR-F was lower than their level of DR-M and, in contrast, men's level of DR-M was lower than their level of DR-F. These findings question the unidimensionality of DRS as a measure of LPF, which should yield equal levels of DRS for the different representations, whereas DR-S seems to be the prime indicator of LPF.

2.2.3 The Present Study

As the psychometric features of the DRS in nonclinical samples are poorly understood, the present study aimed to explore the validity of the DRS as a measure of LPF in a nonclinical sample. To that general aim, we focused on three specific objects of research: possible sex differences; the relationship of the DRS with symptoms, psychopathology, and interpersonal functioning; and the unidimensionality of the DRS. After ensuring interrater reliability, we first investigated gender differences in DRS. In a standard sample of young adult first-year psychology students, we did not expect to observe gender differences in DR-S, DR-M or DR-F.

Second, we aimed to determine whether a linear association exists, as has been found in clinical samples, between levels of DRS and indices of interpersonal and intrapersonal functioning, or whether relationships of DRS with psychopathology could be categorical, with features differing below and above a specific cut-off point. Our first hypothesis was that in a non-clinical population, the DRS shows no linear associations with symptoms. The second hypothesis was that lower DRS levels are associated with more depressive,

dissociative features, more dependency and self-criticism, but with less support and depth in relationships, and more conflict. Our third hypothesis was that a cut-off of DR-S 7 differentiates between normal and impaired LPF.

Third, we investigated the unidimensionality of the DRS in a non-clinical population. To this end, we studied the relationships between the levels of DR-S, DR-M, and DR-F

2.2.4 Methods

2.2.4.1 *Participants and Procedures*

In this study, 371 young adults, taking a course in psychology at a large university in Belgium, were asked to participate in a study on personality and emotions in exchange for course credits. After giving informed consent, participants were first asked to complete a demographic questionnaire and a series of self-report questionnaires. They were then asked to complete the written version of the Object Relations Inventory (ORI; Levy, Blatt, & Shaver, 1998; see below). Initial screening of the ORIs resulted in the exclusion of 38 participants (10.24%) who did not complete the ORI. We analyzed the scores of the remaining 333 participants (275 females and 56 males; 12 participants did not report their gender). Age ranged from 17 to 24 years (M = 18.62; SD = 1.24). Most participants had attained higher secondary education (97.2%).

2.2.4.2 *Questionnaires*

The written form of the ORI (Levy et al., 1998) assesses "the ability to understand both oneself and one's intersubjective interpersonal matrix" (Huprich et al., 2016, p. 30). Participants were presented with three blank pages and instructed to describe their father, their mother, and themselves in as much detail as possible, with one description per page, and to use as much of the available space as possible. For assessment of the reliability of the ORI, two samples of 15 randomly selected descriptions of mother, father, and self were scored by final-year master's students of psychology with the English version of the DRS after a two-phase training. First, after training in comprehensively distinguishing the levels by reading and discussing them, 15 random protocols were scored. A two-way random effects model (Shrout & Fleiss, 1979) was used to calculate the Intra-class Correlation Coefficient (ICC) (range 0–1), assuming that the same raters, as a sample of all possible raters, scored all ORI protocols. The F-test was then applied to the ICC and detected no significant differences between the raters, $F(1, 15) = 15.14$, but the ICC was not sufficient (ICC =.697). Discrepancies between coders were discussed and solved based on consensus. Both raters then scored another set of 15 randomly selected cases, resulting in sufficient inter-rater reliability for clinical significance (Cicchetti et al., 2011), with an ICC of .73 and no significant difference between raters, $F(1, 15) = .88$.

The Diagnostic Inventory for Depression (DID; Zimmerman, Sheeran, & Young, 2004) assesses the severity of depression, the frequency of symptoms, the psychosocial impact

of depression, and quality of life, as well as a diagnosis of depression according to the DSM-IV algorithm (American Psychiatric Association, 1994). Estimates of internal consistency (Cronbach's α) were .887, .755, and .850 for severity of depression, psychosocial impairment, and quality of life, respectively.

The Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986) assesses the frequency of different dissociative symptoms. The scale consists of three subscales evaluating the degree of amnesia, depersonalization or derealization, absorption, and imaginative involvement. Cronbach's αs were .796, .812, and .855, respectively.

The Depressive Experiences Questionnaire (DEQ; Blatt, D'Afflitti, & Quinlan, 1976) assesses two personality dimensions (dependency and self-criticism) that have been shown to confer vulnerability to a wide range of types of psychopathologies. High scores for dependency are suggestive of maladaptive relatedness, and high scores for self-criticism reflect maladaptive levels of differentiation. The Dutch version of the DEQ used in this study (Luyten, Corveleyn, & Blatt, 1997) has similar psychometric characteristics to the original version. Scores were calculated using the factor scores and loadings of the original DEQ (Blatt et al., 1976). The reliability of the DEQ, as measured with Cronbach's α, was .769.

The Quality of Relationships Inventory (QRI; Pierce, Sarason, Sarason, Solky-Butzel, & Nagle, 1997; Verhofstadt, Buysse, Rosseel, & Peene, 2006) measures support, conflict, and depth with a specific self-designated relationship in mind. Cronbach's αs were .891, .901, and .836, respectively. Relational dysfunction is a core feature of PDs.

2.2.4.3 *Statistical Analysis*

Because the DRS is an ordinal scale in which each level is only an indication of ranking in the ordered levels, a normal distribution cannot be assumed; therefore, we used nonparametric calculations, which we performed using SPSS 25.00. First, we calculated descriptive statistics and distributions of DR-S, DR-M, and DR-F in both genders, as well as the difference between these distributions. Kendall's τ provided correlations of the DRS scales with gender, educational level, and age.

Next, we formally tested with contrast linearity tests in ANOVA the linearity of associations between the DRS and indices of intrapersonal and interpersonal functioning. We also tested whether a quadratic U-shaped relationship outperformed linear relationships. Curve fitting was used to estimate and visualize both types of relationships for the total population, because trends inferred from a selected range of a full population can be misleading (e.g., Mendoza & Mumford, 1987).

Relationships of the DRS with target variables were calculated by using Kendall's τ correlations. Differences in the distributions of psychopathology and interpersonal features between the different levels of DRS were computed with the Kruskal–Wallis test to test the predictive value of the DRS levels for these features. Next, to empirically investigate the theoretical cut-off of DR-S level 6 or 7, the DRS was categorized into levels, and contrasts between categorical regressions with dummy variables of DRS level on symptoms at each ordinal level were used to explore the possibility of a cut-off. We also calculated the sensitivity and specificity (Area Under the Curve, AUC) of DR-S for the discriminative threshold at level 5 versus level 6. Two groups could be delineated based on a cut-off score, with a significant contrast between DRS ≥6 (high group) and DRS <6 (low group). Then, differences in symptoms and psychopathology dimensions between the high- and low-level DRS groups were tested using the Mann–Whitney U test with Bonferroni correction for multiple comparisons. Eta squared η2 or ε2 (Lenhard & Lenhard, 2016) were used as effect sizes to compare differences.

To test the unidimensionality of the DRS and the relationships between DR-S and the subscales DR-M and DR-F, we calculated the inter-scale correlations with Kendall's τ and tested whether DR-M and DR-F were associated with DR-S by testing the concordance in the ranks between DR-S, DR-M, and DR-F in the related samples. Subsequently, associations of DR-S and categorical high and low DR-S with dependency, self-criticism, and the interaction of dependency and self-criticism were calculated.

2.2.5 Results

2.2.5.1 *Descriptive Statistics*

The distribution of DRS in the sample ranged from DR-S 4 to DR-S 8, with only one count each for level 2 and level 9 (Table S2 [Supplement]). Distributions of DRS in males and females were not significantly different (U DR-S = 7641, SE = 491.43, p = .438; U DR-M = 7249, SE = 531.11, p = .666; U DR-F = 6778, SE = 474.62, p = .113). There were no significant relationships between DR-S, DR-M, or DR-F and the demographic variables age and gender (Table S3 [Supplement]), and only a small positive correlation between level of education and DR-S (r = .121, p = .022).

2.2.5.2 *Relationships of DRS with Symptoms, Psychopathology, and Interpersonal Functioning*

Investigation of linearity with ANOVA linearity contrast tests (Table S4 [Supplement]) revealed that only nonlinear or combined linear and nonlinear associations of DR-S were significant after Bonferroni correction. Specifically, nonlinear associations of DR-S with indices of dissociation and conflict in relationships and combined linear and nonlinear associations with indices of dissociation and depression were significant, with moderate effect sizes (Lenhard & Lenhard, 2016) of η2 > .06. However, curve fittings on the scatter plots (Figure S1 [Supplement]) showed that the relationships were neither linear nor

quadratic, and hence not continuous. Therefore, the DRS appeared to be an ordinal scale requiring nonparametric analyses in this sample.

Nonparametric Kendall's τ correlations between DRS and symptoms, psychopathology dimensions, and interpersonal functioning (Table S3 [Supplement]) showed that there was only one, and small, significant correlation, namely between DR-F and dissociative features. With the Kruskal–Wallis test (Table S5 [Supplement]), we investigated whether distributions of the features differed over the different levels of DRS. Only the level of DR-M was significantly associated with the depth in relationships after Bonferroni correction.

Categorical regression of DRS subscales on the selected features (Table 1) revealed that only categorical regression models of DR-S were significant after Bonferroni correction, with significantly different effects between the levels of DR-S on severity of depression, dissociative features, and conflict in relationships.

Model summary	Adj. R^2	*SE* Estimate	F_{Change}	(df1,df2)	*p*	Durbin-Watson
DR-S						
DID-SEV	.048	7.731	3.777	(6, 323)	.001*	1.732
DES	.077	11.406	7.533	(4 , 311)	.000**	1.734
DEQ-DEP	–.004	0.915	0.709	(4, 312)	.587	0.803
SC	.018	0.844	2.436	(4, 312)	.047	0.852
QRI-S	.012	0.683	1.979	(4, 312)	.098	1.868
QRI-C	.046	0.553	4.790	(4,312)	.001*	1.767
QRI-D	.002	0.607	1.166	(4,312)	.326	1.836
DR-M						
DID-SEV	.028	7.813	3.362	(4, 325)	.010	1.702

DES	.017	11.641	2.425	(4, 324)	.048	1.658
DEQ-DEP	–.007	0.914	0.445	(4, 325)	.776	0.805
SC	.015	0.851	2.275	(4, 325)	.061	0.861
QRI-S	.037	0.678	3.087	(4, 325)	.016	1.924
QRI-C	.014	0.561	2.179	(4, 325)	.071	1.733
QRI-D	.023	0.023	2.897	(4, 325)	.022	1.786
DR-F						
DID-SEV	.014	7.870	2.141	(4, 325)	.076	1.741
DES	.015	11.654	2.228	(4, 324)	.066	1.663
Model summary	Adj. R^2	*SE* Estimate	F_{Change}	(df1,df2)	*p*	Durbin-Watson
DEQ-DEP	.000	0.910	1.034	(4, 325)	.390	0.810
SC	.004	0.856	1.312	(4, 325)	.265	0.894
QRI-S	–.003	0.687	0.777	(4, 325)	.541	1.824
QRI-C	.000	0.565	0.980	(4, 325)	.481	1.728
QRI-D	.005	0.605	1.422	(4, 325)	.226	1.767

Table 1 Categorical prediction models of regressions with dummies

Note. DRS = Differentiation–Relatedness Scale; DR-S = DRS in descriptions of the self; DR-M = DRS in descriptions of mother; DR-F = DRS in descriptions of father; DID = Diagnostic Inventory for Depression; DID_sev = severity of depression; DES = Dissociative Experiences Scale, frequency; DEQ = Depressive Experiences Questionnaire; DEQ_sc = DEQ self-criticism; DEQ_dep = DEQ dependency; QRI = Quality of Relationships Inventory; QRI_s = QRI support; QRI_c = QRI (conflict; QRI_d = QRI depth.

** p <.05; **p <.01; ***p <.001 after Bonferroni correction*

Effect sizes: R2 < .01, no effect; .01 ≥ R2 < .06, small effect; .06 ≥ R2 < .14, intermediate effect; R2 ≥ .14, large effect (Lenhard & Lenhard, 2016).

Contrasts (Table S6 [Supplement]) between the dummy regressions of the different levels of these three features showed similar patterns. The same similarity of level in patterns of contrasts applied for DR-S and DR-M, but the regression models of DR-M were not significant. Importantly, there were no contrasts in levels of DR-F. DR-S and DR-M effects on severity of depression or dissociative features, and DR-S effects on conflict in relationships, differed significantly between levels 5 and 6 or 7 (or 8). From level 6 on, the association between DR-S and severity of depression, dissociative features, and conflict in relationships decreased significantly (Table S6 [Supplement]). Unexpectedly, the association of DR-S with conflict in relationships increased again slightly between levels 7 and 8. All other contrasts suggested a cutoff between levels <6 and ≥6. Indeed, the Research Operating Curve (ROC) at levels 5 and 6 (Figure S2 [Supplement]) confirmed that only the models for depression, dissociation, and conflict in relationships were significant after Bonferroni correction at level 5, with AUCs (sensitivity and specificity) of, respectively, 69% (p = .002), 71% (p = .001), and 68% (p = .003) probability of correct positive prediction, while the predictive power at level 6 decreased to 41% (p = .012), 43% (p = .054), and 46%, (p = .22), respectively. Hence, from level 6 onwards, the predictive power of DR-S for symptoms and problematic relational functioning disappeared. Differences between the effects of DR-S on groups with DR-S <6 and DR-S ≥6 were calculated with the Mann–Whitney U test (Table 2). High- and low-level DR-S and DR-M groups differed significantly in their severity of depression. These two groups also differed significantly in their dissociation symptoms and conflict in relationships after Bonferroni correction. The effect sizes were small (<.14) however; the grouping explained 2.9–3.6% of the variance in ranks, but the power was diminished by the difference in sample size because of the restriction of range at the impaired end.

	Mann–Whitney *U*	*Z*	*p*	*η2= Z2/N*
DR-S				
DID severity	2562.0	–3.458	.001**	.036
DES total	2993.0	–3.199	.001**	.031
DEQ				
DEP	3577.0	–1.348	.178	.006
S-C	2943.0	–2.661	.008	.022
QRI				
support	2859.5	–2.845	.004*	.025
conflict	2974.0	–2.601	.009	.021
depth	3902.5	–.677	.498	.001
DR-M				
DID severity	6539.5	–3.104	.002**	.029
DES total	8521.0	–2.010	.044	.012
DEQ				

DEP	7831.0	–1.227	.220	.007
S-C	7585.0	–1.583	.113	.007
QRI				
support	8057.0	–.904	.366	.002
conflict	8115.5	–.817	.414	.002
depth	7655.5	–1.488	.137	.007
DR-F				
DID severity	7278.5	–1.430	.153	.006
DES total	8371.0	–1.798	.072	.010
DEQ				
DEP	8018.0	–.303	.762	.000
S-C	7960.0	–.391	.696	.000
QRI				
support	7580.0	–.972	.331	.003
conflict	7806.0	–.626	.531	.001
depth	7604.5	–.935	.350	.003

Table 2 Mann-Whitney U tests of categorical differences between DRS above and below the cut-off level of 6.
Note. DRS = Differentiation-Relatedness Scale; DR-S = DRS in descriptions of self; DR-M = DRS in descriptions of mother; DR-F = DRS in descriptions of father; DID = Diagnostic Inventory for Depression; DES = Dissociative Experiences Scale; DEQ = Depressive Experiences Questionnaire; QRI = Quality of Relationships Inventory; DEP = dependency; S-C = Self-Criticism. * p < .05; **p < .01; ***p < .001 after Bonferroni correction. Effect sizes: η^2 < .01 = no effect, .01 ≥ η^2 < .06 = small effect, .06 ≥ η^2 < .14 = intermediate effect, η^2 ≥ .14 = large effect (Lenhard & Lenhard, 2016)

2.2.5.3 *Unidimensionality of DRS*

The inter-scale Kendall's correlations between DR-S, DR-M, and DR-F (Table S3 [Supplement]) were all large but not perfect, indicating that they were related, but also measuring differing features. The concordance of the ranks of DR-S with DR-M and DR-F was low (Kendall's W DR-M = .063, p < .001 and DR-F = .084, p < .001) but comparable for DR-M and DR-F, and not significantly different for DR-M and DR-F (z = –.263, p = .396). Hence, DR-S varied in the same direction as DR-M and DR-F, but the distribution of the ranks was not the same. Associations of dependency and self-criticism with DR-S were not significant, and the effect size indicated no effect. However, self-criticism was related significantly to high versus low DR-S (τ = .146, p = .008), but dependency (τ = .001, p = .988) was not, and nor was the interaction of dependency with self-criticism (τ = .033, p = .461).

2.2.6 Discussion

This study aimed to further validate the DRS in a sample of nonclinical young adults. We first investigated relationships of DRS with demographic features and differences in distributions between the two genders. Secondly, we studied linear and potential categorical relationships of the associations of DRS with self-report measures of depressive and dissociative features of personality psychopathology and of interpersonal functioning. Finally, we investigated the unidimensionality of the DRS by studying the possible

redundancy of the parallel DRS subscales of DR-S and DR-M and DR-F. We also investigated whether a DRS score reflected general LPF as the outcome of the dialectics between differentiation and relatedness, or whether impairments in the constituting latent dimensions, dependency, and self-criticism had unique direct contributions to lower DRS.

First, the distributions of DRS did not differ between the genders. There were no relationships with the demographic features age and gender, and only a small correlation of DR-S with educational level. This positive correlation may be consistent with findings that level of education is related to mentalizing abilities (Pino & Mazza, 2016), and thus to the capacity to represent mental states.

Secondly, results showed only nonlinear relationships between the DRS and indices of interpersonal and intrapersonal functioning. There were only few and small associations between levels of DRS and indices of interpersonal and intrapersonal functioning in the current sample. However, young adults scoring below 6 on the DR-S seemed to be more vulnerable to psychopathology than those scoring above this cut-off were. Hence, the investigation of linear and categorical relationships in this study suggested that the assumed theoretical level of 6 might be an adequate cut-off to differentiate adaptive from maladaptive functioning. Furthermore, although DR scores have been shown to be linearly associated with clinical features in patients with PDs (e.g., Lowyck et al., 2013), in this sample of nonclinical young adults, this was not the case as, from level 6 upward, higher levels on the DR-S seem to be relatively independent of indices of psychopathology.

The finding that, at least in community samples, relationships between DRS and indices of psychopathology and interpersonal functioning may not be merely linear reveals an essential limitation of the DRS. Most theories of personality hypothesize that vulnerability to psychopathology is dimensionally distributed (e.g., Berghuis, Kamphuis, & Verheul, 2014). It also follows that in samples with a low proportion of individuals with lower levels of personality organization, studies using the DRS and dimensional analyses may fail to detect underlying vulnerability in subsamples of individuals within that larger sample.

Finally, the study suggested that DRS is not unidimensional because the DR-S, DR-M, and DR-F subscales correlated only moderately and showed marked differences in associations with psychopathology. Neither DR-M nor DR-F was predictive for DR-S, but associations of DR-M and DR-F with DR-S did not differ. They varied in the same direction as DR-S, but the distribution of the ranks of DR-S was significantly different from both subscales. There were substantial differences in the associations between the different types of representations and indices of intrapersonal and interpersonal functioning investigated. Only DR-S and DR-M differentiated those with high versus low levels of dissociative features, self-criticism, and supportive relationships. Hence, the representation of the father appeared to be less related to indices of functioning in this sample. At least in Western societies, over the last decades, there has been a shift in the role of mothers and fathers in child development

(Luyten & Blatt, 2013), with more balance between the parents in terms of the extent to which they are involved in parenting and child development. Therefore, it is surprising that in the current study, the representation of fathers was not associated with personality functioning. Hence, particularly in young adulthood, representational structures related to mothers as primary caregivers may be more important than those related to fathers. Further research in this context is needed.

Furthermore, the LPF construct assessed by the DRS seems not to be unidimensional. Latent LPF dimensions of (maladaptive) relatedness and differentiation had different and independent contributions to impaired DR-S. DR-S was not associated with the integration of both, but reflected only maladaptive levels of differentiation, that is, self-criticism. This finding may be due to achievement issues playing a central role in this sample of university students (Tosevski, Milovancevic, & Gaijic, 2010). Students whose developmental history is marked by an absence of warmth and understanding in the relationship with their mother may be particularly vulnerable to achievement-related distress in the transition to young adulthood (Pagura, Cox, Sareen, & Murray, 2006). Studies have suggested that adults who have been neglected may develop excessive self-criticism and achievement strivings to compensate for feelings of inferiority and conflict related to attachment problems (Shahar, 2015), putting them at increased risk of depression during a life stage when there is an intense focus on achievement. One study showed that the impact of the interaction between self-criticism and achievement stress was more than 20 times as strong at age 25 as in late adulthood, while, in contrast, vulnerability associated with dependency peaked later in life (Mazure & Maciejewski, 2003). An alternative explanation is that the lack of relationship between DR-S and dependency is due to the outweighing protective and maladaptive effects of dependency, as dependency has been shown to have both elements of risk but also protection (Abuin & de Rivera, 2015).

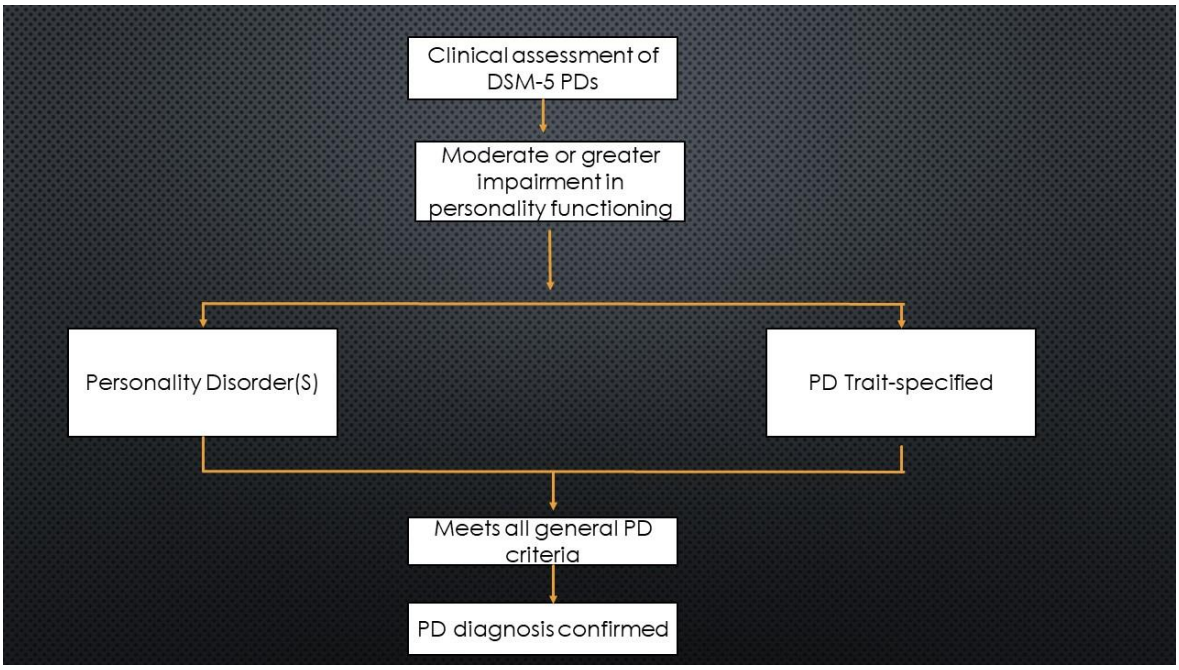
From a clinical perspective, this study further emphasizes the value of a focus on impaired representation of the self and others. Severe impairments in the representation of the self in particular appear to be associated with feelings of depression, despair and dissociative features, even in a community sample of young adults. Furthermore, open descriptions of self and parents may be easily integrated in routine screening and diagnostic procedures as a reliable and valid assessment of the LPF.

One limitation of the current study is its cross-sectional nature. Longitudinal studies are needed in this context to disentangle possible reciprocal relationships between levels of DRS and psychopathology. Second, the study focused on university students. Although university students may on average show higher functioning than their peers, studies have revealed high levels of psychopathology among university students (e.g., Ibrahim, Kelly, Adams, & Glazebrook, 2013). Hence, the absence of linear relationships between the DRS and intrapersonal and interpersonal functioning can most likely not be attributed to the nature of the sample, given the considerable range in scores on the DR-S and the other

measures. About 10% of participants in this sample showed impaired LPF (DR-S scores <6), and only 20% showed higher levels of LPF (DR-S scores >6). Nonparametric analyses showed a pattern with no continuous effects of DR-S, but significant categorical differences.

Despite these limitations, this study suggests that although the DRS may be used to detect emerging personality pathology in young adults, relationships between the DR-S and LPF are most probably nonlinear in non-clinical samples. Longitudinal research is needed to substantiate these conclusions. Further, the variance of DR-S is mostly independent of the differences in representations of the parents. This finding could be surprising from an object-relations perspective but is consistent with the limited enduring effects of early attachment experiences across the lifespan in normative samples (Fraley, 2002; Fearon, Shmueli-Goetz, Viding, Fonagy, & Plomin, 2014).

DSM-5 ASSESSMENTS OF THE LEVEL OF PERSONALITY FUNCTIONING: INTRAPERSONAL AND INTERPERSONAL FUNCTIONING



10

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[10] Note. Oldham, J.M. (2018, November 26). Workshop Alternative Model of Personality Disorders. In Congress Zorgstandaard Personality Disorders, De Viersprong Academy, Haarlem, The Netherlands

2.3 DSM-5 Assessments of the Level of Personality Functioning: Intrapersonal and Interpersonal Functioning

2.3.1 Abstract

Background: In DSM-5, Section III (American Psychiatric Association, 2013), the Level of Personality Functioning (LPF) was proposed as a severity index of personality disorders (PDs), but as it reflects both trait-like (availability) and state-like (accessibility) features, of which, moreover, the relationship with the experience of patients is unclear, we critically examined LPF in patients with general psychopathology.

Methods: This study compared the validity of the direct Inventory of Personality Organization (IPO), and the indirect Differentiation-Relatedness Scale (DRS) LPF-measure, in relation to measures of intrapersonal and interpersonal functioning. The sample consisted of 70 inpatients with general psychopathology and no primary PDs. Associations of both measures with DSM-PDs were examined, with and without controlling for clinical distress.

Results: The IPO was significantly related to age and clinical distress. When controlling for clinical distress, the IPO was still associated with cluster A (bizarre) and B (erratic) PD features, high levels of self-criticism, conflict in relationships and low levels of adaptive coping strategies. The DRS was only related to the schizotypal PD.

Limitations: In general psychopathology patients, both the IPO and the DRS, appear to have limitations in measuring LPF. The IPO seems to be prone to state effects, although correlations with PDs remained significant when controlling for clinical distress. Conclusion: The DRS seemed to be more independent from clinical distress but was unexpectedly unrelated to features of personality pathology. The DRS reflects availability, while IPO also reflects different degrees of accessibility of LPF in PDs.

Keywords: personality disorders, severity, interpersonal functioning, coping, Level of Personality Functioning

To overcome problems of categorical classification of personality disorders (PDs) such as lack of therapeutic specificity, a dimensional Alternative Model of Personality Disorders (AMPD) was proposed in DSM-5, Section III (Diagnostic and Statistical Manual of Mental Disorders, 5th edition; American Psychiatric Association, 2013). It consisted of a hybrid system of the Level of Personality Functioning (LPF, criterion A), indicating presence and severity of PDs with impairments in mental representations of self and interpersonal functioning, and the style of PDs with maladaptive traits (criterion B). The proposal of the AMPD suggests the independence of criteria A and B, but the debate about the relationship between the two dimensions remains unresolved (Widiger et al., 2018). Evidence is

accumulating that impairments in mental representations of the self in relation to that of others as developed in object relations hamper personality integration and thus underlie personality pathology (Lowyck, Luyten, Verhaest, Vandeneede, & Vermote, 2013). However, it is not clear yet whether LPF could be implied by the maladaptive traits, form a separate trait or could be a general factor of psychopathology underlying both traits and symptoms (Widiger et al., 2018). As, however, the state-trait model of Zuroff, Blatt, Sanislow, Bondi, & Pilkonis (1999) suggested that the availability (content and structure) of mental representations is quite stable but that the accessibility may fluctuate in temporary (mood) states and context, we investigated the impact of clinical distress on a direct and indirect LPF measure. Because a range of newer instruments is still being validated, we compared an already extensively investigated self-report measure to a performance-based measure of LPF (Huprich, Auerbach, Porcerelli, & Bupp, 2016), to refine the construct as called for by the Hierarchical Taxonomy Of Psychopathology - consortium (Widiger et al., 2018). The Inventory of Personality Organization (IPO; Lenzenweger, Clarkin, Kernberg, & Foelsch, 2001) as the direct measure, reveals a conscious representation of LPF, while the Differentiation and Relatedness Scale (DRS; Diamond et al., 2014) as the indirect measure, reveals the object-related representation of LPF.

2.3.2 Differentiation Relatedness Scale and Inventory of Personality Organization

2.3.2.1 *The Object Relations Inventory-Differentiation and Relatedness Scale as an indirect measure of LPF.*

Diamond and Blatt's DRS (Diamond et al., 2014) is a 10-level ordinal subscale of the Object Relations Inventory (ORI) (inter-rater reliability of ORI is .70, p=0.0005, Vermote, 2005). It assesses the LPF as representational levels for mother, father, (therapist), peer, and self, resulting from dialectics between relatedness and self-definition. Blatt's theory and assessment have influenced the proposed two-dimensional LPF-Scale in DSM-5, Section III. The DRS measures the transition from impairments in basic differentiation between self and others, with lack (level 1) or confusion (level 2) of boundaries (e.g. flood of details with a sense of confusion), over attempts to establish and maintain object and self-constancy by the use of mirroring (level 3) idealization and denigration (level 4) or oscillation between both (level 5) (e.g. extreme one-sided description), to differentiated and integrated concepts of self and others (level 6), with increasing tolerance for ambiguities (level 7; e.g. integration of disparate aspects), and the capacity for empathic (level 8), reciprocal (level 9) relationships with a mutual reflective construction of meaning (level 10; e.g. understanding the perspective of the other) (Diamond et al., 2014). Reliability of the DRS is good, DRS ICC = .83 (Shrout & Fleisch) (Diamond et al., 2014), and concurrent and discriminant validity is solid (Calamaras, Reviere, Gallagher, & Kaslow, 2016). Because Blatt's theory is rooted in object-relational thinking and attachment theory, it is assumed that the levels of representation of significant others might differ, depending on differing dyads with the self.

2.3.3 The Inventory of Personality Organization as a direct measure of LPF.

The IPO is a self-report measure of LPF, assessing features seen as typical key dimensions in LPF (Widiger et al., 2018, p.3). The IPO derives from the theory of Kernberg, stating that the quality of object relations results in a continuum of ego functioning from normal to severe, with three organization levels. Combinations of impairments in three key subscales of IPO determine the levels. These scales measure 1) identity confusion (ID, 21 items) as poor understanding of self and others (e.g. 'I pick up hobbies and interests and then drop them'), 2) the use of primitive defenses (PrD, 16 items) as splitting and projection (e.g. 'I feel I don't get what I want'), and 3) problems with reality testing (RT, 20 items) as maintaining empathy with ordinary social criteria of reality (e.g. 'I feel that my wishes or thoughts will come true as if by magic'). While the neurotic level may show avoiding defenses against inner conflicts, the borderline level shows impairments in ID and PrD, and the psychotic level shows problems in RT moreover. Studies have revealed excellent internal consistency and test-retest reliability (r = .72-.83, Lenzenweger et al., 2001) and supported convergent, concurrent and discriminant validity (e.g., Lenzenweger et al., 2001; Lowyck et al., 2013; Smits, Vermote, Claes, & Vertommen, 2009).

While existing research has provided evidence for the reliability and validity of both the DRS and the IPO, the only study that directly compared the relationship between both instruments and features of clinical functioning (Lowyck et al.,2013) found that correlations between IPO and DRS were only small to medium and therefore initiated the measurement of complementary personality aspects. DRS predicted depression severity, clinical symptoms, and self-harm, IPO predicted clinical symptoms, interpersonal problems, and self-harm. As, however, this study included a sample of disordered personality patients, it remains unclear to what extent these findings generalize to patients with general psychopathology and only secondary personality pathology and to what extent these associations reflect clinical distress, PD traits or/and impaired personality functioning.

Therefore, in this study, we investigated associations of IPO and DRS with features of possible cognitive, intrapersonal and interpersonal dysfunction in a sample of patients with general psychopathology, with and without controlling for clinical distress. In this sample, PDs were less severe, and chronic psychosis was excluded, but functional impairment and subjective distress, two prerequisites for diagnosis of PD in DSM-5, were present.

We expected more severe personality pathology traits and PDs, more self-criticism and dependency, and more maladaptive interpersonal functioning and coping with higher IPO scores and lower DRS scores. Indeed, impairments of LPF can be understood as impaired object relations, manifested in impaired identity, self-directedness, interpersonal empathy, and intimacy (see AMPD). Following previous findings with IPO and DRS, we did not expect relationships with age, gender, or educational level. In keeping with the nature of PD, we hypothesized no influence of clinical distress in the relationship between PDs and DRS and IPO.

2.3.4 Methods

2.3.4.1 *Participants*

Seventy inpatients (Caucasian, 35 males) aged 18 to 60 ($\bar{x}$ = 36.6, SD 11.9) were included, consecutively admitted for specialized diagnosis and brief psychotherapy. The only inclusion criterion was general psychopathology (Supplement S1), but patients with manifest psychosis, cognitive deterioration, were selected out before admission to the ward. The mean level of education was higher secondary education (level 3, from 1= primary education to 6 = university).

2.3.4.2 *Measurements*

a. Clinical Distress

The Symptom Checklist-90 (SCL-90; Arindell & Ettema, 1986) is a 90 items self-descriptive scale with eight subscales and a total scale. Patients rate each item on a 5-point Likert scale. The subscales are summed up.

b. Psychiatric Symptoms

Beck Depression Inventory (BDI; Van der Does, 2002) is a 21-item self-descriptive 4-point (0-3) scale multiple-choice inventory with three subscales. Total severity score is the sum (max. 63) and can be minimal (0-13), light (14-19), moderate (20-28) or severe (29-63).

Dissociation Questionnaire (DIS-Q; Vanderlinden, Van Dyck, Vertommen, Vandereycken, & Verkes, 1993) is a 63-item self-descriptive questionnaire with a 5-point Likert scale for degrees of dissociative experiences with four subscales. The total score is summed up.

c. Personality pathology

Descriptive DSM - IV -TR

ADP-IV (Schotte & De Doncker, 1996) consists of 94 trait-distress items, each criterion of DSM-IV (American Psychiatric Association, 1994) scoring the typicality of the trait on a 7-point Likert scale. If score , > 5, then distress is scored on a 3-point Likert scale. Trait and distress scores are summed up for every dimension, and a categorical score is calculated following a DSM-IV-TR algorithm with combinations of cut-offs for traits and distress. After that, the diagnosis of clusters A, B, and C is calculated.

Criterion A DSM-5, Section III

The *Depressive Experience Questionnaire* (DEQ; Luyten, Corveleyn, & Blatt, 1997) is a 66-item self-descriptive questionnaire, with a 7-point Likert scale with three factors; self-criticism and dependency were used as dimensions of LPF. Scores were calculated using factor scores and loadings of the original DEQ (same psychometric characteristics).

The DRS (Blatt, Wein, Chevron, & Quinlan, 1979) is a 10-point ordinal clinician rating scale of LPF. It is indirect because the aim is obscure for the subject. The performance-based LPF is scored on the ORI, a semi-structured interview in which subjects are asked to describe important others (i.e., mother (DR-M), father (DR-F), peers (DR-P) and self (DR-

S)) as detailed as possible. Then, DRS is used to assess the ability to understand both oneself and one's interpersonal matrix. For a full description of the use of DRS and ORI, see Diamond et al. (2014). The same levels can be clinically rated (after training for reliability) for different significant others like the mother (DR-M), the father (DR-F), the self (DR-S), a peer (DR-P) or a therapist (DR-T).
The IPO is a self-report instrument and hence a direct measure of LPF with 136 items on a 5-point Likert scale and 9 subscales of which ID, PrD and RT are keys to determine the organization level by different combinations (see introduction).

d. Functional outcome

Progressive Matrices (PM; Raven, 2006) estimates IQ by 60 multiple-choice items in 5 sets of visual pattern detection with increasing difficulty. The rough score is converted into a percentile according to a set of criteria such as age.

Quality of Relationships Inventory (QRI; Pierce, Sarason, & Sarason, 1991) is a self-report scale with 25 items scored on a 4-point Likert scale with three calculated subscales: support, conflict, and depth.

Utrechtse Coping Lijst (UCL; Schreurs & van de Willige, 1988) is a self-report scale with 47 items scoring on a 5-point scale the frequency of using a specific coping (seven subscales).

2.3.4.3 *Procedures*

The ethics committee of NPO Emmaus, Mechelen, and the University of Antwerp, Belgium, approved this study. The assessment was part of the routine treatment, except for the ORI. Patients were informed about the study, filled in coordinates and demographical data, and provided written informed consent. Then, in the first two weeks of admission, they got a psychiatric diagnosis (S1), an interview with the ORI, and they digitally filled in the clinical questionnaires.

2.3.4.4 *Statistical analysis*

Statistical analyses were performed using SPSS 22.00 (IBM corp., 2013). Pearson's correlations between DRS levels rated on ORI descriptions of self, mother, father and peer and IPO-ID, IPO-PD and IPO-RT were calculated (*p< .05, **p<.01). Next, correlations were calculated for DR-S, DR-M, DR-F, DR-P and IPO- ID, PD and RT as aspects of LPF measures and clinical distress and symptoms (SCL-90, BDI, DIS-Q), differentiated criterion A dimensions of AMPD (DEQ), DSM-IV-TR PDs (ADP-IV) and functional relational (QRI) and coping (UCL) measures. Partial correlations were calculated to control for clinical distress covarying for SCL-90. Comparison of correlations was tested with Fisher z or Hoerger Z-scores for dependent correlations. Comparison of categorical groups (gender) was calculated for IPO-ID, IPO-PD, and IPO-RT with ANOVA and Bonferroni correction for multiple comparisons.

2.3.5 Results

2.3.5.1 *Convergent validity of DRS and IPO*

Results indicated that DRS and IPO do not correlate (DR-S: r IPO-ID = .11, r IPO-PD = .12, r IPO-RT = .09, p>.05) (S2). But, while subscales of IPO correlated comparably high (r IPO-ID/RT = .54**, r IPO-PD/RT = .58**, r IPO-ID/PD = .66**), correlations between DRS representations diverged in very small correlations with DR-P (r DR-F = .27*), moderate correlations with DR-S (all = .34**) and a high correlation between DR-M and DR-F (r = .54**).

2.3.5.2 *Associations of DRS and IPO with stable and fluctuating variables*

Neither DRS nor IPO correlated with gender, level of education, or IQ (Table S3), i.e. stable factors in personality development. Temporary and dynamic measures such as age (r = .28-.31*) (Table S3), clinical distress (r SCL-90 = .57-.61**), symptoms of depression (r BDI = .436-.558**) and especially the more fluctuating symptoms of dissociation (r DISQ = .717-.786**) all correlated with IPO (Table S4).

2.3.5.3 *Controlling for clinical distress in associations of DRS and IPO with functional measures*

Therefore, we re-ran correlations with traits of PD, coping, and relational functioning, controlling for clinical distress (see Table 1). While DRS was not related to coping measures and relational functioning (Table 2), all IPO measures were related to self-criticism and dependency (Table 3), to most coping measures, and conflict in relationships (Table 2). Although there was a significant impact of clinical distress for self-criticism and dependency, only correlations between IPO and self-criticism remained after controlling for clinical distress (r IPO-ID = .528**, r IPO-PD = .452**, r IPO-RT = .215*). Hence, self-criticism appeared to be a structural deficit in impaired IPO (LPF), while dependency seemed to be explainable by contextual, interpersonal, and distress features.

	DRS				**IPO**				
ADP-IV	**DR-M**	**DR-F**	**DR-P**	**DR-S**	**IPO-PD**	**IPO-ID**	**IPO-RT**	*M*	*SD*
	N = 67	**N = 67**	**N = 47**	**N = 65**	**N = 64**	**N = 64**	**N = 64**		
CLUSA	-.048	-.209	-.332*	-.309*	*.778***	*.749***	*.714**	67.26	21.97
control	.006	-.123	-.242	-.242	*.618***	*.557***	*.505***		
CLUSB	.072	-.056	-.246	-.234	.794**	.760**	.735**	97.85	35.65
control	.147	.141	-.168	-.108	.693**	.639**	.600**		
CLUSC	-.008	-.113	-.223	-.184	*.624***	*.648***	*.542***	79.89	24.43
control	.136	.156	-.104	-.079	*.314***	*.337***	*.159*		
PARD	-.018	-.065	-.128	-.200	.747**	*.696***	*.702***	21.74	9.54
control	.067	.094	-.121	-.073	.590**	*.496***	*.514***		
SZD	-.181	*-.256**	-.341*	.181	.399**	.431**	.280**	19.93	7.71
control	-.156	*-.289*	-.285	-.172	.213	.253*	.040		
STD	..036	-.219	-.340*	-.375**	*.775***	*.734***	*.755***	28.59	10.62
control	..084	-.089	-.252	-.302*	*.622*	*.543***	*.587***		
ASD	-.002	.036	-.102	-.092	.654**	.318**	.538	17.70	9.29
control	-.029	-.06	-.053	-.01	.613**	.581**	.462**		
BLD	.012	-.063	-.318*	-.279*	*.764***	*.759***	*.696***	39.67	13.78
control	.110	.124	-.227	-.151	*.604***	*.589***	*.488***		
HISD	.178	-.030	-.326*	-.210	.690**	.645**	.672**	23.28	9.90
control	.236	.215	-.254	-.174	.559**	.487**	.532**		
NARD	.081	-.052	-.010	-.160	.609**	.573**	.614**	21.05	9.36
control	.192	.192	-.005	-.015	.531**	.480**	.538**		
AVD	.002	-.088	-.025	-.186	.564**	.527**	*.417***	26.21	10.05
control	.117	.102	.057	-.086	.354**	.286**	*.125*		
DEPD	-.001	-.089	-.243	-.102	*.553***	*.617***	*.475***	27.41	10.56
control	0.103	.178	-.125	-.038	*.194*	*.285**	*.047*		

	DRS				**IPO**				
	DR-M	**DR-F**	**DR-P**	**DR-S**	**IPO-PD**	**IPO-ID**	**IPO-RT**	*M*	*SD*
	N = 67	**N = 67**	**N = 47**	**N = 65**	**N = 64**	**N = 64**	**N = 64**		
ADP-IV									
OCD	-.026	-.119	-.303*	-.179	*.498***	*.538***	*.529***	26.41	8.56
control	.100	.088	-.210	-.060	*.173*	*.219*	*.220*		
DED	-.013	-.149	-.182	-.291*	*.536***	*.560***	*.421***	27.13	10.53
control	.171	.105	-.058	-.159	*.230*	*.251**	*.033*		
PAD	.098	-.049	-.088	-.179	.686**	.673**	.636**	19.39	7.34
control	.279	.155	.013	.055	.519**	.491**	.438**		
DEQ									
DEP	.089	.062	-.235	.057	*.43***	*.464***	*.404***	.096	0.966
control	.159	.189	-.125	.176	*.115*	*.163**	*.04*		
SC	.013	-.14	-.053	-.165	*.687***	*.7327***	*.572***	.380	1.033
control	.087	-.03	.141	-.072	*.452***	*.528***	*.215**		

Table 1 Correlations of the DRS and the IPO with personality disorders as measured with ADP-IV, according to DSM-IV-TR criteria, with interpersonal functioning, as measured with the QRI, and with Coping as measured with the UCL. Also, partial correlations are added, controlled for clinical distress (SCL-90 total score) and significant differences between correlations and partial correlations, calculated with Fisher z, in italics.

CLUS A = cluster A PD's, CLUS B = cluster B PD's, CLUS C = cluster C PD's, PARD = paranoid PD, SZD = schizoid PD, STP = schizotypal PD, ASD = antisocial PD, BLD = borderline PD, HISD = histrionic PD, NARD = narcissistic PD, AVD = avoidant PD, DEPD = dependent PD, OCD = obsessive-compulsive PD, DED = depressive PD, PAD = passive-aggressive PD, DEQ = Depressive Experience Questionnaire, DEP = DEQ dependency dimension, SC = DEQ self-criticism dimension.

	DRS				IPO				
	DR-M	**DR-F**	**DR-P**	**DR-S**	**IPO-ID**	**IPO-PD**	**IPO-RT**	**MEAN**	**SD**
QRI (N)									
SUPPORT	.144	.115	-.018	.100	.017	-.005	.076	2.811	.737
DEPTH	.082	.032	.054	.204	.100	.093	.143*	2.984	.680
CONFLICT	-.153	-.239	.122	-.090	.291**	.311**	.205**	2.192	.696
UCL (N)									
ACT	-.045	-.067	.094	.026	-.379**	-.292**	-.157*	2.111	.624
PALL	-.064	-.087	-.001	.137	.276**	.253**	.331**	2.328	.547
AVOID	.023	-.119	-.049	-.021	*.452***	*.366***	*.376***	2.289	.525
SOCIAL	.126	.271*	.059	.238	-.180*	-.077	-.069	2.002	.711
PASS	-.082	-.132	-.221	-.189	*.686***	*.61***	*.542***	2.506	.583
EXPR	-.056	.066	-.073	.001	.278**	.357**	.336**	2.050	.675
COMF	.046	.093	.049	.048	-.189*	-.151*	.038	2.281	.583
TOTAL	-.022	-.022	-.025	.119	.211**	.239**	.342**	2.243	.284

Table 2 Correlations of DRS and IPO with interpersonal functioning, as measured with the QRI, and with Coping as measured with the UCL. Significant differences of correlations and partial correlations between IPO and coping measures are marked with italics

Subscales of UCL (Utrecht Coping List): ACT = active problem solving, PALL = palliative coping, AVOID = avoidant coping, SOCIAL = seeking social support, PASS = passive reaction, EXPR = expression of emotions, COMF = comforting thoughts, TOTAL = UCL total score

2.3.5.4 *Correlations of DRS and IPO with PDs controlling for clinical distress: three types*

Correlations of DRS (DR-S and DR-P) with PDs were surprisingly limited to cluster A, the schizoid, schizotypal, borderline, and histrionic PD and, after controlling for clinical distress, only DR-S was related with exclusively the schizotypal PD. This PD has been questioned as

a PD and would rather suggest a genetic vulnerability like schizophrenia (Lenzenweger, 2015). IPO correlated with all PDs, but after controlling for clinical distress, three types appeared. First, correlations of the IPO with cluster C seemed to be merely state-dependent, while second, correlations with cluster A or B remained strong, even if they too showed important impact of clinical distress. Third, PDs typically associated with extreme internalizing (schizoid and avoidant) and externalizing (antisocial, histrionic, passive-aggressive, and narcissistic) traits, seemed to be independent of clinical distress. Thus, descriptive PDs showed three types, according to susceptibility to distress.

2.3.6 Discussion

2.3.6.1 *Availability and accessibility of LPF*

In summary, in this sample, the DRS appeared to be associated with psychotic vulnerability and was not associated with clinical measures of PD-severity (distress, symptoms, traits, or functioning in relationships or coping). DRS measured the availability of personality functioning, the structural vulnerability that gives rise to disturbances in the self (Zuroff, Sadikaj, Kelly, & Leybman, 2015). IPO, in turn, was state dependent and was associated with interpersonal functioning, clinical distress, coping, functioning of self, and with all PDs. However, comparisons of correlations between descriptive PDs and IPO (LPF) before and after controlling for clinical distress differentiated three types of PDs by the impact of clinical distress.

2.3.6.2 *DRS and IPO complement in differentiating identity integration from clinical distress*

In all, the present research reveals an impact of clinical distress on PDs. But the impact differs depending on the type of PD. The three types revealed in the comparison of the results for IPO and DRS in the present sample indicate that DRS is only useful to detect psychotic PDs (availability), IPO is complementary (availability and accessibility). IPO shows in high LPF (cluster C) a relationship with PDs determined by clinical distress, in medium LPF (with extreme internalizing or externalizing traits), the presence of clinical distress shows no impact, but in low LPF (cluster A and B), there is a clear impact of both hampered identity integration and clinical distress. Even if the present study is limited in scope due to the specificity of the sample, which is limited to general psychopathology patients, it opens a perspective for reliable measurement of PDs, independent of clinical distress.

BIFACTOR MODELING SUGGESTS DIFFERENT FUNCTIONS FOR GENERAL AND SPECIFIC FACTORS IN PERSONALITY DISORDERS

11

Beheydt, L., Jansen, B., De grave, C., & Luyten, P. (In Review)

[11] *Note. (2021). Vulnerability or adaptability symbol. Turned wooden cubes and changed words 'vulnerability' to 'adaptability'. Grey background, copy space. Business, vulnerability or adaptability concept (graphic). Retrieved june, 8, 2022 from https://assets.adobe.com/libraries/urn:aaid:sc:EU:931bf7c9-e698-452e-81a8-a328ac2cc6e1/bd920e09-52d1-4d8c-a110-cb22d1b86bc1*

2.4 Bifactor modeling suggests different functions of general and specific factors in personality disorders

2.4.1 Abstract

Background: The factor structure underlying DSM-IV-TR (American Psychiatric Association, 2000) personality disorders (PDs) was investigated with confirmatory factor analysis (CFA) in 241 affective-spectrum patients, assessed for all DSM-IV-TR PDs, and for intrapersonal and interpersonal functioning. Methods: The model fits and concomitant factor loadings were evaluated for a one-factor, correlated-factors, a hierarchical, and a nested bifactor model. Then, regression models of the best-fitting model were used to predict descriptive PDs. Finally, correlations between specific factors and variables of intrapersonal and interpersonal functioning were calculated, controlling for the p-factor.
Results: The bifactor PD model, with specific loading on clusters A (bizarre), B (erratic), and C (anxious) traits, and a general p-factor, was the only acceptable model with a good fit and good composite reliability for the p-factor. Limitations: Important limitations were the cross-sectional design of the study and the limited share of psychotic patients in the sample. Conclusion: The bifactor model enabled independent prediction of descriptive PDs by the p-factor and the traits and differentiated the borderline from narcissistic PDs.

2.4.2 Introduction

In the past decade, a general psychopathology factor (the p-factor), common to all psychiatric disorders, has been proposed to underlie the structure of mental disorders (e.g., Caspi et al., 2014; Kotov et al., 2017; Lahey et al., 2012; Patalay et al., 2015), similar to the general factor in intelligence. A recent review of research (Smith et al., 2020) suggests that the p-factor represents an index of functional impairment with the potential to inform decisions regarding the duration and intensity of care required for patients with psychiatric disorders.

The p-factor was tested first with a bifactor model, to differentiate between the impact of the general p-factor and that of specific trait factors (Caspi et al., 2014) in psychiatric disorders. Criticism of the use of the bifactor model subsequently led to the promotion of other types of models, such as hierarchical and network models (e.g., van Bork et al., 2017; Widiger & Oltmans, 2017). Moreover, criticism was formulated concerning the meaning of the p construct. In what follows, we review evidence concerning the p-factor and current limitations of research in this area. Next, we outline the aims of the current study.

2.4.2.1 *Evidence for the p-factor*

The considerable association between internalizing and externalizing symptoms, with correlations ranging between r = .51 and r = .72 (e.g., Conway et al., 2019), has led to the view that general and specific factors explain the structure underlying psychopathology.

From a factor analytic perspective, a bifactor model was suggested as the appropriate model to study the unique contribution of general and specific features of psychopathology (Reise, Moore, & Haviland, 2010). In addition, the bifactor model would enable the combination of discrete aspects of the latent structure with continuity of dimensions and common traits for normal and abnormal personality in one model (Eaton, Krueger, Docherty, & Sponheim, 2014). The theoretical benefit of a bifactor model would thus be the possibility to integrate discrete and continuous views on personality disorders (PDs). The assumption that a general p-factor underlies the structure of psychopathology has been relatively well established in children (e.g., Sallis et al., 2019; Waldman et al., 2016), adolescents (Castellanos-Ryan et al., 2016; Laceulle, Volleberg, & Ormel, 2015; Patalay et al., 2015) and adults (Caspi et al., 2014; Lahey et al., 2012 ;). Moreover, these studies suggest three lower order factors, that is, an internalizing factor associated with distress and fear (Greene & Eaton, 2017; Lahey et al., 2012; Martel et al., 2017), an externalizing factor associated with substance use, conduct disorder, and antisocial behavior, and a third factor representing psychotic or thought disorder (Caspi et al., 2014; Stochl et al., 2015). This latter factor typically shows the highest loading on the p-factor (e.g., Caspi et al., 2014).

2.4.2.2 *Criticism of the bifactor model*

Three types of criticism have been formulated regarding the bifactor model. First, from a statistical perspective, the preference of a bifactor model over a hierarchical factor model (e.g., Widiger & Oltmans, 2017) based on comparison of model fit has been criticized, because model fit in structural equation modeling is an assessment of the similarity of the observed variance–covariance matrix with that implied by the model. The bifactor and hierarchical models are almost equivalent, with very similar variance–covariance matrices in spite of their widely differing theoretical bases. Although they can be statistically distinguished in theory, they are both highly subject to sampling variability (van Bork et al., 2017), which explains the resistance to using model fit as a basic argument in favor of a specific model. However, it may be argued that one step to evaluate whether the bifactor model makes sense as a model is to quantify the absolute strength of covariation of individual PDs and the general factor, as well as relative to the variance shared by individual PDs that belong to a common specific factor (i.e., cluster A, B, and C). The covariance of the general factor and the individual PDs is a necessary condition for a genuine bifactor model, and is not quantified by model fit indices, which could be good without fulfilling this condition (Murray et al., 2019, p. 2).

Second, from a theoretical perspective, the p-factor has been criticized because it lacks a theoretical foundation. Specific PDs may partially overlap as different multidimensional samples of the same pool of specific dimensions and not necessarily as different expressions of the same underlying common latent factor (e.g., Borsboom & Cramer, 2013). Alternatively, symptoms could causally influence each other, as; in general, problems lead to more problems, explaining the many positive correlations between symptoms or between individual PDs (e.g., Borsboom & Cramer, 2013). Network models

are better suited to identifying such causal interplay between variables, according to proponents of network approaches (Borsboom & Cramer, 2013). Any structural equation model with hierarchical relationships can be characterized as an equivalent network model that explains the relationships directly. Even a network of latent factors and direct relationships without a general factor is possible (e.g., van Bork, et al., 2017). Since, from a mathematical point of view, models hypothesizing a common cause, reciprocal effects, or common effects appear to be variants of the same mathematical Ising model (Kruis & Maris, 2016), statistics cannot be used to prove a model. Regarding PDs in particular, the p-factor has been criticized because it explains an insufficient amount of the variability in PDs beyond personality traits to justify a hybrid model as proposed in the Alternative Model of Personality Disorders (AMPD) in Section III of DSM-5 (American Psychiatric Association, 2013). The AMPD claimed the p-factor as a factor of variability independent from traits in PDs. In our study, we will therefore investigate the common, the differential, and the weighted explained variance of the general p-factor and the traits.

The p-factor has also been criticized for failing to offer guidance for therapeutic interventions, as it would not yield associations with specific treatment targets. This criticism gave rise to hypotheses concerning a general factor as an artifact or as an evaluative bias. In this context, the present study will test the validity of the constructs in the bifactor model, the specific trait factors cluster A (bizarre), cluster B (erratic), and cluster C (anxious), and the p-factor by covariance analyses to sort out the error variance. Furthermore, the correlations between the specific factors and a wide range of clinical features of patients will be tested, controlling for the p-factor. If the p-factor has no unique impact, the partial correlations should remain substantial.

2.4.3 The present study

Previous studies investigated the bifactor structure of psychopathology in adults by comparing the model fit with other types of factor analytic models. However, all these studies focused on symptom disorders (Lahey et al., 2012; Caspi et al., 2014) or patients diagnosed with PDs (Sharp et al., 2015). To date, no studies have investigated the p-factor in psychiatric patients presenting both state and trait-like affective-spectrum disorders. This is the first study to do so as it will compare different factor models of general psychopathology—a one-factor, a correlated-factors, a hierarchical, and a bifactor model—in a sample of inpatients (N = 241) presenting with a wide range of psychopathology. If the bifactor model fits best, the predictive value of the different factors for each PD will be calculated. In addition, in response to the doubts about the construct reliability, discussed above, different covariance indices will be calculated to sort out sources of error variance and to justify that the variance in the PDs covaries with the p-factor, controlled for covariance due to specific factors. In addition, the relationships between the specific factors and indices of intrapersonal (e.g., clinical distress, symptoms of depression and dissociation, coping, personality pathology) and relational functioning will be investigated.

2.4.4 Methods

2.4.4.1 *Sample/participants*

This study is part of a larger study, LEDAS (Leuven-Duffel Assessment), which was conceived to develop a feasible evidence-based clinical assessment battery to assess PDs and psychotherapeutic indications. The study was approved by the regional ethical committee of Emmaus and by the ethical committee of the University of Antwerp. The sample consisted of 241 patients (100 males, 141 females, aged 18–60) consecutively admitted to a psychiatric service at the University Psychiatric Center, Duffel. Patients had a mean age of 36.6 years (SD = 11.9). Their median level of education was 3 on a 6-point scale (1 = primary education, 2 = lower secondary education, 3 = higher secondary education, 4 = higher education short course, 5 = higher education long course, 6 = university education).

Based on the algorithm of the Assessment of DSM-IV Personality Disorders (ADP-IV) questionnaire for obtaining a DSM-IV-TR Axis II diagnosis, 84 of 241 patients fulfilled criteria for a PD: 84 patients for cluster B PDs (66 borderline, 1 narcissistic, 8 antisocial, and 9 histrionic), 84 patients for cluster C PDs (21 obsessive-compulsive, 15 dependent, 48 avoidant, and 34 not otherwise described), and 46 patients for cluster A PDs (21 paranoid, 12 schizoid, and 13 schizotypal). Of the 241 patients, 99 had clinical depression, with a Beck Depression Inventory (BDI-II) score of more than 20, indicating moderate depression, and 66 patients scored positive for dissociation on screening with the DIS-Q (see below).

2.4.4.2 *Procedure*

Written informed consent was obtained from all patients. In the first 2 weeks of admission, the intelligence of all patients was screened with the Progressive Matrices (PM; Raven, 2006). In a second stage of the research, 80 patients were also assessed by more differentiated intelligence testing with subtests of the Wechsler Adult Intelligence Scale (WAIS III; Wechsler, 1981) and the Groninger Intelligence Test (GIT; Luteyn & Barelds, 2004). The mean estimated IQ was 96.7 (range 58–126). Seven patients with an IQ of less than 70 (indicative of intellectual disability) were excluded from the study. A share of 241 patients completed a set of questionnaires digitally; these comprised the Inventory of Personality Organization (IPO, Lenzenweger et al., 2001), the Depressive Experiences Questionnaire (DEQ, Blatt, D'Afflitti and Quinlan, 1976), the Beck Depression Inventory (BDI-II, van der Does, 2002) for assessment of the severity of depression, the Dissociation Questionnaire (DIS-Q, Vanderlinden et al., 1993) for screening of dissociative problems, the Symptom Checklist 90 (SCL-90; a screening tool for clinical complaints; Arindell & Ettema, 1986), and the Utrechtse Coping Lijst (UCL, Schreurs & van de Willige, 1988). These were all part of the routine assessment of patients.

2.4.4.3 *Measures*

The IPO (Lenzenweger et al., 2001) is a 136-item self-report measure, of which all items are scored on a 5-point Likert scale (1 = never true, 5 = always true). In this study, the three

main scales—identity diffusion (21 items), primitive defense (16 items), and deficits in reality testing (20 items)—were used. Studies have reported excellent internal consistency and test–retest reliability for the IPO (e.g., Berghuis et al., 2009; Lenzenweger et al., 2001). Regarding validity, several studies have supported the convergent, concurrent, and discriminant validity (e.g., Lenzenweger et al., 2001; Smits et al., 2009). In this study, Cronbach's α was .875 for the primitive defense subscale, .917 for the reality testing subscale, .884 for the identity diffusion subscale, and .962 for all items.

The SCL-90 (Arindell & Ettema, 1986) is a 90-item self-report symptoms checklist in which patients are asked to rate each symptom on a 5-point Likert scale ranging from 0 (not at all present) to 4 (extremely present). The SCL-90 consists of nine subscales: somatization (α = .892), obsessive-compulsive symptoms (α = .860), interpersonal sensitivity (α = .917), depression (α = .908), anxiety (α = .901), hostility (α = .832), phobic anxiety (α = .861), paranoid ideation (α = .767), and psychoticism (α = .808), as well as a total scale (α = .975). The Cronbach's α in this study was .975.

The BDI-II (van der Does, 2002) is a 21-item, multiple-choice self-report inventory measuring the severity of depression during the past week. Factor analysis has shown that the BDI-II consists of three factors: a somatic factor (α = .686), an affective factor (α = .745), and a cognitive factor (α = .709) (Vanheule, 2008). Cronbach's α for the total BDI-II was .864.

The DIS-Q (Vanderlinden et al., 1993) is a 63-item questionnaire, which uses 5-point Likert scales ranging from 1 (not at all) to 5 (applies very strongly to me) to measure to what degree dissociative experiences apply to the subject. Four subscales are measured: identity confusion and fragmentation (α = .946), loss of control over behavior, thoughts, and emotions (α = .911), amnesia (α = .879), and absorption (α = .477). The scale has good to excellent internal consistency (Cronbach's α in this study = .964) and test–retest reliability, as well as good construct and congruent validity (Sno, 2004; Vanderlinden et al., 1993).

The PM (Raven, 2006) assesses IQ by 60 items in 5 sets of visual pattern detection or series completion tasks with multiple-choice answers comprising 6 or 8 options. Each set has a different cognitive theme and items are ranked following an increasing level of difficulty. The rough score is converted into a percentile according to a set of criteria such as age.

The UCL (Schreurs & van de Willige, 1988) is a 47-item instrument, in which patients are asked to score on a 5-point scale (0 = never, 4 = very frequently) how often they use specific coping strategies in difficult situations. The UCL consists of seven subscales: active problem solving (α = .776), palliative reaction (α = .727), avoidance (α = .711), seeking social support (α = .870), passive reaction (α = .730), expression of emotions (α = .614), and reassuring thoughts (α = .660). Test–retest reliability (0.45–0.85) is satisfactory. Cronbach's α in this study was .787.

The DEQ (Blatt et al., 1976) is a 66-item questionnaire based on phenomenological experiences of depressed patients. Subjects must rate each item on a 7-point Likert-type scale. Initial principal components analyses with VARIMAX rotation in a sample of 660 students yielded three factors, dependency, self-criticism, and efficacy (Blatt et al., 1976). The Dutch version of the DEQ, which was used in this study, has similar psychometric characteristics to the original DEQ (Luyten et al., 1997). Scores were calculated using the factor scores and loadings of the original DEQ (Blatt et al., 1976). According to Blatt et al. (1976), each of the standardized scores of the 66 items should be multiplied by the factor weight coefficient obtained in the normed sample for the loadings on self-criticism and dependency. In this unit weight scoring system, all 66 items, relative to their factor weight coefficients, contribute to form the final scores of each factor. Thus, the internal consistency reliability coefficient α is reported only for the entire DEQ questionnaire. This α was 0.807 in the current study.

The QRI (Pierce, Sarason, & Sarason, 1991) is a self-report questionnaire consisting of 25 items evaluated on a 4-point scale, measuring three dimensions of experience in a specific relationship: experience of support (7 items), of conflict (12 items), and of depth (6 items). The Cronbach's α in this study was 0.836 for the depth subscale, 0.901 for the conflict scale, and 0.891 for the support scale.

The ADP-IV (Schotte & De Doncker, 1996) provides a dimensional as well as a categorical scoring of PDs, following the criteria of DSM-IV-TR (American Psychiatric Association, 2000). It consists of 94 items assessing each criterion of DSM-IV-TR for PDs. Patients must score all criteria on a 7-point Likert scale indicating the typicality of the trait (1 = totally disagree, 7 = totally agree). In cases with a dimensional score above 5, there is an accompanying distress and impairment score (1 = totally not, 3 = most certainly). Dimensional scores are calculated by summing ADP-IV trait scores and distress scores for each dimension (the different PDs of DSM-IV-TR, namely, paranoid PD, schizoid PD, schizotypal PD, borderline PD, narcissistic PD, histrionic PD, obsessive-compulsive PD, avoidant PD, depressive PD, and passive-aggressive PD). A categorical score is calculated using an algorithm based on combinations of cut-offs for the trait and distress scores. Finally, scores for each cluster of PDs are calculated (cluster A, bizarre; cluster B, erratic; cluster C, anxious). Concordant validity with the Structured Clinical Interview for DSM-IV Axis II Disorders is good, with κ = 0.5 (Schotte et al., 1998; Schotte et al., 2004). Cronbach's α in this study was .985 for the total scale, .951 for cluster A, .957 for cluster B, and .940 for cluster C.

2.4.4.4 *Statistical analyses*

First, confirmatory factor analysis (CFA) models were estimated of a one-factor higher order (p) model and a first order correlated-factors model with the DSM-IV-TR cluster A (bizarre), B (erratic), and C (anxious) PDs as lower order factors. Further, following state-of-the-art guidelines for testing bifactor models (Brunner et al., 2012), a higher order

hierarchical model with the three cluster factors as dependent from a latent hierarchical overarching p-factor, and a confirmatory bifactor model (CBM), were estimated. The CBM consisted of a model with a p-factor that was independent from the cluster factors A, B, and C. The correlations between the clusters were set to zero in the hierarchical and the bifactor models. Hence, we compared the model fits of a model with only one general psychopathology factor explaining the variance in PDs of DSM-IV-TR (American Psychiatric Association, 2000); a first-order model with three correlated factors, namely, the trait clusters of DSM-IV-TR (i.e., the bizarre, erratic, and anxious traits); a higher order model suggesting that the three trait factors are independent but correlated by an hierarchical overarching common general psychopathology factor; and finally, the bifactor or nested-factor model in which the PDs are defined by specific traits that are nested in the general psychopathology factor.

The models were estimated using weighted least squares estimator (WLSE) in Amos (Arbuckle, 2014). Models were identified by fixing the variance of each factor to 1, and freely estimating the first factor loading. Model fit was assessed (Sivo et al., 2019) by the Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) with values of .90 and .95 reflecting acceptable and good fit, respectively (Hu & Bentler, 1999). In addition, the root mean square of error of approximation (RMSEA) (Steiger, 1990) was compared for the different models, for which values lower than .06 are acceptable (Hu & Bentler, 1999).

Further, to evaluate the construct reliability of the psychological measures hypothesized in the bifactor model following state-of-the-art guidelines (Rodriguez, Reise, & Haviland, 2016a, 2016b), we analyzed the covariance between items, factors, and the general factor on the level of the bifactor model, on the level of the factors (p and clusters A, B, and C), and on the level of the items (the individual PDs) to control for construct bias. To do so, the Bifactor Indices Calculator was used (Dueber, 2017).

First, we evaluated with the Percent of Uncontaminated Correlations (PUC) the percentage of covariance that reflects variance only from the p-factor, and with the Explained Common Variance (ECV) the proportion of PD covariance relative to the variance of general and specific factors (i.e., clusters A, B, C, and p) in the model. ECV NEW is ECV calculated only for the items loading on that specific factor, that is, p or cluster A, B, or C. With ECV > .70, the relative bias is estimated below the 10% benchmark (Bonifay, Reise, Scheines, & Meijer, 2015). An ECV > .70 of p and PUC > .70 suggest that the common variance is essentially unidimensional. With ω hierarchical > .70 as a measure of PD covariance in absolute terms, that is, the percentage of systematic variance in unit-weighted total scores that can be attributed to individual differences on the general factor, Reise, Scheines, Widaman, and Haviland (2013) indicate that the “presence of some multidimensionality is not severe enough to disqualify the interpretation of the instrument as primarily unidimensional” (p.22). Therefore, ω was calculated as the estimate of internal reliability of the multidimensional composite. For ω of p, all items were considered; for cluster A (bizarre),

cluster B (erratic r) and cluster C (anxious), only item loadings relevant for ω S (= ω specific) were considered. Relative ω then was calculated as the percentage of reliable variance in the composite due to the component, for the general factor independent of the specific factors, and for the specific factors independent of the general factor. We also evaluated the Average Relative Parameter Bias (ARPB), the difference between an item's loading in the unidimensional p-model and the loading on p in the bifactor model divided by the general factor loading in the bifactor model. An ARPB < 10–15% would be acceptable and indicate no concern for bias (Rodriguez, Reise, & Haviland, 2016b). Construct reliability (H) was assessed based on the guidelines of Hancock and Mueller (2001). H evaluates how well a latent variable is represented by its given items, and, as such, how suitable and replicable a structural equation model is likely to be (Rodriguez, Reise, & Haviland, 2016a). H is calculated as the ratio of variance left unexplained. It represents the construct replicability, the correlation between a factor and an optimally weighted item composite; H > .80 indicates a well-defined latent variable (Hancock & Mueller, 2001, p. 230) and is likely stable across studies (Hancock & Mueller, 2001). However, we first checked the Factor Dominance (FD), the correlation between factor scores and the factors. Factor scores should be used only when FD > .90 (Gorsuch, 1983, p. 260). Finally, the Item Explained Common Variance (IECV) was calculated, indicating the unidimensionality on the item level. IECV > .80 indicates a fairly unidimensional item set reflecting the content of the general factor (Stucky & Edelen, 2015, p. 51).

For the estimation of the predictive regressions in the model, according to Bring (1994), standardized regression weights can best be computed by dividing the t-values of the regressions, because with the β weights, the unstandardized weights are partitioned but not the accompanying SEs, which could bias the results significantly. Next, we calculated correlations of the obtained factors with indices of intrapersonal and interpersonal functioning, controlling for the p-factor. Because of the numerous correlations, we applied a Bonferroni correction. Imputation with multiple complex imputation (Enders, 2017; van Ginkel, 2014) prevented bias in the results by attrition and produced a single imputed data set based on all participants who provided at least some PD data (N = 241). All analyses were done using SPSS 25 (IBM Corp., 2013).

2.4.5 Results

As shown in Table 1, only the bifactor model provided a good fit to the data.

2.4.5.1 *Structural Equation Modeling*

Statistics		Model Fit			
	Evaluation criteria	One Factor	First-Order Model	Higher Order Model	Nested-Factor Model (Bifactor)
Absolute Fit Indices					
N Parameters		35	33	33	47
χ^2		766.771	1405.471	1405.471	42.720
df		55	32	32	18
p	<.01	< .001	<.001	<.001	.001
χ^2/df	< 3	13.941	43.921	43.921	2.373
Hoelter .05	>200	27	49	49	184
ECVI		3.076	.990	.503	.503
ECVI CI		[2.76-3.42]	[.91-1.08]	[.45-.59]	[.45-.59]
MECVI	smallest	3.09	.99	.50	.52
AIC	smallest	836.77	1471.47	1471.47	136.72
BCC	smallest	840.28	1471.96	1471.96	140.68
Relative Fit Indices:					
NFI	>.95	.672	.866	.866	.975
RFI	>.95	.535	.811	.811	.925
IFI	>.95	.688	.868	.868	.986
TLI	>.95	.553	.814	.814	.955
Noncentrality-based Indices					
CFI	>.90	.685	.868	.868	.985
RMSEA	< .10 acceptable >.05 good <.08 <.01 excellent	.218	.170	.170	.071
[RMSEA, 90% CI]		[.205,.232]	[.162,.178]	[.162,.178]	[.044,.099]
PClose	.05	.000	.000	.000	.096

Table 1 Model fit of the four factor analytic models

Note. ECVI = Expected Cross Validation Index; CI = Confidence Interval; MECVI = Modified Expected Cross Validation Index; AIC = Akaike Information Criterion; BCC = Brown-Cudeck Criterion; NFI = Normed Fit Index; RFI = Relative Fit Index; IFI = Incremental Fit Index; TLI = Tucker Lewis Index; CFI = Comparative Fit Index; RMSEA = Root Mean Square of Error Approximation; Pclose = p value of null hypothesis that estimate is below .05.

All PDs showed significant loadings on the p-factor (see Figure 1). Interestingly, borderline PD features showed very high loadings on the p-factor, suggesting that borderline PD features were most indicative of a high p-factor.

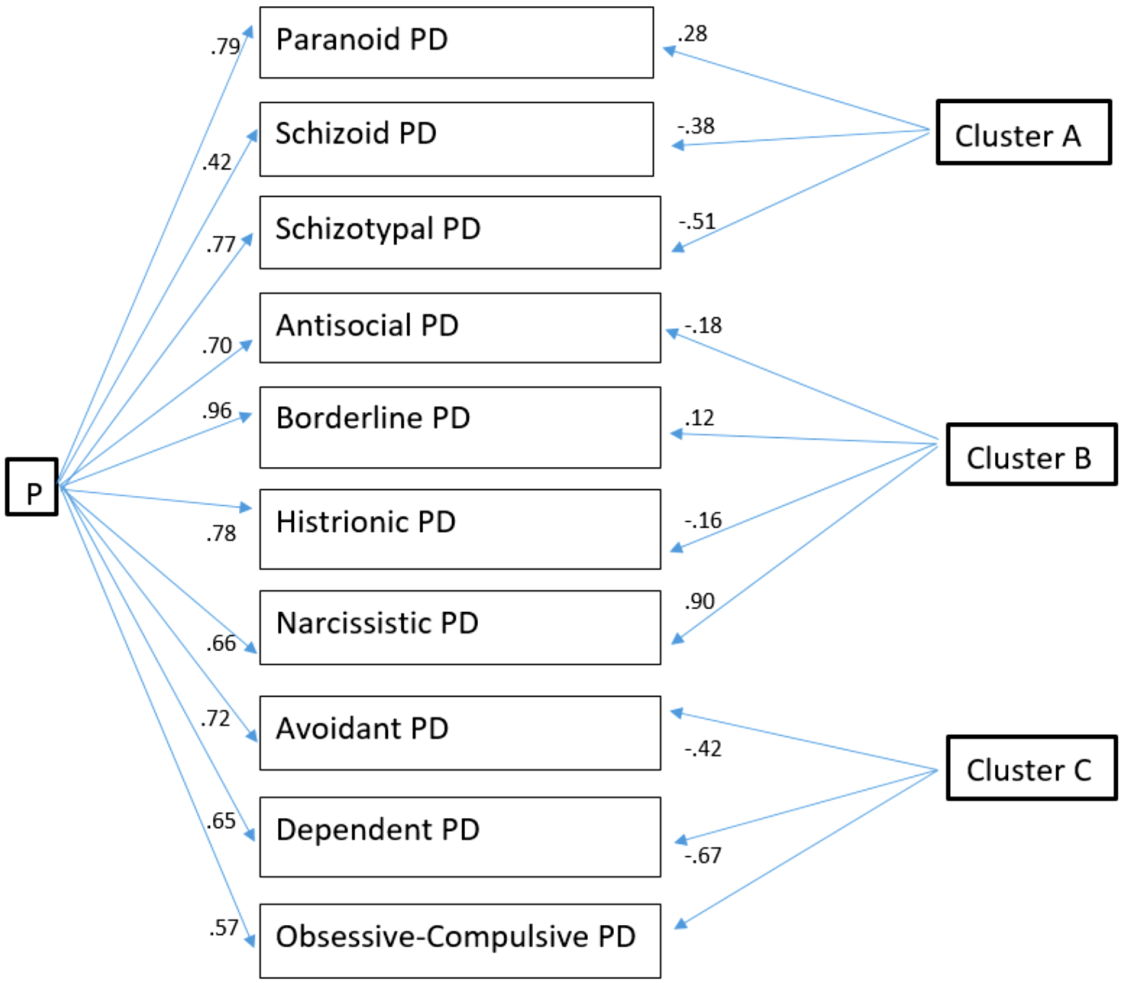

Figure 1 Factor loadings of the bifactor model

All PD features were predicted by p and trait factors (see Table 2). For borderline, narcissistic, schizotypal, and avoidant PD features, the bifactor models explained almost all of the variance in PD features. The p-factor explained about one-third of schizoid, antisocial, and obsessive-compulsive PD features.

Predicted PD	Bifactor	Cluster A				General Psychopathology				Standardized Regression Weights (Bring, 1994)
	R^2_{A+P}	R^2_A	*B*	*SE*	t_A	R^2_P	*B*	*SE*	t_P	$t_A/t_P = B_A/B_P$
Paranoid	.70	.08	-2.68	.50	-5.35***	.62	7.44	.51	14.48***	-.37
Schizoid	.32	.14	-2.78	.54	-5.16***	.18	3.00	.45	6.66***	-.78
Schizotypal	.85	.26	-5.64	.73	-7.69***	.59	8.50	.61	13.97***	-.55
	Bifactor	Cluster B				General Psychopathology				Standardized Regression Weights
	R^2	R^2	*B*	*SE*	t_B	R^2	*B*	*SE*	t_P	$t_B/t_P = B_B/B_P$
Antisocial	.32	.03	-1.56	.55	-2.83**	.49	5.97	.49	12.22***	-.23
Borderline	.94	.01	1.66	.77	2.16*	.92	13.10	.68	19.30***	.11
Histrionic	.64	.03	-1.57	.54	-2.92**	.61	7.54	.52	14.42***	-.20
Narcissistic	1.25	.81	-7.48	2.07	-3.62***	.44	5.53	.52	10.69***	-.03
	Bifactor	Cluster C				General Psychopathology				Standardized Regression Weights
	R^2	R^2	*B*	*SE*	t_C	R^2	*B*	*SE*	t_P	$t_C/t_P = B_C/B_P$
Dependent	.70	.18	-4.57	1.07	-4.28***	.52	7.84	.61	12.89***	-.33
Avoidant	.87	.45	-7.22	1.62	-4.47***	.42	6.94	.62	11.11***	-.40
Obsessive-Compulsive	.35	.03	-1.463	.56	-2.60**	.32	5.02	.53	9.49***	-.27

Table 2 Variance explained and coefficients of the regression models in the bifactor model

Second, analysis of indices based on the covariance matrix (Table 3) suggested that the construct or composite reliability (Netemeyer, 2003; Brunner & Süß, 2005) of the p-factor was good. On the model level, the PUC, which reflected only variance from p, was 73%. Because the ECV was .717, indicating a relative bias below the benchmark of 10% (Bonifay, Reise, Scheines, & Meijer, 2015), the common variance can be regarded as essentially unidimensional. Moreover, the ARPD of .077 (i.e., < 10%) indicated that there was no reason to assume parameter bias by standard error (Muthén et al., 1987). On the factor level, factor score estimates should be used only when the factor dominance is above .90 (Gorsuch, 1983, p. 260). This was the case only for the p-factor and cluster B, with FDs of

.978 and .988, respectively. Indeed, the calculated H for all factors suggested that only p and cluster B were well-defined factors (H > .80), with H = .954 and .813, respectively. The relative ω showed that the reliable variance was 95% for p and only 27% for cluster B. This was not due to problems with the internal reliability of the factors, because these were good: 95% for p, 79% for cluster A, 94% for cluster B, and 83% for cluster C. It is possible that in clusters A and C another source of variability was producing variance. All in all, 72% of the common variance was explained by the p-factor, while only 26–34% of the variance was explained by the specific factors A, B, and C. On the individual level, the paranoid, antisocial, borderline, histrionic, and obsessive-compulsive PDs seemed to form a fairly unidimensional item set that reflects the p-factor (Stucky & Edelen, 2015). In contrast, the narcissistic, dependent, and schizoid PDs appeared to be rather multidimensional concepts.

Construct Evaluation Level	PUC	IECV	ECV (S&E)	ECV (NEW)	RPB	ARPB	ω/ ωS	ωH/ ωHS	Relative ω ω/ω H	*H*	FD
Model											
	.733		.717	.717		.077					
Factor											
p			.717	.717			.947	.903	.953	.954	.978
Cluster A			.068	.257			.793	.069	.087	.377	.820
Cluster B			.124	.264			.938	.043	.046	.813	.988
Cluster C			.092	.341			.832	.247	.297	.514	.878
Item/PD											
Paranoid		***.888***			-.038	.038					
Schizoid		.550			-.238	.238					
Schizotypal		.695			-.104	.104					
Antisocial		***.938***			.043	.043					
Borderline		***.985***			-.031	.031					
Histrionic		***.960***			.013	.013					
Narcissistic		.350			.015	.015					
Dependent		.746			-.028	.028					
Avoidant		.485			-.169	.169					
Obsessive-Compulsive		***.918***			-.088	.088					

Table 3 Evaluation of construct reliability of the model with bifactor covariance indices on the level of the model, the factors, and the individual personality disorders

Note. PUC = Percent of Uncontaminated Correlations; IECV = Item Explained Common Variance, assessment of unidimensional construct in bold; ECV = Explained Common Variance; ECV NEW = ECV calculated only on the items loading on that specific factor;[A]RPB = [Average] Relative Parameter Bias; ω = estimate of internal reliability of the multidimensional composite; ωS = ω Specific; ω H = ω Hierarchical; ω HS = ω Hierarchical Specific; Relative ω = ω divided by ω H; H = construct replicability; FD = Factor Dominance.

2.4.5.2 *Correlations with indices of functioning*

After Bonferroni correction, controlling for the p-factor decreased the large positive associations of specific traits with clinical distress, with severity of depression, and with dissociation, to only a small negative association of cluster B with depressive symptoms and with clinical distress, and a small positive association of cluster A with reality testing, and of cluster C with depression (See supplement 1). Overall, the p-factor alone explained about 70% of the variance.

Variable	*r* CLUS A *(r controlled for p)*	*r* CLUS B *(r controlled for p)*	*r* CLUS C *(r controlled for p)*
Age	-,236**	-,119	-,167*
	-171	***.132****	*.031*
IQ	.055	-,065	,10
	-.05	*-.025*	*.073*
SCL -90	,602***	.527***	,.636***
	.061	***-.197****	*.123*
BDI –II	,513***	.426***	,563***
	.043	***-.230*****	***.172****
DIS-Q	,614***	.587***	.617***
	.045	*-.056*	*-.036*
UCL	,140*	,164*	.114
	.009	*.069*	*-.095*

Variable	*r* CLUS A *(r controlled for p)*	*r* CLUS B *(r controlled for p)*	*r* CLUS C *(r controlled for p)*
IPO-PD	.087	.045	.017
	.111	*.005*	*-.093*
IPO-ID	.125	.066	.074
	.107	*-.042*	*-.038*
IPO-RT	.123	.067	.047
	.131*	*-.011*	*-.091*
QRI-S	.257	.255	.252
	.021	*.014*	*-.023*
QRI-C	-.058	-.051	-.091
	.024	*.044*	*-.068*
QRI-D	.173	.182	.197
	-.019	*.002*	*.032*
Dependency	.108	.076	.124
	.013	*-.071*	*.056*
Self-Criticism	.077	.064	.023
	.071	*.040*	*-.091*

Table 4 Zero-order correlations of the specific factors of the bifactor model with external variables, and their partial correlations, controlled for the general factor

Note. IQ = Intelligence Quotient; SCL-90 = Symptom Checklist-90; BDI = Beck Depression Inventory; DIS-Q = Dissociation Questionnaire; UCL = Utrechtse Coping Lijst; IPO = Inventory of Personality Organization, IPO-PD = primitive defense, IPO-ID = identity problems, IPO-RT = reality testing disturbances; QRI = Quality of Relationships Inventory, QRI-S = support, QRI-C = conflict, QRI-D = depth. Bonferroni correction: p<.05 = .05/14=.004, p<.01 = .01/14 = .0007. Italics: partial correlations; bold: significant after Bonferroni correction

2.4.6 Discussion

This study investigated the structure of psychopathology in a sample of inpatients with affective-spectrum disorders and with a wide range of personality pathology by comparing the model fits of different factor models. A bifactor model provided the best fit to the data and was the only statistically acceptable factor model. It explained almost all of the variance in the borderline, narcissistic, schizotypal, and avoidant PDs, but only about one-third of the variance in the schizoid, antisocial, and obsessive-compulsive PDs. Hence, the bifactor model does not appear the best model for modeling all PDs. Still, all PDs loaded significantly on the p-factor.

State-of-the-art evaluation indices of bifactor models showed that the common variance of the bifactor model was essentially unidimensional, reflecting the variance of the hypothesized independent p-factor. In this sample, the p-factor accounted for 73% of the variability in PDs, while only about 30% of the variance was due to specific factors. No bias by error variance was found. PDs appear to be in essence p-factor disorders. Reliability tests showed that in this population of patients with affective-spectrum disorders, only the p-factor and cluster B appeared to be reliable constructs. Other sources of variance seemed to influence cluster A and cluster C disorders. On the individual PD level, the paranoid, antisocial, histrionic, borderline, and obsessive-compulsive PDs seemed to form an item set reflecting the content of the p-factor. The schizoid, schizotypal, and avoidant PDs showed parameter bias. One might consider here a factor of detachment as a common factor shared by clusters A and C.

However, the main finding was that the bifactor model made it possible to determine the proportion of traits and p-factor simultaneously within each PD, making it clear that borderline PD is almost entirely a p-factor disorder. In contrast, narcissistic PD seems an actual trait disorder. It suggests that the p factor should not be dispersed in the traits but remain an independent model factor.

We may have identified a reporting bias of clinical burden in schizoid, obsessive-compulsive, avoidant, and narcissistic PD; incidentally, these are disorders that have been empirically associated with the use of attachment deactivating strategies (e.g., Levy et al., 2015; Luyten et al., 2021). It seems that deactivating attachment is associated with underreporting distress. Studying the relationship between deactivating attachment and the p-factor seems to be a promising future path of investigation.

2.4.6.1 *Limitations and future directions*

A major limitation of this study is that, due to the cross-sectional design of the study, it does not allow causal conclusions to be made. It also did not assess potentially important other independent variables, such as genetic factors. Secondly, although the sample size

was relatively large, some of the analyses might have been underpowered, particularly as there were relatively few patients with psychosis in the sample.

2.4.7 Conclusions

This study provides further evidence for the validity of a general psychopathology factor in a sample of inpatients with affective-spectrum disorders and with a broad range of PD severity. Future research is needed to further replicate these findings and investigate potential implications for therapeutic intervention.

Chapter 3 Part three: General Discussion

12

[12] *Note. Freshidea (photographer). (n.d.). Living with anxiety (jpeg). Retrieved june, 8, 2022 from https://stock.adobe.com/be_fr/Library/urn:aaid:sc:EU:931bf7c9-e698-452e-81a8-a328ac2cc6e1?asset_id=335196375*

The clinical perspective

3.1 Psychomotor retardation in late-life depression

3.1.1 Findings on psychomotor retardation in depressed elderly

Starting from a typical in-hospital case of a depressed elderly woman with psychomotor retardation (PR), and the state of the art of research on PR, we presumed that PR is clinically a very important feature of depression. However, in our research PR appears – different from core symptoms of negative affect or anhedonia – not to be exclusively related to severity of depression in all cases. Zooming in on PR in elderly depressed patients, the phenomenology of PR seemed different from and more prevalent than PR in depression in younger people. In elderly depressed patients, PR proved to be a distinct feature, predicting relapse, independent from stressors or physical illness. For the explanation of PR in elderly depressed patients, we were referred to and informed by some major competing hypotheses presenting explanations such as vascular depression, degenerative processes, a scar of past depressions, bipolarity, but also depression-aging interaction. We selected the last hypothesis, because it was the hypothesis that was best amenable to behavioral research that could generate directly translatable clinical findings due to the parallel in the method of research and the method of clinical practice. As, however, due to our restrictive inclusion criteria, the sample we investigated could not be considered as a representative probe of the natural population of depressed elderly, we only had the opportunity of selecting out confounding variables of medication or degeneration. A valuable control, of course, as these confounding variables constitute important biases that are to be considered in hypotheses concerning PR in elderly depressed patients.

Comparing PR to cognitive functioning and to the concomitant effect of depression and aging in non-demented and medication-free elderly patients with major depression to a matched control group of healthy elderly (Beheydt et al., 2015a), revealed that depressed elderly patients were significantly slower, both in scores on rating scales and in performance on copying tasks and cognitive performance tasks. Post hoc comparison with a similar study with younger depressed patients revealed, moreover, an additive effect of depression and aging in fine motor skills. Interestingly, the study led to a reinterpretation of initiation time as cognitive time and of motor execution time as motor time. Indeed, increasing the complexity and thus increasing the cognitive share of the psychomotor tasks did not increase the initiation time but it increased the motor time and – to a lesser degree – the reinspection time. The cognitive component of PR as measured with performance tasks involves more motor circuitry than as measured with standard cognitive tasks. As PR became more visible in more complex tasks with unmedicated patients, while it had been more obvious in simple tasks with medicated elderly depressed patients in a previous study

(Pier et al., 2004), it may be assumed that PR due to depression is different in quality from medication induced PR. PR in depression is predominantly cognitive, thus observable in complex tasks, provoking cognitive executive impairment, while PR due to medication is motor retardation, observable in simple copying tasks, and provoking abnormal velocity. In all, the conclusion was that PR is a complex symptom that may show important qualitative differences subsequently leading to different clinical treatment targets.

Medication is known to be an important influence on PR. But less well known is the extent and the nature of the effect of medication. Therefore, the next study attempted to assess selectively the effect of medication in elderly depressed. We thereto studied longitudinally the effect of a monotherapy with 5-20mg escitalopram in depressed patients (Beheydt et al., 2015; see Chapter 1.3.). As mentioned before, on every assessment point, results were compared to those of a healthy control-group receiving no treatment. The main finding was that a treatment of 12 weeks with escitalopram improved mood symptoms (depression and anxiety) to a moderate level, but much less cognitive and psychomotor symptoms. A longer follow-up study is needed in elderly to assess the effects of escitalopram, because slower and delayed timeframes and varying stages of change were found for different symptoms. Though interaction effects of group and time appeared, they could not be proved, due to large variance in the patient group. However, dividing the patient group in high and low processing speed revealed that whereas the high functioning group did not differ from the control group, the low group followed a significantly different trend with lower cognitive and psychomotor performance. Processing speed and executive function were bad predictors for the prognosis of treatment with escitalopram in the first 12 weeks.

3.1.2 General discussion on psychomotor retardation in elderly

3.1.2.1 *The 'general age-related slowing' put in perspective*

In the past two decades ‘age-graded decrements in accuracies and maximum speed of fine motor movements have nurtured the assumption of general age-related slowing of central cognitive processes’ (Krampe, 2002: p. 769). Basic processing steps are assumed to be proportionally slower in elderly, therefore, in complex tasks, requiring more steps, increase in negative age-related effects can be expected (Krampe, 2002).

Our results confirmed a general age-related slowing of central cognitive processes of the elderly compared to the younger population. The adult and older population of healthy controls differed significantly on all information processing-, executive- and fine motor-performance tasks, with effect sizes that ranged from very large (hedge’s g=1.784) to huge (hedge’s g=11.62) (Sawilowsky, 2009). The exception was a small effect size for the initiation time of copying simple lines. Indeed, the initiation time appeared a reaction or activation time, but not a cognitive time. The initiation time of complex figures reflected a ceiling effect in the slowing by aging. It differed significantly between healthy younger and

elderly subjects, but not between younger and elderly depressed patients, who were both already slowed down by depression.

Interestingly, we found a differentiation in the slowing of the basic steps between the adult and the elderly population. Differences in initiation time of copying simple lines were small, differences in motor execution and reinspection times were very large (hedge's g> 1.7), differences for inhibition in Stroop tasks (hedge's g>2.6) and the executive function of WCST (g>3.6) or the matching time of SDST were huge, but differences in the writing time of the SDST or the initiation time of copying complex figures were 'gigantic' [m.i.] (g>10). We can summarize these findings as the cumulative age-related negative effect on complex tasks.

Also, elderly differed more from each other on specific tasks than the younger population. The variability between subjects is larger in elderly, standard errors being systematically larger than in the adult population. These results seem to underpin the first challenge of the general slowing hypothesis in favor of a differential aging hypothesis, viz. that general PR is dissociable in different processes of age and the specific individual training of motor skills during life (playing piano, soccer, …) and that the behavioral outcome of these processes reflects long-term adaptations to internal and external constraints and, hence, gives rise to significant stable interindividual differences (Krampe, 2002). It would be interesting for future research to investigate whether the stable interindividual differences in processing speed are more dependent on decrease by degenerative processes or on gain by motor expertise. More precisely, the question whether loss of motor and psychomotor skills due to aging can partly be compensated by active training deserves more research. As does the question whether depression affects well-trained psychomotor skills less than regular skills.

Elderly seem to be slower than younger subjects, but some aspects of that slowing are likely due to qualitative differences such as increased (motor) inhibition in case of sensory conflict, as shown in the comparison of the Stroop-task results. Evidence for such qualitative differences in elderly can be observed in the increasing time needed for action preparation as shown in the longer initiation time of complex motor tasks. Investigation of associations of aging with cognitive functioning (Hoogendam et al., 2014) showed that fine motor skill, processing speed and visuospatial capacity, but not memory were most affected by age. Our results suggest that decline in visuospatial capacity might be a major cause of extremely longer planning of execution of visuospatial complex movements in fine motor skill. Inhibition is larger (g=2.66) in elderly relative to younger adults in case of sensory conflict in the Stroop, but the difference in time for output between the populations is almost five times larger (g=10.93) in tasks with higher visuospatial load, as in drawing or writing complex figures. Even if we do not have data on differences between adults and elderly in pure visuospatial capacity, we can conclude that initiation (timing),

inhibition (cognitive control), planning (sequencing) and execution (motor control) of complex visuospatial movements are differentially affected by age.

3.1.2.2 *Subtypes of PR in elderly depressed*

In depressed elderly with PR, the unique feature is the double burden of age and depression on the cognitive control network. Young never depressed subjects are faster than young, depressed subjects, who in turn are faster than older never depressed, still faster than older depressed subjects. In our results, however, it was clear that depending on the task, there were two discernable patterns in PR, a general slowing pattern and an executive burden pattern. The general slowing pattern is similar to the one described by Rao and colleagues (Rao et al., 2015). Depressed patients are slower than controls in processing speed, in reading, in motor time. In the executive pattern, the younger subjects are better than the elderly, independent of depression, in tasks demanding higher cognitive control. Hence, young, depressed subjects are faster than older depressed (Rao et al., 2015) are, which is significantly confirmed by our results.

Comparison between older never depressed subjects and older depressed subjects reveals that on a task of sustained attention and inhibitory control, older depressed people recruit more fronto-striatal regions in order to perform on the same level as their never depressed peers (Rao et al., 2015). In the sample of our research, however, we found two cognitive subtypes of elderly depressed patients, characterized by differences in processing speed, divided by the central tendency as a cut-off. Depressive elderly patients of the high processing speed group did not significantly differ from healthy elderly, while patients of the low functioning group showed a significant lower performance both in motor and in cognitive functioning.

In summary, longer reaction times in elderly depressed were not explainable by PR alone, but also by altered inhibition, a specificity of depressed elderly patients (Carvalho et al., 2014). Altered inhibition has important clinical relevance, since it may explain the lack of inhibition of suicidal intrusions and may thus be related to higher suicidality in older depressive patients. Alexopoulos (2005) had suggested that inhibitory problems were predictive of lack of response to antidepressants and Malsert and colleagues (Malsert et al., 2012) reported that reaction times and error rates were predictive of response to transcranial magnetic stimulation over the dorsolateral prefrontal cortex. The type of inhibitions Alexopoulos and Malsert are referring to are typical visuospatial inhibition problems involving the CBTC or corticobasal ganglia-thalamo-cortical network (Figure 1).

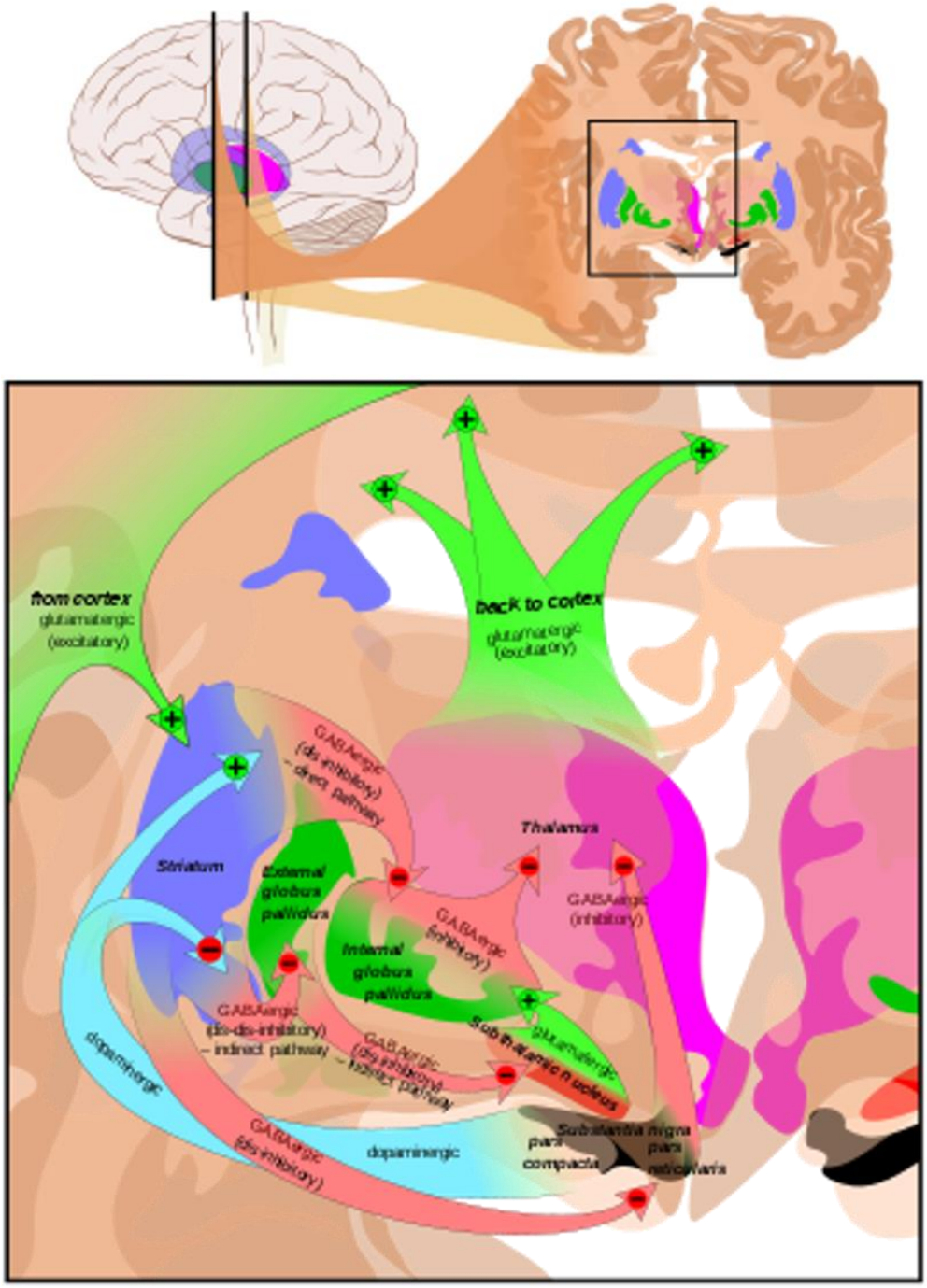

Figure 1 The Corticobasal ganglia-thalamo-cortical network, a possible biological marker for psychomotor retardation

Note. *Cortico-basal* *ganglia-thalamo-cortical loop. Reprinted from anatomical term of neuroanatonomys in Wikipedia,april 2019, retrieved march 20, 2022 from:* *https://www.wikidata.org/wiki/Q28456996*

The inhibition data revealed by our research have clinical relevance in the sense suggested by Alexopoulos, but they are also interesting from the functional process perspective we want to take. The observed differential inhibition development (just like the highly differential PR development) in aging suggests a developmental point of view on the functioning of the patient, suggests in other words a complementary functional approach to evaluate adequately the meaning of observed differences in PR.

Although PR appeared as a distinct feature in depressed elderly in our results, on a diagnostic level, it could only be taken in consideration for the diagnosis of depression, but it was not a reliable indicator for prognosis or remission. At least not over the period of 12 weeks of beginning treatment, as it caused no significant improvement in PR during that period, even when there were observable (mood) signs of remission of the depression. This result is clearly in line with the finding by Vrieze et al. (2013) that psychomotor change, though a key MDD dimension, is not related to treatment outcome.

3.1.3 Conclusion

PR is associated with aging as well as with depression. Depressed elderly, then, show an additive effect. A refined analysis of PR suggests that the initiation time is a motivational and compensatory time. In younger depressed patients or in healthy elderly, a cognitive more complex task demands more initiation time for more effortful control or more frontal recruitment. However, the initiation time does not lengthen in elderly depressed in which this compensatory frontal recruitment is not possible. The contribution of the motivational aspect can be tested by the difference between simpler and more complex visuospatial fine motor tasks. In contrast to the original assumption that the initiation time was the cognitive time, in our studies, the cognitive time was incorporated in the motor time. The cognitive time involved two important and different aspects. First, there is the inhibitory capacity, which is impaired by aging processes and lengthens the motor time in PR of elderly, healthy and depressed. Secondly, there are stable cognitive impairments which may be an indication of impaired cognitive reserve, due to pharmacological effects (e.g., benzodiazepines), but also due to degenerative processes (e.g. demential, vascular,). This aspect becomes clear by prolongation of the processing time as in tasks of symbol substitution. Finally, there is the real motor time, which we called neuromotor, and which manifests itself as velocity, the speed of drawing a simple line without cognitive load. It is strongly related to the Widlöcher rating of PR (Widlöcher, 1983), which may be an indication of the CBTC circuit functioning and of intra-individual changes in the dopaminergic state. PR appears to be too much used as an umbrella term for a motivational, two kinds of cognitive and a neuromotor deficit. Even if they interact, many times, the three kinds of PR may cover different aspects of a disease, demanding different treatments if possible.

3.1.4 Assessment of the different aspects of PR

New ways of assessment of PR (Figure 2) have focused on the differentiated aspects of PR. Specific psychomotor alterations of the inhibition process in depressed elderly were discovered through the assessment of PR with saccadic and anti-saccadic reaction times of eye movements by Carvalho and colleagues (2014). These alterations were associated to severity of depression and not to cognitive deficits. Considering in more detail our findings, inhibitory problems appeared to be an aging effect. It is possible that the additive effect of aging and severity of depression is an interaction effect. We agree, however, that inhibition problems are not indicative of cognitive deficits of degenerative processes. The cognitive executive psychomotor deficit or acquired lack of cognitive reserve has been measured, for example, by measures of verbal fluency in bipolar patients (Thomas-Ollivier et al., 2017), and was associated to "MOCA", a well-known screening of executive deficits. In our findings, an easy marker for elderly with or without cognitive reserve appeared to be the measure of processing speed as measured with the symbol substitution task. It may be an interesting future investigation to find out what could be a useful cut-off in this task to decide on the availability of cognitive reserve. Especially because processing speed differentiated strikingly the trajectory of patients during treatment with escitalopram. Finally, the motor core of PR is velocity. This has traditionally been measured by posture-cognitive dual-tasking (e.g., Deschamps et al., 2016), associated with the non-cognitive retardation in the execution of simple movements. Hence, assessment of postural performance, which is easy to perform and quick to assess in clinical settings - for instance, by maintaining quiet standing balance during two trials, with versus without vision, and combined with backward counting (dual task)- is sometimes used as a reliable and objective marker of PR in MDD patients (Deschamps et al., 2016). In line with our findings, however, we are inclined to dispute the interpretation of this task, and to claim instead that this kind of task is a marker of a velocity syndrome, from a physiopathological point of view a non-cognitive PR disorder. It is associated with motor times of simple lines, with postural sway, and with the retardation rating of Widlöcher. This type of retardation was also confirmed in research using actigraphy, differentiating depressed patients with or without PR, and was associated with intra-individual changes and the CBCT network connectivity (Krane-Gartiser, Henriksen, Vaaler, Fasmer, & Morken, 2015).

Generalizing, we can state that PR consists of a motivational component, associated with compensating frontal recruitment, of an inhibitory component, associated with the double burden of aging and severity of depression leading to alterations in the inhibitory process, of a cognitive deficit component due to lack of cognitive reserve, and finally, of a neuromotor or velocity component, indicating the higher connectivity of the CBCT network with varying dopaminergic states. All of these aspects can be measured separately and may lead to the development of new assessment instruments, specific treatment options and a better understanding of depression in elderly.

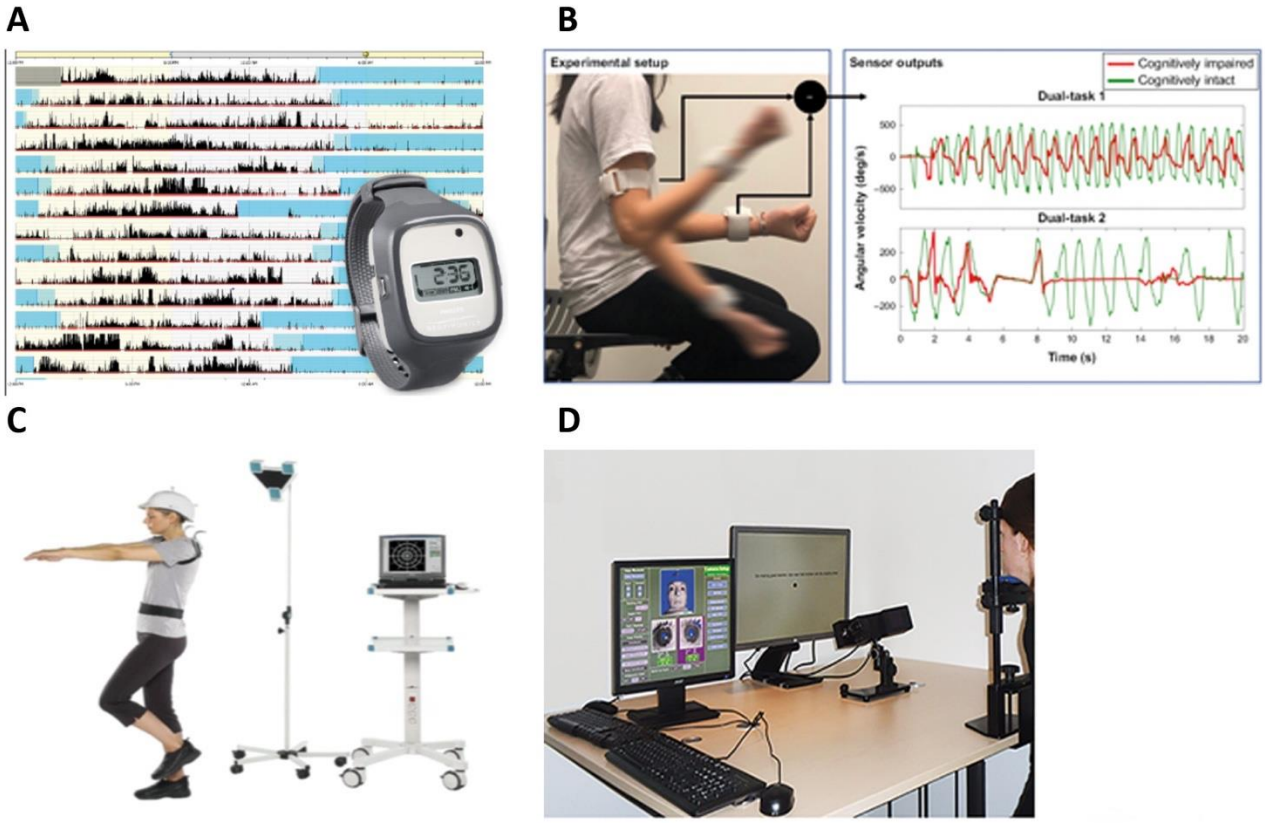

Figure 2 New assessment tools for psychomotor retardation: actigraphy (e.g. Krane-Gartiser, 2015), posture dual tasking (e.g. Deschamps et al., 2016), cranio-corpography (e.g. Terziivanova et al., 2018), and measurement of saccadic eye movements (e.g. Carvalho et al., 2014)[13]

[13] *Note. (a) Actigraphy. From 'Research Facilities' by (n.n.)(n.d.). 'Sleep Research Clinic and Laboratory' Retrieved june 8 2022 from https://sleep.hku.hk/facilities/ © Pok Fu Lam, University Hongkong. (b) UEF experimental setup, sensor and parameters. From 'The Association between cognition and dual-tasking among older adults: the effect of motor function type and cognition task difficulty.by H. Ehsani, M.J. Mohler, K. O' Connor, E., Zamrini, C. Tirambulo & N.*

3.1.5 Confounding factors

Of course, as indicated in the introduction, the studies in this thesis are subject to limitations in generalizability because of the limitations for inclusion, particularly the stringent requirements for inclusion that are almost non-existent in the natural population such as the exclusion of patients with comorbidities or the requirement of being medication-free at baseline. However, these requirements served a different goal than wanting to observe depression with PR in a natural elderly population. The primary goal was the almost experimental set-up of qualitative performance observation of the essence of depression with a large assessment battery in an aging population. The filtering out of confounding factors such as comorbidity, medication and the interaction of comorbidity and medication could of course not prevent the intrusion of other confounding factors such as, for instance, possible bias of motivation. Indeed, as the demands made on included patients were relatively high, patients with seriously impaired motivation dropped out and only sufficiently motivated patients remained included. That bias became apparent in the results, which showed no main effect of motivation with varying complexity of the tasks. The stringent inclusion requirements also led to the exclusion of patients with somatic adversities that, admittedly, play an important role in depression in elderly, such as cerebrovascular disease, causing severe disability, dementia or delirium. As, moreover, seriously depressed elderly show most of the time a multiplicity of complaints that need to be treated, and they also show a multiplicity of dysfunctions resembling dementia, even cognitively, we suppose that some exclusions because of positive dementia screening on the Mini Mental State Examination, could have been a sign of severe depression leading to extreme cognitive disability along with severe impairments in motivation (apathy). But these subjects were not the focus of the present investigation. Another restriction on the generalizability of our study is that none of the inpatients and residents of the homes we screened met the inclusion criteria. On the other hand, their exclusion indirectly proved once more the general disabling effect of depression in elderly patients. In all, the stringent inclusion requirements cause our conclusions to be reframed as well-controlled experimental results of a small scale, in depth, 'in vitro' study of the relation between depression and PR in aging patients.

Toosizadeh,2019,. Clinical Interventions in aging, 14, p 661. (https://www.dovepress.com/getfile.php?fileID=48969). Copyright 2019 by Dovepress, (c) Computerized ultrasonographic cranio-corpo-graphy-test of Untenberger-Fukuda. From 'Objective quantification of psychomotor disturbances in patients with a major depressive disorder' by.P. Terziivanova, E. Haralanova, E. Milushev, R. Dimitrov and C.-F. Clausse, 2018, Journal of evaluation in clinical practice, 24, p 828, (https://doi.org/10.1111/jep.12916), Copyright 2018 by Wiley & Sons, Ltd. (d) Demonss set up. Picture of the device set-up. Adapted from J.A. Bijvank, A. Petzold, L.J. Balk, H.S. Tan, B.M. Uitdehaag, M. Theodorou, & L.J. van Rijn 'A standardaized protocol for quantification of saccadic eye movements; DEMoNS' Plos One, 2018, 13(7), p 4, (https://doi.org/10.1371/journal.pone.0200695.q002). Copyright 2018 by Nij Bijvank et al.

variable	Used as measurement of	After investigation appeared a measure of	investigated in one of the papers Possible clinical marker
Motor time Simple lines	**Motor speed**	**Acceleration, no significant difference between not-depressed and depressed medication-free patients** (observed in neuromotor retardation for instance due to medication in findings of M. Pier in medicated /deteriorated patients)	Yes marker of retardation by **medication** (or deterioration)
Initiation time complex figures	**Cognitive load**	**Response time** (time to start, was not longer with more cognitive load **in depressed medication-free patients**, BUT measuring **compensating desinhibition in normal elderly**)	yes marker of brain **aging** (=compensating frontal recruitment) in elderly
Motor time complex figures	**Motor speed**	**Cognitive load**, also psychomotor (due to depression and to aging cognitive AND motor) retardation	yes marker of **depression** cognitive AND motor retardation = PR
Processing speed: Categorical difference (by central tendency) = interindividual difference	**Cognitive speed**	**Prognostic subtypes of change with escitalopram** in depressed patients independent of severity of depression: different evolution normal processing speed same progress in PR as normal controls	yes Interesting marker, **might** be marker of **deterioration in elderly**
Intra-individual variability in motor time of simple lines	Variability, **reliability of prognosis (regression trend)**	**Fronto-striatal connectivity?** Differing balance between striatal dopaminergic state and frontal control of the circuit, independent of severity of depression	No, calculation based on literature … marker, **might** be marker of **fronto-striatal instability, a transdiagnostic factor**

Table 1 Five observable clinical aspects in one symptom of PR

3.1.6 Overall relevance of the study

To conclude, we want to stress the complementary relevance of this specific type of small-scale investigation in addition to the necessary large-scale studies (Denscombe, 2014). The carefully chosen restrictions in the inclusion requirements, the well-focused research purposes, and the possibility of applying a large battery of tests on a select sample made it possible to focus a study, within a clear theoretical framing, on clinical observations that could be directly relevant in clinical practice and treatment.

3.1.7 Clinical guidelines for treatment of elderly patients with PR

Starting from a typical in-hospital case of a depressed elderly woman with PR, we presented a clinical picture showing that PR is more than a simple symptom in elderly patients. It is a symptom that requires careful assessment.

On admission, Lily was agitated. The accompanying symptoms of fear and movements and the fact that she did not talk anymore, could readily suggest a diagnosis of anxiety or psychosis. However, the clinician should bear in mind the common trunk of non-interactiveness in PR and in psychomotor agitation. As was apparent in this case, agitation turns into retardation very easily, probably because of a common etiology. In DSM, no trajectories of symptoms turning into other symptoms are described, even when clinicians recognize the dynamics of symptoms. These dynamics of symptoms require that severe disorders be evaluated on a very frequent basis, independent from interventions performed.

Further, the case illustrates that very often, in hospital, it is not possible to have a useful diagnostic conversation to check DSM criteria systematically.

Also, remaining silent in a conversation may prove clinically ambiguous, as it may as well be a symptom of PR, as a psychological symptom of refusing to talk. For psychotherapeutic treatment, it is important to understand the difference, because interpreting a temporary deficit or impairment as a relational or motivational issue can be stress inducing and may destroy feelings of trust and comfort in the relationship with the therapist.

But how to know that it is not a relationship issue but rather a suffering from the circumstances? The circumstances that induce the silence could be manifold. In this case, she could be cross at her sons who forced her to accept help. Or perhaps they gave her the impression of wanting to get rid of her due to her difficulties of functioning independently, whereas she and her husband had always helped people and ‘never gave up on family’…

Further, how to know that it is not a stubborn keeping silent as a test of the relationship? After all, her life story indicates several possible sources for attachment problems.

Emotionally, losing a father and missing a mother are important threats for a safe start in life. These threats can be triggered repeatedly in stressful emotional and relational situations.

Yet, it is unusual to combine the taxonomic approach of DSM and the descriptive interpretive approach as I have endeavored here. And it is even more unusual to integrate the two approaches. One of the causes of this lack of integration of the two approaches is not the overriding influence of the use of DSM, as is sometimes asserted, but rather an unintended side effect of DSM usage, which caused the 'death of phenomenology' (Andreasen, 2007) and thus excluded a process approach. Replacing the 'thick description' by thoughtless enumeration of consensus symptoms too easily resulted in a lack of understanding with clinicians of what they observed. Exemplary for that lack of understanding, for instance, is the diagnostic uncertainty created by symptoms of depression that are not included in the consensus description of DSM, especially the cognitive ones. Still, such diagnostic uncertainties resulting from nomothetic consensus descriptions did not turn clinicians to more phenomenological approaches. These were, as yet, assumed to be lacking sufficient empirically testable evidence.

However, in the last decade, with larger studies and more different methodologies available, it has become possible to investigate empirically the value of clinicians' presumptions in more phenomenological impressions such as the cardinal importance of PR in depression. Depression remains puzzling because even the symptoms cannot be unambiguously interpreted. In the symptoms, affective and motor dysregulations in different brain structures are involved as much as mutual interactions between them. To be more successful in the interpretations of the observations and measurements, psychiatry ought to recur to more integrated approaches. In what follows, we will make some suggestions for clinicians regarding psychomotor retarded depression.

First, we would suggest clinicians to use objective measures for PR. As yet, psychomotor disturbances have been evaluated subjectively in clinical practice, by clinical observation or by using rating scales. Objective measures such as the drawing tasks in our studies, or postural performance cranio-corpo-graphy (Terziivanova, Haralanova, Milushev, Dimitrov, & Claussen, 2018); reveal that the group of depressive patients is heterogeneous at the psychomotor level. With cranio-corpography increased psychomotor activity and reactivity was found in some of the depressed patients. In our study, processing speed (measured with a symbol substitution task) was a marker of change in PR symptoms in treatment with SSRI, independent of mood changes. However, investigating different PR measures, we found that for measuring non-cognitive retardation, the speed of drawing simple lines was accurately measuring velocity. But also, that this objective PR measure correlated very well with the more subjective Salpetrière Retardation Rating Scale (SRRS), in contrast with measures of PR that included more cognitive aspects. An important suggestion,

contributing to clinical efficiency, would be to create an extended SRRS including a part for rating increased velocity, because the SRRS did well in measuring retarded velocity.

A second practical suggestion would be to use PR as a prognostic symptom in clinical depression. Indeed, other research confirmed our finding that severity of PR at baseline is negatively associated with clinical remission (Cao et al., 2016).

Third, like Terziivanova and Haralanov (2013), we also observed differentiation of different depression symptoms with PR. Anxiety or medication status – our patients were medication free - are not associated with PR severity (Terziivanova & Haralanov, 2013). A problem with PR is that both patients and assessing clinicians, tend to assume too easily that nervousness or tension are circumstantial states or personality traits and not symptoms of the disease.

Reappraisal of PR and the other symptoms of depression as having a different relationship with genetic, environmental, and childhood environmental risk factors, may lead to better clinical insight and yield better hints for what should be treated. Many symptoms are predicted by several factors, as expected. Yet, Kendler and Aggen (2017) found that appetite loss, psychomotor agitation and feeling tired were symptoms exclusively predicted by genetic and temperamental factors, while anhedonia, weight loss and trouble sleeping were predominantly predicted by childhood environmental risks. Interestingly, notwithstanding a common trunk, agitation was exclusively determined by genetic and temperamental factors, while PR was multidetermined. The authors thereby confirmed again the important role of transient influences and multi-determination of individual symptoms. That is where psychology and personality development come in.

Finally, I would like to stress the importance of using different perspectives. Taking account of differences between signs and symptoms, between a clinician's perspective and that of the patient, may lead to a more differentiated interpretation. Thus, Vares, Salum, Spanemberg, Caldieraro, and Fleck (2015) found that bringing together the perspectives is clinically helpful in the treatment of depression. It results in a multidimensional depressive construct that can be organized into a continuum of severity within ascending order of severity: sexual, cognitive, insomnia, appetite, PR, and agitation symptoms. In our study, the use of a multidimensional construct of PR resulted in the identification of five different clinical aspects (see Table 1) that are candidates for further future clinically relevant research. These five clinical features were simple motor speed as related to medication effects, cognitive load as related to aging, complex motor speed as related to depression, cognitive speed as related to deterioration, and variability of simple motor speed as psychomotor instability possibly related to hyperdopaminergic or bipolar features. They help a clinician in functionally understanding what is observed, but they also illustrate the possible ambiguity of one symptom.

We are living in a fascinating era and the science of psychiatry progresses fast with new insights and models. Further evolution may lead to a real integration of the two strands explored in this thesis.

3.2 The Level of Personality Functioning

3.2.1 Findings on the level of personality functioning

Until today, personality disorders (PDs) remain a controversial issue in clinical decision-making in residential psychiatry, although they are highly prevalent, with up to 40% in psychiatric patients (Herpertz et al., 2007). This study's central assumption is that the difficulty is partly due to the current gaps in our knowledge concerning the nature of PDs. Further exploration of the nature of PDs is therefore needed to foster clinical decision-making in treating PDs.

Recent findings on the nature of PDs fuel doubts about the validity and clinical utility of descriptive PD diagnoses. First, longitudinal studies indicate much less stability in PD diagnoses than expected, suggesting that current classification systems of PDs mainly reflect clinical distress (Herpertz et al., 2007). Indeed, the fundamental difference between axis I and axis II disorders of DSM-IV-TR has not been empirically confirmed. Some categories cover highly heterogeneous patient profiles with limited clinical utility (e.g. Kotov et al., 2011). The neurobiological underpinnings of PDs point to a close interaction between nature and nurture in their etiology but without evidence for polypharmacy for the broad spectrum of symptoms (e.g., Herpertz et al., 2007). Psychotherapy for specific PDs is effective, but only if it focuses on treating transdiagnostic maladaptive characteristics rather than on specific treatment for each descriptive PD (for an overview, see the evidence-based psychological interventions of the Australian Psychological Association, 2018).

As a result of the practical limitations in the descriptive approach to PDs, the Alternative Model of Personality Disorders (DSM-5, section III; American Psychiatric Association, 2013) proposed seven individual criteria, A to G, to determine PDs. The first, A, refers to personality functioning, understood as impairment in self and relatedness to others. Criterion B refers to pathological traits, organized around five domains, in analogy with the Big Five-factor model of normative personality. The criteria C and D refer to inflexibility and stability across time, and E, F, and G are exclusion criteria of other mental disorders (E), of effects of substance abuse or a medical condition (F) or features normative for a development stage (G). In AMPD, a PD is a combination of significant problems in functioning and more than one pathological trait (Krüger & Hobbs, 2020). The A criterion is an impairment criterion, and by consequence, a severity criterion; the B criterion is the style of disturbance criterion, which refers to the big five personality styles of the normal temperament. Zimmermann et al. (2019) published a comprehensive review on the

operationalization, reliability, and validity of criterion A and B and the relationship between the two, the association between functioning and traits. Krüger and Hobbs (2020), in turn, reviewed remaining questions on criterion A and necessary future research on PDs. Clarkin, Caligor, and Sowislo (2020), as proponents of the object-relational theory underlying the A criterion of the AMPD, proposed a grid based on the theory of object relations to underpin optimized clinical applicability of the AMPD. The studies discussed below will shed more light on the two defining PD dimensions of the AMPD to assess the severity of PDs and add the necessary comments from the clinician's perspective.

Three sets of findings emerged from our studies on Level of Personality Functioning (LPF).

The first set of findings (2.2.) came from a study of the use of the Differentiation Relatedness Scale (DRS; Diamond et al., 2014) to assess the LPF in a sample of nonclinical young adults (N=333). The DRS is a reliable and valid 10-level scale designed to rate levels of personality functioning on narrative descriptions of self and significant others. However, to date, most studies of the DRS have been done in clinical samples; little is known about its psychometric properties in nonclinical samples. Since the instrument is based on clinician rating of performance and not on self-report, the instrument is less sensitive to the egosyntonic characteristics attributed to some PDs. As Krüger and Hobbs (2020) pointed out, performance-based assessment measures may, therefore, provide a unique value in assessing criterion A and should be studied more frequently. This study examined linear and potential categorical relationships of DRS with demographic features and with indices of intrapersonal and interpersonal functioning (i.e., depressive, and dissociative symptoms, dependent and self-critical personality features, and warmth, conflict, and depth of intimate relationships) in a nonclinical sample of young adults (N = 333). Results showed less evidence for linear relationships between levels of DRS and intrapersonal and interpersonal functioning indices than were found in patients with PDs (e.g., Lowyck et al., 2013). By contrast, a cut-off of DRS level 6 differentiated young adults at risk for psychopathology from those with more adaptive levels of functioning. Most theories of personality hypothesize a dimensional distribution of vulnerability to psychopathology (e.g., Berghuis, Kamphuis, & Verheul, 2014). Using the DRS and dimensional analyses may fail to detect such underlying vulnerability if present in small subsamples of individuals within larger samples. However, the present result might also indicate a linear relationship between personality functioning and psychiatric symptoms in PDs and none in normal personality. An ordinal cut-off point instead of a graduated transition would then mark the difference between the two. Such a categorical difference indicating personality pathology would open new avenues for developing measures and assessments for screening for emerging PD in normal young adults before personality gets consolidated and distortions in personality become harder to treat.

Another issue in the study was the unidimensionality of the DRS in the convergence of the level of self-representation (DR-S) and representation of the mother (DR-M) and father

(DR-F) in personality development. Surprisingly, the representation of fathers was not associated with personality functioning. Hence, particularly in young adulthood, representational structures related to mothers as primary caregivers may be more important than those related to fathers, even if there has occurred a shift in the role of mothers and fathers in child development (Luyten & Blatt, 2013), with more balance between the parents regarding the extent to which they are involved in parenting and child development. Further research in this context is needed. Also, the LPF-construct assessed by the DRS seems not to be unidimensional. Latent LPF dimensions of (maladaptive) relatedness and differentiation made different and independent contributions to impaired DR-S. DR-S was not associated with the integration of both dimensions but reflected only maladaptive levels of differentiation, that is, self-criticism. This finding may be due to achievement issues playing a central role in this sample of university students (Tosevski, Milovancevic, & Gajic, 2010). Another possible explanation is that dependency outweighs protective and maladaptive effects (Abuín & de Rivera, 2015). Thus, these findings provide a partial answer to the debate about the organizational structure of criterion A indices in the AMPD, subdivided into self and other. Although the DSM-5 section III AMPD (American Psychiatric Association, 2013) structure could not readily be replicated by Sleep et al. (2019), in our study with normal young adults, the LPF appeared not unidimensional, supporting instead the structure of LPF conceptualized as the two dimensions of relatedness and self-definition.

The second set of findings (3.3), in 70 inpatients with general psychopathology and no primary PDs, showed that in assessing LPF, both the IPO as a self-report instrument and the DRS as a performance-based rating show different limitations in measuring LPF. Although correlations of descriptive PDs with LPF remained significant when controlling for clinical distress, the IPO seemed prone to state effects. The DRS seemed to be more independent from clinical distress but was unexpectedly unrelated to features of personality pathology, self-criticism (differentiation), and dependency (relatedness), constituting the LPF. This finding encourages consideration of momentary clinical distress when assessing a PD. It shows that performance-based instruments may be helpful to control for the effects of clinical distress. The LPF as a severity index of PDs reflects both trait-like (availability) and state-like (accessibility) features, of which, moreover, the relationship with the experience of patients is unclear.

The self-report IPO (Lenzenweger et al., 2001) was significantly related to age and clinical distress. When controlling for clinical distress, the IPO was still associated with cluster A (bizarre) and B (erratic) PD features, high levels of self-criticism, conflict in relationships, and low levels of adaptive coping strategies. The DRS was only related then to the schizotypal PD. DRS seemed to reflect availability, the structural vulnerability that gives rise to disturbances in the self (Zuroff, Sadikaj, Kelly, & Leybman, 2015), while IPO also reflects different degrees of accessibility of LPF in PDs. Comparisons of correlations between descriptive PDs and IPO (LPF) before and after controlling for clinical distress differentiated

three types of PDs according to the impact of clinical distress. This difference in impact could be due to the accessibility of personality functioning, the fluctuation of mental structures by mood, social context, or biological factors. The IPO showed in high LPF (cluster C) a relationship with PDs determined by clinical distress. In medium LPF (with extreme internalizing or externalizing traits), the presence of clinical distress showed no impact. However, in low LPF (clusters A and B), there was a clear impact of both hampered identity integration and clinical distress. DRS was only helpful to detect psychotic PDs (availability), IPO was complementary (availability and accessibility). Unlike LPF, traits seemed independent of clinical distress. Our results thus confirm the suggestion of Sharp et al. (2015, p.306) that "melding personality functional impairment and pathological traits into specific types, as is done in DSM-5 III (American Psychiatric Association, 2013), needs rethinking". The LPF, as measured by the DRS, is likely to assess the psychotic personality organization as conceptualized by Kernberg (Kernberg & Caligor, 2005), defined by thought disorders and disturbed reality testing. LPF was differentiated from the severity measurement in terms of extremity of traits in patients with general psychopathology, reflecting a lack of adaptability. The schizoid, the narcissistic, and the avoidant PD were independent of clinical distress or other dynamic variables such as age or educational level. Once controlled for distress, they were also independent of the LPF.

The third set of findings in this study (3.4) concerns the structure of psychopathology in PDs and the relationship between criterion A and B. Krueger and Hobbs (2020) rightly call the correlations between criterion A and B 'a key issue regarding the empirical evaluation of the AMPD', because they reflect different PD scholarship traditions. A reflects clinical inferences about psychological mechanisms that go awry in PD patients, and B reflects PD trait characteristics from patient reports (Krueger & Hobbs, 2020). Some think criteria A and B may be two sides of the same coin and simplify the AMPD by concatenating features described in A and B. In discussions concerning the relationship, remarkably, most of the arguments are methodological, for example, about the incremental validity of criterion A beyond criterion B. Nevertheless, as Morey appropriately replies: "If our goal is not simply prediction, but also understanding—to be able to parse different aspects of personality problems into aspects that may have different causal pathways, such as temperamental versus maturational elements of personality—then confounding such potentially distinct aspects may impede progress" (Morey, 2019, p.1197). Overall, there is a growing consensus that one general psychopathology (p-factor) and several specific trait factors may form the structure of psychopathology (see Caspi et al., 2014; Kotov et al., 2011). We investigated the factor structure underlying descriptive DSM-IV-TR PDs (American Psychiatric Association, 2000) with confirmatory factor analysis (CFA) in 241 affective-spectrum patients, assessed for all DSM-IV-TR PDs, for intrapersonal and interpersonal functioning. The model fits and concomitant factor loadings were assessed for a one-factor, a correlated-factors, a hierarchical, and a nested bifactor model. Then, regression models of the best-fitting model were used to predict descriptive PDs. Finally, correlations between specific factors and variables of intrapersonal and interpersonal functioning were calculated, controlling for the p-factor. CFA showed that a bifactor model, with specific

loading on cluster A (bizarre), B (erratic), and C (anxious) traits, and a general p-factor, was the only tested model providing a good fit to the data. All PDs loaded on the p-factor, but the paranoid, antisocial, borderline, histrionic, and obsessive-compulsive PDs appeared to be a unidimensional item set of the p-factor. The bifactor model predicted almost all the variance (>94%) in borderline and narcissistic PDs; however, while the p-factor explained almost all the variance in borderline PD, the share of trait variance was more influential in narcissistic PD. This outcome aligns with Sharp et al.'s finding that "narcissistic PD criteria's average loading on the general factor was rather weak (Mr=.31)" (Sharp et al., 2015; p. 396). The bifactor PD model was the only acceptable model with a good fit and good composite reliability for the p-factor. It enables independent prediction by the p-factor and the traits. It differentiates the borderline and narcissistic PDs on a qualitative level, even if mathematical differences between competing factor models are modest.

3.2.2 General discussion of the Level of Personality Functioning

3.2.2.1 *Availability, Accessibility, Susceptibility, Vulnerability, and Attachment style?*

Our studies showed that personality functioning is a promising concept for a better understanding of PDs. They have made clear that the non-adaptability of traits is independent of the level of personality functioning. In contrast with traits, the LPF intertwines with a predisposition for thought disorders and clinical symptoms. We attempted to substantiate the qualitative difference between the two AMPD dimensions empirically in these studies. Interestingly, the findings were consistent with those by Caspi and colleagues who suggested that the p-factor could be related to brain integrity and symptoms (Caspi et al., 2014) and those by Snyder and Hankin (2019) that the p-factor could be related to chronic stress, which it generates and predicts. Whether there is a causal link between these findings is an issue for further research.

The measurement of LPF in different ways revealed different functional aspects of a PD. First, as with DRS, the measurement of LPF with the performance-based assessment alone proved to be independent of the clinical burden. Independent of clinical burden, LPF measured psychotic vulnerability to thought disorders. Such resulted from the measurement of the first functional dimension, that of the availability of personality functioning. The discovery of that dimension seems to confirm the existence of the psychotic personality organization, as suggested by Kernberg (Kernberg & Caligor, 2005). Second, LPF measured by DRS appeared to categorically distinguish between well-functioning personality functioning and personality functioning intertwined with symptoms of psychological distress in a standard population. A cut-off for impaired personal functioning could be defined in a standard population of young adults.

Measuring LPF with IPO in patients with general psychopathology and a considerable variation in severity of PDs provided more insight into the role of clinical burden. IPO is the

instrument that measures personality functioning as derived from Kernberg's personality organization, focusing on the capacity for reality testing, identity problems, and the degree of primitive defenses. Control for clinical burden clarified that the relationship between descriptive PDs and the level of personality functioning as measured by IPO is not always comparable. Thus, there appeared to be types of PDs depending on the nature of the relationship between clinical burden, personality traits, and personality functioning. PDs in which the association with personality functioning is determined solely by symptoms are comparable to neurotic disorders as defined by Kernberg. They are typical cluster C PDs and show a strong association with dependence, which seems to be determined by clinical burden. One could justifiably call this type a susceptibility PD. The tremendous impact of clinical stress determines them. PDs thoroughly intertwined with personality functioning then resemble Kernberg's borderline organization, revealing cluster A and cluster B PDs. They also exhibit disruption of all IPO dimensions characteristic of the borderline organization, and they exhibit high levels of self-criticism, conflict in relationships, and low levels of coping strategies. These are PDs that disrupt the accessibility of general personality functioning, either through lack of availability, as in schizotypal PD, or through intertwining with clinical burden, such as schizotypal, borderline, and paranoid PDs. Finally, there is the adaptability type of PDs, unrelated to clinical burden but essentially typified by extremity of specific characteristics such as internalization and externalization. Even if one could argue that in this case, there are chronic problems in interpersonal functioning due to extreme traits, there is no connection with the clinical burden.

These findings together may suggest that it makes theoretical sense to distinguish and not mix traits and personality functioning, as these independently determine a PD at a descriptive level.

Furthermore, there was some indication that the p-factor, the degree of general psychopathology, is also a structuring factor in PDs. Indeed, in the three samples - the population with severe PDs, the one with general psychopathology, and the one without clinical problems - a different relationship was found between the level of personality functioning and inter- and intrapersonal functioning, the so-called dimensions of personality functioning. One might assume that these are three populations with a different p-factor, a different degree of general psychopathology. Unfortunately, we were unable to vary those three degrees within one sample. That is, therefore, a subject for further research.

It is helpful to clarify here that the degree of general psychopathology and personality functioning is still not the same. The p-factor underlying PDs appeared to be about the same as the borderline functioning described by Kernberg (Kernberg & Caligor, 2005). The general p-factor, common for state and trait disorders, appears in the literature to be a reasonably stable factor over time, predicting the degree of care and treatment required the vulnerability of the personality. However, analysis of the structure of psychopathology

with the bifactor model also revealed that a model with the general p-factor and specific traits, as in AMPD, was not predictive for some PDs. It was striking that these were PDs with deactivating attachment. It is possible that activating or deactivating attachment is a forgotten dimension in the alternative model of PDs. If so, the p-factor might coincide with borderline PD only insofar as activating attachment is concerned. This attachment style as a choice to deal with relationships and with the natural environmental factors in one's life, the relational coping, is logically dynamic according to the circumstances (Luyten & Fonagy, 2021). However, as LPF was also dynamic, the hypothesis that LPF is the association of the p-factor with the attachment choice might be a subject for further investigation. We may thus ultimately arrive at a model that also explains the deactivating PDs well.

3.2.2.2 *Limitations*

Of course, we are aware that some limitations of this study preclude firm conclusions. Complementary longitudinal studies monitoring the stability of the p-factor and the level of personality functioning together would be highly informative. Large subsamples with all degrees of psychopathology well represented in one overall sample would render the results of the complex analyzes less hypothetical. Finally, several replications would be required to make the persuasiveness of the concept of a cut-off for good personality functioning more reliable.

3.2.2.3 *Conclusion on the Level of Personality Functioning*

All in all, this research has yielded new solid arguments to believe that LPF is an interesting two-dimensional concept that divides PDs into three types, according to the degree of psychopathology, remarkably analogous to Kernbergs levels of personality organization. There is the susceptibility type, the accessibility type, and the availability type (Figure 1). These types may imply specific therapeutic indications. LPF is independent of the extremity of the traits, especially internalizing and externalizing, which mainly determine adaptability. The thought disorder dimension is to some degree associated with the p-factor. However, the share of p (gPD) and of traits differ hugely, with the borderline disorder as almost an equivalent of a p disorder and the narcissistic disorder as almost the equivalent of a trait disorder.

The gPD or p-factor seems to coincide with the borderline personality functioning, but such a model does not explain PDs with a deactivating attachment. Hence, the AMPD model seems to lack one more dimension, possibly activating or deactivating attachment. In that sense, this work mainly offers clues for unraveling the essence of a PD that provide insight and indications for appropriate clinical treatment.

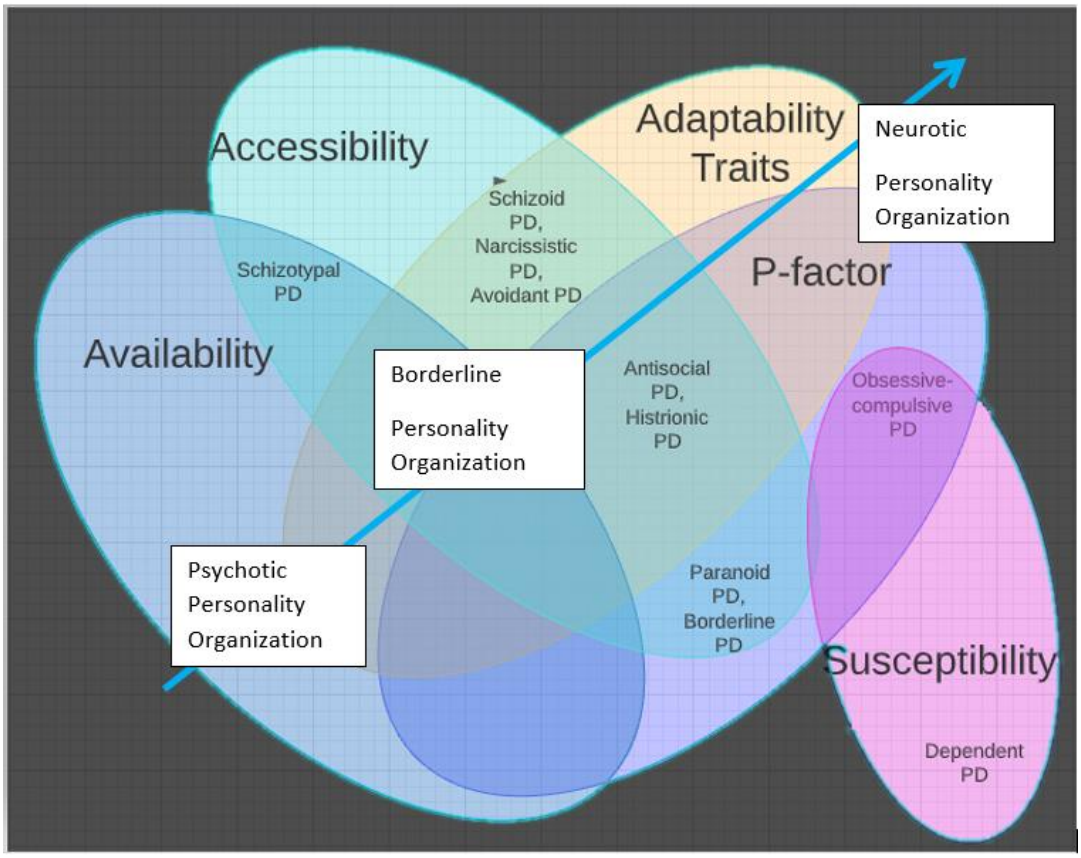

Figure 1. Functional types of personality disorders

3.3 The clinical perspective in the investigation of patients with complex affective disorders

In the preface, reference was already made to the fact that this is primarily the investigation of a clinician. The question may arise in which way a clinical approach can provide scientific benefit. In what follows it will be shown that the strength of the clinical approach is that it counterbalances compartmentalization in research. Even if disciplinary rigor is necessary to produce controllable research questions, it may in some cases limit the validity of the results, especially when dealing with complex issues such as human psychopathology. Thus, mood and PDs are two independently developed areas of research, but in clinical practice, they are highly comorbid. The importance of reducing barriers between disciplines will be exemplarily illustrated below in the discussion of the relationship between mood disorders and PDs.

Compartmentalization also hinders the scientist because it yields contradictory results that are difficult to explain. Again, the clinician's intuition, the knowledge by experience, may contribute here to hypotheses that better fit the complex reality. Such experiential knowledge proves especially valuable concerning gender differences that occur but in other cases are markedly absent, concerning the differences in psychopathology according to the degree of severity and, in the overall observation that despite the dimensional approach and the idea of graded continuity, there is an experience of a rift that marks severe psychopathology. However, since intuition is particularly sensitive to personal bias, these hypotheses need to be substantiated or contradicted one by one in methodologically reliable research.

The hypotheses in the two parts of this thesis concerned very different areas, so we reviewed the findings in two separate discussions. Clinically, however, it is very important to break down the traditional barriers between the disciplines and to reconsider the stereotypes created by the compartmental research. Specifically, we need to pay much more attention to the impact of PDs in the elderly or to the presence of psychomotor or cognitive retardation in young people.

A clinical framework is necessarily an overarching framework in which psychomotor symptoms and personality pathology share attention, and normal development, aging, longitudinal perspectives, and influences of context receive due consideration. These affect the same patient that needs to be understood. Therefore, in addition to necessary research into sub-aspects, complementary research into associations and interactions is required. Recognizing such connections, and seeing order, sequence and priority, part and whole in the phenomenological clinical multiplicity of how a patient presents, will produce more accurate hypotheses about cause and effect that in turn need further investigation, but are crucial for clinical prognosis and planning of treatment. Clinical phenomenology is yet an underdeveloped domain in research, in spite of the rich clinical tradition with a variety of

theoretical backgrounds. There is an enormous amount of knowledge available, but there is a surprising lack of scientific investigation of how to frame or structure the multiplicity and diversity of clinical observations. Neglecting cross-disciplinary observation in discipline specific research often deprives clinical practice of essential scientific information.

What the two parts of this thesis have in common is that they are two studies of long-term trait vulnerability in depression. This vulnerability appears as well in personality as in cognition and psychomotor functioning. Investigating these vulnerabilities together teaches us that it is not so easy to say that one vulnerability is a cause and the other an effect. Further longitudinal research should deepen our understanding here.

In what follows, we will discuss the relationship between affective disorders and PDs and indicate how this discussion affects the interpretation of the results obtained in our research and reveals how bringing together two seemingly independent subjects in the same clinical picture, could produce new insights for further investigation that may prove clinically relevant.

In a recent review Behn, Herpertz and Krause (2018) indicate that the intersection between affective disorder and PD opens a new direction in clinical research. The relevance of this type of research is that it calls for emphasis on underlying functional domains and personality characteristics, and that it challenges the effectiveness of usual treatment protocols for complex depression. New treatments should be based on interventions that map as well onto deficits in functional domains [such as cognition, psychomotor functioning (cfr. Rdoc domains, ICF, positive health domains), or personality functioning (cfr. AMPD), mi.] as onto personality characteristics [such as personality traits (cfr. Hitop), mi.] that influence clinical presentation and responses to treatment. Thus, Behn et al. (2018) invoke the findings that first line psychotherapeutic and pharmacological interventions to alleviate depressive mood are less effective in patients with borderline PD and that depressive symptoms in these patients remit with treatment of the PD, to suggest different pathogenesis, even if the two pathologies would not be totally independent.

In the same vein, Klein, Kotov & Bufferd (2011), formulated seven hypotheses about the relationship between affective disorders and PDs, with borderline PD in particular. The proposed relationships were that personality and depression would have a common cause (1), that they would form one continuous spectrum (2), that personality is a precursor of depression (3), that personality predisposes to developing a depressive disorder (4), that personality has pathoplastic effects on depression (5), that personality features are state-dependent concomitants of depressive disorders (6), and that personality features are consequences or scars of depressive disorders (7).

In general, their hypotheses lead to two entities of evidence. First, there is evidence for depression and borderline PD as distinct, yet commonly co-occurring entities. Aligning with

the common cause hypothesis, major depressive disorder (MDD) and borderline PD share common disease mechanisms, they overlap in the functional domain of affect dysregulation, and share affect symptomatology. Borderline PD often presents together with MDD. The diathesis-stress model has often explained the co-occurrence. Neuroticism or negative affectivity is a heritable trait characterized with exacerbated negative emotionality, sensitivity, and reactivity to stress (Tacket & Lahey, 2016), explaining 30% of the variability in BPD and 22% of the variability in MDD. The original assumption that depression would be more episodic and less reactive to the environment (Gunderson, 2007) and interpersonal stressors as rejection (Staeble et al., 2011), was not warranted by evidence. Depression frequently shows inter-episodic residual symptoms or even a chronic course from the beginning (Frödl, Möller, & Meisenzahl, 2008; Klein, 2010). The chronic early onset form of depression goes along with severe impairment in interpersonal functioning. On the other hand, BPD patients typically stop presenting BPD symptomatology as time passes (Zanarini et al., 2005). Also, MDD in BPD patients did not present with higher affective instability compared to MDD patients (Kohling et al., 2015). A phenotypic differentiation may not be as strong as initially thought.

The foregoing discussion can be directly linked to the results of the two investigations presented here. It leads us to a first crosslink between the two investigations. Indeed, large scale studies have shown that the most stable predictor of phenotypic stability is the p-factor, one factor of psychopathology that underlies severity in formerly called state or DSM-IV-TR axis I mental disorders as depression (Caspi, 2014) and in PDs (Sharp et al., 2015). It led to giving up the classification of PDs in ICD -11, presenting dimensional criteria for one single PD, based on domains of functioning and a five-factor model of personality traits, as in AMPD (Criterion A and B). A single criterion of dysfunction may thus present phenotypic variability as a function of maladaptive personality traits.

A second group of evidence reveals a specific phenotype of depression in personality pathology (Behn, Herpertz, & Krause, 2018). Specific depressive phenotypic profiles would stem from specific personality vulnerabilities. These findings fit in with the polarities theory of Blatt, as we studied it in the second part. The theory of polarities of differentiation and relatedness stems from Blatts' effort to understand the difference between two sorts of depression, the introjective depression and the anaclitic depression. The first type of depression would be disrupted in self-integrity and self-esteem (differentiation) with typically extreme self-criticism, the second type would be disrupted in relationships (relatedness) with typically fears of abandonment or dependency. The distinction was developed connecting personality predispositions with stressful life events. The relationship between both depression and borderline PD with adverse childhood events is pertinent and could explain the relationship between these two conditions as a common cause (Heim and Binder, 2012).

Herewith a second cross-link between the two parts of our research is suggested. Silva et al. (2017) showed that the two polarities of personality as integrated in the AMPD model, the positioning in an anaclitic (relatedness) or introjective (differentiation) experience, modulate the biological stress reactivity. Patients with more impairment in the self-differentiation exhibit more biological stress reactivity, and patients with more impairment in relationships show higher scores in self-report. The vulnerability for depression seems thus to be specific for the two types of patients, with impairments in self versus relatedness, especially when confronted with environmental events (Behn et al., 2018).

Within the predisposition model, depression in borderline PD has often been studied as a specific phenotype with accelerated patterns of emotional vulnerability (Mneimne et al., 2018), hardly identifiable with self-reports, because these are not suitable to consider the moment-to-moment variability in mood. Borderline PD depressive patients would exhibit greater impulsivity, aggressiveness, and interpersonal hypersensitivity (Fertuck et al., 2013), and greater risk of self-injury or suicidal behavior (Lieb et al., 2004). All that would contribute to the differential diagnosis of MDD and borderline PD.

Beyond phenotypes, functional domains underlying symptomatic representations are being examined and posit avenues towards etiopathogenetic models and treatment development with modular interventions, precision psychotherapy. The review of Behn et al. (2018) shows how common cause and common predispositions may explain both heterogeneity and differential response of depressive patients to standard treatment packages. Further development of measures of LPF, as we have discussed before, are important, especially because assessment of LPFS seems sensitive in distinguishing between borderline depression and MDD. The LPF as measured with the LPFS of the AMPD, or as measured with the DRS, shows high correlations with clinical ratings of PD, but not with DSM-IV axis 1 disorders. It suggests that depression is a tributary to but not a concurrent syndrome of PD. But although the findings suggest different physiopathology for depression and borderline PD, the clinical importance is, however, that change of trajectories in both depression and PD interact and do not follow parallel or independent tracks.

For treatment, it is crucial to know the critical components of change to be able to develop precision psychotherapy. Therefore, a variety of methodological approaches is needed, quantitative as well as qualitative strategies, in different samples, with different developmental levels, in different sets of outcomes, as self-report, from performance testing or as neurobiological outcomes. For instance, specific affective and socio-cognitive processing deficits associated with psychopathology markers of complex depression require add-on modules as dialectical behavior therapy for affect regulation or mentalization based treatment for social cognition and interpersonal hypersensitivity.

Thus, a first conclusion can be that criterion A of the AMPD, the LPF, has a significant value as a personality trait in the vulnerability for depression. Criterion A creates the possibility of differentiating MDD from depression in borderline PD, and the two dimensions of the LPF, differentiation and relatedness, differentiate between the share of biological stress reactivity, in response style on depression self-report scales, or in chance of drop out, and give indications for precision treatment of complex depression.

A second conclusion is that further developing the concept of the p-factor may eventually provide us with an important prognostic feature, a feature that can explain comorbidity and heterogeneity in PDs and in DSM-IV-TR axis 1 disorders. Apparently, controlling for the general psychopathology factor makes sense and helps to better understand the critical differences between personality traits, paving the way to understanding different functional physiopathologies. Such understanding is essential for the clinician wanting to find out how to treat functional problems instead of diagnoses, and to further develop a precision cure/care in psychotherapy. What is needed for the clinician, following Krueger & Hobbs (2020), are case conceptualization flow charts linked to the multidimensional nature of PD variation [and of depression, mi.]. Reducing dimensional information to singular labels (e.g., 'high on detachment') may be as problematic for reliability and case conceptualization as classical labels (Krueger and Hobbs, 2020, p 130). Therefore, the structure of psychopathology should be a first area of further examination, as it should serve as a reliable guide into multimodal, transdiagnostic, and transdisciplinary findings.

One obviously interesting feature in the present research has been psychomotor retardation (PR). From a clinical perspective, PR is especially interesting in that it is assessable by objective performance testing as well as by self-report and rating by clinicians or neurobiological measures. It proved to be one of the features that can disclose relationships between different types of outcomes collected by different disciplines of an identical construct. Moreover, it is a typical symptom of melancholic depression. It was as such known as a severity marker of depression. However, recent evidence is more ambiguous about the latter statement. We too, found in our research evidence for PR that was manifest without concurrent severity. Both were partly independent in elderly; PR could remain severe while depressive mood diminished. Also, research by Arbabi et al. (2016) suggested that PR was an indication of a psychomotor phenotype in borderline PD, predicted by impulsivity scores. Interestingly and more in general, research suggests that non-focal neurological soft signs such as PR might play a role in pathogenesis of chronic psychiatric disorders (e.g., Toro & Schröder, 2019). In borderline PDs, for instance, an important hypothesis was that the typical disturbances in emotion regulation would be associated with sleep disturbances. However, solely retardation in activity had negative associations with sleep disturbances (Arbabi et al., 2016). Also, PR assessed with corpography, showed that the lateral sway and the number of steps in elderly, was not associated with medication status. Yet, PR was negatively associated with remission in unipolar major depression.

All in all, the large range of investigations in two areas of research has provided new insights into complex disorders and leads to methodological conclusions for future research paths.

First, it leads to the conclusion that in scientific research of multidimensional layered phenomena such as PR, different types of assessment should be used and caution should be exercised when drawing conclusions stemming from different assessments such as self-rating, rating by a clinician, different types of performance assessments or neurobiological assessments. Typically, neurological soft signs were originally associated with schizophrenia and PR with severity of depression, now, a common pathway of PR associated with other neurological soft signs and brain functioning is suggested as an indicator of a more transdiagnostic vulnerability for psychopathology. Obviously, relationships between different assessments should be further investigated, as we proved in exemplary trails in the two parts.

Second, an important methodological shortcoming is the absence of consideration of bipolar depression in the comparison of borderline PD and MDD. The usual features indicated to serve differential diagnosis between unipolar depression and borderline PD, appear to differentiate less specific in the differentiation with bipolar 2 depression, another type of affective disorder. Indeed, a recent review posited childhood trauma history, deliberate self-harm, comorbidity rates, neurocognitive features, treatment response and impulsivity parameters to be less specific differentiators between bipolar 2 depression and borderline PD. The best predictors were the family history, the onset pattern, the clinical course, the phenomenological pattern of depressive and elevated mood states, and symptoms of emotional dysregulation (Bayes, Parker, & Fletcher, 2014). Our results suggest that the PR in elderly that is not associated with severity of depression could be associated with bipolar depression. This finding is in line with the suggestion of Tyrer and Brittlenbank (1993) to assess the presence of PR in depression in case of adolescent onset repeated depressions, to prevent misdiagnosis of bipolar disorder as borderline.

Finally, from a clinical perspective the results of our research motivate the overall conclusion that a deeper understanding of two unresolved discussions is to be pursued. Both the discussion on the nature of PR in psychiatric disorders and the discussion on the role of general psychopathology in PDs are at the heart of clinical psychiatry and related to the fundamental question of the nature of psychiatric disorders. Future study of the nature of psychiatric disorders is fueled by intriguing findings such as recent biological evidence “that the p factor may represent fundamental deficits in the ability to effectively integrate, coordinate, and monitor information such as is necessary for executive control” (Hairi 2019, p. 19) which motivates to further explore interdisciplinary research into the nature of general psychopathology in PDs, just as the findings that the cerebello-thalamo-cortical circuit (CTCC) is implicated in the emergence of liability for all common forms of psychopathology (Hairi 2019) and that it is known as the unique biomarker of PR at the

same time (Liberg & Rahm, 2015; Yin, 2017) invite to further pursue the nature of PR and its relation to psychiatric disorders.

It may have become clear by now that such study will involve interdisciplinary thinking, breaking down rigid partition of research and taking account of clinical experience.

3.4 A clinician's retrospective view through a dual lens

In retrospect, the two lines of the research described in the current study opened a perspective for future research and may prove fruitful for clinical practice. The experimental research and the theoretical analysis results reveal practical possibilities for a more differentiated clinical approach.

3.4.1 From symptoms to processes and vice versa

The first, experimental line of research, focusing on psychomotor functioning as a symptom, led to the finding of differentiation of psychomotor retardation (PR) into different functional types according to distinct clinical profiles of cognitive and motor characteristics, PR by depression, by pharmacological effects, by aging and through a degenerative process. This type of research is in line with psychiatric research that links symptoms to processes. Investigating the differentiation in phenomenological quality of symptoms is the first direction in such research, such as retardation due to depression (Sabbe, 1997) or chronic fatigue (Schrijvers et al., 2009) and changes in symptoms due to specific types of pharmacological treatment (e.g., Sabbe, 1997). Another direction of such research is to investigate relationships between particular symptoms and processes, such as the association of PR with action monitoring (Schrijvers et al., 2009) or aging (Pier, 2004).

The second line of research was a more theory-driven approach that focused on LPF. It revealed that empirical examination of traits referencing the complex processes of identity development, mental representation of attachment figures, or relational functioning could lead to meaningful subtyping of descriptive disorders. Availability, accessibility, adaptability, susceptibility, and personality vulnerability are functional and clinically relevant organizational principles of these subtypes. This approach revealed the qualitative difference between extreme traits and the LPF or vulnerability for each type of psychopathology.

3.4.2 The advantages of double vision in psychopathology research

Both perspectives, i.e. a more descriptive and more theory-driven approach, are valid and complementary approaches in studying psychopathology.

In addition, the current study suggests that this kind of research can bring the seemingly dead horse of phenomenology to life. It enables the clinician to reunite evidence-based practice with a functional approach that identifies tailored treatment indications in an intimate restorative psychotherapeutic contact with the patient by expert Phenomenological unfolding, Hermeneutical analysis and Dynamic analysis (PHD). This PHD (Messas et al., 2018) includes explanation of the patient's perception, explanation of the patient's attitude to his/her experience, and explanation of the life history in which experiences and positioning are embedded. The functional clinical approach advocated here is valuable because, besides fundamental biological research in psychiatry, there is an urgent need for studies into clinical practice as presented rather than in fragmentary sub-aspects. This study should target small sample groups and focus on specific problems, such as PR in younger and older patients, the changing importance of dependence versus criticism with age, or specific cultures or environments and their interaction with them. Subsequently, treatments can be tailored to comorbidities and complex problems by considering the availability of potential growth and its accessibility, the susceptibility to clinical need and relational conditions, and the vulnerability, apart from the lack of adaptability due to extremity of traits.

Thus, applying our findings to the case studies of Lily and Jimmy leads us to the diagnosis that Jimmy's symptoms may manifest the impaired integration of self and relatedness. In contrast, Lily’s PR symptoms indicate that biological treatment of the major depression is needed first.

Jimmy suffered from lower levels of personal functioning, intertwined with clinical problems and complicated by his detachment. The impairment of functioning started in young adulthood with parental separation, although there were some signs of psychomotor problems (stuttering, mild motor delay) in childhood. From adolescence, he had trouble with self-direction, such as the inability to achieve anything after high school despite high standards and awareness of his responsibility. He also reported problems with identity; he doubted whether he could be gay. Furthermore, he encountered difficulties in empathy; he had to drink to feel capable of social behavior and had long had social anxiety and problems in intimacy; involuntarily, he felt a sense of detachment. There are pathological features of detachment, antagonism, and disinhibition. On a descriptive level, he was diagnosed with an avoidant PD. There was a longer-standing personality functioning disorder associated with social ineptness that was inflexible and pervasive and not just because of substance abuse. The problems led to suicidality and depression and were too severe to be confined to a standard difficult stage of young adulthood.

Lily showed specific signs of biological depression, with several severe episodes during her life, accompanied by PR. The agitation and inability to respond, the fluctuating moods with substantial mood instability, the rumination and freezing show that this is much more likely to be early depression rather than depression due to incipient deterioration such as

incipient dementia. PR calls for the importance of biological treatment of the complaints and clinical distress. After that, psychotherapeutic treatment of grief could be a focus. The traumatic event of the early loss of her father and her mother's emotional unavailability had a substantial impact on the loss experience. In addition to the recurring depressive episodes and the disturbed coping with loss, Lily had experienced long periods of good psychosocial functioning in a socially satisfying life and a very intimate relationship with her husband. The level of personality functioning did not appear too disrupted because self and interpersonal functioning seemed adequate when not in clinical distress. The two cases demonstrate the need to use a two-pronged approach, starting from clinical symptoms to processes and vice versa. Then differences in treatment priorities appear.

Samenvatting

In de dagelijkse praktijk in de residentiële psychiatrie zijn er twee benaderingen om patiënten klinisch te beoordelen. De eerste benadering is de verplichte DSM-diagnose, een symptoom- en classificatiegerichte diagnose. In deze benadering worden symptomen systematisch beoordeeld, gekwantificeerd, kwalitatief geanalyseerd en begrepen als het resultaat van verschillende pathologische processen. De tweede benadering is een ontwikkelingsprocesbenadering die uitgaat van zowel normale als abnormale functionele processen. Die begint meestal met het opbouwen van psychotherapeutisch contact en het klinisch luisteren naar het verhaal van een patiënt om vervolgens op zoek te gaan naar tekenen van verstoring en vervorming of tekenen van veerkracht in het functioneren. In het voorbehandelingsonderzoek voor de daaropvolgende psychotherapie wordt een relatie opgebouwd.

De centrale stelling van dit werk is dat het mogelijk is om de huidige communicatiekloof tussen deze twee prominente benaderingen te overbruggen door de functionele benadering. Daarvoor zijn in dit onderzoek twee richtingen gevolgd: één van beschrijvende –mogelijks multidimensionale- symptomen en classificatie naar een dynamisch proces en, omgekeerd, één van een moeilijk samen te vatten proces naar beschrijvende symptomen en classificaties. Zo werd het streven om de kloof te overbruggen methodologisch vorm gegeven in een tweerichtings onderzoek.

In het eerste deel werd één symptoom, psychomotorische vertraging (PR), bestudeerd. We hebben het symptoom nauwgezet bestudeerd in een populatie van depressieve ouderen die medicatievrij waren. Dit onderzoek in kleine steekproeven was bedoeld als een in vitro onderzoek. Een steekproef van 40 depressieve ouderen werd getest met een batterij van verschillende klinische, cognitieve en psychomotorische retardatietesten op vier momenten in een drie maanden lopende monobehandeling met escitalopram max. 20mg. Uit die eerste studie bleek dat, met de verschillende maten van vertraging, verschillende soorten vertraging konden worden onderscheiden, zoals cognitieve vertraging, vertraging van initiatie van een beweging, motivationele vertraging en motorische vertraging. Vertraging bij depressieve ouderen uitte zich overwegend in de uitvoering van complexe fijn-motorische tekentaken met gecombineerde cognitieve en motorische belasting. In het vervolgonderzoek werden we geconfronteerd met een significante uitval door somatische comorbiditeiten, typisch voor depressieve ouderen. Maar van de 20 patiënten met op de vier momenten een beoordeling, bleek de depressieve stemming eerst te verbeteren, terwijl de psychomotorische verbetering pas later en langzamer optrad. Bovendien leken er op basis van de scores voor verwerkingssnelheid bij baseline twee subgroepen te ontstaan. In de discussie leidden de gecombineerde resultaten van beide onderzoeken ons tot de hypothese van een classificatie van etiologisch verschillende PR-proces-typen. Die

classificatie kon worden gerealiseerd op basis van fenomenologisch verschillende klinische profielen.

In het tweede deel bestudeerden we het niveau van persoonlijkheidsfunctioneren (NPF; AMPD, DSM-5, III, American Psychiatric Association, 2013) met de theoriegestuurde Differentiation Relatedness Scale (DRS; Diamond, Blatt, Stainer, & Kaslow, 1991) bij 330 studenten en 70 patiënten die het semi-gestructureerde ORI-interview (Object Relations Inventory; Blatt, Wein, Chevron, & Quinlan, 1979) hadden gedaan, een subgroep van de 240 onderzochte opgenomen patiënten. De ORI is een schriftelijk of mondeling interview waarin proefpersonen wordt gevraagd belangrijke anderen te beschrijven. Daarop wordt de DRS beoordeling gegeven. DRS is een beoordelingsschaal gebaseerd op Blatts 'polariteiten'-model (2008), dat persoonlijkheid definieert als het product van een progressieve dialectische interactie van de dimensies zelfdefinitie en interpersoonlijke verbondenheid. Eerst werden schriftelijke of mondelinge klinische beschrijvingen (n = 330 studenten, n = 70 opgenomen patiënten) van representaties van zichzelf en belangrijke anderen als moeder, vader en een leeftijdsgenoot (vb. partnerrelatie) beoordeeld. Vervolgens werden de beoordelingen geassocieerd met scores van depressie, dissociatieve kenmerken en relationeel functioneren bij jongvolwassenen (n = 330) en bij opgenomen patiënten met algemene psychopathologie (n = 240). Bij de patiënten werden deze beoordelingen bovendien aangevuld met geslacht, leeftijd, IQ, en rapportage van klachten (SCL-90), coping (UCL) en indices van persoonlijk functioneren (IPO, DEQ, ADP-IV).

De resultaten gaven aan dat de DRS vanwege een beperking in het bereik geen adequaat instrument was om het NPF te meten. De test bleek goed onderscheid te maken tussen niveaus van NPF bij ernstige persoonlijkheidsstoornissen, maar niet in een standaardpopulatie. De test gaf echter een duidelijke indicatie voor een mogelijke afkapwaarde voor voldoende persoonlijkheidsfunctioneren, wat inhoudt dat het NPF niet verweven is met symptomen of lijden. Bovendien bleek de DRS in een populatie van opgenomen patiënten met algemene psychopathologie ook niet de optimale keuze te zijn, omdat deze niet voldoende onderscheid maakte tussen niveaus van NPF. DRS werd wel niet beïnvloed door symptomen. Het bleek problemen te detecteren met beschikbaarheid van persoonlijkheid (de beperking van het beschikbare potentieel voor persoonlijkheidsgroei), denkstoornissen en onthechting, maar niet met de toegankelijkheid van persoonlijkheid (de moeilijke tijdelijke toegang tot de beschikbare persoonlijkheid door klinische last die het NPF beïnvloedt). In een daaropvolgend onderzoek naar persoonlijkheidsstoornissen bij 240 opgenomen patiënten met algemene psychopathologie werd het NPF gedefinieerd als de gemeenschappelijke factor van alle specifieke persoonlijkheidsstoornissen, in analogie met het Alternatieve Model van Persoonlijkheidsstoornissen (AMPS, DSM-5 IIII, 2013). Het hybride AMPS-model van persoonlijkheidspathologie werd vervolgens geëvalueerd met een bifactormodel, dat bestond uit specifieke trekken volgens de clusters van persoonlijjkheidsstoornissen (bizar, grillig, angstig) en een algemene persoonlijkheids-stoornis-factor, gPD, waarvan wordt

aangenomen dat het het NPF is. Het bifactormodel bleek het enige passende model. Het maakt onafhankelijke voorspelling van persoonlijkheidsstoornissen door de gPD of p-factor en de trekken mogelijk. Het model maakte zo een betekenisvol onderscheid mogelijk tussen borderline en narcistische persoonlijkheidsstoornissen. Bovendien voorspelde het model beide PS bijna perfect. Het niveau van persoonlijkheidsfunctioneren blijkt een interessant tweedimensionaal concept van zelf- en interpersoonlijk functioneren te zijn. Het NPF is onafhankelijk van de extremiteit van de trekken, met name internaliserende en externaliserende, die voornamelijk het aanpassingsvermogen bepalen. De dimensie gedachtestoornis is tot op zekere hoogte geassocieerd met de p-factor. Het aandeel van p (gPD) en van trekken verschilt echter enorm tussen beschrijvende persoonlijkheidsstoornissen. De gPD of p-factor lijkt samen te vallen met de borderline persoonlijkheidsstoornis. Maar zo'n hybride bifactormodel met één algemene en meerdere specifieke trekfactoren, verklaart geen persoonlijkheidsstoornissen met gedeactiveerde gehechtheid. De variantie van persoonlijkheidsstoornissen met gedeactiveerde gehechtheid wordt niet zo sterk voorspeld door het bifactormodel. Mogelijks leidt gedeactiveerde gehechtheid tot rapportagebias, met onderrapportage van klachten.

Samengevat bieden de huidige studies een voorbeeldmodel van evidence-based klinisch relevant onderzoek. De diepgaande analyse van één symptoom – psychomotorische vertraging – leidde tot indices voor een functionele classificatie van verschillende processen (veroudering, depressie, degeneratieve cognitieve stoornis) door verschillende symptoomprofielen. Een complementaire analyse van het dimensionele proces van persoonlijkheidsontwikkeling (zelf en interpersoonlijk functioneren) leidde tot een functionele classificatie van persoonlijkheidsstoornissen (beschikbaarheidstype, toegankelijkheidstype). De convergentie van beide richtingen in een geïntegreerde functionele benadering zou vervolgens bijdragen aan het overbruggen van de kloof tussen beschrijvende dimensies van signalen en symptomen en dynamische procesdimensies van affectieve spectrumstoornissen. Het zou kunnen bijdragen aan klinische bruikbaarheid dankzij de fenomenologische herkenbaarheid voor de clinicus. Het bestuderen van klinische fenomenen in verschillende populaties, in verschillende ontwikkelingsstadia over de levensloop, in verschillende specifieke contexten, met behulp van zowel kwantitatieve als kwalitatieve benaderingen, blijkt een noodzakelijke aanvulling te zijn op nosologische beschrijvingen van symptomen en classificaties en komt de klinische praktijk ten goede met het oog op precisietherapie. Het geeft ook een meer gedifferentieerd begrip van de aard en het ontwikkelingsproces van persoonlijkheidsstoornissen en draagt zo bij aan een innovatieve dynamische conceptualisering van stemmingsstoornissen.

Summary

This thesis is the natural outcome of my clinical interest in patients with affective spectrum disorders. Out of the many characteristics that typify these patients, two stood particularly out for me. The first was the psychomotor retardation (PR) that is so typical in this patient group. The second was the purported role of severity of personality disorders (PDs; also referred to as personality pathology) as a predictor of onset, course, and treatment response in these disorders. Unfortunately, as a clinician, both features belong to two different clinical perspectives that result in different treatment options. The first perspective is the –possibly multidimensional- symptom or descriptive classification perspective (sorting, defining principle), the second is the dynamic process dimensions perspective (developmental principle).

The central thesis of the present work is that it is possible to bridge the current communication gap between the two prominent perspectives by the development of a functional approach that connects classifications and processes. To do so, this research followed two directions: one from symptoms and classification to dimensions and processes and, inversely, one from dimensional processes to symptoms and classifications. Thus, the endeavor to bridge the gap was methodologically shaped into a two-way investigation.

In the first part, one symptom, psychomotor retardation (PR), was studied. We studied the symptom meticulously in a population of depressed elderly that were medication-free. This small-sample study served as an in vitro study. A sample of 40 depressed elderly was tested with a battery of various clinical, cognitive, and PR tests at four moments, within a three-months monotherapy period with escitalopram max. 20mg. That first study revealed that, with the different measures of retardation, different types of slowing could be distinguished, such as cognitive slowing, slowing of initiation of a movement, slowing by motivation, and motor slowing. Slowing in depressed elderly was predominantly evident in the execution of complex fine motor drawing tasks with combined cognitive and motor load. In the follow-up study, we were confronted with a significant dropout due to somatic comorbidities typical in depressed elderly. Still, of the 20 patients who fulfilled all assessments at the four moments, the depressive mood decreased first, while the psychomotor improvement came later and was delayed. In addition, two subgroups, based on processing speed scores at baseline, showed a different evolution. In the discussion, the combined results of both studies led us to the hypothesis of a functional classification of PR types referring to different etiological processes. That classification could be established based on phenomenologically differing clinical profiles, i.e., combinations of critical PR features.

In the second part, we studied the Level of Personality Functioning (LPF; AMPD, DSM-5, III) with the theory-driven Differentiation Relatedness Scale (DRS; Diamond, Blatt, Stainer, & Kaslow, 1991) in 330 students and in 70 patients who had done the semi-structured ORI interview, a subgroup of the 240 researched inpatients. The ORI is a written or oral interview in which subjects are asked to describe important others (Object Relations Inventory; Blatt, Wein, Chevron, & Quinlan, 1979) and on which the DRS is rated. DRS is a rating scale based on Blatt's 'polarities' model (2008), defining personality as the product of a progressive dialectic interaction of the dimensions of self-definition and interpersonal relatedness (n = 330 students, n = 70 inpatients). First, written or oral clinical descriptions of representations of self and significant others as mother, father, and a significant peer were rated. Then the ratings were associated with assessments of depression, dissociative features, and relational functioning in young adults (n = 330) and in inpatients with general psychopathology (n = 240). With the patients these ratings were additionally supplemented with assessments of gender, age, IQ, clinical distress (SCL-90), coping (UCL), and indices of personality functioning (IPO, DEQ, ADP-IV). The results indicated that, due to a restriction of range, the DRS was not an adequate instrument to measure the LPF. The test was found to differentiate well between levels of LPF in severe PDs, but not in a standard population. However, the test yielded a clear indication for a possible cut-off value for good enough personality functioning, which implies that the LPF is not associated with symptoms or suffering. Furthermore, in a population of inpatients with general psychopathology, the DRS appeared not to be the optimal choice either, as it did not sufficiently differentiate between levels of LPF. However, DRS was not influenced by symptoms. It appeared to detect problems with availability (the limitation of available potential for personality growth), thought disorders, and detachment, but not with the accessibility (the difficult temporary access to the personality by clinical distress influencing the LPF). In a subsequent investigation of PDs in 240 inpatients with general psychopathology, LPF was defined as the common factor of all specific PDs. The hybrid AMPD model of personality pathology was then evaluated with a bifactor model, which yielded specific traits of clusters A, B, and C (bizarre, erratic and anxious) and a general PD factor (gPD), assumed to be the LPF. The bifactor model appeared to be the only fitting model. It enables independent prediction by the gPD or p-factor and the traits. Thus, the model allowed for a meaningful distinction between borderline and narcissistic PDs. Moreover, the model predicted both PDs almost perfectly.

The level of personality functioning proves to be an interesting two-dimensional concept of differentiation and relatedness. LPF is independent of the extremity of the traits, especially internalizing and externalizing, which mainly determine adaptability. The thought disorder dimension is to some degree associated with the p-factor. However, the share of p (gPD) and of traits variance explained differ hugely between descriptive PDs. The gPD or p-factor seems to coincide completely with the borderline PD. However, such a hybrid bifactor model with one general and several specific trait factors, does not explain PDs with deactivating attachment. The variance of PDs with deactivating attachment is not

predicted by the bifactor model. Deactivating attachment could possibly lead to reporting bias, with underreporting of distress.

Taken together, the present studies provide an exemplary model of evidence-based clinically relevant research. The in-depth analysis of one symptom – PR – led to indices for a functional classification of different processes (aging, depression, degenerative cognitive disorder). A complementary analysis of the dimensional process of personality development (self-definition and relationship) led to a functional classification of PDs (availability type, accessibility type, susceptibility type). The convergence of both directions into an integrated functional approach may subsequently contribute to bridging the gap between descriptive nosological classification and developmental process approaches of affective spectrum disorders. It will also contribute to clinical utility thanks to the development of readily accessible evidence-based tools for the clinician. Studying clinical phenomena in different populations, in different stages of development across the life span, in different specific contexts, using both quantitative and qualitative approaches, turns out to be a necessary complement to nosological descriptions of symptoms and classifications and obviously benefits clinical practice. It also provides a more differentiated understanding of the nature and the developmental process of PDs and thus contributes to an innovative dynamic conceptualization of affective spectrum disorders.

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List of Abbreviations

AMPD	Alternative Model of Personality Disorders
DRS	Differentiation Relatedness Scale
IPO	Inventory of Personality Organization
SCL-90	Short Complaints List 90 items
DSM	Diagnostic Statistical Manual
UCL	Utrecht Coping List
BDI	Beck Depression Inventory
DISQ	Dissociation Questionnaire
DES	Dissociative Experiences Scale
PD	Personality Disorder
WAIS	Wechsler Adult Intelligence Scale
GIT	Groninger Intelligence Test
PM	Progressive Matrices
QRI	Quality of Relationships Inventory
WBPS	Psychological Wellbeing Scale
DEQ	Depressive Experience Scale
PAR	Paranoid
SZD	Schizoid

STD	Schizotypal
BDL	Borderline
NAR	Narcissistic
HIS	Histrionic
ASOC	Antisocial
AVD	Avoidant
DEP	Dependent
OCD	Obsessive- Compulsive
ORI	Object Relations Interview
P-factor	General factor of psychopathology
gPD	General factor of personality disorders
gP	General factor of Personality
g	General factor of intelligence
GDS	Geriatric Depression Scale
TMT	Trailmaking Test
WCST	Wisconsin Card Sorting Test
15W	15 words
LC	Line copying
FC	Figure copying
CFC	Complex Figure copying

SDST	Symbol Digit Substitution Test
PR	Psychomotor Retardation
CBGTC loop	Cortico-Basal Ganglia-Thalamo- Cortical Loop
CCC	Cortico-celebellar circuit

List of definitions

Psychomotor Retardation	A complex cognitive and motor slowing of movements, of speech, of expression and a common symptom of depression (and also a negative symptom of psychosis)
Level of Personality Functioning	The dynamic normal and abnormal developmental level of personality functioning (cfr. Blatt, 2008), also reflecting the momentary severity of dysfunction of personality disorders
Differentiation	Articulation and stabilization of a consolidated, individuated and integrated sense of self and other
Relatedness	Appreciation of mutual, empathically attuned, reciprocal, interpersonal relatedness
Representation	Imaginary image of the cognitive and affective characteristics of a person and also the relationship with that person; in psychoanalysis: the object relationship, in behavioral treatments: a cognitive-affective schema of a person
Interpersonal Matrix	The network of significant persons (by kinship or emotional) in a persons life and/or context

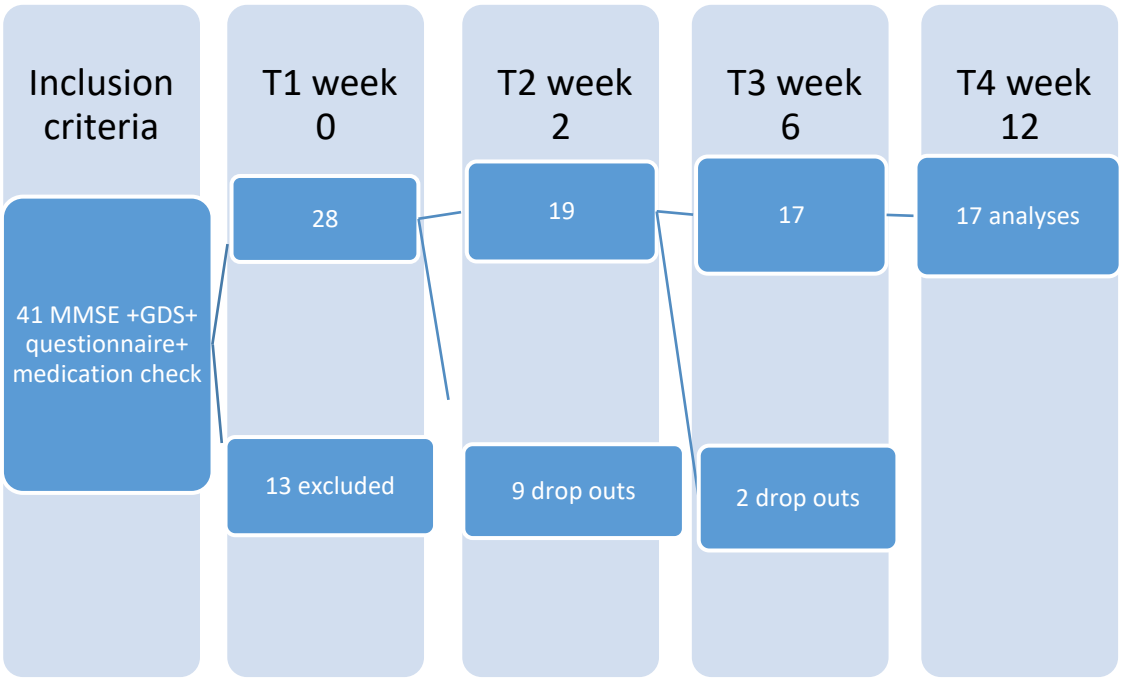
Phenomenology	The science of phenomena as distinct from that of the nature of being. An approach that concentrates on the study of consciousness and the objects of direct experience. For instance: how psychopathology is presented to the clinician in behavior, i.e., what a patient does, tells to feel, think, want, perceives, remembers, that can be registered, and is not directly interpreted.
Hitop	Hierarchical Taxonomy of Psychopathology
Trait	A distinguishing quality or characteristic, typically one belonging to a person. A genetic determined characteristic
State	the particular condition that someone or something is in at a specific time.
Adaptability	The possibility to adapt flexibly to problems, or changing circumstances, as a contrast to rigidity
Availability	The presence of cognitive/affective or personality level potential, the stable limitations on growth
Accessibility	The possibility to use the available potential of cognition/affect or developmental personality level

Psychological Vulnerability	The combination of problems with availability, accessibility, susceptibility. Also, the likeliness of mental health problems by nature (genetics) and nurture (adverse life events)
Severity	Double concept of quality of intense suffering and of poor prognosis
Susceptibility	Likeliness /proportion of being influenced by a pathological (biological) state, disease or momentary (environmental) condition
Mentalization	mentalization is the ability to understand the mental state – of oneself or others – that underlies overt behaviour. Mentalization can be seen as a form of imaginative mental activity that lets us perceive and interpret human behaviour in terms of intentional mental states (e.g., needs, desires, feelings, beliefs, goals, purposes, and reasons).
Clinical Distress	The acute dysregulation of baseline functioning by suffering because of complaints or disease
Thought Disorder	Formal thought disorder refers to an impaired capacity to sustain coherent discourse, and occurs in the patient's written or spoken language. Examples are jumping to conclusions, wrong attributions, chaotic speech, over certainty, lack of reflectiveness…

SUPPLEMENTS

Paper PR2

Beheydt, L. L., Schrijvers, D., Docx, L., Bouckaert, F., Hulstijn, W., & Sabbe, B. (2015b). Cognitive and psychomotor effects of three months of escitalopram treatment in elderly patients with major depressive disorder. Journal of Affective Disorders, 188, 47–52. https://doi.org/10.1016/j.jad.2015.08.041



Supplement Figure 1. Patients dropped out due to physical and psychiatric adverse events

Supplement Table 1. Effects were approximately the same in all types of analysis (pale gray). Standard mixed models analysis appeared too liberal in effects and was more reliable after multiple imputation, which produced extra memory group effects (dark grey).

MEASURE	**TIME EFFECT**		**TIME*GROUP EFFECT**		**GROUP EFFECT**	
	p	***η²***	***p***	***η²***	***p***	***η²***
GDS						
Completers	<0.001**	0.312	<0.001**	0.223	<0.001**	0.725
LOCF	<0.001**	0.216	0.001**	0.151	<0.001**	0.712
Mixed Models	<0.001**		<0.001**		0.008**	
Mixed Models significant at level 0.05 after Multiple Imputations (MI)	M.I.		M.I.		M.I.	
STAI						
Completers	0.001**	0.151	0.049*	0.081	<0.001**	0.509
LOCF	0.004**	0.120	0.055	0.069	<0.001**	0.475
Mixed Models	0.031*		0.185		<0.001**	
Mixed Models MI			M.I.		M.I.	

MEASURE	TIME EFFECT		TIME*GROUP EFFECT		GROUP EFFECT	
	p	η^2	p	η^2	p	η^2
SSRS						
Completers	0.850	0.017	0.832	0.019	0.325	0.065
LOCF	0.158	0.045	0.155	0.046	<0.001**	0.580
Mixed Models	0.891		0.526		0.039*	
Mixed Models MI						
WTOT						
Completers	0.001**	0.157	0.485	0.025	0.185	0.054
LOCF	<0.001**	0.165	0.866	0.006	0.037*	0.109
Mixed Models	0.007**		0.602		0.001**	
Mixed Models MI	M.I.				M.I.	
WRECALL						
Completers	0.001**	0.157	0.252	0.042	0.389	0.023
LOCF	<0.001**	0.170	0.432	0.024	0.182	0.048
Mixed Models	0.019*		0.729		0.02	
Mixed Models MI	M.I.					

MEASURE	TIME EFFECT		TIME*GROUP EFFECT		GROUP EFFECT	
	p	η^2	p	η^2	p	η^2
WRECOG						
Completers	0.018*	0.109	0.305	0.041	0.061	0.061
LOCF	0.004**	0.117	0.418	0.026	0.04*	0.115
Mixed Models	0.035*		0.600		<0.001**	
Mixed Models MI	M.I.				M.I.	
STROOP1						
Completers	0.542	0.020	0.773	0.009	0.005**	0.232
LOCF	0.460	0.022	0.395	0.026	0.003**	0.221
Mixed Models	0.526		0.709		<0.001**	
Mixed Models MI					M.I.	
STROOPINT						
Completers	0.113	0.061	0.195	0.046	0.012*	0.167
LOCF	0.113	0.061	0.195	0.046	0.012*	0.012
Mixed Models	0.104		0.428		<0.001**	
Mixed Models MI					M.I.	

MEASURE	TIME EFFECT		GROUP EFFECT		TIME*GROUP EFFECT	
	p	η^2	p	η^2	p	η^2
WCSTCAT						
Completers	0.089	0.062	0.202	0.044	0.002**	0.252
LOCF	0.079	0.066	0.249	0.041	0.003**	0.243
Mixed Models	0.509		0.314		<0.001**	
Mixed Models MI					M.I.	
CLIT						
Completers	0.135	0.058	0.185	0.050	0.003**	0.258
LOCF	0.041*	0.09	0.308	0.035	0.001**	0.270
Mixed Models	<0.001**		<0.001**		<0.001**	
Mixed Models MI					M.I.	
CLMT						
Completers	0.010*	0.164	0.364	0.027	<0.001**	0.189
LOCF	0.010*	0.164	0.364	0.027	0.009**	0.189
Mixed Models	<0.001**		<0.001**		<0.001**	
Mixed Models MI						

MEASURE	TIME EFFECT		GROUP EFFECT		TIME*GROUP EFFECT	
	p	η^2	p	η^2	p	η^2
CCIT						
Completers	0.133	0.057	0.630	0.015	0.019*	0.157
LOCF	0.010*	0.164	0.364	0.027	0.009**	0.189
Mixed Models	0.194		0.672		0.010*	
CCMT						
Completers	0.015*	0.108	0.822	0.008	0.017*	0.162
LOCF	0.015*	0.108	0.822	0.008	0.017*	0.162
Mixed Models	0.297		0.497		0.001**	
Mixed Models MI					M.I.	
SDSTIT						
Completers	0.646	0.010	0.127	0.067	0.469	0.016
LOCF	0.122	0.066	0.178	0.055	0.189	0.057
Mixed Models	0.015*		0.271		<0.001**	
Mixed Models MI						

MEASURE	TIME EFFECT		GROUP EFFECT		TIME*GROUP EFFECT	
	p	η^2	p	η^2	p	η^2
SDSTMT						
Completers	0.009**	0.134	0.446	0.025	0.026*	0.145
LOCF	0.019*	0.115	0.205	0.051	0.122	0.078
Mixed Models	0.500		0.240		<0.001**	
Mixed Models MI	M.I.					

Supplement Table 2

Comparison SDST HIGH/LOW scoring patient groups based on central tendency (median= 28; High≥28, Low <28)

Measure	df	F	p	η^2
CLIT				
Time	(3,21)	0.673	0.578	0.088
Time*Group	(3,21)	1.025	0.402	0.128
Group	(1,7)	3.452	3.452	0.330
T1-T2 Time	(1,7)	0.239	0.64	0.033
T1-T2 Time*Group	(1,7)	0.125	0.734	0.018
CLMT				
Time	(3,21)	6.863	0.002**	0.495
Time*Group	(3,21)	1.480	0.249	0.175
Group	(1,7)	16.137	0.005**	0.697
T1-T2 Time	(1,7)	7.427	0.30	0.515
T1-T2 Time*Group	(1,7)	0.238	0.64	0.033
CCIT				
Time	(3,27)	1.562	0.222	0.148
Time*Group	(3,27)	0.958	0.427	0.096
Group	(1,9)	0.999	0.346	0.099
T1-T2 Time	(1,9)	0.714	0.420	0.073
T1-T2 Time*Group	(1,9)	0.354	0.566	0.038

Measure	df	F	p	η^2
CCMT				
Time	(3,27)	1.188	0.333	0.117
Time*Group	(3,27)	1.034	0.393	0.103
Group	(1,9)	3.011	0.117	0.251
T1-T2 Time	(1,7)	4.587	0.061	0.338
T1-T2 T*Group	(1,7)	3.043	0.115	0.253
SDSTIT				
Time	(3,21)	1.042	0.394	0.130
Time*Group	(3,21)	0.612	0.614	0.080
Group	(1,7)	3.771	0.093	0.350
T1-T2 Time	(1,7)	0.083	0.781	0.012
T1-T2 Time*Group	(1,7)	1.083	0.333	0.134
SDSTMT				
Time	(3,21)	1.846	0.170	0.209
Time*Group	(3,21)	0.322	0.809	0.044
Group	(1,7)	5.041	0.06	0.419
T1-T2 Time	(1,7)	1.998	0.310	0.146
T1-T2 Time*Group	(1,7)	0.437	0.530	0.059

Comparison SDST Low scoring patient group and control group

Measure	Df	F	p	η^2
CLIT				
Time	(3,72)	0.624	0.602	0.025
Time*Group	(3,72)	3.366	0.023	0.123
Group	(1,24)	13.917	0.001**	0.367
T1-T2 Time	(1,24)	1.588	0.220	0.021
T1-T2 Time*Group	(1,24)	0.223	0.641	0.009
CLMT				
Time	(1.251, 30.013)	9.438	<0.001**	0.282
Time*Group	(1.251, 30.013)	3.867	0.05*	0.139
Group	(1,24)	26.07	<0.001**	0.521
T1-T2 Time	(1,24)	7.278	0.013*	0.013
T1-T2 Time*Group	(1,24)	3.174	0.087	0.117
CCIT				
Time	(1.587, 38.080)	0.638	0.499	0.026
Time*Group	(1.587, 38.080)	0.815	0.425	0.033
Group	(1,24)	5.394	0.029*	0.183
T1-T2 Time	(1,24)	0.000	0.986	0
T1-T2 Time*Group	(1,24)	1.129	0.299	0.045

Measure	Df	F	p	η^2
CCMT				
Time	(2,325, 55.803)	7.908	0.001**	0.248
Time*Group	(2,325, 55.803)	3.018	0.049*	0.112
Group	(1,24)	13.112	0.001**	0.353
T1-T2 Time	(1,24)	16.093	0.001**	0.401
T1-T2 Time*Group	(1,24)	6.877	0.015*	0.223
SDSTIT				
Time	(1.326, 31.822)	0.969	0.357	0.039
Time*Group	(1.326, 31.822)	3.503	0.059	0.127
Group	(1,24)	1.704	0.204	0.066
T1-T2 Time	(1,24)	0.132	0.720	0.005
T1-T2 Time*Group	(1,24)	4.122	0.054	0.147
SDSTMT				
Time	(1.909, 45.809)	6.081	0.005**	0.202
Time*Group	(1.909, 45.809)	1.415	0.253	0.056
Group	(1.24)	16.281	<0.001**	0.404
T1-T2 Time	(1,24)	1.399	0.248	0.055
T1-T2 Time*Group	(1,24)	0.001	0.978	0.00

Comparison SDST High Group and Controls

Measure	df	F	p	η^2
CLIT				
Time	(1.569, 32.94)	4.375	0.028*	0.172
Time*Group	(1.569, 32.94)	0.742	0.453	0.034
Group	(1,21)	0.366	0.366	0.039
T1-T2 Time	(1,21)	3.153	0.09	0.131
T1-T2 Time*Group	(1,21)	0.782	0.387	0.036
CLMT				
Time	(3,78)	6.692	<0.001**	0.205
Time*Group	(3,78)	1.493	0.223	0.054
Group	(1,26)	0.614	0.441	0.441
T1-T2 Time	(1,26)	5.319	0.029*	0.170
T1-T2 Time*Group	(1,26)	4.084	0.054	0.136
CCIT				
Time	(2.370, 61.623)	3.018	0.048*	0.104
Time*Group	(2.370, 61.623)	1.05	0.365	0.039
Group	(1,26)	3.16	0.087	0.108
T1-T2 Time	(1,26)	0.819	0.374	0.031
T1-T2 Time*Group	(1,26)	0.286	0.597	0.006

Measure	df	F	p	η^2
CCMT				
Time	(2.264, 58.869)	1.775	0.174	0.064
Time*Group	(2.264, 58.869)	0.815	0.461	0.03
Group	(1,26)	0.143	0.143	0.08
T1-T2 Time	(1,26)	0.332	0.332	0.036
T1-T2 Time*Group	(1,26)	0.119	0.119	0.091
SDSTIT				
Time	(2.095, 52.377)	2.027	0.140	0.075
Time*Group	(2.095, 52.377)	1.090	0.346	0.042
Group	(1,25)	0.137	0.714	0.005
T1-T2 Time	(1,25)	6.627	0.016	0.21
T1-T2 Time*Group	(1,25)	0.744	0.397	0.029
SDSTMT				
Time	(2.171, 54,284)	2.727	0.07	0.098
Time*Group	(2.171, 54.284)	1.530	0.225	0.225
Group	(1,25)	0.136	0.715	0.005
T1-T2 Time	(1,25)	6.942	0.014	0.217
T1-T2 Time*Group	(1,25)	3.192	0.086	0.113

Supplement Table 3 Effects of GLM repeated measures after imputation with last observation carried forward

Measure	df	F	p	η^2
GDS				
Time	(2.214,84.138)	10.463	<0.001**	0.216
Time*Group	(2.214, 84.138)	6.751	0.001**	0.151
Group	(1,38)	93.763	<0.001**	0.712
SRRS				
Time	(2.614, 99.349)	1.809	0.158	0.045
Time*Group	(2.614, 99.349)	1.826	0.155	0.046
Group	(1,38)	52.527	<0.001**	0.580
WTOT				
Time	(3, 114)	7.531	<0.001**	0.165
Time*Group	(3,114)	0.244	0.866	0.006
Group	(1,38)	4.651	0.037*	0.109
WRECALL				
Time	(3,111)	7.563	<0.001**	0.170
Time*Group	(3,111)	0.924	0.432	0.024
Group	(1,37)	1.851	0.182	0.048

Measure	df	F	p	η^2
WRECOG				
Time	(3,105)	4.658	0.004**	0.117
Time*Group	(3,105)	0.952	0.418	0.026
Group	(1,35)	4.548	0.04*	0.115
STROOP1				
Time	(2.091, 73.199)	0.796	0.460	0.022
Time*Group	(2.091, 73.199)	0.948	0.395	0.026
Group	(1,35)	9.938	0.003**	0.221
STROOPINT				
Time	(1.901, 66.521)	2.280	0.113	0.061
Time*Group	(1.901, 66.521)	1.680	0.195	0.046
Group	(1,35)	7.004	0.012*	0.012
WCSTNCORR				
Time	(3,99)	1,600	0.194	0.046
Time*Group	(3,99)	2.059	0.121	0.059
Group	(1,33)	7.687	0.009**	0.189

Measure	df	F	p	η^2
SDSTNCORR				
Time	(2.264, 65.657)	8.215	<0.001**	0.221
Time*Group	(2.264, 65.657)	0.753	0.490	0.025
Group	(1,29)	9.615	0.004	0.249
SDSTIT				
Time	(2.309, 69.260)	2.106	0.122	0.066
Time*Group	(2.309, 69.260)	1.743	0.178	0.055
Group	(1,30)	1.806	0.189	0.057
SDSTMT				
Time	(2.373, 71.181)	3.916	0.019*	0.115
Time*Group	(2.373, 71.181)	1.602	0.205	0.051
Group	(1,30)	2.533	0.122	0.078
CLIT				
Time	(2.132, 70.363)	3.261	0.041*	0.09
Time*Group	(2.132, 70.363)	1.204	0.308	0.035
Group	(1,33)	12.236	0.001**	0.270
CLMT				
Time	(1.273, 42.002)	6.479	0.010*	0.164
Time*Group	(1.273, 42.002)	0.927	0.364	0.027
Group	(1,33)	7.674	0.009**	0.189

Measure	df	F	p	η^2
CCIT				
Time	(1.273, 42.002)	6.479	0.010*	0.164
Time*Group	(1.273, 42.002)	0.927	0.364	0.027
Group	(1,33)	7.674	0.009**	0.189
CCMT				
Time	(2.494, 82.315)	3.989	0.015*	0.108
Time*Group	(2.494, 82.315)	0.255	0.822	0.008
Group	(1,33)	6.369	0.017*	0.162
CLIT T1-T2				
Time	(1,33)	4.839	0.035*	0.128
Time*Group	(1,33)	2.516	0.122	0.071
CLMT T1-T2				
Time	(1,33)	4.153	0.05*	0.112
Time*Group	(1,33)	0.478	0.494	0.014

Measure	df	F	p	η^2
CCIT T1-T2				
Time	(1,33)	4.153	0.05*	0.112
Time*Group	(1,33)	0.478	0.494	0.014
CCMT T1-T2				
Time	(1,33)	6.658	0.015*	0.168
Time*Group	(1,33)	0.558	0.460	0.017

Paper LPF1

Beheydt, L. L., Van Liefferinge, D., Lowyck, B., Schrijvers, D., Sabbe, B., & Luyten, P. (2020). Levels of relatedness and self-definition in young adults: Associations with psychopathology and interpersonal functioning. *Psychoanalytic Psychology, 37*(3), 232–240. https://doi.org/10.1037/pap0000297

Table S1 *The 10 levels of the Differentiation-Relatedness Scale (see also Huprich, Auerbach, Porcerelli, & Bupp, 2016)*

Level	Comments
1. Self/other boundary compromise (physically)	*Basic physical cohesion/integrity of representations is compromised*
2. Self/other boundary confusion (intellectual, affective)	*Affective/intellectual boundaries are confused, fused, or compromised*
3. Self/other mirroring	*Consolidation and stabilization of representations based on mirroring*
4. Self/other idealization or denigration	*Consolidation and stabilization of representations based on unitary, unmodulated idealization or denigration*
5. Semi-differentiation	*Tenuous, semi-differentiated consolidation of representations achieved through primitive splitting and/or rigid adherence to concrete properties to achieve a tenuous cohesion*
6. Emergent, ambivalent constancy (cohesion) and an emergent sense of relatedness	*Emergent differentiated, constant, integrated representation of self and other*
7. Consolidated, constant (stable) self and others in unilateral relationship	*Increasing tolerance for ambiguity*
8. Cohesive, individuated, empathically related self and other	*Representations of self and others as empathically interrelated*
9. Reciprocally related, integrative unfolding self and other	*Representations of self and other in reciprocal and mutually facilitating interactions*
10. Integrative, creative constructions of self and other in empathically and reciprocally attuned relationships	*Reflectively constructed, integrated representations of self and others in reciprocal and mutual relationships*

Supplement S2: Distribution of DRS in young adult students

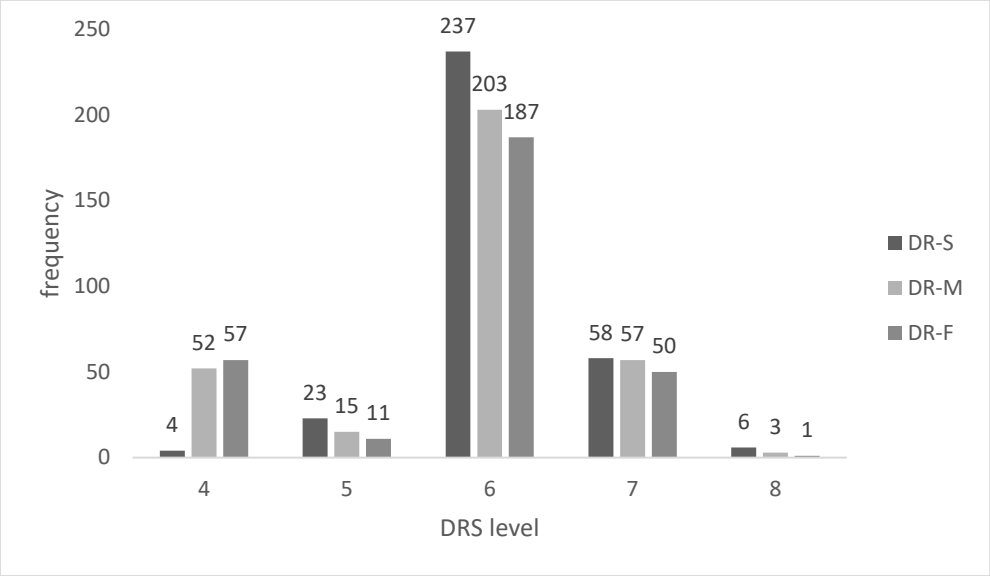


Figure S2

Distribution of levels of DR-S (DR based on Self representation). DR-M (DR based on Mother representation) and DR-F (DR based on Father representation) in a sample of young adult students

Table S2

Percentages of DRS levels

DRS %	4	5	6	7	8	Mean	Median	SD
DRS	1.2	6.9	70.7	17.3	1.8	6.12	6	0.597
DRM	15.5	4.5	60.6	17	0.9	5.83	6	0.927
DRF	17	3.3	55.8	14.9	0.3	5.76	6	0.951

Note. In the first year psychology students. 90% of the students show a DR-S level above 6. frequencies of high levels of DR-M and DR-F are more modest. respectively 78.5 and 71% . According to former studies in community samples. we expected 15% suffering of psychopathology. As expected in a normal sample, a restriction of range is shown. No students were found with a DR-S of 1. 2 or 3 but also high levels of 9 or 10 were not present. The possibility that lower representations of mother and father reflect real problems in their interactions deserves further investigation.

Table S3

Testing for linearity versus deviance of linearity in relationships between DRS and symptoms, psychopathology dimensions or relational functioning measures: linearity contrast tests with F-tests in ANOVA and effect sizes.

Interaction		***df***	***F***	***p***	***R^2(linear)***	***η^2***
DR-S*DID_sev	linear	1	5.225	.023	.015	.015
	dev linear	5	3.488	.004		
	combined	6	3.777	.001*		.066
DR-S*DES	linear	1	7.861	.005	.022	.022
	dev linear	5	4.769	.000**		
	combined	6	5.285	.000**		.090
DR-S*DEQ_dep	linear	1	.140	.708	.000	.044
	dev linear	5	.805	.547		
	combined	6	.694	.654		.013
DR-S*DEQ_sc	linear	1	1.052	.306	.003	.003
	dev linear	5	1.904	.093		
	combined	6	1.762	.106		.032
DR-S*QRI_s	linear	1	.298	.586	.000	.000
	dev linear	5	2.930	.013		
	combined	6	2.491	.023		.044
DR-S*QRI_c	linear	1	.911	.341	.003	.002
	dev linear	5	4.215	.001*		
	combined	6	3.665	.002		.064
DR-S*QRI_d	linear	1	.622	.431	.002	.002
	dev linear	5	1.657	.144		
	combined	6	1.485	.183		.027
DR-M*DID_sev	linear	1	7.572	.006	.023	.023
	dev linear	3	2.562	.055		
	combined	6	3.815	.005		.046
DR-M*DES	linear	1	3.428	.065	.010	.010
	dev linear	3	2.124	.097		
	combined	6	2.450	.046		.029
DR-M*DEQ_dep	linear	1	.004	.949	.000	.000
	dev linear	3	.710	.546		
	combined	6	.534	.711		.007
DR-M*DEQ_sc	linear	1	.586	.445	.002	.002
	dev linear	3	2.911	.035		
	combined	6	2.330	.056		.028
DR-M*QRI_s	linear	1	.180	.672	.001	.001
	dev linear	3	4.041	.008		

Interaction		*df*	*F*	*p*	R^2*(linear)*	η^2
	combined	6	3.076	.017		.037
DR-M*QRI_c	linear	1	.043	.836	.000	.000
	dev linear	3	2.907	.035		
	combined	6	2.191	.070		.027
DR-M*QRI_d	linear	1	2.121	.146	.006	.006
	dev linear	3	3.196	.024		
	combined	6	2.927	.021		.035
DR-F*DID_sev	linear	1	3.464	.064	.011	.011
	dev linear	3	.465	.707		
	combined	6	1.214	.305		.016
DR-F*DES	linear	1	6.970	.009	.023	.023
	dev linear	3	.184	.907		
	combined	6	1.881	.114		.024
DR-F*DEQ_dep	linear	1	.428	.513	.001	.001
	dev linear	3	1.239	.296		
	combined	6	1.036	.389		.014
DR-F*DEQ_sc	linear	1	.000	.994	.000	.000
	dev linear	3	1.287	.279		
	combined	6	.965	.427		.013
DR-F*QRI_s	linear	1	.437	.509	.001	.001
	dev linear	3	1.012	.388		
	combined	6	.868	.483		.012
DR-F*QRI_c	linear	1	.432	.512	.001	.001
	dev linear	3	1.159	.326		
	combined	6	.977	.420		.013
DR-F*QRI_d	linear	1	.934	.335	.003	.003
	dev linear	3	1.434	.233		
	combined	6	1.309	.267		.017

Note. DR-S (DR-self); DR-M (DR-mother); DR-F (DR-father); dev. linear (deviance from linearity); DID (Diagnostic Inventory for Depression), DID_sev (severity of depression); DES (Dissociative Experiences Scale, frequency); DEQ (Depressive Experience Scale) DEQ_dep (dependency); DEQ_sc (self-criticism); QRI (Quality of Relationships Inventory), QRI_s (support in relationships); QRI_c (conflict in relationships); QRI_d (depth in relationships).

Small effects are marked in grey. Moderate effects are underlined. *$p \leq .05$, **$p \leq .01$ after Bonferroni correction.Table S4

Pearson and Kendall's tau zero-order correlations of DRS subcales with depressive and dissociative symptoms, with psychopathology dimensions and with interpersonal functioning

	Parametric: Pearson *r*			Non-parametric : Kendall's τ		
	DR-S (N=330)	DR-M (N=325)	DR-F (N=302)	DR-S	DR-M	DR-F
DID_sev	-.123*	-.150**	-.107	-.062	-.080	-.034
DES	-.149**	-.101	-.150**	-.078	-.067	-.106*
DEQ_sc	-.056	-.042	0	-.039	-.006	.038
DEQ_dep	-.021	.004	.038	-.005	.0	.034
QRI_s	.030	.023	.038	.044	.014	.029
QRI_c	-.051	-.011	.038	-.062	.016	.045
QRI_d	-.043	-.08	-.056	-.029	-.083	-.058

Note. DRS (Differentiation-Relatedness Scale); DR-S (DRS in descriptions of the self); DR-M (DRS in descriptions of mother); DR-F (DRS in descriptions of father); DID (Diagnostic Inventory for Depression), DID_sev (severity of depression); DES (Dissociative Experiences Scale, frequency); DEQ (Depressive Experiences Questionnaire), DEQ_sc (self-criticism), DEQ_dep (dependency); QRI (Quality of Relationships Inventory), QRI_s (support), QRI_c (conflict), QRI_d (depth).

Table S5 *Statistics of change of a linear regression model with addition of a binomial DRS term as independent variable in the regression (quadratic model) of DRS on features of psychopathology and interpersonal functioning. Non-parametric analysis with Kruskal-Wallis tests in grey.*

DR-S	*Statistics of change*					*Coefficients*	
Linear and Quadratic model	R^2_{adjust}	R^2_{change}	F_{change}	*(df 1, df2)*	*p*	*β*	*t*
Non-parametric analysis with Kruskal- Wallis test	$\varepsilon^2=\eta^2$		*H*	*df*	*p*		
DR-S*didsev	.012	.015	4.933	(1,323)	.027	-.123	-2.221
DR-S^2*didsev	.041	.032	10.78	(1,322)	.001*	2.028	3.283
Non-parametric DRS*didsev	.043		14.13	6	.028		
DR-S*des	.026	.029	9.688	(1,322)	.002*	-.171	-3.113
DR-S^2*des	.050	.027	9.20	(1,322)	.003*	1.868	3.034
Non-parametric DR-S*des	.049		16.17	6	.013		
DR-S*dep	-.003	.000	.092	(1,323)	.762	.017	.303
DR-S^2*dep	.001	.007	2.36	(1,322)	.126	.968	1.535
Non-parametric DR-S*dep	.015		4.846	6	.564		
DR-S*SC	.000	.003	1.131	(1,323)	.288	-.059	-1.064
DR-S^2*SC	.025	.028	9.30	(1,322)	.002*	1.899	3.049
Non-parametric DR-S*SC	.032		10.65	6	.100		
DR-S*qris	-.003	.000	.086	(1,323)	.769	.016	.293
DR-S^2*qris	.034	.040	13.44	(1,322)	.000**	-2.273	-3.665
Non-parametric DR-S*qris	.036		11.97	6	.063		
DR-S*qric	.002	.005	1.630	(1,323)	.203	-.071	-1.277
DR-S^2*qric	.056	.056	19.37	(1,322)	.000**	2.698	4.401
Non-parametric DR-S*qric	.050		16.57	6	.011		
DR-S*qrid	-.003	.000	.094	(1,323)	.760	-.017	-.306
DR-S^2*qrid	.014	.020	6.50	(1,322)	.011	-1.597	-2.549
Non-parametric	.023		7.48	6	.278		

DR-S*qrid							
DR-M	Statistics of change					*Coefficients*	
Linear and Quadratic model	R^2_{adjust}	R^2_{change}	F_{change}	*(df 1, df2)*	*p*	*β*	*t*
Non-parametric analysis with Kruskal- Wallis test	$\varepsilon^2=\eta^2$		*H*	*df*	*p*		
DR-M*didsev	.017	.020	6.612	(1,318)	.011	-.143	-2.571
DR-M²*didsev	.015	.001	.374	(1,317)	.541	.312	.612
Non-parametric DR-M*didsev	.040		13.04	4	.011		
DR-M*des	.006	.009	2.983	(1,324)	.085	-.096	-1.727
DR-M²*des	.003	.000	.134	(1,323)	.715	-186	-.365
Non-parametric DR-M*des	.030		9.72	4	.045		
DR-M*dep	-.003	.000	.114	(1, 318)	.735	.019	.338
DR-M²*dep	-.001	.005	1.519	(1, 317)	.219	.633	.219
Non-parametric DR-M*dep	.012		3.90	4	.420		
DR-M*SC	-.002	.002	.509	(1, 318)	.476	-.040	-.713
DR-M²*SC	.002	.007	2.150	(1,317)	.144	-.787	-1.535
Non-parametric DR-M*SC	.023		7.47	4	.113		
DR-M*qris	-.003	.000	.056	(1,318)	.814	.013	.236
DR-M²*qris	.000	.006	1.999	(1,317)	.158	-.726	-1.414
Non-parametric DR-M*qris	.029		9.42	4	.052		
DR-M*qric	-.003	.000	.038	(1,318)	.845	-.011	-.196
DR-M²*qric	.003	.009	2.828	(1,317)	.094	-.868	.091
Non-parametric DR-M*qric	.031		10.30	4	.036		
DR-M*qrid	.001	.004	1.371	(1,318)	.243	-.066	-1.171
DR-M²*qrid	.000	.002	.624	(1,317)	.430	-.406	.790
Non-parametric DR-M*qrid	.046		15.05	4	.005*		

DR-F	*Statistics of change*					*Coefficients*	
Linear and Quadratic model	R^2_{adjust}	R^2_{change}	F_{change}	*(df 1, df2)*	*p*	*β*	*t*
Non-parametric analysis with Kruskal- Wallis test	$\varepsilon^2=\eta^2$		*H*	*df*	*p*		
DR-F*didsev	.006	.009	2.730	(1,295)	.100	-.096	-1.652
DR-F2*didsev	.006	.004	1.077	(1,294)	.300	.594	1.038
Non-parametric DR-F*didsev	.003		5.19	4	.268		
DR-F*des	.018	.021	6.429	(1, 300)	.012	-.145	-2.536
DR-F2*des	.015	.000	.097	(1,299)	.756	-.177	-.311
Non-parametric DR-F*des	.024		7.73	4	.102		
DR-F*dep	.000	.003	.879	(1,295)	.349	.055	.938
DR-F2*dep	.006	.010	3.050	(1,294)	.082	.999	1.746
Non-parametric DR-F*dep	.014		4.55	4	.337		
DR-F*SC	-.003	.000	.008	(1,295)	.930	.005	.088
DR-F2*SC	.005	.012	3.432	(1,294)	.065	1.060	1.853
Non-parametric DR-F*SC	.017		5.47	4	242		
DR-F*qris	-.003	.001	.240	(1,295)	.624	.029	.490
DR-F2*qris	.000	.006	1.816	(1,294)	.179	-.773	-1.348
Non-parametric DR-F*qris	.005		1.95	4	.744		
DR-F*qric	-.002	.002	.495	(1,295)	.482	.041	.703
DR-F2*qric	-.003	.002	.607	(1,294)	.436	.448	.779
Non-parametric DR-F*qric	.012		4.07	4	.397		
DR-F*qrid	-.002	.002	.468	(1,295)	.495	-.040	-.684
DR-F2*qrid	-.005	.000	.104	(1,294)	.747	-.323	.747
Non-parametric DR-F*qrid	.019		6.10	4	.192		

Note. DR-S (DR-self); DR-M (DR-mother); DR-F (DR-father); DID = Diagnostic Inventory for Depression; DES = Dissociative Experiences Scale; didsev (severity of depression); des (dissociative experiences); dep (dependency);SC (self-criticism); qris (support of relationships); qric (conflict in relationships); qrid (depth in relationships). *$p < .05$, **$p < .01$ after Bonferroni correction.

Table S6

Pearson Correlations of Depression features as measured with DID and psychopathology dimensions as measured with DEQ. Intermediate effects are underlined, small effects in italics

	DEPENDENCY	SELF-CRITICISM
DID-severity	*.240***	.527***
DID-frequency	*.239***	.454***
DID-psychosocial	*.233***	.495***
DID-quality of life	*.162***	*.298***
DID-weight	*.154***	*.208***
DID-sleep	.008	*.142**
DID-psychomotor	*.109**	.333***
DID-fatigue	*.183***	.328***
DID-guilt	.338***	*.261***
DID-concentration	*.206***	*.195***
DID-suicide thoughts	*.147***	.346***
DID-symptoms (number)	*.299***	.461***
DID-diagnosis	*.219***	.422**

Note. *p<.05. **p<.01 ***p<.001

Paper LPF 2

Beheydt, L., Schrijvers, D., Sabbe, B., Jansen, B., De Grave, C., & Luyten, P. (2020). DSM-5 Assessments of the Level of Personality Functioning: Intrapersonal and Interpersonal Functioning. Psychiatry, 83(1), 84-93. https://doi.org/10.1080/00332747.2019.1650411

DSM-IV-TR Diagnosis	N=71
DSM-IV-TR Diagnosis Axis I	N=71
Affective Disorder	56
Anxiety Disorder	5
Substance Abuse Disorder	4
Psychotic Disorder	4
Adjustment Disorder	1
Disorder due to CVA	1 [1]
DSM-IV-TR Diagnosis Axis II (SCID-II)	
Cluster A	
Paranoid PD	2
Schizoid PD	4
Cluster B	
Borderline PD	15
Narcissistic PD	7
Histrionic PD	5
Antisocial PD	5
Cluster C	
Avoidant PD	14
Dependent PD	8
Obsessive Compulsive PD	7
Not Otherwise Specified PD	7

[1] Note This patient was excluded. PD = Personality Disorder.

Table S1 Overview of the DSM-IV-TR diagnoses of the patients

Table S2

Correlations between DRS and IPO measures

	DRS_ORI				IPO			*M*	*SD*
	DR-M	DR-F	DR-P	DR-S	IPO-ID	IPO-PD	IPO-RT		
DR-M		.54**	.20	.34**	.10	-.02	.15	5.18	1.16
DR-F			.27*	.34**	.03	-.07	-.04	5.34	1.30
DR-P				.34**	-.19	-.24*	-.23	5.25	1.14
DR-S					-.11	-.12	-.09	5.29	1.06
IPO-ID						.66**	.54**	58.34	11.56
IPO-PD							.58	38.84	9.66
IPO-RT								41.43	1.38

DR_mother = DR-M, DR_father = DR-F, DR_peer = DR-P, DR_self = DR-S, *IPO_Identity Diffusion = IPO-ID, IPO_Primitive Defense = IPO-PD, IPO_disturbed Reality Testing = IPO-RT*

*p< .05 **p< .01

Table S3

Demographic features and descriptive statistics: medians and ranges, correlations of DRS (Kendall's r) and IPO (Pearson r) with age and educational level and differences between gender-groups as assessed with t-tests.

		Gender	**Age**	**Level of education**	**IQ**
N = 68	Median, range	50.7% male	*M* = 36.85 (11.99), 17-57	*M* = 3 (1.04)	*M* = 107.07 86-126
DR-S, N = 66	6, 2-8	*t* = .369, *p* = .71	.026, *p* = .781	.028, *p* = .792	-.030, *p* = .749
DR-M, N = 68	5.5, 2-7	*t* = .510, *p* = .61	-.063, *p* = .577	-.120, *p* = .340	.001, *p* = .992
DR-F, N = 68	5.5, 2-7	*t* = -.413, *p* = .68	-.061, *p* = .512	-.055, *p* = .600	-.071, *p* = .449
DR-P, N = 47	5, 2-8	*t* = .936, *p* = .36	-.035, *p* = .714	-.099, *p* = .346	-.104, *p* = .272
IPO-PD, N = 65	38, 16-70	*t* = -1.092, *p* = .279	-.300, *p* = .016*	-.135, *p* = .286	-.143, *p* = .261
IPO-ID, N = 65	58, 27-100	*t* = -.977, *p* = .332	-.284, *p* = .023*	-.136, *p* = .282	-.203, *p* = .107
IPO-RT, N = 65	40, 22-92	*t* = -.990, *p* = .326	-.312, *p* = .012*	-.124, *p* = .330	-.234, *p* = .063

DR_mother = DR-M, DR_father = DR-F, DR_peer = DR-P, DR_self = DR-S, IPO_Identity Diffusion = IPO-ID, IPO_Primitive Defense = IPO-PD, IPO_disturbed reality testing = IPO-RT
*p < .05 **p < .01

Table S4

Correlations of the DRS_ORI and the IPO with clinical measures, total clinical distress as measured with the SCL-90, depression as measured with the BDI and dissociative features as measured with the DIS-Q

	DRS				**IPO**				
	DR-M	DR-F	DR-P	DR-S	IPO-ID	IPO-PD	IPO-RT	M	SD
SCL-90	-.078	-.113	-.117	-.109	.617**	.574**	.573**	2.572	.71
BDI	-.031	-.041	-.304	-.155	.558**	.497**	.436**	2.0737	.56
DISQ	-.003	-.120	-.214	-.092	.767**	.717**	.786**	1.9992	.54

SCL-90 = total score of clinical distress, BDI = total depression score on the Beck Depression Inventory, DISQ = dissociation Questionnaire total score.

*P < .05 **p < .01

Paper LPF 3

Beheydt, L., Jansen, B., De Grave, C., & Luyten, P. (in review). Bifactor modeling suggests different functions of general and specific factors in personality disorders.

Supplement Table 1. *Zero-order correlations of the specific factors of the bifactor model with external variables, and their partial correlations, controlled for the general factor*

Variable	r CLUS A (r controlled for p)	r CLUS B (r controlled for p)	r CLUS C (r controlled for p)
Age	-,236**	-,119	-,167*
	-171**	**.132***	.031
IQ	.055	-,065	,10
	-.05	-.025	.073
SCL -90	,602***	.527***	,.636***
	.061	**-.197****	.123
BDI –II	,513***	.426***	,563***
	.043	**-.230*****	**.172****
DIS-Q	,614***	.587***	.617***
	.045	-.056	-.036
UCL	,140*	,164*	.114
	.009	.069	-.095
IPO-PD	.087	.045	.017
	.111	.005	-.093
IPO-ID	.125	.066	.074
	.107	-.042	-.038
IPO-RT	.123	.067	.047
	.131*	-.011	-.091
QRI-S	.257	.255	.252
	.021	.014	-.023
QRI-C	-.058	-.051	-.091
	.024	.044	-.068
QRI-D	.173	.182	.197
	-.019	.002	.032

Dependency	.108	.076	.124
	.013	-.071	.056
Self-Criticism	.077	.064	.023
	.071	.040	-.091

Note. IQ = Intelligence Quotient; SCL-90 = Symptom Checklist-90; BDI = Beck Depression Inventory; DIS-Q = Dissociation Questionnaire; UCL = Utrechtse Coping Lijst; IPO = Inventory of Personality Organization, IPO-PD = primitive defense, IPO-ID = identity problems, IPO-RT = reality testing disturbances; QRI = Quality of Relationships Inventory, QRI-S = support, QRI-C = conflict, QRI-D = depth. Bonferroni correction: p<.05 = .05/14=.004, p<.01 = .01/14 = .0007. Italics: partial correlations; bold: significant after Bonferroni correction

List of Tables

List of Figures

THE LEVEL OF PERSONALITY FUNCTIONING

Figure 1. Functional types of personality disorders

126

Curriculum Vitae

Personal Data

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Education

Master in Clinical Psychology, major clinical psychology, minor theoretical psychology (language psychology)	Catholic University of Louvain, Leuven, 1996
PhD Student	University of Antwerp 2014-2021

Professional Certified Training

Certificate of specialisation in Family Therapy	University Hospitals of Catholic University of Louvain, UZ Leuven, 1999
Certificate Post-Academic training (PAV) Clinical Assessment	Inter University Training of the Catholic University Louvain, Ghent University, & VUB Brussels, 2005
Training in Residential Psychotherapy (multidisciplinary)	Delft (Holland), 2008

"What is it to be a researcher in the 21th century?"	Inter University Training of the Catholic University Louvain, Ghent University, & VUB Brussels 2014-2015
Training as supervisor in de psychotherapy	Training and ongoing Learning supervision, Uantwerpen, 2020-2021

Professional career

Clinical function

PC Sint-Norbertushuis Duffel/ PZ Duffel/ UPC Duffel	Psychologist, psychotherapist (100%)	1997-2018
PC Bethaniënhuis Zoersel, Rivendel studio	Teamsupervisor	2006-2007
PC Sint Amedeus Mortsel, De Link	Teamsupervisor	2009-2012
Similes en Fonds Geert Noël	Skills trainer for families	2011-2014
Private Practice	Psychotherapist, supervisor, consultant	2009-2014 2016-2021
UPC Duffel	Staff member Clinical Assessment	2019-2021

Teaching and training

Training and in company education of bachelors and masters in Psychology in psychotherapy and assessment,	University Psychiatric Center, Duffel,	2010-2021
Guest College Dialectical Behavior Therapy, Faculty of Health and Medicine, Antwerp University	University of Antwerp	2011
Practicum Neuroscience, 3rd bachelor year in Medicine	University of Antwerp	2014
Thesis advisor of Bachelor thesis (Laura Lemmens) 'Cognitive Remediation' and 'The association of coping and the Level of Personality Functioning' Thomas More (Thomas Tack et al.,)	Thomas More College, Antwerp	2012, 2020
Trainer: dealing with parents with psychiatric problems in family counseling	ITA, Brasschaat Sint Vincentius Halen-Zelem	2012 2013

Training in integrated psychosocial treatment of patients with affective spectrum disorders and personality disorders	UPC Duffel, Treatment Programme of affective spectrum Disorders	October 2018, January 2019
Supervision of professional psychotherapists in UPC Duffel	UPC Duffel	2020-2021

Publications

Schrijvers, D., Destoop, M., Beheydt, L., De Bruijn, E., Hulstijn, W., Sabbe, B. (2012). - Research in the Collaborative Antwerp Psychiatric Research Institute on cognitive and psychomotor dysfunctions in mood disorders in: *Acta psychiatrica Belgica* - ISSN 0300-8967 – 112(4), p. 25-29.

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Proceedings

Beheydt, L., Sabbe, B., De Picker, L., Goetschalckx, J., Daelemans, W. (2017). Computational language analysis for assessment of schizophrenia. *Proceedings 6th International Conference on Cognitive and Behavioral Psychology* (CBP) (2017), p. 118-127. https://doi.org/10.5176/2251-1865_CBP17.37

Beheydt, L., Bouckaert, F., Sabbe, B. (2011). Cognitive and fine motor function in depressed elderly patients. *International Psychogeriatrics 2011, 23*, supplement 1, 15th International congress of the International Psychogeriatric Association, 6-9 September 2011, The Hague, The Netherlands.

Reviewer invited by peer-reviewed journals

Psychoanalytic Psychology, 2019

Service

Advisory Board	member	2011
Jury Astra Zeneca Psychiatry Award		
UPC Duffel (university of Antwerp) advisory board of Evidence Based Practice	member	2012-2018/2019-2021
Vlaams Forum Diagnostiek, Flemish Advisory board for clinical assessment	Board of directors and executive committee	2020-2021
Superior Health Council of Belgium	Expert	2022-2028
Member of scientific association		
Belgian Federation of Psychology, Flemish	Member of division board	2019-2020

Association of Clinical Psychology, Division for clinical assessment		
Certificate number 722106055		
Visum of Federal Agency of Health, Food Chain and Environment 266085	certified	2020-2021
Belgian College of Neuropsychopharmacology and Biological Psychiatry (BCNBP)	member	2011-2019
Belgian Association of Couple and Family Therapy and System Counseling	member	2020-2021

Presentations

Beheydt, L. (2002). *Zin of onzin van residentiële behandeling van borderline persoonlijkheidsstoornissen*. [Sense or nonsense of residential treatment of borderline personality disorders]. Oral presentation at Symposium Holding environment, seminar of the Foundation of Psychotherapy, Holland.

Beheydt, L. (2006). *Herstel- en terugvalfactoren bij borderlinepersoonlijkheidsstoornissen* [Factors of recovery and relapse in borderline personality disorder]. Oral presentation at Symposium *Major psychiatric disorders: critical factors for recovery and relapse.* Psychiatric Hospital, Duffel

Beheydt, L. & Vandenbroeck, C.(2009). *Het recht op Informatie*. [The right of information] Evening seminar, Similes (association of family members of psychiatric patients).

Beheydt, L. (2009). *Als er tijd is…het metaforisch gebruik van tijd* [If there was time…the metaphoric use of time]. Oral presentation at Symposium of the psychotherapeutic environment in residential psychiatry *Take Your Time*, Duffel.

Beheydt, L. (2010). *Depressie bij ouderen*[Depression in elderly]. Symposium with Didier Schrijvers as president *'Psychomotorische symptomen bij de majeur depressieve stoornis* [Psychomotor symptoms in major depression]*'* at Mental Health Congress (CGG), Gent.

Beheydt, L. (2010). *Op eieren lopen. Omgaan met BPS* [walking on eggshells, dealing with borderline personality disorder]. Oral presentation at seminar, Similes.

Beheydt, L. (2011). *Inleiding.* Symposium *Implicaties van DSM-5 voor diagnostiek en behandeling van persoonlijkheidsstoornissen* [Introduction. Implications of DSM-5 for assessment and treatment of personality disorders]. *Oral presentation at Assessment Symposium*, Duffel.

Beheydt, L. (2011*). Omgaan met Borderline Persoonlijkheidsstoornis*[Dealing with borderline personality disorder]. Oral presentation at seminar, Similes

Beheydt, L. (2011). *De Trein der Traagheid. Cognitieve en psychomotorische vertraging bij ouderen met depressie* [The train of retardation -dutch alliteration-. Cognitive and psychomotor retardation in elderly depressed]. Oral presentation at Researchclub CAPRI

Beheydt, L. (2012). *Burn out. Een modeverschijnsel?* [Burn out, just a hype?] Oral presentation for De Link, team assertive community treatment, Psychiatric Center, Amedeus, Mortsel

Beheydt, L. (2012). *Pesten of gepest worden* [Bullying or being bullied]. Seminar at Elementary School De Kriekelaar (Private Elementary School, Hofstade)

Beheydt, L. (2012). *Error monitoring in major depressive disorder with psychomotor retardation: an fMRI study*, Oral presentation at Research in Psychiatry day of the Belgian College of Neuropsychopharmacology and Biological Psychiatry (BCNBP) and the Flemish Association of Psychiatry.

Beheydt, L. (2012). *Psychomotorische vertraging bij ouderen met depressie* [Psychomotor Reatardation in elderly depressed]. Oral presentation at Symposium Onderzoek naar stemmingsstoornissen [Research into affective spectrum disorders], presided by prof.dr. Schrijvers. Lustrum symposium of 10 years CAPRI (Collaborative Antwerp Research Institute).

Beheydt, L. (2013). *Cognitieve Remediatie ter bevordering van Ziekte-inzicht* [Cognitive Remediation for development of insight in psychotic and bipolar disorder]. Oral presentation of new treatment program, UPC Duffel.

Beheydt, L. (2013). '*The aging mind: neuroplasticity in response to cognitive training*', Oral presentation at the Journal club of the Academic Clinical Staff, University Center, St. Norbertushuis, Duffel, July, 2013

Beheydt, L. (2013). *Inleiding/ Diagnostiek in Interactie* [Introduction/ Assessment in interaction]. Oral presentation of Symposium Diagnostiek in de klinische praktijk [Assessment in clinical practice]. Assessment Symposium, University Center, Duffel

Beheydt, L. & Oldenburg, J. (2014). *De Life-Chart methode en bipolaire stoornis: Inzicht en behandeling*. [The Life Chart Method and bipolar disorder] Oral presentation at the BCNBP (Belgian College of Neuropsychopharmacology and Biological Psychiatry) Hot topics course. October, 17, 2014.

Beheydt, L. & Boost, A. (2017). *Functionele remediatie voor bipolaire stoornis* [Functional Remediation for Bipolar Disorder]. Oral presentation for Seminar Course at UPC Duffel.

Beheydt, L. (2019). *Het Niveau van persoonlijkheidsfunctioneren.* [The level of persoanlity functioning], oral presentation at the scientific seminars of Sinaps (Scientific Initiative for Neuropsychiatric and Psychopharmacological Studies), research unit, UPC Duffel and University of Antwerp.

Beheydt, L. (2019). *Psychodiagnostiek. Sleutel voor therapiewinst* [Assessment. The key for gain in psychotherapy]. Oral presentation of implementation of new clinical assessment strategies at the medical Staff meeting, UPC Duffel. March, 29, 2019.

Beheydt, L. (2019). *Het niveau van persoonlijkheidsfunctioneren* [The level of persoanlity functioning], oral presentation at the Researchclub of CAPRI (Collaborative Antwerp Psychiatric Research Institute), University of Antwerp.

Beheydt, L. (2020). *Precision Care.* Seminar in Psychiatry Artevelde College, January, 31, 2020.

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