CHAPTER 7

DISCUSSION

The results of this empirical study indicate the following:

- Noncognitive sequelae are present in the disease profile of Alzheimer's patients and the findings of this study support the literature indicating a higher prevalence of neurobehavioural in comparison with neuropsychiatric disturbances.
- The noncognitive symptoms are related to specific patient characteristics such as age and cognitive status.
- There is a significant association between primary and secondary caregiver ratings on premorbid temperament. This indicates that retrospective bias has a limited influence on premorbid and current ratings.
- There is a significant association between premorbid temperament traits and the occurrence of noncognitve symptoms.

The following discussion addresses the above results in the context of the specific theoretical approaches that have been outlined in the preceding chapters.

7.1 Noncognitive manifestations

Neurobehavioural symptoms seem to occur more frequently than neuropsychiatric symptoms in this group of Alzheimer's patients. Moreover, specific symptom clusters share significant links with certain patient characteristics. For example, neurovegetative disturbances are significantly associated with age, indicating that advancing age adds to the cumulative effects of the disease in disrupting regulatory behaviours. In terms of noncognitive symptoms, specific neurobehavioural and neuropsychiatric manifestations

are related to the level of cognitive functioning. The latter appears to be inversely associated with cognitive status and the former positively associated. This variable relationship may shed light on the underlying manifestations of noncognitive disturbances.

7.1.1 Neuropsychiatric symptoms and cognitive status

The neuropsychiatric manifestations may be related to the neurological substrates that are dysfunctional and these may cause specific cognitive deficits. In the case of depression, impairment in specific domains may influence a patient and the underlying frustration at his/her inability may induce this type of reaction (Harwood et al., 2000). Support for this conjecture is derived from findings obtained for anosognosic Alzheimer's disease patients. This group of patients has shown an unusually low incidence of depressive symptoms, which suggests a link between observed depressive symptoms and awareness of declining abilities (Green, 2000; Zec, 1993). Moreover, neuropsychiatric phenomena have been linked to the preservation of particular cortical areas and associated neurochemicals (Harwood et al., 2000). The existence of depressive symptoms early as opposed to late in the disease course may suggest that this is a reactive response to awareness of one's declining abilities (Cummings & Black, 1998; Payne et al., 1998). Insight and awareness diminishes with disease severity thus, the reactivity to declining abilities also diminishes. This contention provides a psychological substrate for the occurrence of depressive symptomatology.

Further evidence for a psychological substrate comes from studies comparing the clinical profiles of late-onset Alzheimer's disease patients with early-onset Alzheimer's disease patients. Lawlor et al. (1994) found that early-onset age predicted the severity of depressive symptoms more robustly than late-onset age. Several studies have shown that

dementia onset at an early age is accompanied by greater atrophy and neurochemical disturbances, and the greater impairment in function may adversely influence the younger Alzheimer's patient as long as awareness and insight into the progressive deficits is retained (Green, 2000).

7.1.2 Neurobehavioural symptoms and cognitive status

In terms of the neurobehavioural symptoms and declining cognitive ability, the results show that the irritability/aggression subscale scores share a significant inverse relationship with the level of cognitive functioning. It appears that patients with greater cognitive impairment and widespread atrophy may be more likely to act out and engage in repetitive and disruptive behaviour because of a disruption in general information processing mechanisms. Based on the deduction that greater cognitive inability translates into more acute neuropathology, the atrophy of the prefrontal cortex may be the underlying substrate for dysregulatory behaviour.

The degenerative progression of Alzheimer's disease disrupts the adaptive algorithmic sequences that have developed over the life span and insidiously allows the devolution of the self. Interestingly, the pathological degeneration follows a pattern that is opposite to that of myelination during brain development. In other words, the atrophy commences in sparsely myelinated areas and proceeds to areas of dense myelination paralleling a hierarchical devolution beginning with lower order functions and ending with deterioration in frontal cortex and a complete unravelling of behaviour and affect (Braak & Braak, 1997). The correlates of this unravelling process are an inability to maintain complex interactions, regulate emotions, and conform to societal expectations, all of which reflect aspects of executive functioning. Studies have shown that the

components of disinhibition (restlessness, irritability, agitation) were significantly associated with executive abilities in subjects with moderate and severe Alzheimer's disease (Chen et al., 1998; Paulsen et al., 2000).

In this sample, the neurobehavioural symptoms of irritability and agitation co-occurred with the neuropsychiatric manifestations of anxiety and depressive characteristics. Mega et al. (1996) shows a similar profile and suggests that the co-occurrence of neurobehavioural and neuropsychiatric symptoms illustrates that the antecedents of these manifestations may be multifactorial and related to both underlying neuropathology and a psychosocial attempt to emulate premorbid communication with the environment. Agitated activity, for example, may be a self-soothing mechanism for the patient. In addition, motor activity may be a patient's way of providing self-stimulation in order to overcome the paucity of environmental stimuli (Niesten & Siegal, 1996). The anger, anxiety, emotional lability, and inappropriate behaviours may be masks of despair, a form of primitive communication emerging from an unravelling self (Lawlor, 1996).

The results of the frequency analysis therefore, shows that noncognitive symptoms manifest in varying degrees among Alzheimer's patients, and its significant correlation with patient characteristics such as cognitive status indicates that its multifactorial aetiology may derive from both neurological and psychological substrates.

7.2 Retrospective bias

One of the most challenging aspects of research on premorbid assessment is the need to hark back in time and sketch portraits of individuals prior to the disease onset. This

challenge is compounded by the incapacity of Alzheimer's patients to narrate their own life history and introspect about their own character and predispositions.

The results of several studies provide evidence for and against retrospective bias colouring the images and impressions of premorbid disposition (Brandt et al., 1998; Malinchoc, Rocca, Colligan, Offord, & Kokmen, 1997; Strauss et al., 1997; Swearer et al., 1996). Bearing in mind the equivocal results and the challenge posed by possible recollection bias in caregiver ratings, this study attempted to minimise reporting biases by employing a reliable instrument and by procuring information about premorbid disposition and current behaviour from different informants. The findings of the present study show that the premorbid recollections of different observers correlate significantly, and primary caregivers who live with the patient are able to distinguish between their recollections of a person's demeanor in the past without the impingement of current problematic behaviours. The researcher, however, bears in mind that the difficulty in determining the extent of the preclinical phase may introduce an ad hoc type of recollection bias.

Manifestations of specific noncognitive symptoms can be attributable to particular premorbid temperament characteristics and not only to recollection bias. The premorbid temperament traits that appear to be significant prognostic factors are discussed below.

7.3 Premorbid temperament and noncognitive symptoms

Individual differences in temperament in nonpathological samples are associated with biological substrates (Boyce et al., 1991). The theories of inter alia Pavlov, Eysenck, Zuckerman, and Strelau explore and expand on these based on the underlying premise that differences in temperament are driven by the idiosyncratic functional organisation of

the central nervous system. Temperament also features as a possible salutogenic factor that protects against certain psychopathological behaviours in nonclinical samples (Cederblad et al., 1995). In neurological patient groups, temperament traits serve as possible predictors of onset of such diseases as Parkinson's or Alzheimer's, as well as playing a role in molding the symptom profile of these diseases (Furukawa, Hori, Yoshida, Tsuji, Nakanishi, & Hamanaka, 1998; Glosser et al., 1995; Meins & Dammast, 2000). As an elaboration of the latter premise, the following sections attempt to elucidate the multidimensional role of temperament in predicting noncognitive symptoms in Alzheimer's disease.

In our attempts to understand the relationship between premorbid temperament and behaviour within a disease context, one is reminded that the relationship is not confined to a unitary neural-behavioural interchange, but rather interactions of multilevel and multidimensional neural and behavioural systems. The empirical evidence supports the contention held among researchers that the interplay on a neural and behavioural continuum is multidimensional.

7.3.1 A multidimensional lens: Legacy of a neuropsychological approach

A neuropsychological approach takes into account the dimensional and categorical explanations as espoused by the psychological and medical models, respectively. The psychological model assumes that symptoms lie on a continuum for a given trait, and the medical model assumes that pathology is defined in the absence of normal behaviour. Claridge (1997) argues that there is an inextricable link between normality and disorder, illness and health in psychological terms, and in order to unravel the disorder it is necessary to comprehend the nuances of normality. In other words, the dimensional as

opposed to the discontinuous view may provide a structural and descriptive illustration on predisposition and behaviour. For example, the dimensional view provides some insight about the extent to which a person's depressive state or their aggressive manifestations may stem from a perseverative temperament or by its underlying biological substrate such as an inherently low threshold of arousal and high reactivity. Several authors, who argue that noncognitive symptoms in Alzheimer's disease are merely an exaggeration of premorbid traits, have used the dimensional approach (Bozolla et al., 1992; Chatterjee et al., 1992; Kolanowski et al., 1997; Welleford et al., 1995).

7.3.1.1 Noncognitive symptoms: Merely caricatures of premorbid traits

In Alzheimer's disease, according to Chatterjee et al. (1992), persons who occupy the extremes on the temperament continuum are considered more vulnerable to noncognitive manifestations as the extremes or nonnormative behaviours are likely to emerge because of the diseased profile. The dimensional approach regards abnormal behaviour as an extreme manifestation of specific traits. In contrast, the medical model considers individuals designated with pathological labels as qualitatively distinct groups. There is growing recognition that symptoms underlying Alzheimer's disease are merely an exacerbation of stable, inherent temperament tendencies that are measurable along dimensions (Zuckerman et al., 1984).

Utilising the dimensional approach, the assumption that patients at extremes on the temperament continuum premorbidly are more likely to display pathologic signs can be applied as an explanation for certain patients displaying specific noncognitive symptoms. In other words, a patient's temperament interacts with the neurologically imposed stereotypic profile, thus creating differential increments at which noncognitive symptoms

are likely to emerge. Studies have shown that many demented patients maintain a semblance of their unique patterns of traits and therefore, their premorbid traits act as catalysts for any manifestations that may occur, and the premorbid temperament "gives drama and intensity to life-long traits" (Kolanowski et al., 1997).

This pathoplastic aspect of temperament is addressed by Berrios (1989), who attempts to explain the occurrence of noncognitive symptomotology. He suggests that the exaggeration of premorbid traits caused by disease processes only explains the quantitative alterations in noncognitive symptoms, whereas the qualitative changes (hallucinations, delusion) have no obvious counterpart in premorbid actions. In other words, pathoplasticity may serve as explanations for neurobehavioural symptoms but not for neuropsychiatric manifestations. The latter, according to Berrios (1989) may be explained by recourse to the occurrence of cortical disinhibition. The neuropsychiatric manifestations may be the 'released behaviour' that arises when higher inhibitory functions cannot contain the processes arising from lower cortical areas. This mechanism, however, cannot fully explain the neuropsychiatric phenomenon in the absence of neuroanatomical knowledge about the specific structures of the brain whose release is assumed to cause hallucinations and delusions.

7.3.1.2 The flip side of the coin: Salutogenic properties of extreme traits

Further criticism of the view that noncognitive symptoms are merely exaggerations of extreme premorbid disposition arises from the research of Cederblad et al. (1995). They utilized categories of sub and super dimensions with the former classified on the low end of the continuum and the latter on the high end of the continuum. They hypothesised that persons occupying the extremes of the continuum are less likely to be vulnerable to

pathological conditions and maladaptive stress reactions when compared to persons who occupy a middle position on the continuum. They motivate their idea that extremes on a continuum act as salutogenic factors by assuming that persons on the extreme are more likely to alter the aversive environment or seek out a more suitable niche environment when compared to persons in the middle position. They use dimensions of super- and sub- capacity, validity, solidity, and stability. They associate their dimensions of super-validity and super-solidity to low emotionality (low arousal to stimulation), low impulsivity, high persistence, high exploratory, and high energy behaviours. Sub-stability is associated with high reward dependence, high sociability, high adaptation level, and openness. Depression is associated with validity (level of energy); and high energy correlates with super-validity, active, energetic, persevering, and tenacious traits (sub-validity associated with cautious, tense, and hesitant). They conclude that super-validity is a salutogenic factor or custodial factor for mental health against depression.

According to Cederblad et al. (1995), psychosis correlates with a person's capacity (intelligence) and solidity (flexibility). A high capacity translates into a high intelligence and super-solidity translates into low flexibility and good impulse control. On the other hand, sub-solidity translates into extreme flexibility, impulsive, and a need for novelty and avoidance of monotony. They conclude that super-solidity is a salutogenic factor against psychiatric disturbances. Overall, they conclude that protection from psychosis comes from super-validity (low emotional reactivity), super-solidity (low impulsivity), and capacity. These factors increase the coping capacity of an individual because the adaptive temperament disposition functions as a coping resource. Certain extreme temperament traits therefore, seem to be advantageous and may act as protective-reactive or protective-enhancing. Of note, their hypothesis was based on subjects that were not neurologically impaired but at different risk levels for psychopathology. The interjection of a brain

disease, however, may reduce the salutogenic benefits of temperament extremes. Alzheimer's patients lose the ability to negotiate their environment as the disease progresses. Hence, the salutogenic benefits of extreme traits may be dependent on the disease stage and its accompanying cognitive consequences and not merely a positioning on a continuum.

Adding to the general criticism of the account of noncognitive symptoms as exaggerated manifestations, Strelau (1987b) considers the interaction between predisposition and behaviour to be more complex and involving systems that regulate arousal and reactivity. It is the interaction of these systems together with disease processes, which may produce certain noncognitive profiles in Alzheimer's patients. The following premise is discussed below, utilizing the general principles of the Regulative Theory of Temperament (Strelau, 1994).

7.3.2 Theoretical stance revisited

One of the central themes that permeate the temperament theories discussed in chapter four pertained to the concepts of arousal or activation. Furthermore, according to Klonowicz (1986, 1987) and Strelau (1987b) reactivity as a key component of arousal mechanisms, is a fundamental mediator of environmental, biological and behavioural relations. In the following sections, the discussion focuses on the nature of reactivity and its implications in the context of the empirical findings.

7.3.2.1 Reactivity

In the context of this study, one considers reactivity to mediate relations between the disease influences (environmental), biological predisposition (temperament) and behaviour outcomes (noncognitive symptoms). To recapitulate, the reactivity concept is primarily derived from Pavlov's nervous system types such as strength of excitation. Strength of excitation incorporates the idea that there are individual differences in the level of excitation to stimuli of a given intensity. Many underlying anatomical substrates have been proposed as the driving mechanism for these processes: Robinson (1987) stipulates that strength of excitation corresponds to the activities of the diffuse thalamocortical system; Meccaci (1987) speaks of the reticular formation; Gray and McNaughton (2000) focus on the behavioural inhibition and activation systems, and Zuckerman (1995) considers the neurochemical interactions. Strelau (1987b) however, argues that the underlying physiological functional systems are complex and although all these systems may contribute to an individual's intensity and magnitude of reactions, the dominance of a particular anatomical system or neurochemical is determined by type of activity and situation.

The reactivity continuum consists of two extremes: high reactivity (high sensitivity) and low reactivity (low sensitivity). Due to the complex and multilevel physiological mechanisms that determine an individual's magnitude and intensity of reaction and the outcome of Alzheimer's on these processes, it is incumbent on the reader to understand the role of reactivity in regulating the impact of stimulation.

7.3.2.2 Reactivity and the resting level of arousal

According to Strelau (1987b) and Klonowicz (1987) the level of arousal is higher in high reactive individuals when compared to low reactive individuals. By implication, one can deduce that reactivity controls the level of arousal and a high level of resting arousal enables the individual to detect stimuli easily because the cortex has sufficient tonus. The impact of the stimuli magnitude and novelty is greater for people with high reactivity and higher resting arousal levels.

7.3.2.3 Impact of altered stimulative values

Among Alzheimer's patients who showed a high reactive profile, it is assumed that the impact of stimulation would collude with the pathological correlates of the disease. This assumption is based on the knowledge that individuals have a requisition-competence ratio, which helps with filtering input in proportion to a person's handling capacity (Strelau, 1983). The higher the reactivity levels, the more susceptible the system processes to entropy and disruption of balance. Moreover, in higher reactives, changes in performance and energy expenditure are commensurable with increases in the stimulation load. Due to this susceptibility, high reactive Alzheimer's disease patients would be unable to process input and with altered filtering mechanisms, the stimulation load on brain processing would probably cause certain pathological symptoms to manifest.

These manifestations may reflect an imbalance in the requisition-competence ratio. On a practical level, caregivers interact with patients who are mild or moderately affected utilising similar premorbid communication patterns. However, with a high reactive, the disease would most likely disrupt the requisition-competence balance and this change in

stimulative value could result in difficult and challenging behaviour. This implies that in certain situations caregivers may benefit from a modification in patterns of interaction depending on premorbid temperament of the patient. Among low reactives, the ratio is not as susceptible to imbalances, although decreases in stimulation load can cause imbalances in the system (Strelau, 1987b). However, a more drastic attenuation of stimuli is needed for restoring the balance in high reactives when compared to low reactives. In other words, discrepancies between optimal and actual levels of stimulative intensity is more likely to impact on the high reactive rather than the low reactive, and adverse behavioural outcomes may underscore the disruptive processes (Eliasz, 1987).

7.3.2.4 Exteroceptive influences and self-regulation

Persons with Alzheimer's disease have to endure potentially noxious stimulation (overload or underload) because of the malfunctioning of their cognitive filtering mechanisms. The degree of nonspecific bombardment of the cortex by stimuli depends on a person's level of reactivity, and they compensate for the deficits arising from inadequate task demands and capacity according to their reactivity level (Strelau, 1983).

Self-regulation mechanisms are in place to control the stimulation in order to maintain the optimal level of stimulation and arousal, and these mechanisms function at a physiological and behavioural level. The latter engages trigger mechanisms that activate transient alterations in stimulation processes in order to deal with change increments in inputs.

Studies on animals reflect the self-regulatory mechanisms and show that rats deprived of stimulation select a more stimulating environment, whereas over-stimulated rats choose a more mundane environment (Klonowicz, 1987). The intensity of the seeking behaviour (in

this case pressing a button) is associated with the need for stimulation, which depends on reactivity levels. Perhaps high reactive Alzheimer's patients, who experience an increased level of chaotic input stimuli, react in dysregulated ways to shut off the input. Moreover, high reactives are more susceptible to changes in the social milieu and this is accompanied by a general lack of adaptive behaviour to altered environmental stimulation (Eliasz, 1987). The higher the reactivity the more attention is paid to others and the need for caregiver supervision among Alzheimer's patients creates contexts in which the patient and caregiver interact very closely. Consequently, a high reactive would be more likely to misinterpret the interactions and because of their limited capacity to function appropriately within broad ranges of stimulus intensities are more likely to manifest with challenging behaviours such as aggression and general behavioural dysregulation. Hence, the impact of temperament on noncognitive manifestations may depend on the balance between the need for stimulation (inherent) and the stimulation received from the environment (acquired and malleable to disease processes).

7.3.2.5 Functional structure of behaviour

Behaviour that is goal directed utilises two pivotal operation systems to organize the optimal output. The first is considered the basic operations and the second the auxiliary operations. According to Strelau (1987b) in low reactives the basic operations dominate over the auxiliary operations and in high reactives the relationship is reversed.

From the assumptions underlying the notion of reactivity and response to stimulation, the psychological significance of this pertains to a protective role of auxiliary operations. For example, the auxiliary mechanisms sustain the target activity and minimise stress and circumstances that appear to create tension. Moreover, high reactives fatigue easier than

low reactives in terms of work capacity and low reactives make fewer attempts at planning and control. In other words, the more reactive a person is, the greater the need for protection from adverse stimuli because of the lower capacity to deal with stress and the higher capacity to tire more easily when dealing with novel situations. Hence, an Alzheimer's patient with high premorbid emotional reactivity may react more adversely as the disease progresses because of his/her capacity to deal with environmental stimuli. Caregivers of such patients may have to regulate the environmental demands more stringently as the function of behaviour is more dependent on the progression of disease because as the disease progresses the Alzheimer's patients cognitive capacity to deal with environmental stressors also deteriorates.

7.3.2.6 Interoceptive influences and self-regulation

Unlike the self-regulation that responds to environmental stimulation, interoceptive influences involve another stimulation source namely individual behaviour. The modification of activity and the preference for certain contexts depend on the stimulation value of those contexts and a person's level of reactivity (Eliasz, 1987; Robinson, 1987). In less stimulating contexts, high reactives modify activity levels and respond with more stimulating activities. Conversely, low reactives expend higher levels of activity commensurate with the higher stimulation values of the context. It would appear that low reactives are more attuned to their environments, whereas high reactives display a lesser aptitude for behavioural regulation in relation to changing environmental stimulation. In other words, activity modification is dependent on reactivity, which in turn determines an individual's capacity for regulation, and the optimal functioning of reciprocal feedback lopes between intrinsic and extrinsic stimulation (Klonowicz, 1987).

7.3.2.7 Reactivity and anticipation

In addition to a difference in arousal levels between high and low reactives, Klonowicz (1987) postulates that high and low reactives differ in emotional tone of arousal with high reactives displaying a negative emotional bias. It appears that the negative bias displayed by high reactives may be the result of their resistance to the differential between their optimal arousal and current levels of arousal. This generalization, however, depends on the principle of Occam's razor. The juxtaposition of simplicity and complexity reflects Bateson's (1979) notion of Occam's razor or the rule of parsimony, which states that the simplest solution/hypothesis is adequate to explain certain phenomenon.

In this case, however, the phenomenon under consideration has complex links with neurophysiology, particularly the reciprocal regulative principles of feedback, therefore a more complex explanation is warranted. Klonowicz (1987) hypothesises that interactions with the environment produce different levels of uncertainty and anticipation in high and low reactives. This results in the differentiation of emotional tone that is observed between the two groups. Interestingly, Klonowicz (1987) found that anxiety and uncertainty decrease proportionately in both groups, but that high reactives are more sensitive to the effects of information when compared to low reactives. She concludes that high reactives have a greater capacity for anticipatory stress and a smaller repertoire of coping resources, thus the stress is often interpreted as aversive stimuli and the reactions are imbued with a negative emotional tone.

7.3.2.8 Summary

The reactivity principle according to the Regulative Theory of Temperament is therefore a central concept in theories of temperament and personality that espouse a biological base. Individual differences in behaviour can be explained utilising the idea of reactivity as a mechanism that influences modes of regulation. Figure 7-1 summarises the main principle of reactivity and its influence on behavioural outcomes.

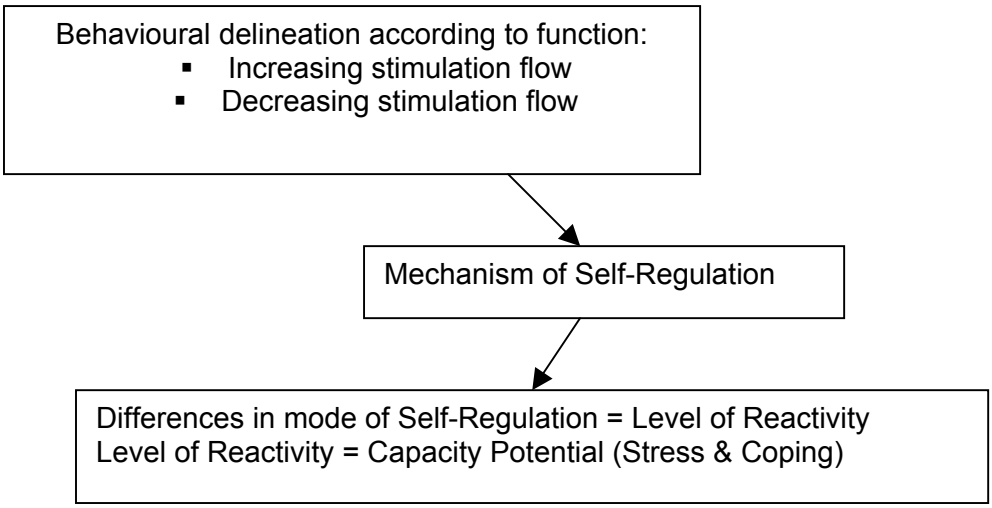

Figure 7-1: Reactivity mechanisms

7.4 Empirical and theoretical coupling

As a pair, the first variate indicates that Alzheimer's disease patients with a proclivity for aggressive behaviours and inappropriate behaviours but lower depressive profiles, were premorbidly more emotionally reactive, had a low sensory threshold (high sensitivity), and impaired cognitive abilities. The second significant variate shows that patients with Alzheimer's disease who tend to manifest with depressive and dysregulatory behaviour appear to have a perseverative temperament with a low neuronal sensory threshold (high sensitivity) and a propensity to maintain and attain a low level of stimulation (low activity

level). These significant results will be discussed in relation to the general Regulative Theory of Temperament principles mentioned above.

7.4.1 The premorbid antecedents of aggressive symptoms

Higher levels of inner instability and nervous tension are regarded as unspecific vulnerability factors for various mental disorders (Andrews, 1996). Therefore, individuals with Alzheimer's disease and high levels of emotional reactivity and sensory sensitivity may be at special risk for a broad range of subsequent neurobehavioural and neuropsychiatric pathologies. Emotional reactivity, according to Strelau (1987b), regulates the intensity of reactions and is expressed as negative affect intensity. Extrapolations from this assumption would suggest that high levels of emotional reactivity would translate into more intense reactions and the reverse would hold for low levels of emotional reactivity.

7.4.1.1 Low thresholds

The canonical analysis reveals that emotional reactivity and sensory sensitivity correlate positively with irritability and aggressive symptoms thereby indicating that high reactives would more likely respond intensively to what they would punctuate as aversive stimuli. To reiterate, according to biological based theories of temperament persons classified as augmenters, introverts, weak nervous system types, and high reactives (high sensory sensitivity) are considered to have a low arousal threshold (Strelau, 1987b). Consequently, they have a lower capacity to deal with altered stimulative values. In other words, high emotional reactivity levels In the presence of high sensitivity (low thresholds) would likely result in intense behaviours being triggered more easily because of the low arousal thresholds.

Several studies have shown that many of the Regulative Theory of Temperament traits are significantly associated with other temperament and personality dimensions (Strelau & Zawadzki, 1995; Zuckerman, 1987a). Significant correlations were found for the emotional reactivity scale of the FCB-TI and the NEO-FFI neuroticism scale and due to the paucity of research dealing with specific temperament dimensions one has to utilize these studies as comparative parameters. One such study by Kolanowski et al. (1997) found a significant association between premorbid neuroticism and aggressive behaviours and between extraversion and aggression. If one assumes a correlation between neuroticism and emotional reactivity, then Kolanowski's et al. (1997) results would support the findings in the present study. On the other hand, the second significant association reported by Kolanowski et al. (1997) contradicts the results of the present study. The Regulative Theory of Temperament proposes that persons with high emotional reactivity and low sensory thresholds are more likely to be introverted and more prone to the inimical effects of altered stimulative value and more likely to act out when compared to people with low emotional reactivity.

A possible explanation from an Regulative Theory of Temperament perspective involves the following: in relation to the stimulative value of the behavioural output, it is likely that the extroverts with Alzheimer's disease respond with behaviour that has a high stimulative value such as aggressive acts and behavioural dysregulation such as pacing. One would assume that the deterioration in cognition alters the intensity and magnitude with which input is perceived and processed, and persons who are categorized as extroverts may display these behaviours as attempts to provide self stimulation to overcome the lack of environmental stimulation, which is required because of their high arousal thresholds (Neistein & Siegal, 1996). Although explanations can be provided for the significant

association between extroversion and aggression, in essence this result of Kolanowski et al. (1997) is contradictory to the findings of the present study.

7.4.1.2 Disease concomitants

In the present study, the profile of Alzheimer's patients tended toward a moderately affected group, whereas Kolanowski et al. (1997) used a severely impaired group. One can assume that the disease process impact more on the severely affected group than on the moderate group, and disrupts more acutely the complex relationships between predisposition and behaviour. Lopez et al. (2003) identify aggressive behaviours as one of the noncognitive symptoms that manifests as a function of disease severity. This provides support for the notion that occurrence does not necessarily follow a linear pattern for all noncognitive symptoms. Furthermore, later in the disease process, low reactives may be more in need of enhancing stimulation because of the decreasing stimulation load (cognitive processing failure), and persons with this predisposition may react more adversely to the discrepancy between optimal and current levels of functioning. Notwithstanding the contradictory results, it is important to consider that perhaps the relationship between predisposition and symptom occurrence may change because of the disease process and longitudinal studies are needed to address the probability of these changes. Therefore, the equivocal results may reflect the methodological deficiencies in sample categorization that do not include the possibility that reactivity, as a trait is changeable and malleable in disease states (Eliasz & Reykowski, 1986).

Welleford et al. (1995), in addition to the exacerbation or exaggerated symptom profile, also found that Alzheimer's patients may manifest with a stereotypic profile where they display similar increases and decreases in certain traits while maintaining individual

variability. There has been some concordance among researchers regarding the latter profile (Bozolla et al., 1992; Kolanowski et al., 1997; Welleford et al. 1995). Studies of Alzheimer's patients indicate that traits such as neuroticism tend to increase and extraversion, openness agreeableness, and conscientiousness tend to decrease relative to premorbid levels, and this appears to reflect a uniform and stereotypic change. If one considers these changes to be a natural manifestation of the disease and its unique neuropathological profile, then one must consider how biological disposition may interact with this stereotypic profile.

Strelau and Zawadzki's (1995) study may provide some insight into the interaction between the disease profile and disposition. Using Factor analysis, they located the Regulative Theory of Temperament traits among other temperament dimensions, and found that sensory sensitivity and openness share a significant positive relationship (.35). Utilising the findings of Bozolla et al. (1992), Kolanowski et al. (1997) and Welleford et al. (1995) on stereotypic changes, one can deduce that if a stereotypic temperament profile is superimposed onto individual differences in temperament then different noncognitive manifestations may arise for different individuals.

A reduction in openness, as stipulated in the stereotypic profile, may influence an introverted temperament. For example, decreased levels of openness (biological disease changes) impact on high sensory sensitivity (an inherent disposition), because people with high sensory sensitivity are categorised as high reactives and introverts, thus with changes in openness they are more introverted augmenters. Welleford et al. (1995) found that the fantasy facet of openness increased and the facets of ideas and aesthetics decreased. Thus, the reactions of patients with high sensory sensitivity and lower openness may arise from the loss of cognitive and functional abilities (low cogstat score) that are needed to

negotiate the environment. These patients may require more structure and consistent routine and any ambiguity may be perceived as a novel situation to which they will react adversely. Furthermore, resilience is also associated with openness and if the typical profile is a decrease in openness, then persons who have limited structure in their environment and a decrease in resilience to stress are more likely to respond negatively (Garmezy & Rutter, 1983).

7.4.2 The premorbid antecedents of behavioural dysregulation

Features of behavioural dysregulation may arise from a patient's attempt at behavioural adaptation in the event of his or her neurological condition co-habitating with the inherent temperament disposition.

7.4.2.1 Low threshold

Person's with a low sensory threshold and unstable predisposition (high emotional reactivity) are more likely to act out quickly and intensely and their attempts at adaptation in the light of cognitive breakdown may manifest as dysregulated behaviour. Gray (1991) suggests that the efficiency of learning is associated with certain temperament dimensions and reinforcements, and lower cognitive status may have a more profound impact on high reactives than on low reactives. Due to the compromised efficiency of the cognitive system, the interactions between caregiver and Alzheimer's disease patients is more likely to be interpreted as threatening and high in punishment value, and since high reactives are more sensitive to punishment stimuli they are more likely on a behavioural level to react in maladaptive ways.

On the level of behaviour, for example, an anxious individual (high reactive with high sensitivity) tends to be sensitive to punishment because of the dominance of the behavioural inhibition system. On the other hand, the impulsive individual (low reactive) is more sensitive to reward because of the workings of the behavioural activation system. In the context of Alzheimer's disease therefore, the high reactives are more likely to perceive caregiver reproaches as punishment and thus, respond in an inappropriate manner. With the accompanying neurological profile, the person is unlikely to purvey any semblance of an adaptive response and the reaction is likely to be intense even in situations that would normally be deemed as one with a low evocative potential.

The energy systems model expands on the general regulatory principles of Strelau (1987b), and uses the capacities of four energy systems and their set points of efficiency as a model of extrapolation relating to predisposition and behaviour (Gale, 1987). Individuals with high levels of emotional reactivity tended to show greater behavioural dysregulation and irritable and aggressive behaviours. If one equates high neuroticism with high emotional reactivity, high reactives would have an inefficient energy store characterised by a "leaky" system with a high set point. Energy is drained in a disorganised and chaotic manner, creating a scenario where the control system has to work at a maximum rate to counter the anomalous energy expenditure.

The system's attempt at restoring homeostasis involves focusing on the acquisition system and input to this system. Neurotic individuals have problems in both the storage systems and acquisition systems. This attempt at avoiding inimical stimuli attenuates the opportunities to learn and hence conserve energy. Individuals with porous energy stores cannot, in terms of attention sustain continuous focus. The symptoms of dysregulated behaviour can be a reflection of the disorganised energy store. Furthermore, individuals in

the mild to moderate stages of dementia experience significant levels of anxiety at their condition and this may enhance the occurrence of symptoms because the psychological "experience of anxiety may include amalgams of correlates of both energy loss, and the effort required to sustain the energy level of the system" (Gale, 1987, p.300). Dementing individuals with impaired cognitive abilities face the consequences of disrupted input-output mechanisms with inefficient storage processes as well as diminished compensatory strategies to control further energy loss evoked by discrepant output activities.

7.4.2.2 Disease concomitants

In Alzheimer's disease, one has to ask whether there will be a direct influence of temperament predispositions that may be impervious to external influences, or whether the predispositions may be ameliorated by caretaker intervention. This interaction between disease, temperament, and caregiver responses is documented by Ware et al. (1990). Collating the circumstances in which dysregulatory behaviour occurs lends itself to the expedient classification of these behaviours. Most of the occurrences tend to be triggered during times of intimate care (bathing, dressing) where the Alzheimer's patient has to respond to instruction. Ware et al. (1990) conclude that although noncognitive behaviours differ from normal behaviour, the changes experienced still follow ordinary rules of behaviour. Events can serve as antecedents or triggers and responses can reinforce them. As with any other behaviour, changing the pattern of antecedents and consequences will affect the rate of any particular behaviour (Zarit, 1996). In some cases, the inappropriate behaviour may be explained by moderating variables that provide the contextual link between predisposition and behaviour. For example, if an Alzheimer's patient with a certain predisposition is not comfortable in novel situations (perhaps social interactions with strangers) and caregivers impel the person to engage in these, then the

reaction for a person who had a inherent aversion for novelty would likely be more pronounced than one who's predisposition is based more on an approach than withdrawal tendency.

Calkins and Fox (1994) support the idea that emotion and behaviour that is displayed is a function of both the event and the process of socialisation. Although individuality of temperament plays a role in behaviour and physiological responsivity there is also a clear process of caregiver feedback, which allows for the modulation of emotional responses. They propose a model that describes how caretaking premorbidly influences the display of particular reactive tendencies. They begin with the premise that emotional reactivity influences premorbid caretaking style, which in turn affects a patient's ability to cope with emotion-eliciting events.

Wachs and King (1995) identify two factors that may moderate the association between predisposition and behaviour and label this reactive and active covariance. The former states that a person with specific behavioural patterns would educe specific types of behaviours from caregivers. In the case of active covariance, persons with a specific predisposition would select specific situations and environmental contexts. Active covariance refers to the tendency of individuals to approach, explore, and express positive effect under conditions of novelty. The opposite tendency would include persons who are more reticent in such circumstances and in extreme cases may display extremely negative affect and withdrawal when confronted with novel situations.

The subcortical asymmetries in amygdala functioning have been demonstrated by Calkins and Fox, (1992), who show that during approach behaviours there seems to be greater left frontal activation, whereas with withdrawal behaviours there is a right frontal activation. In

Alzheimer's disease, researchers have demonstrated impairment in fear conditioning and emotional responsivity that is linked to the functioning of the amygdala (Hamann et al., 2002; Mori et al., 1999). The importance of these results lies in its association with pathological features especially anxiety related symptoms and uninhibitory behaviour. Negative reactivity, for example, as reflected in accentuated motor activity (behavioural dysregulation), is often accompanied by irritability, fearfulness and behavioural inhibition (right frontal activation). Thus, asymmetrical frontal activation may be a neural substrate of the predisposition to display behavioural dysregulation in the face of novelty among Alzheimer's patients. The expression of temperament dispositions among Alzheimer's patients may involve both an expressive component and a regulatory component, which incorporates the effects of the environment.

7.4.3 The premorbid antecedents of depressive symptoms

It would appear that persons who are classified as high reactives should be more susceptible to depressive symptoms because high emotional reactivity is associated with negative emotion and high sensory sensitivity renders a person less capable of behavioural regulation and enhances their susceptibility to anticipatory stress. Interestingly, there is a contradictory relationship between sensory sensitivity and depression when one interprets the two significant variates. In the first variate sensory sensitivity and depression share a negative correlation and in the second variate they share a positive association. The negative relationship between sensory sensitivity and depression is probably more a function of decreased insight and high sensory sensitivity than emotional reactivity. In this case, disease effects could alter the reactivity thresholds and cause more systemic cortical changes.

If a person is more susceptible to environmental stimuli (high arousal/low threshold) as well as inept at intrinsic self-regulation (high reactives), then a more advanced disease state (low cogstat) may interrupt more sensitive positive and negative feedback systems and produce a devolution of the person's metacognitive ability to monitor subjective feelings. In other words, in the absence of disease induced cognitive deficits, a high reactive may be likely to display depressive symptoms, whereas in the context of this brain disease the cognitive devolution may desensitize the high reactive to anticipatory stress and the ability to monitor his/her coping repertoire (see Klonowicz, 1987). Even in the presence of altered capacity to deal with stimuli, the anosognosic orientation would negate the manifestation of depressive features. On a neurobiological level one can hypothesise that the more advanced the cognitive impairment the more acetylcholine depletion is evident. Cummings and Black (1998) show that lower levels of acetylcholine may protect Alzheimer's patients against depression irrespective of a predisposition for occurrence.

The relationship between symptoms and cognitive status helps to identify symptoms that are stage specific and characteristic of the disease process (Teri et al., 1998). These symptoms can therefore, be anticipated because of the information that is available on the stages of the disease and the atrophy accompanying these stages, and the alleviation of such symptoms are more amenable to pharmacological intervention. On the other hand, the genesis of symptoms that have no relationship with cognitive status are more likely to occur at variable stages and to be more idiosyncratic. These symptoms may prove more problematic for caregivers and their anticipated behavioural pattern may be predicted more by correlates such as premorbid temperament. The management of these symptoms are more amenable to psychological interventions dealing with adaptive behavioural patterns and caregiver reactivity.

Ross et al. (1998) argue against this reactive categorization of depressive symptoms in Alzheimer's disease, and contend that depressive symptoms are not necessarily a function of relatively intact cognition but are rather associated with the specific topography of Alzheimer's lesions. This proposition implies that depression is not confined to the early stages of the disease, but can occur at all stages. In the latter case, one assumes that the disease process and biological based temperament parameters interact at a neural level, and in the former case patients premorbid disposition influence their stress responses and resilience strategies on a psychological level.

7.4.3.1 Low thresholds

Based on the principles of arousability and reactivity, one can hypothesise that high arousability would likely lead to neurobehavioural problems rather than neuropsychiatric disturbances i.e., depression and psychosis (Strelau, 1994). Our findings, however, are contradictory to Chatterjee et al. (1992) who suggest that in Alzheimer's disease the more neurotic patients become (higher emotional reactivity), the more likely they are to manifest with depressive symptoms. Chatterjee et al. (1992) themselves note that one of the flaws in their design was the use of the same caregiver to rate premorbid and current behaviour and this may have allowed retrospective bias to colour the recollections of caregivers. In this instance, caregivers of a depressed patient may have recalled the more neurotic inclinations of the patient. Alternatively, high emotional reactivity (neuroticism) may contribute to depression but deteriorating cognitive status may temper this relationship.

7.4.3.2 The activity trait and depressive symptoms

The activity and sensory sensitivity traits are action-oriented energetic traits that have their physiology and biochemistry related to cortico-reticular structures, as opposed to the emotional traits, which have their underlying physiology linked to the limbic and autonomic systems (Strelau & Zawadzki, 1993). Activity is reported to share a positive association with extraversion, openness and strength of excitation and an inverse correlation with neuroticism. According to Strelau and Zawadzki (1993, 1995), a high level of activity serves as organiser of stimulation and a low level allows one to maintain or attain low levels of stimulation. Thus, this trait serves in a regulatory capacity to temperament disposition. Activity is mainly expressed in social situations/interactions and as a source of stimulation refers to all kinds of behaviour and events including behaviour in risky situations (sensation seeking).

By means of activity, the individual regulates the stimulation value of behaviour or situations in such a way as to satisfy his/her need for stimulation and it is co-determined by the level of reactivity. The need to maintain or attain an optimum level of arousal is a standard for the regulation of stimulation. The stimulation value of activity includes a component whereby activity itself is a source of stimulation and the more complex and different the activity, the higher the stimulation being generated (Strelau & Zawadzki, 1995). One of the most efficient generators of stimulation is the emotional content of activity. Activity is not only the direct source of stimulation but by means of activity, the individual may also modify the stimulation value of the environment. Activities aimed at avoiding or approaching stimuli from the environment illustrates this ability to self-regulate stimulative intensities. Thus, on a behavioural level it serves as an organiser of stimulation and an indirect source of stimulation.

The results of the present study showed that there was an inverse relationship between activity and depression. Perhaps, individuals who display inhibitory behaviour are less likely to generate both internal and external stimulation of any considerable intensity, and in the light of the disease processes, would likely be inept at organizing stimulation and limiting sources of stimuli and responses to these sources. On a neurochemical level, Zuckerman (1987b) and Zuckerman et al. (1984) found a negative relationship between norepinephrine and sensation seeking and hence the activity trait identified by Strelau and Zawadzki (1995).

The neurotransmitter norepinephrine, according to Netter (1991), plays a role in mediating responses to stimuli of punishment and novelty. Zuckerman et al. (1984) also associate this neurotransmitter with responses to reward signals and regulation of fear responses in novel and unfamiliar situations. Therefore, the behavioural role of the norepinephrine system appears to be nonspecific and functions in a mediatory role to all emotive systems. Due to the assumption of an inverse relationship between activity and norepinephrine levels, the deduction that high levels of this neurotransmitter appears to bias attention to the external sources of stimuli and high levels of norepinephrine release is related to the inhibition and disorganisation characterised by anxiety (fear) responses. Although the activation of the norepinephrine system is normal in anxious individuals during sedate states, Zuckerman et al. (1984) found that this system responds intensely when individuals encounter unavoidable stressful situations and activities.

Person's with a low activity trait are considered as hyper-responsive and would thus, normally avoid novel situations because of the high stimulatory potential in these situations. In essence, this interpretation is similar to the arousal construct underlying introversion, where introverts seek lower levels of stimulation in order to maintain an

essentially low level of arousal without exceeding the threshold. Furthermore, because neurotransmitter effects do not occur in isolation it is important to consider the effect of increased norepinephrine levels on dopamine discharges in the cortex. Basic neurochemistry suggests that high levels of norepinephrine could result in lower levels of dopamine because norepinephrine is in essence synthesized from dopamine (Kalat, 2001).

The role of dopamine involves the stimulation or activation of behaviour and together with the norepinephrine system enhances the organisms ability to "adapt to physiological and psychological stress, tolerate strong stimulation, and enhance the capacity to respond adaptively to weak stimulation" (Zuckerman et al., 1984, p.414). An imbalance in this system often translates on a behavioural level to a motivational deficit, which is characteristic of depressive features. Considering the disruptive nature of the disease on a neurochemical level, one can see that persons who are premorbidly inhibited would be likely to respond with negative affect because of the maladaptive responses (both psychological and physiological) to the stressful disease situation brought on by the synaptic changes accompanying the disease. Moreover, dopamine is linked to the rewards systems in the brain and to positive emotions. Consequently, low dopamine activation translates into low stimulation of the reward centers of the brain and thus, less positive affect.

Disruption in dopamine levels can either aggravate levels of spontaneous activity, or enhance the rigidity and diminish the plasticity of adaptive behaviour. In the vignettes of the Alzheimer's patients outlined in Chapter 5, one can clearly see the perseverative and rigid thought patterns that permeate their present experiences. Netter (1991) elaborates on the aspect of rigidity but employs the notion of reflexivity to indicate aspects of cognitive

flexibility. He suggests that low dopamine levels may reduce the capability of individuals to alter their strategies for adapting to changing stimuli. He concludes that the dimensions of flexibility and adaptability may have robust associations with the functioning of the dopamine system. Similarly, the acetylcholine system influences cognitive functions and the use of adaptive strategies for negotiating environmental stressors.

In an intact cortex, serotonin usually mediates responses to fearful and aversive situations. An Alzheimer's patient with a particular premorbid profile (low activity and high perseverative disposition) perceives neutral stimuli as novel, and they are likely to react adversely to this. In situations of stress, serotonin mediates fear responses and sensitivity to punishment signals. As an identifier of aversive stimuli, during stress situations, serotonin reduces activity and preserves energy through behaviours resembling apathetic immobility mixed with fearfulness and hyper-vigilance with a negative accompanying tonus (Zuckerman et al., 1984).

Support for the neurochemical correlate of activity is enhanced by the results, which show that this occurs in the absence of gross cognitive deficit, which signifies minimal structural damage to the cortex. The existence of depressive features in the absence of severe cognitive deterioration implies that the complicated emotions and abstract thought processes, which are required to experience some of these symptoms, are dysfunctional in the later stages because of greater brain atrophy (Burns et al., 1990c). Therefore, the results of this study, which found that depressive features in Alzheimer's disease has a negative relationship with rate of cognitive decline supports the contention that depressive symptoms may be a reactive manifestation that includes coping processes, which underscore attempts at energetic and temporal regulation of behavioural and emotional responses and therefore, aspects of temperament.

7.4.3.3 The perseverative trait and depressive symptoms

The perseverance trait is regarded as a temporal characteristic and is linked to emotion (Strelau & Zawadzki, 1995). As a temporal trait, the underlying neurophysiological mechanisms include the speed of elicitation, termination, and mechanism of neuronal interactions. The perseverance trait has two components namely recurrence and persistence. The former refers to repetitive behaviour after termination of stimulation and the latter to the maintenance of these behaviours. Perseverance is regarded as a secondary function of the nerve cells in the cortex. Secondary effects occur as long lasting states after the original content has withdrawn from the center of consciousness. Stimulation generating emotions are effective in developing secondary effects thus, there exists a strong link between perseveration and emotional components. Individuals with a dominance of secondary functions (perseverative temperament) can be described as being for a long time under the influence of formerly acting stimuli, persistent in emotions, and 'stuck' on old recollections. Perseveration also alludes to lability, degree of cognitive inertia, and rigidity of habits. The components of perseveration include ideational, behavioural, and emotional perseveration.

A person with a perseverative temperament cannot easily unhook from emotional states and this implies a rigidity of habits and an inflexible disposition. In the absence of gross cognitive impairment, perseverative dispositions may accentuate the depressive features that are manifested and together with low activity trait and high sensory sensitivity, a person experiencing changes in the synaptic functions may still maintain the use of old learning and coping strategies and enhance the anticipatory stress that a changing environment is delivering. The results also show that high perseverative tendency can result in behavioural dysregulation as well. This would likely be the mediatory effects of

dopamine, which if disrupted can cause a rigidity in activity behaviour, which is evident when Alzheimer's patients tend to repeat certain motor behaviours and appear to have problems with initiation, volition and termination of repetitive motor behaviour.

7.4.4 Summary

Whatever the probable explanations (psychological or neurobiological) the study highlights the complex relationship between premorbid disposition and noncognitive pathology in Alzheimer's disease and the interaction between the neurobiological correlates of the disease and the behavioural/psychological manifestations that arise from the interaction of predisposition, disease, and symptoms.

7.5 Conclusion

The increase in the volume of studies related to noncognitive features attests to their wider recognition as an important concomitant of Alzheimer's disease and a contributing factor to the psychological distress and burnout among caregivers and nurses (Heyns, Venter, Esterhuyse, Bam, & Odendaal, 2003; Welleford et al., 1995). There is still debate, however, as to whether it is a core symptom of the disease or a secondary correlate of cognitive impairment. Notwithstanding the current debates on the cognitive-noncognitive relationship in Alzheimer's disease, research has shown that noncognitive sequelae are the primary motivators underlying the institutionalization of patients because of their adverse consequences on the psychological morbidity of caregivers. A derivative of the challenging consequences of the noncognitive manifestations is a wealth of new research on the correlates of these manifestations.

Utilising a national sample of Alzheimer's patients, this study attempted to elucidate the role of premorbid temperament in the genesis of noncognitive disturbances. The subjects were 63 community-dwelling patients with probable Alzheimer's disease. They were required to reside in the community with a reliable primary caregiver who agreed to act as informant, to have no history of neurological or psychological diseases or disorders, and be younger than 80, and not on any regimented experimental drug trials. The results of this study indicate that the relationship between premorbid temperament and noncognitive symptoms is multidimensional. For example, cognitively impaired Alzheimer's disease patients with a proclivity for aggressive behaviours and inappropriate behaviours but lower depressive profiles, were premorbidly emotionally reactive and sensitive to sensory stimuli, whereas Alzheimer's patients who tend to manifest with depressive and dysregulatory behaviour displayed a premorbid perseverative temperament with low neuronal sensory thresholds and a tendency to maintain low stimulative levels (low activity). Moreover, specific neuropsychiatric features such as depression has premorbid correlates, but patient characteristics such as cognitive status mediate the influence of the correlates. These findings underscore the necessity for future research that deals with the specific mechanisms of noncognitive manifestations. In this manner, one can anticipate behavioural patterns and caregiver reactivity thereby enhancing the adaptive potential of the caregiver-patient relationship during the disease course.

The relationship between premorbid temperament and noncognitive symptoms is complex at a biological and psychological (functional) level. A reflective look at the results yield insight into our constructions of meaning attached to aging and the neurologically impaired aged population. When we consider this group of people our understanding of both biological and behavioural processes is likely to be couched in terms denoting a loss of complexity. Although on a molecular biological level the latter holds true on a

psychological level one is wont to concur with Vaillancourt and Newell's (2002) idea that the relationship between aging and complexity can reflect a bi-directional alteration in complexity that is determined by the nature of the change requirements.

In a psychological sense, hallucinations and wandering behaviour can reflect an increase in behavioural complexity that is underscored by an adaptive need to communicate or compensate for a lack of environmental stimulation. Apathetic behaviour on the other hand, could reflect a decrease in complexity brought about by connectivity and coupling anomalies indicative of the disease processes. A question that arises from this idea of complexity is whether symptoms that are related to premorbid temperament have a specific directional profile (conceptualized as more or less complex) compared to symptoms that are not related to premorbid disposition. In other words, are their specific clusters of symptoms that are direct concomitants of the disease process and specific clusters that are predicted through the disease course by premorbid disposition? Studies combining electrophysiological and psychological angles would likely yield some insight into these questions.

The importance of addressing these questions is linked to the idea that interventions for challenging behaviour should not necessarily lean towards the elimination of symptoms especially if some symptoms reflect an increase in complexity and an attempt at adaptation in the light of a debilitating brain disease. Perhaps a nursing home resident who manifests with frequent hallucinations is attempting to stave off feelings of fragmentation and maintain a semblance of continuity, which enhances their sense of belonging or familiarity (Lazarus et al., 1996). The results reported by Kolanowski et al. (1997), which show that Alzheimer's patients continue to utilize patterns of adaptation that served them premorbidly, support the contention that interventions need to incorporate the

idea that noncognitive symptoms are not a homogeneous cluster that require alleviation via pharmacological intervention.

In sum the results of this study:

1. Provides empirical evidence for the prevalence of neuropsychiatric and neurobehavioural symptoms in a group of Alzheimer's patients and indicates that symptoms are heterogeneous in terms of prevalence and association with patient characteristics.
2. Supports a multidimensional relationship between premorbid temperament and noncognitive symptoms.
3. Suggests that the relationship between premorbid temperament and noncognitive symptoms could more likely reflect a premorbid diasthesis for these symptoms than a retrospective bias imposed by caregiver informants.
4. Supports a relationship between specific premorbid symptoms and patient characteristics such as cognitive status thus, implying that temperament influences may be causal in the early stages of the disease but their effects may be mitigated by the neurological potency of the disease in the later stages.

7.6 Limitations

The following limitations pertain to the study:

- Although the researcher set out to capture a large sample, after stringent exclusion criteria a relatively small sample formed part of the target group that was involved in this study.

- The target group was confined to caregivers of Alzheimer's patients and not to patients with other dementias and neurological conditions (Parkinson's).
- Due to the nature of the study, a convenience sample was used instead of a random sample and the community-dwelling nature of the sample limits the generalisation of results to patients at care-facilities.
- The sample is comprised mainly of english and afrikaans speaking individuals, and is therefore, not representative of all groups with Alzheimer's disease.
- Interaction between caregiver characteristics (temperament, gender, status), patient characteristics, and noncognitive manifestations was not addressed in this study. The inclusion of this dynamic context allows for the avoidance of the reductionistic fallacy that all challenging noncognitive symptoms arise from underlying pathology.
- This study quantified the occurrence of noncognitive symptoms, but did not contextualise these changes as meaningful to caregivers. The reactivity of caregivers to specific symptoms is an important factor when considering therapeutic interventions. The researcher has initiated a follow-up study that addresses the impact of the temperament-symptom relationship on caregivers and their experiential punctuation of the changes they encounter and endure during the disease process.
- Due to the nature of the study neurological data was not included as a diagnostic aid, and informants provided the relevant information.

7.7 Recommendations

The following recommendations are pertinent to future studies:

- Neuropsychiatric and neurobehavioural symptoms are not a homogenous cluster. In this light, research dealing with specific noncognitive manifestations may contribute more substantially to the correlates, both psychological and neurobiological, of specific manifestations.
- Future studies should include a longitudinal assessment because of the curvilinear nature of noncognitive manifestations. This would render valuable data on the stability of premorbid contributions to the occurrence of noncognitive symptoms and the specific disease mechanisms that underlie emergent pathologic behaviour.
- In terms of instrumentation, a more comprehensive battery for cognitive assessment should be used so that a more specific information can be attained and used to understand the aetiology of noncognitive manifestations.
- To enhance the robustness of the relationship between premorbid temperament and noncognitive occurrences in Alzheimer's disease, comparisons to normal age-matched controls need to be made. In this way, one can determine if the relationship is unique to Alzheimer's dementia or common in the process of normal aging.
- Studies on the salutogenic properties of temperament should complement studies on predispostion to pathology. This would create valuable insights into the topography of temperament traits and their role as antagonists or agonists for pathologic behaviour.
- Interactions between caregivers and patients form a key component of the relationship, considering the progressive nature of the disease. Furthermore,

temperament influences the way in which caregivers interact and these nuances are relayed into the post-disease relationship. The interaction between the disease factors, caregiver responses, and patient reactions should be considered in future studies.

- By including the latter, researchers would be able to develop tangible programmes for caregivers regarding the management of both the challenging behaviours of their wards and their own levels of stress, which are endemic to this caregiving process.

"A student asked his guru what does the proverbial turtle rest on. The master replied that it was another giant turtle…the student pressed on: and what does that turtle rest on? The guru answered as before. The student persisted with the same question and the guru, growing more testy with each query, responded with the same answer. When they reached the seventh turtle, the guru stopped the regress by proclaiming: and there it ends because seven is a magic number."

(Zuckerman, 1995, p. 219).