





**POST-TRAUMATIC STRESS DISORDER: AN EPIDEMIOLOGICAL  
STUDY IN THE ROCKLANDS COMMUNITY**

Submitted in partial fulfilment for the M.A. degree in Clinical Psychology in the  
Department of Psychology, Faculty of Social Sciences at the University  
of the Orange Free State

**Susarah C.W. Blignaut**

Supervisor: Prof. D.A. Louw, Department of Psychology,  
Faculty of Social Sciences, UOFS

Co-supervisor: Prof. C.A. Gagiano, Department of Psychiatry,  
Faculty of Medicine, UOFS

30 November 1995

**UOVS - SASOL-BIBLIOTEEK**



199602096001220000018

Financial aid for this study was received from the CENTRE FOR SCIENCE DEVELOPMENT (HSRC, South Africa). However, opinions expressed and conclusions arrived at, are those of the author and not necessarily those of the Centre for Science Development.

## STATEMENT

I hereby declare that this essay submitted in compliance with the requirements for the M.A. degree in Clinical Psychology at the University of the Orange Free State is my own work and has not previously been submitted for degree purposes at any university. I hereby forgo any author's rights in favour of the University of the Orange Free State.

Signed \_\_\_\_\_

Date \_\_\_\_\_

This essay was written in the form of two articles, which is acceptable in terms of the regulations of the University of the Orange Free State. The first article comprises of a literature study, while the second article deals with the empirical research.

## ACKNOWLEDGEMENTS

I would like to thank the following persons and departments for the important part they played in the realisation of this project.

- Prof. D.A. Louw at the Centre for Behavioural Studies in the Department of Psychology at the UOFS for his help as supervisor and coordinator of this project.
- Prof. C.A. Gagiano and the staff of the Department of Psychiatry at the UOFS for their organizational and financial support.
- Me Gina Joubert and Me Riette Nel at the Department of Biostatistics for the statistical analyses.
- The C.S.D. (HSRC, South Africa) for financing the project.
- Mr. C. Nienaber of the Bloemfontein Housing Board for making available the statistical information on Rocklands.
- Warrant officer N.G. Nomyayi at the Batho police station for making available the crime statistics with regard to Mangaung.
- Sister M. Morabello and the nurses at the Rocklands clinic for their help as interpreters and submitting statistics regarding the Rocklands clinic.
- The master's degree Psychology students of the UOFS for their assistance in collecting data and in fieldwork.
- Mrs. E. Faber for her patience and immense help in the typing of the manuscript.

- Mr. H. Koch for attending to the linguistic aspects of this essay.

**ARTICLE ONE**

**POST-TRAUMATIC STRESS DISORDER: A LITERATURE REVIEW**



## CONTENTS OF ARTICLE ONE

	Page
INTRODUCTION.....	1
HISTORY OF THE DISORDER.....	4
PATHOGENIC FACTORS.....	8
- Predisposing factors.....	8
- Modulating factors.....	11
- Precipitating factors .....	13
- Reinforcing factors .....	16
ETIOLOGICAL THEORIES REGARDING SPECIFIC SYMPTOMS .....	18
REVIEW OF PREVIOUS EPIDEMIOLOGICAL STUDIES ON PTSD.....	22
CONCLUSION.....	24
REFERENCES .....	26

## POST-TRAUMATIC STRESS DISORDER: A LITERATURE REVIEW

***Abstract:** The objective of this article was to present an overview of the existing literature concerning Post-traumatic stress disorder (PTSD). This article focuses on the history, pathogenesis, etiological theories regarding specific symptoms and a review of previous community-based epidemiological studies of PTSD. Literature suggested that the pathogenesis of PTSD is multifactorial. Within the biopsychosocial model, certain predisposing, modulating, precipitating and reinforcing factors can be identified. Biological, psychoanalytical and cognitive theories explain certain symptoms of PTSD. Four community-based epidemiological studies of PTSD could be found. No epidemiological data are available on the prevalence of PTSD in non-caucasian communities. It was concluded that further research is necessary to refine diagnostic criteria of PTSD.*

### INTRODUCTION

In the course of negotiations between the community leaders of Rocklands and the University of the Orange Free State, the urgent need for mental health services for the Rocklands community was identified. However, it is generally accepted that without biographical and epidemiological data it is impossible to render effective services (Strauss, Van Rensburg & Gagiano, 1994). Fox, Hall and Elveback (1970, p.2) stated that "in epidemiology, increased understanding of the natural occurrence of disease may be followed, with more than usual predictability, by the development of effective methods for disease prevention".

Although blacks constitute 85% of the total population in South Africa, there is little or no psychiatric epidemiological information on the black community in general or the Rocklands community in particular. This study thus developed out of the

perceived need for a biographical and epidemiological data base of the Rocklands community.

The Rocklands community is a low socio-economic community exposed to violence and poverty. In the light of the known relation between extreme stressors and the development of post-traumatic stress disorder (PTSD), it seemed important to determine the prevalence of PTSD in this community. Authors such as Horowitz (1986) argued that people accustomed to anxiety or sad states may be more resilient to trauma. It may be argued that in these communities violence, in almost all its forms, may be a common phenomenon to which people get accustomed and thus become more resilient to trauma. Taking into consideration the above-mentioned fact, it might be supposed that the prevalence of PTSD in a trauma-tormented community (e.g. Rocklands) will be within the normal range and not higher than in any other community.

The author did not hold this view and hypothesised that the prevalence of PTSD in the Rocklands Community would be in accordance with the stressors experienced. The purpose of this study was therefore to investigate the degree to which PTSD occurred in the Rocklands community and to correlate biographical variables to the prevalence of PTSD.

The editor of the American Journal of Psychiatry (Anderson, 1995) underlined the importance of epidemiological and biographical data by pointing out that recent diagnostic criteria were founded and validated by clinical descriptions and epidemiological data. He argued that a clinical description of symptoms and epidemiological studies will eventually draw closer to the actual etiology so as to improve diagnostic criteria, treatment and prevention. Epidemiological and biographical data therefore not only assist in the planning of intervention methods, but also shed light on etiology and contribute to the development of more refined diagnostic criteria (Anderson, 1995; MacMahon, 1970).

Although the present study was not in-depth epidemiological research, the results will hopefully begin to shed light on the prevalence of PTSD in a low socio-economic black community. The correlation of biographical variables to the prevalence of PTSD might raise questions in relation to etiology and instigate further research that could contribute to the development of more refined diagnostic criteria. The results could also assist in the planning of more effective mental health services.

The epidemiological research will be discussed in the following article. As an introduction to the research, this article focuses on the literature concerning the history and pathogenesis of PTSD.

## HISTORY OF THE DISORDER

According to DSM IV (APA, 1994), PTSD develops in persons who were exposed to, witnessed or learned about a traumatic event that evoked a response of intense fear, helplessness or horror. The essential features of PTSD are the persistent re-experiencing of the traumatic event (e.g. by means of dreams or flashbacks), an avoidance of stimuli associated with the trauma and emotional numbing (e.g. diminished interests or participation in and avoidance of places) and associated symptoms of increased arousal (e.g. irritability and difficulty in concentrating).

Although the term "post-traumatic stress disorder" was first used in DSM III (APA, 1980), the description of symptoms after a stressful life event dates back to the late 19th century when Da Costa described an anxiety response after experiencing trauma. This reaction to trauma was called "irritable heart", "soldier's heart", the "effort syndrome" and "neurocirculatory asthenia" (Kaplan & Sadock, 1991).

During these early years the primary causes were considered to be of physical origin, such as functional cardiac disturbance and organic brain damage (Kaplan & Sadock, 1991). With the development of psychodynamic theory in the early 20th century, unresolved intra-psychic conflict and fear awakened by trauma were seen as the causes of the reaction after a severe stressor (Horowitz, 1986).

It was not until after World War II that psychological morbidity as a result of extreme trauma was acknowledged (Green, Lindy & Grace, 1985). In 1952 DSM I included the diagnosis of a "gross stress reaction", and later DSM II used the term "transient situational disturbance" or "anxiety neuroses" (Green et al., 1985).

Nevertheless, these early descriptions of trauma-related-disorders tended to see the reaction as acute time-limited distress rather than a disorder. Unless pre-trauma patho- psychology was present, the distress reaction was seen as short-lived. It is clear that the assumption was also made that unless something else was wrong with

the individual, the trauma would not cause a disorder in a person. The primary etiological role of the trauma in the development of a disorder was not as yet accepted. It was only after research and long-term studies of war and trauma veterans had been undertaken that the role of trauma in the development of a disorder in a "normal" person was acknowledged and a diagnosis of post-traumatic stress disorder included in the DSM III (APA, 1980) (Green et al. 1985).

The view and classification of PTSD in the DSM III (APA, 1980) and DSM III-R (APA, 1987) stayed very similar. DSM IV (APA, 1994) improved on DSM III (APA, 1980) and DSM III-R (APA, 1987) in that it described the trauma that precipitated PTSD differently. The ambiguities in the definition of trauma as "an event outside the range of usual human experience" that would be distressing to "almost anyone", gave rise to many questions. For example: In the case of long-term warfare where killing becomes within the range of usual experience, will such traumas not lead to PTSD? Furthermore, if it is accepted that the perception of a stressor determines the experienced intensity of the stressor, a "small" trauma, not distressing to "almost anyone", could lead to symptoms of PTSD in a specific person. In such case a diagnosis of PTSD will not be made, because Criterion A has not been met (APA, 1980, p.236; APA, 1987, p.247). DSM IV (APA, 1994) replaced the definition of trauma and changed Criterion A to an extreme stressor that provokes fear, helplessness and/or horror.

DSM III-R (APA, 1987) and DSM IV (APA, 1994) specified that the symptoms must be present for at least one month. For the interim period right after the stressor, DSM IV (APA, 1994) introduced the diagnosis of Acute Stress Disorder (ASD). The disorder closely resembles PTSD, but lasts for a minimum of 2 days and a maximum of 4 weeks after the traumatic event.

Recently, questions arose about the classification of PTSD. In DSM III (APA, 1980), DSM III-R (APA, 1987) and DSM IV (APA, 1994), PTSD is classified with the anxiety disorders. ICD 10 however, classifies PTSD with the adjustment disorders. It seems that ICD 10 classifies PTSD with the adjustment disorders because of the

etiological similarity, namely a stressful event. The difference between PTSD and adjustment disorders is that in the case of PTSD a recognisable extreme stressor must be present. In adjustment disorders any identifiable stressor that leads to "marked distress" must be present. The disorders in this section can thus be regarded as "maladaptive responses to severe or continued stress" (WHO, 1992, p.343).

It seems that in DSM IV (APA, 1994) the differentiation between adjustment disorders and anxiety-related disorders such as PTSD is made according to the constellation of symptoms. The substrate for PTSD is fear that leads to anxiety symptoms. This is not the case with adjustment disorders.

Genetic studies also suggest that PTSD is more closely related to anxiety disorders than adjustment disorders (Davidson & Swartz, 1985). In the light of available data it seems wise to classify PTSD with anxiety disorders.

Literature also questions the diagnostic refinement of PTSD. Many symptoms of PTSD overlap with symptoms of other diagnostic criteria. For example, "numbing" may be associated with a loss of interest in a depressive episode and intrusive images may be mistaken for hallucinations which are symptomatic of e.g. schizophrenia (Green et al., 1985). These symptoms will be seen as part of the symptomatology of PTSD if the symptoms can be related to a traumatic event. If not, another diagnosis should be made.

However, other symptoms such as substance misuse are seen as comorbid secondary syndromes, even though researchers such as Green et al. (1985) noted that 56 % of all Vietnam veterans at an inpatient PTSD unit had an additional Axis 1 diagnosis of depression or substance abuse. The high comorbidity rate of this psychiatric diagnosis and PTSD was also noted by Sierles, Chen, McFarland and Taylor (1983). The question can be asked if these disorders are part of PTSD and thus related to the stress response, like numbing or intrusive images, or comorbid secondary syndromes. DSM IV (APA, 1994) suggests that these are additional

diagnoses (even though stressor-related) and must be made in addition to a PTSD diagnosis on Axis 1.

It is clear that there are still certain loopholes in the diagnostic refinement of PTSD, but the alterations made in DSM IV (APA, 1994) present a positive step towards the improvement of the diagnostic criteria. In spite of the above-mentioned questions, PTSD seems to be a valid and important diagnosis that merits further research and refinement.



## **PATHOGENIC FACTORS**

The value of a multifactorial pathogenesis of PTSD is widely accepted. Green et al. (1985, p.407) suggested that "the nature of the response to a stressful event is multiply determined". Along the same line Kaplan and Sadock (1991) were of the opinion that the pathogenesis of post-traumatic stress disorder is "probably multifactorial". This view is also held in the present article. The multifactorial pathogenesis of PTSD will be presented in order of predisposing, modulating, precipitating and reinforcing factors within the framework of the Biopsychosocial Model.

### **Predisposing factors**

Predisposing factors are variables that cause an individual to be vulnerable or at risk of developing a disorder under certain conditions (Carson, Butcher & Coleman, 1991). These factors include components of genetic and familial factors as well as childhood trauma (Gagiano, Van Rensburg & Le Roux, 1993). With reference to PTSD, the role of genetic, familial and personality factors as predisposing agents will be discussed.

In the late 19th century it was speculated that a hereditary weakness of the nervous system predisposes an individual to develop symptoms after being exposed to a stressful life-event but this theory came into disregard. The role of hereditary factors that may predispose an individual to develop PTSD has not been experimentally proven (Horowitz, 1986). However, researchers such as Davidson et al. (1985) indicated that there is a genetic substrate for anxiety-related disorders. In this regard research showed that 93 % of all patients with generalised anxiety had a positive family history of anxiety disorders.

Anxiety may, in turn, be a predisposing factor for the development of PTSD. Davidson et al. (1985) stated that patients with PTSD had a positive family history of

enhance a person's resilience to trauma. However, he also stated that "preexisting neurotic conflicts may be impediments to processing stressful life events" (Horowitz, 1986, p.246).

Neither the theory of hereditary vulnerability nor the theory of individual psychopathology should be considered the sole explanation for the development of PTSD. Research indicated that certain personality factors may also lead to resilience or vulnerability in the development of mental disorders after being exposed to a traumatic event (Horowitz, 1986).

Horowitz (1986) identified the following five premorbid personality traits that predispose an individual to develop PTSD:

- (i) enduring and irrational attitudes;
- (ii) active and conflicting sets of values, and schemas that lead to emotional ambivalence;
- (iii) the use of pathological defences that lead to habitual information-distortion;
- (iv) an excessive preoccupation with what was threatened or lost in the event;
- (v) schemas and beliefs that the self is bad or incompetent, and a feeling of being unable to cope.

Mineka (1979) speculated that a feeling of unpredictability and uncontrollability may predispose an individual to be vulnerable to the effects of trauma. Frye and Stockton (1982) held that feelings of unpredictability and uncontrollability are enhanced by an external locus of control that intensifies the negative effects of combat and leads to the development of PTSD.

In conclusion one may state that a genetic predisposition to anxiety might predispose an individual to be more vulnerable to react with anxiety in the face of an extreme stressor. Pretrauma anxiety-related disorders and certain personality traits might also impede an individual's capability to process a stressful life event and thus predispose the individual to develop PTSD.

### **Modulating factors**

Modulating factors refer to cultural disposition, socio-economic factors, age and gender. According to Gagliano et al. (1993), a modulating factor not only intensifies vulnerability, but also modulates the format and presentation of the symptoms.

The role of these factors in the development of PTSD will be discussed.

**Cultural disposition.** Research findings indicate that ethnicity plays an important role in the development of PTSD. In a study done by Penk, Robinowitz, Black, Dolan, Bell, Dorsett, Ames and Noriega (1989) post-war adjustment and PTSD in minority groups were examined. Results indicated that African-American Vietnam veterans scored higher on PTSD than white or Hispanic veterans.

The researchers found that minority status does not account for the above-mentioned results. "These findings indicate that ethnicity contributes importantly to PTSD in selected instances, but that minority group status alone does not account for observed differences" (Penk et al., 1989, p.729). Nevertheless Penk et al. (1989) warned that the contribution of ethnicity could not at that stage be fully explained and that further research with more refined measures would be necessary.

The role of ethnicity might be explained against the background of the manner in which ethnicity and cultural disposition will influence a person's world view, ideologies and perceptions regarding trauma. Straker and Moosa, (1988) suggested that meanings and values ascribed to a trauma will modulate the impact

of the trauma and thus the development of PTSD. For example, some African-Americans identified with the oppressed Vietnamese and developed guilt feelings regarding the killing of Vietnamese. This view modulated their vulnerability to the developing of PTSD (Neal & Turner, 1991).

Ethnicity not only modulates the development of PTSD, but also the manifestation of symptoms (Neal & Turner, 1991). According to literature, African-Americans presented with more violent behaviour, homicidal ideation and behaviour, suspiciousness and unstable relationships (Fabrega, Mezzich & Ulrich, 1988). White and Faustman (1989) also reported that Vietnam-war African-Americans had a higher rate of comorbid disorders of substance abuse and depression. It thus seems that the modulating role of culture can be explained in the way it influences perceptions of the trauma and the manifestation of symptoms.

**Socio-economic factors.** It is widely accepted that socio-economic status is a valid determinant of the prevalence of psychiatric symptoms (Adebimpe, 1984). In their study on anxiety disorders in children Last and Perrin (1993) noted that there was a greater prevalence of PTSD in children from lower socio-economic backgrounds than in those from higher socio-economic backgrounds. Neal and Turner (1991) explained the greater prevalence and risk of developing PTSD as a result of the more frequent occurrence of stressors in low socio-economic communities.

In contrast, Lima, Pai, Santacruz and Lozano (1991) were of the opinion that higher levels of violence in poor communities do not necessarily produce higher levels of PTSD. These authors quoted the research done by Harding, Baltazar and De Arango, who found that adult patients in primary care clinics in low socio-economic communities did not present with more PTSD symptoms.

However, more research on socio-economic status and the development of PTSD is needed to clarify this point. At this stage, findings are contradictory and methods used are not refined enough to give clear answers to questions.

**Age.** In research done by Straker and Moosa (1988) developmental age modulated the impact of the trauma. The younger group of 12 - 15 years experienced more anxiety when they were separated from family members and friends after a police raid. The events were differently interpreted by the group of 16 years and older. The police actions affirmed their status as freedom fighters, raised their self-esteem and reduced the negative impact of the events.

Kaplan and Sadock (1991) also stated that age would interact with personal factors to mediate PTSD. The older the person, the more rigid his coping mechanisms and thus the more difficult it would be to deal with trauma. The very young still have inadequate coping mechanisms and thus also cannot deal with the adjustment demands placed on them by trauma.

The above-mentioned sources therefore suggest that cultural disposition, socio-economic factors and age act as modulating factors in the development of PTSD.

### **Precipitating factors**

A precipitating factor is an event or stressor that "triggers" the onset of a disorder (Carson et al., 1988). In the case of PTSD the precipitator or trauma plays an important role in the pathogenesis of PTSD. According to the residual stress model the nature and extent of the trauma must be considered the primary factor in the development of PTSD (Green et al., 1985, Straker & Moosa, 1988). In contrast to the stress evaporation model, the residual stress model postulates that PTSD will develop in any individual subjected to severe enough stress (Straker & Moosa, 1988).

There is substantial incongruence in the literature as to whether trauma or premorbid personality characteristics should be emphasized as the primary pathogenic factor. Green et al. (1985) suggested that there was enough research evidence highlighting the fact that trauma that is stressful enough, can lead to

chronic disorders in previously healthy individuals. It is thus important to focus on the nature of the trauma or stressor acting as the precipitator of PTSD.

DSM IV (APA, 1994) described a traumatic event as a "situation where the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury or a threat to the physical integrity of self or others". The person's response to this event must involve intense fear, helplessness or horror (APA, 1994, p.427-428). The question arises as to what is the nature of the stressors that would cause a response of fear, helplessness or horror.

**Nature of stressors:** Solomons (1989) identified three characteristics of the stressors experienced by ex-detainees suffering from PTSD. The trauma/stressor caused

- a) fear of death,
- b) a feeling of impotence to avoid the coming trauma, and
- c) a forced passivity that left the detainees with no way of dealing with the trauma.

Carson et al. (1991) was of the opinion that the suddenness of trauma, the amount of life threat, and the intensity of fear emanated by the trauma are important aspects that will determine the intensity of the trauma and the reactions of the victim. Kaplan and Sadock (1991) supported this view and also suggested that the suddenness of trauma may enhance the intensity of the stressor. The impact of the stressor is also determined by the imminence and cumulative effect of the stressor (Neal et al., 1991, Straker & Moosa, 1988).

With regard to the imminence of the stressor, research has shown that the number of PTSD symptoms is related to the closeness (imminence) of the child's exposure

to the traumatic event (Neal & Turner, 1991). Several other studies also indicated that the extent and severity of combat exposure correlate positively with the development of PTSD (Foy et al., 1984; Laufer, Brett & Gallops, 1985).

The cumulative effect of repeated traumatic events in previous exposure to violence in relation to the development of PTSD has as yet not been confirmed (Neal & Turner, 1991). However, following their contact with township refugees, Straker and Moosa (1988) hypothesised that PTSD does not always develop as a response to a single stressor, but often is the result of the cumulation of several stressors.

It is hypothesised that the nature of the trauma has an effect on the type of symptoms that develop (Shore, Vollmer & Tatum, 1989). Feelings of guilt are absent among victims of natural disasters and riots, but present among victims of human-induced violence such as rape (Shore et al., 1989). Human-induced disasters also seem to complicate PTSD, producing more symptoms, whereas natural disasters lead to a less chronic form of PTSD with fewer symptoms.

According to Neal and Turner (1991) the nature of the trauma that led to the development of a specific symptom differed for African-American and white soldiers. African-American soldiers suffered from intrusive imagery if they participated in abusive violence, whereas white soldiers presented with intrusive imagery if they witnessed abusive violence. At the moment research is still inconclusive as to whether certain types of trauma lead to a specific symptomatology or whether the individual's experiencing of the trauma determines the symptomatology.

In conclusion it can be said that, although it seems that trauma is a primary factor in the development of PTSD, it should not be seen as the sole factor in the pathogenesis of PTSD. Both the characteristics of the individual and the environment act as important determinants in the development of PTSD (Green et al., 1985; Foy et al., 1984).

## Reinforcing factors

Reinforcing factors are variables that maintain and reinforce an existing disorder and thus chronize such disorder (Carson et al., 1991). Factors that may serve as maintaining and reinforcing variables to complicate and prolong post-traumatic symptoms include premorbid personality factors, post-trauma social environment and ineffective treatment (McFarlane, 1989; Okura, 1975; Neal & Turner, 1991).

McFarlane (1989) suggested that **personality factors** influence the perpetuation of PTSD. Contrary to belief, McFarlane's research showed that the severity and exposure to the disaster as well as the losses sustained, played an important role in the immediate post-traumatic morbidity, but that pretrauma psychiatric illness predicted chronic PTSD. "This suggested that premorbid factors were a more important cause of chronic post-traumatic morbidity than acute disorder" (McFarlane, 1989, p.227). Other researchers such as Helzer, Robins, Wish and Hesselbrock (1979) also came to the conclusion that premorbid personality factors determine the longitudinal outcome of PTSD. The prognosis thus seems to be more determined by premorbid personality factors than by the intensity of the stressor. Literature suggests that a pretrauma personality characterised by anxiety is especially vulnerable to the development of a chronic PTSD (McFarlane, 1989).

Along the same line Horowitz (1986) and McFarlane (1989) hypothesized that cognitive and emotional preoccupation with disaster will reinforce and perpetuate PTSD. However, the results of McFarlane's study indicated that cognitive and emotional preoccupation per se, did not reinforce PTSD. They stated that pretrauma anxiety underlies cognitive preoccupation, and that this anxiety may serve as a reinforcing factor and lead to chronic PTSD (McFarlane, (1989). Kaplan and Saddock (1991) seem to support the view that personality factors influence the perpetuation of PTSD, stating that persons with premorbid dependency needs may enjoy assuming the sick role and that a chronic PTSD may develop as a result.



The **post-trauma social environment** also seems to play an important role in the chronizing of PTSD (Okura, 1975). African-American war veterans not only experienced the trauma differently but on their return to America found themselves jobless and amidst racial conflict. This might have complicated their "healing" environment and lead to chronizing of PTSD (Neal & Turner, 1991). Literature suggests that the absence of social support increases the likelihood of developing psychiatric illnesses. This also seems to be the case with the development of PTSD.

It is also speculated that social environment may play a role in the reinforcement of certain symptoms, whereas other symptoms are extinguished (Straker & Moosa, 1988). Feelings of increased aggressiveness, hatred and revenge, for example, have a survival function in a war situation, but this is not acceptable in a normal situation. The opposite is true as far as anxiety is concerned. Symptoms of aggressiveness will thus be reinforced in a war situation, while the symptom of anxiety will be more acceptable in time of peace. The latter will be contra-productive in a war situation.

**Ineffective treatment** of PTSD is also mentioned in the literature as a variable that plays an important role in the chronizing of PTSD. The high prevalence of comorbid diagnosis such as depression and alcoholism may mask PTSD, in which case the disorder may go undiagnosed and persist over a long period of time (Green et al., 1985; Sierles et al., 1983).

Other factors that may complicate and prolong PTSD, include physical mutilation, disability, damage suits, withdrawal from social contact and the patient's and other people's reactions to the psychological effect of the disorder (Okura, 1975).

Literature is inconclusive as to what exactly reinforces PTSD. For the purpose of this study it is important to acknowledge the role of the different determinants that may reinforce the disorder and lead to chronic PTSD.

## ETIOLOGICAL THEORIES REGARDING SPECIFIC SYMPTOMS

Different theories try to shed light on the etiology of the symptoms associated with PTSD. A synopsis of these theories is presented.

The initial shock symptoms occur as a result of the stress-response where the autonomic nervous system activates a massive sympathetic discharge that leads to hyperalertness, increased cardiac rate, perspiration, muscular tension and a subjective sensation of anxiety. In the case of severe stressors and PTSD the autonomic arousal may occur every time a person remembers the trauma. This psychological flashback will thus lead to a physiological re-experiencing of the trauma (Kaplan & Sadock, 1991).

According to Jones and Barlow (1990), Van der Kolk, Boyd, Krystal and Greenburg suggested that **startle responses** and **aggressive behaviour** are the result of chronic and exaggerated noradrenergic activity caused by the exposure to a stressor. The aforementioned authors suggested that re-exposure to shock may lead to an endogenous opioid-mediated stress-induced analgesia. After the trauma, opioid levels decrease, resulting in adrenergic hyperactivity causing symptoms such as **anxiety, hyperalertness and sleep disturbances**.

**Intrusive imagery** and repetitive **nightmares** might be explained by the model proposed by Kolb (1987). Kolb (1987) hypothesized that cortical neuronal and synaptic changes occur as a result of exposure to an extreme stressor. Prolonged and excessive stimulation cause cortical and subcortical changes that lead to failure of perceptual discrimination and impulse control causing **intrusive thinking** and **repetitive nightmares**.

In relation to this, Pitman (1989) suggested that when a stressful event leads to the overstimulation of endogenous stress response hormones and neuromodulators, it

modulates the overconsolidation of the memory trace of the event. A deeply engraved traumatic memory that manifests itself in **intrusive thinking**, will thus develop.

The afore-mentioned models introduced important aspects in the understanding of the symptoms of PTSD. Nevertheless, certain aspects, for example questions concerning the delayed onset of PTSD or the reasons why some people are not affected while others are, have not been satisfactorily explained by these biological models.

Some psychoanalytic theories have tried to shed light on the dynamics of the symptoms of PTSD. Psychoanalysis distinguishes between a traumatic syndrome and a traumatic neurosis. The former is viewed to be an autonomic response occurring after the trauma. The latter occurs when the ego regresses as a result of being overwhelmed by the traumatic stimuli (Solomons, 1989).

The ego regresses when there is a libidinal withdrawal from the object world into the self-preserving narcissistic core (Solomons, 1989). If the narcissistic defences are overwhelmed, aggressive drives of the id become more dominant, which leads to anxiety (Kaplan & Sadock, 1991; Solomons, 1989). Defence mechanisms such as repetition compulsion, denial regression and conversion estimations are used to protect the ego. If these defence mechanisms fail, they are transformed into symptoms. For example, the repetition-compulsion defence mechanism enables the individual to regain control over the circumstances by repeating the traumatic event. In this way the trauma becomes less overwhelming and can be integrated into the ego. However, if the person cannot reduce the intensity of the trauma or integrate the experience, the repetition-compulsion mechanism becomes a symptom in the form of **nightmares and flashbacks** (Solomons, 1989).

Research on Vietnam veterans and fire survivors illustrated that certain symptoms (especially nightmares and re-enactments) may fluctuate as a result of external stimuli such as anniversaries of the trauma (Green et al., 1985). In the same way

stimuli that remind the individual of the trauma may intensify certain symptom clusters (Green et al., 1985). This phenomenon could be explained within the cyclic view of PTSD. According to this model there is an oscillation between intrusion and avoidance symptoms (Horowitz, 1986). Intrusions, e.g. **nightmares** and **flashbacks**, occur when the defences are let down or fail.

Through the intrusive representations the cognitive structure tries to assimilate this experience which falls outside the realm of normality. When it is impossible/ difficult to assimilate the experiences, **numbing** symptoms (e.g. amnesia, emotional numbness) serve to reduce or strip the cognitive processing and reduce anxiety. The person will oscillate between intrusion and numbing until the experiences are integrated into the cognitive structures (Horowitz, 1986; Green et al., 1985). This coping mechanism should not be viewed as pathological unless the oscillation process is blocked or prolonged (Green et al., 1985).

A well-known feature of PTSD is the way in which "normal" stimuli remind the victim of the trauma. This symptom might be explained by the cognitive information processing models. Lang (1979) hypothesized that a fear network may exist within the memory network. Foa, Steketee and Olatrav-Rothbaum (1989) elaborated on this view and contended that the fear network associated with the trauma is larger, more intense and easily activated by other information structures. Furthermore, as a result of the violation of safety assumptions, and the blurring of boundaries between safety and danger, situations that were previously considered safe, now remind the victim of the trauma. Thus many stimuli, even those not directly connected, may activate the fear network and remind the victim of the trauma (Foa et al., 1989).

In the same way Chemtob, Roitblat, Hanada, Carlson and Twentyman (1988) tried to explain the **re-experiencing** phenomena of PTSD. Chemtob et al. (1988) postulated that a fear-structure in the form of a cognitive schematic network is continually activated. The activation of the fear structure, which includes

information about neurochemical muscular activity, thoughts, behaviour and emotions, leads to the re-experiencing of the trauma (Chemtob et al., 1988).

The **re-experiencing** phenomena of PTSD can also be explained as a conditioned anxiety response to neutral stimuli such as smells, sounds and places (Keane, Zimering & Caddel, 1985). According to Keane et al. (1985) the principal of generalization of stimulus and second-order conditioning may explain how a conditioned physiological and psychological response can occur in reaction to associated stimuli.

Psychological theories, for example cognitive and psychoanalytic theories as well as biological theories, tried to explain the process of symptom formation. It would seem wise to have these theories integrated to explain the symptomatology of PTSD.

## REVIEW OF PREVIOUS EPIDEMIOLOGICAL STUDIES ON PTSD

Although an extensive literature search was done, only four community-based epidemiological studies of PTSD could be found. The reason for this is that existing studies on PTSD were done on samples or communities recently exposed to trauma, for example research concerning Vietnam war veterans and survivors of the Mount St Helena eruption. Furthermore, studies on PTSD did not make use of random sampling in the broad community and thus cannot be regarded as accurate indices of the prevalence of PTSD. Prevalence figures based on in- and outpatient diagnosis will also be a misrepresentation of the true situation, since most individuals suffering from mental disorders do not seek or receive help from mental health services (Lerner, 1992). Nevertheless, these studies did indeed contribute greatly to the existing knowledge of PTSD.

Even less epidemiological data are available on the prevalence of PTSD in non-caucasian communities (Neal & Turner, 1991). The existing research mainly focused on African-American and Hispanic veterans of the Vietnam war, but no studies done in the general non-caucasian population could be found.

The following community-based epidemiological studies on PTSD were found:

In the USA a community-based epidemiological study on psychiatric disorders was launched through the National Institute of Mental Health (NIMH) Epidemiological Catchment Area Survey (ECA) (Neal & Turner, 1991). As part of the ECA project Davidson, Hughes, Blazer and George (1991) studied the prevalence of PTSD in the Piedmont region of North Carolina and found a 13 % lifetime prevalence among 2 985 subjects.

In contrast to this finding Helzer et al. (1979) found a 1 % prevalence rate of PTSD in 2 493 St Louis inhabitants. These results are in accordance with the results of Shore et al. (1989) who reported a 2,6 % prevalence of PTSD in a USA community.

The difference in the prevalence rate could be explained by the fact that the study done by Davidson et al. (1991) reported on a lifetime prevalence, while the study by Helzer et al. (1979) reported on a point prevalence of PTSD.

All of the above-mentioned studies used the Diagnostic Interview Schedule (DIS). According to Davidson et al. (1991) the DIS might underestimate the actual prevalence of PTSD, but it is almost certain that an identified case does have PTSD. However, the Structured Clinical Interview for Diagnosis (SCID) proved to be more effective in non-treatment seeking individuals in the community (Davidson et al., 1991).

In a study conducted by Breslau, Davis and Andreski (1991) a revised version of the DIS was used. A 7 % prevalence of PTSD was found in a young adult study in Detroit. This higher prevalence rate was accounted for by the greater sensitivity of the revised DIS.

It is clear that there is little information concerning community-based epidemiological studies. Nevertheless, it is very important that such studies be undertaken. Epidemiological data will provide useful information that can help the mental health services predict the needs of the community and plan effective intervention and prevention services accordingly (Lerner, 1992).

## CONCLUSION

Since the introduction of the term "post-traumatic stress disorder" in DSM III (APA, 1980) different views concerning the disorder has been held. The view held in DSM IV (APA, 1994) is that PTSD is a disorder that develops in persons who were exposed to, witnessed or learned about a traumatic event that evoked a response of intense fear, helplessness or horror. The DSM IV (1994, APA) thus acknowledges the primary etiological role of trauma in the development of a disorder in an otherwise healthy individual. This does not mean that the role of predisposing and modulating factors in the development of PTSD should be ignored. As the research, reviewed in the literature study shows, the value of a multifactorial pathogenesis of PTSD is widely accepted.

Although the diagnostic refinement of PTSD in the DSM IV (APA, 1994) has improved, certain questions concerning comorbid secondary syndromes and symptom specificity still need to be answered. At present, certain symptoms (e.g. flashbacks) will be seen as part of the symptomatology of PTSD if the symptoms can be related to a traumatic event. In contrast, a symptom such as substance abuse, are seen as an additional diagnosis to a PTSD diagnosis even when this symptom is stressor-related. In this regard it is suggested that case studies with more refined descriptions of the symptomatology of PTSD are conducted.

Even though biological, psychoanalytic and cognitive theories produce valuable insights into the etiology of specific symptoms, these theories, taken individually, fail to explain all aspects of PTSD. It is thus recommended that these theories should be integrated to explain the symptomatology of PTSD.

Four community-based epidemiological studies of PTSD could be found, but no epidemiological data on the prevalence of PTSD in a non-caucasian community (not recently exposed to a trauma) could be found. The total absence of research concerning the prevalence of PTSD in the general non-caucasian community is a



serious hiatus in research on PTSD. Research in this field, for example the influence of culture on the manifestation of PTSD, may enhance the existing knowledge on PTSD.

Furthermore, it is important to use quantitative and qualitative methods to determine whether PTSD is a mere brainchild of caucasian intellectuals or whether it is a universal phenomenon. The research presented in the following article is an effort to achieve this goal.

## REFERENCES

- Adebimpe, V.R. (1984). American Blacks and psychiatry. Transcultural Psychiatric Research Review, 21, 83-111.
- American Psychiatric Association (APA) (1980). Diagnostic and Statistical Manual of Mental Disorders (3rd ed.). Washington DC: APA.
- American Psychiatric Association (APA) (1987). Diagnostic and Statistical Manual of Mental Disorders (3rd ed., Revised). Washington DC: APA.
- American Psychiatric Association (APA) (1994). Diagnostic and Statistical Manual of Mental Disorders (4th ed.). Washington DC: APA.
- Anderson, N.C. (1995). The validation of Psychiatric Diagnosis: New models and approaches. The American Journal of Psychiatry, 152, 161-162.
- Breslau, N., Davis, G.C., Andreski, P. (1991). Traumatic events and Posttraumatic stress disorder in an urban population of young adults. Archives of General Psychiatry, 48, 216-222.
- Carson, R.C., Butcher, J.N. & Coleman, J.C. (Eds) (1991). Abnormal Psychology and Modern Life (9th ed.). United States of America: Harper Collins.
- Chemtob, C., Roitblat, H.C. Hamada, R.S., Carlson, J.G. & Twentyman, C.T. (1988). A cognitive action theory of post-traumatic stress disorder. Journal of Anxiety Disorders, 2, 253-275.
- Davidson, J. & Swartz, J. (1985). A diagnostic and family study of Posttraumatic stress disorder. American Journal of Psychiatry, 142, 90-95.

Davidson, J.R.T., Hughes, D., Blazer, D.G. & George, L.K. (1991). Posttraumatic stress disorder in the community: an epidemiological study. Psychological Medicine, 21, 713-721.

Eysenck, H.J. (Ed.) (1967). The biological basis of personality. Springfield, Il: Charles C. Thomas.

Fabrega, H., Mezzich, J. & Ulrich, R.F. (1988). Black-White differences in psychopathology in an urban psychiatric population. Comprehensive psychiatry, 201, 285-297.

Foa, E.B., Steketee, G. & Olasov-Rothbaum, B. (1989). Behavioral/Cognitive conceptualizations of post-traumatic stress disorder. Behavior Therapy, 20, 155-176.

Fox, J.P., Hall, C.E. & Elveback, L.R. (1970). Epidemiology Man and disease. London: MacMillan Company.

Foy, D.W., Siprelle, R.C., Rueger, D.B. & Carroll, E.M. (1984). Etiology of posttraumatic stress disorder in Vietnam veterans: Analysis of premilitary, military and combat exposure influences. Journal of Consulting and Clinical Psychology, 52, 79-87.

Frye, J.S. & Stockton, R.A. (1982). Discriminant analysis of posttraumatic stress disorder among a group of Vietnam veterans. American Journal of Psychiatry, 139, 52-56.

Gagiano, C.A., van Rensburg, P.H.J. & le Roux, J.F. (1993, August). The Orange Free State model for community psychiatry. Paper presented at the UNP-WHO workshop, Namibia.

Green, B.L., Lindy, J.D. & Grace, M.C. (1985). Posttraumatic Stress Disorder Toward DSM-IV. Journal of Nervous and Mental Disease, 173, 406-411.

Helzer, J.E., Robins, L.N., Wish, E. & Hesselbrock, M. (1979). Depression in Vietnam veterans and civilian controls. American Journal of Psychiatry, 136, 526-529.

Horowitz, M.J. (1986). Stress response syndromes: A Review of Posttraumatic and Adjustment Disorders. Hospital and Community Psychiatry, 37, 241-249.

Jones, J.C. & Barlow, D.H. (1990). The Etiology of posttraumatic stress disorder. Clinical Psychology Review, 10, 299-328.

Kaplan, H.I. & Sadock, B.J. (Eds.) (1991). Synopsis of psychiatry, behavioral Sciences, clinical Psychiatry (5th ed., 1 vol). Baltimore: Williams and Wilkens.

Keane, T.M., Zimering, R.T. & Caddell, J.M. (1985). A behavioral formulation of posttraumatic stress disorder in Vietnam veterans. The Behaviour Therapist, 8, 9-12.

Kolb, L.C. (1987). A neuropsychological hypothesis explaining Posttraumatic stress disorder. American Journal of Psychiatry, 144, 989-995.

Lang, P.J. (1979). A bio-information theory of emotional imagery. Psychophysiology, 16, 495-512.

Last, G.C. & Perrin, S. (1993). Anxiety disorders in African-American and white children. Journal of Abnormal Child Psychology, 21, 153-164.

Laufer, R.S., Brett, E. & Gallops, M.S. (1985). Dimensions of posttraumatic stress disorder among Vietnam veterans. Journal of Nervous and Mental Disease, 173, 538-545.

Lerner, Y. (1992). Psychiatric Epidemiology in Israel. Isr J Psychiatry Relat Sci, 29, 218-228.

Lima, B.R., Pai, S., Santacruz, H. & Lozano, J. (1991). Psychiatric disorders among poor victims following a Major disaster, Armero, Colombia. Journal of Nervous and Mental Disease, 179, 420-426.

MacMahon, B. & Pugh, T.F. (1970). Epidemiology principles and methods, (1st ed.) London: Churchill.

McFarlane, A.C. (1989). The aetiology of Post-traumatic Morbidity: Predisposing, precipitating and perpetuating factors. British Journal of Psychiatry, 154, 221-228.

Mineka, S.M. (1979). The role of fear in theories of avoidance learning, flooding and extinction. Psychological Bulletin, 86, 985-1010.

Neal, M. & Turner, S.M. (1991). Anxiety disorders Research with African Americans: Current status. Psychological Bulletin, 109, 400-410.

Okura, K.P. (1975). Mobilizing in response to a major disaster. Journal of Community Health, 2, 136-144.

Penk, W.E., Robinowitz, R., Black, J., Dolan, M., Bell, W., Dorsett, D., Ames, M. & Noriega, L. (1989). Differences among black, white, and hispanic veterans who differ in degrees of exposure to combat in Vietnam. Journal of Clinical Psychology, 45, 729-735.

Pitman, R.K. & Orr, S.P. (1990). The black hole of trauma. Biological Psychiatry, 27, 469-471.

Pitman, R.K. (1989). Post-traumatic stress disorder, hormones and memory. Biological Psychiatry, 26, 221-223.

Shore, J.H., Vollmer, W.M. & Tatum, E.L. (1989). Community pattern of posttraumatic stress disorders. Journal of Nervous and Mental Disease, 177, 681-685.

Sierles, F.S., Chen, J.J., McFarland, R.E., & Taylor, M.A. (1983). Posttraumatic stress disorder and concurrent psychiatric illness: a preliminary report. American Journal of Psychiatry, 140, 1177-1179.

Slater, E. & Cowe, V. (1971). The genetics of mental disorders. London: Oxford University Press.

Solomons, K. (1989). The dynamics of posttraumatic stress disorder in South African political Ex-detainees. American Journal of Psychotherapy, XLIII, 208-217.

Straker, G. & Moosa, F. (1988). Posttraumatic stress disorder: A reaction to state supported child abuse and neglect. Child Abuse and Neglect, 12, 383-395.

Strauss, P.R., van Rensburg, P.H.J.J. & Gagiano, C.A. (1994). Psigiatrise epidemiologie. Geneeskunde, 36, 17-23.

White, P.A. & Faustman, W.O. (1989). Posttraumatic stress disorder in minorities. Hospital and Community Psychiatry, 40, 86-87.

World Health Organization (WHO) (1992). International statistical classification of Diseases and Related Health Problems (10th ed., 1st vol). Geneva: WHO.

**ARTICLE TWO**

**POST-TRAUMATIC STRESS DISORDER: AN EMPIRICAL STUDY**

**CONTENTS OF ARTICLE TWO**

	Page
INTRODUCTION.....	1
METHODOLOGY .....	2
- Selecting of sample group.....	2
- Survey methods and procedure .....	3
- Statistical analysis.....	4
RESULTS AND DISCUSSION .....	5
- Demographic description of the sample.....	5
- Point prevalence of PTSD.....	13
- Types of symptoms experienced.....	16
- Symptom distribution.....	17
- Types of trauma experienced.....	18
- Association studies .....	22
CONCLUSION AND RECOMMENDATIONS.....	25
SUMMARY .....	28
OPSOMMING.....	30
REFERENCES.....	32



## POST-TRAUMATIC STRESS DISORDER: AN EMPIRICAL STUDY

*Abstract: The objective of this study was to determine the prevalence of Post-traumatic stress disorder (PTSD) in the Rocklands community, a low socio-economic black community near Bloemfontein. The grid method was used to draw a random sample (n=100). A structured interview schedule, based on DSM IV criteria for PTSD, was administered. Results indicate a 5% point prevalence of PTSD in the Rocklands community. Correlations between demographic variables and certain aspects of PTSD were calculated. The association studies showed that more male respondents and respondents with an educational level below standard six, experienced traumatic stressors. The conclusion was made that PTSD is a relevant diagnosis in the Rocklands community. It is recommended that this type of research should be conducted and the results used to promote primary health care in the community.*

## INTRODUCTION

As discussed in the previous article, there is little or no psychiatric epidemiological information on the black community in South Africa or the Rocklands community in particular. This article focuses on this serious hiatus in the South African epidemiological database. In order to address the afore-mentioned problem, the purpose of this study was to investigate the degree in which PTSD occurred in the Rocklands community.

## **METHODOLOGY**

In April 1994 negotiations between Rocklands community leaders and the Centre for Behavioural Sciences of the Department of Psychology at the University of the Orange Free State were opened to form a collaborative relationship from which both the University and Mangaung could benefit. At that meeting it was decided that research results must be used to promote primary health care at the Rocklands Community Centre. The community leaders gave their approval and promised their support for the planning and continuation of this study. After the research proposal was finalised, it was submitted to the Ethical Committee of the Faculty of Medicine and the Rocklands community leaders for approval and recommendations. The study formally commenced in August 1994.

### **Selection of sample group**

Rocklands is situated in the Free State, 10 km outside Bloemfontein and is officially known as Kagisanong 1. It forms part of greater Mangaung and hosts a population of 54 534 Black South Africans (Nienaber, 1995). According to Nienaber (1995) there are 24 540 men and 29 995 women in Rocklands. These numbers include 17 423 male inhabitants above eighteen years and 21 596 female inhabitants above eighteen years.

At the time of the study, Rocklands consisted of three squatter camps and 10 138 formal housing units. Two health clinics serve the community (Nienaber, 1995).

The study was conducted in the Rocklands community. The sample consisted of 100 individuals, above 18 years, from 100 housing units, selected at random by means of the grid-method. A grid map of Rocklands was prepared by drawing an x/y axis. By randomly selecting 2 numbers at a time, x/y coordinates were chosen. These coordinates identified each plot that had to be visited. At every coordinate 2 houses were visited, that is, the marked plot and the house to its left. Before

entering the house, the gender of the respondent was determined by casting lots. The opposite sex was then to be interviewed in the neighbouring house. If a person of the selected sex was not at home, someone of the opposite sex was approached. If that person was not at home or refused to be interviewed, the next house to the left was selected. If there was more than one person in the house that met the age criterion (18 years), a respondent was selected randomly.

### **Survey methods and procedure**

In this study a structured clinical diagnostic approach based on DSM IV (APA, 1994) criteria was used. Two English questionnaires were applied. The questionnaires were compiled by the author and approved by the Ethical Committee of the Faculty of Medicine.

The first questionnaire consisted of a consent form and a biographical questionnaire. The consent form explained the procedure and aim of the study. Forms were signed by the respondent and researcher after the latter had explained and read the form to the respondent. Consent was given verbally if the respondent could not write. The aim of the biographical questionnaire was to gather information about the age, gender, language, employment, occupation, education, socio-economic status, marital status, number of children, living conditions, family of origin and drug and alcohol habits of participants. The data were later compared with data obtained in the second questionnaire.

The second questionnaire consisted of a checklist of stressors and symptoms of PTSD as it is described in DSM IV (APA, 1994). Questions on the duration of the symptoms were included to specify the acute and chronic subtypes of PTSD as well as PTSD in remission and acute stress disorder. For a positive diagnosis of PTSD, all DSM IV (APA, 1994) criteria had to be met.

Before finalising the questionnaires, a pilot study was conducted at the Pelonomi Psychiatry Unit. Questions that were not clear to respondents were adjusted so as to improve the "user-friendliness" of the questionnaire.

The surveys were done during the first two weeks of November 1994, i.e. between 08:00 and 17:00 on Monday to Friday. The questionnaires were administered by four master's degree students in Psychology. Criteria were discussed and the students were trained so that the procedures were standardized as far as possible. Student nurses in their third year were engaged as interpreters, but since all the respondents could speak Afrikaans or English, translation proved to be unnecessary.

### **Statistical analysis**

Frequencies and percentages were calculated to summarise categorical variables: Means, medians, minimums and maximums were calculated to summarise continuous variables. A 95 % confidence interval (CI) was calculated to determine the main outcome of PTSD. To compare subgroups with respect to the prevalence of PTSD, relative risks with 95 % confidence intervals were calculated. The analysis was done by the Department of Biostatistics at the Medical Faculty of the UOFS.

## **RESULTS AND DISCUSSION**

The data presented are the result of a community study and thus a population-based sample of PTSD. In the description of aspects of PTSD found in the Rocklands community attention has been given to the demographic aspects of the sample, point prevalence, type of symptoms experienced, symptom distribution between the four criteria for PTSD and the type of trauma that occurred. Associations between the experiencing of a stressor as traumatic and gender, marital status and level of education were drawn.

### **Demographic description of the sample**

The main demographic findings are the following.

**Table 1** Demographic description of sample

<b>GENDER</b>	<b>PERCENTAGE</b>
Male	45,0
Female	55,0
<b>AGE</b>	<b>PERCENTAGE</b>
18-25	27,0
26-35	25,0
36-45	15,0
46-55	13,0
56-65	15,0
66-75	3,0
76-80	2,0
<b>CURRENT MARITAL STATUS</b>	<b>PERCENTAGE</b>
Married	34,0
Never married	41,4
Divorced	6,1
Widow/Widower	12,1
Living together	5,1
Estranged	2,0
Frequency missing = 1	
<b>INCOME</b>	<b>PERCENTAGE</b>
R 0 - R 100	57,0
R 101 - R 500	22,0
R 501 - R1 000	15,0
R1 001 - R1 500	4,0
R1 501 and over	2,0
<b>JOINT INCOME PER MONTH</b>	<b>PERCENTAGE</b>
R 0 - R 100	8,3
R 101 - R 500	18,8
R 501 - R1 000	18,8
R1 001 - R1 500	10,4

Table 1 continues

R1 501 and over	6,3
Not applicable	37,5
Frequency missing = 4	
NUMBER OF DEPENDANTS	PERCENTAGE
0	29,0
1	14,0
2	18,0
3	17,0
4	8,0
5	6,0
6	4,0
7	1,0
8	1,0
9	1,0
11	1,0

The sample (n=100) consisted of 45 males and 55 females between the age of 18 and 80 years, the mean age being 35 years. Most of the respondents were either married (34%) or have never been married (41%). Most respondents were of a low socio-economic status. Fifty-seven per cent had an individual income below R100,00 per month, with a median number of two dependants. See Table 1.

**Table 2** Demographic description of sample

<b>LANGUAGE</b>	<b>PERCENTAGE</b>
Sotho	57,0
Tswana	29,0
Afrikaans	1,0
Xhosa	13,0
<b>EMPLOYMENT</b>	<b>PERCENTAGE</b>
Full-time	18,0
Part-time	9,0
Unemployed:	
own free will	3,0
unable to find employment	38,0
Disabled with income	1,0
Pensioned	11,0
Other	19,0
<b>LEVEL OF EDUCATION</b>	<b>PERCENTAGE</b>
No formal education	4,2
Std. 2	2,1
Std. 3	2,1
Std. 4	6,3
Std. 5	6,3
Std. 6	23,2
Std. 7	8,4
Std. 8	9,5
Std. 9	7,4
Matric	23,2
College/Technicon	6,3
University	1,1
	Frequency missing = 5
<b>OCCUPATION</b>	<b>PERCENTAGE</b>
Housewife	8,1
Student	10,1
Scholar	8,1



**Table 3** Demographic description of sample

DESCRIPTION OF LIVING CONDITIONS	PERCENTAGE
Running water on site	71,0
Running water in house	43,0
Electricity	66,0
Toilet	81,0
NUMBER OF ROOMS IN HOUSE (EXCLUDING KITCHEN AND BATHROOM)	PERCENTAGE
1	23,0
2	26,0
3	33,0
4	8,0
5	3,0
6	3,0
7	3,0
9	1,0
NUMBER OF RESIDENTS	PERCENTAGE
1	6,0
2	18,0
3	18,0
4	19,0
5	15,0
6	8,0
7	6,0
8	5,0
9	3,0
10	1,0
11	1,0
DESCRIPTION OF RESIDENTS	PERCENTAGE
Nuclear family	62,0
Extended family	28,0

Table 3 continues

Boarders	5,0
Alone	5,0
<b>DESCRIPTION OF FAMILY OF ORIGIN</b>	<b>PERCENTAGE</b>
Mother	19,4
Father	2,2
Mother and father	62,4
Grandmother	9,7
Grandfather	1,1
Grandfather and grandmother	3,2
Other family members	2,2
Frequency missing = 7	
<b>DESCRIPTION OF FAMILY OF ORIGIN</b>	<b>PERCENTAGE</b>
Nuclear family	74,5
Extended family	25,5
Frequency missing = 6	
<b>NUMBER OF RESPONDENTS WHO EXPERIENCED AGGRESSION</b>	18,0
<b>WHERE RESPONDENTS WERE EXPOSED TO VIOLENCE</b>	<b>PERCENTAGE</b>
Family of origin	17,6
Present family	23,5
Strangers	58,8
Frequency missing = 83	
<b>ABUSES OF ALCOHOL OR DRUGS BY RESPONDENTS</b>	<b>PERCENTAGE</b>
	13,0

Table 3 continues

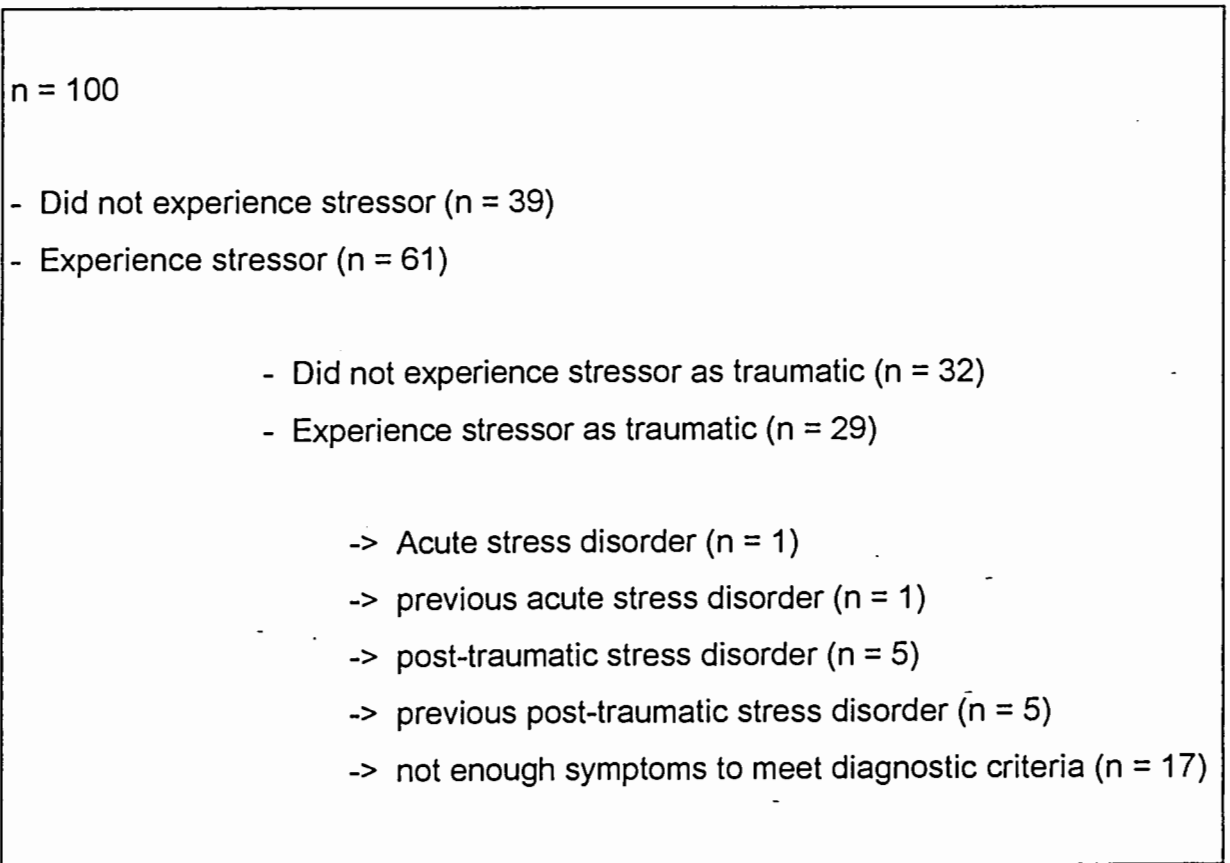
RESPONDENTS' ESTIMATION OF NUMBER OF PEOPLE IN THE COMMUNITY ABUSING ALCOHOL OR DRUGS	PERCENTAGE
Few Average Above average  Frequency missing = 2	18,4 30,6 51,0
<b>RESPONDENTS WHO GREW UP IN A HOUSE WHERE ALCOHOL OR DRUGS WERE ABUSED</b>	24,0
<b>RESPONDENTS LIVING WITH SOMEONE ELSE IN A HOUSE WHERE ALCOHOL OR DRUGS ARE ABUSED</b>	12,4
Frequency missing = 3	

Even though the sample group can be classified a low socio-economic group, most of the respondents (71%) lived in houses with running water on the site, 66 % had electricity and 81% had a toilet in the house or on the site. Most of the houses (33%) consisted of three rooms, with a mean number of four residents per house. Sixty two per cent of the residents in a house were members of the nuclear family. Contrary to popular belief, 74% grew up with the nuclear family. Only 18% experienced violent aggression at home, but the aggressors were mostly people outside the family (58,8%). Although only a small percentage (13%) admitted that they abused alcohol and drugs, 51% of the respondents said that alcohol and drugs were abused in the community on an above-average basis. Most of the respondents (76%) reported that they grew up in a family where alcohol was not abused, while 12% were living with someone who abused alcohol or drugs. (For a more detailed analysis, see Table 3.)

## Point prevalence of PTSD

As Figure 1 shows, 61 respondents experienced a stressor of some sort. Only 29 experienced the stressor as traumatic, that is, with a feeling of helplessness, fear and horror. Two of these respondents suffered from acute stress disorder (ASD), one still presenting with the symptoms and one who previously had the symptoms of ASD. A total of 5 respondents had PTSD symptoms and 5 respondents previously might have had PTSD, but no longer showed the symptoms. Seventeen respondents had some of the symptoms, but not enough to meet the diagnostic criteria of PTSD.

**Figure 1** The experiencing of trauma and the subsequent development of disorders by the respondents



It can thus be stated that a 5% point prevalence of PTSD was found in the Rocklands community. If the 95% confidence interval (CI) is taken into account, the point prevalence might vary between 1,6% and 11,3%. These results seem to correspond with results reported by Davidson, Hughes, Blazer, and George (1991), Shore, Vollmer and Tatum (1989) and Helzer, Robins, Wish and Hesselbrock (1979) who found a PTSD prevalence rate of between 1% and 13% in different USA communities. However, it is important to remember that the results of the aforementioned studies should be compared with great caution, taking into consideration the multitude of variables that were not controlled. Nevertheless, it seems that the prevalence rate of PTSD in non-caucasian, low socio-economic communities is the same as in Western communities of a normal socio-economic status. Important questions about the role of different variables arise from this finding.

One would expect that in a low socio-economic community with a higher prevalence of stressors a higher incidence of PTSD would be found. A possible explanation might be that there is not a higher incidence of stressors in the Rocklands community, or that an unknown variable that modulates the effect of the stressor, is present. However, 17% of the people that experienced stressors as traumatic, developed PTSD. Considering the confidence interval, these results are in accordance with the findings of Breslau, Davis and Andreski (1991) who reported that 23% of a number of persons exposed to trauma developed PTSD. Thus, the percentage of people who developed PTSD after trauma in the Rocklands community appeared to be in accordance with the percentage of people developing PTSD from trauma in other communities. The possibility of a variable modulating the effect of trauma is thus unlikely.

The low prevalence figures for PTSD might have been influenced by the questionnaire used in this research. If one takes into account that there is a difference in the presentation of certain diagnoses (e.g. depression) between culture groups, one may ask whether the symptom presentation of PTSD does not also differ between cultures (Adebimpe, 1981). In this regard, Allen (1986) was of the

opinion that the manifestation of PTSD varies in black patients and that such disorders are often masked by frequent alcohol and drug abuse. This leads to the tendency to misdiagnose black patients. White and Faustman (1989) supported this view and stated that published literature is incomplete in regard to ethnic differences in the expression of PTSD. Further research with regard to the cultural specific expression of PTSD is thus needed. The stated prevalence rate of PTSD should therefore be viewed with caution, considering the fact that the symptom checklist might have excluded certain PTSD sufferers due to the cultural difference in the presentation of symptoms. The stated prevalence of PTSD may therefore be lower than the actual occurrence of the disorder in the Rocklands community.

However, by using a structured clinical diagnostic questionnaire in the form of a checklist of symptoms with well-defined criteria, misdiagnoses as a result of clinical impression or interpretation were minimised. Adebimpe (1994) stated that the sole use of a clinical impression to make a diagnosis, proved to lead to a misdiagnosis in 46% of black patients. It is known that clinical impressions and interpretations are often coloured by the researcher's own paradigms and stereotypes. Adebimpe (1994) was of the opinion that a diagnosis of patients in a transcultural situation would be much more reliable if a structured, well-defined interview schedule would be used.

In conclusion one might speculate that the 5% prevalence of PTSD found in the Rocklands community could be lower than the actual occurrence of PTSD as a result of the nature of the interview schedule. However, by using this structured schedule, misdiagnoses as a result of clinical impression or interpretation were minimised.

Considering all these factors, one may conclude that the stated prevalence of 5% may be an accurate indicator of the occurrence of the disorder in Rocklands. If Rocklands hosts 39 019 people over 18 years, a 5% prevalence rate (95% CI 1.6 to 11.3) would indicate that 624 - 4 409 residents suffer from PTSD. However, the

prevalence of PTSD is subject to the prevalence of stressors. Thus any predictions concerning the prevalence of PTSD should be made with caution.

### Types of symptoms experienced

Since the sample group was small, comparisons should be made with caution. Nevertheless, interesting trends could be detected. For example, when the symptoms of respondents with PTSD were compared to the symptoms of respondents with PTSD in remission, more respondents with PTSD in remission reported symptoms in Criteria C and D. On the other hand, more respondents who suffered from PTSD symptoms at the time of the research reported symptoms in Criteria B. (See Table 4.)

**Table 4** Symptoms reported by respondents with PTSD and with PTSD in remission

Symptoms	Number of respondents suffering from symptoms	
	PTSD (n = 5)	PTSD in remission (n = 5)
<b>Criteria B</b>		
Recurrent images	5	4
Nightmares	5	4
Reliving of the experience	4	4
Extreme distress when reminded	5	5
Psychological reactivity	3	4
<b>Criteria C</b>		
Efforts to avoid associated thoughts	5	5
Avoidance of associated places	3	3
Trouble in remembering important aspects	1	3
Diminished interest in activities	2	4
Feeling of estrangement from others	3	4
Inability to experience loving feelings	3	5

Table 4 continues

Foreshortened future	1	0
No sense of future	4	4
<b>Criteria D</b>		
Trouble in sleeping	5	5
Irritability combined with outbursts of anger	2	4
Difficulty in concentration	3	4
Extreme cautiousness	2	3
Hyperalertness	5	5

Although the results seem to indicate that the symptoms reported by patients with PTSD in remission, mainly fall under Criteria C and D one should be cautious to draw any conclusions. The data were retrospectively obtained from interviews and it might be that the reported symptoms were those that were more chronic and thus best remembered. Support for this argument is found in reports by Davidson et al. (1991) which stated that patients with chronic PTSD pointed out more symptoms in Criteria C.

### Symptom distribution

Of the 17 respondents who experienced stressors with feelings of fear, helplessness and horror but did not show enough symptoms to meet diagnostic criteria for PTSD or ASD, nine persons gave the clinical impression that they might have or have had either PTSD or ASD. These nine respondents were not diagnosed as having PTSD or ASD because they did not meet the diagnostic criteria for one symptom in Criteria B, three symptoms in Criteria C and two symptoms in Criteria D.

Of these nine respondents, three were not diagnosed with PTSD because they lacked one symptom. Two respondents lacked two symptoms, three lacked three



symptoms and one lacked four symptoms. Eight of these respondents did not meet the criteria for three symptoms in Criteria C and seven of these respondents did not meet the criteria for two symptoms in Criteria D. All respondents met the required one symptom in Criteria B; one respondent showed one symptom in Criteria B, four symptoms in Criteria C and one in Criteria D. Although this respondent presented with enough symptoms, he could not be diagnosed as having PTSD, since he did not meet the required number of two symptoms in Criteria D.

These findings question the validation of diagnoses based on a quantitative amount of symptoms in comparison with the qualitative presence of a symptom. Some researchers such as Green, Lindy and Grace (1985) and Horowitz (1986) even suggest that symptoms fluctuate over time and are often episodic. This statement has certain important implications for making a diagnosis and determining a point prevalence of PTSD. This means that only through repeated assessment can a true diagnosis of PTSD be made. One must also bear in mind that in such a case a point prevalence might not necessarily indicate the actual prevalence of PTSD in the community.

Green et al. (1985) suggest that longitudinal research on early symptom development and fluctuation of symptoms should be conducted, since almost nothing is known about the sequence of symptom development. Such research could be helpful in the understanding of the course and stress response process. According to Lerner (1972) a basic aspect of epidemiological research is to shed light on the course and its natural history of a psychiatric disorder. Thus in order to broaden our knowledge of the epidemiology of PTSD, research on symptom development and fluctuation should be conducted.

### **Types of trauma experienced**

If the types of trauma experienced by people with PTSD and PTSD in remission are compared to the types of trauma experienced by people who do not show enough symptoms to meet diagnostic criteria for PTSD, the following results are obtained.

A higher percentage of people with PTSD (currently and in remission) experienced trauma such as a fire, robbery, physical attack, death threats, assault and the observance of dead body parts. A higher percentage of people with insufficient symptoms to meet diagnostic criteria for PTSD experienced serious injury, automobile accidents or imprisonment (see Table 5). In this regard literature suggests that certain types of trauma are more likely to lead to PTSD. For example, 80 % of rape victims developed PTSD, but only 11% of persons who experienced sudden injury (Davis & Breslau, 1994).

**Table 5** Types of trauma experienced by respondents with PTSD (currently and in remission) and respondents showing not enough symptoms to meet diagnostic criteria

	Respondents that experienced the trauma	
	Respondents showing not enough symptoms (n=17)	Respondents with PTSD and PTSD in remission (n=10)
	Percentage	Percentage
Fire	5,0	50,0
Flood	18,0	10,0
Forced removal	12,0	10,0
Tornado	0,0	10,0
Earthquake	0,0	10,0
War	5,0	10,0
Robbery	0,0	30,0
Attack	12,0	40,0
Mugging	0,0	10,0
Kidnapping	0,0	20,0
Being taken hostage	0,0	10,0
Sexual assault	0,0	0,0
Rape	0,0	0,0
Death threat	12,0	30,0
Injury	40,0	20,0
Explosion	0,0	10,0
Terrorist attack	0,0	0,0
Torture	12,0	0,0
Imprisonment	24,0	20,0
Being held in a concentration camp	0,0	0,0
Automobile accident	47,0	20,0
News about serious injury	24,0	40,0
News about death	47,0	50,0
News about assault	24,0	30,0

Table 5 continues

Observance of dead body parts	24,0	50,0
Having a child with a disease	0,0	10,0
Observance of a motor vehicle accident	12,0	0,0
Any other trauma		

Due to the fact that most PTSD sufferers did not want to disclose the nature of the trauma, it was not possible to determine which type of trauma leads to the development of PTSD and which not. This is an important shortcoming in the present study. Green et al. (1985) suggested that research in this direction could lead to the refinement of the diagnosis and description of PTSD.

Nevertheless, this research may indicate that the experiencing of any stressor as traumatic, that is, with a feeling of helplessness, fear and horror, may lead to symptom development. If one looks at Figure 1, it is clear that although 61 respondents experienced a stressor of some sort, only 29 experienced the stressor as traumatic. All of these 29 respondents showed symptoms, but only five of them presented with PTSD symptoms and five might have had PTSD, but no longer presented with the symptoms. One may draw the conclusion that responding to a stressor with fear, helplessness and horror almost always leads to symptom development, but not necessarily to the development of PTSD.

It can thus be argued that the way in which stressors are experienced are an important etiological factor in the development of psychiatric symptoms in general and stress-response symptoms in particular. In this regard Horowitz (1986) stated that 60% of those persons diagnosed as having a mental disorder experienced a, for them, traumatic life-event in the two weeks preceding the onset of the disorder. In comparison only 20% of those people not diagnosed as having a mental disorder experienced a traumatic event in the preceding two weeks.

## Association studies

**Table 6** Association with Gender

Respondents who experienced a stressor	
Male (n = 45) 73,33% (n = 33)	
Female (n = 55) 50,91% (n = 28)	
Respondents who experienced a stressor as traumatic	
Male (n = 33) 48,48% (n = 16)	
Female (n = 28) 46,43% (n = 13)	

Table 6 shows that a higher percentage of males than females experienced a stressor of some sort. A higher percentage of males than females also experienced the stressor as traumatic. These results are in contrast to the results reported by Davidson et al. (1991) who found that more than two thirds of respondents who experienced a stressor as traumatic, were female. A possible explanation for this finding may be that black South African males in Rocklands are more exposed to stressors that may lead to the development of PTSD.

**Table 7** Association with Marital status

Respondents who experienced a stressor
Persons with partners (n = 39) 61,54% (n = 24) Single (n = 61) 60,66% (n = 37)
Respondents who experienced a stressor as traumatic
Male (n = 33) 48,48% (n = 16) Female (n = 28) 46,43% (n = 13)

There was a positive correlation between the percentage of persons with partners and the percentage of single persons who experienced stressors. Similarly the percentage of single persons and persons with partners experiencing stressors as traumatic correlated positively. This finding is in contradiction to the view that the absence of social support increases the likelihood of developing PTSD (Okura, 1975).

**Table 8** Association with educational level

Respondents who experienced a stressor
Above Std. 6 (n = 20) 60,00% (n = 12) Below Std. 6 (n = 75) 62,67% (n = 47)
Respondents who experienced a stressor as traumatic
Above Std. 6 (n = 12) 33,33% (n = 4) Below Std. 6 (n = 47) 51,06% (n = 24)

Frequency missing = 2

The results in Table 8 seem to indicate that people with a lower education level and people with an education level above standard six are exposed to similar stressors, but that people with an educational level below standard six experience more stressors as traumatic. The results of these findings are in contrast to the results obtained by Davidson et al. (1991), namely that a larger percentage of people with a high-school education presented with PTSD. In this study it might have been that socio-economic status (as a result of the level of education) influenced the results. Therefore, before any conclusions can be drawn, the variable of socio-economic status should first be controlled.

In conclusion, the results of the association studies in this research should be used and interpreted with great caution. Certain variables such as socio-economic status were not controlled. One may therefore only speculate about the contribution and role of these variables in the etiology of PTSD. Further research is needed to verify any hypotheses and trends.

## CONCLUSION AND RECOMMENDATIONS

The results obtained in this study indicated a 5% point prevalence of PTSD in the Rocklands community. This finding was supported by figures found in other community-based studies on the prevalence of PTSD.

With regard to the characteristics of PTSD in Rocklands, more symptoms in Criteria C and D of the DSM 1V (APA, 1994) were reported by respondents with PTSD in remission, while respondents who suffered from PTSD symptoms at the time of the research reported more symptoms in Criteria B.

If one looks at the symptom distribution between the three criteria for PTSD, results show that nine respondents gave a clinical impression that they might have or have had PTSD or ASD. Nevertheless, these nine respondents were not diagnosed as having PTSD or ASD because they did not meet the diagnostic criteria for PTSD.

Results also seem to suggest that stressors play an important etiological role in the development of psychiatric symptoms in general and stress-response symptoms in particular.

The association studies showed that more male respondents and respondents with an education level below standard six experienced traumatic stressors. Contrary to the general belief, results of this study showed no significant differences between single persons and persons with partners in relation to the experiencing of a stressor as traumatic.

Since accurate prevalence figures, or an accurate description of characteristics of a disorder can only be obtained from population-based samples, further population-based epidemiological research is needed to enrich our knowledge of PTSD and to obtain accurate prevalence figures (McFarlane, 1989). The development of a large-scale community survey in South Africa similar to the



epidemiological catchment area project in America is suggested. This study must include all population groups.

However, care must be taken to implement an epidemiological study that will be sensitive to the unique experience of blacks in South Africa. Trained interviewers sensitive to black culture should conduct the research by using culturally sensitive interview schedules. In this regard, Adebimpe (1994) found that diagnosis were more accurate when black clinicians assessed black patients and vice versa. Furthermore, in South Africa the additional language barrier and feelings of being intimidated by white researchers may influence results. Allen (1986) reported that blacks often wear a "black mask" of blunted affect in anticipation of frustration in dealing with whites, and that this adds to the diagnostic confusion. It is thus recommended that black clinicians accept the challenge to do this kind of research in the black communities. Nevertheless the culture issue should not be overdramatised, nor should cultural aspects that might influence research results be ignored.

In this research, samples were restricted to the Rocklands community and to respondents aged 18 years and older. Caution must therefore be taken not to generalise the results to all black South Africans. There are subtle but substantial socio-cultural and socio-economic differences between blacks belonging to different cultural groups.

Regarding this research, no real inferences about race and culture variables in regard to results should be drawn since these variables were not controlled. Tendencies and differences in results might be the result of socio-economic factors and not necessarily race or culture. Green, Lindy and Grace (1990) reported that blacks often show higher levels of symptoms in community studies, but that when social factors are controlled, the differences disappear. Some studies have even indicated that whites in urban areas are slightly more symptomatic (Green et al., 1990). According to Penk, Robinowitz, Black, Dolan, Bell, Dorsett, Ames and Noriega (1985) future research must address the social and socio-economic factors

embedded in the vague variable of ethnicity before conclusions about the role of culture in the pathogenesis of PTSD can be made.

This study made use of a too small number of respondents to draw any conclusions. However, possible tendencies could be noted, but more extensive studies involving a larger number of respondents need to be conducted before any conclusions about tendencies can be made.

Finally, the results should be used to promote primary health care in the Rocklands community. According to Fox et al. (1970) the long-term objective of epidemiological research must be to implement strategies for the training of primary care workers in the management of the most frequently occurring psychiatric disorders. In this regard it is strongly suggested that primary health care workers be advised on the presentation of PTSD. The urgent need for such a programme is illustrated by the fact that during the period August to December 1994 no diagnosis of PTSD was made, nor were patients with PTSD treated at the Rocklands clinic (1995).

## SUMMARY

The objective of this study was to determine the prevalence of Post-traumatic stress disorder (PTSD) in the Rocklands community. Correlations between demographic variables and certain aspects of PTSD were calculated.

An extensive literature study on the pathogenesis and history of the disorder was undertaken. Literature suggested that the pathogenesis of PTSD is multifactorial. Within the Biopsychosocial model certain predisposing (e.g. genetic, familial and personality factors), modulating (e.g. culture, socio-economic status and age) and precipitating factors (e.g. trauma) can be identified. Reinforcing factors such as premorbid personality factors, post-trauma social environment and ineffective treatment seem to lead to the chronic subtype of PTSD.

Biological, psycho-analytical and cognitive theories explain certain symptoms of PTSD. However, it would seem necessary to have these theories integrated to explain the symptomatology of PTSD.

The grid method was used to draw a random sample (n=100) in the Rocklands community. A structured interview schedule, based on DSM IV criteria for PTSD, was administered. A 95% confidence interval (CI) was calculated to determine the main outcome of PTSD. To compare subgroups with respect to the prevalence of PTSD, relative risks with 95% CI were calculated.

Results indicate a 5% point prevalence of PTSD in the Rocklands community. Considering the confidence interval (95% CI 1.6 to 11.3) results would indicate that between 624 and 4407 residents may suffer from PTSD. However, the prevalence of PTSD is subject to the prevalence of stressors. Thus predictions concerning the prevalence of PTSD should be made with caution.

The association studies showed that more male respondents and respondents with an educational level below standard six, experienced traumatic stressors. Results showed no significant differences between single persons and persons with partners in relation to experiencing of a stressor as traumatic.

The conclusion was made that PTSD is a relevant diagnosis in the Rocklands community. It is recommended that this type of research should be made to ameliorate primary health care in the community by training health care workers in the management and identification of PTSD.

## OPSOMMING

Die doel van hierdie studie was om die voorkoms van post-traumatiese stresversteuring (PTSV) in die Rocklands gemeenskap te bepaal. Korrelasies tussen demografiese veranderlikes en sekere aspekte van PTSV is bereken.

'n Uitgebreide literatuurstudie is oor die patagonese en geskiedenis van die versteuring gedoen. Dit het geblyk dat die patagonese van PTSV multifaktoriaal is. Binne die biopsigososiale model kan sekere faktore identifiseer word naamlik: predisponerende (byvoorbeeld genetiese, gesins- en persoonlikheidsfaktore), modulerende (byvoorbeeld kultuur, sosio-ekonomiese status en ouderdom) en presipiterende faktore (byvoorbeeld trauma). Versterkende faktore byvoorbeeld premorbiede persoonlikheidsfaktore, post-trauma sosiale omgewing en oneffektiewe behandeling blyk tot 'n kroniese sub tipe van PTSV te lei.

Biologiese, psigo-analitiese en kognitiewe teorieë poog om sekere simptome van PTSV te verklaar. Dit blyk egter nodig te wees om die verskillende teorieë te integreer ten einde die simptomatologie van PTSV te verklaar.

Die rooster metode is gebruik om 'n ewekansige steekproef ( $n=100$ ) in die Rocklands gemeenskap te trek. 'n Gestruktureerde onderhoudskedule, gebaseer op die DSM IV kriteria vir PTSV, is op die steekproef gevoer. 'n Vyf en negentig persent vertrouens interval (VI) is bereken om die voorkoms van PTSV te bepaal. Om subgroepe ten opsigte van die voorkoms van PTSV te vergelyk, is relatiewe risikos met 'n 95% VI bereken.

Resultate het aangedui dat daar 'n 5% voorkoms van PTSV in die Rocklands gemeenskap is. Met in ag neming van die vertrouensinterval (95% VI 1.6 tot 11.3) toon resultate dat tussen 624 en 4409 inwoners van Rocklands moontlik aan PTSV

lei. Die voorkoms van PTSV is egter onderheweig aan die voorkoms van stressore. Voorspellings rakende die voorkoms van PTSV moet gevolglik met die nodige sorg gemaak word.

Korrelasiestudies toon dat meer manlike respondente en respondente met 'n onderwyspyl laer as standerd ses, stressors as traumaties beleef het. Resultate dui op geen betekenisvolle verskil tussen enkellopendes en persone met 'n gesel, ten opsigte van die belewing van 'n stressor as traumaties nie.

Die gevolgtrekking is gemaak dat PTSV 'n relevante diagnose in die Rocklands gemeenskap is. Daar word aanbeveel dat hierdie tipe navorsing gebruik moet word om primêre gesondheidsorg te bevorder deur primere gesondheidswerkers op te lei in die identifisering en hantering van PTSV.

## REFERENCES

- Adebimpe, V.R. (1981). Overview : White norms and Psychiatric Diagnosis of Black Patients. American Journal of Psychiatry, 138, 279-285.
- Adebimpe, V.R. (1994). Race, Racism, and Epidemiological Surveys. Hospital and Community Psychiatry, 45, 27-31.
- Allen, I.M. (1986). Posttraumatic Stress Disorder among Black Vietnam Veterans. Hospital and Community Psychiatry, 37, 55-60.
- American Psychiatric Association (APA) (1994). Diagnostic and Statistical Manual of Mental Disorders (4th ed.). Washington DC: APA.
- Anderson, N.C. (1995). The validation of Psychiatric Diagnosis: New models and approaches. The American Journal of Psychiatry, 152, 161-162.
- Breslau, N., Davis, G.C., Andreski, P. (1991). Traumatic events and Posttraumatic stress disorder in an urban population of young adults. Archives of General Psychiatry, 48, 216-222.
- Davidson, J.R.T., Hughes, D., Blazer, D.G. & George, L.K. (1991). Posttraumatic stress disorder in the community: an epidemiological study. Psychological Medicine, 21, 713-721.
- Davis, G.C. & Breslau, N. (1994). Psychiatric clinics of North America, 2, 289-297.
- Fox, J.P., Hall, C.E. & Elveback, L.R. (1970). Epidemiology Man and disease. London: MacMillan Company.

Green, B.L., Grace, M.C., Lindy, J.D. & Leonard, A.C. (1990). Race differences in response to combat stress. Journal of Traumatic Stress, 3, 379-393.

Green, B.L., Lindy, J.D. & Grace, M.C. (1985). Posttraumatic Stress Disorder Toward DSM-IV. Journal of Nervous and Mental Disease, 173, 406-411.

Helzer, J.E., Robins, L.N., Wish, E. & Hesselbrock, M. (1979). Depression in Vietnam veterans and civilian controls. American Journal of Psychiatry, 136, 526-529.

Horowitz, M.J. (1986). Stress response syndromes: A Review of Posttraumatic and Adjustment Disorders. Hospital and Community Psychiatry, 37, 241-249.

Lerner, Y. (1992). Psychiatric Epidemiology in Israel. Isr J Psychiatry Relat Sci, 29, 218-228.

MacMahon, B. & Pugh, T.F. (1970). Epidemiology principles and methods, (1st ed.) London: Churchill.

McFarlane, A.C. (1989). The aetiology of Post-traumatic Morbidity: Predisposing, precipitating and perpetuating factors. British Journal of Psychiatry, 154, 221-228.

Nienaber, C. (1995). Statistics of Mangaung. Housing manager, Bloemfontein Regional Transitional Council.

Okura, K.P. (1975). Mobilizing in response to a major disaster. Journal of Community Health, 2, 136-144.



Penk, W.E., Robinowitz, R., Black, J., Dolan, M., Bell, W., Dorsett, D., Ames, M. & Noriega, L. (1989). Differences among black, white, and hispanic veterans who differ in degrees of exposure to combat in Vietnam. Journal of Clinical Psychology, 45, 729-735.

Shore, J.H., Vollmer, W.M. & Tatum, E.L. (1989). Community pattern of posttraumatic stress disorders. Journal of Nervous and Mental Disease, 177, 681-685.

Strauss, P.R., van Rensburg, P.H.J.J. & Gagiano, C.A. (1994). Psigiatrise epidemiologie. Geneeskunde, 36, 17-23.

White, P.A. & Faustman, W.O. (1989). Posttraumatic stress disorder in minorities. Hospital and Community Psychiatry, 40, 86-87.