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Influence of Traumatic Life Experiences on Cortisol Levels in Patients at the Psychiatric Hospital of Bingerville (Côte d'Ivoire)

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Abstract | **Original Research Article**

Cortisol, a key hormone in the stress response, is frequently disrupted in psychiatric disorders. However, few studies have examined the impact of traumatic life experiences on cortisol levels in the African context. This research aimed to assess the influence of traumatic life experiences on blood cortisol levels among patients followed at the Psychiatric Hospital of Bingerville (Côte d'Ivoire). This cross-sectional and analytical study involved 170 patients, of whom 42 were selected for hormonal assays. Traumatic experiences were assessed using the Trauma History Questionnaire (THQ), the Posttraumatic Stress Disorder Checklist-Specific (PCLS), and the Clinician-Administered PTSD Scale for DSM-IV (CAPS-IV). Cortisol measurement was performed by automated immunoassay using the VIDAS® system. The results revealed significantly higher cortisol levels in patients who had experienced trauma compared to those without such history. This phenomenon was particularly marked among men. Conversely, among trauma-exposed patients, those suffering from Post-Traumatic Stress Disorder (PTSD) showed significantly lower cortisol levels than those without PTSD. These findings suggest that traumatic life experiences alter the functioning of the HPA axis in psychiatric patients, leading to initial hypercortisolemia followed by hypocortisolemia in cases of PTSD. Identifying these biological profiles could help improve understanding and clinical management of patients in Sub-Saharan Africa.

Keywords: Cortisol, Traumatic Experiences, Post-Traumatic Stress Disorder, Psychiatry, Côte d'Ivoire.

INTRODUCTION

Cortisol, often referred to as the "stress hormone," plays a central role in the body's response to stressors and is involved in numerous physiological functions (energy mobilization, immune modulation, circadian rhythm, cognitive functions, etc.) (Kalafatakis *et al*., 2018; Anliana *et al*., 2025; Knezevic *et al*., 2023). It is the main glucocorticoid secreted by the adrenal glands under the control of the Hypothalamic-Pituitary-Adrenal (HPA) axis (Papadimitriou & Priftis, 2009; Lightman *et al*., 2020). In both clinical and research contexts, blood cortisol level (cortisolemia) is a valuable indicator of the functioning of the stress axis. Alterations of the HPA axis, often reflected by abnormalities in cortisolemia, are among the most frequently observed neurobiological findings in psychiatry. Several studies highlight atypical cortisol profiles in various mental disorders. For example, Zhu *et al*., (2022) reported that mood disorders and schizophrenia are frequently associated with hypercortisolemia or disrupted cortisol rhythms, while, conversely, Pan *et al*., (2020) showed that Post-Traumatic Stress Disorder (PTSD) is often associated with persistent hypocortisolemia. This dysregulation of cortisol can have significant clinical consequences, since abnormal cortisol levels are linked to more severe symptoms, such as suicidal ideation in depression (Peeters *et al*., 2004; Doolin *et al*., 2017; O'Connor *et al*., 2019) and poorer treatment response (Klimes-Dougan *et al*., 2022). The study of cortisol is therefore of major scientific and clinical interest, particularly among patients with mental disorders, in order to better understand the impact of stress and improve patient care.

Furthermore, individuals with psychiatric disorders are very often exposed to traumatic life experiences (violence, abuse, disasters, conflicts, etc.). Research indicates that most patients with severe psychiatric disorders have experienced at least one traumatic event during their lifetime (Ng *et al*., 2023), which can worsen their clinical condition and complicate recovery (Frueh *et al*., 2005; Hogg *et al*., 2022). In Africa in particular, psychological trauma is a major public health issue. For instance, the prevalence of PTSD exceeds 50% in certain populations displaced by conflicts (Tesfaye *et al*., 2024). However, the biological impact of such trauma remains poorly understood in this context. The central issue raised by this research is the extent to which traumatic life experiences influence cortisolemia in psychiatric patients, specifically those treated in Côte d'Ivoire. The importance of this question lies in the high prevalence of trauma within this population and the need to identify potential physiological markers, both to advance neurobiological knowledge and to improve clinical monitoring of traumatized patients.

It is well established that cortisol is a key indicator of stress and that psychiatric disorders are often accompanied by dysregulation of cortisolemia. Individuals who have experienced trauma, in particular, frequently display abnormalities of the stress axis, whether through initial hyperactivation or, more commonly, long-term compensatory hypoactivity (Smeeth *et al*., 2022). However, despite these advances in current knowledge, important gaps remain. Indeed, most studies on cortisol and trauma have been conducted in Western countries or on specific populations (e.g., veterans, victims of targeted violence), and they often focus on a single type of disorder at a time (for example, comparing PTSD patients to healthy controls). In contrast, there is little data on psychiatric patients in Sub-Saharan Africa, who face both high trauma exposure and severe mental illness. In Côte d'Ivoire, for example, the socio-political context (history of conflicts, poverty) may expose the population to various traumas, and limited access to healthcare could amplify the physiological impact of stress (Hossain *et al*., 2014; Bissouma, 2017; Yaro & Kipo-Sunyehzi, 2024; Tesfaye *et al*., 2024). Yet, it remains unknown whether cortisol profiles described in other contexts, such as hypocortisolemia associated with PTSD, are also observed in Ivorian patients, whose cultural, genetic, and environmental specificities may influence this biological marker. Moreover, in patients already suffering from mental disorders (schizophrenia, bipolar disorder, etc.), the additional effect of traumatic life experiences on the HPA axis is unclear: do they exacerbate the existing dysregulation of cortisol? Do they induce specific changes? These questions remain unanswered in the current literature.

In light of this, it is highly relevant to study the effect of traumatic life experiences on cortisolemia in Ivorian psychiatric patients. The general objective of this study is to assess the impact of traumatic life experiences on cortisol profiles in patients treated at the Psychiatric Hospital of Bingerville. To achieve this, the following two specific objectives were pursued:

- To compare blood cortisol levels between patients with a history of traumatic life experiences and those without such a history in this population;
- To determine the specific effect of PTSD on cortisolemia among trauma-exposed patients.

I. MATERIAL AND METHODS

I.1 TYPE AND DURATION OF THE STUDY

The study is cross-sectional, quantitative, and analytical in design. It was conducted in two (02) phases:

1. The first phase, lasting four (04) months, consisted of a clinical assessment of the participants' traumatic life experiences, immediately followed by blood sampling;
2. The second phase, lasting one (01) month, was dedicated to measuring cortisol levels in the collected blood samples.

I.2 MATERIAL

I.2.1 Subjects

This study was conducted in Côte d'Ivoire, involving patients receiving care at the Psychiatric Hospital of Bingerville. It specifically targeted clinically stabilized subjects.

I.2.2 Technical material

I.2.2.1 Data Collection Material

I.2.2.1.1 Trauma History Questionnaire

The Trauma History Questionnaire (THQ), developed by Green (1996) and validated by Hooper *et al*. (2011), is a self-administered questionnaire designed to assess Criterion A1 of Post-Traumatic Stress Disorder (PTSD) according to the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (APA, 1994). It is intended to identify lifetime exposure to one or more traumatic events. This questionnaire lists 24 traumatic events, divided into four categories:

- Criminal acts (items 1 to 4);
- Disasters and general traumatic events (items 5 to 17);
- Physical and sexual violence (items 18 to 23), including forced sexual intercourse, non-consensual touching, or any other form of assault;
- Traumatic events not classified elsewhere (item 24).

For each item, the respondent must indicate whether they have been exposed, and if so, specify the frequency as well as their age at the time of the event.

I.2.2.1.2 PTSD Checklist-Specific

The Posttraumatic Stress Disorder Checklist-Specific (PCLS) is a self-assessment tool developed by Blanchard *et al*., (1996) and later validated in French by

Yao *et al*., (2003). This questionnaire consists of 17 items, each corresponding to a PTSD symptom as defined in the DSM-IV (APA, 1994). The items are grouped into three dimensions reflecting the main PTSD syndromes:

- Intrusion (items 1 to 5);
- Avoidance (items 6 to 12);
- Hyperarousal (items 13 to 17).

Each item is rated from 1 to 5 according to the intensity and frequency of symptoms experienced over the past month. The total score therefore ranges from 17 to 85. In the general population, a score above 44 suggests the presence of clinically significant PTSD (Blanchard, 1996). For individuals already suffering from a psychiatric disorder, a threshold of 42 is recommended due to their greater vulnerability (Grubaugh *et al*., 2006). Although the PCLS is easy to use, it only provides a preliminary assessment. To confirm a PTSD diagnosis, a structured clinical interview remains essential (Paul *et al*., 2013).

I.2.2.1.3 Clinician-Administered PTSD Scale for DSM-IV (CAPS-IV)

Originally, the Clinician-Administered PTSD Scale (CAPS) is a structured clinical interview developed by Blake *et al*., (1990). It was designed to assess the frequency and intensity of PTSD symptoms according to the criteria of the Diagnostic and Statistical Manual of Mental Disorders, 3rd revised edition (DSM-III-R) (APA, 1987). It was later adapted to correspond to the DSM-IV (APA, 1994). Through its standardized questions aligned with DSM-IV diagnostic criteria, the CAPS allows the identification of PTSD, whether current or past. Considered the reference tool for clinicians and researchers in the evaluation of PTSD (Guay & Marchand, 2006), its most recent version, CAPS-V, remains little used. This is explained both by its limited dissemination and, above all, by the controversies surrounding the DSM-V (Cosgrave *et al*., 2006; Demazeux, 2013; Carpenter *et al*., 2010). For these reasons, the CAPS-IV version (Saintonge, 2000) is preferred in the framework of this study.

I.2.2.1.4 Cortisol Assay Device

Cortisol measurement is carried out using the automated VIDAS® system (Vitek® Immuno Diagnostic Assay System), a fully automated multiparametric immuno-analyzer **(Figure 1)**. This system is composed of two main units:

- **Analytical unit:** It includes five independent compartments, each with six positions. These compartments allow simultaneous and automatic immunoassays.
- **Computer unit:** It integrates a computer equipped with:
  - VIDAS PC, a multitasking software that manages subject and test data entry, calibration storage, result display and validation, system operation monitoring and self-tests, as well as patient file and interface management;
  - a dedicated printer for test result reports;
  - a barcode reader.

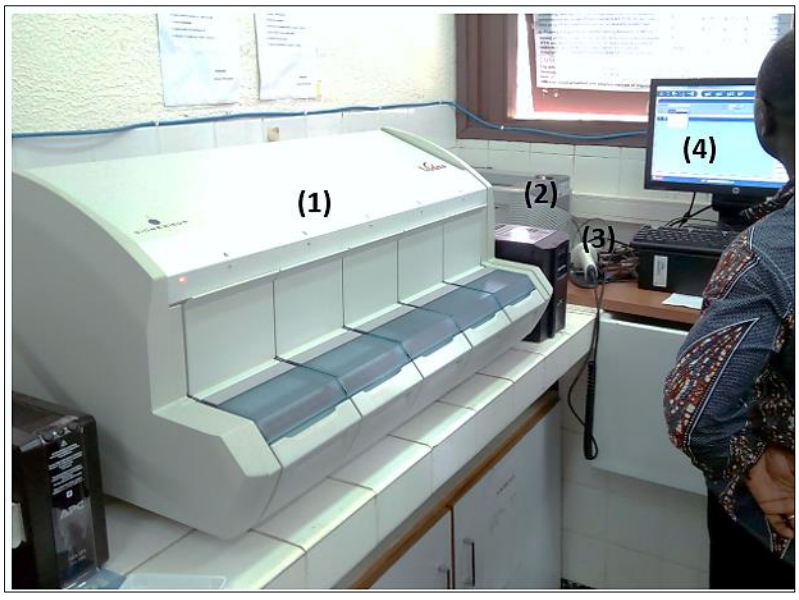

Figure 1: VIDAS Automated System

(1): VIDAS Automaton; (2): Printer; (3): Barcode Reader; (4): Computer screen displaying the VIDAS PC software interface.

I.2.2.1.5 Reagents for Cortisol Assay

Manufactured by Biomérieux SA, the reagents are supplied in the form of a VIDAS® Cortisol S (CORS) reagent kit, ready-to-use for 60 tests. This kit includes the following components:

- 60 reactive strips (STR): Each cartridge contains ten wells sealed under an aluminum sheet and labeled. The label includes a barcode indicating the test code, batch number, and expiration date. The first well is pre-perforated

to facilitate sample introduction, while the last serves as a cuvette for fluorimetric reading. The reagents required for the analysis are distributed among the intermediate wells;

– 60 reactive cones (SPR): Ready-to-use, each cone is pre-coated during manufacture with rabbit polyclonal anti-cortisol immunoglobulins;
– 1 control (C1): Ready-to-use, composed of human serum, cortisol, and sodium azide (1 g/L);
– 1 calibrator (S1): Ready-to-use, also composed of human serum, cortisol, and sodium azide (1 g/L).

I.2.2.2 Data Processing Material

Statistical analyses of the data were performed using XLSTAT (version 2024), an add-in integrated into Microsoft Excel. This tool, directly accessible from the Excel interface, simplifies data processing by allowing intuitive entry of raw data, execution of appropriate statistical analyses, and automatic generation of results in the form of tables and graphs. It also facilitates the application of various statistical tests, all without leaving the spreadsheet environment.

I.3 METHODS

I.3.1 Sampling Method and Sample Description

The sampling process followed two stages. At each stage of the study, a specific sampling method was applied. In the first stage, a sample of 170 subjects was constituted using a convenience sampling method. In the second stage, among the blood samples collected from these 170 subjects, 42 were selected by stratified random sampling, taking into account two stratification criteria: sex and diagnostic category. The diagnostic category was divided into three groups **(Figure 2)**:

- Subjects without traumatic experience (not meeting Criterion A of the CAPS);
- Subjects with traumatic experience but without PTSD (PTSD absent);
- Subjects with traumatic experience and PTSD (PTSD present).

In total, the 42 selected blood samples were divided into three groups of 14 samples each, with a balanced distribution of 7 samples per sex in each group, for cortisol assay.

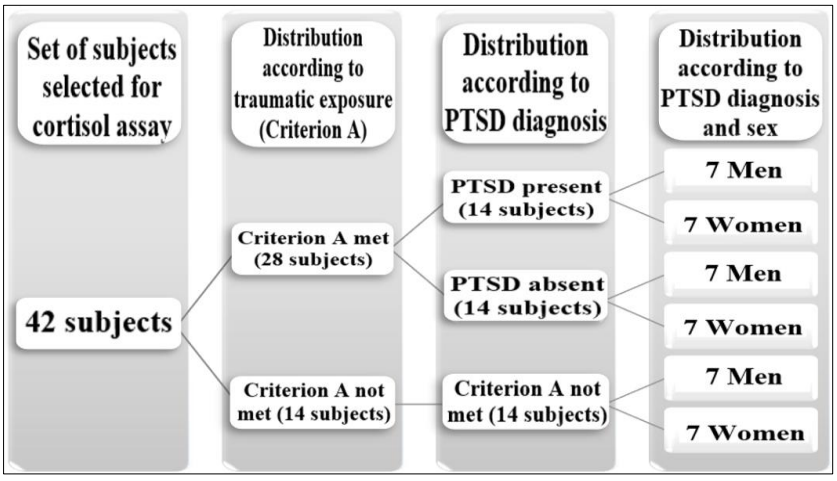

Figure 2: Descriptive diagram of subjects selected for cortisol assay

I.3.2 Method of Subject Selection

I.3.2.1 Inclusion Criteria

Patients included in this study had to meet the following criteria:

- Be a patient monitored and treated at the Psychiatric Hospital of Bingerville, with a clinical condition considered stabilized by the medical team at the time of evaluation;
- Have provided free, voluntary, and informed consent to participate in the study, either personally or through a legal representative;
- Be available for the entire research protocol, including the clinical assessment of traumatic life experiences and the blood sampling for cortisol measurement.

I.3.2.2 Exclusion Criteria

Patients presenting any of the following characteristics were excluded from the study:

- Being in the acute phase of a psychiatric disorder, making it impossible to participate in the assessments or compromising the reliability of the responses;
- Having a known somatic or endocrine pathology (e.g., diabetes, Cushing's syndrome, adrenal insufficiency) likely to alter cortisol secretion;
- Refusing or discontinuing participation in the research protocol, particularly during the data collection phase or at the time of blood sampling.

I.3.3 Data Collection Method

I.3.3.1 Method for Assessing Traumatic Exposures and Post-Traumatic Stress Symptoms

The assessments took place in a quiet consultation room. Each participant was received individually, possibly accompanied by a relative, especially in cases of linguistic difficulty. Three questionnaires were used for these evaluations. First, the THQ was administered to identify the most traumatic life event experienced by the subject. Once this event was determined, the resulting post-traumatic distress was measured using the PCLS, and a global score was calculated immediately. This score was interpreted according to a threshold set at 42, in line with the recommendations of Grubaugh *et al*., (2006) for psychiatric patients. If the score was below this threshold, the subject was considered not to have experienced a significant traumatic event. Conversely, if the score was equal to or greater than 42, the subject was considered to have been exposed to a major traumatic event. In such cases, an in-depth assessment of Post-Traumatic Stress Symptoms (PTSS) was conducted using the CAPS-IV, in order to establish an accurate diagnosis.

I.3.3.2 Cortisol Assay Method

The measurement of cortisol in blood serum samples is carried out according to a strict protocol, detailed as follows:

- **Preparation of Reagents and Equipment:** The necessary reagents are removed from the refrigerator. For each sample, one cone and one CORS cartridge are prepared, as well as for the control or the S1 calibrator. It is essential to ensure that the cone package is properly resealed after each use.
- **Identification and Preparation of Samples:** The test, the calibrator (used in triplicate), and the control are identified respectively by the codes « CORS », « S1 » and « C1 ». The calibrator, the control, and the serum samples are homogenized using a vortex mixer.
- **Sampling and Installation:** A volume of 100 microliters is taken for the calibrator, the control, and each sample. The CORS cones and cartridges are then installed in the analyzer, ensuring that the codes (colors and letters) on the labels match.
- **Launching the Analysis:** The analysis is started without delay. All subsequent steps are automated by the device.
- **Post-Pipetting Management:** After pipetting, the microtubes are sealed and stored at a temperature between 2 and 8 °C.
- **Reading Results and Cleaning:** The results are available after approximately 40 minutes of analysis. The cones and cartridges used are removed from the analyzer and disposed of in an appropriate container.
- **Data Recording:** The results are recorded digitally and printed.

The concentration of cortisol in the blood is expressed in nanograms per milliliter (ng/ml).

I.3.4 Data Processing Method

The statistical analysis of the data was adapted to the objectives of the study. First, an analysis compared cortisol levels between subjects exposed to traumatic events (meeting criterion A) and those not exposed (not meeting criterion A). This comparison was carried out initially on the entire sample, then by distinguishing the results according to sex. The second analysis compared cortisol levels among the three diagnostic categories defined during the second stage of subject sampling.

The results of the analyses are presented as box plots and subjected to statistical tests. For bivariate analyses, the Student's t-test for independent samples was used to compare two groups, while one-way ANOVA was applied to compare three groups. For three-way analyses, a one-way ANOVA was first performed, followed by Student's t-tests stratified by sex. The significance threshold was set at $\alpha = 0.05$, and significance was indicated by asterisks.

I.3.5 Ethical Considerations

The present study was conducted in accordance with basic ethical principles applicable to research involving human subjects. Several key documents were established to ensure the protection of participants and the scientific validity of this study:

- **Research Authorization Request:** Addressed to the Director of the Psychiatric Hospital of Bingerville (HPB), this request obtained the institution's prior approval to carry out the study on site;
- **Research Authorization:** Issued by the Director of the HPB, it officially validated the research protocol and authorized access to participants and clinical data;
- **Information Sheet:** Provided to participants, it outlined the objectives, methods (including blood sampling for cortisol measurement), benefits, and risks of the study, thereby ensuring informed participation;
- **Consent Form:** Signed by each participant (or their legal representative, if applicable), it formalized their free and informed agreement, while specifying their right to withdraw at any time.

Furthermore, the anonymity and confidentiality of the data were strictly maintained throughout the study. All collected information was coded and processed in such a way as to prevent any individual identification.

II. RESULTS AND DISCUSSION

II.1 RESULTS

II.1.1 Analysis of Cortisol Levels According to Traumatic Experiences

Analysis of cortisol levels in relation to traumatic life experiences shows that mean cortisol was markedly higher in participants who had experienced trauma (120.44 ± 47.41 ng/ml) compared with those without such experiences (68.56 ± 18.56 ng/ml). This difference was statistically significant according to the Student's t-test (p = 0.000) **(Figure 3)**. When sex was considered in the analysis, results indicated a significant interaction (ANOVA: p = 0.000). Male participants with a history of trauma exhibited the highest cortisol levels (156.23 ± 34.75 ng/ml), significantly greater than those of the three other categories combined, whose mean values were relatively similar. Further sex-specific comparisons showed that women exposed to traumatic experiences had significantly higher mean cortisol levels (84.65 ± 26.92 ng/ml) than those without trauma exposure (58.99 ± 19.00 ng/ml) (Student's t-test: p = 0.040). A similar pattern was observed among men: traumatized men displayed a mean cortisol level of 156.23 ± 34.75 ng/ml, which was significantly higher than that of their non-traumatized counterparts (74.14 ± 11.90 ng/ml) (Student's t-test: p = 0.000) **(Figure 4).**

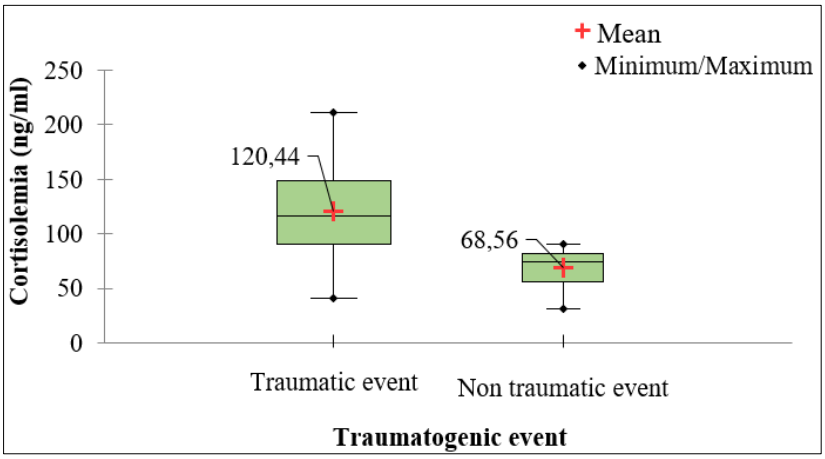

Figure 3: Cortisol levels of subjects according to the characteristic of their main traumatogenic life experience

Student's t-test: p = 0.000 (significant difference between the two mean cortisol levels observed).*

The life event identified by the subject in the THQ is considered traumatic when the subject obtains a PCLS score ≥ 42. In this case, the subject meets Criterion A1 of the PTSD diagnosis according to the DSM-IV (exposure to a traumatic event) and is assessed with the CAPS for the diagnosis of PTSD.

The traumatic event is considered to have a traumatic nature when the subject meets Criterion A2 of the PTSD diagnosis according to the DSM-IV, meaning that the subject's reaction to the traumatic event involved one or more negative emotions such as intense fear, helplessness, horror, or distress.

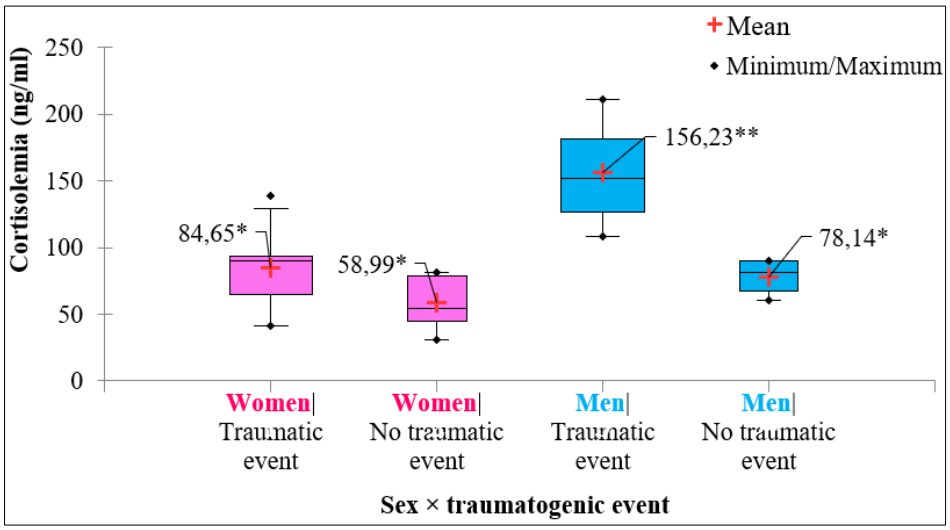

Figure 4: Cortisol levels of subjects according to sex and the characteristic of their main traumatic life experience

ANOVA: p = 0.000 (at least one of the mean cortisol levels observed differs significantly from the three others);*
Independent Student's t-test (Women): p = 0.040 (significant difference between the mean cortisol levels observed in women);*
Independent Student's t-test (Men): p = 0.000 (significant difference between the mean cortisol levels observed in men).*

II.1.2 Analysis of Cortisol Levels According to PTSD Diagnosis

The analysis of cortisol levels according to PTSD diagnosis first shows that the mean cortisol level is significantly higher (ANOVA: p = 0.000) in subjects without PTSD (145.13 ± 44.46 ng/ml) compared to subjects with PTSD (95.76 ± 36.05 ng/ml) and those without traumatic experience (68.56 ± 18.52 ng/ml) **(Figure 5).** Furthermore, comparisons by sex and PTSD diagnosis indicate that the two highest mean cortisol levels are found in men who experienced traumatic events that did not lead to PTSD (184.11 ± 24.16 ng/ml) and in men with PTSD (128.36 ± 16.66 ng/ml). Regarding sex-based comparisons, the results show that the mean cortisol level among women who experienced traumatic events without PTSD (106.15 ± 18.16 ng/ml) is significantly higher (ANOVA: p = 0.000) compared to that observed in the other two groups of women. Similarly, the mean cortisol level in men without PTSD (184.11 ± 24.16 ng/ml) is significantly higher (ANOVA: p = 0.000) compared to that observed in the other two groups of men. Finally, it is noted that for each category of the variable "PTSD diagnosis," the mean cortisol level is higher in men than in women **(Figure 6).**

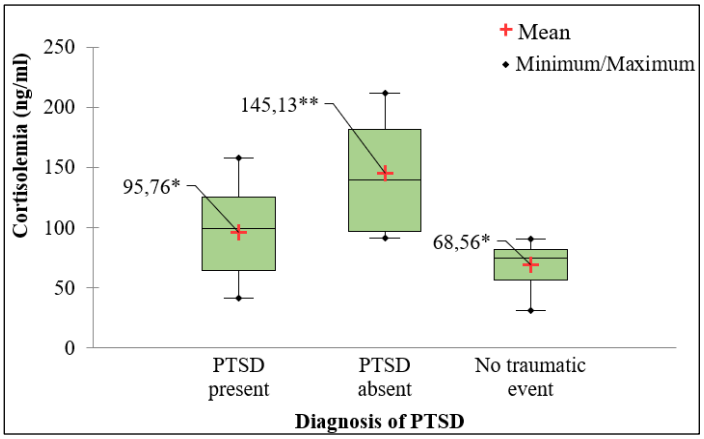

Figure 5: Cortisol levels of subjects according to their PTSD diagnosis

ANOVA: p = 0.000 (at least one of the mean cortisol levels observed differs significantly from the three others).*

A subject is considered to suffer from PTSD if he or she has been exposed to a traumatic event (criterion A) and has, for at least one month after the event (criterion E), the three (03) characteristic post-traumatic syndromes:

- *intrusions and re-experiencing (Criterion B);*
- *avoidance and emotional numbing (Criterion C);*
- *hypervigilance and neurovegetative hyperarousal (Criterion D).*

In addition, these symptoms must cause the subject severe disruption of psychological and socio-professional balance (Criterion F). In short, a subject suffers from PTSD if he or she meets criteria A, B, C, D, E, and F of the PTSD diagnosis as defined by the DSM-IV and CAPS-IV.

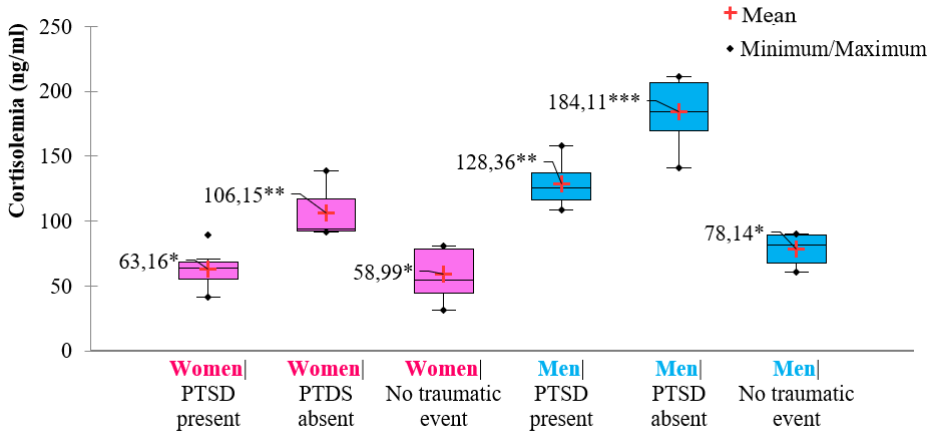

Figure 6: Cortisol levels of patients according to sex and PTSD diagnosis

ANOVA (Overall sample): p = 0.000 (In the overall sample, at least one of the mean cortisol levels differs significantly from the five others);*

ANOVA (Women): p = 0.000 (Among women, at least one of the mean cortisol levels differs significantly from the three others);*

ANOVA (Men): p = 0.000 (Among men, at least one of the mean cortisol levels differs significantly from the three others).*

II.2 DISCUSSION

The present study analyzes the impact of traumatic experiences on variations in blood cortisol levels among patients treated at the Psychiatric Hospital of Bingerville, Côte d'Ivoire. The discussion is structured around two main points: the effect of the traumatic experiences themselves, and the effect of a PTSD diagnosis on cortisolemia.

II.2.1 Influence of Traumatic Experiences on Subjects' Cortisol Levels

According to the analysis of cortisolemia based on traumatic life experiences, subjects who had lived through traumatic events showed significantly higher cortisol levels compared with subjects not exposed to such events. In other words, the recollection of a psychological trauma triggers an acute rise in circulating cortisol levels in these subjects, reflecting an intense stress response. This result suggests that the resurgence of traumatic memory, maintained by the amygdala rather than properly regulated by the hippocampus, provokes a neuroendocrine survival reaction similar to that triggered at the moment of the initial trauma (Salmona, 2018; Roesler *et al*., 2021; Ben-Zion *et al*., 2023). For example, a former combatant exposed to the sound of an explosion may instantly relive the associated war scene. His hypothalamic-pituitary-adrenal (HPA) axis is then abruptly activated, releasing cortisol to prepare his body for the perceived danger. This reactive hypercortisolemia, observed among the traumatized patients in the present study, aligns with findings in the literature. Indeed, Djouini *et al*. (2014), in a study of road accident victims in Algeria, demonstrated that men who experienced physical trauma exhibited significantly higher cortisol levels than uninjured controls at various points following the accident. This phenomenon may be explained by the physiological imprint left by trauma on the stress-response system (Bremner, 2006). Any later exposure to a stimulus associated with the trauma is enough to trigger a substantial hormonal surge, exceeding that observed in individuals who had not experienced such events (Van Der Kolk, 1994; Seo *et al*., 2018).

The results of this study further indicate that this elevation in cortisol levels is more pronounced in men who experienced trauma compared with women. In acute stress situations, men tend to produce more cortisol than women, as reported by numerous previous studies (Reschke-Hernández *et al*., 2016; Lovallo *et al*., 2019; Paris *et al*., 2009). In our sample, traumatized men exhibited on average nearly twice the cortisol levels of their female counterparts. This gap may be explained by differences in hormonal regulation and HPA axis reactivity between the sexes. Indeed, studies confirm that post-stress cortisol profiles are generally higher in men. For example, Liu *et al*., (2017), in a meta-analysis of 34 studies including 1,350 participants and using the Trier Social Stress Test (TSST), concluded that men reached significantly higher peaks of salivary cortisol than women. Male sex hormones (androgens) may raise the threshold of stress tolerance while promoting a stronger hormonal response, whereas estrogens may down-regulate cortisol secretion through more effective feedback mechanisms in women (Moisan, 2021; Zuloaga *et al*., 2024). For instance, in laboratory stress tests, men typically show a much steeper rise in cortisol (an average increase of about 15 nmol/l) compared with women subjected to the same test (around 7 to 8 nmol/l) (Reschke-Hernández *et al*., 2016; Kirschbaum & Hellhammer, 1994). This sexual dimorphism in stress reactivity corroborates the observations of the present study and highlights the importance of considering gender in the analysis of neuroendocrine mechanisms following trauma.

Having established the direct effect of traumatic life experiences on cortisolemia in the study population, it is now necessary to examine the extent to which a formal PTSD diagnosis modulates this hormonal response. This second analysis refines the understanding of the neuroendocrine mechanisms associated with trauma.

II.2.2 Influence of PTSD diagnosis on Subjects' Cortisol Levels

The analysis of cortisol levels according to PTSD diagnosis reveals that patients actually suffering from PTSD display overall lower basal cortisol levels than trauma-exposed patients without PTSD (or trauma-free control subjects). This result is consistent with many previous comparative studies, which reported hypocortisolemia in PTSD patients compared with non-traumatized controls or trauma-exposed but resilient individuals (Yehuda, 2006; Pan *et al*., 2018). For example, Pan *et al*., (2018) found significantly lower average morning salivary cortisol levels in PTSD patients than in control groups (standardized difference of about - 0.3, $p = 0.022$), confirming the trend of hypocortisolemia in PTSD. Similarly, Dekel *et al*., (2017) reported that trauma survivors who developed chronic PTSD had significantly lower basal cortisol concentrations than comparable survivors without PTSD. This paradoxical reduction in cortisol, sometimes described as "counterintuitive" given the intense stress associated with PTSD, is now explained by neurobiological adaptations of the HPA axis. Indeed, repeated or prolonged exposure to extreme stress is associated with hypersécrétion of hypothalamic corticotropin-releasing hormone (CRH) and sensitization of glucocorticoid receptors in key brain regions (notably the hippocampus) (Yehuda, 2006; Herman *et al*., 2016; Stratilov *et al*., 2024). As a result, the negative feedback mechanism of cortisol on the HPA axis becomes excessively effective in PTSD patients: the slightest increase in cortisol rapidly inhibits axis activity, hence a lower-than-expected baseline circulating level (Yehuda, 2006). To illustrate this phenomenon, dexamethasone suppression tests of the corticotropic axis can be cited. Numerous studies have shown that when a

low dose of dexamethasone (a synthetic glucocorticoid) is administered in the evening, PTSD patients exhibit greater suppression of endogenous cortisol the following morning compared with control subjects (Yehuda *et al*., 1993; Lange *et al*., 2005; Wingenfeld *et al*., 2007; Michopoulos *et al*., 2017). This hyperactive suppression of the HPA axis reflects the heightened sensitivity of corticosteroid receptors and corresponds to the chronic hypocortisolemia observed in these patients.

The results of this study also highlight that within the PTSD population, women have even lower basal cortisol levels than men with PTSD. This finding supports the hypothesis of increased female vulnerability to PTSD, a phenomenon widely demonstrated in epidemiological research (Meewisse *et al*., 2007; Olff, 2017; Pan *et al*., 2020). Indeed, women are about twice as likely to develop PTSD during their lifetime as men, despite often experiencing lower or different overall exposure to traumatic events (Olff *et al*., 2007; Haskell *et al*., 2010). For example, Olff *et al*., (2007) reported lifetime prevalence rates of about 10 - 12% for PTSD in women compared to 5 - 6% in men, across all types of trauma. Several biological factors may explain this disparity. It is suspected that the female HPA response, modulated by estrogens, promotes faster post-trauma feedback, leading to more marked hormonal depletion (relative hypocortisolemia), whereas androgens in men tend to limit this feedback and maintain slightly higher cortisol levels (Gill *et al*., 2005; Hellhammer *et al*., 2008). In practice, it is therefore not surprising to observe PTSD female patients in our cohort with very low cortisol levels, likely reflecting an inability to mobilize sufficient amounts of this hormone in the face of chronic stress. This pathophysiological feature could contribute to the greater chronicity and severity of symptoms observed in many women with PTSD (Olff *et al*., 2007).

Finally, it should be emphasized that other uncontrolled factors in the present study could influence cortisol levels independently of trauma or PTSD. For example, age is known to modulate cortisol secretion (older subjects tend to have higher basal cortisol levels) (Wilkinson *et al*., 2001; Yiallouris *et al*., 2019). Similarly, the use of psychotropic medications (antidepressants, anxiolytics, neuroleptics) may alter HPA axis dynamics (Subramaniam *et al*., 2019; Romanova *et al*., 2022). The lack of strict control of these confounding variables represents a limitation of this work. Future research in Côte d'Ivoire or elsewhere would benefit from including these factors in order to better isolate the specific effect of psychological trauma and PTSD on neuroendocrine functioning.

CONCLUSION

The present study aimed to analyze the influence of traumatic life experiences on cortisol levels among patients monitored at the Psychiatric Hospital of Bingerville. The findings reveal that individuals who had experienced trauma showed a significant increase in cortisol levels compared with those who had not. This reactive hypercortisolemia reflects the lasting imprint of trauma on the HPA axis, particularly in men, whose neuroendocrine response proved more pronounced. Conversely, the analysis of PTSD diagnosis indicates an opposite trend. Indeed, patients with PTSD exhibited lower basal cortisol levels than trauma-exposed patients without PTSD, thus confirming the hypocortisolemia described in the literature. These observations highlight the complexity of the neuroendocrine mechanisms associated with psychological trauma, where an initial phase of stress system hyperactivity may evolve into chronic hypoactivity in established cases of PTSD.

These results emphasize the importance of considering cortisol levels as a useful biomarker for understanding and clinically monitoring psychiatric patients exposed to trauma, while taking into account sex differences and differential diagnoses. They also suggest integrating biological evaluation into care protocols, alongside neuropsychological and pharmacological approaches.

Finally, it would be relevant to extend this work with studies exploring in greater detail the neurophysiological and neuroendocrine mechanisms underlying these variations in cortisol levels. The use of tools such as dexamethasone suppression tests, longitudinal monitoring of the circadian rhythm of cortisol, or combined neuroimaging and hormonal assays would deepen the understanding of stress-axis adaptations in the African context. Such perspectives would contribute to enriching knowledge of trauma biology and to developing more personalized care strategies for psychiatric patients in Côte d'Ivoire.

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