Neuropsychiatric Disease and Treatment

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ORIGINAL RESEARCH

Pro-Inflammatory Biomarkers And Depression: A Study Performed In A Group Of Health Sciences University Students

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Introduction: Depression is one of the most prevalent mental illnesses worldwide, and its etiology is still the subject of multiple studies that strive to clearly identify the causes of this disorder and guide the appropriate form of treatment. The present study aimed to establish the correlation between elevated levels of circulating proinflammatory cytokines such as interleukin 6 (IL-6) and tumor necrosis factor-alpha (TNF-α) with depression.

Materials and Methods: An analytical correlational study was conducted in which 300 medicine and nursing students from a university in the city of Loja, Ecuador participated. They were assessed with the Beck Depression Inventory II (BDI II) to determine the presence of depression, and peripheral levels of interleukin 6 (IL-6) and tumor necrosis factor-alpha (TNF-α) were measured using the ELISA technique.

Results: 65% of the participants reported depression according to the Beck Depression Inventory II, which in turn were divided into mild (23%), moderate (20.3%) and severe depression (21.7%) according to the score obtained. A statistically significant correlation was found between elevated levels of IL-6 and TNF-α with depression in the group of nursing students. However, no such correlation was found in the group of medical students.

Discussion: Elevations in both inflammatory cytokines measured in this study were evident in both study groups. This could be explained by the high emotional stress generally associated with healthcare career training, which in turn could reinforce the hypothesis of inflammatory states and their relation to the pathophysiology of depression. However, the percentage of depression cases was higher in the nursing student group, in which the female gender accounted for 83.3% of the group, a fact that could explain the reason why in this group there was a statistically significant relationship between elevated cytokines and the presence of depression.

Keywords: depression, cytokines, neuroinflammation, interleukin 6, tumor necrosis factor-alpha

Introduction

Depression is one of the most common mental disorders worldwide. According to the World Health Organization, 3.8% of the global population suffers from this condition. It is more prevalent among individuals who have experienced abuse, severe loss, or prolonged intense stress, making them more susceptible to developing it.[1]

In Ecuador, in 2015, approximately 2.088 people received medical care for depression in healthcare facilities. However, many cases of depression in the country continue to go unnoticed, leading to underreporting. Those who suffer from it often become accustomed to living in a state of persistent sadness and hopelessness, alternating with brief relatively calm periods. A significant percentage, however, ultimately end up in suicide. According to national statistics by 2023, 4.9% of deaths among young people aged 18 to 29 in Ecuador were due to self-inflicted injuries.[2]

Received: 28 May 2025
Accepted: 30 July 2025
Published: 5 September 2025

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However, the studies carried out so far in Ecuador have focused on determining the frequency of depression or its correlation with other variables such as anxiety and suicide risk in particular. Very few studies have been carried out on university students, and even fewer have established a correlation between elevated levels of proinflammatory cytokines and depression in university students of healthcare careers, a population group that is known to experience continuous and mounting stress levels due to the academic demands and emotional burdens inherent to these careers.

Despite being one of the most significant mental health disorders worldwide, the causes of this condition are still under investigation. In recent years, new theories have emerged that provide a better understanding of its complex pathophysiology; one of these is the neuroinflammation theory, which has proposed an alternate explanation of the role of systemic inflammatory processes, triggered by either an innate or acquired immune response, in the development of disorders such as depression.

Clearly understanding the causes of this disease will help down the road to develop effective prevention and treatment strategies, allowing for timely interventions and potential full recoveries. Taking all these facts into account, the objective of this study was proposed as finding if there is a correlation between elevated levels of circulating proinflammatory cytokines such as interleukin 6 (IL-6) and tumor necrosis factor-alpha (TNF-α) and depression.

Materials and Methods

A cross sectional analytical correlational study was conducted. The study population consisted of 1200 medical and nursing students from a university in Loja – Ecuador.

Sample: the sample size was calculated using the finite population formula with a 95% confidence interval, a Z value of 1.96 and a margin of error of 5%, alpha significance level (α) of 0.05 and a statistical power of 80%. The total calculated sample was 291 participants, with an additional 9 participants included to account for potential losses, resulting in a final sample of 300 participants (150 from medical school and 150 from nursing school).

Inclusion criteria were applied: presently being a regularly enrolled student in the medical or nursing schools and duly signing of an informed consent. Exclusion criteria included students undergoing medical treatment with corticosteroids at the time of the study, also those with an immunosuppressive medical condition, and individuals belonging to vulnerable groups (elderly adults, children and adolescents, pregnant women, people with disabilities, individuals deprived of liberty, or those suffering from catastrophic or highly complex diseases).

The Project was approved by the Ethics Committee of the San Francisco General Hospital of the IESS (Ecuadorian Social Security Institute) under approval number N°18, following the ethical guidelines of the WHO (World Health Organization) and the Declaration of Helsinki as ethical principles for medical research. Prior to sample collection, informed consent was obtained from each participant.

To assess the presence of depression, the Beck Depression Inventory II was used. This is a self-report questionnaire consisting of 21 Likert type items. The original version was developed by Aaron Beck and has since become one of the most widely used instruments for detecting and assessing the severity of depression. Its items describe the most common clinical symptoms experienced by individuals with depression. This clinical tool is designed to evaluate both the presence and severity of depression in adults and adolescents aged 13 and older, with a sensitivity of 94% and a specificity of 92%.[3]

It is a self-administered inventory consisting of 21 items. In each item, the respondent must choose from four alternatives, rated from 0 to 3 points, ordered from least to greatest severity, based on the statement that best describes their mood over the past two weeks. The test evaluates symptoms associated with depression, such as mood variations, changes in habits, and somatic conditions. Once completed, the total score ranges from 0 to 63, with interpretation based on the following categories: 0–13 (no depression), 14–19 (mild depression), 20–28 (moderate depression), and 29–63 (severe depression).

After administering the Beck Depression Inventory II, blood samples were collected to assess inflammatory cytokines IL-6 and TNF– α in serum using the ELISA technique, following the instructions of the DIAsource IL-6-ELISA Kit and

DIAsource TNF-α- ELISA Kit. The reference values for these cytokines were as follows: TNF-α: 0–7,2 pg/mL; IL-6: 0–5 pg/mL.[4,5]

Statistical Analysis: Data normalization was performed using the Kolmogorov–Smirnov test. Levene's test was used to assess homoscedasticity. Spearman correlation test and the Chi-square test were applied for the analysis of non parametric data, respectively. A p value <0.05 was considered statistically significant. The statistical analysis was conducted using IBM SPSS Statistics, Version 20.0.[6]

Results

The study population consisted of 300 university students from medical school and nursing school. The average age was 21.19 years among the participants, 71.66% were women while 28.33% were men (In nursing school, 81.3% of the students were of female gender, while in medicine, only 61.3% of the students were of female gender).

Depression was reported in 65% of participants (Table 1). A statistically significant correlation was found between elevated levels of proinflammatory cytokines and depression in the nursing student group, but it was not found in the medical student group (Tables 2 and 3).

Table 1 Beck Depression Inventory-II

		Frequency	Percentage %
Valid	No depression	105	35
	Mild depression	69	23
	Moderate depression	61	20.3
	Severe depression	65	21.7
	Total	300	100

Notes: In Table 1 it can be observed that only 35% of the study population showed no signs of depression, while the remaining 65% exhibited varying levels of depression, ranging from mild to moderate and severe, with percentages fluctuating between 20% and 23%.

Table 2 Correlation Between Inflammatory Cytokines IL-6 and TNF-α and Depression in Nursing Students

Variable	With Depression	Without Depression	Total	p value
Normal IL-6	61	18	79	
Elevated IL-6	43	28	71	0.023*
Total	104	46	150	
Normal TNF-α	35	12	47	
Elevated TNF-α	69	34	103	0.039*
Total	104	46	150	
*p ≤0.05 (Spearman's Rho)				

Notes: Table 2 shows a statically significant relationship between depression and elevated IL-6 and TNF-α levels in the group of nursing students.

Table 3 Correlation Between Inflammatory Cytokines IL-6 and TNF-α and Depression in Medical Students

Variable	With Depression	Without Depression	Total	p value
Normal IL-6 Elevated IL-6 **Total**	61 30 91	46 13 59	107 43 150	*0.101
Normal TNF-α Elevated TNF-α **Total**	47 44 91	33 26 59	80 70 150	0.160
*p ≤0.05				

Notes: Table 3 shows that in the study group corresponding to medical students, no statistically significant correlations were found between elevated IL-6 and TNF-α levels and depression.

Discussion

High levels of depression (mild, moderate and severe) were observed in more than half of the study population. This finding is consistent with the results of studies conducted by Ngin et al,[7] who reported similar percentages of depression among university students in Cambodia (2018). Their study also found an association between persistent depression and poor academic performance, unhealthy eating habits, limited physical activity, among other factors.

Similarly, the findings align with a study conducted on university students at the University of Santiago de Compostela by Blanco et al, where considering various types of depression from mild to very severe, the prevalence reached 48%, along with equally alarming levels of anxiety (2021). Additionally, Volken et al reported that the prevalence of depression was 43% among French students and 53.7% among Bangladeshi students.[8]

The high percentage of depression reported in this study is noteworthy not only due to the prevalence of this condition itself but also, as indicated by Anbesaw et al,[9] because of the consequences that mental health issues have on university students. These consequences range from poor academic performance and an increased risk of dropping out of the university to higher consumption of alcohol, tobacco, and other drugs, engagement in risky sexual behaviors, physical inactivity, deterioration of physical health, self-harming behaviors and a greater risk of suicide, among others. These challenges are not uncommon in our context, particularly in healthcare career training, where high academic demands, combined with the emotional strain of interacting with ill patients in stressful hospital environments, exposure to frankly hostile treatment from patients, patient's relatives and even peers, and highly competitive atmospheres, contribute to the onset and persistence of conditions such as depression. Additionally, the very limited hours of rest and proper night sleep required to meet academic demands, further exacerbate the stress building situations.[10]

A similar setting is explained by Zhang et al, who reported that academic stress is one of the most significant sources of chronic stress among university students. Academic stress is a psychological state experienced by students as a result of continuous social and self-imposed pressure in a school environment, leading to depletion of their psychological reserves (2020). This condition often results in feelings of frustration and hopelessness, which are particularly common among students in healthcare careers such as medicine and nursing.[10]

However, these results contrast with the study conducted by Anbesaw et al, where the depression rates in both studies are around 28%[9] It is important to note that the first study used the PHQ-2 (Patient Health Questionnaire), which identifies depressive symptoms, while the second study is a systematic review. In our study, we applied the Beck Depression Inventory II, which allows for detecting and assessing the severity of depression. This difference in methodology may have influenced the variation in results between the studies. Additionally, our study only included medical and nursing students, whereas Anbesaw's study included university students in general from Germany and Ethiopia, covering all academic fields. As explained by Milicet al, (2024), healthcare career students tend to exhibit higher levels of depression compared to the general population as these fields are academically, psychologically and emotionally demanding,[11] due to multiple risk factors such as an adverse academic environment, the competitive nature

of the field, academic overload and increasing exposure to suffering and the reality of death, also certain neurotransmitters like serotonin can be affected potentially leading to depression in students pursuing health related careers as indicated in the study by Dhanoa et al.[12]

Nevertheless, the objective of this study was not only to determine the prevalence of depression in the study group but also to explore its origins. In this regard, previous research has shown that monoamine depletion such as serotonin depletion is not sufficient to cause depression in a healthy individual. Likewise, sustained serotonin depletion does not necessarily worsen depressive symptoms in untreated patients. This suggests that other mechanisms are involved in the pathophysiology of depression, including the GABAergic and glutamatergic systems, increased levels of pro-inflammatory cytokines and adrenal glucocorticoids, to hippocampal volume reduction and a chronic low-grade inflammatory state mediated by elevated levels of inflammatory interleukins such as IL-6 and TNF-α.[13] This aligns with the findings of our study, which demonstrated a statistically significant correlation between elevated serum levels of IL-6 and TNF-α, and depression among nursing students (Table 2). We believe this can be explained by the high levels of chronic stress experienced by students of healthcare careers, which lead to an increase in pro-inflammatory cytokines and the presence of conditions such as depression.

Authors such as Kim et al, who state that inflammatory cytokines play a role in depression by triggering an inflammatory response in the brain, explain this. This response interferes with the activity of neurotransmitters such as adrenaline, serotonin and dopamine, affecting the functions of the hypothalamic-pituitary-adrenal (HPA) axis. As a result, early physical symptoms of depression may appear, including generalized and nonspecific pain, fatigue, appetite disturbances, reduced physical activity, sleep disorders, decreased academic performance and lower work productivity. Furthermore, they demonstrated statistical significance of the findings observed in their study. (Table 2).[14–16]

In this regard, Beurel et al state that in the central nervous system, microglial cells function as immune cells and depending on stimuli can induce neuroinflammation. This process is mediated by cytokines produced not only by microglial cells but also by other immune system cells, including inflammatory cytokines such as interleukin-6, tumor necrosis factor-alpha, interferons, and interleukin-10, among others. This inflammatory response disrupts neuronal functions, leading to impaired neurotransmitter signaling, as well as interference with the synthesis and reuptake of serotonin, contributing to the development of depression.[17] In fact, this inflammatory state also induces cellular apoptosis, increases oxidative stress, and causes metabolic disorders that impact neuroplasticity and neuronal function.[18–21] Regarding to this, researchers such as Li et al and Ruiz et al state that the brain contains two types of microglial cells: type 1 (M1), which produce inflammatory cytokines, and type 2 (M2), which produce anti-inflammatory cytokines. Therefore, the imbalance in M1/M2 activity not only influences brain inflammation but the persistence of the depressive disorders.[22,23]

This link between depression and inflammatory processes is also highlighted by other researches, such as Zhan et al, who reported elevated IL-6 and TNF-α level in major depressive disorders. These elevations are associated with hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis, leading to increased corticosteroid secretion and subsequent dysfunction of immunocompetent cells. This dysfunction primarily affects brain function, particularly the turnover of 5-hydroxy-tryptamine (serotonin), reducing its availability in synaptic clefts, impairing neuroplasticity and contributing to depression.[24]

Nevertheless, as explained by researchers such as Harsanyi et al, while elevated levels of pro-inflammatory cytokines can be found in depression, individuals with additional clinical conditions may, on the contrary, exhibit decreased levels of these cytokines. This occurs due to the activation and subsequent suppression of the immune response, which normally functions as part of the body's homeostatic process. This phenomenon may explain the findings of the present study (Tables 2 and 3) where nursing students showed a statistically significant correlation between elevated IL-6 and TNF-α levels and depression, whereas this correlation was not observed in medical students.[25] Furthermore, as indicated by Paganin et al and Baurel et al, it is the persistence of the inflammatory process mediated by these cytokines that contributes to the continuation of depressive symptoms, failure of the antidepressant response and the emergence of atypical depression symptoms.[17,26,27]

This variability in the results between the two study groups could be explained by the fact that the participants were university students in health sciences who were apparently healthy. However, the study reported not only the presence of

depression but also elevated levels of pro-inflammatory cytokines.[27] This leads us to consider the possibility that unidentified health issues in this population may be primarily a response to sustained psychological stress rather than stress of organic origin, particularly in the case of nursing students (Table 2). Meanwhile, medical students may exhibit a compensatory homeostatic response (Table 3).

We also considered the interesting fact that in our study group, in nursing students there is a predominant feminine population (81.3%) compared to medical students (61.3%), and according to the study of Milić J, et al;[11] they also found an increased level of depression in women compared to men.

It should also be noted that one of the limitations of this study is its cross-sectional design, which did not include other variables that could affect the stress response, such as the development of a greater degree of resilience in female medical students compared to male medical students, which would also explain the difference observed between the two groups. Likewise, other variables such as sleep or stress levels, which can also influence elevated levels of proinflammatory cytokines and depression, were not included in this study.

Finally comparing the activities related to nursing and medical training, we considered that the much more intimate relationship at the human level that nursing students tend to have with their patients compared to medical students, could perhaps lead to higher chronic stress levels which could account for the higher levels of elevated cytokines and depression in this group.

Based on the findings presented, the relationship between the inflammatory process driven by the production of pro-inflammatory cytokines such as IL-6 and TNF-α and the onset and persistence of depression is evident, as demonstrated in this study among nursing students. Nevertheless, the results of this study are not conclusive, highlighting the need for further research on this topic in larger populations while considering a broader range of variables.

Conclusions

The prevalence of depression found in this research study was 65% of the study population, categorized into mild depression (23%), moderate depression (20.3%), and severe depression (21.7%).

A statistically significant correlation was observed between elevated levels of IL6 y TNF-α and depression among nursing students. However, no such correlation was found in the group of medical students.

Therefore, the correlation between elevated levels of inflammatory cytokines and depression remains inconclusive.

Studies are Required with a Larger Population and a Similar Proportion of Men and Women in the Study Groups.

Ethics

The authors confirm that the research presented in this article met the ethical guidelines, including adherence to the legal requirements, of Ecuador and received approval from the Ethics Committee of the San Francisco General Hospital of the IESS (Ecuadorian Social Security Institute) under approval number N°18.

Funding

The Vice-Rectorate for Research of Universidad Técnica Particular de Loja funded the purchase of reagents and supplies for the determination of Interleukin-6 and tumor necrosis factor-alpha cytokines.

Disclosure

The authors have no conflicts of interest to report.

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