International Journal of Research and Review
[Indexed, Peer-Reviewed Journal]
Volume 13; Issue: 9; September 2026
Website: www.ijrrjournal.com
Review Paper *E-ISSN: 2349-9788; P-ISSN: 2454-2237*

Phase Angle and Psychological Stress: A Review of Physiological Mechanisms, Cellular Health and Mental Well-being

Dr Kavita Gupta[1], Dr Lalita Chaudhary[2], Dr Megha Kulshreshtha[3], Dr Alka Srivastava[4], Dr Sanjana Chahar[5]

[1]Senior Resident, Department of Physiology, UNS ASMC, ABVP, Jaunpur, India.
[2]Principal, Department of Physiology, MV, ASMC, ABVP, Bijnor, India
[3,4]Associate Professor, Department of Physiology, LLRM, CCSU, Meerut, India.
[5]Junior Resident, Department of Physiology, LLRM, CCSU, Meerut, India.

Corresponding Author: Dr Kavita Gupta

DOI: https://doi.org/10.52403/ijrr.20260919

ABSTRACT

Psychological stress is increasingly recognised as an important factor influencing both mental and physical health. Persistent activation of the hypothalamic–pituitary–adrenal (HPA) axis and sympathetic nervous system (SNS) during chronic stress elevates cortisol and catecholamine levels, promoting oxidative stress, inflammation, and alterations in cellular hydration and membrane integrity. These physiological changes may be reflected by phase angle (PhA), a bioelectrical impedance analysis (BIA)-derived parameter that serves as a non-invasive indicator of cell membrane integrity, body cell mass, and overall cellular health. Emerging evidence suggests that lower PhA values are associated with poor nutritional status, reduced muscle quality, chronic inflammation, and several disease conditions. More recently, researchers have explored its relationship with mental well-being, reporting reduced PhA in individuals experiencing psychological distress, anxiety, and depression. The observed decline in PhA is believed to result from stress-induced shifts in intracellular and extracellular fluid balance, impaired cellular function, and loss of membrane integrity. As PhA integrates nutritional, metabolic, and physiological information, it may provide valuable insight into the biological consequences of psychological stress beyond conventional psychological assessments. This narrative review summarises the physiological mechanisms linking psychological stress with alterations in phase angle, examines current evidence supporting PhA as a potential biomarker of mental well-being, discusses its clinical relevance, and highlights future research directions for incorporating phase angle into comprehensive mental health assessment and monitoring.

Keywords: Bioelectrical impedance, Phase angle, Psychological Stress, Mental well-being, Cellular health, Stress biomarkers

1. INTRODUCTION

Mental health is a state of well-being that enables people to cope with the stresses of life, realise their abilities, learn and work well, and contribute to their community(1). Mental well-being is vital to overall health,

supporting clear thinking, emotional balance, effective coping, healthy relationships, and productive functioning. However, increasing academic, occupational, financial, and social demands have made psychological stress a common experience in modern life. Psychological stress arises when individuals perceive that the demands placed upon them exceed their ability to cope. Although short-term stress can enhance motivation and performance, persistent or excessive stress can negatively affect mental well-being. Chronic psychological stress is associated with anxiety, depressive symptoms, sleep disturbances, impaired concentration, emotional exhaustion, and a reduced quality of life. It may also contribute to physical health problems, including cardiovascular disease, obesity, diabetes, and impaired immune function. Conversely, good mental well-being, supported by healthy lifestyle habits, regular physical activity, relaxation practices, and strong social relationships, improves resilience and the ability to cope with stress. Thus, mental well-being and psychological stress share a dynamic, bidirectional relationship, with each significantly influencing the other.

The number of people diagnosed with mental disorders has risen significantly over the years (2). Globally, more than a billion people live with a mental health condition(1). In 2019, about 970 million people worldwide suffered from a mental disorder, with anxiety and depression being the most common, which together accounted for more than two-thirds of all mental health conditions in 2021. The prevalence of mental disorders varies by sex, with females experiencing a higher rate than males(3). The burden of psychological stress has increased considerably in recent years, affecting individuals of all ages and backgrounds. Modern lifestyle changes, increasing work and academic demands, financial uncertainty, and social pressures have contributed to higher levels of stress in daily life. When stress becomes persistent, it can negatively influence both mental and physical health, reducing overall well-being and increasing the likelihood of stress-related disorders. These growing concerns highlight the need for greater attention to stress prevention and early intervention strategies.

In recent years, Phase Angle (PhA), obtained through bioelectrical impedance analysis, has attracted increasing attention as a simple, non-invasive indicator of cellular health and overall physiological status(4) (5) . Unlike conventional anthropometric measures, PhA reflects cell membrane integrity, body cell mass, and the distribution of intracellular and extracellular fluids, making it a valuable marker of nutritional status, physical fitness, disease severity, and clinical outcomes(6) . Growing evidence indicates that psychological stress produces measurable physiological changes by activating the body's stress response systems. Prolonged stress can disrupt hormonal balance, promote inflammation and oxidative stress, alter fluid distribution, and impair cellular integrity and function. Since PhA reflects cell membrane integrity, body cell mass, and cellular health, the stress-induced physiological alterations may be accompanied by changes in PhA values. Consequently, PhA has emerged as a potential objective marker for evaluating the biological effects of chronic psychological stress. Investigating the relationship between psychological stress and Phase Angle may provide valuable insights into the physiological basis of mental well-being and support the use of PhA as a non-invasive biomarker for the early identification and monitoring of stress-related health changes.

2. BIOELECTRICAL IMPEDANCE ANALYSIS AND PHASE ANGLE

2.1 Bioelectrical Impedance Analysis: Principle and Measurement of Phase Angle

PhA is a bioelectrical parameter derived from bioelectrical impedance analysis (BIA) that reflects the electrical properties of body tissues. It provides an indirect measure of

cell membrane integrity, body cell mass, and the balance between intracellular and extracellular water(4) (5). Higher Phase Angle values generally indicate healthy cell membranes, better cellular function, and good nutritional and functional status, whereas lower values may suggest impaired cellular integrity, reduced body cell mass, inflammation, or underlying disease. Because it is a rapid and non-invasive PhA has gained increasing recognition as a valuable biomarker for assessing overall health, nutritional status, physical performance, and prognosis in various clinical and non-clinical populations.

The physiological basis of BIA is founded on the electrical properties of biological tissues. Biological tissues do not behave as simple electrical conductors because they comprise conductive intra- and extracellular fluids separated by phospholipid cell membranes that possess capacitive properties. Consequently, the human body can be represented as an equivalent electrical circuit, commonly described by the Cole–Cole model, which incorporates both resistance and capacitance to explain the flow of alternating current through tissues(4). This model provides the theoretical framework for understanding impedance components, including resistance and reactance, and forms the basis for the derivation of phase angle as an indicator of cellular integrity and body composition.

BIA is a widely used, non-invasive field method for assessing body composition, which measures the electrical characteristics of the human body either at 50 kHz (single-frequency BIA) or at several frequencies in the range 1–1000 kHz (multifrequency BIA and BIS = bioimpedance spectroscopy) (4). The biological tissues have important electrical properties such as conductivity, resistivity and permittivity. These properties depend on the movement of ions and the presence of cell membranes. When an electrical current passes through the body, tissues oppose the flow of current and can also store electrical charge. Because of these properties, tissues behave like electrical circuits. Understanding them helps in techniques such as bioelectrical impedance analysis, which is used to assess body composition and health status. The technique was originally developed to provide data-driven prediction of total body water (TBW) and fat free mass (FFM) or lean body mass (LBM) based on a two-compartment model of the human body. This technique is generally considered to provide clinically acceptable predictions of body composition.

When a low-amplitude alternating electrical current is passed through the body during BIA, it encounters different biological tissues that influence its flow according to their electrical properties. Body fluids, which are rich in water and electrolytes, conduct electricity readily, whereas fat and bone offer greater opposition because of their lower water content. This opposition to current flow is known as resistance and primarily reflects the body's hydration status. In addition to resistance, the electrical current also interacts with cell membranes, which are composed of phospholipid bilayers that behave as biological capacitors. Rather than allowing the current to pass instantly, these membranes temporarily store electrical charge and release it with a slight delay, producing reactance. Consequently, body tissues function like countless microscopic resistor-capacitor circuits, where extracellular and intracellular fluids serve as conductive pathways and cell membranes provide capacitive properties. The combined effect of resistance and reactance is termed impedance, which represents the total opposition to the flow of alternating current through the body(4). Because the capacitive effect of cell membranes causes a delay between the applied current and the resulting voltage, a measurable angular difference known as the phase angle is generated. Phase angle is calculated from the ratio of reactance to resistance and reflects both the quantity and functional integrity of cell membranes as well as the distribution of body water. Hence, PhA is an

indirect marker of cellular health that reflects cell membrane integrity, body cell mass (BCM), and the balance between intracellular and extracellular water. Healthy cells with intact membranes and adequate intracellular water exhibit higher Phase Angle values, whereas aging, malnutrition, inflammation, oxidative stress, chronic disease, and psychological stress can reduce membrane integrity and intracellular water, leading to lower Phase Angle. Consequently, PhA has emerged as a non-invasive biomarker of nutritional status, cellular function, and overall physiological health, with growing evidence supporting its potential role in assessing the physiological impact of chronic psychological stress.

2.2 Device-Related Variability in Phase Angle Measurement

Phase angle measurements obtained through bioelectrical impedance analysis may vary according to the characteristics and configuration of the BIA device used(7) (8). Differences in measurement frequency, single- versus multifrequency technology, electrode configuration, current pathway, body position, and the algorithms applied by different analysers can influence resistance and reactance and, consequently, the derived PhA (6) (9). Comparative studies have demonstrated that PhA values obtained from different BIA systems may not be directly interchangeable, with some devices showing substantial differences despite assessing the same individuals(9) (10). Variations have also been reported between supine and standing measurements and with changes in electrode placement, emphasizing the importance of a consistent measurement protocol(11). **P**hA has been reported to be higher in the supine than in the standing position(8) (10). Electrode type, anatomical placement, and the side of the body assessed can also produce measurable differences in PhA. In addition, hydration status, recent food and fluid intake, physical activity, bladder volume, skin temperature, ambient temperature, time of measurement, and duration of rest before assessment may modify tissue conductivity and fluid distribution, resulting in variability in BIA parameters(6) (11). Therefore, for reliable comparison of PhA values, measurements should preferably be performed using the same device, at a consistent time of day, under standardised conditions, with the same body position and electrode configuration, following an appropriate period of rest and controlled food, fluid, and exercise intake. Therefore, clinical and research studies should preferably use the same BIA analyser, measurement frequency, electrode configuration, body position, and standardised pre-measurement conditions throughout the study. Device-specific reference values and appropriate equipment calibration should also be considered when interpreting PhA, particularly when comparing results across studies or populations(7) (12). Standardised PhA measurement protocols are essential for obtaining reliable and comparable results in clinical research and for accurately monitoring low PhA and treatment-related changes. It has prognostic value in health and disease(10).

2.3 Determinants and Factors Affecting Phase Angle

Phase angle is influenced by multiple physiological and pathological factors that reflect cellular health, body composition, and fluid distribution. Since phase angle is derived from the relationship between resistance and reactance, any condition that alters cell membrane integrity, body cell mass, or the balance between intracellular and extracellular water can modify its value. Therefore, understanding the determinants and influencing factors of phase angle is essential for its accurate interpretation in both healthy individuals and clinical populations.

At the cellular level, the electrical properties of biological tissues are primarily determined by their water content, electrolyte concentration, structural macromolecules, and metabolically active

cell mass. These components collectively influence tissue conductivity and membrane capacitance, thereby affecting the resistance and reactance measured during bioelectrical impedance analysis.

Body water and inorganic ions constitute the primary conductive medium within the body. Approximately 60% of adult body weight consists of water, although this proportion varies with age, sex, and adiposity. Water itself is a poor conductor; however, its dissolved electrolytes, including sodium, potassium, chloride, calcium, and magnesium, enable the transmission of electrical current. Consequently, body fluids with higher ionic concentrations exhibit greater electrical conductivity than relatively pure water. Differences in total body water, such as the lower water content typically observed in women because of higher body fat percentage, can influence impedance measurements and ultimately affect PhA(6). Adequate cellular hydration increases PhA, while dehydration and fluid imbalance lower it(13).

Earlier studies have shown that organic cellular components contribute substantially to the electrical characteristics of tissues. Phospholipid-rich cell membranes function as biological capacitors by temporarily storing electrical charge, thereby generating reactance. Similar capacitive properties are exhibited by the membranes of intracellular organelles, further contributing to the overall electrical behaviour of cells. Membrane-associated proteins, ion channels, and transporters regulate ion movement across the membrane and maintain cellular electrical activity. Structural alterations resulting from ageing, oxidative stress, chronic disease, or abnormal protein accumulation may impair membrane integrity, reduce membrane capacitance, and consequently lower PhA. Intracellular energy substrates such as glycogen and creatine also indirectly enhance conductivity by promoting intracellular water retention and preserving muscle cell integrity.

To the present date, most evidence shows that higher levels of PhA are conceptually associated with increased Body Cell Mass (BCM). BCM accounts for nearly 20–55% of body mass and is considered one of the strongest physiological determinants of PhA because it represents the body's metabolically active cell compartment. Approximately 85% of BCM consists of intracellular water, making it highly conductive to electrical current. An increase in BCM enhances intracellular conductivity and is generally associated with higher PhA values(6). However, PhA reflects not only the quantity of body cell mass but also its functional quality. Healthy muscle cells with intact membranes generate greater reactance owing to their superior capacitive properties, whereas muscle wasting, ageing, inflammation, or disease impairs membrane integrity and lowers PhA. Although adipocytes are included within BCM, their relatively low water content and limited conductive pathways result in a much smaller contribution to conductivity than skeletal muscle. Therefore, PhA should be interpreted as a comprehensive marker of cellular integrity, hydration status, and metabolically active body cell mass rather than as a direct measure of muscle or adipose tissue alone.

Beyond the intrinsic cellular determinants, PhA is influenced by a wide range of physiological and pathological factors that modify body composition, hydration status, cellular integrity, and membrane function. These factors include age, gender, ethnicity, BMI, fat free mass , Physical activity, athelets, nutrition, infection, inflammation, disease , oxidative stress , psychological stress and smoking(13) (6) (14) (15) (5) .

Gender also influences PhA, with males generally exhibiting higher values than females due to their greater skeletal muscle mass, body cell mass, and intracellular water content. Although PhA increases in both sexes after puberty, the increase is more pronounced in males**(6)** (16).

Ethnicity is another physiological factor contributing to variations in PhA.

Differences in body composition and fluid distribution among ethnic populations influence bioelectrical properties. For example, lower PhA values have been reported in Japanese individuals, which may be attributed to a higher extracellular water-to-total body water ratio and lower skeletal muscle mass. In contrast, higher PhA values observed in Mexicans compared with Germans have been associated with greater fat-free mass related to differences in body size and height (16) (17).

Nutritional status has a significant impact on PhA and is widely recognised as an indicator of functional nutritional status (18). Adequate nutrition preserves body cell mass, intracellular water, and cell membrane integrity, thereby maintaining higher PhA values. Conversely, disease-associated malnutrition promotes a shift of fluid from the intracellular to the extracellular compartment, increasing the extracellular water-to-body cell mass ratio and reducing PhA (14). In healthy adults, phase angle values typically range between 5° and 7°**,** whereas values below 5° have been associated with malnutrition (19) (15).

Physical activity is positively associated with phase angle, as regular exercise promotes skeletal muscle mass, muscle strength, and body cell mass while preserving cellular integrity. These physiological adaptations are accompanied by increased intracellular water content and improved membrane function, leading to higher PhA values. Consequently, physically active individuals generally exhibit higher PhA than sedentary individuals, reflecting better body composition and overall cellular health(20) (21) (22) (16).

Pathological conditions such as infection, inflammation, oxidative stress, psychological stress, chronic diseases and cancer, adversely affect PhA by impairing cell membrane integrity, reducing body cell mass, and altering intracellular–extracellular fluid distribution. These changes modify tissue resistance and reactance, resulting in lower PhA values that reflect compromised cellular health and function.

3. PSYCHOLOGICAL STRESS: PHYSIOLOGICAL AND CELLULAR EFFECT

Stress has a different meaning for different people under different circumstances. In 1936, the term Stress was coined by Hans Selye, which means ‘the non-specific response of the body to any demand for change’(23) . Cannon coined the term the flight or fight to describe animal’s response to threat(24). It is also known as the acute stress response, which leads to activation of sympathetic nervous system (SNS). This response was later recognized as first stage of General Adaptation Syndrome (GAS), which was postulated by Hans Selye. Another definition in terms of neuroendocrinology: Eugene Yates defined stress any stimulus that will provoke the release of ACTH and adrenal glucocorticoids(24).

Stress is experienced by everyone at some point in their lifespan. Stress affects both mental and physical health. Stress often triggers emotional responses such as anxiety, frustration, or irritability. It may also reduce our ability to concentrate and think clearly. When stress continues for a long time, it can aggravate existing health conditions and increase the likelihood of relying on alcohol, tobacco, or other substances as coping mechanisms. Ongoing or intense stress can also contribute to the development or worsening of mental health disorders, particularly anxiety and depression, which often require appropriate medical or psychological support. In many cases, a mental health condition develops when stress-related symptoms persist over time and begin to interfere with everyday activities, including work, education, and personal responsibilities.

Many scientists have noticed that the stress system contains several elements, such as a stressful stimulus, a stressor, and a stress response(25). Stressors are the factors that affect the internal environment and vary

widely from acute to chronic stressors. By repetitive and prolonged exposure to stressors, the stress response leads to maladaptation, leading to detrimental effects on health. Chronic stressors, in particular, can provoke maladaptive responses such as depression, anxiety, cognitive impairment, and cardiovascular disease(26). A specific stressor may potentially bring about specific local stress, while the intensity of stress beyond a threshold may commonly activate the hypothalamic-pituitary-adrenal(HPA) axis and result in a systemic stress response(25). Selye defined "GAS" as, in fact, both stress response and stress effect: the activation of the HPA axis is a systemic stress response, while physical and mental disorders produced by prolonged stress are stress effects (25). There are two different types of stress in humans, such as psychological stress and physiological stress(27).

Psychological stress arises when an individual perceives emotional, cognitive, or social demands as exceeding their ability to cope, leading to feelings such as anxiety, fear, frustration, or emotional tension. Adverse life experiences, such as poverty, losing a job, serious illness, or the death of a loved one, can negatively affect psychological well-being (28). In contrast, physiological stress results from physical challenges such as infection, injury, strenuous exercise, inflammation, surgery, or exposure to extreme environmental conditions, which activate protective biological responses. Although psychological and physiological stressors differ in their origin, both stimulate interconnected neuroendocrine and autonomic pathways that help the body maintain homeostasis(29).

Stress may be classified as acute or chronic depending on its duration and persistence. Acute stress occurs during a particular time or event and is isolated to that incident. It includes events like a near-miss car accident, or preparing for an important presentation at work. Common symptoms of acute stress include: heart palpitations, shortness of breath, feeling lightheaded, headaches, stomach pain or indigestion, sweating and chest pain. Acute stress disorder symptoms occur within 3 days to 1 month of the stressor and last at least 3 days (according to Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5))(30). Once the stressor is removed, physiological functions generally return to baseline, minimising adverse effects. Chronic stress is ongoing. Chronic stress could increase or decrease in severity but is relatively consistent in its presence, in which stressors persist for weeks, months and even years. Common symptoms of chronic stress include: isolation or emotional withdrawal, low energy, aches and pains, trouble sleeping, trouble staying focused and change in appetite. Persistent activation of the HPA axis and sympathetic nervous system results in sustained elevations of stress mediators, promoting chronic inflammation, oxidative stress, metabolic disturbances, and impaired immune regulation(31). Over time, these alterations contribute to structural and functional changes in multiple organ systems and increase the risk of cardiovascular disease, diabetes, depression, and other chronic disorders. Consequently, while acute stress is often adaptive and protective, chronic stress represents a maladaptive state that can significantly compromise overall health and well-being.

The stress response is initiated in the brain when sensory signals indicating a potential threat are processed by the amygdala, which activates the hypothalamus, which is the master command center for homeostasis to initiate downstream neuroendocrine and autonomic responses. Hypothalamus regulate functions such as body temperature, hunger, thirst, sleep, stress, and reproduction. When someone experiences a stressful event, the amygdala, an area of the brain that contributes to emotional processing, sends a distress signal to the hypothalamus (32). The hypothalamus activates the autonomic nervous system (ANS) and HPA axis through corticotropin

releasing hormone (CRH) release, along with vasopressin (ADH) secretion (33) (34). The sympathetic and parasympathetic nervous systems are the two components of the ANS. The sympathetic system drives the rapid fight-or-flight response, whereas the parasympathetic system promotes rest, digestion, energy conservation, and recovery (35). Threat perception activates the amygdala–hypothalamic pathway, triggering sympathetic stimulation of the adrenal medulla and rapid epinephrine release. Epinephrine rapidly enhances cardiovascular and respiratory activity while mobilising glucose and fatty acids to meet the increased energy demands of acute stress(36) . The stress response is further coordinated through activation of the HPA axis, resulting in cortisol secretion(37) (38) Cortisol helps sustain the body's response to prolonged stress by maintaining blood glucose levels, supporting cardiovascular function, and ensuring a continuous supply of energy to essential organs. As long as the brain perceives the stressor to be present, cortisol secretion remains elevated, keeping the body in a state of heightened readiness. Once the threat has resolved, cortisol levels gradually decline, and the parasympathetic nervous system (PNS) becomes dominant. This system slows heart rate, reduces blood pressure, conserves energy, and restores normal physiological function, thereby returning the body to a state of homeostasis.
Psychological stress activates the HPA axis and sympathetic-adrenal-medullary(SAM)axis, increasing cortisol and epinephrine release(39). Although stress hormones support acute adaptation, their prolonged elevation during chronic stress can disrupt cellular and physiological functions (31). Epinephrine and cortisol mobilise energy substrates and enhance metabolic activity; prolonged stress may increase reactive oxygen species (ROS) generation and promote oxidative stress(40) (41). Excessive ROS initiate lipid peroxidation of membrane phospholipids and damage membrane proteins, including ion channels, transporters, and receptors, thereby disrupting membrane structure and function(42) (43). At the same time, chronic cortisol exposure impairs the repair and replacement of damaged membrane components, allowing cellular injury to accumulate(44). Persistent stress is also associated with low-grade inflammation, characterised by increased production of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumour necrosis factor-α (TNF-α), which further amplify oxidative damage(41) (45) (46). Collectively, these changes compromise cell membrane integrity, alter intracellular water distribution, and reduce the capacitive properties of the cell membrane. Since PhA derived from bioelectrical impedance analysis reflects cell membrane integrity and body cell mass, chronic stress-induced oxidative damage and impaired membrane function may contribute to a reduction in PhA. It is important to note that cortisol and epinephrine do not directly damage the cell membrane; rather, their chronic elevation indirectly affects membrane integrity through oxidative stress, inflammation, and impaired cellular repair.

4. LINK BETWEEN PHASE ANGLE AND PSYCHOLOGICAL STRESS

Recent studies indicate a possible relationship between phase angle and mental health, particularly depression, anxiety, and psychological distress(47). Together, fear- and anxiety-based psychiatric disorders are at the same time the most prevalent and the most costly of mental health disorders(48). Anxiety and depressive disorders are highly prevalent mental health conditions that often coexist and arise from interacting biological, psychological, and social factors(49). Anxiety is a normal stress response, but anxiety disorders involve excessive and persistent fear or worry that can impair daily functioning (50). By causing persistent fear and excessive worry, anxiety disorders can impair multiple aspects of life, including personal relationships, social participation, educational achievement, and professional

performance. In 2021, 359 million people in the world had an anxiety disorder, making anxiety disorders the most common of all mental disorders (50). According to the World Health Organisation (WHO), anxiety disorders are the world's most common mental disorders (50). These disorders include generalised anxiety disorder (GAD), panic disorder, social anxiety disorder, separation anxiety disorder, agoraphobia, specific phobias, selective mutism, and other specified anxiety disorders. More women are affected by anxiety disorders than men(50). Depressive disorder is a common mental disorder. It involves a depressed mood or loss of pleasure or interest in activities for long periods of time(51). Approximately 332 million people in the world have depression. Depression is about 1.5 times more common among women than among men(51).

Prolonged exposure to stressful stimuli that elicit fear and anxiety in PTSD, GAD, PD, and phobias activates both central and peripheral immune cells to release cytokines (48) . Psychological stress engages the hypothalamic PVN and LC–noradrenergic system, activating the HPA and SAM axes and increasing cortisol and catecholamine release. Persistent activation of these pathways may contribute to maladaptive physiological changes(52) (53).

Psychological stress activates the HPA axis and SNS, increasing cortisol and catecholamines. These hormones then communicate with immune cells and regulate inflammatory responses(54). Immune cells, including monocytes, macrophages, neutrophils, dendritic cells, T lymphocytes, B lymphocytes, and natural killer cells, express glucocorticoid and adrenergic receptors, allowing them to respond directly to stress hormones(55) .

Under acute conditions**,** cortisol generally acts as a feedback mechanism that limits excessive inflammation by suppressing pro-inflammatory cytokine production such as TNF-α, IL-1β, and IL-6, and suppressing the activity of pro-inflammatory transcription factors such as nuclear factor-kappa B (NF-κB) and activator protein-1 (AP-1), thereby limiting the production of inflammatory cytokines and supporting inflammatory control and homeostasis(56). Glucocorticoids can inhibit inflammatory signalling and promote mechanisms that resolve inflammation. However, chronic psychological stress is different**.** Persistent exposure to cortisol can impair glucocorticoid receptor sensitivity, a phenomenon known as glucocorticoid resistance, which reduces the ability of immune cells to respond to cortisol's anti-inflammatory actions and simultaneously, persistent sympathetic activation through β-adrenergic receptor signalling further alters immune cell function and promotes the release of inflammatory mediators(57) (58). Repeated stress has been associated with altered cytokines, microglial activation, and changes in immune-cell activity(54). Consequently, NF-κB signalling remains active, leading to sustained production of pro-inflammatory cytokines, including tumour necrosis factor-α (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6), thereby establishing a state of chronic low-grade inflammation(57) .

Prolonged psychological stress can disturb the redox balance by increasing ROS production and reactive nitrogen species (RNS), primarily through activation of enzymes such as NADPH oxidase, xanthine oxidase, and inducible nitric oxide synthase, as well as increased mitochondrial oxidative activity(59). When the production of these reactive molecules exceeds the capacity of endogenous antioxidant defences, oxidative stress develops. This provides a bridge between chronic stress and cellular injury(60). Excess ROS can attack the polyunsaturated fatty acids in membrane phospholipids, causing lipid peroxidation. This can alter membrane fluidity, permeability, lipid–protein interactions, ion transport, and membrane signalling(61). There is also experimental evidence that chronic psychological stress can alter membrane fatty-acid composition**.** A rat study found changes in platelet membrane

fatty acids associated with chronic psychological stress and described the connection between oxidative/inflammatory processes and membrane phospholipid oxidation(62). Progressive oxidation of membrane lipids and proteins reduces membrane fluidity, increases membrane rigidity, and compromises the structural and functional integrity of the plasma membrane(63). Oxidative injury also extends to mitochondrial membranes, resulting in impaired ATP synthesis, dysfunction of energy-dependent ion pumps, intracellular ionic imbalance, and activation of apoptotic pathways(64). Collectively, these alterations contribute to loss of cellular integrity and body cell mass. Since the phase angle measured by bioelectrical impedance analysis primarily reflects cell membrane integrity and cellular health, chronic stress-induced inflammation and oxidative membrane damage may reduce membrane capacitance and reactance, ultimately leading to lower phase angle values(65). These mechanisms support the potential role of phase angle as an indirect biomarker of cellular dysfunction associated with chronic psychological stress.

5. CLINICAL APPLICATIONS AND POTENTIAL IMPLICATIONS

Phase angle (PhA) is a non-invasive indicator of cellular integrity, body cell mass, and fluid balance that may reflect physiological changes associated with psychological stress. The available evidence indicates that prolonged psychological stress can disrupt cellular homeostasis through sustained activation of the HPA axis and SNS, resulting in inflammation, oxidative stress, and alterations in membrane function. These physiological disturbances are often accompanied by a reduction in PhA, suggesting that this parameter may serve as an indirect marker of stress-related cellular dysfunction. Thus, PhA may have potential as an indirect marker for monitoring stress-related physiological changes. It offers a practical approach for evaluating overall physiological status in both healthy individuals and patients with chronic diseases. In addition, lower PhA has been associated with poorer nutritional status, reduced functional capacity, and adverse clinical outcomes, supporting its potential role in risk assessment. Serial PhA measurements may also complement nutritional and rehabilitation monitoring by tracking changes in cellular health and recovery. However, PhA is influenced by age, sex, body composition, hydration, and disease status; therefore, further prospective studies are needed to establish its specific association with psychological stress and determine its usefulness for stress assessment and longitudinal monitoring. Such evidence could support the use of PhA in stress assessment and personalised healthcare.

6. FUTURE DIRECTIONS

Future research should establish the direct relationship between psychological stress and phase angle through large-scale, longitudinal studies across diverse populations. Mechanistic studies should explore how neuroendocrine activation, inflammation, oxidative stress, mitochondrial dysfunction, and membrane alterations influence PhA. Combining PhA with stress-related measures such as cortisol, inflammatory cytokines, and heart rate variability may strengthen its interpretation. Standardised reference values based on age, sex, ethnicity, body composition, and health status are also needed. Finally, research should assess PhA responses to stress-reducing interventions and integrate it with physiological and digital health measures to determine their potential for stress monitoring, risk assessment, and personalised healthcare.

Declaration by Authors

Ethical Approval: Not applicable
Acknowledgement: None
Source of Funding: None
Conflict of Interest: No conflicts of interest declared.

Abbreviations-

BIA-Bioelectrical Impedance Analysis
PhA- Phase Angle
HPA Axis- Hypothalamic–Pituitary–Adrenal Axis
ANS- Autonomic Nervous System
SNS- Sympathetic Nervous System
PNS- Parasympathetic Nervous System
TBW- Total Body Water
FFM- Fat Free Mass
LBM- Lean Body Mass
BCM- Body Cell Mass
GAS- General Adaptation Syndrome
ACTH- Adrenocorticotrophin Hormone
CRH- Corticotropin-Releasing Hormone
ADH- Antidiuretic Hormone

REFERENCES

1. Mental health [Internet]. [cited 2026 Jul 3]. Available from: https://www.who.int/news-room/fact-sheets/detail/mental-health-strengthening-our-response
2. Mental health. In: Wikipedia [Internet]. 2026 [cited 2026 Jul 3]. Available from: https://en.wikipedia.org/w/index.php?title=Mental_health&oldid=1362298843
3. World mental health today: latest data [Internet]. [cited 2026 Jul 24]. Available from: https://www.who.int/publications/i/item/9789240113817
4. Ward LC, Brantlov S. Bioimpedance basics and phase angle fundamentals. Rev Endocr Metab Disord. 2023;24(3):381–91. doi:10.1007/s11154-022-09780-3 PubMed PMID: 36749540; PubMed Central PMCID: PMC10140124.
5. Vincenzo OD, Marra M, Scalfi L. Bioelectrical impedance phase angle in sport: a systematic review. Journal of the International Society of Sports Nutrition. 2019 Nov 6; 16:49. doi:10.1186/s12970-019-0319-2 PubMed PMID: 31694665.
6. Rosa GB, Lukaski HC, Sardinha LB. The science of bioelectrical impedance-derived phase angle: insights from body composition in youth. Rev Endocr Metab Disord. 2025;26(4):603–24. doi:10.1007/s11154-025-09964-7 PubMed PMID: 40208410; PubMed Central PMCID: PMC12316808.
7. Dellinger JR, Johnson BA, Benavides ML, Moore ML, Stratton MT, Harty PS, et al. Agreement of bioelectrical resistance, reactance, and phase angle values from supine and standing bioimpedance analyzers. Physiol Meas. 2021 Apr 6;42(3). doi:10.1088/1361-6579/abe6fa PubMed PMID: 33592586.
8. Jensen B, Braun W, Both M, Gallagher D, Clark P, González DL, et al. Configuration of bioelectrical impedance measurements affects results for phase angle. Med Eng Phys. 2020 Oct 1;84(1):10–5. doi: 10.1016/j.medengphy.2020.07.021 PubMed PMID: 32977906; PubMed Central PMCID: PMC12781646.
9. Dupertuis YM, Jimaja W, Beardsley Levoy C, Genton L. Bioelectrical impedance analysis instruments: how do they differ, what do we need for clinical assessment? Curr Opin Clin Nutr Metab Care. 2025 Jul 15;28(5):379–87. doi:10.1097/MCO.0000000000001142 PubMed PMID: 40667712; PubMed Central PMCID: PMC12337901.
10. Bellido D, García-García C, Talluri A, Lukaski HC, García-Almeida JM. Future lines of research on phase angle: Strengths and limitations. Rev Endocr Metab Disord. 2023;24(3):563–83. doi:10.1007/s11154-023-09803-7 PubMed PMID: 37043140; PubMed Central PMCID: PMC10090740.
11. Kyle UG, Bosaeus I, De Lorenzo AD, Deurenberg P, Elia M, Manuel Gómez J, et al. Bioelectrical impedance analysis—part II: utilization in clinical practice. Clinical Nutrition. 2004 Dec 1;23(6):1430–53. doi: 10.1016/j.clnu.2004.09.012
12. Bosy-Westphal A, Danielzik S, Dörhöfer RP, Later W, Wiese S, Müller MJ. Phase angle from bioelectrical impedance analysis: population reference values by age, sex, and body mass index. JPEN J Parenter Enteral Nutr. 2006;30(4):309–16. doi:10.1177/0148607106030004309 PubMed PMID: 16804128.
13. da Silva1 BR, Orsso1 CE, Gonzalez2 MC, Sicchieri3 JMF, Mialich3 MS, Jordao3 AA, et al. Phase angle and cellular health: inflammation and oxidative damage. Rev Endocr Metab Disord. 2023;24(3):543–62.

doi:10.1007/s11154-022-09775-0 PubMed PMID: 36474107; PubMed Central PMCID: PMC9735064.

14. Franco-Oliva A, Ávila-Nava A, Rodríguez-Aguilar EA, Trujillo-Mercado A, García-Guzmán AD, Pinzón-Navarro BA, et al. Association between phase angle and the nutritional status in pediatric populations: a systematic review. Front Nutr. 2023 Jun 21; 10:1142545. doi:10.3389/fnut.2023.1142545 PubMed PMID: 37415913; PubMed Central PMCID: PMC10320581.
15. Małecka-Massalska T, Popiołek J, Teter M, Homa-Mlak I, Dec M, Makarewicz A, et al. Application of phase angle for evaluation of the nutrition status of patients with anorexia nervosa. Psychiatr Pol. 2017 Dec 30;51(6):1121–31. doi:10.12740/PP/67500
16. Cancello R, Brunani A, Brenna E, Soranna D, Bertoli S, Zambon A, et al. Phase angle (PhA) in overweight and obesity: evidence of applicability from diagnosis to weight changes in obesity treatment. Rev Endocr Metab Disord. 2023 Jun;24(3):451–64. doi:10.1007/s11154-022-09774-1 PubMed PMID: 36484943; PubMed Central PMCID: PMC9735068.
17. Ethnic differences in fat and muscle mass and their implication for interpretation of bioelectrical impedance vector analysis. Applied Physiology Nutrition and Metabolism. 2019 Apr 29;44(6):619–26. doi:10.1139/apnm-2018-0276
18. Rinaldi S, Gilliland J, O'Connor C, Chesworth B, Madill J. Is phase angle an appropriate indicator of malnutrition in different disease states? A systematic review. Clinical Nutrition ESPEN. 2019 Feb 1; 29:1–14. doi: 10.1016/j.clnesp.2018.10.010
19. Mattiello R, Amaral MA, Mundstock E, Ziegelmann PK. Reference values for the phase angle of the electrical bioimpedance: Systematic review and meta-analysis involving more than 250,000 subjects. Clin Nutr. 2020 May;39(5):1411–7. doi: 10.1016/j.clnu.2019.07.004 PubMed PMID: 31400996.
20. Norman K, Stobäus N, Pirlich M, Bosy-Westphal A. Bioelectrical phase angle and impedance vector analysis--clinical relevance and applicability of impedance parameters. Clin Nutr. 2012 Dec;31(6):854–61. doi: 10.1016/j.clnu.2012.05.008 PubMed PMID: 22698802.
21. Lukaski HC, Raymond-Pope CJ. New Frontiers of Body Composition in Sport. Int J Sports Med. 2021 Jun;42(7):588–601. doi:10.1055/a-1373-5881 PubMed PMID: 33621995; PubMed Central PMCID: PMC8421000.
22. Marini E, Campa F, Buffa R, Stagi S, Matias CN, Toselli S, et al. Phase angle and bioelectrical impedance vector analysis in the evaluation of body composition in athletes. Clin Nutr. 2020 Feb;39(2):447–54. doi: 10.1016/j.clnu.2019.02.016 PubMed PMID: 30850270.
23. Baik JH. Stress and the dopaminergic reward system. Exp Mol Med. 2020 Dec 1;52(12):1879–90. doi:10.1038/s12276-020-00532-4 PubMed PMID: 33257725; PubMed Central PMCID: PMC8080624.
24. ResearchGate [Internet]. [cited 2026 Jul 9]. (PDF) Stress: Definition and History. Available from: https://www.researchgate.net/publication/285784528_Stress_Definition_and_history doi:10.1016/B978-008045046-9.00076-0
25. Lu S, Wei F, Li G. The evolution of the concept of stress and the framework of the stress system. Cell Stress. 5(6):76–85. doi:10.15698/cst2021.06.250 PubMed PMID: 34124582; PubMed Central PMCID: PMC8166217.
26. Ghasemi F, Beversdorf DQ, Herman KC. Stress and stress responses: A narrative literature review from physiological mechanisms to intervention approaches. Journal of Pacific Rim Psychology. 2024 May 1; 18:18344909241289222. doi:10.1177/18344909241289222
27. Kovács KJ, Miklós IH, Bali B. Chapter 6.1 - Psychological and physiological stressors. In: Steckler T, Kalin NH, Reul JMHM, editors. Handbook of Stress and the Brain [Internet]. Elsevier; 2005 [cited 2026 Jul 28]. p. 775–92. (Techniques in the Behavioral and Neural Sciences). Available from: https://www.sciencedirect.com/science/article/pii/S0921070905800410 doi:10.1016/S0921-0709(05)80041-0
28. Psychological Resilience: An Affect-Regulation Framework - PMC [Internet]. [cited 2026 Jun 22]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC12009612/

29. Ulrich-Lai YM, Herman JP. Neural regulation of endocrine and autonomic stress responses. Nat Rev Neurosci. 2009 Jun;10(6):397–409. doi:10.1038/nrn2647
30. Psych Central [Internet]. 2022 [cited 2026 Jul 9]. Acute Stress vs. Chronic Stress: Is There a Difference? Available from: https://psychcentral.com/stress/chronic-stress-vs-acute-stress
31. Russell G, Lightman S. The human stress response. Nat Rev Endocrinol. 2019 Sep;15(9):525–34. doi:10.1038/s41574-019-0228-0
32. Harvard Health [Internet]. 2011 [cited 2026 Jul 10]. Understanding the stress response. Available from: https://www.health.harvard.edu/healthy-aging-and-longevity/understanding-the-stress-response
33. (PDF) Physiology of Stress and its Management. ResearchGate. 2026 Jun 10. doi:10.24966/MSR-5657/100001
34. Physiology and Neurobiology of Stress and Adaptation: Central Role of the Brain | Physiological Reviews | American Physiological Society [Internet]. [cited 2026 Jul 10]. Available from: https://journals.physiology.org/doi/full/10.1152/physrev.00041.2006
35. Tindle J, Tadi P. Neuroanatomy, Parasympathetic Nervous System. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 [cited 2026 Jul 28]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK553141/ PubMed PMID: 31985934.
36. Khalil B, Rosani A, Warrington SJ. Physiology, Catecholamines. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 [cited 2026 Jul 28]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK507716/ PubMed PMID: 29939538.
37. James KA, Stromin JI, Steenkamp N, Combrinck MI. Understanding the relationships between physiological and psychosocial stress, cortisol and cognition. Front Endocrinol (Lausanne). 2023 Mar 6; 14:1085950. doi:10.3389/fendo.2023.1085950 PubMed PMID: 36950689; PubMed Central PMCID: PMC10025564.
38. Saad HA, Marzouk M, Abdelrahman H, Moradikor N. Mechanisms underlying stress effects on the brain: Basic concepts and clinical implications. Prog Brain Res. 2025; 291:21–47. doi: 10.1016/bs.pbr.2025.01.008 PubMed PMID: 40222781.
39. Turner AI, Smyth N, Hall SJ, Torres SJ, Hussein M, Jayasinghe SU, et al. Psychological stress reactivity and future health and disease outcomes: A systematic review of prospective evidence. Psychoneuroendocrinology. 2020 Apr; 114:104599. doi: 10.1016/j.psyneuen.2020.104599 PubMed PMID: 32045797.
40. Marik PE, Bellomo R. Stress hyperglycemia: an essential survival response! Crit Care. 2013;17(2):305. doi:10.1186/cc12514 PubMed PMID: 23470218; PubMed Central PMCID: PMC3672537.
41. Noushad S, Ahmed S, Ansari B, Mustafa UH, Saleem Y, Hazrat H. Physiological biomarkers of chronic stress: A systematic review. Int J Health Sci (Qassim). 2021;15(5):46–59. PubMed PMID: 34548863; PubMed Central PMCID: PMC8434839.
42. Kiselyov K, Muallem S. ROS and intracellular ion channels. Cell Calcium. 2016 Aug;60(2):108–14. doi: 10.1016/j.ceca.2016.03.004 PubMed PMID: 26995054; PubMed Central PMCID: PMC4996699.
43. Juan CA, Pérez de la Lastra JM, Plou FJ, Pérez-Lebeña E. The Chemistry of Reactive Oxygen Species (ROS) Revisited: Outlining Their Role in Biological Macromolecules (DNA, Lipids and Proteins) and Induced Pathologies. Int J Mol Sci. 2021 Apr 28;22(9):4642. doi:10.3390/ijms22094642 PubMed PMID: 33924958; PubMed Central PMCID: PMC8125527.
44. Harnessing the power of nutritional antioxidants against adrenal hormone imbalance-associated oxidative stress - PMC [Internet]. [cited 2026 Jul 28]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10720671/?utm_source=chatgpt.com
45. Rohleder N. Stress and inflammation - The need to address the gap in the transition between acute and chronic stress effects. Psychoneuroendocrinology. 2019 Jul; 105:164–71. doi: 10.1016/j.psyneuen.2019.02.021 PubMed PMID: 30826163.

46. Marsland AL, Walsh C, Lockwood K, John-Henderson NA. The effects of acute psychological stress on circulating and stimulated inflammatory markers: A systematic review and meta-analysis. Brain Behav Immun. 2017 Aug; 64:208–19. doi: 10.1016/j.bbi.2017.01.011 PubMed PMID: 28089638; PubMed Central PMCID: PMC5553449.
47. Huang L, Zhang Y, Wang J, Zhou J. Investigating the association of bioelectrical impedance analysis–derived phase angle and depressive symptoms in patients receiving maintenance hemodialysis: a cross-sectional study. Front Nutr. 2025 Sep 9;12. doi:10.3389/fnut.2025.1662484
48. Michopoulos V, Powers A, Gillespie CF, Ressler KJ, Jovanovic T. Inflammation in Fear- and Anxiety-Based Disorders: PTSD, GAD, and Beyond. Neuropsychopharmacology. 2017 Jan;42(1):254–70. doi:10.1038/npp.2016.146 PubMed PMID: 27510423; PubMed Central PMCID: PMC5143487.
49. Mental disorders [Internet]. [cited 2026 Jul 28]. Available from: https://www.who.int/news-room/fact-sheets/detail/mental-disorders
50. Anxiety disorders [Internet]. [cited 2026 Jul 21]. Available from: https://www.who.int/news-room/fact-sheets/detail/anxiety-disorders
51. Depressive disorder (depression) [Internet]. [cited 2026 Jul 21]. Available from: https://www.who.int/news-room/fact-sheets/detail/depression
52. Tian R, Hou G, Li D, Yuan TF. A Possible Change Process of Inflammatory Cytokines in the Prolonged Chronic Stress and Its Ultimate Implications for Health. The Scientific World Journal. 2014;2014(1):780616. doi:10.1155/2014/780616
53. Godoy LD, Rossignoli MT, Delfino-Pereira P, Garcia-Cairasco N, de Lima Umeoka EH. A Comprehensive Overview on Stress Neurobiology: Basic Concepts and Clinical Implications. Front Behav Neurosci. 2018 Jul 3; 12:127. doi:10.3389/fnbeh.2018.00127 PubMed PMID: 30034327; PubMed Central PMCID: PMC6043787.
54. Johnson JD, Barnard DF, Kulp AC, Mehta DM. Neuroendocrine Regulation of Brain Cytokines After Psychological Stress. J Endocr Soc. 2019 May 14;3(7):1302–20. doi:10.1210/js.2019-00053 PubMed PMID: 31259292; PubMed Central PMCID: PMC6595533.
55. Shimba A, Ikuta K. Control of immunity by glucocorticoids in health and disease. Semin Immunopathol. 2020 Dec;42(6):669–80. doi:10.1007/s00281-020-00827-8 PubMed PMID: 33219395.
56. Immune regulation by glucocorticoids - PMC [Internet]. [cited 2026 Jul 28]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC9761406/?utm_source=chatgpt.com
57. Bekhbat M, Rowson SA, Neigh GN. Checks and balances: The glucocorticoid receptor and NFκB in good times and bad. Front Neuroendocrinol. 2017 Jul; 46:15–31. doi: 10.1016/j.yfrne.2017.05.001 PubMed PMID: 28502781; PubMed Central PMCID: PMC5523465.
58. Slavich GM, Irwin MR. From stress to inflammation and major depressive disorder: a social signal transduction theory of depression. Psychol Bull. 2014 May;140(3):774–815. doi:10.1037/a0035302 PubMed PMID: 24417575; PubMed Central PMCID: PMC4006295.
59. Liguori I, Russo G, Curcio F, Bulli G, Aran L, Della-Morte D, et al. Oxidative stress, aging, and diseases. Clin Interv Aging. 2018; 13:757–72. doi:10.2147/CIA.S158513 PubMed PMID: 29731617; PubMed Central PMCID: PMC5927356.
60. Geddie H, Cairns M, Smith L, van Wyk M, Beselaar L, Truter N, et al. The impact of chronic stress on intracellular redox balance: A systems level analysis. Physiol Rep. 2023 Apr 5;11(7):e15640. doi:10.14814/phy2.15640 PubMed PMID: 37020327; PubMed Central PMCID: PMC10076515.
61. Catalá A, Díaz M. Editorial: Impact of Lipid Peroxidation on the Physiology and Pathophysiology of Cell Membranes. Front Physiol. 2016 Sep 22; 7:423. doi:10.3389/fphys.2016.00423 PubMed PMID: 27713704; PubMed Central PMCID: PMC5031777.
62. Bikulčienė I, Baleišis J, Mazgelytė E, Rudys R, Vosyliūtė R, Šimkūnaitė-Rizgelienė R, et al. Impact of chronic psychological stress on

platelet membrane fatty acid composition in a rat model of type 1 diabetes Mellitus. Lipids Health Dis. 2024 Mar 8; 23:69. doi:10.1186/s12944-024-02067-3 PubMed PMID: 38459494; PubMed Central PMCID: PMC10921692.

63. Niki E. Lipid peroxidation: physiological levels and dual biological effects. Free Radic Biol Med. 2009 Sep 1;47(5):469–84. doi: 10.1016/j.freeradbiomed.2009.05.032 PubMed PMID: 19500666.
64. De Domenico I, Ward DM, di Patti MCB, Jeong SY, David S, Musci G, et al. Ferroxidase activity is required for the stability of cell surface ferroportin in cells expressing GPI-ceruloplasmin. EMBO J. 2007 Jun 20;26(12):2823–31. doi: 10.1038/sj.emboj.7601735 PubMed PMID: 17541408; PubMed Central PMCID: PMC1894773.
65. Ayala A, Muñoz MF, Argüelles S. Lipid Peroxidation: Production, Metabolism, and Signaling Mechanisms of Malondialdehyde and 4-Hydroxy-2-Nonenal. Oxid Med Cell Longev. 2014; 2014:360438. doi:10.1155/2014/360438 PubMed PMID: 24999379; PubMed Central PMCID: PMC4066722.

How to cite this article: Kavita Gupta, Lalita Chaudhary, Megha Kulshreshtha, Alka Srivastava, Sanjana Chahar. Phase angle and psychological stress: a review of physiological mechanisms, cellular health and mental well-being. *International Journal of Research and Review*. 2026; 13(9): 166-180. DOI: *https://doi.org/10.52403/ijrr.20260919*

******