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The Communication-Endocrine-Stress Adaptive Regulation (CESAR) Model: A Biopsychosocial Framework for Understanding Health Outcomes in Sensory Impairment

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Abstract

Sensory impairment affects over 1.3 billion people worldwide, yet the field still lacks a mechanistically specified theoretical framework explaining how communication inaccessibility contributes to stress-related health disparities in these populations. Existing models remain fragmented, addressing biological, psychological, and social factors in isolation rather than as interconnected systems. This concept paper presents the Communication-Endocrine-Stress Adaptive Regulation (CESAR) Model, an integrative biopsychosocial framework that integrates communication access, social support, stress regulation, and neuroendocrine function into a unified causal pathway. The CESAR model proposes that sensory impairment creates communication barriers may reduce social support, increase perceived stress, dysregulate the hypothalamic–pituitary–adrenal (HPA) axis, and ultimately impact reproductive health and psychological well-being. This integrative framework synthesizes evidence from disability studies, stress physiology, and communication sciences to provide a comprehensive theoretical foundation for understanding adaptation in sensory-impaired populations. The model incorporates feedback loops, moderating factors (sex, age, impairment type, duration), and environmental contexts (accessibility policies, healthcare access) that influence adaptive outcomes. By proposing specific causal pathways and testable hypotheses, the CESAR model provides a roadmap for future empirical research and targeted interventions that address the root causes of health disparities in sensory-impaired populations rather than merely treating symptoms.

Keywords: sensory impairment; biopsychosocial model; communication barriers

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1. Introduction

1.1. The Challenge of Sensory Adaptation in a Biopsychosocial Context

Sensory impairment represents one of the most prevalent forms of disability worldwide, affecting over 1.3 billion people with hearing loss and 2.2 billion people with vision impairment or blindness [1]. Despite this enormous global burden, theoretical frameworks for understanding the complex health consequences of sensory impairment remain fragmented and inadequate. Current approaches typically address biological, psychological, and social factors in isolation, failing to capture the dynamic interplay between communication barriers, stress physiology, and health outcomes that characterizes the lived experience of sensory-impaired individuals.

The central problem is not whether sensory impairment is associated with poorer health outcomes, but how recurrent communication inaccessibility becomes transformed into chronic psychosocial stress, neuroendocrine dysregulation, and measurable psychological and reproductive health consequences.

The specific problem addressed in this paper is the absence of a mechanistically specified framework explaining how sensory impairment-related communication inaccessibility becomes biologically embedded as chronic stress and, in turn, contributes to adverse health outcomes. Although communication barriers, social isolation, psychological distress, and neuroendocrine dysregulation have each been documented in sensory-impaired populations, these processes have rarely been integrated into a single directional model with clearly defined mediating pathways. Consequently, the field still lacks a theory capable of explaining not only whether health disparities occur in hearing- and vision-impaired populations, but also through which psychosocial and psychoneuroendocrine mechanisms they emerge and persist.

Sensory impairment, whether auditory or visual, represents far more than a physiological deficit. It is a condition that fundamentally reconfigures an individual's interaction with their social and physical environment, demanding continuous and effortful adaptation [2]. Individuals who are Deaf, Hard-of-Hearing (DHH), or Visually Impaired (VI) navigate a world predominantly designed for sensory-typical perception, encountering daily challenges that extend beyond the primary sensory loss itself. These challenges are profoundly psychosocial, influencing social integration, educational attainment, and occupational opportunities. Consequently, these populations exhibit a significantly higher prevalence of stress-related conditions, including depression and anxiety, compared to their sensory-typical peers [3]. However, an important conceptual gap persists: the field lacks a model that explains, in a mechanistically coherent way, how sensory impairment-related barriers at the communicative and social level are translated into sustained physiological dysregulation and downstream health consequences. Existing biopsychosocial frameworks have been instrumental in shifting the paradigm away from a purely biomedical, deficit-oriented perspective. However, they often remain at a high level of abstraction, functioning more as philosophical orientations than as testable scientific models with clearly defined causal pathways [4]. This paper argues that to advance both understanding and intervention, a new, more mechanistic model is required.

The experience of living with a sensory impairment can be viewed as a unique natural experiment for investigating fundamental principles of human psychobiology. Communication is a strong, non-pathological model for seeing how a specific, long-lasting problem at the person-environment interface may affect the body's main physiological control systems, like the neuroendocrine stress response. By studying this population, it is possible to gain insights not only into the specific needs of individuals with sensory loss but also into the universal processes by which social experience becomes biologically embedded, or "gets under the skin," for all humans [5].

The need for an integrative theoretical framework is particularly urgent given mounting evidence of health disparities in sensory-impaired populations. Individuals with hearing loss or visual impairment demonstrate elevated rates of depression, anxiety, social isolation, and various physical health conditions compared to their sensory-intact counterparts [6,7]. Moreover, emerging research suggests that sensory impairment may be associated with reproductive health challenges, including menstrual irregularities, fertility issues, and sexual dysfunction, though the mechanisms underlying these associations remain poorly understood [8,9].

1.2. Defining Core Constructs

Precise operational definitions of its core constructs are crucial for developing a strong model.

Sensory Deprivation vs. Adaptation: It is crucial to distinguish between the initial loss of sensory input (*deprivation*) and the subsequent, lifelong process of adaptation. Adaptation leads to complex changes in the brain, helping it compensate for the loss of a sense and often improving the sensitivity of the remaining senses [2]. However, this adaptation is not always beneficial; the constant effort required to navigate a sensorially mismatched environment can lead to maladaptive outcomes, including chronic stress and cognitive fatigue [10].

Communication Access: This paper introduces communication access as a central construct, moving beyond the simple transmission and reception of information. It refers to the level of clarity, ease, and mental exertion needed to establish mutual understanding. This multidimensional concept encompasses environmental barriers (e.g., background noise, poor lighting), accessibility tools (e.g., interpreters, assistive technology), and the cognitive load imposed on the individual during an interaction [11]. It is the primary environmental interface directly and chronically disrupted by sensory impairment.

Psychoneuroendocrine Pathways: These are the biological systems that form the bridge between psychological experience and physiological function. The principal pathway of interest for this model is the Hypothalamic–Pituitary–Adrenal (HPA) axis. The HPA axis is the body's primary neuroendocrine stress response system, which, upon perception of a stressor, initiates a hormonal cascade culminating in the release of cortisol. This system is designed for acute responses, but chronic activation can lead to its dysregulation, with systemic health consequences [12].

1.3. Limitations of Current Theoretical Approaches

Existing theoretical frameworks for understanding sensory impairment and health outcomes suffer from several critical limitations. The medical model focuses primarily on sensory deficits and rehabilitation technologies, neglecting the broader psychosocial context that shapes adaptation outcomes [13]. Conversely, social models emphasize environmental barriers and discrimination while minimizing biological factors that may contribute to health disparities [14]. Biopsychosocial approaches to sensory impairment often view biological, psychological, and social aspects as distinct entities rather than interconnected components with dynamic interactions [15].

Most importantly, existing frameworks lack a thorough examination of communication's pivotal role as a mediator between sensory impairment and health results. Communication barriers represent a primary pathway through which sensory impairment impacts social relationships, stress levels, and ultimately, physiological functioning [16]. Yet no existing model systematically integrates communication access with stress physiology and neuroendocrine regulation to explain health disparities in sensory-impaired populations.

Taken together, these frameworks provide important but partial explanations. What remains insufficiently addressed is the absence of a model that specifies how sensory impairment-related communication inaccessibility functions as a chronic psychosocial stressor, how it erodes perceived social support, and how these processes become linked to measurable neuroendocrine dysregulation and downstream health outcomes. The research gap addressed by the CESAR model is therefore not the absence of biopsychosocial thinking per se, but the absence of a directional, mechanistically articulated, and empirically testable framework tailored to sensory impairment.

In summary, the specific gap addressed by the CESAR model is threefold. First, communication access has rarely been conceptualized as the primary person–environment

stress interface in sensory impairment. Second, perceived social support and perceived stress have seldom been positioned as sequential psychosocial mediators linking communication barriers with physiological adaptation. Third, existing frameworks have not explicitly connected these psychosocial processes with HPA-axis dysregulation and downstream psychological and reproductive health outcomes. CESAR addresses this gap by specifying a directional, mechanistically articulated, and empirically testable pathway from communication inaccessibility to stress-related health consequences.

1.4. The Need for an Integrative Framework

The complexity of adaptation in sensory impairment requires a theoretical framework that can account for multiple levels of analysis—from molecular mechanisms to social systems—while specifying the causal pathways through which sensory impairment influences health outcomes. Such a framework must integrate insights from disability studies, stress physiology, communication sciences, and neuroendocrinology to provide a comprehensive understanding of adaptive processes.

Accordingly, what is needed is not another general biopsychosocial description of disability, but a model that specifies the pathway through which communication inaccessibility operates as a chronic psychosocial stressor and becomes linked to measurable neuroendocrine and health outcomes. The CESAR model is proposed to address this specific theoretical gap by integrating communication access, perceived social support, stress appraisal, HPA-axis dysregulation, and downstream psychological and reproductive health consequences within one testable framework.

1.5. Thesis Statement and Aims

More specifically, this paper addresses the unresolved theoretical problem of how chronic communication barriers associated with sensory impairment are translated into cumulative psychosocial stress and biologically consequential dysregulation.

The central thesis of this paper is that existing frameworks relevant to sensory impairment each explain important parts of the problem, yet remain insufficient to account for the specific mechanistic pathway through which communication inaccessibility becomes translated into chronic stress-related physiological and health consequences. The proposed Communication-Endocrine-Stress Adaptive Regulation (CESAR) model addresses this limitation by positing a specific, integrated, and empirically testable causal pathway that links chronic communication barriers directly to HPA axis dysregulation and, subsequently, to adverse psychological and reproductive health outcomes.

The aims of this conceptual paper are threefold:

1. To critically review and synthesize disparate bodies of literature from communication sciences, stress psychology, and neuroendocrinology within the context of sensory impairment.
2. To formally present the CESAR model, detailing its constituent pathways, underlying causal hypotheses, moderators, and feedback dynamics.
3. To outline the model's significant implications for future research, clinical practice, and public health policy, thereby providing a roadmap for its empirical validation and practical application.

2. Materials and Methods

2.1. Study Design and Conceptual Approach

This study was designed as a concept paper aimed at theory development rather than empirical hypothesis testing. The purpose of the paper was not to report new primary data, but to construct an integrative biopsychosocial framework capable of explaining how

sensory impairment-related communication barriers may become translated into chronic psychosocial stress, neuroendocrine dysregulation, and downstream health consequences. The paper therefore follows a conceptual synthesis approach, combining insights from communication science, disability studies, stress psychology, and neuroendocrinology to develop a directional and empirically testable explanatory model.

2.2. Model Development Process

The CESAR model was developed through a structured conceptual process comprising four stages. First, the relevant interdisciplinary literature was reviewed to identify recurring domains associated with health disparities in populations with sensory impairment, with particular attention to communication barriers, social support, perceived stress, and stress physiology. Second, these domains were examined comparatively in light of existing theoretical frameworks, including biopsychosocial, stress-appraisal, and salutogenic models, in order to identify their explanatory strengths and unresolved limitations in the sensory impairment context. Third, based on this synthesis, a directional sequence of model components was conceptually derived, with communication inaccessibility positioned as the upstream person–environment stress interface, psychosocial mediators specified as intermediate mechanisms, and HPA-axis dysregulation identified as the principal biological embedding pathway. Fourth, the model was refined by incorporating feedback loops, contextual moderators, and potential downstream outcomes in order to increase its theoretical completeness and future empirical testability.

Model components were retained when they met three criteria: conceptual relevance to the sensory impairment context, theoretical plausibility within an ordered biopsychosocial stress pathway, and the existence of prior empirical or theoretical literature supporting their inclusion as central rather than peripheral processes. This approach was intended to balance interdisciplinary breadth with sufficient mechanistic specificity.

Within this framework, communication access was retained as the upstream component because it represents the most immediate and chronic person–environment interface disrupted by sensory impairment. Perceived social support and perceived stress were retained as central psychosocial mediators because they provide the most theoretically coherent link between communication barriers and longer-term adaptation outcomes. HPA-axis dysregulation was retained as the principal biological pathway because it offers a mechanistically specified account of how repeated communicative strain may become biologically embedded and translated into downstream psychological and reproductive health consequences. Together, these components were selected not as an exhaustive list of all relevant factors, but as the most conceptually central and sequentially integrable elements for a testable biopsychosocial model.

Scope and Boundaries

The CESAR model is intended primarily as a framework for understanding chronic adaptation processes in adolescents and adults with hearing impairment, visual impairment, or dual sensory impairment who are exposed to recurring barriers in communication access and social participation. Its central application is to contexts in which sensory impairment creates sustained person–environment mismatch rather than isolated or acute episodes of difficulty. The model is not intended to explain all possible health outcomes associated with sensory impairment, nor does it assume that the proposed pathways operate uniformly across all populations, developmental stages, or sociocultural contexts. Rather, it is proposed as a bounded biopsychosocial framework whose applicability is expected to vary according to impairment type, age of onset, communication environment, and contextual accessibility conditions.

2.3. Communication Pathways in Sensory Deprivation

Communication represents a fundamental human capacity that extends far beyond the simple transmission of information. In the context of sensory impairment, communication encompasses the complex processes of comprehension, expression, and mutual understanding that enable social connection and participation in community life [17]. When sensory function is compromised, these communication processes become significantly more challenging, creating barriers that can have profound consequences for psychological and physical health.

2.3.1. Communication Access as a Primary Stressor

The CESAR model posits that the cascade of adverse outcomes begins with a chronic, pervasive environmental stressor: diminished communication access. This stress is not an occasional event but a fundamental feature of daily life for many individuals with sensory impairments. The nature of this stressor differs qualitatively between DHH and VI populations, taxing different cognitive and emotional systems, yet both converge on a common outcome of increased psychosocial strain.

For DHH individuals, communication stress often arises from the immense cognitive load required to process a degraded auditory signal. Lip-reading, a common strategy, is notoriously unreliable, with only about 30% of spoken English being visually distinct, forcing the individual to engage in constant, effortful guesswork to fill in the gaps [11]. The use of written notes, often assumed to be an adequate substitute, fails to account for the fact that literacy development can be different for pre-lingually deaf individuals, making this mode of communication less effective than is often presumed. Systemic barriers, such as the chronic shortage of qualified sign language interpreters in critical settings like healthcare, further compound this stress, transforming routine interactions into high-stakes challenges. These are not mere inconveniences; they are daily micro-stressors that demand heightened vigilance and deplete finite cognitive resources. The stress is one of frustration and cognitive exhaustion, stemming from a constant struggle for signal clarity gaps [11].

For VI individuals, communication stress originates from a different source: the loss of the rich, parallel stream of nonverbal information that contextualizes verbal exchange. Facial expressions, eye gaze, gestures, and body posture provide a continuous flow of data about a speaker's intentions, emotions, and focus of attention [18,19]. Without this visual channel, social interactions become fraught with ambiguity. A VI person may not know if a question was directed at them, struggle with the rhythm and turn-taking of a group conversation, or misinterpret the emotional tone of a comment, leading to situations that are socially awkward or embarrassing [20]. This may create a stress rooted in social anxiety and uncertainty—a constant effort to infer the hidden social landscape that is effortlessly perceived by sighted individuals. While the origins differ—signal degradation for DHH populations versus loss of social context for VI populations—the outcome is a shared experience of "communication stress." This can be defined as a persistent state of heightened physiological and psychological arousal arising from a mismatch between the communicative demands of a situation and the individual's resources to meet those demands [18–23]. This chronic stressor is the critical initiating factor in the CESAR model.

More broadly, these barriers also extend to environmental navigation and access to visual information that supports social participation [24].

2.3.2. Impact on Social Connection and Belonging

Communication barriers are hypothesized to impact the quality and quantity of social relationships available to sensory-impaired individuals. Meta-analytic evidence indicates that individuals with sensory impairments experience higher rates of loneliness and social

isolation compared to their sensory-intact peers [25]. This social isolation is not simply a function of reduced social contact, but reflects the quality of communicative interactions and the sense of belonging that emerges from successful communication.

The relationship between communication access and social connection is bidirectional and dynamic. Poor communication access may reduce opportunities for meaningful social interaction, which in turn is proposed to limit the development of communication skills and confidence. This provides a plausible pathway for a self-perpetuating cycle in which communication barriers lead to social withdrawal, further reducing communication opportunities and exacerbating social isolation [21,23].

2.4. Stress and Neuroendocrine Regulation

The chronic nature of communication barriers in sensory impairment positions these challenges as potent activators of stress response systems. Understanding the physiological consequences of chronic communication stress requires examination of the primary stress response systems and their regulation under conditions of persistent environmental challenge [12,26,27].

2.4.1. The Hypothalamic–Pituitary–Adrenal (HPA) Axis

The HPA axis represents the primary neuroendocrine system responsible for coordinating the body's response to stressful challenges [28]. Under normal circumstances, HPA activation follows a predictable pattern: stressor perception triggers hypothalamic release of corticotropin-releasing hormone (CRH), which stimulates pituitary secretion of adrenocorticotropic hormone (ACTH), leading to adrenal cortisol release. Cortisol then provides negative feedback to terminate the stress response while mobilizing energy resources and modulating immune function.

However, chronic stress exposure can dysregulate this carefully orchestrated system, leading to patterns of HPA dysfunction that have been implicated in numerous health conditions [29]. Chronic stress may result in either hypercortisolism (elevated cortisol levels) or hypocortisolism (blunted cortisol responses), both of which are associated with adverse health outcomes including depression, immune dysfunction, metabolic disorders, and reproductive health problems.

2.4.2. Allostatic Load and Cumulative Stress Burden

The concept of allostatic load, introduced by McEwen and Stellar [26], provides a framework for understanding how chronic stress exposure leads to cumulative physiological dysregulation. Allostatic load represents the "wear and tear" on biological systems that results from repeated activation of stress response systems in the absence of adequate recovery periods.

In the context of sensory impairment, communication barriers may function as chronic stressors that contribute to allostatic load through several mechanisms. First, the cognitive effort required to navigate communication challenges may chronically activate stress response systems [30]. Second, the social isolation that often accompanies communication barriers represents a well-established risk factor for HPA dysregulation [27]. Third, the unpredictability and uncontrollability of many communication challenges may be particularly potent activators of stress response systems [31].

2.4.3. Evidence for HPA Dysfunction in Sensory Impairment

Emerging research provides preliminary evidence for HPA axis dysfunction in sensory-impaired populations, though this literature remains limited. Studies of individuals with hearing loss have documented altered cortisol patterns, including flattened diurnal cortisol rhythms and abnormal cortisol awakening responses [32]. These patterns are consistent

with chronic stress exposure and may partially explain the elevated rates of depression and other health problems observed in this population.

Research with individuals with visual impairment has similarly documented evidence of chronic stress, including elevated perceived stress levels, altered sleep patterns, and increased inflammatory markers [33]. While direct measures of HPA function are limited in this population, the constellation of findings suggests chronic stress system activation that may contribute to health disparities.

2.5. Integrative Psychosocial Mechanisms

The translation of sensory impairment into adverse health outcomes is proposed to occur through complex psychosocial mechanisms that mediate and moderate the relationships between communication barriers, stress response, and physiological function. Understanding these mechanisms requires examination of both mediating pathways (through which effects are transmitted) and moderating factors (that influence the strength of relationships).

2.5.1. Mediating Pathways: From Communication to Health

The CESAR model proposes a specific sequence of mediating relationships that connect sensory impairment to health outcomes (Table A1). This pathway begins with sensory impairment creating communication access barriers, which reduce perceived social support, increase perceived stress, dysregulate HPA function, and ultimately impact reproductive health and psychological well-being [34–36].

Social Support as a Key Mediator

Social support represents a critical mediator in the pathway from communication barriers to health outcomes. Extensive research demonstrates that social support functions as a buffer against the adverse effects of stress on physical and mental health [34]. The "stress-buffering" model proposes that social support protects health by reducing the perceived severity of stressors and by providing resources for coping with stressful challenges.

For individuals with sensory impairments, communication barriers directly impact both the availability and quality of social support. Difficulty communicating with family members, friends, and healthcare providers can reduce the instrumental support (practical assistance) and emotional support (empathy, caring) that are crucial for managing life challenges. Moreover, communication barriers may prevent individuals from effectively seeking or accepting support when it is offered [14,20,37].

Perceived Stress as a Proximal Mediator

Perceived stress, defined as the degree to which individuals appraise situations as unpredictable, uncontrollable, and overwhelming relative to their coping resources, represents a proximal mediator between social support and physiological outcomes [34]. The transactional model of stress and coping emphasizes that stress responses depend not on objective characteristics of stressors, but on individuals' cognitive appraisals of their ability to manage challenging situations.

Communication barriers may increase perceived stress through multiple pathways. First, communication challenges are often unpredictable and uncontrollable, characteristics that are particularly likely to trigger stress responses. Second, communication barriers may reduce individuals' confidence in their ability to manage social and practical challenges, increasing stress appraisals. Third, the social isolation that often accompanies communication barriers may reduce access to coping resources, further increasing perceived stress [11].

2.5.2. Moderating Factors

The strength of relationships within the CESAR model is influenced by numerous moderating factors that may amplify or attenuate the effects of sensory impairment on

health outcomes. Understanding these moderators is crucial for identifying individuals at highest risk and for developing targeted interventions.

Demographic and Individual Factors

Sex and gender represent important moderators of stress responses and health outcomes in sensory impairment. Research suggests that women may be more vulnerable to the adverse effects of chronic stress on reproductive health, while men may show greater susceptibility to cardiovascular and metabolic consequences [38]. Age at onset of sensory impairment also moderates outcomes, with congenital impairments associated with different adaptation patterns compared to acquired impairments [36].

The type and severity of sensory impairment influence the nature and extent of communication barriers experienced. Individuals with profound hearing loss may face different challenges compared to those with mild hearing loss, while the combination of hearing and vision impairment (dual sensory impairment) presents unique communication challenges that may have particularly severe consequences for health outcomes [36,38].

Cultural and Contextual Factors

Cultural factors significantly moderate the relationship between sensory impairment and health outcomes. Deaf culture, for example, provides a framework for positive identity formation and social connection that may buffer against the adverse effects of communication barriers in hearing-dominant environments [39]. Similarly, cultural attitudes toward disability and the availability of cultural accommodations influence adaptation outcomes.

Socioeconomic status represents another crucial moderator, as financial resources influence access to assistive technologies, healthcare services, and educational opportunities that can mitigate communication barriers. Environmental factors, including accessibility policies, workplace accommodations, and community support services, also moderate the relationship between sensory impairment and health outcomes [39].

Taken together, these pathways illustrate the central interdisciplinary logic of the CESAR model. Communication access is conceptualized as the primary person–environment interface at which sensory impairment becomes socially consequential. Perceived social support and perceived stress serve as the key psychosocial translation mechanisms through which communication inaccessibility is transformed into sustained stress appraisal. HPA-axis dysregulation, in turn, provides the biological embedding mechanism by which chronic communicative strain becomes linked to measurable downstream effects on psychological and reproductive health. In this way, the CESAR model does not merely place social, psychological, and biological factors side by side, but specifies how they are theoretically and temporally connected within a one-directional framework.

2.6. Limitations of Existing Frameworks in Sensory Impairment

The CESAR model is proposed not to replace foundational biopsychosocial theories but to build upon them by addressing specific limitations that become apparent when applied to the context of sensory impairment. A critical comparison reveals the conceptual gaps that CESAR is designed to fill.

George Engel's Biopsychosocial (BPS) Model (1977): Engel's model was a revolutionary and necessary paradigm shift, urging medicine to move beyond a narrow biomedical focus and consider the whole person in their social context [13]. Its enduring contribution is its holistic philosophy. However, its primary limitation is its lack of mechanistic specificity. The BPS model is a "blueprint" or a "framework for teaching" rather than a testable scientific model [40]. It posits that biological, psychological, and social domains interact but fails to specify how they interact, what the key mediating pathways are, or what the relative importance of each domain might be in a given condition [4]. For sensory impairment,

it allows one to state that social barriers matter, but it provides no formal mechanism to explain how a communication barrier leads to endocrine changes.

Lazarus & Folkman's Transactional Model of Stress and Coping (1984): This model made the crucial contribution of centering the stress process on an individual's cognitive appraisal. Stress is not inherent in an event but in the transaction between the person and the environment, as appraised by the individual (primary appraisal of threat/challenge; secondary appraisal of resources/coping options). This is highly relevant to sensory impairment, where a social situation may be appraised as highly threatening due to communication challenges. The model's limitation, however, is that it remains largely at the psychological level. It expertly describes the cognitive and emotional processes of coping but does not explicitly integrate the downstream biological consequences of these processes, particularly the specific neuroendocrine pathways that are activated by a persistent appraisal of threat and a failure of coping resources [41].

Aaron Antonovsky's Salutogenic Model and Sense of Coherence (SOC): In contrast to pathogenic models focused on risk factors, Antonovsky's salutogenic model asks what creates health [42]. Its core concept, Sense of Coherence (SOC), is a global orientation that life is comprehensible, manageable, and meaningful. A strong SOC allows individuals to mobilize resources to cope with stress effectively. This is a valuable framework for understanding resilience. Its limitation in the present context is that it describes a protective psychological state but does not detail the physiological mechanisms that are compromised when a person's sense of coherence is systematically eroded by a chronic stressor like communication inaccessibility. It explains who might be resilient but not what breaks down physiologically in those who are not [42,43].

The CESAR model seeks to bridge these gaps by providing the missing mechanistic link—the HPA axis—and by specifying a directional, causal pathway that originates in the social domain (communication access), is mediated by the psychological domain (social support, perceived stress), and results in concrete, measurable biological outcomes. A comparison of the discussed models, together with the proposed CESAR model, is presented in Table 1 (and additional frameworks in Table A2).

Table 1. A Comparative Analysis of Biopsychosocial Models Applied to Sensory Impairment.

Feature	Engel's Biopsychosocial Model	Lazarus & Folkman's Transactional Model	Antonovsky's Salutogenic Model (SOC)	The Proposed CESAR Model
Core Concept	Holistic, systems-based view of the patient and illness.	Stress as a transaction between person and environment, mediated by cognitive appraisal.	Focus on origins of health (salutogenesis) and resources for resilience (SOC).	A psychoneuroendocrine cascade linking communication to physiological dysregulation.
Primary Locus of Stress	Unspecified interaction between biological, psychological, and social domains.	Individual's subjective appraisal of a situation as threatening or exceeding coping resources.	A breakdown in one's sense of life as comprehensible, manageable, and meaningful.	Chronic friction at the person–environment interface, specifically in communication access.
Role of Biology	An acknowledged, co-equal domain alongside psychological and social factors.	Primarily an unspecified outcome of the psychological stress and coping process.	The "health ease/dis-ease continuum" as a general outcome of successful coping.	A core, specified mechanistic pathway (HPA axis) that mediates between psychosocial stress and health outcomes.

Table 1. *Cont.*

Feature	Engel's Biopsychosocial Model	Lazarus & Folkman's Transactional Model	Antonovsky's Salutogenic Model (SOC)	The Proposed CESAR Model
Primary Contribution	A crucial paradigm shift away from pure biomedical reductionism.	A process-oriented view of stress that emphasizes cognition and individual differences.	A positive psychology framework focusing on resources and resilience rather than deficits.	A testable, mechanistic integration of communication, social psychology, and neuroendocrinology.
Key Limitation in Sensory Impairment Context	Lacks testable causal pathways; is more of a philosophy than a predictive model.	Under-specifies the precise biological mechanisms that are affected by chronic appraisal of threat.	Describes a resilient state but does not detail the specific physiological breakdown when SOC is eroded.	Proposed as a testable framework; its predictive validity remains to be empirically confirmed.

Authors' compilation based on sources [4,41,43,44].

As shown in Table 1, prior frameworks each capture an important part of the problem, yet none specify the full communication-to-stress-to-neuroendocrine pathway that CESAR formalizes.

Accordingly, the CESAR model does not replace these foundational frameworks, but rather translates their broad insights into a directional pathway specific to sensory impairment. By formalizing communication access as the primary person–environment stress interface, the model specifies a sequence from communication barriers to social support, perceived stress, HPA-axis dysregulation, and downstream health outcomes. This is the specific theoretical gap the present model seeks to address.

Beyond conceptual distinction, the advantage of the CESAR model lies in its greater explanatory and translational specificity. Compared with broader biopsychosocial formulations, it identifies a concrete upstream mechanism—communication inaccessibility—as the recurring stress interface through which sensory impairment becomes socially and biologically consequential. Compared with appraisal-based and salutogenic frameworks, it offers a more explicit account of how psychosocial strain is converted into measurable neuroendocrine dysregulation and downstream health outcomes. This gives the model practical value not only for theory development, but also for the design of empirical studies, the selection of mediators and biomarkers, and the identification of intervention targets at the levels of communication access, social support, stress regulation, and contextual accessibility.

3. The CESAR Model: Theoretical Framework and Hypotheses

3.1. Model Overview

The Communication-Endocrine-Stress Adaptive Regulation (CESAR) Model provides an integrative framework for understanding how sensory impairment influences health outcomes through interconnected biological, psychological, and social pathways. The model proposes that sensory impairment initiates a cascade of effects that ultimately impact health through dysregulation of stress response systems and their downstream consequences for reproductive and psychological health (Figure 1). More specifically, the CESAR model integrates three levels of explanation that are often treated separately in the literature: the communicative level (access barriers and interactional mismatch), the psychosocial level (social support, appraisal, and perceived stress), and the biological level (HPA-axis activation and dysregulation). The model therefore specifies the sequential links among these levels rather than treating them as parallel correlates of sensory impairment.

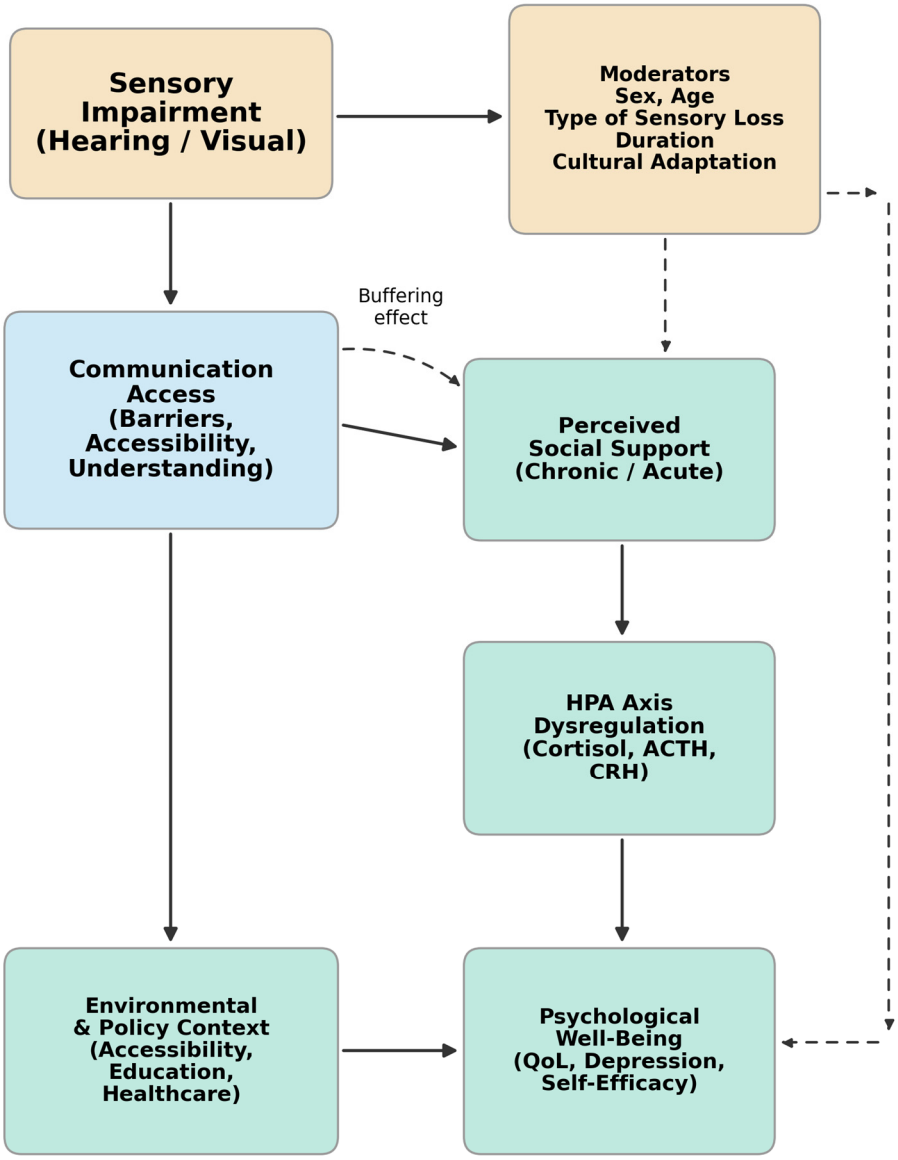

Figure 1. The CESAR Model: Communication-Endocrine-Stress Adaptive Regulation Framework for Sensory Impairment. Solid arrows represent hypothesised direct effects, while dashed arrows indicate feedback loops, buffering effects, and moderating relationships. Colour coding: yellow indicates Sensory Impairment and Moderators, blue indicates Communication Access, and green indicates the remaining model components.

As shown in Figure 1, the model is organized around a primary directional pathway linking sensory impairment to communication access barriers, reduced perceived social support, increased perceived stress, HPA-axis dysregulation, and downstream health outcomes. In the figure, psychological well-being and reproductive health are represented as the principal outcome domains, while factors such as sex, age, impairment type, duration of impairment, and contextual accessibility conditions are positioned as moderators of pathway strength. The figure also distinguishes direct hypothesized pathways from feedback dynamics: solid arrows represent the proposed primary causal sequence, whereas dashed arrows indicate feedback loops and context-sensitive modifying influences. This structure is intended to make explicit not only which constructs are included in the model, but also how they are ordered and related. Each of these components is elaborated in the subsequent sections addressing communication access, stress physiology, psychosocial mediators, moderators, and feedback mechanisms.

The CESAR framework conceptualizes sensory adaptation as a dynamic self-regulating system rather than a set of static correlations. Communication barriers act as primary perturbations that trigger cascading neuroendocrine and social feedback loops. Through these bidirectional interactions, the system continuously recalibrates stress responses and social engagement, maintaining adaptive balance under chronic communicative stress.

The CESAR model is grounded in several key theoretical principles:

1. **Systems Integration:** Biological, psychological, and social factors are conceptualized as interconnected systems rather than independent domains.
2. **Causal Specification:** The model proposes specific causal pathways with directional relationships that can be empirically tested.
3. **Dynamic Feedback:** The model incorporates feedback loops that capture the bidirectional and self-reinforcing nature of adaptation processes.
4. **Contextual Sensitivity:** The model acknowledges that relationships vary across individuals and contexts through moderating factors.
5. **Intervention Implications:** The model identifies multiple points of intervention that could disrupt maladaptive pathways and promote positive adaptation.

Importantly, "systems integration" in the CESAR model refers to more than the co-presence of multiple domains. It refers to a theoretically ordered sequence in which communication barriers function as chronic environmental demands, psychosocial processes shape their appraisal and buffering, and neuroendocrine mechanisms biologically encode repeated exposure to these demands. This ordering is essential to the model's interdisciplinary coherence and distinguishes CESAR from broader but less mechanistically specified biopsychosocial formulations.

Micro-mechanism Focus: From Communication Stress to Cortisol Release

To make the link between the psychosocial and biological domains more tangible, it is useful to trace the physiological cascade of a single, typical stressful event as predicted by the model.

1. **The Scenario (Environmental Stressor):** A visually impaired individual is in a dynamic group conversation at a social gathering. They are unable to use eye gaze or head turns to track who is speaking to whom, leading to confusion about when it is their turn to speak and whether comments are directed at them [20,22].
2. **Appraisal (Psychological Processing):** The situation is appraised as socially threatening, uncertain, and difficult to manage, consistent with transactional stress theory. This aligns with the primary appraisal process described by Lazarus and Folkman [41].
3. **Limbic System Activation (Neural):** This appraisal recruits neural threat-detection systems, particularly the amygdala, thereby increasing salience and vigilance.
4. **HPA Axis Initiation (Neuroendocrine):** Amygdala-driven signaling to the hypothalamus triggers corticotropin-releasing hormone (CRH) release into the hypophyseal portal system.
5. **Pituitary Response:** CRH stimulates the anterior pituitary to secrete adrenocorticotropic hormone (ACTH) into the systemic circulation.
6. **Adrenal Response:** ACTH stimulates the adrenal cortex to release cortisol [12].
7. **Physiological Consequences:** Cortisol mobilizes metabolic resources, heightens vigilance, and modulates immune and affective processes as part of the acute stress response.
8. **The Chronic Effect:** In isolation, this is a normal adaptive response. However, for an individual with a sensory impairment, this type of scenario may occur multiple times per day. The constant repetition of this micro-level cascade leads to the macro-level state of chronic HPA axis activation and eventual dysregulation hypothesized in the CESAR model.

While the previous scenario illustrates the psychoneuroendocrine cascade in the context of visual impairment, the CESAR model is intended to apply across different types of sensory loss. To further clarify the relationships among model components, the following examples illustrate how similar mechanisms may operate in other contexts.

A second example concerns a DHH individual navigating a critical medical consultation in a noisy clinical environment. The cognitive effort required to lip-read and interpret degraded auditory signals represents a communication access barrier that increases cognitive load, uncertainty, and perceived lack of control. When adequate interpreting support is unavailable and time constraints limit opportunities to ask for clarification, the individual may perceive reduced informational and instrumental support from healthcare providers. This situation may then be appraised as threatening or difficult to manage, increasing perceived stress and, when such high-stakes encounters are recurrent, contributing to repeated HPA-axis activation.

A third example concerns dual sensory impairment, where auditory and visual communication channels are simultaneously compromised. In this context, communication inaccessibility may reduce access not only to verbal information, but also to nonverbal cues, environmental signals, and spontaneous social interaction. The model would therefore predict a stronger indirect pathway from communication barriers to perceived stress through reduced social support and reduced coping resources, particularly in environments lacking specialized accessibility accommodations.

Together, these examples clarify that CESAR does not assume a simple direct effect of sensory impairment on health. Rather, it proposes that health consequences emerge through repeated, context-dependent interactions among communication access, perceived social support, stress appraisal, and neuroendocrine regulation.

3.2. Core Hypotheses

The CESAR model generates six primary hypotheses that specify the causal relationships between model components:

Hypothesis 1 (H1). *Sensory Impairment → Communication Access Barriers.*

Theoretical Rationale: Sensory impairment directly compromises the sensory channels (auditory, visual) that support effective communication, creating barriers to comprehension, expression, and mutual understanding across communicative contexts.

Operational Definition: Communication access barriers encompass difficulties in understanding spoken or written communication, expressing thoughts and needs effectively, participating in group conversations, accessing environmental information, and achieving mutual understanding with communication partners.

Empirical Support: Extensive research documents communication challenges across sensory impairment types, including reduced speech perception in noise for individuals with hearing loss [45], difficulty accessing visual communication cues for individuals with visual impairment [46], and compound communication barriers for individuals with dual sensory impairment [47].

Hypothesis 2 (H2). *Communication Access Barriers → Reduced Perceived Social Support.*

Theoretical Rationale: Communication barriers are hypothesized to impede the development and maintenance of social relationships by reducing the quality of social interactions, limiting opportunities for emotional expression and understanding, and creating challenges in accessing and providing social support.

Operational Definition: Perceived social support includes emotional support (empathy, caring, love), instrumental support (practical assistance, resources), informational support (advice, guidance), and appraisal support (feedback, affirmation) as perceived by the individual.

Empirical Support: Research consistently demonstrates associations between communication difficulties and reduced social support in sensory-impaired populations [48,49]. Qualitative studies reveal that communication barriers create challenges in developing intimate relationships, maintaining friendships, and accessing support during times of need.

Hypothesis 3 (H3). *Reduced Social Support → Increased Perceived Stress.*

Theoretical Rationale: Social support functions as a buffer against stress by providing resources for coping with challenges, reducing the perceived severity of stressors, and enhancing individuals' confidence in their ability to manage difficult situations. Reduced social support therefore increases vulnerability to stress.

Operational Definition: Perceived stress refers to the degree to which individuals appraise their life situations as unpredictable, uncontrollable, and overwhelming relative to their perceived coping resources.

Empirical Support: The stress-buffering effects of social support are among the most robust findings in health psychology [38]. Meta-analytic evidence demonstrates that social support reduces both subjective stress responses and physiological markers of stress activation across diverse populations and stressor types.

Hypothesis 4 (H4). *Increased Perceived Stress → HPA Axis Dysregulation.*

Theoretical Rationale: Chronic stress exposure may lead to dysregulation of the HPA axis through mechanisms including altered negative feedback sensitivity, disrupted circadian rhythms, and changes in receptor function. This dysregulation manifests as abnormal cortisol patterns and altered stress reactivity.

Operational Definition: HPA axis dysregulation includes alterations in diurnal cortisol rhythms (flattened or elevated patterns), abnormal cortisol awakening responses, altered cortisol reactivity to acute stressors, and disrupted negative feedback regulation.

Empirical Support: Extensive research demonstrates that chronic stress leads to HPA dysregulation across multiple populations and stressor types [50]. Longitudinal studies show that sustained stress exposure predicts the development of abnormal cortisol patterns over time.

Hypothesis 5 (H5). *HPA Axis Dysregulation → Reproductive Health Impairment.*

Theoretical Rationale: HPA axis hormones interact with the hypothalamic-pituitary-gonadal (HPG) axis through multiple mechanisms, including direct effects of cortisol on gonadotropin-releasing hormone (GnRH) secretion, modulation of gonadotropin sensitivity, and interactions at the level of gonadal steroid production.

Operational Definition: Reproductive health impairment includes menstrual cycle irregularities, ovulatory dysfunction, reduced fertility, sexual dysfunction, and alterations in reproductive hormone levels.

Empirical Support: Research demonstrates that chronic stress and HPA dysregulation are associated with reproductive health problems in women, including menstrual irregularities, anovulation, and reduced conception rates [51]. Similar effects on male reproductive function have been documented, including reduced testosterone levels and impaired spermatogenesis.

Hypothesis 6 (H6). *HPA Axis Dysregulation → Impaired Psychological Well-Being.*

Theoretical Rationale: HPA dysregulation influences psychological well-being through multiple pathways, including direct effects of cortisol on brain regions involved in mood regulation, indirect effects through sleep and immune function, and bidirectional relationships with depression and anxiety.

Operational Definition: Psychological well-being encompasses multiple domains including life satisfaction, positive and negative affect, depression and anxiety symptoms, self-efficacy beliefs, and overall quality of life.

Empirical Support: Meta-analytic evidence demonstrates robust associations between HPA dysregulation and psychological disorders, particularly depression and anxiety [52]. Longitudinal studies suggest bidirectional relationships, with HPA dysregulation both predicting and resulting from psychological distress.

A detailed description of the hypotheses is provided in Table A3.

3.3. Feedback Mechanisms and Dynamic Processes

The CESAR model incorporates several feedback mechanisms that capture the dynamic and self-reinforcing nature of adaptation processes in sensory impairment:

3.3.1. Stress–Communication Feedback Loop

Chronic stress and its physiological consequences can impair communication effectiveness through multiple pathways. Stress-related fatigue, cognitive dysfunction, and emotional dysregulation can reduce individuals' capacity for effortful communication tasks. Additionally, stress-related social withdrawal may reduce opportunities for communication practice and skill development, further exacerbating communication barriers.

3.3.2. Health–Stress Feedback Loop

Reproductive health problems and psychological distress can themselves become sources of stress, creating additional burden on already dysregulated stress response systems. This feedback mechanism may explain why health problems in sensory-impaired populations often become chronic and resistant to intervention.

3.3.3. Social Support Buffering Effects

The model incorporates the well-established finding that social support can buffer against the adverse effects of stress on health outcomes. This buffering effect operates at multiple levels, including cognitive (altering stress appraisals), behavioral (promoting healthy coping strategies), and physiological (directly modulating stress response systems).

4. Implications and Future Research Directions

The implications of the CESAR model follow directly from its ordered causal architecture. Because the model identifies communication inaccessibility as the upstream stress interface, psychosocial mediators as intermediate mechanisms, and HPA-axis dysregulation as the principal biological embedding pathway, it implies that research, clinical assessment, and policy should be organized around these sequential levels rather than around isolated outcomes alone. In practical terms, this means that communication barriers should be treated not merely as functional inconveniences, but as potential initiating determinants of chronic stress-related health risk.

Because the CESAR model is organized as a sequential pathway, its implications should be interpreted in relation to specific model components. Communication access implies the need for environmental and interactional interventions; perceived social support indicates the importance of interpersonal and community-based resources; perceived stress

identifies the need for psychological assessment and stress-regulation strategies; HPA-axis dysregulation supports the inclusion of biomarkers in future empirical studies; and reproductive and psychological outcomes define the distal health domains in which the model's explanatory and clinical relevance should be tested.

4.1. Empirical Testing of the CESAR Model

The CESAR model generates specific, testable hypotheses that can be evaluated using multiple research methodologies. The comprehensive empirical testing of this model will require a coordinated research program employing diverse approaches:

4.1.1. Structural Equation Modeling Approaches

The causal pathways specified in the CESAR model are ideally suited for testing using structural equation modeling (SEM) techniques. SEM allows for the simultaneous examination of multiple relationships while accounting for measurement error and testing competing model specifications. Key considerations for SEM testing include:

Cross-Sectional Studies: Initial model testing can employ cross-sectional designs to examine the pattern of relationships among model variables. While cross-sectional data cannot establish causality, they can provide evidence for the plausibility of proposed pathways and identify relationships that warrant longitudinal investigation.

Longitudinal Studies: Definitive testing of causal relationships requires longitudinal data that can establish temporal precedence. Multi-wave longitudinal studies should measure all model variables at multiple time points to examine both cross-lagged effects and within-person changes over time.

Multi-Group Analyses: The CESAR model's emphasis on moderating factors suggests the need for multi-group SEM analyses that test whether model relationships vary across different populations (e.g., by type of sensory impairment, sex, age of onset).

4.1.2. Intervention Studies

The CESAR model's specification of causal pathways provides a roadmap for intervention development and testing. Interventions that target proximal causes in the causal chain should produce downstream effects on more distal outcomes:

Communication Access Interventions: Interventions that improve communication access (e.g., assistive technology training, communication partner training, environmental modifications) should reduce perceived stress and improve health outcomes if the CESAR model is correct.

Social Support Interventions: Programs that enhance social support (e.g., peer mentoring, support groups, family intervention) should buffer against stress and improve physiological and psychological outcomes.

Stress Management Interventions: Direct stress management interventions (e.g., mindfulness training, cognitive-behavioral therapy) should improve HPA function and downstream health outcomes.

4.1.3. Biomarker Integration

Comprehensive testing of the CESAR model requires integration of biological markers that can objectively assess HPA function and other physiological processes:

Cortisol Assessment: Multiple cortisol measures should be employed, including diurnal cortisol profiles, cortisol awakening responses, and cortisol reactivity to standardized stressors. Salivary cortisol collection provides a non-invasive method for repeated sampling.

Inflammatory Markers: Given the role of chronic stress in promoting inflammation, studies should include markers of systemic inflammation such as C-reactive protein, interleukin-6, and tumor necrosis factor-alpha.

Reproductive Hormones: Testing reproductive health pathways requires assessment of relevant hormones including estradiol, progesterone, testosterone, luteinizing hormone, and follicle-stimulating hormone.

Full comparison of biomarkers is presented in Table A4.

4.2. Clinical and Policy Implications

Accordingly, CESAR implies that effective intervention may require targeting different points of the same pathway: reducing communication strain at the environmental level, strengthening social support at the interpersonal level, attenuating maladaptive stress appraisal at the psychological level, and monitoring stress-related physiological burden at the biological level. This multilevel implication is specific to the CESAR model's sequential design and distinguishes it from broader recommendations that focus on distal symptoms alone.

The CESAR model has significant implications for clinical practice and public policy related to sensory impairment:

4.2.1. Healthcare Integration

The model suggests that healthcare for sensory-impaired individuals should adopt a more integrated approach that addresses communication, social, and stress-related factors rather than focusing solely on sensory rehabilitation (Table A5):

Screening and Assessment: Healthcare providers should routinely screen for communication barriers, social isolation, and stress-related symptoms in sensory-impaired patients. Validated instruments for assessing these domains should be incorporated into standard care protocols.

Multidisciplinary Care: The complex, interconnected nature of factors in the CESAR model suggests the need for multidisciplinary care teams that include audiologists, ophthalmologists, mental health professionals, and social workers.

Prevention Focus: The model's emphasis on cascading effects suggests that early intervention to address communication barriers and social support may prevent the development of more serious health problems.

4.2.2. Policy and Environmental Interventions

The CESAR model highlights the importance of environmental and policy factors in determining health outcomes for sensory-impaired individuals:

Accessibility Legislation: Policies that mandate communication accessibility (e.g., sign language interpreters, captioning, assistive listening devices) may have far-reaching health benefits by addressing root causes of stress and social isolation.

Healthcare Access: Ensuring that healthcare services are fully accessible to sensory-impaired individuals may improve both direct health outcomes and reduce stress related to healthcare navigation.

Educational and Employment Policies: Policies that support inclusive education and employment may improve long-term health outcomes by enhancing communication skills, social connections, and socioeconomic status.

From a public health perspective, the CESAR model provides a strong rationale for policies that mandate and fund robust accessibility infrastructure. Policies ensuring the availability of qualified interpreters in healthcare and educational settings, the implementation of universal design principles in public spaces, and the funding of assistive

technologies should be framed not just in the language of civil rights, but also as evidence-based public health initiatives [49]. By reducing the chronic, systemic communication stress experienced by a significant portion of the population, such policies can lower the collective allostatic load, potentially leading to long-term reductions in healthcare costs associated with stress-related chronic diseases.

4.3. Future Research Priorities

Several research priorities emerge from the CESAR model that could significantly advance understanding of health outcomes in sensory impairment:

4.3.1. Mechanistic Research

Neurobiological Pathways: Research is needed to elucidate the specific neurobiological mechanisms through which communication stress influences HPA function. This includes examination of brain regions involved in stress and communication processing, neurotransmitter systems, and epigenetic factors.

Developmental Trajectories: Longitudinal research examining how CESAR model relationships develop and change across the lifespan could inform targeted interventions for different age groups.

Gene-Environment Interactions: Investigation of genetic factors that moderate susceptibility to stress-related health problems in sensory impairment could identify high-risk individuals and personalized intervention approaches.

4.3.2. Intervention Development

Technology-Enhanced Interventions: Development and testing of technology-based interventions that address multiple CESAR model components simultaneously (e.g., apps that provide communication support, social connection, and stress management).

Family and Community Interventions: Research on interventions that target the broader social environment rather than just the individual with sensory impairment.

Cultural Adaptations: Development of culturally adapted interventions that account for diverse community values and practices related to disability and health.

Emerging AI-assisted tools, including real-time captioning, speech-to-text systems, visual scene interpretation, and communication support technologies, may further reduce communication strain and thereby attenuate downstream psychosocial stress and its mental health consequences.

4.3.3. Population-Specific Research

While developed within the context of sensory impairment, the core principles of the CESAR model—that impaired communication access is a potent chronic stressor that drives physiological dysregulation—are likely applicable to other populations.

Dual Sensory Impairment: Individuals with both hearing and vision impairment may face particularly severe communication barriers and health risks that warrant specialized investigation [20].

Aphasia: Individuals who acquire aphasia, often following a stroke, experience a sudden and dramatic loss of communication ability. This leads to profound social isolation, frustration, and high rates of post-stroke depression [53]. The CESAR model provides a ready-made framework for investigating the psychoneuroendocrine consequences in this population, exploring how the acute disruption of communication impacts the HPA axis and contributes to the complex health challenges faced by stroke survivors.

Autism Spectrum Disorder (ASD): A core diagnostic feature of ASD is persistent challenges in social communication and interaction [54]. While the origin of the communication difficulty is neurodevelopmental rather than sensory, the psychosocial consequences—

social isolation, bullying, and chronic stress—are strikingly similar [55]. The CESAR model could be adapted to explore how these innate differences in communication style and social processing act as a chronic stressor that contributes to the high rates of anxiety, depression, and other stress-related health problems observed in the autistic population.

Pediatric Populations: Research on CESAR model relationships in children and adolescents could inform early intervention approaches and prevent the development of chronic health problems.

Aging Populations: Given the high prevalence of sensory impairment in older adults, research on age-related factors that influence CESAR model relationships is crucial for promoting healthy aging.

5. Conclusions

The Communication-Endocrine-Stress Adaptive Regulation (CESAR) Model is proposed as a potentially important advance in the theoretical understanding of health outcomes in sensory impairment. The CESAR model reframes communication inaccessibility as a mechanistic driver of stress-related health disparities rather than as a merely contextual or functional limitation. By positioning communication access as the upstream person–environment stress interface, the model specifies how sensory impairment may become socially consequential, psychologically appraised, biologically embedded through HPA-axis dysregulation, and ultimately linked to psychological and reproductive health outcomes. More specifically, the model is intended to address the central theoretical problem identified in the Introduction: the absence of a mechanistically specified framework explaining how sensory impairment-related communication inaccessibility becomes biologically embedded as chronic stress and translated into downstream health consequences. By integrating insights from disability studies, stress physiology, communication sciences, and neuroendocrinology, the CESAR model provides a comprehensive framework for understanding the complex pathways through which sensory impairment influences health and well-being.

The model's key contributions include: (1) specification of causal pathways linking sensory impairment to health outcomes through communication barriers, social support, and stress physiology; (2) integration of biological and psychosocial factors within a unified theoretical framework; (3) incorporation of feedback mechanisms and moderating factors that capture the dynamic nature of adaptation processes; and (4) generation of specific, testable hypotheses that can guide empirical research and intervention development. This explicitly ordered explanatory pathway also gives the model a practical advantage over broader theoretical frameworks by identifying more specific hypotheses, mediators, biomarkers, and intervention points.

The CESAR model has significant implications for research, clinical practice, and public policy. For researchers, the model provides a roadmap for systematic investigation of health disparities in sensory-impaired populations and offers specific hypotheses for empirical testing. For clinicians, the model suggests the need for more integrated, multidisciplinary approaches to care that address communication, social, and stress-related factors. For policymakers, the model highlights the importance of accessibility policies and environmental modifications in promoting health outcomes for sensory-impaired individuals.

A central tenet of this work is the reframing of sensory impairment as more than a condition to be managed; it is a powerful natural model for investigating the fundamental mechanisms of mind–body interaction. The daily challenges of communication and social navigation faced by individuals with hearing or visual loss are not unique pathologies but rather an amplification of universal human processes. The CESAR model uses this amplified context to illuminate the profound and continuous influence of our social and

communicative environment on the core neuroendocrine systems that regulate our health. The insights gained from studying this population have the potential to advance the broader fields of social neuroscience, psychosomatic medicine, and public health. Recent studies documenting mental health burden and communication-related difficulties in hearing- and vision-impaired populations further support the relevance of a framework linking communication inaccessibility with downstream biopsychosocial outcomes [3,6,7,11,12,20].

Perhaps most importantly, the CESAR model shifts focus from treating the symptoms of health disparities to addressing their root causes. By identifying communication barriers as a primary driver of stress and health problems, the model suggests that interventions targeting communication access may have far-reaching benefits for multiple health outcomes. This prevention-focused approach has the potential to improve quality of life for millions of sensory-impaired individuals while reducing healthcare costs and societal burden. At this stage, the significance of the CESAR model lies primarily in its capacity to organize a previously fragmented field into a coherent, testable framework; its broader scientific and practical value will need to be established through future empirical validation.

The presentation of the CESAR model is not an endpoint but a call to action. The next critical phase of this research program must be empirical, longitudinal, and deeply interdisciplinary, as outlined in this paper. Validating the model's hypothesized pathways will require collaboration between psychologists, communication scientists, endocrinologists, and public health researchers. The planned subsequent empirical studies will represent the initial steps in this validation process. Ultimately, the goal is to translate the insights from this model into effective, evidence-based interventions and policies. By targeting the root cause of the stress cascade—communication barriers—it may be possible to develop strategies that not only enhance quality of life but also promote physiological resilience and improve long-term health for the millions of individuals worldwide living with sensory impairments and other communication challenges.

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Abbreviations

The following abbreviations are used in this manuscript:

ACTH	Adrenocorticotropic Hormone
ASD	Autism Spectrum Disorder
BPS	Biopsychosocial
CASDA	Communication Assessment Scale for Deaf Adults
CESAR	Communication-Endocrine-Stress Adaptive Regulation
CIQ	Community Integration Questionnaire
CRH	Corticotropin-Releasing Hormone
DHH	Deaf, Hard-of-Hearing
DIDS	Deaf Identity Development Scale

FSH	Follicle-stimulating hormone
GnRH	Gonadotropin-releasing hormone
GSE	General Self-Efficacy Scale
HPA	Hypothalamic–Pituitary–Adrenal
HPG	Hypothalamic-pituitary-gonadal
ICF	International Classification of Functioning, Disability and Health
IL-6	Interleukin-6
ISEL	Interpersonal Support Evaluation List
LH	Luteinizing hormone
PSS-10	Perceived Stress Scale
QoL	Quality of Life
SEM	Structural equation modeling
SNI	Social Network Index
SOC	Sense of Coherence
TNF-α	Tumor necrosis factor-alpha
VI	Visually Impaired

Appendix A. Supplementary Tables for CESAR Model Concept Paper

Table A1. Proposed Mediators and Moderators for Future CESAR Model Research.

Mediating Variables			
Domain	**Variable**	**Measurement Approach**	**Hypothesized Role**
Communication	Communication effectiveness	Communication Assessment Scale for Deaf Adults (CASDA)	Mediates sensory impairment → social support
	Communication confidence	Self-efficacy questionnaires	Moderates communication barriers → stress
	Technology use	Assistive technology usage surveys	Moderates sensory impairment → communication
Social	Social network size	Social Network Index (SNI)	Mediates communication → social support
	Relationship quality	Interpersonal Support Evaluation List (ISEL)	Mediates social network → perceived support
	Social participation	Community Integration Questionnaire (CIQ)	Mediates social support → stress
Psychological	Perceived stress	Perceived Stress Scale (PSS-10)	Mediates social support → HPA function
	Coping strategies	Brief COPE Inventory	Moderates stress → HPA function
	Self-efficacy	General Self-Efficacy Scale (GSE)	Moderates stress → health outcomes
Moderating Variables			
Domain	**Variable**	**Measurement Approach**	**Hypothesized Moderation**
Demographic	Sex/Gender	Self-report + biological sex	All pathway relationships
	Age	Chronological age	Stress → HPA, HPA → reproductive health
	Socioeconomic status	Composite SES index	Communication → support, stress → health

Table A1. *Cont.*

Moderating Variables			
Domain	**Variable**	**Measurement Approach**	**Hypothesized Moderation**
Impairment	Type of sensory loss	Audiometry, visual acuity testing	Sensory impairment → communication
	Severity of loss	Degree of hearing/vision loss	All primary pathway relationships
	Age of onset	Congenital vs. acquired	Communication → support → stress
	Duration of impairment	Years since diagnosis/onset	Stress → HPA dysregulation
Cultural	Deaf culture identification	Deaf Identity Development Scale (DIDS)	Communication → support (DHH only)
	Cultural attitudes	Disability attitudes measures	Environmental context effects
	Language preference	Primary communication modality	Communication effectiveness

Table A2. Comparison of Existing Biopsychosocial Models in Sensory Impairment Research.

Model/Framework	Primary Focus	Key Components	Strengths	Limitations
WHO ICF Model	Disability classification	Body functions, activities, participation, environmental factors	Comprehensive, internationally recognized	Static, lacks causal pathways
Medical Model	Sensory deficits	Hearing/vision loss, rehabilitation, assistive technology	Clear intervention targets	Ignores psychosocial factors
Social Model	Environmental barriers	Discrimination, accessibility, social attitudes	Emphasizes social justice	Minimizes biological factors
Stress and Coping Model (Lazarus)	Cognitive appraisal	Primary/secondary appraisal, coping strategies	Well-validated, process-oriented	Limited biological integration
Biopsychosocial Model (Engel)	Multiple domains	Biological, psychological, social factors	Holistic approach	Lacks specific causal pathways
CESAR Model	**Integrated pathways**	**Communication → stress → neuroendocrine → health**	**Causal specification, feedback loops, testable**	**Requires empirical validation**

Table A3. CESAR Model Hypotheses and Corresponding Statistical Tests.

Hypothesis	Statistical Approach	Key Variables	Expected Effect Size
H1: Sensory Impairment → Communication Barriers	Linear regression, SEM	Audiometry/visual acuity → communication scales	$\beta = 0.40–0.60$
H2: Communication Barriers → Reduced Social Support	Mediation analysis, SEM	Communication effectiveness → social support measures	$\beta = -0.30–0.50$

Table A3. *Cont.*

Hypothesis	Statistical Approach	Key Variables	Expected Effect Size
H3: Reduced Social Support → Increased Stress	SEM, multilevel modeling	Social support → perceived stress scales	β = −0.40–0.60
H4: Increased Stress → HPA Dysregulation	Linear mixed models	Perceived stress → cortisol patterns	β = 0.25–0.40
H5: HPA Dysregulation → Reproductive Health	SEM, mediation analysis	Cortisol → reproductive hormones/outcomes	β = 0.20–0.35
H6: HPA Dysregulation → Psychological Well-being	SEM, multilevel modeling	Cortisol → depression, QoL measures	β = 0.30–0.45
Moderator Analyses			
Moderator	Analysis Approach	Hypothesized Pattern	Statistical Test
Sex/Gender	Multi-group SEM	Stronger reproductive effects in women	$\Delta\chi^2$ test, parameter comparisons
Type of SI	Multi-group SEM	Different pathway strengths	Measurement invariance testing
Age of Onset	Continuous moderator	Stronger effects for acquired SI	Interaction terms in SEM
Cultural Identity	Multi-group SEM	Buffering effects in strong cultural identity	Moderated mediation analysis
Moderator	Analysis Approach	Hypothesized Pattern	Statistical Test
Sex/Gender	Multi-group SEM	Stronger reproductive effects in women	$\Delta\chi^2$ test, parameter comparisons
Type of SI	Multi-group SEM	Different pathway strengths	Measurement invariance testing
Age of Onset	Continuous moderator	Stronger effects for acquired SI	Interaction terms in SEM
Cultural Identity	Multi-group SEM	Buffering effects in strong cultural identity	Moderated mediation analysis

Table A4. Recommended Biomarkers for CESAR Model Testing.

Primary Neuroendocrine Markers			
Biomarker	**Collection Method**	**Measurement Protocol**	**Clinical Significance**
Cortisol	Saliva	Diurnal profile (4–6 samples/day)	HPA axis function
		Cortisol awakening response (0, 15, 30, 45 min)	Stress reactivity
		Dexamethasone suppression test	Negative feedback sensitivity
ACTH	Plasma	Morning fasting sample	Pituitary function
CRH	Plasma	Research protocol only	Hypothalamic function

Table A4. *Cont.*

Secondary Stress Markers			
Biomarker	**Collection Method**	**Measurement Protocol**	**Clinical Significance**
Alpha-amylase	Saliva	Same as cortisol sampling	Sympathetic nervous system
Inflammatory markers	Serum/Plasma	C-reactive protein, IL-6, TNF-α	Chronic inflammation
Oxidative stress	Plasma/Urine	8-isoprostane, MDA	Cellular damage
Reproductive Health Markers			
Estradiol	Serum	Cycle-specific timing (women)	Ovarian function
Progesterone	Serum	Luteal phase (women)	Ovulation confirmation
Testosterone	Serum	Morning fasting (men)	Gonadal function
LH/FSH	Serum	Baseline and stimulation tests	Gonadotropin function
SHBG	Serum	Fasting sample	Hormone bioavailability

Table A5. Clinical and Policy Implications of CESAR Model Components.

Healthcare System Implications			
CESAR Component	**Clinical Implication**	**Implementation Strategy**	**Outcome Measures**
Communication Barriers	Routine communication assessment	Standardized screening tools	Communication effectiveness scores
Social Support	Social support interventions	Peer mentoring programs	Social network size, quality
Stress Management	Integrated stress care	Multidisciplinary teams	Perceived stress, cortisol levels
HPA Monitoring	Biomarker screening	Point-of-care cortisol testing	Diurnal cortisol patterns
Reproductive Health	Specialized reproductive care	SI-informed fertility counseling	Reproductive hormone profiles
Policy and Environmental Implications			
CESAR Component	**Policy Implication**	**Legislative/Regulatory Action**	**Expected Population Impact**
Communication Access	Universal communication access	ADA enhancement, captioning mandates	Reduced communication barriers
Social Integration	Inclusive community programs	Funding for accessibility programs	Increased social participation
Healthcare Access	SI-competent healthcare	Provider training requirements	Improved health outcomes
Workplace Support	Employment accommodations	Enhanced workplace accessibility	Reduced occupational stress
Workplace Support	Employment accommodations	Enhanced workplace accessibility	Reduced occupational stress
Educational Support	Inclusive education policies	Early intervention mandates	Better developmental outcomes

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