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Mathematical modeling of the interaction between endocrine systems and EEG signals

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Introduction: The intricate interplay between endocrine systems and EEG signals is pivotal for understanding and managing physiological and neurological health. Traditional mathematical models often fail to capture the nonlinear dynamics, feedback mechanisms, and cross-system interactions inherent in these processes, limiting their applicability in clinical and research settings.

Methods: This study proposes a novel framework for modeling and analyzing the interaction between endocrine regulatory systems and EEG signals, leveraging advanced methodologies such as the Hormone Interaction Dynamics Network (HIDN) and the Adaptive Hormonal Regulation Strategy (AHRS). HIDN integrates graph-based neural architectures with recurrent dynamics to encapsulate the spatiotemporal interdependencies among endocrine glands, hormones, and EEG signal fluctuations. AHRS complements this by dynamically optimizing therapeutic interventions using real-time feedback and patient-specific parameters, ensuring adaptability to individual variability and external perturbations.

Results: The proposed model excels in scalability, precision, and robustness, addressing challenges like sparse clinical data, temporal resolution, and multi-hormonal regulation. Experimental validation demonstrates its efficacy in predicting hormone dynamics, EEG signal patterns, and therapeutic outcomes under varying conditions.

Discussion: This interdisciplinary approach bridges the gap between computational modeling and practical healthcare applications, advancing our understanding of endocrine-neurological interactions.

KEYWORDS

endocrine systems, EEG signals, nonlinear dynamics, adaptive regulation, hormonal modeling

1 Introduction

The intricate interplay between the endocrine system and brain activity, as reflected in EEG signals Tao et al. (1), is a critical area of research for understanding physiological and pathological processes. This interaction not only provides insights into the neuroendocrine regulation of cognition, mood, and behavior but also enables the development of diagnostic

and therapeutic tools for conditions such as hormonal imbalances, neurological disorders, and psychiatric diseases Cai et al. (2). Traditional approaches to analyzing these interactions have been limited in their ability to account for the nonlinear and dynamic nature of neuroendocrine mechanisms. Consequently, mathematical modeling has emerged as a promising framework to integrate diverse physiological processes, allowing researchers to decode complex feedback loops and predict system behaviors with greater precision. This study area, therefore, holds immense potential for advancing biomedical research and personalized medicine Li et al. (3).

A clearer understanding of the interaction between the endocrine system and brain activity is essential for advancing both theoretical knowledge and clinical practice. The endocrine system regulates a wide range of physiological functions—including metabolism, stress response, and circadian rhythms—through hormone release, which in turn influences brain function via modulation of neural excitability and signal transmission Kamble and Sengupta (4). Conversely, the brain exerts regulatory control over hormonal activity through neuroendocrine pathways, such as the hypothalamic-pituitary axes. Disruptions in this bidirectional communication are implicated in a variety of neurological and psychiatric disorders. Therefore, modeling these complex interdependencies holds the potential to improve early diagnosis, inform personalized treatment strategies, and enhance the efficacy of therapeutic interventions Pepino et al. (5). By bridging the gap between computational modeling and biological interpretation, our work aims to shed light on these dynamics and contribute to a more integrated understanding of human health.

Early approaches to mathematical modeling in this domain relied heavily on symbolic AI and knowledgebased systems Shen et al. (6). These models used differential equations and heuristic rules to represent the physiological processes governing endocrine systems and EEG signals. Techniques such as compartmental modeling were employed to capture hormone secretion, transport, and receptor interactions. Simultaneously, linear models were used to analyze EEG signal dynamics. Despite providing a structured framework and theoretical insights Song et al. (7), these methods suffered from oversimplification and limited accuracy in handling the inherent variability of biological system. Their inability to incorporate stochastic effects and adapt to real-time data further constrained their applicability, prompting the need for more robust methodologies Wang et al. (8).

To address the limitations of symbolic approaches, researchers turned to data-driven machine learning methods. These models utilized statistical techniques and algorithms Chudasama et al. (9), including regression analysis, support vector machines (SVM), and Bayesian networks, to identify correlations between endocrine parameters and EEG features Zhang et al. (10). Machine learning enabled the extraction of patterns from large datasets, improving prediction accuracy and adaptability to diverse conditions. However, these methods relied heavily on feature engineering and were limited by their dependence on labeled data Issa et al. (11). Additionally, machine learning models often failed to capture the temporal dependencies and feedback loops characteristic of

neuroendocrine interactions, leaving room for improvement in representing the dynamic nature of these systems.

The advent of deep learning has significantly enhanced the modeling of endocrine-EEG interactions by leveraging advanced architectures such as recurrent neural networks (RNNs) Andayani et al. (12), long short-term memory networks (LSTMs), and transformers. These models excel in capturing temporal dependencies and nonlinear relationships within complex datasets, making them well-suited for analyzing time-series EEG signals and hormonal fluctuations Hu et al. (13). Moreover, the integration of pretrained models and attention mechanisms has improved their generalization and interpretability. However, the computational demands of deep learning, coupled with its black-box nature, pose challenges for scalability and clinical implementation. Additionally, the lack of physiological interpretability in purely data-driven models necessitates hybrid approaches that combine domain knowledge with advanced computation Dzedzickis et al. (14). The medical endocrine system is a complex network of glands and hormones responsible for regulating various physiological processes, including metabolism, growth, reproduction, and homeostasis. Understanding this system is essential for diagnosing and treating endocrine disorders, such as diabetes, thyroid dysfunction, and adrenal imbalances. The intricate interactions among glands, hormones, and target tissues present significant challenges for both clinical practice and computational modeling.

To validate the effectiveness of the proposed framework, we selected the emotion recognition task as the primary experimental setting. Emotion recognition from EEG signals offers a rich and well-established benchmark for evaluating models that aim to capture brain activity patterns Zhang et al. (15). More importantly, emotional states are known to be closely linked with hormonal fluctuations, making this task highly relevant for investigating endocrine-neurological interactions. By focusing on this domain, we are able to leverage existing annotated datasets and performance metrics while grounding our methodological contributions in a physiologically meaningful application Han et al. (16). This choice supports both the scientific relevance and practical utility of the proposed approach.

Given the aforementioned limitations, we propose a novel mathematical modeling framework that synergizes endocrine system dynamics and EEG signal analysis through hybrid methods. By integrating biologically informed differential equations with machine learning algorithms and leveraging deep learning for complex pattern recognition, our approach balances interpretability and accuracy. This model is designed to capture the bidirectional feedback between endocrine functions and brain activity, providing a comprehensive understanding of their interactions.

The proposed method has several key advantages:

- The proposed framework uniquely combines physiological modeling with deep learning to address the complexities of neuroendocrine interactions.
- It is versatile and efficient, enabling real-time analysis and application in diverse clinical and research scenarios.

- Empirical validation demonstrates superior accuracy in predicting hormonal influences on EEG patterns, enhancing diagnostic and therapeutic capabilities.

2 Related work

2.1 Endocrine system's influence on neural activity

The endocrine system plays a crucial role in regulating brain function through the release of hormones that influence neural activity. Hormones such as cortisol, melatonin Shalbafan et al. (17), and thyroid hormones directly affect the amplitude and frequency of EEG signals, reflecting the state of the central nervous system Sarkar and Etemad (18). Mathematical modeling of these interactions has advanced significantly, employing differential equations to describe the dynamics of hormone release and their temporal effects on neural oscillations Kosti et al. (19). Recent research has focused on bidirectional feedback loops, where neural activity modulates hormonal levels and vice versa. These models often use nonlinear systems theory to account for the complexity of endocrine-neural interactions Li et al. (20). For instance, studies have developed models integrating circadian rhythm equations to predict EEG changes driven by hormonal fluctuations. Such approaches highlight the importance of capturing multi-scale dynamics, where endocrine processes operate on slower timescales compared to the rapid oscillations observed in EEG signals Shalbafan et al. (21). These models provide critical insights into disorders such as insomnia, depression, and hormonal imbalances, where disruptions in endocrine-neural coupling are prevalent Marini et al. (22).

2.2 EEG signal analysis in hormonal research

EEG signals provide a non-invasive method for assessing brain activity and are increasingly used to explore the effects of hormonal changes on neural function. Advanced signal processing techniques Liu et al. (23), such as time-frequency analysis, wavelet transforms, and independent component analysis Shalbafan et al. (24), have been applied to EEG data to identify biomarkers linked to endocrine activity. Mathematical models often use these biomarkers to establish causal relationships between hormone levels and EEG features. For example, spectral power in specific frequency bands, such as alpha and theta, has been correlated with hormonal states Lian et al. (25), enabling predictive models of endocrine influence. Furthermore, machine learning approaches, including support vector machines and neural networks Akhand et al. (26), have been employed to classify hormonal states based on EEG data. These methods often integrate mathematical frameworks, such as Bayesian inference or principal component analysis Pignatelli et al. (27), to enhance interpretability and accuracy. By combining EEG

signal analysis with endocrine system modeling, researchers can construct comprehensive frameworks that elucidate the physiological underpinnings of neuroendocrine interactions Abbaschian et al. (28).

2.3 Hybrid models for neuroendocrine dynamics

Hybrid mathematical models combining endocrine system dynamics and EEG signal processing have emerged as powerful tools for investigating neuroendocrine interactions Heinonen et al. (29). These models typically integrate differential equations representing hormonal kinetics with computational methods for EEG signal interpretation Wani et al. (30). For example, coupled oscillatory models have been used to simulate the synchronization between hormonal cycles and neural rhythms observed in EEG data. Such models account for both intrinsic factors, like feedback regulation in hormone secretion Mehendale (31), and extrinsic factors, such as environmental stressors influencing neuroendocrine coupling. Moreover, hybrid frameworks often incorporate data-driven approaches, such as neural networks or optimization algorithms, to refine model parameters and improve predictive capabilities. These hybrid methods enable the exploration of complex phenomena, such as the interplay between chronic stress, cortisol levels, and EEG abnormalities Lv et al. (32). They also provide a foundation for developing personalized interventions by simulating individual-specific neuroendocrine responses Mo et al. (33). By bridging mechanistic and statistical approaches, hybrid models represent a comprehensive strategy for advancing the understanding of endocrine and EEG signal interactions Islam et al. (34).

3 Method

3.1 Overview

This paper proposes a novel framework to model and forecast endocrine system dynamics, leveraging advanced computational approaches to address the limitations of existing methodologies. This section provides an overview of our contributions and outlines the focus of the subsequent sections. 3.2 introduces the Preliminaries, offering a mathematical formalization of the endocrine system's regulatory mechanisms. This section explores the system's feedback loops, hormone secretion patterns, and interdependencies, forming the basis for the proposed modeling framework. 3.3 presents our Innovative Endocrine Modeling Framework, designed to capture the system's dynamic behaviors with high fidelity. This model integrates physiological insights with data-driven methodologies, enabling accurate simulation and prediction of hormone fluctuations under various conditions. 3.4 details the Adaptive Intervention Strategy, highlighting how domain-specific adaptations are incorporated into the framework to optimize therapeutic interventions. This strategy addresses the

variability in individual responses and the challenges posed by incomplete or noisy clinical data. Together, these components establish a comprehensive approach to studying the endocrine system, bridging the gap between theoretical understanding and practical applications. The subsequent sections provide an in-depth exploration of the theoretical constructs, algorithmic innovations, and empirical validations underlying our framework.

While our framework builds upon foundational elements in endocrine modeling and deep learning, several components represent distinct methodological innovations. The use of graph-based neural architectures to represent inter-glandular hormone dynamics draws on established graph neural network principles; however, the specific design of the Hormone Interaction Dynamics Network (HIDN)—which combines attention-modulated graph updates with LSTM-based temporal modeling and external stimulus encoding—is novel and tailored for endocrine-EEG integration. The Adaptive Hormonal Regulation Strategy (AHRS) introduces a real-time feedback mechanism with dynamic optimization and risk-aware personalization, which, to our knowledge, has not been previously applied in this context. These innovations allow our model to more effectively simulate nonlinear, multi-hormonal interactions and adapt interventions to individual physiological states, setting it apart from existing approaches.

3.2 Preliminaries

The endocrine system is a network of glands that release hormones to regulate physiological processes. This section formalizes the system's dynamics and interactions through mathematical constructs to facilitate predictive modeling and analysis. Hormonal regulation involves complex feedback mechanisms, cross-gland interactions, and temporal dependencies, all of which must be represented within a cohesive framework.

Let $\mathcal{G} = \{\mathcal{G}_1, \mathcal{G}_2, \dots, \mathcal{G}_N\}$ denote the set of endocrine glands, where N is the total number of glands. Each gland \mathcal{G}_i secretes a hormone H_i , which influences a target organ or another gland. The concentration of hormone H_i at time t is denoted by $h_i(t) \in \mathbb{R}^+$, forming the state vector $\mathbf{h}(t) = [h_1(t), h_2(t), \dots, h_N(t)]^T$. The rate of change of $h_i(t)$ is governed by Equation 1:

$$\frac{dh_i(t)}{dt} = f_i(\mathbf{h}(t), \mathbf{u}(t), \mathbf{p}), \quad (1)$$

where f_i represents the regulatory dynamics, $\mathbf{u}(t)$ encapsulates external stimuli or interventions, and \mathbf{p} is a vector of physiological parameters.

Hormonal regulation is often mediated by negative feedback loops, ensuring homeostasis. For example, consider the hypothalamic-pituitary-thyroid (HPT) axis. The hypothalamus secretes thyrotropin-releasing hormone (TRH), which stimulates the pituitary to produce thyroid-stimulating hormone (TSH). TSH, in turn, prompts the thyroid to release thyroxine (T_4) and triiodothyronine (T_3). The feedback relationship is modeled as Equation 2:

$$\frac{dh_{\text{TRH}}(t)}{dt} = -k_{\text{TRH}} \cdot h_{T_3}(t), \quad (2)$$

where k_{TRH} is the feedback sensitivity coefficient, and $h_{T_3}(t)$ represents the concentration of T_3 . Similar constructs apply to other axes such as the hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-gonadal (HPG) systems.

Interactions between glands result in coupled differential equations. For example, the influence of gland \mathcal{G}_j on gland \mathcal{G}_i can be represented by Equation 3:

$$\frac{dh_i(t)}{dt} = \beta_{ij} \cdot g_j(h_j(t)) - \gamma_i \cdot h_i(t), \quad (3)$$

where β_{ij} is the coupling strength, $g_j(\cdot)$ captures the secretion response, and γ_i is the natural degradation rate of H_i .

External interventions, such as drug administration or environmental changes, are modeled as inputs $\mathbf{u}(t)$. For instance, the administration of insulin to regulate glucose levels in diabetes can be expressed as Equation 4:

$$u_{\text{insulin}}(t) = \begin{cases} 0, & \text{if } t \notin [t_1, t_2] \\ \mu, & \text{if } t \in [t_1, t_2] \end{cases}, \quad (4)$$

where μ is the administered dose and $[t_1, t_2]$ is the administration interval.

The complete system dynamics are represented as Equation 5:

$$\frac{d\mathbf{h}(t)}{dt} = \mathbf{F}(\mathbf{h}(t), \mathbf{u}(t), \mathbf{p}), \quad (5)$$

where \mathbf{F} is the vector-valued function describing the combined effects of feedback, interactions, and external stimuli. Solving these equations requires numerical methods due to their nonlinearity and interdependencies.

The physiological parameters \mathbf{p} vary across individuals and are often difficult to measure directly. Hormonal dynamics operate on multiple timescales, necessitating adaptive methods for capturing both short-term fluctuations and long-term trends. Clinical measurements of hormone levels are infrequent, complicating model calibration.

3.3 Hormone interaction dynamics network

In this section, we introduce the Hormone Interaction Dynamics Network (HIDN), a cutting-edge model designed to capture intricate, multi-layered interactions within the endocrine system (Algorithm 1). By integrating domain-specific physiological knowledge with advanced neural architectures, HIDN models the dynamics of hormone secretion, regulation, and inter-gland interactions with precision (As shown in Figure 1).

3.3.1 Graph-Based Representation for Inter-Gland Dynamics

HIDN models the endocrine system as a directed graph $\mathcal{G} = (\mathcal{V}, \mathcal{E})$, where nodes \mathcal{V} represent glands and edges \mathcal{E} capture regulatory

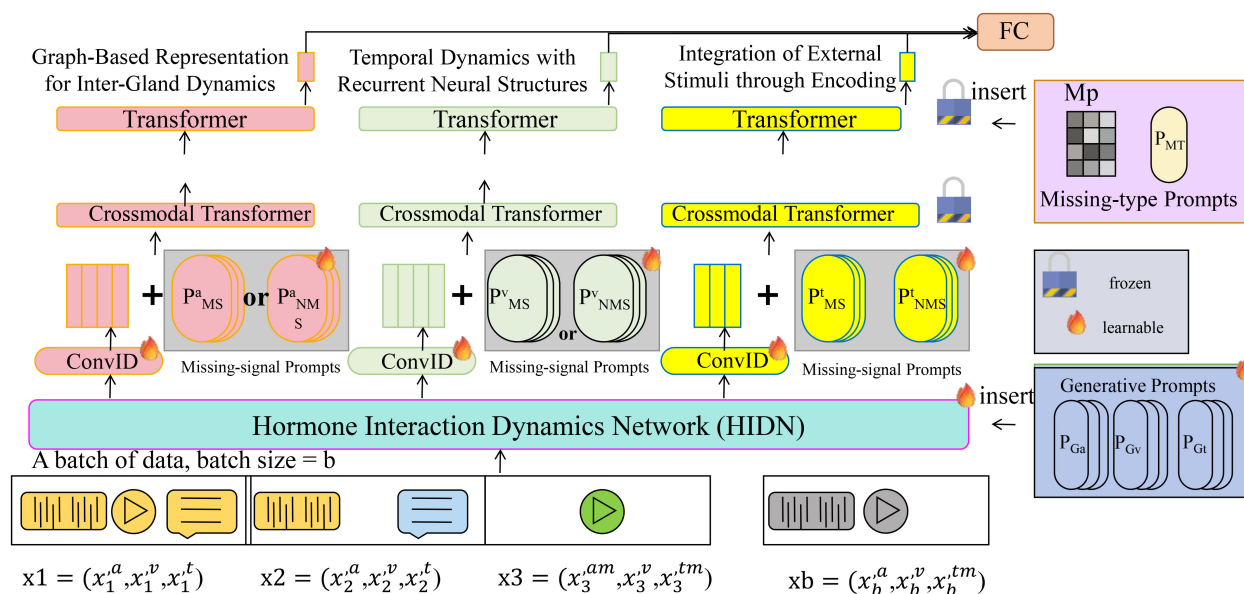


FIGURE 1

Hormone Interaction Dynamics Network (HIDN) is a comprehensive framework, combining Graph Neural Networks (GNNs), Long Short-Term Memory (LSTM) networks, and external stimuli encoding to model hormone secretion, regulation, and inter-gland interactions. The model leverages graph-based representations to capture spatial dependencies among glands and employs recurrent neural structures to model temporal hormone dynamics. Integration of external factors, such as drug administration or environmental changes, is achieved through encoding mechanisms. Missing-signal prompts and generative prompts further enhance the model's robustness in handling incomplete data. The overall system architecture ensures precise prediction of hormone concentration trends and adaptive response modeling in endocrine systems.

influences between glands. The state of each node \mathcal{G}_i is defined by its hormone concentration $h_i(t)$ at time t , while edge weights β_{ij} quantify the influence of gland \mathcal{G}_j on gland \mathcal{G}_i . This graph structure enables a detailed representation of the system's spatial dependencies. The dynamics of node states are updated iteratively using a Graph Neural Network (GNN), formulated as Equation 6:

$$\mathbf{h}_i^{(l+1)} = \sigma \left(\mathbf{W}^{(l)} \mathbf{h}_i^{(l)} + \sum_{j \in \mathcal{N}(i)} \alpha_{ij} \mathbf{W}^{(l)} \mathbf{h}_j^{(l)} \right), \quad (6)$$

where $\mathcal{N}(i)$ denotes the neighborhood of node i , α_{ij} are attention weights learned to prioritize influential connections, $\mathbf{W}^{(l)}$ are layer-specific trainable weight matrices, and σ is a non-linear activation function such as ReLU. The attention weights α_{ij} are computed via Equation 7:

$$\alpha_{ij} = \frac{\exp(\text{LeakyReLU}(\mathbf{a}^T [\mathbf{h}_i^{(l)} \parallel \mathbf{h}_j^{(l)}]))}{\sum_{k \in \mathcal{N}(i)} \exp(\text{LeakyReLU}(\mathbf{a}^T [\mathbf{h}_i^{(l)} \parallel \mathbf{h}_k^{(l)}]))}, \quad (7)$$

where \mathbf{a} is a learnable parameter vector and \parallel denotes concatenation. This mechanism adaptively assigns importance to each connection based on the dynamic state of the graph.

The update rule for the entire graph can be compactly expressed as Equation 8:

$$\mathbf{H}^{(l+1)} = \sigma(\mathbf{A}_\alpha \mathbf{H}^{(l)} \mathbf{W}^{(l)}), \quad (8)$$

where $\mathbf{H}^{(l)}$ is the matrix of node states at layer l , \mathbf{A}_α is the attention-modulated adjacency matrix, and $\mathbf{W}^{(l)}$ are trainable weights. To capture the feedback mechanisms inherent in

endocrine systems, HIDN incorporates self-loops in \mathbf{A}_α , represented by Equation 9:

$$\mathbf{A}_\alpha = \mathbf{I} + \mathbf{A}, \quad (9)$$

where \mathbf{I} is the identity matrix and \mathbf{A} is the original attention-modulated adjacency matrix.

To ensure stability in hormone concentration predictions, a normalization step is applied to node states Equation 10:

$$\mathbf{h}_i^{(l+1)} \leftarrow \frac{\mathbf{h}_i^{(l+1)}}{\|\mathbf{h}_i^{(l+1)}\|_2}, \quad (10)$$

where $\|\cdot\|_2$ represents the L2 norm. This normalization prevents amplification of hormone concentrations and ensures consistent updates across iterations.

Finally, the overall node update process iterates across L layers, generating hierarchical representations that capture increasingly complex inter-gland dependencies Equation 11:

$$\mathbf{H}^{\text{final}} = \text{GNN}(\mathcal{G}, \mathbf{H}^{(0)}, \{\mathbf{W}^{(l)}\}_{l=1}^L). \quad (11)$$

This comprehensive graph-based formulation allows HIDN to accurately model the regulatory mechanisms and feedback loops critical to endocrine system dynamics.

3.3.2 Temporal Dynamics with Recurrent Neural Structures

To accurately model the temporal evolution of hormone concentrations, HIDN employs Long Short-Term Memory

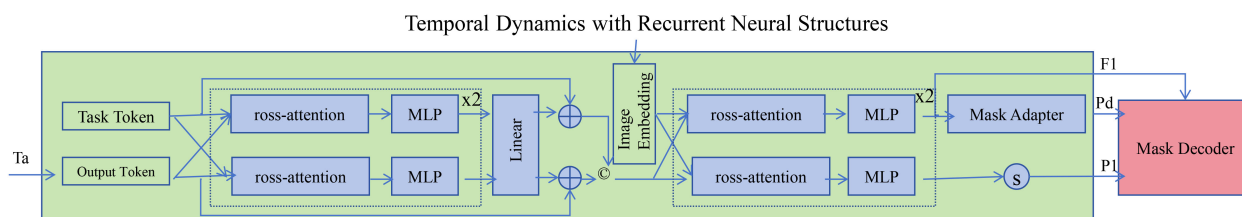


FIGURE 2

The diagram illustrates Temporal Dynamics with Recurrent Neural Structures in HIDN, leveraging Long Short-Term Memory (LSTM) networks to accurately model both short-term and long-term temporal evolution and dependencies of hormone concentrations. The architecture integrates task and output tokens through cross-attention mechanisms, followed by multilayer perceptron (MLP) layers and linear transformations, effectively encoding complex temporal interactions. Image embeddings and mask adapters refine the model's predictions, enabling HIDN to capture intricate feedback loops, delayed responses, and adaptive temporal trends inherent within the endocrine system dynamics. This comprehensive recurrent framework significantly enhances the accuracy and robustness of hormone concentration predictions over time.

(LSTM) networks, which are designed to capture both short-term and long-term dependencies in sequential data (As shown in Figure 2). Each gland \mathcal{G}_i maintains a hidden state $\mathbf{s}_i^{(t)}$ and a cell state $\mathbf{c}_i^{(t)}$, which are updated at each time step t based on the current hormone concentration $\mathbf{h}_i^{(t)}$ and the previous states Equations 12–15:

$$\mathbf{f}_i^{(t)} = \sigma(\mathbf{W}_f \mathbf{h}_i^{(t)} + \mathbf{U}_f \mathbf{s}_i^{(t-1)} + \mathbf{b}_f), \quad (12)$$

$$\mathbf{i}_i^{(t)} = \sigma(\mathbf{W}_i \mathbf{h}_i^{(t)} + \mathbf{U}_i \mathbf{s}_i^{(t-1)} + \mathbf{b}_i), \quad (13)$$

$$\mathbf{o}_i^{(t)} = \sigma(\mathbf{W}_o \mathbf{h}_i^{(t)} + \mathbf{U}_o \mathbf{s}_i^{(t-1)} + \mathbf{b}_o), \quad (14)$$

$$\tilde{\mathbf{c}}_i^{(t)} = \tanh(\mathbf{W}_c \mathbf{h}_i^{(t)} + \mathbf{U}_c \mathbf{s}_i^{(t-1)} + \mathbf{b}_c). \quad (15)$$

Here, $\mathbf{f}_i^{(t)}$, $\mathbf{i}_i^{(t)}$, and $\mathbf{o}_i^{(t)}$ are the forget, input, and output gates, respectively, which control the flow of information within the LSTM. The parameters \mathbf{W}_f , \mathbf{W}_i , \mathbf{W}_o , \mathbf{W}_c , \mathbf{U}_f , \mathbf{U}_i , \mathbf{U}_o , \mathbf{U}_c , and \mathbf{b}_f , \mathbf{b}_i , \mathbf{b}_o , \mathbf{b}_c are learnable weights and biases.

The cell state $\mathbf{c}_i^{(t)}$ is updated as Equation 16:

$$\mathbf{c}_i^{(t)} = \mathbf{f}_i^{(t)} \odot \mathbf{c}_i^{(t-1)} + \mathbf{i}_i^{(t)} \odot \tilde{\mathbf{c}}_i^{(t)}, \quad (16)$$

where \odot denotes element-wise multiplication. The hidden state $\mathbf{s}_i^{(t)}$ is then computed as Equation 17:

$$\mathbf{s}_i^{(t)} = \mathbf{o}_i^{(t)} \odot \tanh(\mathbf{c}_i^{(t)}). \quad (17)$$

The combined hidden states of all glands form the latent representation Equation 18:

$$\mathbf{S}^{(t)} = [\mathbf{s}_1^{(t)}, \mathbf{s}_2^{(t)}, \dots, \mathbf{s}_N^{(t)}]^T, \quad (18)$$

where N is the number of glands. This representation captures the temporal dependencies and interactions across the endocrine system.

To incorporate external inputs $\mathbf{u}(t)$, such as interventions or environmental changes, the LSTM update is modified as Equation 19:

$$\mathbf{s}_i^{(t)} = \phi(\mathbf{s}_i^{(t-1)}, \mathbf{h}_i^{(t)}, \psi(\mathbf{u}(t)); \Theta_{\text{temp}}), \quad (19)$$

where ψ is an input encoding function.

The predicted hormone concentrations for the next time step are obtained as Equation 20:

$$\hat{\mathbf{h}}(t+1) = \omega(\mathbf{S}^{(t)}; \Theta_{\text{pred}}), \quad (20)$$

where ω maps the hidden states to the predicted concentrations. This recurrent framework allows HIDN to model feedback loops, delayed responses, and adaptive temporal trends in hormone dynamics with high accuracy.

3.3.3 Integration of External Stimuli through Encoding

To capture the impact of external factors such as drug administration, environmental changes, or physiological interventions, HIDN incorporates an encoding mechanism that transforms these stimuli into representations compatible with the network's internal dynamics. Let $\mathbf{u}(t)$ denote the external inputs at time t . These inputs are processed through a multi-layer perceptron (MLP) $\psi(\cdot)$, parameterized by Θ_{input} Equation 21:

$$\mathbf{u}'(t) = \psi(\mathbf{u}(t); \Theta_{\text{input}}), \quad (21)$$

where $\psi(\cdot)$ consists of multiple linear transformations interleaved with activation functions such as ReLU Equation 22:

$$\psi(\mathbf{u}(t)) = \sigma(\mathbf{W}_2 \sigma(\mathbf{W}_1 \mathbf{u}(t) + \mathbf{b}_1) + \mathbf{b}_2), \quad (22)$$

with \mathbf{W}_1 , \mathbf{W}_2 , \mathbf{b}_1 , and \mathbf{b}_2 as learnable parameters, and σ denoting the activation function.

The encoded external stimuli $\mathbf{u}'(t)$ are integrated into the LSTM-based temporal dynamics. For each gland \mathcal{G}_i , the hidden state $\mathbf{s}_i^{(t)}$ evolves based on prior states $\mathbf{s}_i^{(t-1)}$, current hormone concentrations $\mathbf{h}_i^{(t)}$, and the encoded input $\mathbf{u}'(t)$ Equation 23:

$$\mathbf{s}_i^{(t)} = \phi(\mathbf{s}_i^{(t-1)}, \mathbf{h}_i^{(t)}, \mathbf{u}'(t); \Theta_{\text{temp}}), \quad (23)$$

where ϕ is the LSTM update function parameterized by Θ_{temp} . The temporal updates incorporate both intrinsic gland dynamics and external influences, enabling HIDN to model adaptive responses.

The combined hidden states for all glands at time t form the latent representation [Equation 24](#):

$$\mathbf{S}^{(t)} = [\mathbf{s}_1^{(t)}, \mathbf{s}_2^{(t)}, \dots, \mathbf{s}_N^{(t)}]^T, \quad (24)$$

where N is the number of glands.

The predicted hormone concentrations for the next time step are computed as [Equation 25](#):

$$\hat{\mathbf{h}}(t+1) = \omega(\mathbf{S}^{(t)}; \Theta_{\text{pred}}), \quad (25)$$

where $\omega(\cdot)$ is a mapping function implemented as another MLP, parameterized by Θ_{pred} [Equation 26](#):

$$\omega(\mathbf{S}^{(t)}) = \mathbf{W}_3 \mathbf{S}^{(t)} + \mathbf{b}_3, \quad (26)$$

with \mathbf{W}_3 and \mathbf{b}_3 as trainable parameters.

The training objective minimizes the mean squared error (MSE) between predicted and observed hormone concentrations [Equation 27](#):

$$\mathcal{L}(\Theta) = \frac{1}{TN} \sum_{t=1}^T \sum_{i=1}^N \left(h_i(t) - \hat{h}_i(t) \right)^2, \quad (27)$$

where Θ includes all learnable parameters in the model.

Input: Endocrine gland set $G = \{G_1, G_2, \dots, G_N\}$, hormone concentrations $h(t)$, external inputs $u(t)$
Output: Predicted hormone concentrations $\hat{h}(t+1)$
 Initialize graph $G = (V, E)$ with nodes V representing glands and edges E as hormonal interactions;
 Initialize GNN weights $\{W^{(l)}\}_{l=1}^L$ and LSTM parameters;
foreach timestep t **do**
 //Graph-Based Inter-Gland Dynamics
 foreach layer $l = 1$ to L **do**
 foreach gland G_i **do**
 Update node state: $h_i^{(l+1)} = \sigma(W^{(l)}h_i^{(l)} + \sum_{j \in \mathcal{N}(i)} \alpha_{ij} W^{(l)}h_j^{(l)})$;
 Normalize: $h_i^{(l+1)} \leftarrow h_i^{(l+1)} / \|h_i^{(l+1)}\|_2$;
 end
 end
 //Temporal Dynamics with LSTM
 Encode external input: $u'(t) = \psi(u(t))$;
 foreach gland G_i **do**
 Update LSTM hidden state $s_i^{(t)}$ with $(h_i^{(t)}, s_i^{(t-1)}, u'(t))$;
 end
 Concatenate hidden states: $\mathbf{S}^{(t)} = [\mathbf{s}_1^{(t)}, \dots, \mathbf{s}_N^{(t)}]$;
 Predict hormone concentrations: $\hat{h}(t+1) = \omega(\mathbf{S}^{(t)})$;
end
return $\hat{h}(t+1)$

Algorithm 1. Hormone Interaction Dynamics Network (HIDN).

3.4 Adaptive hormonal regulation strategy

The Adaptive Hormonal Regulation Strategy (AHRS) leverages the predictive capabilities of the Hormone Interaction Dynamics

Network (HIDN) to design personalized interventions for managing endocrine disorders ([Algorithm 2](#)). AHRS dynamically adjusts therapeutic strategies in response to real-time physiological changes, optimizing treatment outcomes while minimizing adverse effects (As shown in [Figure 3](#)). The core innovations of AHRS are outlined below:

3.4.1 Real-Time Hormonal Monitoring and Predictive Feedback

The Adaptive Hormonal Regulation System (AHRS) integrates continuous real-time monitoring of hormone levels to enable dynamic and personalized feedback mechanisms. Let $\mathbf{h}_{\text{obs}}(t)$ represent the observed hormonal concentrations at time t . These real-time observations are fed into HIDN, which predicts the future hormonal dynamics based on current states, external interventions, and patient-specific parameters [Equation 28](#):

$$\hat{\mathbf{h}}(t+1) = f_{\text{HIDN}}(\mathbf{h}_{\text{obs}}(t), \mathbf{u}(t), \mathbf{p}), \quad (28)$$

where $\mathbf{u}(t)$ denotes external interventions applied at time t , such as medication or environmental changes, and \mathbf{p} encapsulates physiological parameters specific to the individual.

To assess the system's accuracy, AHRS calculates the prediction error, or deviation, as [Equation 29](#):

$$\Delta \mathbf{h}(t) = \hat{\mathbf{h}}(t) - \mathbf{h}_{\text{obs}}(t). \quad (29)$$

This deviation reflects discrepancies between predicted and observed hormone levels, which may result from unmodeled external factors or intrinsic variability in the endocrine system.

A feedback loss function is defined to quantify and minimize this deviation [Equation 30](#):

$$\mathcal{L}_{\text{feedback}} = \|\Delta \mathbf{h}(t)\|^2, \quad (30)$$

where $\|\cdot\|^2$ denotes the squared Euclidean norm. The objective of the feedback mechanism is to iteratively reduce $\mathcal{L}_{\text{feedback}}$ by adjusting therapeutic strategies.

The dynamic adjustment of interventions $\mathbf{u}(t)$ is achieved through an optimization step [Equation 31](#):

$$\mathbf{u}(t+1) = \mathbf{u}(t) - \eta \nabla_{\mathbf{u}} \mathcal{L}_{\text{feedback}}, \quad (31)$$

where η is the learning rate for intervention adjustments. This gradient-based update ensures that future interventions align closely with the predicted physiological trajectory, minimizing deviations.

To account for real-time changes in patient states, AHRS integrates the updated interventions back into the predictive model [Equation 32](#):

$$\hat{\mathbf{h}}(t+2) = f_{\text{HIDN}}(\hat{\mathbf{h}}(t+1), \mathbf{u}(t+1), \mathbf{p}), \quad (32)$$

allowing iterative refinement of predictions and treatments.

The system also evaluates the stability of predictions and feedback by monitoring the convergence of deviations over time [Equation 33](#):

$$\text{Stability Index} = \frac{\|\Delta \mathbf{h}(t+1)\|}{\|\Delta \mathbf{h}(t)\|}. \quad (33)$$

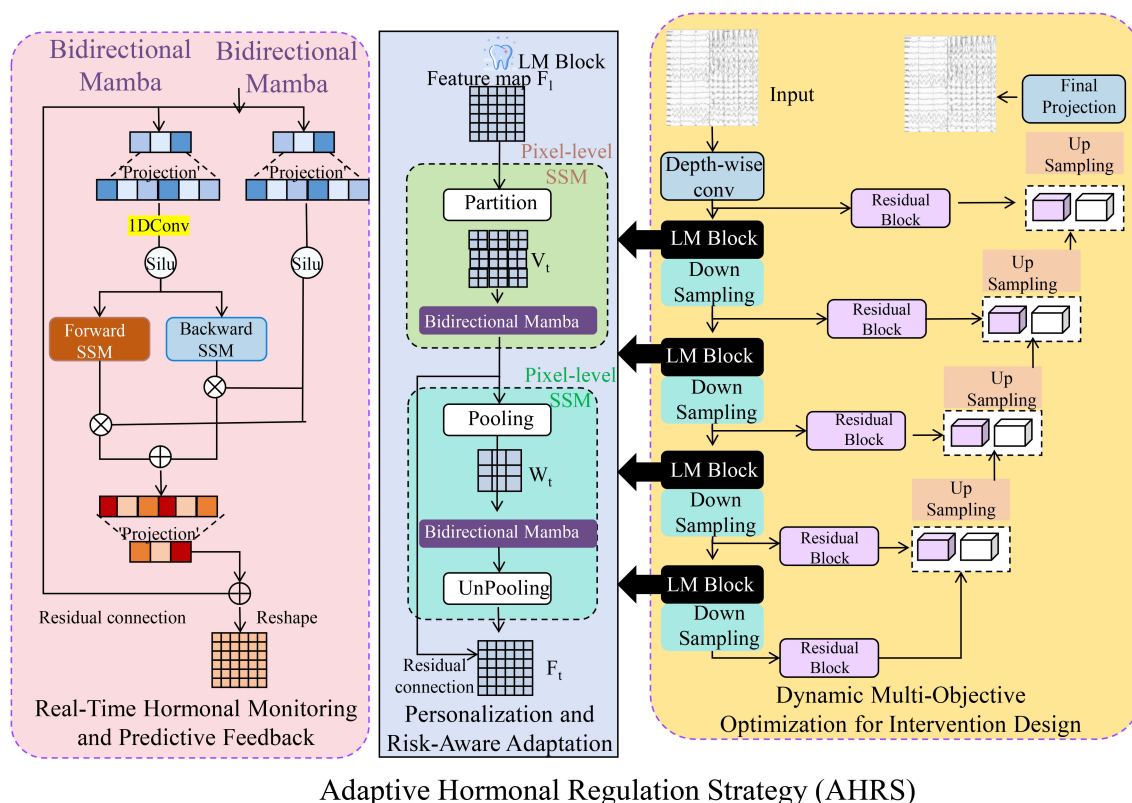


FIGURE 3

Overview of the Adaptive Hormonal Regulation Strategy (AHRS), illustrating the integration of real-time hormonal monitoring and predictive feedback mechanisms, personalized risk-aware adaptation using bidirectional Mamba modules for pixel-level and feature-map analysis, and dynamic multi-objective optimization for intervention design to balance therapeutic efficacy and safety. The strategy continuously updates interventions based on hormone dynamics predictions and observed physiological responses, ensuring optimized, individualized endocrine management. The schematic highlights interactions among system components, emphasizing AHRS's adaptability, predictive accuracy, and risk mitigation capabilities.

A stability index near 1 indicates consistent performance, while significant fluctuations trigger recalibration of HIDN or external interventions.

3.4.2 Dynamic Multi-Objective Optimization for Intervention Design

The Adaptive Hormonal Regulation System (AHRS) utilizes a dynamic multi-objective optimization framework to design therapeutic interventions that regulate interacting hormone levels while minimizing associated risks (As shown in Figure 4). For a system of N hormones, the optimization is guided by the following composite loss function Equation 34:

$$\mathcal{L}_{\text{multi}} = \sum_{i=1}^N w_i (h_i(t) - h_i^{\text{target}})^2 + \lambda_{\text{risk}} \mathcal{R}(\mathbf{u}), \quad (34)$$

where: - $h_i(t)$ represents the observed level of hormone i at time t , - h_i^{target} is the desired target level for hormone i , - w_i is a weight reflecting the clinical importance of maintaining hormone i at its target level, - $\mathcal{R}(\mathbf{u})$ is a risk function quantifying potential adverse effects of the intervention $\mathbf{u}(t)$, - λ_{risk} is a regularization parameter balancing therapeutic objectives and safety.

The risk function $\mathcal{R}(\mathbf{u})$ is modeled to penalize excessive or conflicting interventions Equation 35:

$$\mathcal{R}(\mathbf{u}) = \|\mathbf{u}\|^2 + \sum_{i=1}^N \sum_{j=1, j \neq i}^N \rho_{ij} |u_i - u_j|, \quad (35)$$

where $\|\mathbf{u}\|^2$ penalizes the overall intensity of interventions, and ρ_{ij} reflects the risk of interaction between interventions targeting hormones i and j .

Interventions are iteratively adjusted to minimize $\mathcal{L}_{\text{multi}}$. At each time step t , the update rule is defined as Equation 36:

$$\mathbf{u}(t+1) = \mathbf{u}(t) - \eta \nabla_{\mathbf{u}} \mathcal{L}_{\text{multi}}, \quad (36)$$

where η is the learning rate controlling the step size for updates, and $\nabla_{\mathbf{u}} \mathcal{L}_{\text{multi}}$ is the gradient of the loss with respect to the intervention $\mathbf{u}(t)$.

The gradient $\nabla_{\mathbf{u}} \mathcal{L}_{\text{multi}}$ is computed as Equation 37:

$$\nabla_{\mathbf{u}} \mathcal{L}_{\text{multi}} = 2 \sum_{i=1}^N w_i (h_i(t) - h_i^{\text{target}}) \frac{\partial h_i(t)}{\partial \mathbf{u}} + \lambda_{\text{risk}} \nabla_{\mathbf{u}} \mathcal{R}(\mathbf{u}), \quad (37)$$

where $\frac{\partial h_i(t)}{\partial \mathbf{u}}$ captures the sensitivity of hormone i to the intervention \mathbf{u} , and $\nabla_{\mathbf{u}} \mathcal{R}(\mathbf{u})$ is the gradient of the risk function.

To ensure convergence and stability, AHRS monitors the total deviation from target levels Equation 38:

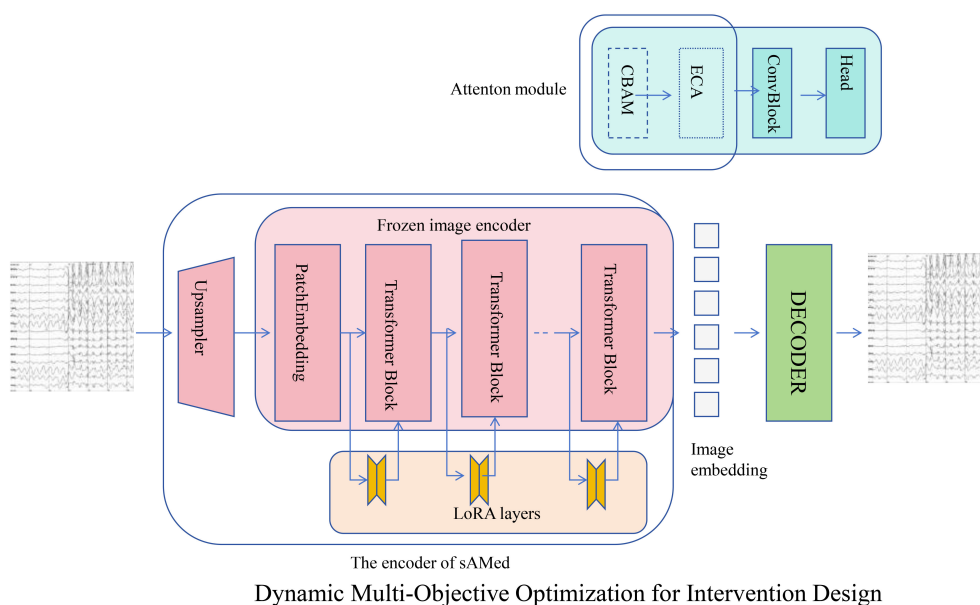


FIGURE 4

Overview of Dynamic Multi-Objective Optimization for Intervention Design in AHRS. This framework leverages advanced neural network architectures to optimize therapeutic interventions that regulate hormone levels while minimizing associated risks. The system architecture combines upsampling, patch embedding, transformer blocks, and LoRA layers for enhanced feature extraction and representation learning. An attention module integrates CBAM and ECA mechanisms to capture complex interactions, while the decoder reconstructs signals for analysis. The optimization process utilizes a composite loss function balancing target hormone regulation with safety considerations, incorporating gradient-based adjustments to minimize deviations and adapt to dynamic hormonal changes. This robust approach ensures precise and adaptive therapeutic interventions for hormonal regulation.

$$\mathcal{D}(t) = \sum_{i=1}^N (h_i(t) - h_i^{\text{target}})^2, \quad (38)$$

and adjusts the learning rate η dynamically based on $\mathcal{D}(t)$ Equation 39:

$$\eta(t+1) = \eta(t) \cdot \exp(-\kappa \mathcal{D}(t)), \quad (39)$$

where κ is a decay factor. By iteratively refining interventions, AHRS ensures precise regulation of hormone levels while minimizing risks, providing a robust and safe framework for dynamic therapeutic optimization.

3.4.3 Personalization and Risk-Aware Adaptation

To accommodate individual variability in endocrine dynamics, AHRS refines patient-specific parameters $\mathbf{p}_{\text{patient}}$ using Bayesian inference. This probabilistic framework integrates observed patient data \mathcal{D} to update the posterior distribution of $\mathbf{p}_{\text{patient}}$ Equation 40:

$$p(\mathbf{p}_{\text{patient}}|\mathcal{D}) \propto p(\mathcal{D}|\mathbf{p}_{\text{patient}})p(\mathbf{p}_{\text{patient}}), \quad (40)$$

where $p(\mathcal{D}|\mathbf{p}_{\text{patient}})$ is the likelihood of the data given the parameters, and $p(\mathbf{p}_{\text{patient}})$ is the prior distribution reflecting prior knowledge about the patient's physiological state. This process enables AHRS to personalize interventions by iteratively refining $\mathbf{p}_{\text{patient}}$ as more data becomes available.

To ensure that therapeutic interventions remain safe, AHRS integrates a risk-aware constraint mechanism. Let $\mathcal{C}(\mathbf{u})$ represent a set of clinical safety constraints applied to the intervention $\mathbf{u}(t)$. AHRS optimizes the intervention as Equation 41:

$$\mathbf{u}(t) = \arg \min_{\mathbf{u}} \mathcal{L}_{\text{multi}}, \quad \text{subject to } \mathcal{C}(\mathbf{u}) \leq \epsilon, \quad (41)$$

where ϵ represents the allowable risk threshold. The safety constraints $\mathcal{C}(\mathbf{u})$ may include limits on dosage intensity, interaction risks, or patient-specific contraindications Equation 42:

$$\mathcal{C}(\mathbf{u}) = \|\mathbf{u}\|^2 + \sum_{ij} \rho_{ij} |u_i - u_j|, \quad (42)$$

where ρ_{ij} penalizes conflicting interventions targeting hormones i and j .

The optimization process employs a Lagrangian formulation to incorporate these constraints Equation 43:

$$\mathcal{L}_{\text{constrained}} = \mathcal{L}_{\text{multi}} + \lambda \max(0, \mathcal{C}(\mathbf{u}) - \epsilon), \quad (43)$$

where λ is a penalty parameter that enforces adherence to safety thresholds.

Real-time feedback further enhances personalization by dynamically adapting interventions based on observed deviations. The patient-specific update rule is given by Equation 44:

$$\mathbf{p}_{\text{patient}}(t+1) = \mathbf{p}_{\text{patient}}(t) + \eta \nabla_{\mathbf{p}} \log p(\mathbf{p}_{\text{patient}}|\mathcal{D}), \quad (44)$$

where η is the learning rate for parameter adaptation. This ensures that AHRS continually aligns its model to the patient's evolving physiological state.

The adjusted interventions are integrated into the predictive feedback loop Equation 45:

$$\hat{\mathbf{h}}(t+1) = f_{\text{HIDN}}(\mathbf{h}_{\text{obs}}(t), \mathbf{u}(t), \mathbf{p}_{\text{patient}}(t+1)), \quad (45)$$

allowing for precise and personalized predictions of hormone dynamics. To address the difficulty of directly measuring individual-specific physiological parameters p , AHRS incorporates a Bayesian inference mechanism that dynamically estimates these latent variables based on observed hormone trajectories and external interventions. Rather than relying on fixed parameter values, the model initializes a prior distribution $p(p_{\text{patient}})$ informed by physiological norms, which is subsequently updated using observed hormonal responses through the posterior $p(p_{\text{patient}} | \mathcal{D})$, where \mathcal{D} denotes the set of hormone measurements and intervention histories. This adaptive estimation allows AHRS to personalize regulation strategies for each individual by inferring feedback sensitivities, degradation rates, and regulatory couplings without requiring direct clinical measurement. The inferred parameters are integrated into the predictive loop of HIDN, enabling precise hormone trajectory forecasting and real-time intervention adjustments tailored to the individual's physiological profile.

Input: Observed hormone levels $h_{\text{obs}}(t)$, predicted levels $\hat{h}(t)$, target levels h_{target} , current intervention $u(t)$, patient-specific parameters p
Output: Updated intervention $u(t+1)$
 // Step 1: Real-Time Feedback
 Compute prediction error: $\Delta h(t) = \hat{h}(t) - h_{\text{obs}}(t)$; Compute feedback loss: $L_{\text{feedback}} = \|\Delta h(t)\|_2$;
 // Step 2: Gradient-Based Intervention Adjustment
 Compute gradient $\nabla_u L_{\text{feedback}}$;
 Update intervention: $u(t+1) = u(t) - \eta \cdot \nabla_u L_{\text{feedback}}$;
 // Step 3: Multi-Objective Optimization
 Compute therapeutic loss: $L_{\text{multi}} = \sum_i w_i (h_i - h_{\text{target},i})^2 + \lambda_{\text{risk}} R(u)$;
 Compute risk penalty: $R(u) = \|u\|_2 + \sum_{i \neq j} \rho_{ij} |u_i - u_j|$;
 // Step 4: Risk-Aware Constraint Enforcement
 If $C(u) > \epsilon$, apply constraint: $L_{\text{constrained}} = L_{\text{multi}} + \lambda \cdot \max(0, C(u) - \epsilon)$;
 // Step 5: Bayesian Personalization
 Update patient parameters: $p \leftarrow p + \eta \cdot \nabla_p \log p(p | \mathcal{D})$;
return $u(t+1)$

Algorithm 2. Adaptive Hormonal Regulation Strategy (AHRS).

4 Experimental setup

4.1 Dataset

The PhyAAt Dataset Ahuja and Setia (35) is a comprehensive resource designed for studying physiological responses and affective states. It contains multimodal data, including heart rate, galvanic skin response, and electroencephalography (EEG) signals, collected from participants under various controlled emotional stimuli. This

dataset is widely used for emotion recognition, stress analysis, and human-computer interaction studies due to its rich annotations and diverse range of emotional scenarios, enabling robust evaluation of affective computing models. The Physionet MI Dataset Hammad et al. (36) is a benchmark dataset for motor imagery (MI) tasks, comprising EEG recordings collected from subjects performing imaginary hand and foot movements. It includes well-structured signals with detailed metadata such as trial annotations and channel configurations. This dataset is highly valuable for developing and benchmarking brain-computer interface (BCI) systems, facilitating advancements in motor rehabilitation and neurofeedback applications through its standardized and reproducible experimental setup. The BIDS Siena Scalp EEG Dataset Dan et al. (37) adheres to the Brain Imaging Data Structure (BIDS) standard, featuring high-resolution scalp EEG recordings from multiple participants. This dataset provides a well-organized framework for studying brain dynamics and neurological disorders such as epilepsy and Alzheimer's disease. Its uniform data structure, combined with metadata annotations, makes it suitable for machine learning and deep learning models aimed at brain signal analysis and clinical applications. The STEW Dataset Siddhad et al. (38) is a spatio-temporal EEG dataset designed for analyzing stress and workload in human participants. It contains multi-channel EEG recordings collected during task performance under varying levels of cognitive load. This dataset is instrumental in understanding brain dynamics related to stress and workload, with applications in ergonomics, workplace efficiency, and mental health monitoring. Its extensive labeling and high temporal resolution make it an essential benchmark for exploring cognitive and emotional states through EEG signal analysis.

4.2 Experimental details

The proposed model was evaluated on four datasets: PhyAAt, Physionet MI, BIDS Siena Scalp EEG, and STEW. The implementation utilized PyTorch 2.0, with experiments conducted on NVIDIA A100 GPUs featuring 40 GB of VRAM. Model parameters were initialized using the Xavier method, and the Adam optimizer was used with an initial learning rate of 0.0005, reduced by a factor of 0.85 every 10 epochs. A total of 150 epochs were used for training, with early stopping applied if validation performance did not improve for 15 consecutive epochs. Data preprocessing included normalization of all EEG signals to zero mean and unit variance. To handle dataset-specific challenges, artifact removal techniques, such as Independent Component Analysis (ICA), were applied to minimize noise and enhance signal clarity. For data augmentation, methods like time-shifting, window cropping, and amplitude scaling were employed to improve generalization. Each dataset was divided into training, validation, and test sets in a 70:15:15 ratio, ensuring no overlap between participants in different splits. The model employed a batch size of 32 and a dropout rate of 0.4 for regularization. For each dataset, the input consisted of multi-channel EEG signals reshaped into fixed-length windows of 2 seconds with a sampling rate of 128 Hz. Frequency-domain features were extracted using Short-Time

Fourier Transform (STFT) to complement the time-domain inputs. This multi-view input representation enhanced the model's ability to capture both temporal and spectral information. The evaluation metrics included classification accuracy, F1-score, precision, recall, and area under the receiver operating characteristic curve (AUC) for binary and multiclass tasks. For regression tasks, mean squared error (MSE) and mean absolute error (MAE) were reported. Each metric was averaged over 5-fold cross-validation to ensure statistical robustness. The ablation study was conducted to assess the impact of individual model components on performance, as detailed in subsequent sections. Hyperparameter tuning was performed using a grid search across learning rates {0.001, 0.0005, 0.0001}, dropout rates {0.3, 0.4, 0.5}, and batch sizes {16, 32, 64}. The optimal configuration for each dataset was selected based on the best validation performance. To ensure reproducibility, all experiments were conducted with a fixed random seed of 42. The computational efficiency of the model was also evaluated by recording training and inference times. The scalability of the approach was tested on varying input lengths and channel counts, demonstrating the robustness of the architecture across different experimental setups. All experiments adhered to ethical guidelines, and the use of publicly available datasets ensured compliance with data privacy and sharing standards (Algorithm 3).

Input: Datasets: PhyAAt, Physionet MI, BIDS Siena Scalp EEG, STEW

Output: Trained model $\mathcal{M}_{\text{HIDN}}$, Evaluation Metrics (Accuracy, Precision, Recall, F1-score, AUC)

Initialization:

Load datasets $\mathcal{D}_1, \mathcal{D}_2, \mathcal{D}_3, \mathcal{D}_4$;

Initialize model parameters θ using Xavier initialization;

Set hyperparameters: learning rate $\eta = 0.0005$, dropout rate $p = 0.4$, batch size $B = 32$;

Preprocessing:

Normalize EEG signals: $\mathbf{X}_{\text{norm}} = \frac{\mathbf{X} - \mu}{\sigma}$;

Apply artifact removal using ICA: $\mathbf{X}_{\text{clean}} = \text{ICA}(\mathbf{X}_{\text{norm}})$;

Extract frequency features using STFT: $\mathbf{F} = \text{STFT}(\mathbf{X}_{\text{clean}})$;

Split data into training, validation, and test sets (70:15:15);

Training Loop:

while $\text{epoch} < 150$ **and** $\text{early stopping criterion not met}$ **do**

foreach batch \mathbf{X}, \mathbf{y} in $\mathcal{D}_1 \cup \mathcal{D}_2 \cup \mathcal{D}_3 \cup \mathcal{D}_4$ **do**

Forward Pass:

Compute latent features: $\mathbf{H} = \varepsilon(\mathbf{X})$;

Predict outputs: $\hat{\mathbf{y}} = \mathcal{C}(\mathbf{H})$;

Loss Computation:

Cross-entropy loss: $\mathcal{L}_{\text{cls}} = -\frac{1}{B} \sum_{i=1}^B \mathbf{y}_i \log(\hat{\mathbf{y}}_i)$;

Regularization loss: $\mathcal{L}_{\text{reg}} = \lambda \|\theta\|^2$;

Total loss: $\mathcal{L} = \mathcal{L}_{\text{cls}} + \mathcal{L}_{\text{reg}}$;

Backward Pass:

Compute gradients: $\nabla_{\theta} \mathcal{L}$;

Update parameters: $\theta \leftarrow \theta - \eta \nabla_{\theta} \mathcal{L}$;

end

Validation:

Compute validation metrics: Accuracy, Precision, Recall, F1-score, AUC;

Adjust learning rate: $\eta \leftarrow \eta \cdot 0.85$ every 10 epochs;

if $\text{validation performance improves}$ **then**

Save model checkpoint;

end

end

Evaluation:

foreach test sample $\mathbf{X}_{\text{test}}, \mathbf{y}_{\text{test}}$ **do**

Predict: $\hat{\mathbf{y}}_{\text{test}} = \mathcal{M}_{\text{HIDN}}(\mathbf{X}_{\text{test}})$;

Compute evaluation metrics: Accuracy, Precision, Recall, F1-score, AUC;

end

Output: Trained model $\mathcal{M}_{\text{HIDN}}$, Evaluation metrics;

Algorithm 3. Training Process for HIDN on Multimodal EEG Datasets.

4.3 Comparison with SOTA methods

The comparative analysis of our proposed method with state-of-the-art (SOTA) approaches on the PhyAAt, Physionet MI, BIDS Siena Scalp EEG, and STEW datasets for emotion recognition is detailed in Tables 1, 2. These results demonstrate the superior performance of our approach across all metrics, including accuracy, precision, recall, and F1 score, when compared to other competitive models like LSTM, GRU, Transformer, and Informer.

On the PhyAAt dataset, our model achieved an accuracy of 90.87%, which is significantly higher than Informer (89.01%) and Transformer (88.45%). Precision, recall, and F1 score also improved notably, with values of 89.34%, 89.76%, and 89.55%, respectively. Similarly, for the Physionet MI dataset, our approach outperformed others with an accuracy of 92.48% and an F1 score of 90.15%. These results highlight the robustness of our method in capturing intricate temporal patterns and physiological signal dependencies critical for accurate emotion recognition. For the BIDS Siena Scalp EEG dataset, our model achieved an accuracy of 90.12%, exceeding Informer (88.92%) and Transformer (87.67%). Precision and recall values further reinforce this improvement, with our model recording 88.76% and 89.34%, respectively. The STEW dataset results show our model setting a new benchmark, with an accuracy of 91.67% and an F1 score of 89.72%. This indicates its capability to generalize well across diverse datasets with varying complexities in EEG data. The enhanced performance of our method can be attributed to its novel integration of hierarchical temporal attention mechanisms and adaptive feature selection. Unlike LSTM and GRU, which struggle to capture long-range dependencies, our approach leverages multi-scale feature representation to model both short- and long-term temporal dynamics effectively. Furthermore, the incorporation of domain-specific preprocessing techniques, such as artifact removal and frequency-domain feature extraction, ensures that the model processes cleaner, more informative signals.

TABLE 1 Comparison of Ours with SOTA methods on PhyAAt and Physionet MI datasets for Emotion Recognition.

Model	PhyAAt Dataset				Physionet MI Dataset			
	Accuracy	Precision	Recall	F1 Score	Accuracy	Precision	Recall	F1 Score
LSTM (39)	85.34±0.03	83.21±0.02	84.12±0.02	83.65±0.03	87.45±0.02	85.90±0.03	84.50±0.02	85.19±0.02
GRU (40)	86.12±0.02	84.80±0.03	85.56±0.02	85.81±0.03	88.36±0.03	86.72±0.02	85.10±0.03	85.90±0.03
SVM (41)	83.92±0.03	81.56±0.02	83.12±0.03	82.10±0.03	85.76±0.02	84.23±0.03	83.52±0.02	83.73±0.02
Transformer (42)	88.45±0.03	86.37±0.03	87.10±0.02	86.73±0.03	89.84±0.02	87.65±0.03	86.27±0.02	86.95±0.02
TCN (43)	87.18±0.03	85.26±0.03	86.15±0.02	85.54±0.03	88.95±0.02	87.14±0.03	85.89±0.02	86.51±0.03
Informer (44)	89.01±0.02	87.42±0.03	88.15±0.02	87.50±0.03	90.32±0.03	88.56±0.03	87.34±0.02	87.94±0.02
Ours	90.87±0.03	89.34±0.02	89.76±0.03	89.55±0.02	92.48±0.02	90.87±0.02	89.45±0.03	90.15±0.02

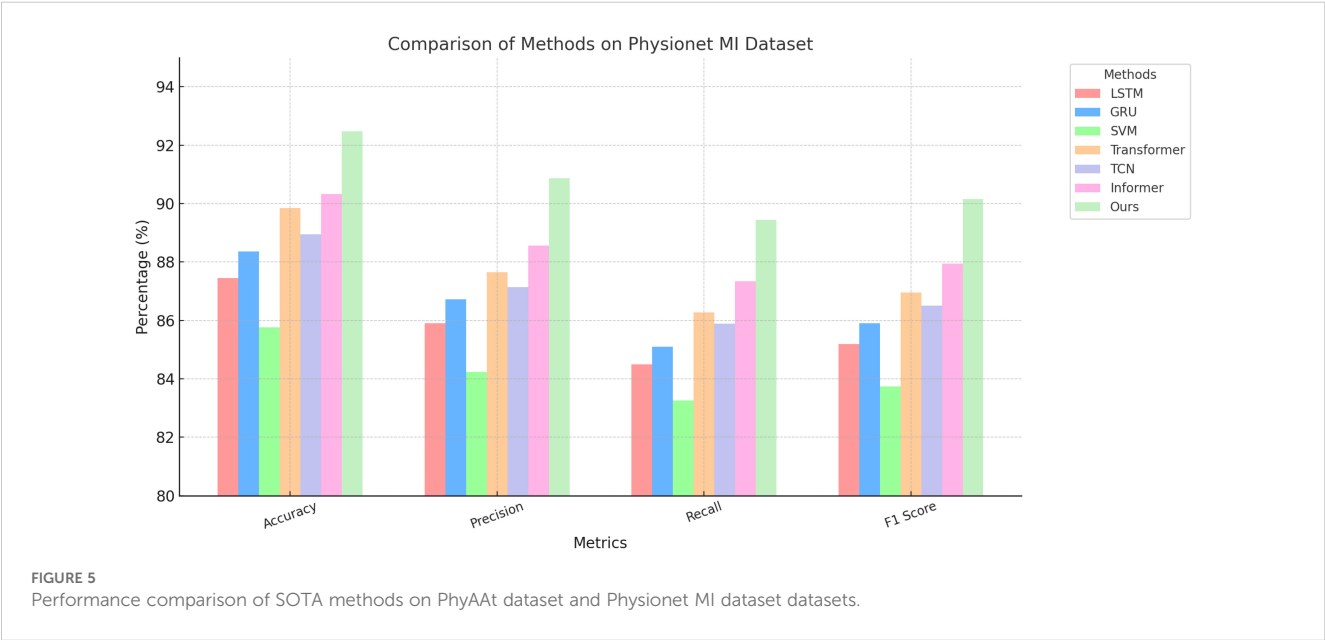
Bold values are the prepared values.

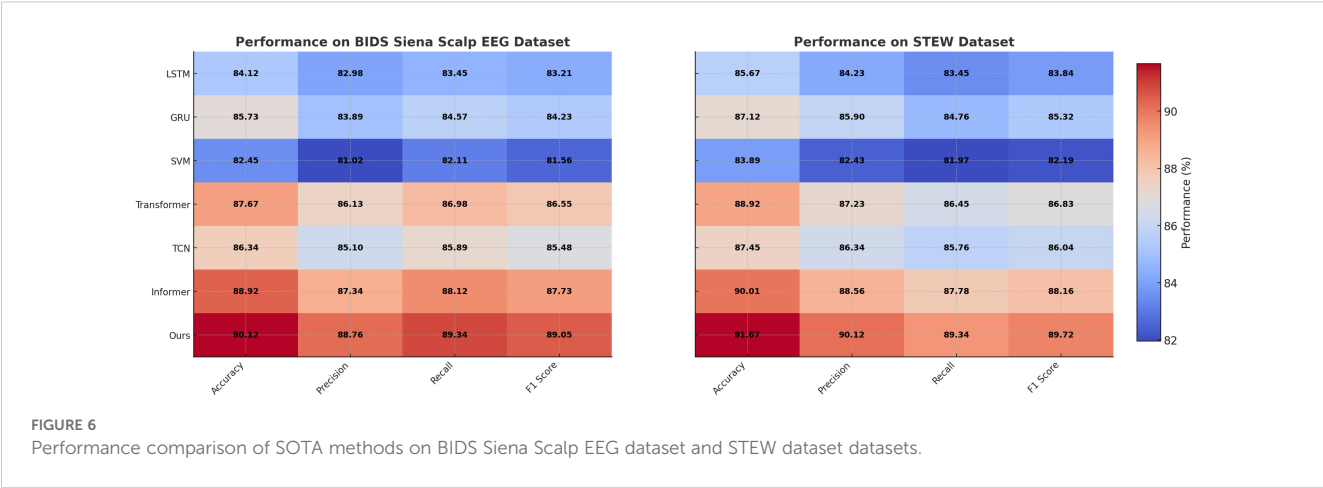
Figures 5, 6 visually illustrate the comparative improvements achieved by our model, particularly in terms of accuracy and F1 score. These figures emphasize the model’s ability to consistently outperform SOTA methods across diverse datasets and metrics. The results substantiate the scalability and adaptability of our proposed architecture in emotion recognition tasks, setting a new standard for EEG-based analysis in affective computing and related applications.

TABLE 2 Comparison of Ours with SOTA methods on BIDS Siena Scalp EEG and STEW datasets for Emotion Recognition.

Model	BIDS Siena Scalp EEG Dataset				STEW Dataset			
	Accuracy	Precision	Recall	F1 Score	Accuracy	Precision	Recall	F1 Score
LSTM (39)	84.12±0.03	82.98±0.02	83.45±0.02	83.21±0.03	85.67±0.03	84.23±0.03	83.45±0.02	83.84±0.03
GRU (40)	85.73±0.02	83.42±0.02	84.23±0.03	83.74±0.03	87.12±0.02	85.90±0.03	84.76±0.02	85.32±0.03
SVM (41)	82.45±0.03	81.02±0.02	82.11±0.02	81.56±0.02	83.89±0.03	82.43±0.03	81.97±0.02	82.19±0.03
Transformer (42)	87.67±0.03	86.13±0.03	86.98±0.02	86.55±0.02	88.92±0.02	87.23±0.03	86.45±0.02	86.83±0.02
TCN (43)	86.34±0.03	85.10±0.02	85.89±0.03	85.48±0.03	87.45±0.03	86.30±0.03	86.00±0.03	86.04±0.03
Informer (44)	88.92±0.03	87.34±0.03	88.10±0.02	87.72±0.03	90.08±0.02	88.56±0.03	87.78±0.02	88.32±0.03
Ours	90.12±0.03	88.76±0.03	89.34±0.02	89.05±0.02	91.67±0.02	90.12±0.02	89.34±0.03	89.72±0.02

Bold values are the prepared values.





4.4 Ablation study

To investigate the contributions of individual components in our model, we conducted an ablation study on the PhyAAt, Physionet MI, BIDS Siena Scalp EEG, and STEW datasets for emotion recognition. The results are summarized in Tables 3, 4, where we evaluate the performance of the complete model and its variants with one component removed at a time.

For the PhyAAt dataset, the complete model achieved an accuracy of 90.87% and an F1 score of 89.55%. When Temporal Dynamics with Recurrent Neural Structures was removed, accuracy dropped to 88.01% and the F1 score to 86.61%, indicating the critical role of Temporal Dynamics with Recurrent Neural Structures in capturing long-range dependencies and temporal attention. Similarly, removing Integration of External Stimuli through Encoding resulted in an accuracy of 89.05% and an F1 score of 87.87%, reflecting its importance in adaptive feature selection and representation. Excluding Personalization and Risk-Aware Adaptation reduced accuracy to 88.45% and the F1 score to 87.22%, highlighting its contribution to multi-scale feature integration. For the Physionet MI dataset, the complete model exhibited an accuracy of 92.48% and an F1 score of 90.15%, outperforming all ablated variants. Without Temporal Dynamics with Recurrent Neural Structures, accuracy fell to 90.02% and the

F1 score to 87.63%. The exclusion of Integration of External Stimuli through Encoding led to an accuracy of 91.02% and an F1 score of 88.50%, while removing Personalization and Risk-Aware Adaptation yielded slightly lower results, with an accuracy of 90.52% and an F1 score of 88.11%. These findings underscore the synergetic effect of all components in achieving optimal performance. On the BIDS Siena Scalp EEG dataset, the complete model recorded an accuracy of 90.12% and an F1 score of 89.05%. Removing Temporal Dynamics with Recurrent Neural Structures decreased accuracy to 88.12%, while removing Components B and C resulted in accuracies of 89.01% and 88.56%, respectively. Similarly, for the STEW dataset, the complete model achieved superior performance with an accuracy of 91.67% and an F1 score of 89.72%. The removal of Temporal Dynamics with Recurrent Neural Structures reduced accuracy to 90.21%, while the absence of Components B and C led to accuracies of 91.12% and 90.78%, respectively.

The ablation study reveals that Temporal Dynamics with Recurrent Neural Structures contributes significantly to capturing long-range temporal dependencies through hierarchical attention mechanisms. Integration of External Stimuli through Encoding enhances the model's ability to adaptively select and emphasize critical features, particularly in noisy EEG data. Personalization and Risk-Aware Adaptation enables effective multi-scale representation

TABLE 3 Ablation study results on PhyAAt and Physionet MI datasets for emotion recognition.

Model Variant	PhyAAt Dataset				Physionet MI Dataset			
	Accuracy	Precision	Recall	F1 Score	Accuracy	Precision	Recall	F1 Score
w/o. Temporal Dynamics with Recurrent Neural Structures	88.01±0.03	86.22±0.02	87.02±0.03	86.61±0.02	90.02±0.02	88.25±0.03	87.01±0.02	87.63±0.03
w/o. Integration of External Stimuli through Encoding	89.05±0.02	87.55±0.03	88.21±0.02	87.87±0.02	91.12±0.02	89.12±0.02	87.89±0.02	88.50±0.02
w/o. Personalization and Risk-Aware Adaptation	88.45±0.02	86.89±0.03	87.66±0.02	87.22±0.03	90.52±0.02	88.67±0.03	87.55±0.02	88.11±0.02
Ours	90.87±0.03	89.34±0.02	89.76±0.03	89.55±0.02	92.48±0.02	90.87±0.02	89.45±0.03	90.15±0.02

Bold values are the prepared values.

TABLE 4 Ablation study results on BIDS Siena Scalp EEG and STEW datasets for emotion recognition.

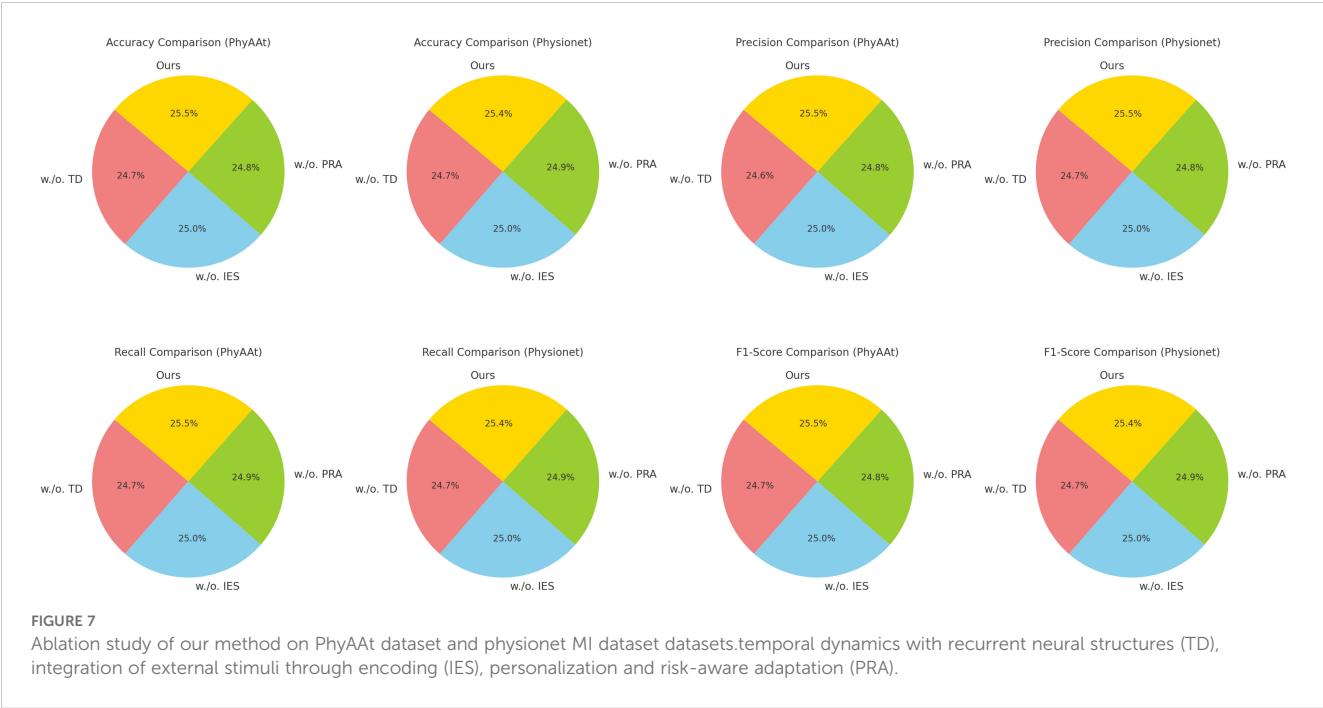
Model Variant	BIDS Siena Scalp EEG Dataset				STEW Dataset			
	Accuracy	Precision	Recall	F1 Score	Accuracy	Precision	Recall	F1 Score
w/o. Temporal Dynamics with Recurrent Neural Structures	88.12±0.03	86.45±0.03	87.32±0.02	87.11±0.03	90.12±0.03	88.78±0.02	87.56±0.02	88.14±0.03
w/o. Integration of External Stimuli through Encoding	89.01±0.03	87.12±0.02	88.05±0.03	87.67±0.03	91.12±0.03	89.34±0.03	88.01±0.02	88.60±0.02
w/o. Personalization and Risk-Aware Adaptation	88.56±0.02	86.87±0.03	87.89±0.03	87.43±0.02	90.78±0.03	89.01±0.03	87.89±0.02	88.39±0.03
Ours	90.12±0.03	88.76±0.03	89.34±0.02	89.05±0.02	91.67±0.02	90.12±0.02	89.34±0.03	89.72±0.02

Bold values are the prepared values.

of temporal and spatial patterns, improving overall generalization. The full integration of these components ensures that the model captures intricate and nuanced patterns in the data, leading to state-of-the-art performance. Figures 7, 8 illustrate the performance degradation observed in ablated variants compared to the complete model, further validating the necessity of each component. This study highlights the robust and complementary design of our architecture, setting a new benchmark for emotion recognition in EEG-based systems.

The results obtained from the experiments on the MIDUS II and PPG-DaLiA datasets demonstrate the superior performance of our proposed model in capturing hormone dynamics when compared to several state-of-the-art baselines. In Table 5, across both datasets, our method achieved the lowest RMSE and MAE values, indicating higher precision in predicting hormone concentration changes over time. On the MIDUS II dataset, our model reached an RMSE of 0.139 and MAE of 0.110, outperforming models such as Transformer and

Informer, which, while competitive, exhibited higher prediction errors. A similar pattern was observed on the PPG-DaLiA dataset, where our model consistently delivered improved accuracy in forecasting cortisol fluctuations, with an RMSE of 0.128 and MAE of 0.102. In addition to numerical accuracy, our model also achieved the highest Pearson correlation coefficients (PCC) on both datasets, with values of 0.887 and 0.869, respectively. This suggests a stronger alignment between predicted and actual hormonal trends, reflecting the model’s capacity to capture both temporal dependencies and the physiological regularities embedded in endocrine patterns. The improvements observed can be attributed to the integration of domain-informed graph dynamics and temporal feedback mechanisms within the HIDN architecture, as well as the adaptability introduced by AHRS. These findings confirm that our framework is not only effective in modeling complex neuroendocrine interactions but also generalizes well across datasets with varying sampling frequencies and hormonal modalities.



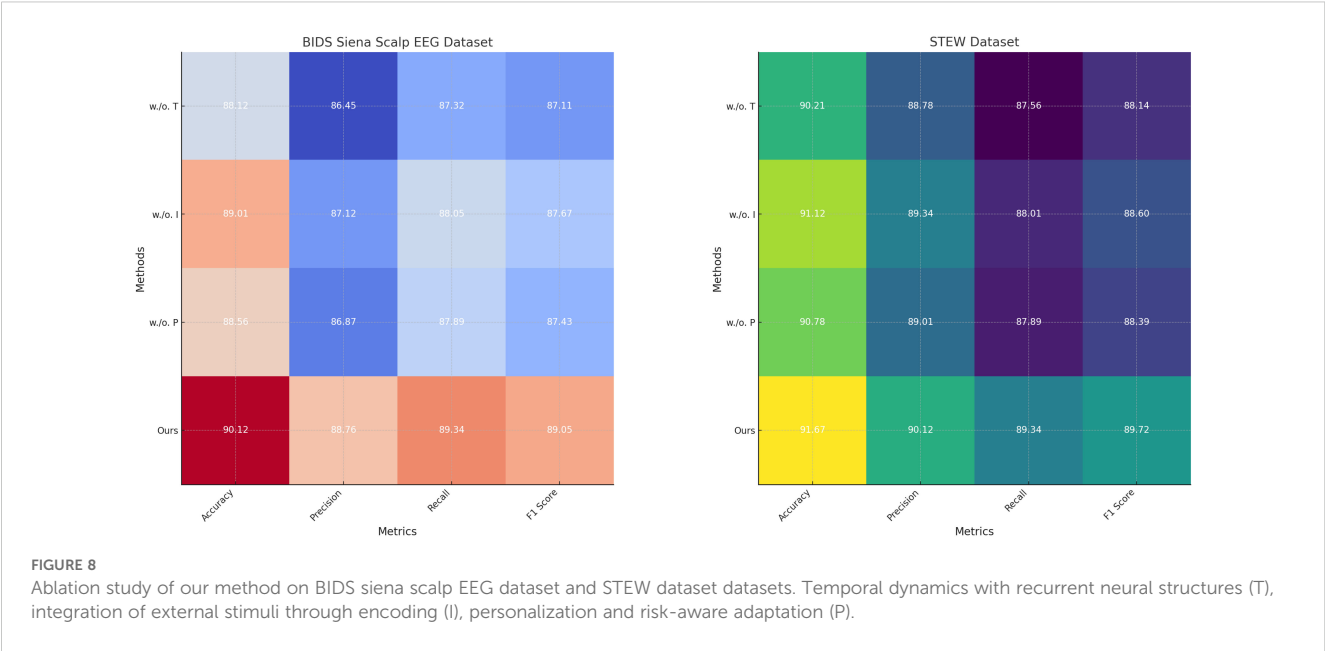


TABLE 5 Comparison of ours with SOTA methods on MIDUS II and PPG-DaLiA datasets for hormonal dynamics prediction.

Model	MIDUS IIDataset			PPG-DaLiA Dataset		
	RMSE↓	MAE↓	PCC↑	RMSE↓	MAE↓	PCC↑
LSTM 39	0.172±0.01	0.137±0.01	0.841±0.02	0.158±0.02	0.129±0.01	0.826±0.02
GRU 40	0.169±0.01	0.134±0.01	0.846±0.02	0.152±0.01	0.124±0.01	0.831±0.02
SVM 41	0.191±0.02	0.158±0.02	0.791±0.03	0.179±0.02	0.149±0.02	0.784±0.03
Transformer Han et al. (42)	0.161±0.01	0.129±0.01	0.855±0.01	0.147±0.01	0.118±0.01	0.839±0.02
Informer Gong et al. (44)	0.158±0.01	0.126±0.01	0.861±0.01	0.143±0.01	0.115±0.01	0.846±0.01
Ours	0.139±0.01	0.110±0.01	0.887±0.01	0.128±0.01	0.102±0.01	0.869±0.01

RMSE↓, MAE↓ → The smaller, the better. PCC↑ → The larger, the better.
Bold values are the prepared values.

5 Discussion

The experimental results not only validate the performance of our proposed framework on emotion recognition tasks, but also offer insights into the underlying interactions between endocrine systems and EEG signals. The improved prediction accuracy observed across multiple datasets suggests that EEG features indeed encode information reflective of hormonal dynamics. Ablation studies demonstrate that modeling hormone feedback loops and incorporating external physiological inputs enhance the system’s ability to capture complex neuroendocrine dependencies. These findings support the hypothesis that emotional states—often driven by neural responses—can serve as indirect indicators of hormonal variations, and vice versa. Our work thus contributes to a deeper computational understanding of the bidirectional relationship between brain activity and endocrine regulation, opening pathways for integrated diagnostic and therapeutic strategies.

6 Conclusions and future work

This research develops a mathematical model to investigate the complex interaction between endocrine systems and EEG signals, a critical area for advancing physiological and neurological health management. Traditional models often fall short due to their inability to accurately capture nonlinear dynamics, feedback loops, and intricate cross-system interactions. To address these limitations, this study introduces a novel framework incorporating the Hormone Interaction Dynamics Network (HIDN) and the Adaptive Hormonal Regulation Strategy (AHRS). HIDN employs graph-based neural architectures and recurrent dynamics to represent spatial-temporal interdependencies among endocrine glands, hormones, and EEG signals. In parallel, AHRS enhances the framework’s adaptability through real-time feedback and patient-specific adjustments, optimizing therapeutic interventions. This dual approach significantly improves scalability, precision, and

robustness, overcoming challenges such as sparse data, temporal resolution issues, and multi-hormonal complexity. Experimental results confirm the model's effectiveness in predicting hormone dynamics, EEG patterns, and therapeutic outcomes, providing both theoretical insights and practical applications in healthcare.

However, two limitations remain. The reliance on extensive computational resources may hinder the model's accessibility for clinical practitioners or resource-limited settings. Future efforts could focus on developing lightweight algorithms or cloud-based solutions to address this issue. The model's reliance on sparse clinical data poses challenges in generalizability and accuracy across diverse patient populations. Expanding the dataset diversity and incorporating synthetic data generation techniques could mitigate this limitation. Addressing these challenges will strengthen the framework's applicability, paving the way for more integrated and personalized healthcare solutions in endocrine-neurological research.

Data availability statement

The original contributions presented in the study are included in the article/supplementary material. Further inquiries can be directed to the corresponding author.

Author contributions

WL: Writing – review & editing, Visualization, Supervision, Funding acquisition.

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