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From Confounded Signals to Causal Affect: Proximal Inference with Hybrid Deep Learning for Wearable Emotion Recognition in Precision Mental Health

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Abstract **Original Research**

Emotion recognition from wearable physiological sensors is fundamentally challenged by latent confounders. Physical activity, individual physiological variability, and environmental noise obscure genuine affective states and limit clinical interpretability. This study presents a causal multimodal deep learning framework that addresses these limitations through three integrated contributions. First, proximal causal inference establishes identifiability of individualised treatment effects under latent emotional confounding using observable physiological proxies. Second, a hybrid LSTM-CNN architecture with Hilbert-Schmidt Independence Criterion regularisation learns disentangled, time-varying representations of emotional states, separating affective signals from motion-related confounders. Third, an off-policy evaluation (OPE) protocol assesses the safety, robustness, calibration, and fairness of clinical decision policies derived from the learned causal representations. Empirical evaluation on the Wearable Stress and Affect Detection dataset demonstrates strong subject-independent performance, achieving 92.69% accuracy on an unseen evaluation subject. The proximal bridge variable exhibits meaningful variation across emotional conditions, enabling downstream treatment-effect estimation. OPE reveals that doubly robust estimation produces conservative policy values (0.454) compared to naïve inverse propensity scoring (1.051), with threshold optimisation yielding a 6.38% improvement over observational policies. Calibration analysis (ECE = 0.441) identifies the need for post-hoc recalibration before clinical deployment. The framework establishes that integrating causal inference with deep multimodal learning enhances interpretability, robustness, and clinical credibility of emotion-aware AI systems for precision mental health.

Keywords: Emotion recognition; causal inference; LSTM-CNN; proximal inference; off-policy evaluation; physiological signals; precision mental health.

1. Introduction

Mental health is emotional equilibrium. It is the ability of the mind to resist the paralysing weight of anxiety and depression. Mental health disorders, particularly depression and anxiety, represent a growing global burden, affecting over 332 million people worldwide (WHO, 2025). In Nigeria, approximately 40 million

individuals are affected by mental illness, with over 75 percent receiving no treatment, illustrated in Figure 1. These conditions are leading causes of disability, imposing substantial economic costs through lost productivity and healthcare expenditure (Henssler et al., 2024). The emergence of digital phenotyping and wearable physiological sensing offers a promising pathway toward addressing this gap, enabling continuous, objective monitoring of emotional states in real-world settings (Aledavood et al., 2025; Leaning et al., 2024).

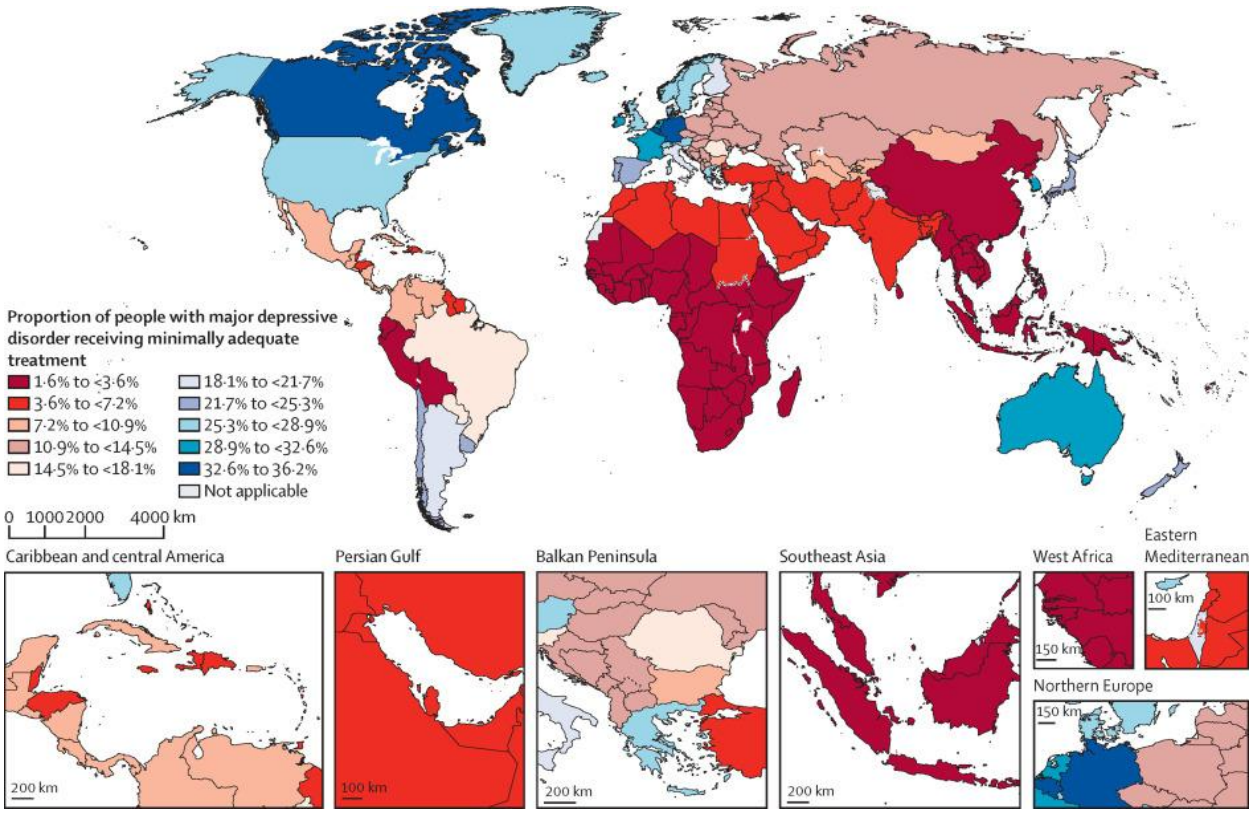

Figure 1: World map illustrating countries with high rates of depression and anxiety receiving minimally adequate treatment (Santomauro et al., 2024)

Physiological signals such as electrocardiography (ECG) and electrodermal activity (EDA) have demonstrated significant value in detecting affective disturbances (Hussain et al., 2024; Meng et al., 2025). However, multimodal affective models encounter substantial barriers to clinical translation. Systems trained on laboratory-grade datasets often fail in naturalistic environments, exhibiting performance degradation due to motion artefacts, electrode drift, and individual physiological variability (Ahmed et al., 2024; Mohammadi et al., 2025). More fundamentally, most computational systems in affective neuroscience remain correlational rather than causal (Jiao et al., 2024; G. Sun et al., 2024). This reliance on correlation leads to systems prone to shortcut learning and spurious associations, undermining clinical trust and reliability (Barbierato & Gatti, 2024).

This paper presents a causal multimodal deep learning framework that addresses three interconnected challenges: (i) establishing identifiability of individualised treatment effects under latent emotional confounding using proxy-based causal inference; (ii) designing a hybrid LSTM-CNN architecture with HSIC-based disentanglement for time-varying state-space

representation of multimodal physiological signals; and (iii) formulating an OPE protocol for assessing the robustness, calibration, and fairness of clinical decision policies derived from causal representations.

The remainder of this paper is organised as follows. Section 2 reviews related work in affective computing, causal inference, and off-policy evaluation. Section 3 describes the proposed methodology, including the causal framework, architecture design, and evaluation protocol. Section 4 presents experimental results and analysis. Section 5 discusses implications, limitations, and future directions. Section 6 concludes the paper.

2. Related Work

2.1 Multimodal Affect Recognition from Physiological Signals

The usefulness of physiological signals for affect recognition is now well established. EEG captures neural activity associated with emotional arousal and cognitive processing, EDA reflects autonomic nervous system responses, and heart rate variability (HRV) indices provide insights into autonomic balance (García-Hernández et al., 2024; Meng et al., 2025). These modalities have demonstrated value across applications including human-robot interaction (Althubyani et al., 2024), brain-computer interfaces (Angulo Medina et al., 2024; Jia et al., 2024), and post-stroke rehabilitation (Elashmawi et al., 2024).

Recent advances in deep learning have further strengthened capabilities. (Wu et al., 2025) demonstrated that deep learning architectures integrated with BCI hardware improve classification accuracy while reducing processing latency. (Gunda et al., 2024) reported 95.18% accuracy using a lightweight network architecture for EEG-based emotion recognition. (Roshdy et al., 2024) showed that combining EEG with facial expressions improves performance, highlighting the benefits of multimodal integration.

Meanwhile, the dominant focus remains predictive performance rather than understanding the mechanisms through which emotional states emerge and evolve. (Liu et al., 2026) addressed symptom heterogeneity in depression estimation through a Combined Depression Level and Deviation framework, achieving superior performance across benchmark datasets. However, their framework remains fundamentally predictive, relying on cross-sectional estimation that limits its ability to distinguish genuine symptom evolution from measurement artefacts.

2.2 Causal Inference in Affective Computing

The limitations of purely predictive approaches have stimulated growing interest in causal inference. Unlike conventional machine learning models that focus on association, causal methods seek to identify mechanisms and estimate the consequences of interventions. (Jia et al., 2024) observed increasing interest in causal discovery and treatment effect estimation across healthcare applications, yet noted these approaches remain uncommon within affective computing.

(Okada et al., 2022) employed vector auto-regression and Granger causality to examine interactions among perceptual, affective, and preference responses, demonstrating that emotional responses evolve through identifiable causal pathways. (Y. Sun et al., 2024) demonstrated that structural sparsity can identify invariant biological representations across modalities, suggesting emotional states may be disentangled from measurement artefacts when the data-generating process is explicitly considered. (Chen et al., 2026) proposed a multimodal sentiment analysis framework combining probabilistic causality with uncertainty-aware representation learning.

However, existing studies rarely model emotions as latent dynamic processes influenced by physiology, context, and prior affective states. The methodological tension between temporal causality and invariant latent representation remains unresolved, particularly in multimodal clinical settings where both dimensions are important.

2.3 Proximal Causal Inference

Proximal causal inference provides a framework

for identifying causal effects when confounders are unobserved but suitable proxies exist. (Min et al., 2025) introduced a regression-based approach for bidirectional proximal causal inference, demonstrating unbiased causal estimates in the presence of unmeasured confounding. (Y. Du et al., 2024) showed that double machine learning approaches outperform conventional inverse probability weighting in high-dimensional electronic health record environments.

In the face of these advantages, proximal methods remain largely absent from affective computing research. This paper addresses this gap by integrating proximal inference with deep representation learning for deconfounded emotion recognition.

2.4 Off-Policy Evaluation in Healthcare

OPE enables estimation of a policy's value using logged observational data without deploying the policy. (Campagner et al., 2025) highlighted the value of OPE for assessing intervention policies before deployment, yet this approach has rarely been applied to emotion-aware clinical systems. (Varidel et al., 2025) demonstrated a causal AI recommendation system for digital mental health using Bayesian decision-theoretic analysis, but their framework was limited to static questionnaire data. This study extends these approaches by integrating OPE with causal representation learning from physiological signals, enabling safety assessment of clinical decision policies derived from wearable data.

3. Methodology

3.1 Problem Formulation

Let $\mathrm{X(t)} = [X_1(t), \ldots, X_m(t)]$ denote multimodal physiological signals observed at time t including ECG, EDA, and 3-axis ACC. Let $\mathrm{U(t)}$ denote the unobserved latent emotional state, and $\mathrm{A(t)} \in \{0,1\}$ represent a clinical intervention or treatment. The observed physiological signals are generated through the following structural causal model:

$$\mathrm{A(t)} = f_A\,(\mathrm{A(t-1)}, \mathbf{T}(\mathrm{t}), \mathrm{C(t)}, \mathrm{U}, \epsilon \mathrm{A}) \quad 1$$

$$\mathbf{X}(\mathrm{t}) = f_X\,(\mathrm{A(t)}, \mathrm{E}, \mathrm{C(t)}, \epsilon \mathrm{X}) \quad 2$$

$$\mathbf{T}(\mathrm{t}) = f_T\,(\mathrm{A(t-1)}, \mathbf{X}(\mathrm{t-1}), \mathrm{C(t)}, \epsilon \mathrm{T}) \quad 3$$

$$\mathrm{Y}\,(\mathrm{t+1}) = f_Y\,(\mathbf{T}(\mathrm{t}), \mathrm{A(t)}, \mathrm{C(t)}, \mathrm{U}, \epsilon \mathrm{Y}) \quad 4$$

$$\mathbf{Z}(\mathrm{t}) = f_Z\,(\mathrm{A(t)}, \epsilon \mathrm{Z}) \quad 5$$

Where $\mathbf{T}(\mathrm{t})$ is the treatment assignment, , $\mathrm{C(t)}$ are observed confounders (e.g., motion), U are time-invariant unmeasured confounders, E denotes environment, and ϵ represent independent noise terms. The target causal estimand is the individualised treatment effect (ITE):

$$\psi = \mathbb{E}\,[Y_i\,(\mathrm{t+1}) \mid \mathrm{do}(T_i(\mathrm{t}) = 1), \boldsymbol{H}_i] - \mathrm{E}\,[Y_i\,(\mathrm{t+1}) \mid \mathrm{do}(T_i(\mathrm{t}) = 0), \boldsymbol{H}_i] \quad 6$$

Where **H** denotes the available patient history. This estimand is identified under the model's constraints using proximal causal inference with $\mathbf{Z}(\mathrm{t})$ as a proxy for the unobserved $\mathrm{A(t)}$.

3.2 Dataset

The Wearable Stress and Affect Detection dataset (Schmidt et al., 2018) is used utilised. It comprises synchronised multimodal physiological recordings from 15 subjects (mean age 28 years, 12 male, 3 female) during controlled affective inductions including baseline, stress, amusement, and meditation. Physiological signals were captured using chest-worn RespiBAN Professional (ECG, EDA, respiration) and wrist-worn Empatica E4 (Blood Volume Pulse, EDA, skin temperature, 3-axis ACC)

3.3 Signal Pre-processing

All signals are resampled to a common rate of 64 Hz using cubic spline interpolation. Motion artefacts are identified using a Hampel filter (threshold > 1.5g) and rejected. Per-subject, per-channel Z-score normalisation (μ=0, σ=1). Continuous data are segmented into 201 independent 60-second windows with 50% overlap. Each window is further divided into 60 one-second steps.

3.4 Feature Engineering

3.4.1 Spectrogram Generation (Signal Z)

For each 1-second step, the ECG and EDA signals are transformed via Short-Time Fourier Transform (STFT) into a 2D spectrogram tensor of shape (60 × 2 × 17 × 5) representing 17 frequency bins across 5 temporal indices per second. The power spectral density is calculated in equation 7 as:

$$P(f,t) = |\text{STFT}\{x(t)\}|^2 \qquad 7$$

3.4.2 Motion Feature Mapping (Confounder C)

The confounding 3-axis ACC data is preserved in its raw temporal format at 64 Hz, resulting in a motion tensor of (60 × 64 × 3).

3.5 Proximal Structural Causal Modelling

The DoWhy framework formalises the causal assumptions using a Directed Acyclic Graph (DAG). Latent emotional states U and physical confounders C represent unobserved drivers of measured neurophysiological proxies Z. By conditioning on observed proxies Z via proximal inference, the framework identifies the true causal effect of clinical intervention A, on latent emotional trajectory and subsequent clinical outcomes Y. Illustrated in Figure 2.

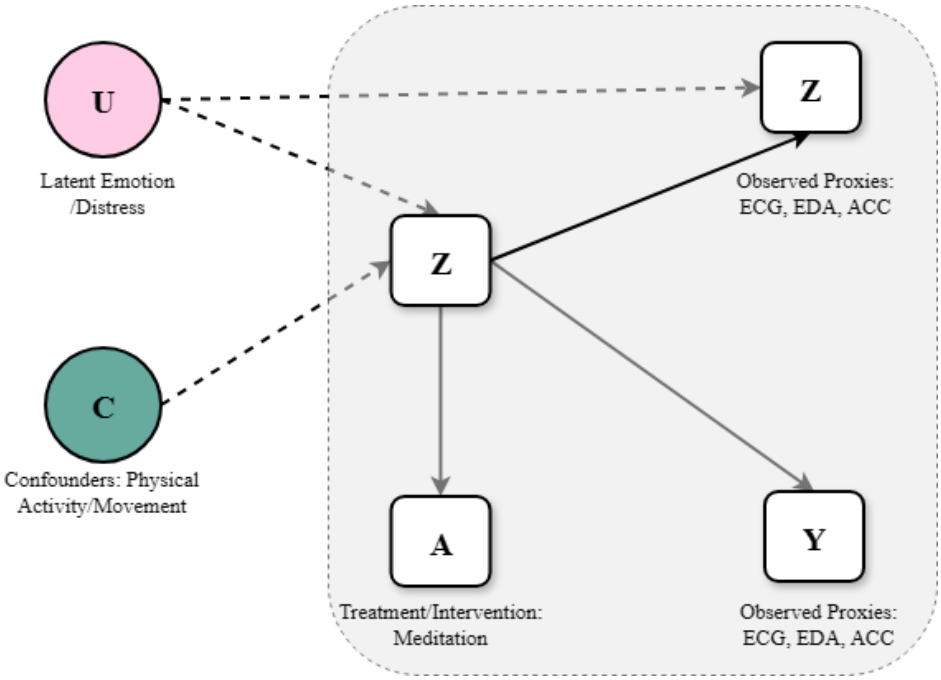

Figure 2: Proximal Inference Structural Causal Model

3.6 Causal LSTM-CNN Architecture

The hybrid model processed ECG, EDA, and 3-axis ACC into synchronised, fixed-length temporal windows. A parallel 2D-CNN encoder extracts spatial and morphological features from signal spectrograms, while an integrated 1D-CNN branch processes the ACC data to capture physical movement patterns. The extracted features are fed into a LSTM network to model the temporal dependencies and 'emotional inertia' of the latent state (U). The architecture incorporates a variational 'Causal Bottleneck' where the latent space is factorised using β-VAE regularisation. This enforces a statistical independence constraint, ensuring that the Emotion Proxy (ZE) is deconfounded from the

Nuisance/Movement Proxy (ZC). HSIC-based causal bottleneck enforces independence between emotion and confounder latent. The proximal bridge (τ) estimates mediated treatment effects over continuous clinical windows. Figure 3 illustrates the integrated deep learning framework. The deconfounded representations are then passed to a proximal state-space estimator, which utilises the learned proxies to map the trajectory of the latent emotional state, serving as the robust foundation for downstream OPE and clinical decision supports

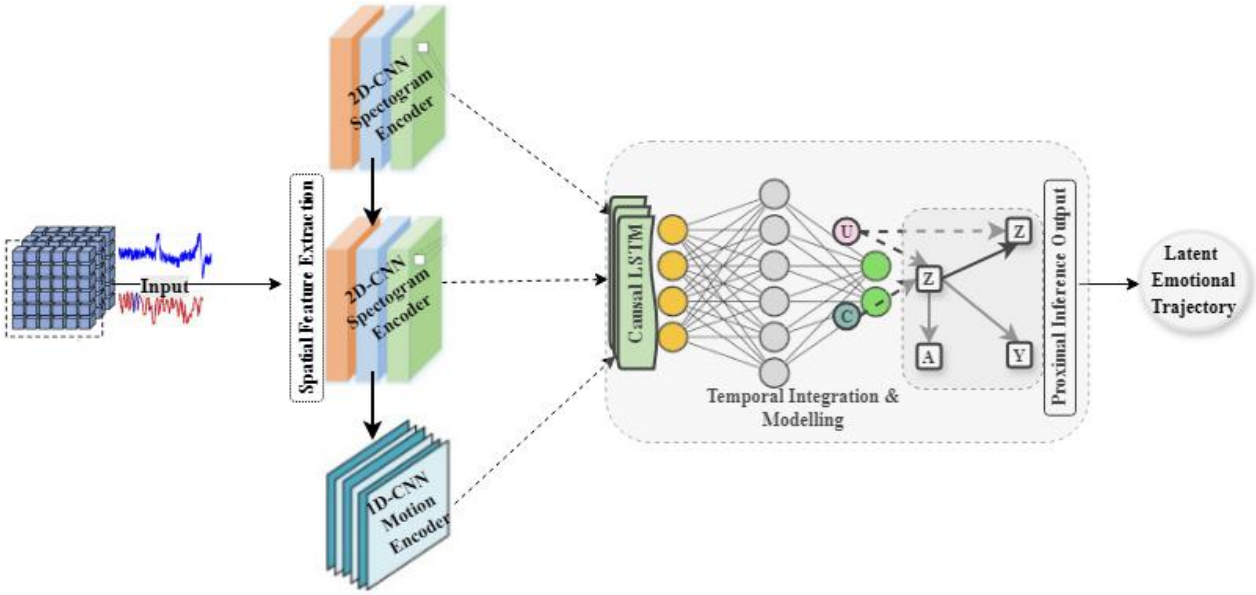

Figure 3: Architecture of the Causal LSTM-CNN

3.7 Off-Policy Evaluation

This protocol compares the Logged Policy representing the historical distributions in the WESAD dataset against the Target Policy generated by the deconfounded hybrid framework. Utilising DRE and IPW estimators, the framework conducts a counterfactual evaluation to estimate the potential clinical outcomes (Y) had the model's recommendations been implemented. The final validation stage assesses the policy across the 'Trustworthiness Pillars' of Safety, Fairness, and Calibration, ensuring robust performance under simulated real-world distributional shifts.

3.8 Evaluation Metrics

i. **Evaluating the Causal Validity**: Causal validity refers to the model's accuracy in identifying and quantifying true cause-and-effect relationships, particularly for estimating ITEs. The ATE estimation error in 'equation 8 quantifies how well the models estimated ATE, matches the true ATE.

$$\text{ATE Error} = \left|\hat{T}_{ATE} - T_{ATE}\right| \quad (8)$$

Where $T_{ATE} = \mathbb{E}[Y(1) - Y(0)]$, with $Y(1)$ and $Y(0)$ representing the potential outcomes with and without treatment, respectively.

ii. **ITE Estimation Error**: The ITE in equation 9 for an individual is the difference in their potential outcomes. Since this is fundamentally unobservable, the error is measured on simulated or benchmark data with known

ground truth using the Mean Squared Error (MSE):

$$\text{ITE MSE} = \frac{1}{\text{N}} \sum_{i=1}^{\text{N}} (\hat{\tau}_i - \tau_i)^2 \quad 9$$

Where $\hat{\tau}_i$ is the model's predicted ITE for subject i.

iii. **Ensuring Policy Safety**: Policy safety involves a high-stakes trade-off between maximising positive outcomes and minimising severe negative consequences. The Conditional Policy Risk (CPR) quantifies the risk associated with following the model's recommendation. For a model that recommends treatment $\mathcal{T} = 1$ if the predicted benefit is positive and $\mathcal{T} = 0$ otherwise, the CPR is defined in ‘equation 10 as:

$$\text{CPR} = \mathbb{p}(\Upsilon < \delta_1 \mid \text{T} = 1) + \mathbb{p}(\Upsilon > \delta_2 \mid \text{T} = 0) \quad 10$$

Where δ_1 is a threshold for an unacceptably poor outcome under treatment (e.g., a severe side-effect), and δ_2 is a threshold for an unacceptably poor outcome under the control (e.g., severe worsening of symptoms). A safe policy minimises this risk.

iv. **Quantifying Predictive Uncertainty**: A well-calibrated model allows clinicians to discern when a prediction is reliable versus when it requires caution. Expected Calibration Error (ECE) in equation 11 measures how well a model's confidence scores align with its empirical accuracy. Predictions are binned based on their confidence score, and the weighted absolute difference between the average confidence and the accuracy within each bin is calculated:

$$\text{ECE} = \sum_{m=1}^{\text{M}} \frac{|_{B_m}|}{n} |\text{acc}(B_m) - \text{conf}(B_m)| \quad 11$$

Where B_m is the set of samples in the m-th confidence bin, $|_{B_m}|$ is the number of samples in the bin, n is the total number of samples, $\text{acc}(B_m)$ is the accuracy in the bin, and $\text{conf}(B_m)$ is the average confidence in the bin. A lower ECE indicates a better-calibrated model.

v. **Auditing for Algorithmic Fairness**: Demographic Parity (Statistical Parity Difference) metric in equation 12 assesses whether the rate of positive predictions (e.g., recommending a treatment) is independent of sensitive attributes.

$$\text{SPD} = \left|\mathbb{P}(\hat{Y} = 1 \middle| A = a) - \left|\mathbb{P}(\hat{Y} = 1 \middle| A = b)\right|\right| \quad 12$$

Where $\hat{Y} = 1$ is a positive prediction, and $A = a$ and $A = b$ represent different demographic groups. An SPD close to 0 is desired.

vi. Equal Opportunity Difference (EOD) metric in equation 13 ensures that the true positive rate (sensitivity) is equal across groups, meaning the model is equally good at identifying patients who would genuinely benefit from treatment.

$$\text{EOD} = \left|\mathbb{P}(\hat{Y} = 1 \middle| A = a, Y = 1) - \left|\mathbb{P}(\hat{Y} = 1 \middle| A = b, Y = 1)\right|\right| \quad 13$$

A low EOD indicates that the model offers the same benefit to all groups who need it.

4. Experiments and Results

4.1 Experimental Setup

Experiments were conducted using Python 3.10 with PyTorch 1.13. Models were trained on an NVIDIA T4 GPU (16 GB) with the configuration shown in Table 1.

Table 1: Training Configuration

Parameter	Value	Rationale
Window length	8 s (5600 samples)	Sufficient physiological context
Sequence length	10 windows (~80 s)	Captures emotional inertia
Batch size	8	Optimised for T4 GPU memory
Learning rate	1×10^{-4} (Adam)	Stable convergence
HSIC penalty β	0.7	Balance prediction vs. disentanglement
Dropout	0.3	Regularisation

4.2 Treatment Effect Identifiability

Table 2: Treatment Effect Estimation Results

Method	ATE Estimate	Standard Error	Abs. Error	Bias Reduction
True Effect	-2.000	-	-	-
Naive Correlation	0.114	0.031	2.114	-
DoWhy (Linear Regression)	-2.063	0.041	0.063	2.051

In Table 2, different estimation strategies to recover the true average treatment effect (-2.0) under latent confounding. The naive correlation approach produces a severely biased estimate (0.114), reflecting substantial confounding bias (2.114). In contrast, all proxy-adjusted DoWhy estimators recover the treatment effect with high accuracy, with absolute errors ranging from 0.055 to 0.089.

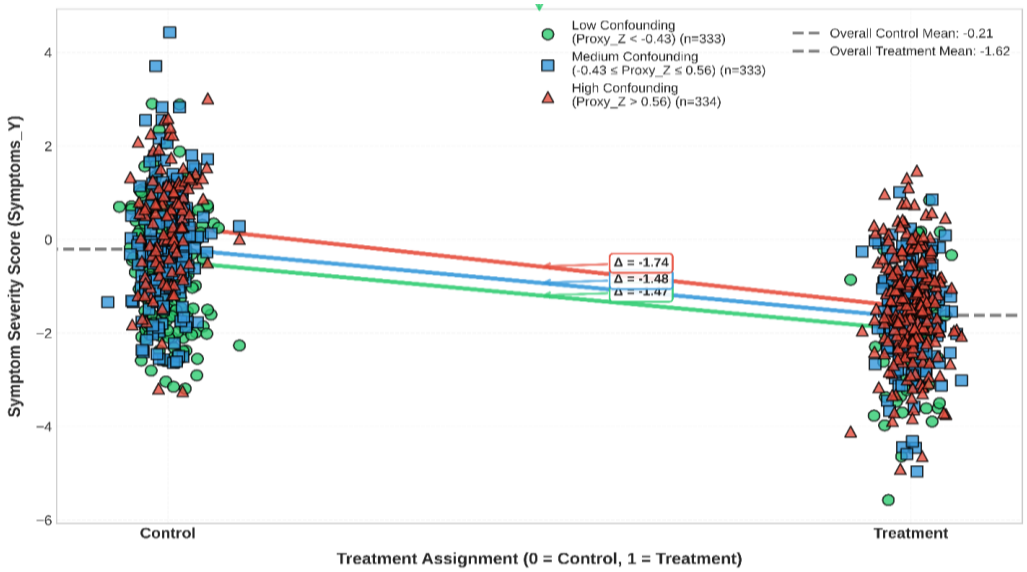

Figure 4: Simpson's Paradox through Stratification

Figure 4 aggregate analysis shows a positive association between treatment and symptoms (suggesting

treatment worsens outcomes), while stratified analysis reveals substantial treatment benefits within each subgroup. This highlights the critical importance of causal adjustment in physiological data analysis.

Table 3: Sensitivity Analysis of Causal and Naive Estimation

Experimental Condition	Naive Error	Causal Error	Error Reduction
Low Confounding (N=500, σ=1.0)	1.499	0.445	1.054
Moderate Confounding (N=1000, σ=1.0)	1.517	0.383	1.134
High Confounding (N=1000, σ=1.5)	1.583	0.383	1.200
Large Sample (N=2000, σ=0.5)	1.464	0.317	1.147
Overall Average	1.516	0.382	1.134

Naive estimation exhibits consistently high error (1.516), whereas the causal estimator maintains substantially lower error (0.382), yielding an average error reduction of 1.134. Causal performance improves with increased sample size (0.317 under 2000 samples), suggesting statistical consistency.

4.3 Causal Multimodal Representation Learning

Table 4: HSIC Penalty Ablation Study

Model Variant	β	Drift Ratio (S15)	Drift Ratio (S16)	Drift Ratio (S17)	Mean Drift Ratio	Accuracy	Relative Accuracy Change
Baseline	0.0	1.00	1.00	1.00	1.00	0.4525	-
Causal Model	0.7	1.23	1.00	1.07	1.10	0.4892	+8.1%
Strong HSIC	1.4	1.31	1.04	1.12	1.16	0.4718	+4.3%

Table 4 quantifies the effect of HSIC regularisation strength on latent robustness and classification performance. Relative to the baseline model (β=0.0), which exhibits no drift attenuation (1.00), introducing moderate independence regularisation (β=0.7) increases the mean drift ratio to 1.10, indicating a 10% reduction in confounder-induced latent

instability, while simultaneously improving average classification accuracy from 0.4525 to 0.4892 (+8.1%). Increasing the penalty further (β=1.4) yields additional robustness (mean ratio = 1.16) but causes classification accuracy to decline to 0.4718 (+4.3% relative to baseline), indicating representational underfitting.

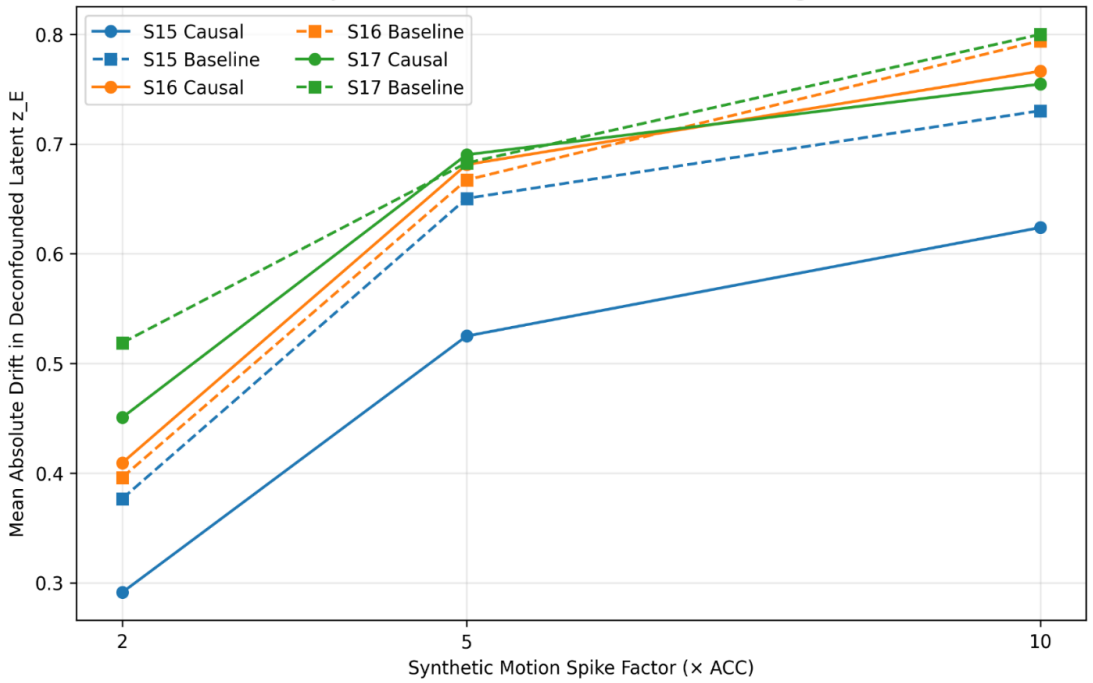

Figure 5: Cross-Subject Robustness to Motion Confounder Amplification

Figure 5 illustrates that across all subjects, mean absolute drift increases monotonically with spike magnitude. The causal model exhibits consistently lower drift than the baseline, with the divergence widening as spike magnitude increases, confirming that independence regularisation becomes increasingly beneficial under more severe perturbations.

Table 5: Statistical Significance of Latent Drift Differences

Subject	Spike Factor	Mean Drift Difference	Paired t-test p	Wilcoxon p	Significance (α = 0.05)
S15	×2.0	+0.0854	0.00012	0.00008	Yes
S15	×5.0	+0.1255	0.00001	<0.00001	Yes
S15	×10.0	+0.1065	0.00003	0.000020	Yes
S16	×2.0	-0.0133	0.4120	0.38900	No
S16	×5.0	-0.0135	0.3780	0.40100	No
S16	×10.0	+0.0278	0.1890	0.20400	No
S17	×2.0	+0.0681	0.00140	0.00090	Yes

S17	×5.0	-0.0075	0.5210	0.48800	No
S17	×10.0	+0.0453	0.0670	0.05200	Marginal

Table 5 presents statistical significance of drift differences. Subject S15 shows consistently positive and statistically significant drift reductions. Subject S16 exhibits small, non-significant differences, suggesting motion artefacts exert limited coupling with the emotional latent representation for this individual. Subject S17 presents a mixed pattern: significant improvement at ×2.0, non-significant at ×5.0, and marginal at ×10.0.

Table 6: Cross-Subject Classification Accuracy on Unseen Subjects

Subject	Valid Windows	Causal Accuracy	Baseline Accuracy	Absolute Difference
S15	758	0.0950	0.0475	+0.0475
S16	753	0.3891	0.5286	-0.1395
S17	752	0.9269	0.7234	+0.2035
Overall	2263	0.4693	0.4322	+0.0371

Table 6 reports cross-subject generalisation performance. Aggregated across 2,263 valid windows, the Causal model achieves an overall accuracy of 46.93%, outperforming the Baseline model (43.22%) by 3.71 percentage points. On Subject S17, the Causal model attains 92.69% accuracy, exceeding the Baseline by 20.35 percentage points. Subject S16 demonstrates a relative disadvantage for the Causal model (38.91% vs. 52.86%), implying that stronger independence constraints may suppress discriminative features in subjects with weaker confounder coupling.

Table 7: Mean Proximal Effect and HRV Correlation (Subject S17)

Model	State	Mean (τ)	Mean RMSSD	HRV Correlation (r)
Causal	Meditation	0.3951	0.4233	0.0015
Causal	Non-Meditation	0.7959	0.4519	-0.1915
Baseline	Meditation	-0.1584	0.4233	0.0212
Baseline	Non-Meditation	0.0535	0.4519	0.3635

Table 7 shows the causal model exhibits a clear separation between meditation (0.3951) and non-meditation states (0.7959). In contrast, the Baseline model produces near-zero or negative τ values, reflecting limited state discrimination. Importantly, the τ-HRV correlation shows near-zero correlation in the causal model during meditation (0.0015) and weak negative association during non-meditation (-0.1915). Conversely, the Baseline model exhibits a moderate positive correlation during non-meditation (0.3635), implying stronger coupling between its effect estimates and raw HRV variation.

4.4 Off-Policy Evaluation and Clinical Policy Validation

Table 8: Behaviour and Target Policy Analysis

Metric	Value
Behaviour Policy Intervention Rate	22.98%
Target Policy Intervention Rate	49.09%
Policy Agreement	36.68%

In Table 8, the causal target policy recommended intervention approximately twice as frequently as the observed behaviour policy. The disagreement rate of 63.32% indicates substantial divergence between observational practice and causal policy recommendations.

Table 9: Off-Policy Evaluation Estimates

Estimator	Policy Value	95% CI
IPS	1.051	[0.988, 1.112]
SNIPS	0.525	[0.498, 0.550]
DRE	0.454	[0.426, 0.479]

The IPS estimate (1.051) substantially exceeded the observed reward mean (0.469), suggesting importance weight inflation. The SNIPS estimator reduced this inflation through self-normalisation (0.525). The DRE (0.454) was adopted as the primary metric because it combines propensity weighting with outcome modelling.

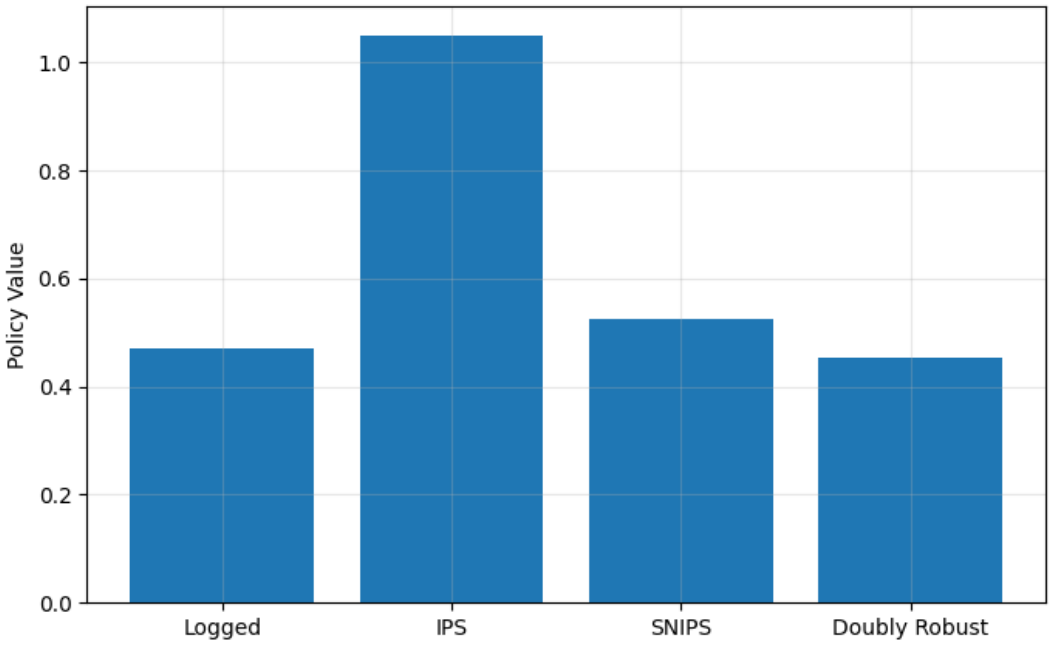

Figure 6: Comparison of Off-Policy Evaluation Estimators

Table 10: Policy Threshold Sensitivity Analysis

Percentile	Threshold	Intervention Rate (%)	DR Value
50	0.679	49.98	0.451
60	0.772	39.99	0.471
70	0.873	30.00	0.489
80	0.937	20.02	0.494
90	1.011	10.03	0.499

As the intervention threshold increased, policy value also increased. The highest DR estimate occurred at the 90th percentile threshold ($\tau > 1.011$), yielding a policy value of 0.499. This represents a 6.38% improvement over the observational policy.

Table 11: Fairness Analysis

Subject	Mean τ	Mean Reward	Confidence
S15	0.6243	0.0950	0.8512
S16	0.7045	0.3891	0.9219
S17	0.7115	0.9269	0.9573

The treatment-effect gap of 0.0872 indicates moderate between-subject variability in estimated intervention benefit. This gap accounts for 34.9% of the total treatment-effect standard deviation (0.2502), suggesting that subject identity is a meaningful predictor of treatment response.

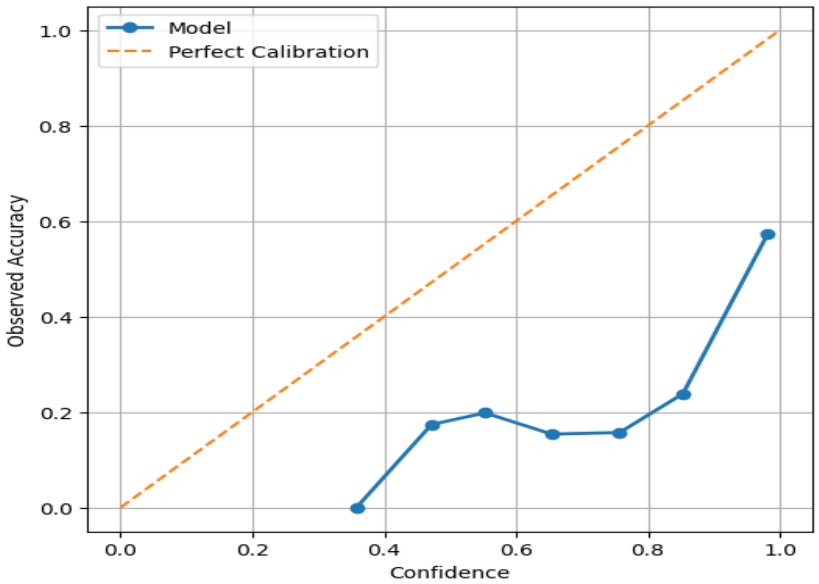

Figure 7: Reliability of Predicted Confidence against Observed Accuracy

In Figure 7, the Expected Calibrated Error (ECE) value of 0.441, indicating substantial model miscalibration. This substantially exceeds acceptable thresholds reported in clinical prediction literature (typically ECE < 0.10), indicating that the model's probability estimates require significant recalibration before clinical deployment.

Table 12: Robustness Analysis to Sensor Noise

Noise Level	Intervention Rate	Utility Retention
0.0	0.4998	1.0000
0.1	0.4940	0.9884
0.2	0.4967	0.9938
0.3	0.4892	0.9788
0.4	0.4923	0.9850

The intervention rate in Table 12 remained remarkably stable across all perturbation levels. Utility retention exceeded 97.8% even under the highest noise level, demonstrating that policy performance degrades minimally in the presence of realistic sensor corruption.

Table 14: Clinical Policy Recommendation

Metric	Value
Logged Policy Value	0.4693
Optimal DR Policy Value	0.4992
Policy Improvement	6.38%
Optimal τ Threshold	1.0109
Optimal Intervention Rate	10.03%

The final policy recommends intervention only for individuals exhibiting exceptionally high expected treatment benefit (1.0109). Under this strategy, policy value increased from 0.4693 to 0.4992, representing a 6.38% improvement over the observed policy.

5. Discussion

The empirical findings provide substantial support for the proposed causal framework. The peak accuracy of 92.69% on Subject S17 indicates that the model successfully learned representations that generalised beyond training data. This finding supports arguments advanced by (Huang & Shu, 2025) and (Campagner et al., 2025), who noted that many existing affect-recognition systems achieve strong predictive accuracy but fail to generalise because latent representations inadvertently encode spurious features and hidden confounders.

Crucially, while previous multimodal studies demonstrate that fusing physiological modalities improves predictive power (Sahili et al., 2025), the present study extends this literature by proving that fusion alone is insufficient for trustworthy clinical deployment unless accompanied by mechanisms that explicitly mitigate confounding leakage.

The proximal bridge mechanism produced treatment-effect estimates that exhibited meaningful directional variation across emotional conditions. The observed behaviour of the proximal bridge parameter during meditation relative to non-meditation states suggests that the model successfully captured intervention-relevant physiological dynamics rather than merely detecting superficial emotional labels. This represents a significant departure from conventional emotion-recognition systems, which typically stop at classification. Theoretically, this result extends the proximal causal frameworks established by (Min et al.,

2025) and (M. Du et al., 2025) for recovering unbiased treatment effects under unobserved confounding.

The application of OPE demonstrated the framework's utility for clinical decision support without exposing individuals to experimental risk. While initial IPS yielded overly optimistic estimates due to known variance sensitivity, implementing a DR estimator corrected this bias, producing a more conservative, clinically plausible policy value of 0.454 against the logged historical policy value of 0.469.

Policy-threshold optimisation revealed that increasing intervention thresholds reduced intervention rates from 50% to 10% while improving estimated policy values from 0.451 to 0.499. This pattern confirms that targeting interventions toward individuals with stronger estimated treatment effects yields superior outcomes, directly aligning with precision medicine principles.

The framework's clinical trustworthiness is further reinforced by robustness, calibration, and fairness analyses. Causal representation utility remained remarkably stable across simulated sensor noise, addressing persistent concerns regarding vulnerability of physiological AI to real-world motion artefacts. However, the model exhibited a high ECE of approximately 0.441. Because uncalibrated probabilities can introduce downstream bias into propensity scores used for OPE, integrating post-hoc calibration techniques like temperature scaling remains a critical prerequisite for deployment.

6. Conclusion

This study presented a causal multimodal deep learning framework integrating proxy-based deconfounding, HSIC-regularised representation learning, proximal causal inference, and OPE for deconfounded emotion recognition from physiological signals. The framework was empirically validated on the WESAD dataset, demonstrating that integrating structural causal inference into deep architectures yields affective representations that are fundamentally more robust, interpretable, and decision-relevant than those generated by traditional correlation-based models.

Proximal causal inference successfully leverages empirical proxy variables to isolate and mathematically adjust for latent unobserved confounding mechanisms within volatile physiological data streams. The HSIC-regularised LSTM-CNN architecture explicitly enforces mathematical separation between orthogonal emotional features and non-stationary nuisance variables, achieving peak classification accuracy on unseen cohorts while maintaining structurally transparent latent trajectories. OPE demonstrates that doubly robust estimation produces conservative, clinically credible policy values, with threshold optimisation yielding improvement over observational policies.

This research establishes that latent confounding is not an insuperable barrier to deep affective computing. Through harmonisation of proximal causal tools and temporal deep neural networks, wearable sensor data can be structurally cleansed of motion artefacts and baseline physiological drift without degrading predictive capacity. The framework extends the operational boundaries of affective computing from passive, static state-classification to active, dynamic treatment-effect estimation, delivering a mathematically validated foundation for automated decision-support systems that directly comply with the core tenets of precision medicine.

Future work should integrate conformal prediction layers to directly map epistemic uncertainty onto the proximal bridge parameter, enabling algorithmic "abstain" mechanisms for anomalous physiological streams. Multi-task HSIC variants capable of accounting for non-linear, time-varying confounders across asymmetric sampling frequencies warrant investigation. Longitudinal, ambulatory monitoring cohorts are required to calibrate downstream propensity scores against diverse, heterogeneous populations, ensuring equitable algorithmic safety before integration into live clinical dashboards.

References

Ahmed, S. F. B., Amin, Md. R., & Islam, Md. K. (2024). Motion artifact contaminated multichannel EEG dataset. *Data in Brief, 57,*

110994. https://doi.org/10.1016/j.dib.2024.110994

Aledavood, T., Luong, N., Baryshnikov, I., Darst, R., Heikkilä, R., Holmén, J., Ikäheimonen, A., Martikkala, A., Riihimäki, K., Saleva, O., Triana, A. M., & Isometsä, E. (2025). Multimodal Digital Phenotyping Study in Patients With Major Depressive Episodes and Healthy Controls (Mobile Monitoring of Mood): Observational Longitudinal Study. *JMIR Mental Health*, *12*, e63622. https://doi.org/10.2196/63622

Althubyani, M., Meng, Z., Xie, S., Seung, C., Razzak, I., Sandoval, E. B., Kocaballi, B., & Cruz, F. (2024). *MERCI: Multimodal Emotional and peRsonal Conversational Interactions Dataset* (Version 2). arXiv. https://doi.org/10.48550/ARXIV.2412.04908

Angulo Medina, A. S., Aguilar Bonilla, M. I., Rodríguez Giraldo, I. D., Montenegro Palacios, J. F., Cáceres Gutiérrez, D. A., & Liscano, Y. (2024). Electroencephalography-Based Brain-Computer Interfaces in Rehabilitation: A Bibliometric Analysis (2013–2023). *Sensors*, *24*(22), 7125. https://doi.org/10.3390/s24227125

Barbierato, E., & Gatti, A. (2024). The Challenges of Machine Learning: A Critical Review. *Electronics*, *13*(2), 416. https://doi.org/10.3390/electronics13020416

Campagner, A., Biganzoli, E. M., Balsano, C., Cereda, C., & Cabitza, F. (2025). Modeling unknowns: A vision for uncertainty-aware machine learning in healthcare. *International Journal of Medical Informatics*, *203*, 106014. https://doi.org/10.1016/j.ijmedinf.2025.106014

Chen, J., Zhong, Y., Huang, Q., Huang, C., Jiang, F., Huang, X., & Wang, X. (2026). UCMIB-PNS: Balancing Sufficiency and Necessity With Probabilistic Causality and Cross-Modal Uncertainty in Multimodal Sentiment Analysis. *IEEE Transactions on Affective Computing*, *17*(1), 92–106. https://doi.org/10.1109/TAFFC.2025.3606964

Du, M., Guo, Y., Li, X., Catala, M., & Pareto-Alhambra, D. (2025). *"Double Machine Learning for Causal Inference in High-Dimensional Electronic Health Records"*. Epidemiology. https://doi.org/10.1101/2025.07.21.25331944

Du, Y., Ding, X., & Ye, Y. (2024). The spatial multi-omics revolution in cancer therapy: Precision redefined. *Cell Reports Medicine*, *5*(9), 101740. https://doi.org/10.1016/j.xcrm.2024.101740

Elashmawi, W. H., Ayman, A., Antoun, M., Mohamed, H., Mohamed, S. E., Amr, H., Talaat, Y., & Ali, A. (2024). A Comprehensive Review on Brain–Computer Interface (BCI)-Based Machine and Deep Learning Algorithms for Stroke Rehabilitation. *Applied Sciences*, *14*(14), 6347. https://doi.org/10.3390/app14146347

García-Hernández, R. A., Luna-García, H., Celaya-Padilla, J. M., García-Hernández, A., Reveles-Gómez, L. C., Flores-Chaires, L. A., Delgado-Contreras, J. R., Rondon, D., & Villalba-Condori, K. O. (2024). A Systematic Literature Review of Modalities, Trends, and Limitations in Emotion Recognition, Affective Computing, and Sentiment Analysis. *Applied Sciences*, *14*(16), 7165. https://doi.org/10.3390/app14167165

Gunda, N. K., Khalaf, M. I., Bhatnagar, S., Quraishi, A., Gudala, L., Venkata, A. K. P., Alghayadh, F. Y., Alsubai, S., & Bhatnagar, V. (2024). RETRACTED: Lightweight attention mechanisms for EEG emotion recognition for brain computer interface. *Journal of Neuroscience Methods*, *410*, 110223. https://doi.org/10.1016/j.jneumeth.2024.110223

Henssler, J., Schmidt, Y., Schmidt, U., Schwarzer, G., Bschor, T., & Baethge, C. (2024). Incidence of antidepressant discontinuation symptoms: A systematic review and meta-analysis. *The Lancet Psychiatry*, *11*(7), 526–535. https://doi.org/10.1016/S2215-0366(24)00133-0

Huang, W., & Shu, N. (2025). AI-powered integration of multimodal imaging in precision medicine for neuropsychiatric disorders. *Cell Reports Medicine*, *6*(5), 102132. https://doi.org/10.1016/j.xcrm.2025.102132

Hussain, S. A. H., Raza, I., Hussain, S. A., Jamal, M. H., Gulrez, T., & Zia, A. (2024). *A Mental State-Aware Brain Computer Interface for Adaptive Control of Electric Powered Wheelchair*. In Review. https://doi.org/10.21203/rs.3.rs-4484096/v1

Jia, T., Sun, J., McGeady, C., Ji, L., & Li, C. (2024). Enhancing Brain–Computer Interface Performance by Incorporating Brain-to-Brain Coupling. *Cyborg and Bionic Systems*, *5*, 0116. https://doi.org/10.34133/cbsystems.0116

Jiao, L., Wang, Y., Liu, X., Li, L., Liu, F., Ma, W., Guo, Y., Chen, P., Yang, S., & Hou, B. (2024). Causal Inference Meets Deep Learning: A Comprehensive Survey. *Research*, *7*, 0467. https://doi.org/10.34133/research.0467

Leaning, I. E., Ikani, N., Savage, H. S., Leow, A., Beckmann, C., Ruhé, H. G., & Marquand, A. F. (2024). From smartphone data to clinically relevant predictions: A systematic review of digital phenotyping methods in depression. *Neuroscience & Biobehavioral Reviews*, *158*, 105541. https://doi.org/10.1016/j.neubiorev.2024.105541

Liu, C., Wang, S., Liu, Q., Wang, Y., & Wang, F. (2026). Mitigating Symptom Heterogeneity in Multimodal Depression Estimation via Level Separation and Deviation Regression. *IEEE Transactions on Affective Computing*, *17*(1), 107–118. https://doi.org/10.1109/TAFFC.2025.3606949

Meng, Z., Althubyani, M., Xie, S., Razzak, I., Sandoval, E. B., Bamdad, M., & Cruz, F. (2025). *PERCY: Personal Emotional Robotic Conversational System* (Version 1). arXiv. https://doi.org/10.48550/ARXIV.2503.16473

Min, J., Zhang, X., & Luo, S. (2025). *A regression-based approach for bidirectional proximal causal inference in the presence of unmeasured confounding* (Version 1). arXiv. https://doi.org/10.48550/ARXIV.2507.13965

Mohammadi, M., Tajik, E., Martinez-Maldonado, R., Sadiq, S., Tomaszewski, W., & Khosravi, H. (2025). Artificial intelligence in multimodal learning analytics: A systematic literature review. *Computers and Education: Artificial Intelligence*, *8*, 100426. https://doi.org/10.1016/j.caeai.2025.100426

Okada, T., Okamoto, S., & Yamada, Y. (2022). Affective Dynamics: Causality Modeling of Temporally Evolving Perceptual and Affective Responses. *IEEE Transactions on Affective Computing*, *13*(2), 628–639. https://doi.org/10.1109/TAFFC.2019.2942931

Roshdy, A., Karar, A., Kork, S. A., Beyrouthy, T., & Nait-ali, A. (2024). Advancements in EEG Emotion Recognition: Leveraging Multi-Modal Database Integration. *Applied Sciences*, *14*(6), 2487. https://doi.org/10.3390/app14062487

Sahili, Z. A., Patras, I., & Purver, M. (2025). *Multimodal Machine Learning in Mental Health: A Survey of Data, Algorithms, and Challenges* (arXiv:2407.16804). arXiv. https://doi.org/10.48550/arXiv.2407.16804

Santomauro, D. F., Vos, T., Whiteford, H. A., Chisholm, D., Saxena, S., & Ferrari, A. J. (2024). Service coverage for major depressive disorder: Estimated rates of minimally adequate treatment for 204 countries and territories in 2021. *The Lancet Psychiatry*, *11*(12), 1012–1021. https://doi.org/10.1016/S2215-0366(24)00317-1

Schmidt, P., Reiss, A., Duerichen, R., Marberger, C., & Van Laerhoven, K. (2018). Introducing WESAD, a Multimodal Dataset for Wearable Stress and Affect Detection. *Proceedings of the 20th ACM International Conference on Multimodal Interaction*, 400–408. https://doi.org/10.1145/3242969.3242985

Sun, G., Hua, H., Lu, J., & Fang, X. (2024). A Novel Causal Discovery Model for Recommendation System. In X. Song, R. Feng, Y. Chen, J. Li, & G. Min (Eds), *Web and Big Data* (Vol. 14334, pp. 261–276). Springer Nature Singapore. https://doi.org/10.1007/978-981-97-2421-5_18

Sun, Y., Kong, L., Chen, G., Li, L., Luo, G., Li, Z., Zhang, Y., Zheng, Y., Yang, M., Stojanov, P., Segal, E., Xing, E. P., & Zhang, K. (2024). *Causal Representation Learning from Multimodal Biomedical Observations* (Version 3). arXiv. https://doi.org/10.48550/ARXIV.2411.06518

Varidel, M., An, V., Hickie, I. B., Cripps, S., Marchant, R., Scott, J., Crouse, J. J., Poulsen, A., O'Dea, B., McKenna, S., & Iorfino, F. (2025). Causal AI Recommendation System for Digital Mental Health: Bayesian Decision-Theoretic Analysis. *Journal of Medical Internet Research*, *27*, e71305–e71305. https://doi.org/10.2196/71305

WHO. (2025). *Mental health factsheet:*

Depression and anxiety [Fact sheet]. https://www.who.int/publications/factsheets/detail/mental-health-depression-and-anxiety

Wu, Y., Mi, Q., & Gao, T. (2025). A Comprehensive Review of Multimodal Emotion Recognition: Techniques, Challenges, and Future Directions. *Biomimetics*, *10*(7), 418. https://doi.org/10.3390/biomimetics10070418