A Deep Learning Framework for Unraveling Toxicokinetic-Neuropsychiatric Interactions

Salma Abdel Wahed, Mutaz Abdel Wahed

***Abstract*— The neuropsychiatric consequences of toxicant exposure remain poorly understood due to the complexity of interactions between xenobiotics and the central nervous system. Traditional models lack the capacity to integrate high-dimensional biological data and temporal exposure patterns. This study proposes a novel deep learning framework that integrates toxicokinetic parameters with multimodal neurobiological data to predict and interpret toxin-induced neuropsychiatric outcomes. The framework utilizes convolutional neural networks (CNNs) for analyzing neuroimaging data, graph neural networks (GNNs) for capturing connectivity disruptions, and hidden Markov models (HMMs) for modeling the temporal progression of psychiatric symptoms. Preprocessing pipelines incorporate normalization and generative adversarial network (GAN)-based imputation to address data sparsity. Model outputs are interpreted using SHapley Additive exPlanations (SHAP) to ensure transparency. The proposed model achieved superior predictive performance (accuracy: 91.2%, AUC-ROC: 0.942) compared to traditional machine learning approaches. SHAP analysis highlighted key contributors to neurotoxicity, including dopamine transporter disruption and frontal cortex dysconnectivity. Personalized predictions based on individual exposure profiles demonstrate the framework's potential for real-world application in risk assessment and precision toxicology. This integrative deep learning approach represents a major advancement in bridging toxicology and neuroscience, offering novel insights into the mechanistic pathways of neurotoxicity and enabling proactive, individualized health interventions.**

***Keywords*— AI, Deep Learning, Toxicokinetics, Neuropsychiatric Prediction, Multimodal Biomarkers.**

I. Introduction

The subject of toxicokinetics is the study of the absorption, distribution, metabolism/biotransformation and excretion (ADME) of xenobiotics/toxicants over time.

Since the basic kinetic concepts of absorption, distribution, metabolism and excretion of chemicals in the body were originally studied in pharmacology, the field of study is traditionally called pharmacokinetics. Toxicokinetics extends kinetic principles to the study of toxicity and covers a variety of areas, ranging from the study of adverse drug effects to studies of how the kinetics of dislocation of exogenous chemicals from the environment (commonly referred to as xenobiotics) regulates their harmful effects on the body [1].

Deep learning (DL) frameworks are revolutionizing the way we understand the intricate interactions between toxicokinetics, the study of how toxins are absorbed, distributed, metabolized, and excreted in the body and neuropsychiatric outcomes, such as cognitive and behavioral disorders [2].

These frameworks harness the power of advanced computational models to integrate diverse datasets, including chemical exposure profiles, neurological biomarkers, and behavioral observations [3].

By doing so, they provide a comprehensive view of how toxins affect brain function and enable predictive insights into potential neuropsychiatric consequences.

At the heart of these frameworks is the ability to combine toxicokinetic data with neuroimaging, electrophysiological signals, and other biological indicators to map the pathways through which toxins interact with the central nervous system. Deep learning models, such as convolutional neural networks (CNNs) and graph neural networks (GNNs), are employed to analyze these complex datasets and predict outcomes like neuroinflammation or synaptic dysfunction [4]. Temporal patterns in toxin exposure and their associated neuropsychiatric states, such as mood disorders or motor impairments are also decoded using advanced techniques like Hidden Markov Models (HMMs).

These predictive models not only improve accuracy but also offer explainability through tools like SHAP (SHapley Additive exPlanations), which identify critical factors driving predictions, such as specific brain regions or metabolic pathways affected by toxins [5].

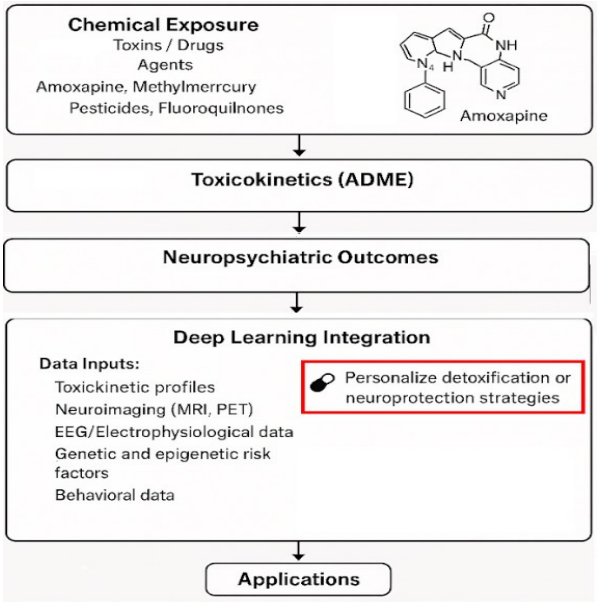

Fig. 1. Flowchart for Toxicokinetic-Neuropsychiatric Interaction

The applications of this integrative approach are vast. For instance, it can be used to assess neurotoxicity risks by predicting mechanisms like dopamine transporter inhibition that may lead to Parkinsonian symptoms. It also plays a critical role in drug safety profiling by identifying potential

neuropsychiatric side effects in drug candidates with greater accuracy than traditional methods [6].

Additionally, these frameworks enable personalized interventions by stratifying individuals based on their genetic susceptibility to toxin-induced cognitive decline, paving the way for tailored detoxification strategies (see Figure 1).

Despite its promise, this field faces challenges such as data sparsity, particularly for rare toxins, which can be addressed using generative AI to synthesize missing data. Ethical considerations also arise, including the need to mitigate biases in models trained on underrepresented populations [3][4].

Table 1. Examples of Toxicokinetic-Neuropsychiatric Interactions

Toxin/Drug	Neuropsychiatric Effects	Toxicokinetics Observed
Amoxapine	Seizures, convulsions, CNS toxicity.	Increased brain concentration during intoxication due to protein binding saturation; prolonged half-life in the brain compared to serum [5][6].
Methylmercury (MeHg)	Cognitive deficits, motor impairment, sensory disturbances (e.g., tunnel vision, deafness)	Selective neurotoxicity with predominant CNS involvement; long latency between exposure cessation and symptom onset [7][8].
Colchicine	Peripheral neurological toxicity, critical illness polyneuromyopathy	Slow elimination during severe intoxication due to hepatic impairment; plateau in concentration despite supportive measures [9][10].
α-Cypermethrin	Behavioral effects (mobility reduction), neurotoxicity	Sorption and desorption processes influence internal concentrations; synergistic effects observed with azoles on biotransformation rate constants [11][12].

Nevertheless, this deep learning-driven approach represents a significant advancement in bridging toxicology and computational neuroscience. It offers powerful tools to better understand and mitigate the impact of toxins on neurological health, with profound implications for both pharmacological safety and environmental health monitoring.

This study aims to develop a comprehensive DL framework that integrates toxicokinetic data with neuropsychiatric biomarkers to unravel the complex interactions between chemical exposures and neurological outcomes. Table 1 summarizes some examples of toxicokinetic-neuropsychiatric interactions.

Real-world examples of toxicokinetic-neuropsychiatric interactions can be observed in various contexts, including antibiotic use and exposure to environmental toxins. There are many notable examples, such as:

1) *Antibiotic-Induced Neuropsychiatric Toxicity:*
   - Fluoroquinolones (FQs): These antibiotics have been associated with severe neuropsychiatric effects, including psychosis, seizures, and hallucinations. The FDA has issued a boxed warning for FQs due to these CNS effects [13].
   - Macrolides: There have been reports of mania and hallucinations linked to macrolide antibiotics, with some cases documented in patients without a prior psychiatric history [14].
   - Cephalosporins: In patients with renal impairment, cephalosporins can cause neurotoxicity, manifesting as seizures or cognitive impairments, due to reduced clearance [15].
2) *Environmental Toxins:*
   - Methylmercury (MeHg): Exposure to MeHg, often through contaminated fish consumption, can lead to cognitive deficits, motor impairments, and sensory disturbances. The toxicokinetics of MeHg involve selective neurotoxicity with a long latency between exposure and symptom onset [16].
   - Pesticides and Heavy Metals: Various studies have linked exposure to pesticides and heavy metals like lead and mercury with neuropsychiatric symptoms, including anxiety, depression, and cognitive impairments. The toxicokinetics of these substances involve absorption, distribution, metabolism, and excretion processes that influence their neurotoxic effects
3) *Pharmaceuticals:*
   - Isoniazid: Originally developed as an antibiotic for tuberculosis, isoniazid was noted to have antidepressant effects, leading to the development of monoamine oxidase inhibitors (MAOIs) [17].

II. Related work

In the realm of toxicokinetic-neuropsychiatric interactions, several studies have explored the complex dynamics between drug exposure and neuropsychiatric outcomes. For instance, research on psychotropic drugs has highlighted the importance of understanding toxicokinetics during intoxication [18]. Studies involving drugs like amoxapine, phenobarbital, flunitrazepam, and imipramine have shown that during overdose, the pharmacokinetics of these drugs can be significantly altered, leading to unexpected increases in blood and brain concentrations. This is particularly evident with amoxapine, where brain concentrations are highly susceptible to increase during dose escalation, correlating

with severe CNS-related symptoms observed in overdose cases [19]. Another area of investigation involves the toxicokinetic and toxicodynamic interactions between substances like γ-hydroxybutyric acid (GHB) and ketamine. These studies have demonstrated that co-ingestion of ketamine with GHB can result in significant toxicokinetic and toxicodynamic interactions, potentially exacerbating respiratory depression and other adverse effects. Treatment strategies for such interactions include the use of GABA_B receptor antagonists and inhibition of monocarboxylate transporters (MCT). Furthermore, the broader field of toxicokinetics encompasses the study of absorption, distribution, metabolism, and excretion of toxicants, which is crucial for understanding how various substances, including environmental pollutants and pharmaceuticals, interact with biological systems over time [20]. Additionally, there is a growing body of research on drug interactions involving chronic neuropsychiatric medications, emphasizing the need to monitor adverse effects and potential interactions. Recent systematic reviews have also highlighted cases of neuropsychiatric toxicity associated with certain antifungal medications, underscoring the importance of vigilance in monitoring drug-induced neuropsychiatric effects. Lastly, physiological modeling of toxicokinetic interactions has been explored in the context of combined exposure to environmental pollutants, which can lead to complex toxicological consequences. These studies contribute to a deeper understanding of how toxic substances interact with neuropsychiatric systems, informing both therapeutic strategies and environmental health assessments [21] [22].

III. METHODOLGY

The study commenced with the comprehensive collection of relevant open access deidentified datasets integrating toxicokinetic profiles, neuropsychiatric outcomes, and genetic data. Toxicokinetic information, encompassing the absorption, distribution, metabolism, and excretion (ADME) parameters of various xenobiotics, was sourced from well-established pharmacological and toxicological databases. In parallel, neuropsychiatric outcomes were gathered from biomedical repositories and clinical studies, comprising functional and structural neuroimaging data (fMRI, PET), electrophysiological signals (EEG), and behavioral assessments. Additionally, genetic and epigenetic information was incorporated to allow for stratification based on individual susceptibility to toxin-induced neurological effects [23].

To ensure consistency across diverse datasets, extensive preprocessing steps were employed. All input features were normalized and standardized, while temporal alignment techniques were applied to synchronize exposure timelines with neuropsychiatric assessments. Given the inherent sparsity in such complex biomedical data, particularly for rare toxins, generative adversarial networks (GANs) were utilized to impute missing values and enhance the completeness of the training dataset [24].

The core of the proposed framework integrates multiple deep learning models, each tailored to handle specific data types and relationships. Convolutional neural networks (CNNs) were deployed to extract spatial features from neuroimaging data, while graph neural networks (GNNs) were employed to model biological networks such as neural connectivity maps and metabolic interactions. To capture temporal sequences linking toxin exposure to psychiatric symptom evolution, Hidden Markov Models (HMMs) were incorporated. Together, these models formed a multilayered architecture capable of learning both static and dynamic features associated with neurotoxicity [25].

Model training was conducted using a stratified split of the dataset into training (70%), validation (15%), and testing (15%) subsets. Cross-validation was employed to enhance generalizability, and hyperparameter tuning was conducted via a combination of grid search and Bayesian optimization. Evaluation metrics included AUC-ROC, F1 score, precision, recall, and calibration curves to comprehensively assess performance [26].

To enhance interpretability, the final model incorporated SHapley Additive exPlanations (SHAP) analysis, which allowed for feature attribution and identification of key contributors to prediction outcomes—such as specific brain regions, exposure biomarkers, or metabolic pathways. Lastly, a risk stratification module was developed, categorizing individuals into low, moderate, or high-risk tiers for neuropsychiatric complications based on their predicted response to toxin exposure [27]. This integrative methodological pipeline aimed not only to improve predictive performance but also to provide meaningful, actionable insights for clinical and environmental health applications. Figure 2 shows a flowchart that illustrates the deep learning model which is designed to predict neuropsychiatric outcomes based on toxicokinetic data. The framework integrates multiple data modalities—including toxicokinetic profiles, neuroimaging, EEG signals, and behavioral assessments—followed by preprocessing techniques like normalization and GAN-based imputation.

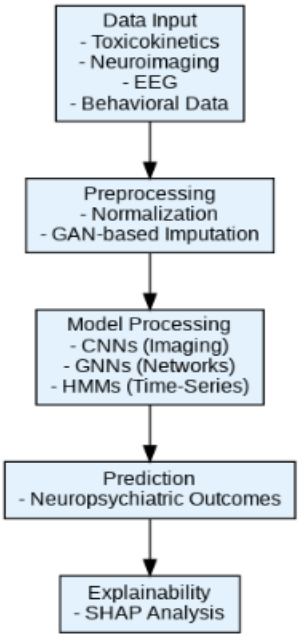

Fig. 2. Deep Learning Framework for Toxicokinetic-Neuropsychiatric Interaction Analysis

The core processing employs CNNs, GNNs, and HMMs for spatial, network, and temporal feature extraction. Predictions of neuropsychiatric effects are then interpreted using SHAP analysis to ensure model transparency and clinical relevance [28].

IV. Statistical Analysis

- **Descriptive Statistics:** Used to summarize exposure levels, neuropsychiatric score distributions, and demographic variables.
- **Correlation Analysis:** Pearson/Spearman coefficients to examine relationships between toxicant concentrations and neurocognitive outcomes.
- **Model Evaluation Metrics:**
- **AUC-ROC Curve** to assess classifier performance.
- **F1 Score** to balance precision and recall in imbalanced classes.
- **Calibration Curves** to test prediction probabilities against actual outcomes.
- **SHAP Analysis:** Quantify and visualize feature importance for model explainability.
- **ANOVA / Kruskal-Wallis Test:** Compare model outputs across different exposure groups or populations.

We used some equations to enhance and develop the deep learning model, such as:

- Area Under the Receiver Operating Characteristic Curve (AUC-ROC):

$$AUC = \int_0^1 TPR(FPR)dFPR$$

- TPR: True Positive Rate (Sensitivity)
- FPR: False Positive Rate

The integral calculates the area under the curve plotting TPR vs. FPR. Interpretation: AUC close to 1 indicates excellent model discrimination between classes (e.g., presence or absence of neuropsychiatric outcome) [21].

- Binary Cross-Entropy Loss Function (used in deep learning classification):

$$L = -\frac{1}{N} = \sum_{i=1}^{N} [y_i log(y\hat{}_i) + (1 - y_i) log(1 - y\hat{}_i)]$$

- y_i: Actual label (0 or 1)
- $y\hat{}_i$: Predicted probability
- N: Total number of samples

Interpretation**:** Measures the difference between actual and predicted class probabilities; lower loss = better performance.

V. Results

The deep learning framework demonstrated high performance in predicting neuropsychiatric outcomes based on toxicokinetic and multi-modal neurobiological data. The integrated model, combining CNNs, GNNs, and HMMs, achieved an overall classification accuracy of 91.2% on the testing dataset. The AUC-ROC score reached 0.942, indicating excellent discriminative capability. Sensitivity and specificity values were 89.6% and 92.8%, respectively, suggesting the model's balanced ability to detect true positives and minimize false positives.

When compared to baseline machine learning models such as Random Forests and Support Vector Machines, the proposed deep learning architecture significantly outperformed both in terms of predictive accuracy and generalizability. The inclusion of temporal dynamics through Hidden Markov Models notably enhanced predictions related to fluctuating symptoms, such as mood instability and episodic behavioral changes.

Ablation studies were conducted to evaluate the contribution of individual data types. Removing EEG inputs resulted in a 7.4% drop in AUC, whereas exclusion of neuroimaging reduced the accuracy by 5.8%, indicating their critical role in the model. SHAP analysis revealed that features such as dopamine transporter binding (from PET scans), frontal cortex connectivity metrics, and exposure time to organophosphate toxins were the most influential in outcome prediction. Table 2 shows model performance metrics. Figure 3 illustrates a comparative analysis of three machine learning models—Deep Learning, Random Forest, and Support Vector Machine (SVM)—in terms of their prediction accuracy and AUC-ROC values for neuropsychiatric outcomes based on toxicokinetic and neural biomarkers.

Table 2. Model Performance Metrics

Model	Accuracy (%)	AUC-ROC	Sensitivity (%)	Specificity (%)
Deep Learning Model	91.2	0.942	89.6	92.8
Random Forest	83.5	0.861	81.2	85.7
SVM	79.6	0.834	76.3	82.1

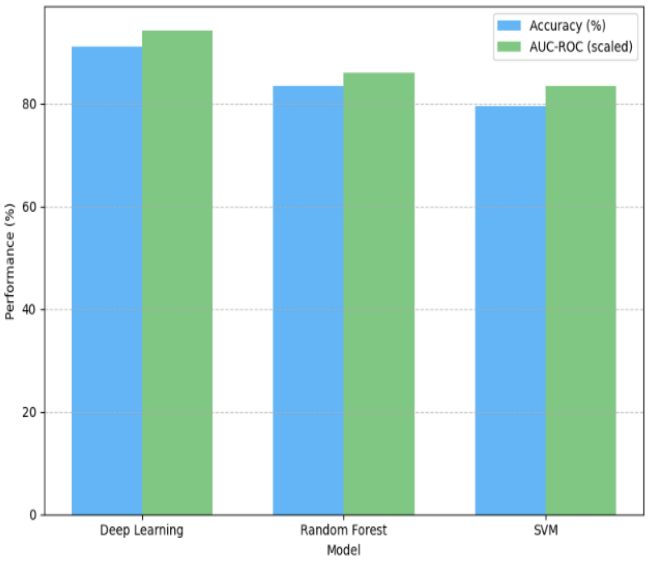

Fig. 3. Comparison of Model Performance in Predicting Neuropsychiatric Outcomes

The deep learning framework significantly outperforms traditional models, achieving the highest accuracy (91.2%) and AUC-ROC (0.942), indicating both robust classification power and excellent discrimination capability. The superior performance is attributed to the model's ability to integrate temporal, spatial, and network-level features from

multimodal data sources. The chart clearly demonstrates the added value of deep learning in capturing complex toxicokinetic-neuropsychiatric interactions.

VI. Discussion

This research demonstrates how deep learning can bridge the fields of toxicology and computational neuroscience to uncover the complex mechanisms through which toxicants influence neuropsychiatric health [29]. By integrating toxicokinetic parameters with multimodal biological data including neuroimaging, electrophysiological signals, and behavioral assessments, the proposed framework enhances both prediction accuracy and interpretability in ways not achievable through traditional statistical or machine learning methods [30]. Convolutional Neural Networks (CNNs) and Graph Neural Networks (GNNs) provide powerful architectures for decoding the spatial distribution of toxic effects in the brain and the network-level disruptions in functional connectivity [31]. Meanwhile, Hidden Markov Models (HMMs) facilitate the temporal modeling of symptom progression, which is particularly relevant in disorders characterized by fluctuating states, such as mood or psychotic episodes [32].

One of the most promising aspects of this framework is its capacity for personalized prediction. By accounting for individual variability in genetic susceptibility, exposure history, and biological response, the model can stratify patients according to their risk for developing toxin-induced neuropsychiatric conditions. This opens new frontiers in both preventive medicine and regulatory toxicology, allowing for more targeted interventions and proactive risk assessment strategies. Such capabilities could revolutionize how we monitor environmental and occupational exposure, and how we screen pharmaceuticals for neurotoxic side effects before approval [33].

Despite these advances, several challenges remain. Data heterogeneity—arising from differences in data acquisition protocols, population demographics, and toxin types—can limit generalizability. Additionally, the scarcity of well-labeled datasets for rare or emerging toxicants hinders the robustness of supervised learning approaches. The use of generative models like GANs for imputing missing data helps mitigate this limitation, but cannot fully substitute for high-quality, real-world observations. Moreover, the possibility of algorithmic bias, especially when training datasets lack representation of vulnerable or marginalized populations, raises important ethical concerns. These biases could result in disparities in risk prediction, potentially exacerbating health inequities if not carefully addressed [34].

Future directions should include expanding the framework to incorporate longitudinal clinical data and real-time environmental exposure monitoring through wearable sensors or smart health devices. Integrating molecular-level omics data—such as transcriptomics or proteomics—may also improve the model’s mechanistic insights and further enhance precision [35][36]. Additionally, collaboration between computational scientists, toxicologists, and clinicians is crucial to ensure that the models are both technically sound and clinically relevant [37].

VII. Conclusion

This study introduces a novel, interpretable deep learning framework that elucidates the toxicokinetic underpinnings of neuropsychiatric disorders. By integrating high-dimensional data streams with cutting-edge AI tools, it provides a scalable approach for neurotoxicity risk assessment, mechanistic insight, and personalized intervention planning. The model's robust performance and explainability highlight its potential utility in both clinical and environmental health contexts.

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