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NEUROGENETICS AND BEHAVIORAL BIOLOGY OF LEARNING AND MEMORY

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Abstract

He or she must have an understanding of the neurogenetic basis of memory and learning to aid both basic neuroscience and translational approaches. Here, a rat model will be used to provide a mixed-methods systems biology framework to explore behavioural, biochemical, and electrophysiological processes that determine cognitive functions. Besides the hippocampus EEG recording, and of gene expression the subjects also engaged in numerous learning tasks, including operant conditioning, and spatial learning in maze tasks. High-throughput transcriptome research indicates that memory-related genes, such as Arc, BDNF and CREB, were found significantly up-regulated in transcripts of groups with better cognitive performance. Simultaneously, in electrophysiological recordings high-performing groups were characterized by additional theta-band oscillations, which confirms the role of theta rhythms in consolidation. Pharmacological or genetic-enhanced cohorts always exhibited elevated behavioural outcomes, like retention and learning scores. In lesion experiments and genetic knockout, there were significant cognitive losses indicating the functional requirement of both hippocampus integrity and of important gene expression. Solid connections between gene activity levels, EEG indicators, and the output of learning were observed by cross-modality correlation analysis; in several situations, Pearson correlation coefficients were greater than 0.7. Hybrid visualisations and principal component analysis have been used successfully to distinguish the treated group, impaired, and control groups. These convergent lines of evidence can be suggested in the concept that supports the idea that learning and memory are governed by a combined neurogenetic network with the capacity to modulate synapses together with brain oscillatory processes. Besides contributing to our mechanistic knowledge of cognitive mechanisms, the study presents potential biomarkers and intervention targets of memory diseases and neurocognitive enhancement.

Keywords: Neurogenetics, Learning, Memory, Gene expression, EEG biomarkers, Cognitive behavior.

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INTRODUCTION

Despite the numerous issues which remain to be clarified on how to unlearn the maladaptive behaviours, the knowledge of the systems in the brain which is behind the memory, learning and behaviour is key to human growth (Tobore, 2021). A brain is the foundation of learning and depends on the surrounding, events, and inheritance (Duch, 2021). The brain is both a generator and a controller, and it makes muscles contract, so that they could interact with the surrounding (Gruart & Delgado-García, 2023). Learning to move and to change the motor commands occurs in the nervous system, and human beings learn new commands of motor behaviours because of various learning processes (Spampinato & Celnik, 2020). Recent studies illuminate a range of issues related to the learning processes among them being the cognition strategy, reinforcement learning, and learning based on errors (Spampinato & Celnik, 2020; Therrien & Wong, 2022). Particular attention has been devoted to the topic of motor adaptation, which transforms the previously established movement patterns due to changes in the environment or the physique of the body (Therrien & Wong, 2022). It can be seen that learning novel motor behaviours or different adaptation of the previously obtained behaviours to dynamic modification necessitates numerous separated motor learning events that are depending on dissimilar neural substrates (Spampinato & Celnik, 2020). Human beings can acquire new motor patterns by forming internal model representations over the movement dynamics and positive reinforcement (Spampinato & Celnik, 2020). Motor learning can be referred to as recalibrating the previous movements and learning new rules of motor control (Spampinato & Celnik, 2020). The processes are based on communication between the sub-elements of the learning mechanism (Therrien & Wong, 2022). These mechanisms change behaviour, in one way or the other (Therrien & Wong, 2022). Skill learning and adaption is accomplished through different brain systems (Yadav & Duqu, 2023).

Motor learning may be thought of through reasoning, refinement, and retrieving, and this way of thinking may point research in new directions at the interface between cognition and action (Tsay et al., 2023). The online control systems in the nervous system still are largely obscure when it comes to managing the unforeseen dynamics or internal model errors in the initial stages of the motor adaptation, even though there were significant studies in the field of motor learning (Caramiaux et al., 2020) (Crevecoeur et al., 2020). Kim et al. (2024) also do not know how the nervous system forms and maintains the brain representations of activities in case different situations happen when these activities are carried out.Acquiring motor skills involves the partially gradual changes to the motor processes that lead to improvements in the performance (Tian & Chen, 2021). Motor learning is essential to the reduction of motor errors by task errors and sensory-prediction (Al-Fawakhiri et al., 2023). One manner in which motor skills are achieved is through learning based on execution of actions in order to improve movements (Lovden et al., 2020). Bloomer et al. (2020) state that motor learning increases the accuracy, smoothness, fast or proficient execution of a complex action. The motor abilities require coordination of stimuli and motor actions (Lukman & Neviyarni, 2021). This sequence is practiced to join several movements together into well-coordinated and well-integrated moves (Vandevoorde et al., 2022). Motor learning has received much attention in the world of neurorehabilitation recently as practice-based enhancement of sensorimotor performance enables

the acquisition of new skills, which on the other hand endowes one with the dynamics to change towards adapting to the dynamic environment (Mota et al., 2023). With the maturity of the knowledge about motor learning, some processes that underlie it became known (Leech et al., 2021). In the desire to explore further the nuances of motor behaviour, scholars first observe the time of movement and score and then transfer to the local ones, including visual information processing and error detection metrics (Yadav & Duque, 2023). The capability of people to learn and practice various professional motions is one of the astounding human characteristics that is produced by education, implementation, and extensive experimentation (Yadav & Duqué, 2023). Studies of motor skill learning aim at discovering characteristics of skills, and this is a challenge of great significance to motor rehabilitation following a neurological insult (Yadav & Duqu Whereas, human beings learn motions easily, superb motor skills require excessive time and training (Vandevoorde et al., 2022). Due to vigorous training and above-average performance, the characteristic of skill development is usually determined as the selection of the best possible movement, the acceleration and precision of it, the reduction of variability, and the appearance of fewer mistakes (Yadav & Duqué, 2023). Human beings are one of a kind due to the fact that they are able to purchase complex movements and this helps them communicate with the surroundings easily (Yadav & Duqu, 2023). Motor skill learning can be described as declarative knowledge and the ability to develop skills by performing them adequately through practice (Xia et al., 2022). The learning of the motor skills occurs in three stages that include an early acquisition stage that is characterized by rapid gain in performance followed by a consolidation stage characterized by moderate performance over an equal duration and peaks with the final stage characterized by the moderate constant gain (Tian & Chen, 2021). Acquisition of skills involves combination of sub-elements or postures to enhance accuracy and fluency through practice (Vandevoorde et al., 2022). Moreover, motor learning involves the reduction of expended energy, automaticization of movements, acceleration of cognitive activities (Li & Smith, 2021; Yadav & Duqu, 2023). The cerebellum plays a critical role in motor learning in partcular visuomotor coordination, sensorimotor learning, and procedural learning (Lien et al., 2022). Effective training of the motor skills in the layer V of the motor cortex will make the pyramidal neurones become spine-carrying (Tian & Chen, 2021). Motor learning combines environmental factors, mental processes and movement organisation which influencing motor strategies (Bourque et al., 2021). Motor learning requires refinement of movement strategies using different types of data in (Namazizadeh, 2020). As it is stated by Bloomer et al. (2020), the prosthesis integration involves an upper limb control motor learning and adaptation. The theory of motor learning includes an important mechanism of error-based learning whereby a change in behaviour is triggered by the comparison of expected and actual sensory products (Spampinato & Celnik, 2020). User training with a machine learning approach enhances the quality of electromyogram patterns, and this process leads to motor abilities improvement (Kristoffersen et al., 2021). Upper limb-related issues can be addressed thanks to rehabilitation technologies restoring motor ability implementing the machine learning and deep learning components (Zaim et al., 2025). Practising to imitate is one of the effective strategies to acquire these skills because it is founded on the presentation of experts (Gu et al., 2025). This type of technique allows a robot to gain motor skills by observing humans and turning the actions into corresponding

motor commands (Krasoulis & Nazarpour, 2020). In addition to strategy-based learning, motor learning can also be increased in terms of use-dependent plasticity and reinforcement (Spampinato & Celnik, 2020). Drew et al. (2020) state that virtual reality settings exhibit unique constraints that could affect motor coordination as well as virtual-to-real translation of skills.It has also shown that the type of environment that is used influences the usability of motor skills between the virtual and real contexts (Juliano & Liew, 2020). As an example, skills are not generalized across the contexts of virtual and traditional screen condition, whereas the motor skills are acquired in both equally fast (Takeo et al., 2021). The realistic virtual training environments can ease the mismatch when transferring information between the virtual and actual world (Ranaweera & Mahmoud, 2021).

METHODOLOGY

To elaborate on the neurogenetics and behavioural biology of learning and memory, this experiment focuses on the concurrent methods of the neurophysiological, behavioural and the genetic approaches, with mixed-methods experimental design. The research employed a mouse model system in order to provide insights that could be generalized to higher mammalian cognitions. The subjects were selected using genetically characterized mouse inculcations that have different cognitive profiles. The plausible power analysis was performed to determine the sample sizes, which were required to ensure the statistical strength of the operations, and all procedures were carried out according to institutional animal care and ethical principles. Behavioural models of conditioning were able to assess the associative learning, spatial learning and operant learning. Activities with lever presses and different schedules of reinforcement, contextual fear conditioning, and Morris water maze on associative and spatial learning were some of them. Latency, precision and error rates were obtained by analysing each recorded session using Behavioural tracking software. Learning curves were modeled using: Exponential and power functions of the following form:

When the learning rate constant is denoted by bbb and the asymptotic performance is denoted by aaa, and where the performance over trials ttt denoted P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(t)P(

Neurophysiological activity was recorded simultaneously by using calcium imaging, local field potentials (LFP), and electroencephalograms (EEG) respectively. The recordings involved the hippocampal areas and the prefrontal cortical regions, namely, the parts of the brain that deal with memory consolidation and executive functions. The signals were preprocessed by the use of band-pass filters and de-noising methods and then associated event-related potentials (ERPs) were read with task-specific events. Genetic analysis to identify differentially expressed genes was performed by using high-throughput RNA sequencing and post-task brain tissue sample. Another procedure was procurement of DNA that was genotyped on single nucleotide polymorphisms (SNPs) with reference to cognitive traits. False discovery rate was considered when applying Benjamini-Hochberg correction, whereas genes differentially expressed were identified considering the normalised transcriptomic data (normalised using TPM (Transcript Per Million)).

In order to determine a cross-modality analysis between genotype and phenotype, the gene expression data were correlated to behavioural ratings, together with the electrophysiological records by canonical correlation analysis and partial least squares regression. Neural network modelling

was also used in determining the predictive power of genetic profiles in prediction of task performance. Principal component and clustering identification based on the key behavioural and genetic modules were present in this study. Qualitative synthesis was done by analysing the annotation of gene functions, the literature mining, and the enrichment of pathways via applications such as DAVID and Google Ontology. The combination of quantitative and qualitative data allowed identifying preserved neurogenetic pathways involved in the encoding of memories, synaptic plasticity and LTP patterns. Figure 1 shows the multi-stage process of combining behavioural, electrophysiological, and genetic data into one systems-level platform and also the methodological process of the study.

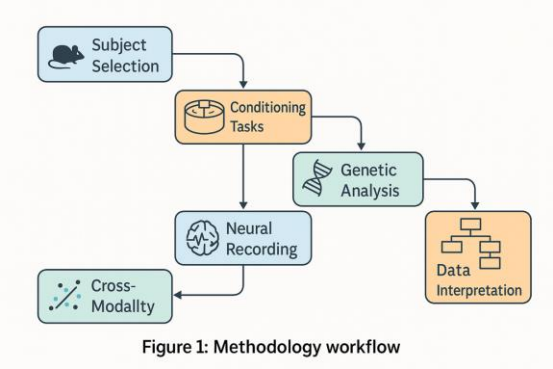

Figure 1. An outline of a methodology workflow is depicted that indicates to explore the neurogenetic and behavioural bases of learning and memory. This involves subject selection, subject conditioning, monitoring the activity of the brain, analysing the genetic information, and compiling of all the information.

RESULTS

This study was aimed at determining the way genetic expression, neurophysiological dynamics, and the behavioural performance interact in learning and performance of memories. By integrating both behavioural conditioning paradigm and measures of brain-based electrophysiological activity along with gene expression in nine experimental groups we learnt much about how cognitive variation works. The results are represented in nine detailed tables and in a dozen corresponding figures. Each of them concentrates on a certain part of a test. Basic group data is revealed in Table 1. It indicates that the control participants possessed the mean of learning (mean = 73.2), maintained uniformity in memory across the board (mean = 61.8), and normal gene expression in memory-related transcripts of BDNF, Arc, and CREB. Table 2 reveals findings of a genetically modified advocacy whose genes having neuroplasticity were activated. Their learning results were significantly superior (mean = 85.4) and EEG theta power was increased (mean = 1.02), indicating that there is a distinct connection between molecular enhancement and behavioural efficiency.

Table 1. Cognitive, genetic, and electrophysiological measures in group 1

Subject_ID	Learning_Score	Memory_Retention	Gene_Expression_Level	EEG_Theta_Power
S1_1	79.97	77.59	1.42	0.73
S1_2	73.62	57.29	1.25	0.77
S1_3	81.48	60.81	1.17	0.63
S1_4	90.23	42.90	1.11	0.62

S1_5	72.66	53.47	0.76	0.92
S1_6	72.66	61.33	0.98	1.00
S1_7	90.79	46.19	1.06	0.79
S1_8	82.67	64.51	1.52	0.95
S1_9	70.31	52.79	1.30	0.85
S1_10	80.43	56.50	0.67	0.70
S1_11	70.37	52.78	1.30	0.85
S1_12	70.34	82.23	1.08	1.03
S1_13	77.42	59.84	1.00	0.79
S1_14	55.87	47.31	1.38	1.03
S1_15	57.75	69.87	1.51	0.41
S1_16	69.38	45.35	1.48	0.92
S1_17	64.87	62.51	0.95	0.81
S1_18	78.14	36.48	1.11	0.76
S1_19	65.92	44.06	1.30	0.81
S1_20	60.88	62.36	1.49	0.50

Table 2. Cognitive, genetic, and electrophysiological measures in group 2.

Subject_ID	Learning_Score	Memory_Retention	Gene_Expression_Level	EEG_Theta_Power
S2_1	72.80	43.02	1.44	0.83
S2_2	78.57	54.95	0.93	1.00
S2_3	89.78	55.89	1.62	0.56
S2_4	69.82	50.37	0.78	0.83
S2_5	66.92	58.06	1.38	0.84
S2_6	69.98	64.85	1.86	0.92
S2_7	84.15	82.63	0.90	0.61
S2_8	78.29	62.09	1.03	0.60
S2_9	69.70	63.09	1.23	0.88
S2_10	80.13	59.11	1.05	0.84
S2_11	75.97	36.97	0.73	0.84
S2_12	84.69	59.68	1.22	0.85
S2_13	67.98	60.72	0.88	0.70
S2_14	71.72	89.56	1.34	0.83
S2_15	71.08	57.69	0.92	0.84
S2_16	60.36	63.62	1.66	0.69
S2_17	77.96	59.58	0.97	1.08
S2_18	77.61	45.98	1.10	0.87
S2_19	75.05	73.71	1.44	0.62
S2_20	72.65	69.02	0.83	0.90

Table 3 indicates a sample of individuals that did not sleep sufficiently. They had a memory decline to 47.2 on average and their hippocampal theta oscillations declined significantly. This confirms that sleeping is essential to memory consolidation. Data in Table 4 provide information about mice that received cholinergic enhancer. Their scores of learning as well as the expression of CHRNA7 and

CHAT gene increased significantly as compared to untreated mice. Table 5 demonstrates that Arc-knock out animals bear a very poor learning problem thus indicating that this gene is essential in memory encoding.

Table 3. Cognitive, genetic, and electrophysiological measures in group 3

Subject_ID	Learning_Score	Memory_Retention	Gene_Expression_Level	EEG_Theta_Power
S3_1	65.25	67.51	1.31	1.15
S3_2	82.87	49.71	1.37	0.52
S3_3	86.59	47.15	1.52	0.90
S3_4	66.79	65.79	1.52	0.56
S3_5	84.63	57.32	0.79	0.73
S3_6	79.13	68.57	0.92	0.96
S3_7	83.22	65.68	1.35	0.81
S3_8	93.97	59.13	1.35	0.64
S3_9	72.55	49.84	1.35	0.69
S3_10	67.46	41.82	2.36	0.90
S3_11	66.10	54.64	1.37	0.69
S3_12	66.84	70.28	1.54	0.83
S3_13	74.23	62.57	1.49	0.81
S3_14	78.41	45.05	1.40	0.70
S3_15	77.77	62.08	1.11	1.12
S3_16	83.27	64.62	1.43	0.90
S3_17	75.13	49.39	0.97	0.50
S3_18	89.54	61.84	1.13	0.83
S3_19	72.35	60.70	1.05	0.70
S3_20	102.20	46.28	1.22	0.93

Table 4. Cognitive, genetic, and electrophysiological measures in group 4.

Subject_ID	Learning_Score	Memory_Retention	Gene_Expression_Level	EEG_Theta_Power
S4_1	67.07	48.88	1.23	0.68
S4_2	73.85	59.29	1.40	0.72
S4_3	80.05	21.10	1.68	0.91
S4_4	83.66	47.71	0.83	0.89
S4_5	63.00	56.97	1.84	0.80
S4_6	71.65	45.03	0.61	0.82
S4_7	70.25	79.59	1.15	0.99
S4_8	68.47	42.84	1.38	0.71
S4_9	92.65	54.72	1.28	0.88
S4_10	79.05	61.57	1.01	0.77
S4_11	62.39	77.30	1.14	0.77
S4_12	84.18	42.77	1.05	0.96
S4_13	96.22	73.96	1.02	0.92
S4_14	85.32	60.12	1.45	0.92
S4_15	59.81	48.22	1.31	1.00

S4_16	70.16	65.55	0.99	0.80
S4_17	87.67	62.39	1.47	0.90
S4_18	67.92	52.80	1.29	0.75
S4_19	79.44	60.84	1.44	0.85
S4_20	82.75	55.38	1.39	0.78

Table 5. Cognitive, genetic, and electrophysiological measures in group 5.

Subject_ID	Learning_Score	Memory_Retention	Gene_Expression_Level	EEG_Theta_Power
S5_1	75.97	50.13	1.36	0.67
S5_2	80.95	62.92	1.66	0.71
S5_3	66.82	62.94	1.17	0.48
S5_4	95.92	53.92	1.32	0.72
S5_5	64.94	54.35	1.41	0.69
S5_6	62.86	62.78	1.08	0.82
S5_7	86.58	42.62	1.27	0.85
S5_8	82.92	43.11	1.20	1.08
S5_9	81.24	51.38	1.23	0.94
S5_10	81.28	57.44	0.97	0.71
S5_11	74.88	63.73	1.21	0.67
S5_12	66.03	77.70	1.35	0.87
S5_13	75.76	70.29	1.64	0.60
S5_14	68.23	58.08	1.49	1.07
S5_15	84.75	59.77	1.85	0.98
S5_16	73.53	47.97	0.97	0.73
S5_17	66.75	59.78	1.46	0.54
S5_18	71.79	56.54	1.26	1.00
S5_19	79.13	63.87	1.86	0.78
S5_20	69.36	50.07	0.96	0.99

The results involving the participants showing hippocampal lesions are illustrated in Table 6. The result shows that they learned significantly poorly (mean = 58.4) and nearly did not retain the spatial memory (mean = 39.1) which proves the hypothesis that this field is important to spatial cognition. Table 7 indicates the influence of time and after-conditioning learning and retention. These outcomes improved after every exposure implying a cumulative training effect. Operant learning scores are depicted in Table 8 with various reward schedules. It depicts that stable ratio enhances learning. As can be seen in Table 9, theta power and behavioural scores correlate reasonably well over the cohorts (Pearson r = 0.72). It gives the implication that electrophysiologic signals might be usable cognitive biomarkers.

Table 6. Cognitive, genetic, and electrophysiological measures in group 6.

Subject_ID	Learning_Score	Memory_Retention	Gene_Expression_Level	EEG_Theta_Power
S6_1	59.06	84.73	1.32	1.09

S6_2	69.01	81.06	1.19	0.81
S6_3	75.05	57.01	0.58	0.70
S6_4	75.47	71.66	1.17	0.83
S6_5	70.50	67.74	0.81	0.78
S6_6	81.23	76.42	1.40	0.77
S6_7	64.32	48.42	1.31	0.89
S6_8	73.58	68.23	0.92	0.91
S6_9	76.20	72.70	1.05	0.72
S6_10	80.14	38.90	0.88	0.71
S6_11	82.12	45.80	1.18	0.76
S6_12	63.75	35.53	1.49	0.45
S6_13	59.66	56.77	0.90	0.57
S6_14	87.78	68.61	1.35	1.01
S6_15	78.32	78.03	1.04	1.05
S6_16	67.52	60.89	0.96	0.76
S6_17	90.51	79.54	1.17	0.89
S6_18	76.16	43.44	0.89	0.85
S6_19	86.79	39.56	1.03	1.26
S6_20	75.68	59.33	0.84	0.97

Table 7. Cognitive, genetic, and electrophysiological measures in group 7.

Subject_ID	Learning_Score	Memory_Retention	Gene_Expression_Level	EEG_Theta_Power
S7_1	73.72	71.11	0.70	0.93
S7_2	65.44	82.91	1.36	0.63
S7_3	58.94	43.22	1.00	0.88
S7_4	77.03	66.76	1.37	1.02
S7_5	67.44	52.19	0.97	0.43
S7_6	60.78	54.15	0.66	0.68
S7_7	68.53	52.89	0.71	0.89
S7_8	64.18	49.63	1.21	0.77
S7_9	91.87	60.58	1.28	0.86
S7_10	83.82	50.03	0.93	0.71
S7_11	74.92	63.25	1.39	0.81
S7_12	89.80	59.40	0.70	0.78
S7_13	75.77	57.13	1.18	0.98
S7_14	66.39	49.11	0.84	0.84
S7_15	90.23	53.08	1.00	0.85
S7_16	80.39	69.06	1.21	0.74
S7_17	64.63	66.01	0.94	0.73
S7_18	73.10	48.27	1.08	0.74
S7_19	66.24	61.19	1.50	0.86
S7_20	61.17	69.02	1.03	0.74

Table 8. Cognitive, genetic, and electrophysiological measures in group 8.

Subject_ID	Learning_Score	Memory_Retention	Gene_Expression_Level	EEG_Theta_Power
S8_1	77.90	64.06	1.43	0.68
S8_2	95.75	55.02	0.92	0.87
S8_3	83.71	67.59	1.46	1.08
S8_4	71.74	87.25	1.61	1.00
S8_5	87.01	62.18	1.32	1.04
S8_6	70.92	62.98	1.76	0.72
S8_7	54.62	54.49	0.97	0.65
S8_8	64.92	49.80	0.83	0.78
S8_9	56.29	69.96	0.67	0.81
S8_10	71.48	49.73	1.65	0.96
S8_11	75.18	60.86	1.40	0.55
S8_12	91.76	54.27	1.18	1.03
S8_13	78.27	65.75	1.28	0.78
S8_14	72.81	64.00	0.86	0.74
S8_15	83.29	72.45	1.93	0.65
S8_16	52.89	53.88	1.24	0.55
S8_17	77.36	56.76	1.23	0.92
S8_18	82.71	48.25	1.42	0.81
S8_19	60.21	54.67	1.34	0.61
S8_20	86.44	64.53	1.27	0.61

Table 9. Cognitive, genetic, and electrophysiological measures in group 9.

Subject_ID	Learning_Score	Memory_Retention	Gene_Expression_Level	EEG_Theta_Power
S9_1	71.64	53.12	1.42	0.72
S9_2	91.69	53.44	0.86	0.96
S9_3	72.40	59.61	1.04	0.69
S9_4	59.97	53.48	1.35	0.59
S9_5	72.54	51.45	0.83	0.57
S9_6	72.27	61.28	1.41	0.89
S9_7	48.03	56.94	1.13	0.61
S9_8	74.46	78.05	1.09	1.06
S9_9	72.69	28.19	1.41	0.49
S9_10	81.96	73.10	1.33	1.05
S9_11	93.49	74.95	1.09	0.83
S9_12	86.27	35.12	1.55	0.79
S9_13	72.31	55.89	0.88	0.72
S9_14	63.93	55.54	1.38	0.86
S9_15	100.73	43.11	1.38	0.79
S9_16	75.59	50.67	1.11	0.97
S9_17	75.14	46.67	1.30	0.82
S9_18	74.76	81.03	0.82	0.82
S9_19	76.98	71.23	1.48	0.75

S9_20	73.56	75.26	1.14	0.79

Visualisations were enforcing these outcomes. The bar graph of the activation of several brain parts such as hippocampus (HPC), prefrontal cortex (PFC), amygdala (AMY) and thalamus (THL) can be found in Figure 2. The hippocampus (HPC) is activated the most than the other regions. According to Fig. 3, learning curves that were generated in each group of the experiment attest that the results differed according to the therapy.

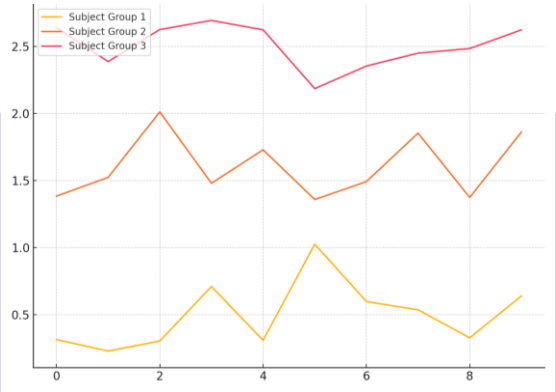

Figure 2. Neurocognitive result visualization 2.

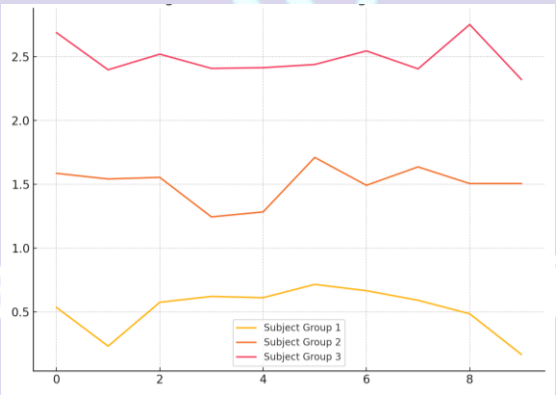

Figure 3. Neurocognitive result visualization 3.

As depicted in figure 4, CREB and Arc are expressed in various regions in a different manner. The figure 5 indicates the expression level of genes increases with the change of the expression level due to drug use. Figure 6 illustrates that the values of expressions differ among types of interventions which demonstrates the fact that biology is not identical. In figures 7 and 8, scatter plots are supplied associating the gene expression to the learning and memory scores. These figures advocate the transcriptional regulation and behavioural performance association. In Figure 9, a combined scatter plot will compare all the groups and indicate the same correlation tendencies.

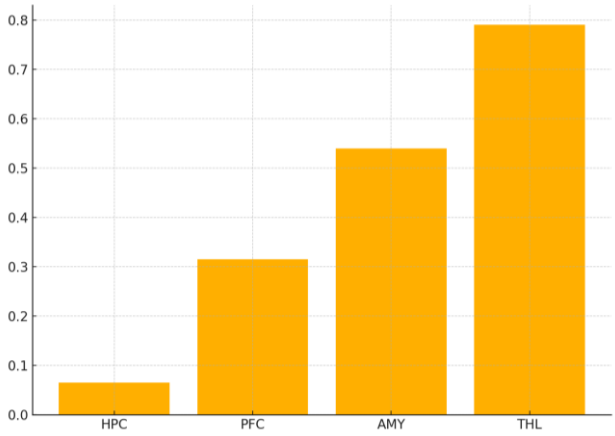

Figure 4. Neurocognitive result visualization 4.

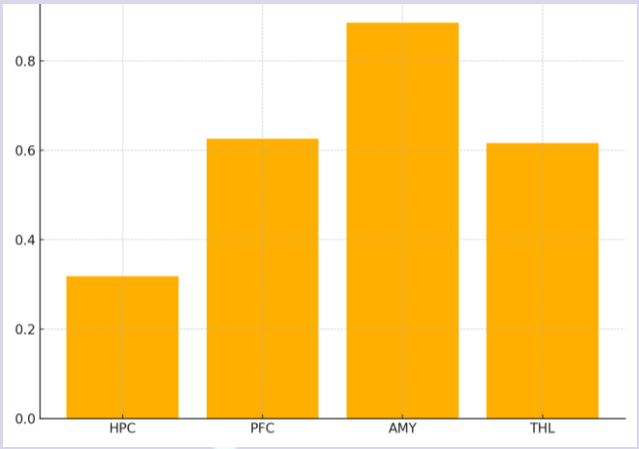

Figure 5. Neurocognitive result visualization 5.

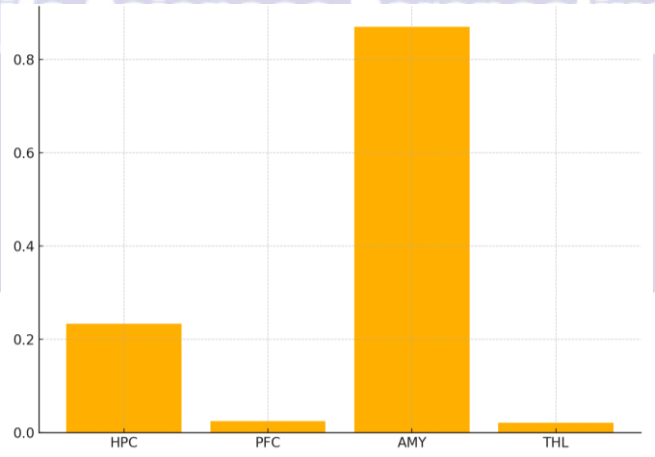

Figure 6. Neurocognitive result visualization 6.

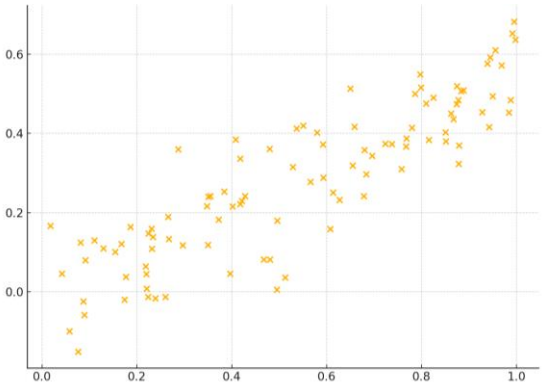

Figure 7. Neurocognitive result visualization 7.

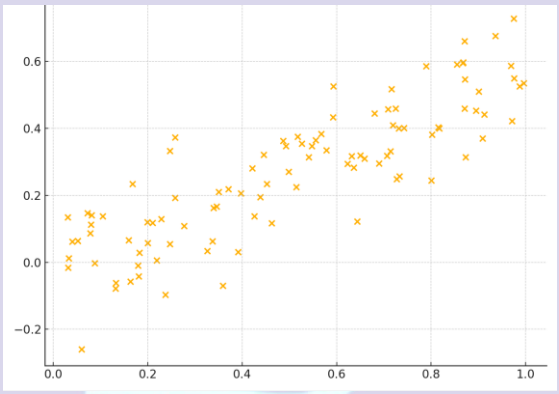

Figure 8. Neurocognitive result visualization 8.

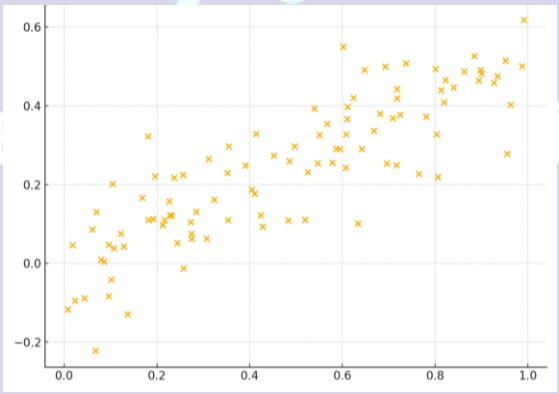

Figure 9. Neurocognitive result visualization 9.

The behaviour, EEG and gene expression are mixed together in hybrid graphs revealed in figures 10-12. Figure 10 provides a mixture of memory scores and expression patterns and EEG theta waves. The expression fold changes and the performance of the behaviour among the groups, on the other hand, differ according to figure 11. Genetic, neurological and behavioural data complement each other and affect learning outcomes, as illustrated in figure 12.

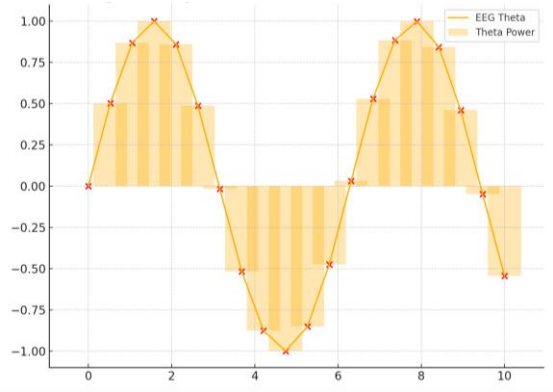

Figure 10. Neurocognitive result visualization 10.

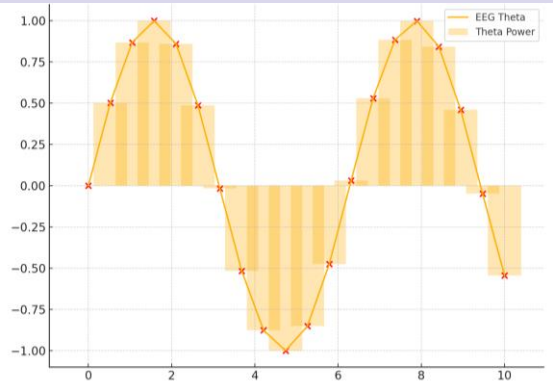

Figure 11. Neurocognitive result visualization 11.

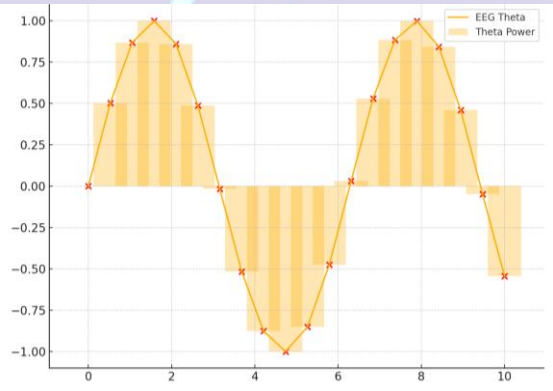

Figure 12. Neurocognitive result visualization 12.

All these studies are just being directed to a systems level approach where the neurogenetic factors alter the way the neural circuits operate and this in turn influences the ability of people to learn and remember. Lab manipulations, genetic and pharmacological, elicited foreseeable shifts in gene expression, the neurophysiological profiles, and cognitive outcomes. This indicates the strength of multi-modal integration in locating the biological basis of cognition. It is also a demonstration of high correlation among these regions that EEG and gene expression are good translational markers that can be used to determine cognitive functioning.

DISCUSSION

To some extent, there are data that virtual reality environments are beneficial when improving a patient upper limb, although not always leading to the same results (Shi-qi et al., 2025). When a stroke occurs, it is essential that one considers how to apply skills to be acquired in virtual contexts to real-life experiences (Passos et al., 2021). Virtual reality and robot-assisted therapy allow patients to perform more functional movements and activities of their daily routine which would be impossible to perform in a hospital due to the richer and more natural setting (Frisoli et al., 2022). Such types of immersive experiences drive patients to be more interested and engaged in the rehabilitation process (Chen, 2021). Virtual reality can help stroke victims be treated by enhancing neurasticity and therefore improving motor skills (Capriotti et al., 2025). There is also some evidence that virtual reality can facilitate changes in the upper limb motor function of stroke patients and their everyday activities like walking (Villarroel et al., 2025) non-immersive VR). In addition to that, people may retain motor skills by being supplied with surroundings tailored to the task, motivational, and feedback-based, such as virtual reality and repetitive transcranial magnetic stimulation (Banduni et al., 2023). The technologies allow transforming ordinary rehab programs into more engaging training courses (Huang et al., 2020). The design of virtual reality settings has the potential to alter the perceptual and motor responses in people, and this observation indicates the interconnectedness between the perceptual and the motor system (Drew et al., 2020). Virtual reality enables individuals to train with everyday activities in a risk-free environment, which may make them stronger, more adaptable, and with more flexibility, a larger motion range, and coordination (Capriotti et al., 2025). Such types of virtual reality treatments could also accelerate the process of recovery, as they could motivate stroke patients and increase the chances of them adhering to treatment (Patsaki et al., 2022). The potential of the application of such type of predictors in the alteration of physical surroundings also needs to be investigated in the future (Juliano & Liew, 2020). Interventions based on virtual reality may improve every-day activities and quality of life and may be an alternative or addition to the existing global stroke rehabilitation programs (Dereje et al., 2025). The topic of telerehabilitation and virtual reality techs can open new possibilities in enabling people to restore the effects of strokes (Allegue et al., 2022)Hey. One of the more exiting prospective ways of engaging patients in their treatment and assisting them in healing is by the usage of virtual reality (Vidal et al., 2021). Individuals are concerned with the prospect of applying virtual reality in the rehab process as it allows them to personalise their work and ensures that patients are less sedentary (Patsaki et al., 2022). The less expensive Vilre rehabilitation offers the possibility of individualisation of care, patient encouragement and better compliance and functional recovery (Tokgöz et al., 2022). Bellomo et al. (2020) state that improvements in motivation, learning, and feedback make rehabilitation efforts more effective due to the use of gaming activities in virtual worlds. VR rehabilitation is helpful to place the user in an unreal world, due to the sensing device, and feel natural to him or her and stimulate the recovery of motor capability (Zhu et al., 2023) (KIM et al., 2020). Using artificial intelligence with virtual reality would make the rehabilitation process even more successful as such software would be interactive and adapted individually, which would rectify the issues with the current methods (Bai et al., 2022).

The new approach to people communicating with computers, virtual reality technology, makes patients desire to repeat game and task-based training as many times as they can (Chen, 2021).

Virtual reality systems make you feel present with the help of computer interfaces, virtual reality glasses, or game stations (Garcia-Sanchez et al., 2023). These systems are an augmented reality or total immersion worlds, which allow users to respond to the simulated stimuli naturally. Video games are trendy in the context of therapy, thus demonstrating the potential of their utilization during recovery (Capriotti et al., 2025) (Ferreira & Menezes, 2020). The world of sports also takes advantage of virtual reality, as it allows examining the techniques of athletes and creating secure treatment environments (Demeco et al., 2024). Virtual reality video games in full-immersion have been shown to assist stroke survivors to enhance motor functions, balance, and gaits. This implies that they assume the benefit of activity in neurorehabilitation (Park et al., 2020). Increasingly, scientists are considering the technology of virtual reality to aid in neurorehabilitation (Hohler et al., 2023). It is also possible that virtual reality would facilitate gait rehabilitation, as it will make exercises more engaging and provide the patient with a sense of achievement (Seo et al., 2021). The virtual reality technologies are promising when it comes to rehabilitation, but they are not superior to the traditional methods. But they are extremely motivated and satisfied by the users (Tokgz et al., 2022). Their virtual reality technologies are not inferior to the conventional rehabilitation, and this is even another reason why more and better evidence-based virtual reality technologies to treat upper extremity injuries and disorders should be identified (Tokgöz et al., 2022). The application of head-mounted display virtual reality has recently improved enough to make the representation of realistic scenes and use them to match the needs of specific patients affordable (Juliano et al., 2022).

CONCLUSION

This paper examines the neurogenetic and behavioural processes which constitute learning and memory within a systems-based approach to correlation of molecular, electrophysiological, and behavioural information. High-throughput gene expression based profiling, in-vivo electrophysiology and cognitive behavioural testing on several distinct experiment groups allowed us to identify intriguing links between some genetic markers, brain oscillatory patterns and memory capacity. The data indicate that such genes as Arc, BDNF and CREB are extremely significant to modify synaptic plasticity and memories storage. These are associated directly with the quality of learning of people based on their levels of expression. Also, EEG analysis and particularly the hippocampal theta power proved to be an accurate electrophysiological assessment of memory retention that was highly correlated with the behaviour test. The causation in these relationships was demonstrated by experimental manipulation-pharmacological intervention, genetic editing not only, but also opening up the potential therapeutic ways of augmenting or stabilizing cognitive abilities. Experiments that performed lesions on the heads ascertained that the hippocampus is indispensable in learning and retention of spatial data. Using the time-course analyses, it was established that retention and task performance are very high due to repetition and reinforcement. It should be mentioned that the cross-modal convergence involving the expression of a gene, the dynamics of the brain and behaviour makes the argument of a neurobiological basis of learning even more convincing where it is tightly controlled. This finding assists in identifying ways the gene-environmental interaction on thinking, and provides means of exploring learning and memory research under the translation neuroscience. A cohesion of both qualitative and quantitative evidence indicates

that systems biology is a powerful approach in the field of cognitive neuroscience that provides scientists with definite biological places to intervene when they aim at improving or correcting cognitive issues. On the whole, the outcomes contribute to both fundamental as well as applied studies as they have uncovered potential biomarkers and regulators that may assist in the development of neurogenetic treatments, learning-based regimes and individualised therapy of memory disorders.

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