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Brain-on-a-Chip Devices and Their Potential for Psychiatric Electrophysiology: A Scoping Review

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Abstract

Introduction: Circuit dysfunction is central to schizophrenia, bipolar disorder, and major depressive disorder, but current models cannot connect human-relevant microcircuit activity to the electrophysiological abnormalities seen clinically. While changes in alpha and gamma activity, coherence, and cross-frequency coupling are well-established biomarkers, traditional in vitro systems lack the controlled architecture needed to study their cellular origins. Brain-on-a-chip platforms offer engineered control over neural geometry and connectivity, making them a promising tool for bridging this gap. This scoping review evaluates how these systems have been used to study neuronal electrophysiology and their relevance to psychiatric biomarker research.

Methods: A structured database search, combined with manual screening and citation tracing, identified studies using chip-based neural platforms that reported direct electrophysiological recordings. Non-neural systems, organoid-only studies, and models lacking electrophysiology were excluded. Four studies met inclusion criteria.

Results: Experimental platforms primarily reported spikes, firing rates, bursting, and basic correlation measures. None captured low-frequency activity, oscillatory power, synchrony, coherence, or local-field-potential-like signals that define psychiatric electrophysiological biomarkers. Most studies used rodent neurons or generic excitatory stem-cell-derived neurons, and none included defined interneuron subclasses required for modelling gamma oscillations or balanced circuit dynamics.

Discussion: Findings show that current brain-on-a-chip systems are architecturally advanced but electrophysiologically limited. Their outputs capture excitability but not the mesoscale dynamics relevant to psychiatric biomarkers.

Conclusion: Progress in this field will require integration of patient-derived neurons, defined inhibitory interneuron types, and analytical pipelines aligned with clinical electrophysiology to enable brain-on-a-chip platforms to model microcircuit dysfunction in psychiatric illness.

Keywords: brain-on-a-chip; organ-on-a-chip; microphysiological systems; electrophysiology; neural biomarkers; psychiatric disorders; schizophrenia; major depressive disorder; bipolar disorder

Introduction

Schizophrenia, major depressive disorder (MDD), and bipolar disorder (BD) are among the most challenging psychiatric conditions, marked by chronicity, complex symptoms, and limited understanding of their underlying mechanisms [3, 5, 6]. Without objective diagnostic biomarkers, clinicians still mainly depend on subjective assessments instead of measurable, biology-based indicators. Traditional research methods, such as post-mortem studies, animal models, and neuroimaging, have improved our understanding of psychiatric diseases. However, they are not enough to address the human-specific, developmental, and microcircuit-level issues that contribute to these disorders [7, 11]. There is a growing need for experimental systems capable of capturing the circuit dysfunctions believed to drive psychiatric symptoms.

Electroencephalography (EEG) is one of the most commonly used tools for studying brain function in psychiatric disorders. Altered alpha power, gamma synchrony, long-range coherence, and cross-frequency coupling are repeatedly reported across schizophrenia, BD, and MDD, reflecting disruptions in excitatory–inhibitory balance and interneuron-mediated synchrony [4–5, 9]. These oscillatory abnormalities are some of the most reliable and reproducible biomarkers we have. They provide insight into the behavior of circuits at a mesoscale level. However, EEG does not show the cellular or synaptic processes that create these rhythms. This gap between mesoscale biomarkers and microcircuit function limits mechanistic interpretation and slows biomarker development.

Compounding this gap, spectral biomarkers show limited consistency in methods. A recent review of schizophrenia, MDD, BD, Obsessive–Compulsive Disorder, and ADHD reveals significant differences in reported changes in delta, theta, alpha, and beta bands. These differences stem from inconsistencies in preprocessing,

referencing, and even frequency-band boundaries [13]. This variability highlights the field's reliance on analytical practices instead of stable biological markers. To address this issue, we need new in vitro systems that can link EEG-scale irregularities to microcircuit-level mechanisms.

Human induced pluripotent stem cell (hiPSC) models, 2D neuronal cultures, and brain organoids have emerged as promising options for bridging this translational gap. These platforms allow for the study of patient-specific genetic risk, developmental issues, and synaptic problems in human-derived neurons [5, 8, 11]. Cerebral organoids, in particular, can show structured oscillations, long-range synchronization, and preterm-like EEG patterns. This offers valuable insights into how circuits develop [1–2]. However, they lack consistent architecture, controlled connectivity, and precise microenvironments. Their electrophysiology is also constrained by reliance on planar MEAs, which cannot map activity to defined anatomical structures.

Brain-on-a-Chip Platforms: Microengineered Control Over Neural Circuitry

Brain-on-a-Chip (BoC) systems offer an alternative, complementary path. These platforms use microfabrication, microfluidics, and patterned substrates to impose geometric and functional organisation on neuronal cultures. Unlike organoids, BoC systems specify the architecture: axons can be directed through microchannels, somata can be segregated into defined compartments, and unidirectional or bidirectional connections can be engineered with high reproducibility [1, 3, 12]. Microelectrode arrays (MEAs), Complementary Metal-Oxide-Semiconductor sensors, and other embedded biosensors enable high-resolution electrophysiological monitoring of spiking, bursting, synchrony, and, in some cases, local-field-potential-like activity. This combination of structural control and functional readout positions BoCs as a uniquely powerful tool for interrogating how defined circuit motifs generate emergent electrophysiological behaviours.

Linking Electrophysiology Across Scales: From EEG to MEA-Based BoCs

EEG abnormalities in psychiatric disorders result from interactions within and between distributed cortical microcircuits. To model these features in vitro, systems must capture not only the intrinsic excitability of neurons but also synchronisation at the network level, long-range connections, and coordinated burst propagation. MEA-integrated BoC platforms are well suited for this task. Their engineered microarchitecture allows researchers to recreate patterns like feedforward inhibition, recurrent excitation, or connections between the prefrontal cortex (pfCx), hippocampus (Hip), and amygdala (Amy). They can then examine how disturbances affect synchrony and coherence, which relate directly to EEG and local field potential (LFP) readouts. Unlike organoids, where structure varies and connections are unpredictable, BoCs provide the control needed to link microcircuit mechanisms with mesoscale biomarkers.

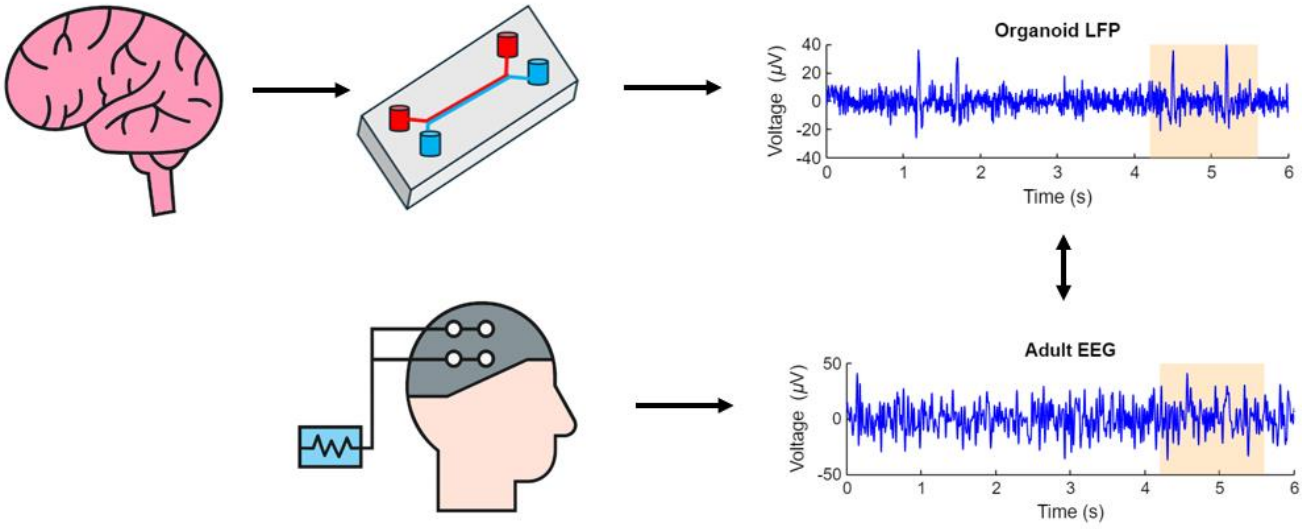

Figure 1. Linking brain-on-a-chip (BoC) neural activity to clinical electrophysiology. (Top) Engineered BoC platforms model simplified neural microcircuits and allow recording of local field potentials (LFPs), which reflect coordinated population activity. (Bottom) In vivo, electroencephalography (EEG) captures analogous oscillatory dynamics at the macroscale. Despite differences in spatial scale and signal origin, shared features such as burst timing, frequency content, and network synchrony enable conceptual comparison between BoC LFPs and human EEG biomarkers relevant to psychiatric disorders.

A Gap in the Literature

Although reviews have examined psychiatric EEG biomarkers, organoid electrophysiology, and the engineering basis of BoC systems, no synthesis has investigated the role of BoC platforms in modeling electrophysiological biomarkers related to psychiatric disorders [1–5, 8–9, 11–13]. Current BoC studies remain scattered and vary widely in biological complexity and electrophysiological readouts. With the growing interest in engineered neural microcircuits and the urgent need for models that can translate circuit ideas into useful biomarkers, a focused scoping review is both timely and needed. This review aims to evaluate how current BoC systems have been used to study neural electrophysiology, assess their relevance to psychiatric biomarker research, and identify future directions to advance the field.

Methods

Search Strategy

This scoping review examined studies from the past 15 years that used BoC or microengineered neural platforms to record neuronal electrophysiology relevant to biomarkers implicated in schizophrenia, BD, or MDD. Searches were conducted in PubMed (n=1) and Google Scholar (n=7), supplemented by targeted manual hand-searching of academic journals (n=29)citation chaining. The Boolean query used was: ("brain-on-a-chip" OR "organ-on-a-chip" OR "brain-on-chip" OR "organ-on-chip") AND ("psychiatric disorder*" OR schizophreni* OR "bipolar disorder" OR depress*) AND (electrophysiolog* OR "local field potential*" OR LFP OR MEA OR "microelectrode array" OR "field potential*")*. Searches were limited to English. Following initial screening, one study identified via database searching met the inclusion criteria, alongside three additional studies identified through manual searching of academic journals including Frontiers in Neuroengineering, Scientific Reports and npj Microgravity. Because BoC electrophysiology studies rarely use psychiatric terminology despite methodological relevance, manual searching was used to capture relevant studies that may not be indexed under psychiatric disease terms. A supplementary sensitivity search incorporating organoid-on-a-chip terminology did not identify additional eligible BoC studies under the defined inclusion criteria.

Eligibility Criteria

Studies were eligible if they met one or more of the following criteria:

1. Used a neural BoC or microfluidic neuroengineering platform in which the chip constituted the primary experimental architecture (2D or 3D; human or animal neurons).
2. Reported electrophysiological data obtained directly from the BoC system, including but not limited to extracellular spikes, firing rates, bursting, or multi-electrode recordings.
3. Demonstrated engineered microcircuit features such as compartmentalisation, guided axonal pathways, patterned connectivity, or integrated biosensing relevant to neural function that were imposed by the chip design.
4. Provided methodological or technical innovations that directly enable neural electrophysiology within BoC platforms (e.g., microelectrode integration, microchannel design).

Studies were excluded if they met any of the following criteria:

1. Used organoids, spheroids, or brain aggregates or organoid-on-chip systems unless the chip acted as a fully engineered BoC by actively imposing microphysiological constraints (e.g., active microfluidics or imposed connectivity), rather than serving as a passive holder or recording interface.
2. Reported non-neural organ-on-a-chip systems or microfluidic devices without neuronal culture.
3. Used standard 2D cultures on planar MEAs or organoid systems mounted on MEAs without microengineered BoC features.
4. Focused on non-psychiatric diseases without mechanisms relevant to circuit-level electrophysiology.
5. Lacked electrophysiology entirely or only used molecular/structural outcomes.
6. Were duplicates, non-English, or lacked accessible full texts.

Study Selection

Records identified through database searching and journal-specific manual searching of core neuroengineering and lab-on-a-chip venues(n = 37) were screened for eligibility. After removing two duplicates, 35 unique records remained. These 35 records were screened at the title and abstract level, of which 31 were excluded for lacking neuronal culture, lacking electrophysiology, or not involving BoC platforms. Four full-text articles were assessed for eligibility, all of which met the inclusion criteria and were included in the review. The study selection process is summarised in the PRISMA-style flow diagram (Figure 2).

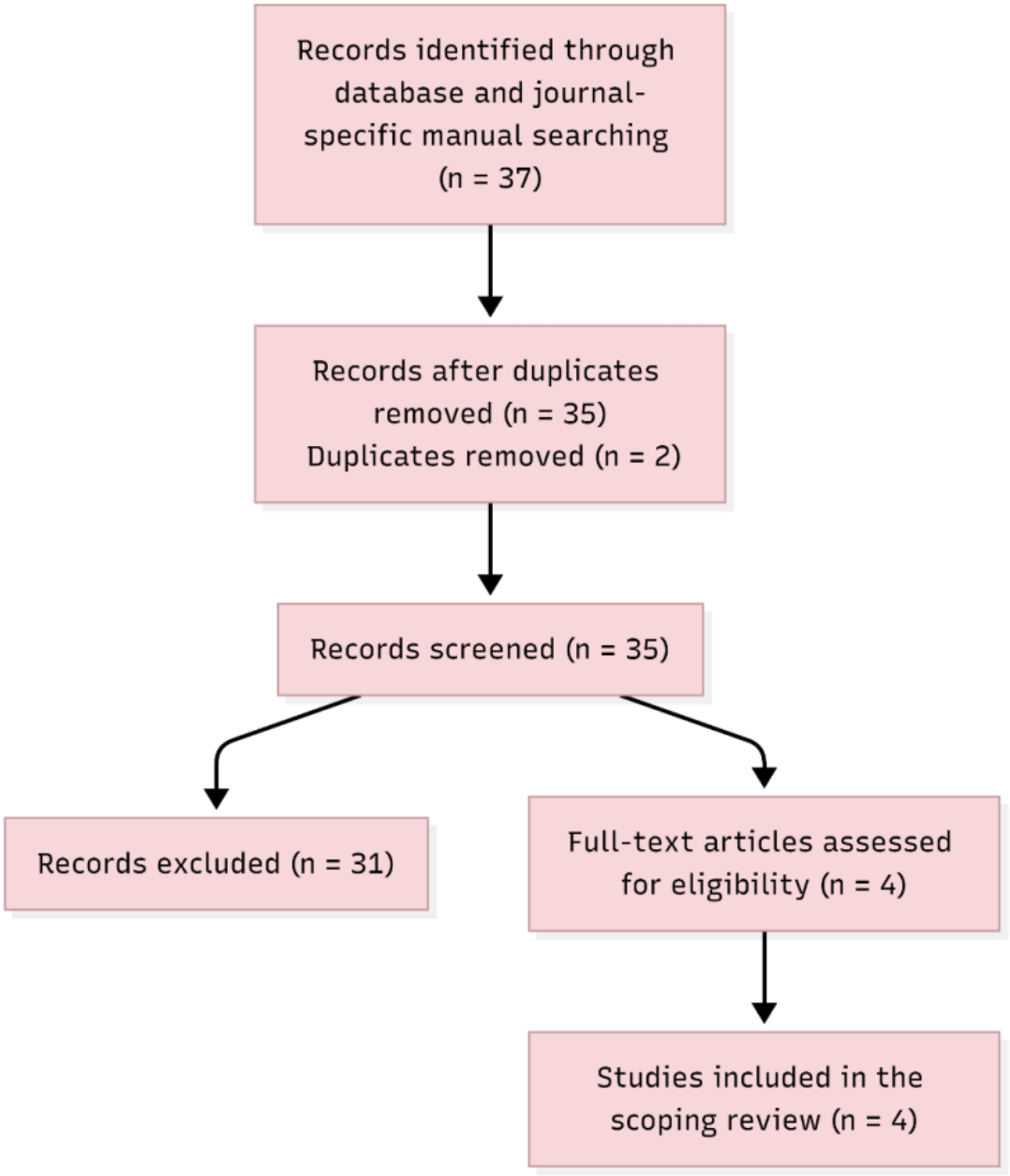

Figure 2. PRISMA-style flow diagram summarising the study selection process. A total of 37 records were identified through database and journal-specific manual searching. After removal of duplicates (n = 2), 35 records underwent title and abstract screening, of which 31 were excluded. Four full-text articles were assessed for eligibility, and all of these met the inclusion criteria and were incorporated into the scoping review.

Data Extraction

Data were charted into a structured Excel workbook. Items captured were: citation; disorder studied; sample size; culture/media; timepoints; electrophysiology modalities (MEA, LFP/oscillations, spectral exponent/1/f, spikes/bursts); key finding; biomarker(s); inclusion decision; study type (experimental/review); and interneuron subclasses where reported.

Results

Characteristics of Included Studies

Four experimental BoC studies met the inclusion criteria [14–17]. In all studies, sample sizes were modest, usually with n = 3-4 devices and 6-60 active electrodes based on the device design. None of the studies specifically modeled a psychiatric disorder, but one study used the antagonist phencyclidine (PCP) to create a schizophrenia-related

change in vitro [17]. All cultures included a mix of excitatory and inhibitory neurons, but none identified specific interneuron subclasses, such as parvalbumin (PV), somatostatin (SST), or Vasoactive Intestinal Peptide (VIP).

Table 1. Overview of studies

Study	Neuronal Source	Device / Culture	Electrophysiology	Psychiatric Relevance
Aydogmus et al. [14]	hiPSC-derived cortical neurons	Silicon–polymer BoC with integrated microelectrodes and pH sensor	Single-electrode spikes; increased firing from ~0.3 to ~1.4 Hz	Excitability only; no network measures or oscillations
Padilla et al. [15]	hiPSC glutamatergic + rat hippocampal neurons in 3D Matrigel	3D hydrogel BoC with Neuropixels probe	113–180 spikes/60s; stable after microgravity; increased VGLUT1/2	Only excitatory neurons; no inhibitory subclasses; no network metrics
Kanagasabapathi et al. [16]	Primary rat cortical neurons (E18)	Dual-compartment microfluidic MEA with 10 µm channels	Spikes, bursts; tetrodotoxin isolation; inter-compartment cross-correlation	Strong architecture control but no oscillations or interneuron data
Dauth et al. [17]	Rat pfCx, Hip, Amy neurons	Tri-region BoC with guided axonal pathways	Spikes, bursts, cross-regional synchrony; PCP perturbation	Anatomical realism but no LFP/oscillatory measures

Neuronal Sources and Culture Paradigms

Two studies used hiPSC-derived neurons. Aydogmus et al. cultured hiPSC-derived cortical neurons for 21 days, which included 7 days of differentiation and 14 days of maturation, on a silicon polymer BoC [14]. Padilla et al. combined hiPSC-derived glutamatergic neurons with rat hippocampal neurons in a 3D Matrigel hydrogel, recording data up to days 8 to 11 [15]. The other studies used rat primary neurons: embryonic day 18 cortical neurons grown for 17 to 35 Day In Vitro (DIV) in a dual-compartment device, and pfCx, Hip and Amy neurons cultured for 14 to 18 DIV in a microengineered multiregional system [16–17]. Overall, these time frames overlap with the developmental period in which dissociated cortical networks typically shift from sparse firing to organized bursting, which occurs around DIV 14 to 21, allowing for the assessment of maturing network electrophysiology.

Device Architectures and Electrophysiological Readouts

Despite the significant differences in design, all platforms used extracellular electrophysiology to study neuronal activity. The devices included planar silicon-polymer hybrids with integrated microelectrodes and pH-sensing FETs, 3D hydrogel-based systems that used probes for depth-resolved recording, dual-compartment MEA platforms that guided axonal projections between chambers, and multiregional setups that modeled prefrontal, hippocampal, and amygdala connectivity [14–17]. Across these systems, recordings were taken at 10 to 25 kHz, focusing on high-frequency spike activity. Common metrics included firing rate, spike count, burst occurrence, and, in one study, inter-regional cross-correlation to measure functional coupling [17]. Notably, no study recorded or analyzed LFPs, oscillatory power or EEG-analog biomarkers, largely due to high-pass filtering of signals above about 200 Hz.

Functional Network Activity Across Platforms

All four studies reported spontaneous neural activity that matched early network development [14–17]. HiPSC-derived cortical neurons showed increased firing and burst-like responses after applying picrotoxin, which demonstrated sensitivity to inhibitory blockade [14]. In 3D hydrogel cultures, spike patterns stayed stable over several days and persisted after suborbital flight [15]. Dual-compartment rat cortical cultures displayed progressively coordinated bursting by DIV 21 to 28, showing the emergence of functional excitatory and inhibitory loops [16]. In multiregional circuits, significant cross-correlation between prefrontal, hippocampal, and amygdala compartments indicated successful engineering of long-range functional interactions [17]. Despite these signs of network activity and responsiveness, all functional outputs remained limited to spike-based measures, with no low-frequency or oscillatory signatures.

Discussion

Aydogmus et al. introduce a silicon-polymer BoC that combines pH sensing with single-electrode recordings from hiPSC-derived cortical neurons that matured for three weeks on-chip [14]. Spontaneous spikes were sampled at 10 kHz with a 200 Hz high-pass filter. The application of the

$GABA_A$ antagonist picrotoxin (50 µM) increased the mean firing rate from about 0.3 to 1.4 Hz across three recordings [14]. This shows that excitability can be modified with drugs and that there is stable electrical performance across repeated measurements. The use of human cortical neurons and a reliable shift in firing rate due to drug application are notable strengths. They provide a basis for pharmacological screening based on excitability, which is relevant to psychiatric research. However, the electrophysiology is confined to a single microelectrode. This limit prevents the assessment of network-level properties like synchronized bursting, functional connectivity, or spatial propagation of activity. The strong high-pass filtering also eliminates low-frequency components. This makes it impossible to measure theta, alpha, beta, or gamma oscillations, which are key electrophysiological markers for schizophrenia, BD, and MDD. Overall, Aydogmus et al. offer a solid proof-of-concept platform, but it currently lacks the complexity needed to model electrophysiological signatures relevant to psychiatric conditions [14].

Padilla et al. describe a BoC that combines a Matrigel-embedded neuronal construct with a high-density Neuropixels probe, capture hundreds of channels simultaneously [15]. HiPSC-derived glutamatergic neurons showed spontaneous spikes within 24 hours and kept their activity for eight days, producing ~113–180 spikes per 60 seconds with mean amplitudes of ~270–295 µV [15]. Spatial decay of spike amplitude across neighbouring electrodes confirmed genuine single-unit activity rather than noise. However, the system only contains excitatory glutamatergic neurons and lacks GABAergic interneuron subclasses. This limits its ability to model excitatory/inhibitory balance, synchrony, or oscillatory patterns important for psychiatric disorders. Electrophysiology in this platform is limited to spike detection and waveform sorting, with no analysis of network dynamics or spectral features. While not specifically designed for psychiatric research, Padilla et al. present a highly stable, high-density 3D electrophysiology platform that lays a strong foundation for future BoC studies focused on psychiatric biomarkers [15].

Kanagasabapathi et al. developed an early dual-compartment neurofluidic MEA system that demonstrated how physically segregated yet neurally connected populations can generate coherent electrophysiological activity [16]. Primary rat cortical neurons were cultured for up to 35 days, producing spontaneous spiking and increasing network bursting during the third week, consistent with typical maturation [16]. A major strength is the demonstration of true fluidic isolation: application of tetrodotoxin to one compartment abolished all spiking locally while leaving activity in the opposite compartment unaffected, with recovery following washout. This confirms the biological origin of signals and validates the platform for region-specific pharmacological perturbation [16]. A second strength is the evidence of functional connectivity across microchannels, with significant inter-compartment cross-correlations across 17 devices indicating formation of effective synaptic projections [16]. However, electrophysiology is limited to spike rates, bursting, and correlation metrics. It does not include oscillatory, spectral, or LFP-like measures. The cultures also lacked interneuron subclass resolution, preventing links to psychiatric biomarkers such as gamma abnormalities or excitation–inhibition imbalance. Despite these limitations, the study provides an important early example of long-term, pharmacologically isolatable, functionally connected microcircuits on-chip, offering conceptual groundwork for future BoC models of disrupted cortical coupling in psychiatric disorders.

Dauth et al. created a multiregional BoC that contains physically separated but axonally connected neurons from the pfCx, Hip and Amy [17]. The primary rat neurons maintained specific characteristics depending on their region in vitro, showing different ratios of GAD^+ inhibitory and $VGLUT^+$ excitatory neurons. These cellular differences showed up electrophysiologically; Amy networks had lower spontaneous firing and less metabolic activity compared to pfCx and Hip [17]. A major advantage of this platform is its ability to show functional long-range connections. Patterned axonal guidance allowed activity to spread across regions, and cross-correlation analysis showed significant synchrony between regions, which indicated the formation of effective synaptic connections [17]. The platform's importance for psychiatric research is enhanced by its pharmacology. When PCP was applied, it caused specific changes in spiking and bursting in different regions, and disturbances in one area affected activity in the connected regions. However, all neurons were derived from rodents, subclasses of interneurons were not defined, and the electrophysiology focused only on spikes, bursts, and cross-correlation without looking at oscillations or LFP-like measures. So, while the system is anatomically detailed, it models circuit interactions instead of the electrophysiological markers typical of psychiatric disorders.

Cross-Study Synthesis

Across the four BoC systems [14–17], electrophysiology is dominated by single-unit or multi-unit spikes, firing rates and bursting, with limited engagement with true LFPs or oscillatory metrics. This contrasts with organoid and clinical literature, where network-level oscillations, phase–amplitude coupling and band-limited power are already standard readouts for psychiatric disease [1–4, 13].

In the included studies identified, currentBoCs therefore sit in a "pre-biomarker" space: they offer excellent control over architecture and robust excitability readouts, but they do not yet implement the spectral, coherence or cross-frequency analyses that would allow direct mapping to EEG/MEG biomarkers in schizophrenia, BD and MDD [3–4, 13].

Limitations of Current BoC Platforms

Biologically, all four BoC studies rely on either rodent primary neurons [16–17] or generic hiPSC lines biased towards excitatory glutamatergic neurons [14–15], without deliberate introduction of PV/SST/VIP interneuron subclasses or systematic control of E/I balance, despite strong evidence from organoid and iPSC work that interneuron dysfunction and E/I imbalance are central to psychiatric pathophysiology [5–9,12]. Sex of donors or animals is rarely reported or analysed, although large EEG/meta-analytic datasets show that spectral abnormalities in psychiatric disorders are modest, heterogeneous and likely moderated by demographic factors including sex [4, 13]. Analytically, BoC readouts stop at spikes, bursts and simple correlations; no study reports canonical band power, coherence, or theta–gamma abnormalities that could be compared quantitatively to the clinical EEG/LFP literature [3–4, 13].

Organoid-On-Chip vs "True" BoC

Organoid-on-chip approaches frequently use chips as passive scaffolds or MEA carriers rather than active microphysiological systems, creating terminological ambiguity rather than overlap in device architecture. Osaki et al. culture reciprocally connected cerebral organoids on a PDMS–MEA chip and demonstrate delta-band LFPs, complex bursting and optogenetically inducible short-term plasticity, but the chip mainly provides mechanical support and axon guidance rather than region-specific microfluidics or controlled inputs [18]. In contrast, BoC platforms [14–17] explicitly engineer compartmentalisation, connectivity and perfusion but currently underperform organoid systems in terms of oscillatory richness and biomarker-like readouts [1–2, 5–11]. The paper by Osaki et al. is therefore best seen as an organoid-on-chip reference point highlighting what BoCs could achieve if similar oscillatory complexity were coupled to engineered architecture [18].

Future Directions

Future work should move BoCs from pre-biomarker architecture to bona fide biomarker platforms by: (i) incorporating human, sex-balanced, patient-derived iPSCs with defined interneuron subclasses; (ii) redesigning electrode and analysis pipelines around clinically used metrics (band power, coherence, cross-frequency coupling, intermittency); (iii) integrating organoid-like 3D architecture and self-organised oscillations with BoC-level control of connectivity and fluidics; and (iv) explicitly benchmarking BoC readouts against clinical EEG/LFP effect sizes and variability in SCZ/BD/MDD [1–13, 18].

Conclusions

This scoping review examined how current BoC platforms have been used to investigate neuronal electrophysiology and evaluated their relevance for modelling biomarkers implicated in schizophrenia, BD, and MDD. While BoCs provide precise control over microcircuit architecture, including compartmentalization, guided axonal pathways, and integrated microfluidics, the electrophysiological outputs of current systems are still limited. Across all four included studies, recordings were restricted to spikes, firing rates, bursting, and basic correlation analyses. No work reported low-frequency activity, oscillatory power, synchrony or coherence, all of which are central EEG/LFP biomarkers in psychiatric illness. As a result, within the included studies identified, current BoCs should be considered pre-biomarker platforms: structurally sophisticated but functionally incomplete from a psychiatric electrophysiology perspective.

Biological constraints further limit translational relevance. These include the use of rodent models or generic iPSC-derived cultures, the absence of specific interneuron subclasses, and the lack of sex-specific or patient-derived neuronal sources. Analytical limitations, especially the lack of EEG-aligned metrics, also prevent meaningful comparisons with clinical electrophysiological signatures.

This review identifies several avenues for progress. Future BoCs must incorporate patient-derived, sex-balanced stem cell lines; introduce PV/SST/VIP interneuron populations; and combine microengineered architectures with 3D cytoarchitectures capable of generating self-organised oscillations. Equally important is the adoption of analytical frameworks that quantify band power, coherence, and cross-frequency coupling, enabling direct comparison to clinical EEG/LFP biomarkers. Advancing BoCs along these lines will move the field from architectural demonstrations toward platforms capable of revealing mechanistic links between microcircuit dysfunction and psychiatric electrophysiology.

List of Abbreviations

ADHD: attention-deficit/hyperactivity disorder
Amy: amygdala
BD: bipolar disorder
BoC: brain-on-a-chip
DIV: days in vitro
E/I: excitatory–inhibitory
EEG: electroencephalography
FET: field-effect transistor
GABA: gamma-aminobutyric acid
GAD: glutamate decarboxylase
Hip: hippocampus
hiPSC / iPSC: human induced pluripotent stem cell / induced pluripotent stem cell
LFP: local field potential
MDD: major depressive disorder
MEA: microelectrode array
PCP: phencyclidine
pfCx: prefrontal cortex
PV: parvalbumin (interneuron subclass)
SST: somatostatin (interneuron subclass)
VGLUT: vesicular glutamate transporter

VIP: vasoactive intestinal peptide (interneuron subclass)

Conflicts of Interest
The author declare that they have no conflict of interests.

Ethics Approval and/or Participant Consent
This study is a scoping review of previously published literature and did not involve the recruitment of human participants, the use of identifiable personal data, or any experimental procedures. As such, ethics approval and participant consent were not required.

Authors' Contributions
MMF: All work contributing to the generation of this manuscript was carried out by MMF. MMF conducted all literature searches, screened and selected studies, extracted and analysed all data, generated tables and figures, and interpreted the findings. MMF drafted, revised, and finalised the manuscript for publication.

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