GLOBAL NEUROPSYCHIATRY GROUP

Monthly Newsletter

CONNECTING MINDS ACROSS NEUROLOGY AND PSYCHIATRY

July 2026 | Clinical discussion, research highlights, polls and events

WELCOME TO THE GNG NEWSLETTER

Warm greetings to colleagues, trainees and researchers across our international community. July brought together thoughtful clinical problem-solving, critical appraisal of emerging evidence, and a lively exchange across the neurology-psychiatry interface. This round-up distils those conversations into practical themes while protecting patient confidentiality.

The month's discussions ranged from catatonia and medication sensitivity to post-encephalitis recovery, dementia biomarkers, acquired brain injury, chronic traumatic encephalopathy and the biological heterogeneity of schizophrenia. We also celebrated new members, congratulated Professor Jon Stone on becoming President of the Functional Neurological Disorder Society, and concluded the month with a stimulating GNG academic meeting.

A NOTE TO READERS

This newsletter summarises professional discussion for education. It is not a clinical guideline and does not replace patient-specific assessment, local policy, prescribing information or multidisciplinary advice.

INSIDE THIS ISSUE

Acute behavioural change, neuroleptic sensitivity and persistent catatonia

A late-onset presentation prompted careful separation of precipitant, complication and perpetuating factors.

The discussion centred on an older adult with abrupt mania/psychosis following a medication change, followed by restraint, high creatine kinase, infection, confusion, rigidity and marked psychotropic sensitivity. Contributors considered whether the initial event and the subsequent syndrome represented different stages of the same illness - or an iatrogenic cascade superimposed on a vulnerable brain.

DIFFERENTIAL FRAMEWORK

- Medication-precipitated affective psychosis, with age, substance exposure and cerebral vulnerability considered as modifiers.
- Catatonia or malignant catatonia, including the significance of partial response to benzodiazepines and whether ECT should be considered.
- Neuroleptic malignant syndrome or a prolonged post-NMS state, balanced against alternative explanations for raised CK such as restraint and injections.
- Delirium and infection-related encephalopathy, autoimmune encephalitis, seizures/non-convulsive status, dementia with Lewy bodies, frontotemporal dementia and rapidly progressive neurological disease.
- An incidental frontal meningioma versus a causally relevant lesion - requiring clinical-radiological correlation rather than attribution by location alone.

PRACTICAL APPROACH DISTILLED FROM THE GROUP

- Reconstruct the timeline carefully: baseline function > drug exposure > autonomic/motor change > infection > treatment response.
- Use repeated structured assessment of catatonia (for example, the Bush-Francis scale) and document response to benzodiazepines.
- Review antipsychotic burden and sedation; avoid adding treatments that obscure the phenotype unless risk requires it.
- Complete an appropriately broad organic work-up, with EEG and advanced imaging considered when clinically indicated.
- Escalate early to multidisciplinary review; ECT remains a key option when catatonia persists or becomes life-threatening.

OUTCOME SHARED WITH THE GROUP

The patient subsequently improved as antipsychotics were reduced and benzodiazepine treatment continued. Autoimmune CSF markers were reported negative, and further imaging was planned. The update reinforced the value of staged reassessment rather than premature diagnostic closure.

Case details have been generalised. Treatment decisions remain with the treating team.

After encephalitis: neuropsychiatric recovery is part of recovery

July discussions linked individual clinical challenges with new evidence on long-term psychiatric and behavioural outcomes.

A systematic review and meta-analysis shared in the group found that depression, anxiety, disinhibition and emotional instability each affect a substantial minority of encephalitis survivors. The paper brought a recurring clinical problem into focus: survival is not the endpoint, and neurological follow-up that does not measure mental health may miss much of the continuing burden.

RESEARCH SIGNAL

Across 101 studies and 4,703 patients, pooled estimates were 26.9% for depression, 22.8% for anxiety, 20.5% for disinhibition and 22.9% for emotional instability. Heterogeneity was substantial, supporting the need for standardised outcomes and prospective cohorts.

CLINICAL DISCUSSION: ANXIETY, AMNESIA AND EPILEPSY

A separate case involved anxiety, memory impairment and misidentification symptoms after recurrent HSV encephalitis in a person with epilepsy and intellectual disability. The group emphasised that treatment choices could not be separated from seizure control, antiseizure-drug effects, cognition, metabolic vulnerability and sensitivity to antipsychotics.

- Review whether antiseizure medication may be contributing to mood, anxiety or behavioural symptoms before layering additional psychotropics.
- Define the dominant target: anxiety, psychosis, episodic memory impairment, executive dysfunction or environmental distress may require different strategies.
- Where cognition drives anxiety, cognitive rehabilitation, environmental adaptation, carer education and compensatory supports remain central - even when medication is considered.
- Use off-label cognitive enhancers only after an explicit benefit-risk discussion and with careful seizure monitoring.
- Prepare families for long recovery trajectories and screen proactively for psychiatric symptoms rather than waiting for crisis presentation.

Source: Watson et al., Brain Communications (2026)

Movement at rest in dementia: naming the phenomenon before treating it

A poll about apparent akathisia after donepezil opened a broader discussion about phenomenology, polypharmacy and diagnostic uncertainty.

In the scenario, a person with Alzheimer disease and behavioural symptoms developed repetitive lower-limb movement after donepezil was added to long-standing venlafaxine and risperidone. Most respondents selected a drug combination rather than a single culprit. Contributors then questioned whether the observed behaviour was truly akathisia.

PHENOMENOLOGY CHECKLIST

- Is there a subjective inner restlessness or urge to move?
- Is the movement goal-directed (for example, repetitive activity) or non-goal-directed?
- Does movement relieve discomfort?
- Are there racing thoughts, anxiety or agitation that may mimic motor restlessness?
- Are hyperreflexia, clonus, diaphoresis or autonomic signs present?
- Could this be stereotypy, restless legs, wandering, dyskinesia or serotonergic restlessness?

WHY THE DISTINCTION MATTERS

In dementia, reduced ability to report subjective distress makes akathisia harder to diagnose. Temporal association is informative but does not prove causation; medication review, observation across contexts and cautious dose adjustment may clarify the picture.

RELATED JULY BIOMARKER DEBATE

The group also explored how to interpret a negative amyloid PET in a clinically convincing MCI-to-Alzheimer trajectory. Most poll respondents felt it should substantially reduce - but not absolutely eliminate - diagnostic confidence. Discussion highlighted the value of quantitative context such as centiloid distance from the positivity threshold, disease stage, alternative pathologies and follow-up over time.

Acquired brain injury: treat the cognitive domain, not a generic “memory problem”

A community poll showed preference for stimulants and modafinil, but the discussion quickly moved beyond rankings.

OPTION	VOTES	SHARE
Methylphenidate (Ritalin)	7	36.8%
Modafinil	5	26.3%
Memantine	4	21.1%
Acetylcholinesterase inhibitors	2	10.5%
Dexamphetamine	1	5.3%
Bupropion / guanfacine / clonidine	0	0%

N = 19 responses. This was an informal professional poll, not comparative evidence of efficacy.

DISCUSSION TAKEAWAYS

- Start with neuropsychological formulation: episodic memory, attention, processing speed, initiation and executive dysfunction are not interchangeable targets.
- Match treatment to lesion pattern, functional goals and the person’s rehabilitation plan.
- Account for seizure risk, sleep, cardiovascular risk, anxiety, agitation and interactions with antiseizure medication.
- Combine pharmacological trials with cognitive rehabilitation, environmental supports and measurable functional outcomes.
- Define a time-limited trial and stopping rule; improvement should be demonstrated in daily function, not assumed from prescription.

What the group chose - and the clinical questions beneath the vote

1. New movement after adding donepezil

Combination of venlafaxine, risperidone and donepezil: 17/27 (63.0%); risperidone: 9/27 (33.3%); donepezil: 1/27 (3.7%). The discussion cautioned against converting plurality into causality and prioritised direct phenomenological assessment.

2. Next diagnostic step in suspected logopenic aphasia

Plasma p-tau217: 6/10 (60%); amyloid PET: 2/10 (20%); CSF Aβ42/phospho-tau: 1/10 (10%); wait and watch: 1/10 (10%). The result reflects the growing appeal of accessible blood biomarkers, while availability and local pathways remain decisive.

3. Negative amyloid PET in a strong MCI-to-AD trajectory

Substantially reduces confidence but does not rule out AD: 10/12 (83.3%); rules out AD: 1/12; modestly lowers probability: 1/12. Contributors stressed quantitative interpretation, timing and consideration of non-Alzheimer pathology.

READING POLLS RESPONSIBLY

These snapshots reveal where discussion is concentrated; they do not establish consensus standards. Small, self-selected samples and differences in local access limit generalisability.

EMERGING BIOMARKER CONTEXT

A July paper reported that plasma p-tau217 stratified future risk of progression among cognitively unimpaired older adults across six cohorts. The promise is clinically important, but risk communication must distinguish pathology, probability and an individual timeline.

SCHIZOPHRENIA BEYOND A SINGLE PATHWAY

Two papers challenged one-size-fits-all accounts of psychosis.

WIDESPREAD SYNAPTIC DENSITY LOSS

Using [11C]UCB-JPET,Chopraand colleagues reportedwidespreadlower synaptic density in schizophrenia, with moderate-to-large effect sizes and prominent involvement of frontal, temporal, cingulate, thalamic, striatal and hippocampal regions. Spatial modelling suggested that molecular distribution and network architecture may shape the observed pattern.

WHY IT MATTERS

The findings support a brain-wide systems account of schizophrenia and may help connect molecular mechanisms with distributed network dysfunction. Cross-sectional imaging cannot establish the timing or causal direction of synaptic change.

Source: Chopra et al., Molecular Psychiatry (2026)

A BVFTD-LIKE PHENOTYPE IN A SUBGROUP

Chang and colleagues applied a previously validated imaging classifier to identify a frontotemporal dementia-like grey-matter pattern in schizophrenia. Greater expression of this pattern was associated with lower striatal dopamine synthesis and higher striatal iron, suggesting biological heterogeneity that may not fit a uniform hyperdopaminergic model.

CLINICAL CAUTION

This is a research phenotype, not evidence that schizophrenia is frontotemporal dementia. Replication, longitudinal study and careful translation are essential before subgroup findings influence routine diagnosis or treatment.

Source: Chang et al., Neuropsychopharmacology (2026)

RELATED VIEWPOINT: DOPAMINE AND BEYOND

A further July discussion considered a pluralistic model of the schizophrenia spectrum: partially overlapping neurochemical and cellular routes to psychosis could support biomarker-guided stratification and mechanism-based trials, while raising questions about what truly counts as “pluralism.”

Source: Keshavan et al., Toward a Pluralistic Model for the Schizophrenia Spectrum

From antibody discovery to the long tail of outcomes

DIMINISHING RETURNS - OR UNFINISHED WORK?

AJAMA Neurologyviewpoint argued that clinicallymeaningful autoimmuneencephalitis antibody discovery has plateaued and that greater gains may now come from disease mechanisms and targeted therapies. Group discussion agreed the allocation question was reasonable but cautioned that history makes certainty difficult: today's low-frequency signal may still become tomorrow's clinically useful syndrome.

- Discovery value depends on phenotype specificity, reproducibility and whether the finding changes care.
- The opportunity cost of poorly targeted discovery must be weighed against the risk of prematurely closing the field.
- Faster treatment and better outcome measurement may yield benefits even without a new antibody.

Source: Takegami et al., Diminishing Returns of Novel Autoantibody Discovery in Encephalitis

TWO-YEAR RISKS AFTER HOSPITALISED INFECTION

A large retrospective multicohort study mapped psychiatric and neurological diagnoses after infections across body systems and age groups. Risk was elevated for many outcomes, with encephalitis showing the highest relative signal and cognitive deficits a prominent absolute burden in some comparisons.

INTERPRETATION

The exposure was hospitalisation with infection, so findings should not be directly extrapolated to every minor community infection. Clinically, the paper supports attention to new or worsening neuropsychiatric symptoms after significant infection while guarding against overtreatment during transient illness.

July moved repeatedly between prediction, uncertainty and person-centred communication.

PLASMA P-TAU217 AND FUTURE COGNITIVE IMPAIRMENT

A multicohort study of 2,684 cognitively unimpaired older adults estimated progression risk across baseline p-tau217 levels. The work strengthens the evidence that blood biomarkers can identify biologically elevated risk before impairment, but a biomarker is not a deterministic forecast.

Source: Buckley et al., JAMA - Prognostic Value of Blood-Based P-Tau217

WHEN DIAGNOSIS BECOMES A DEADLINE

A viewpoint on Alzheimer biomarkers and assisted dying warned that earlier biological certainty may create a perceived shrinking window for autonomous choice. The central ethical task is to communicate what biomarkers can and cannot predict, address stigma and fear, and avoid turning risk information into a foreclosed narrative.

WHO DEMENTIA RISK-REDUCTION UPDATE SHARED FOR WORLD BRAIN DAY

- Strong recommendations included physical activity for adults with normal cognition and tobacco cessation.
- Conditional recommendations included cognitive activity, healthy diet, management of vascular/ metabolic risk, hearing support and tailored multidomain intervention.
- Vitamin and omega-3 supplementation were not recommended specifically for dementia prevention; several other interventions had insufficient evidence for this specific outcome.

Source: WHO, Risk reduction of cognitive decline and dementia: guidelines, 2nd edition

LINKED READING LIST

Papers circulated by the GNG community during July 2026

Each link opens the journal or DOI page; summaries are brief educational overviews of the shared paper.

01

Widespread synaptic density loss in schizophrenia follows molecular and network architecture

Chopra S, et al. Molecular Psychiatry. 2026.

Using [11C]UCB-J PET, the study found widespread lower synaptic density in schizophrenia, with particularly strong effects in left-sided frontal, temporal, cingulate, thalamic, striatal and hippocampal regions. Network and molecular architecture appeared to shape the spatial pattern, supporting a distributed systems model of illness.

READ PAPER >

02

Chronic idiopathic urinary retention and Fowler's syndrome in women

McRae S, et al. BJUI Compass. 2026.

This multidisciplinary review proposes a practical framework for assessment and stepped, nonsurgical management of chronic idiopathic urinary retention and Fowler's syndrome. It integrates urology, neurology, physiotherapy, psychology and lived experience while highlighting the limited randomised evidence base.

READ PAPER >

03

Advances in multiple sclerosis

HauserSL.NewEnglandJournalofMedicine. 2026;395:54-68.

A broad clinical review of MS pathogenesis, diagnosis and treatment, emphasising B- and T-cell biology, highly effective anti-CD20 therapies and the persistent unmet need in progressive disease. It also reviews remyelination, neuroprotection and comprehensive symptom and lifestyle management.

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04

Diminishing returns of novel autoantibody discovery in encephalitis

Takegami N, Day GS, Leypoldt F, Irani SR. JAMA Neurology. 2026.

This viewpoint argues that discovery of high-impact encephalitis antibodies has slowed and that greater patient benefit may now come from clarifying mechanisms and developing targeted treatments. It calls for stronger phenotypic specificity, reproducibility and attention to the opportunity costs of low-yield discovery programmes.

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LINKED READING LIST

Encephalitis, epilepsy and cognition

Papers 05-08

05

Psychiatric and behavioural sequelae following encephalitis: a systematic review and meta-analysis

Watson CJ, et al. Brain Communications. 2026;8(3):fcag175.

A systematic review and meta-analysis of 101 studies involving 4,703 patients found substantial long-term burdens of depression, anxiety, disinhibition and emotional instability. The authors call for prospective cohorts and a standardised mental-health outcome set after encephalitis.

READ PAPER >

06

Tau pathology in epilepsy: emerging mechanisms and translational opportunities

Sen A, et al. Brain. 2026;149:2250-2272.

This review examines tau as a possible mechanistic bridge between epilepsy, cognitive impairment and dementia. It covers experimental and human evidence, biomarkers and trials targeting tau-related pathways, while stressing that causal and disease-modifying claims require deeper tissue and longitudinal validation.

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07

Cognitive follow-up in anti-N-methyl-D-aspartate receptor encephalitis: hospital discharge, 4, 8, and 12 months

Bayliss L, et al. Clinical Neurology and Neurosurgery. 2023;228:107701.

Twenty-one patients underwent repeated assessment from discharge to 12 months. Global cognition improved markedly, but attention fluctuated and domain-specific deficits could persist despite favourable neurological recovery - supporting structured neuropsychological follow-up and rehabilitation.

READ PAPER >

08

Toward a pluralistic model for the schizophrenia spectrum - dopamine and beyond

Keshavan MS, et al. JAMA Psychiatry. 2026.

The authors synthesise evidence for dopamine, glutamate, GABA, serotonin, acetylcholine and immune-metabolic mechanisms in schizophrenia-spectrum disorders. They argue that biomarker-informed subgroups and mechanism-based trials may better address treatment resistance and biological heterogeneity than a universal dopamine pathway.

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LINKED READING LIST

Biomarkers, brain injury and infection

Papers 09-12

09

Prognostic value of blood-based p-tau217 levels for progression to cognitive impairment

Buckley RF, et al. JAMA. Published online July 14, 2026.

Across 2,684 cognitively unimpaired older adults, higher baseline plasma p-tau217 predicted progression and faster cognitive decline. Very high levels were associated with an estimated 38% five-year progression risk, although the authors caution that validation in unselected populations is needed before individual prognostic use.

READ PAPER >

10

Amateur soccer heading and acute elevations in blood-based p-tau217 and S100B

Hoppen MI, et al. JAMA Neurology. 2026;83:635-644.

In 302 amateur players, real-world heading exposure was associated with small acute increases in S100B and p-tau217, with dose-response signals for header number and impact. Biomarker levels normalised within 24-48 hours; the study does not establish long-term neurodegeneration.

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11

A frontotemporal dementia-like phenotype in schizophrenia: links to striatal dopamine and iron accumulation

Chang X, et al.Neuropsychopharmacology. 2026.

A machine-learning grey-matter signature identified a bvFTD-like pattern in a subgroup with schizophrenia. Stronger pattern expression correlated with lower striatal dopamine synthesis and higher striatal iron, suggesting biological heterogeneity without implying that schizophrenia itself is a neurodegenerative dementia.

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12

Mapping 2-year psychiatric and neurologic risks after infections across body systems and age groups

Taquet M, et al. JAMA Psychiatry. Published online July 15, 2026.

This large retrospective multicohort study compared outcomes after hospitalised infections across body systems and ages. Many psychiatric and neurological risks were elevated, with encephalitis prominent in relative-risk analyses and cognitive deficits contributing substantial absolute burden; findings should not be extrapolated directly to minor community infections.

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LINKED READING LIST

Autoimmunity, CTE and dementia ethics

Papers 13-16

13

Autoimmune encephalitis and paraneoplastic neurologic syndrome: clinical diagnosis

Lee S-T. CurrentOpinion in Neurology.2026;39:361-369.

A practical review of subacute clinical patterns, neuroanatomical localisation, MRI, CSF and antibody testing in AE and paraneoplastic syndromes. It emphasises red flags, correct assay interpretation and the integration of clinical, radiological and serological evidence to reduce both missed diagnoses and misdiagnosis.

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14

Chronic traumatic encephalopathy (CTE) - features and forensic considerations

Byard R, et al. Forensic Science, Medicine and Pathology. 2023;19:620-624.

This commentary reviews CTE neuropathology, relevant exposure histories and forensic autopsy sampling. It highlights perivascular phosphorylated-tau pathology in sulcal depths and argues that systematic head-trauma histories and appropriate brain examination are necessary to avoid missed cases.

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15

Alzheimer disease biomarkers and assisted dying: when diagnosis becomes a deadline

Lyndon S, et al. JAMA. 2026.

The viewpoint explores how earlier biomarker-supported diagnosis may create a perceived deadline for end-of-life decisions while capacity remains intact. It urges clinicians to communicate prognostic uncertainty, address identity and stigma, and avoid allowing pathology information to become a deterministic personal narrative.

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16

The risk of Alzheimer's disease and cognitive impairment characteristics in eight mental disorders: a UK Biobank observational study and Mendelian randomization analysis

Liu Y, et al. Alzheimer's & Dementia. 2024;20:4841-4853.

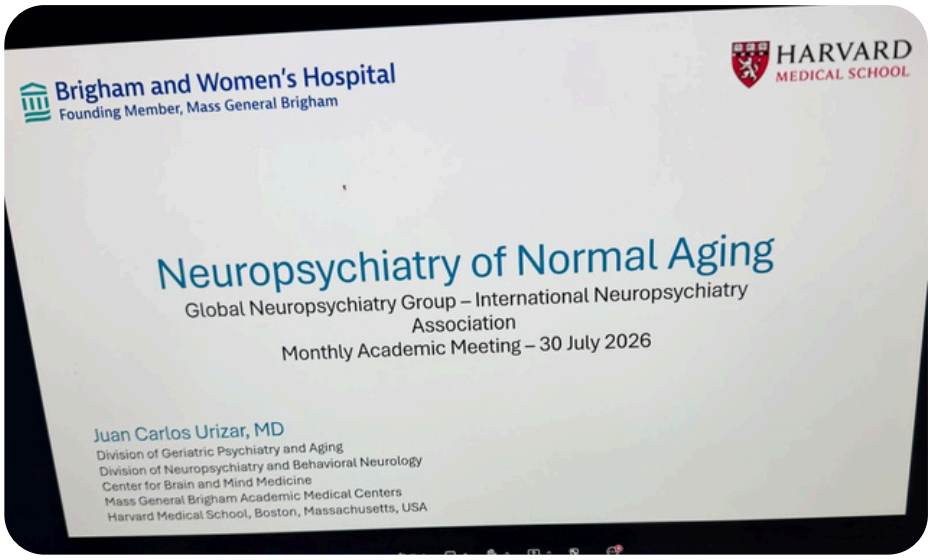
In UK Biobank data, Alzheimer risk was higher among people with bipolar disorder and major depression; Mendelian-randomisation analyses also supported links involving bipolar disorder and OCD. Cognitive profiles differed by diagnosis, suggesting that prevention and monitoring may need to be disorder-specific.

READ PAPER >

Also shared: the July discussion included the WHO guideline Risk reduction of cognitive decline and dementia (2nd edition), as well as GNG educational handouts and meeting material. These are listed elsewhere in this newsletter because they are guidelines or educational resources rather than journal papers.

GNG-INA MONTHLY ACADEMIC MEETING - 30 JULY 2026

Chronic traumatic encephalopathy and the neuropsychiatry of normal ageing



Presentation slide shared during the July academic meeting.

CHRONIC TRAUMATIC ENCEPHALOPATHY

Dr Rowena Mobbs presented clinical cases and the evolving evidence around repetitive head impacts, CTE neuropathology, diagnosis during life and the limits of causal inference. Discussion emphasised the importance of separating compelling personal narratives from the evidentiary questions of exposure measurement, dose-response relationships, susceptibility and appropriate controls.

NEUROPSYCHIATRY OF NORMAL AGEING

Prof Juan Carlos Urizar explored ageing as a dynamic balance across brain, body, cognition and environment. The session considered how age-related change differs from disease and highlighted the interaction between severe mental illness, cognitive risk and the need for prevention-oriented longitudinal care.

MEETING MESSAGE

Stay informed. Stay curious. The July meeting showed why neuropsychiatry is strongest when clinical observation, biological evidence and humane uncertainty are held together.

ON THE HORIZON

Recent reading and forthcoming opportunities

Verified on 15 August 2026

NEW AND RECENT BOOKS

The Neuroscience of Word Meaning - David Kemmerer (Cambridge University Press, online 27 February 2026)

A neuroscience-led account of how conceptual knowledge and word meaning are organised, spanning experiential foundations, semantic hubs and higher-order conceptual structure. Particularly relevant to clinicians and researchers interested in aphasia, semantic cognition and neurodegenerative language disorders. View publisher page >

Charney & Nestler's Neurobiology of Mental Illness, 6th edition - Sklar, Buxbaum, Nestler & Charney (eds.)

A substantially renewed reference covering genetics and molecular, cellular and systems neuroscience across major mental illnesses. Its breadth makes it useful for advanced trainees, clinician-scientists and readers tracking the move toward biologically stratified psychiatry.

View publisher page >

FORTHCOMING EVENTS

DATE	EVENT	FORMAT / LOCATION
27 Aug 2026	GNG Monthly Academic Meeting - Functional Neurological Disorder	Online; details to be circulated by GNG
23-26 Sep 2026	26th World Congress of Psychiatry	Stockholm, Sweden
22-24 Oct 2026	Neuropsychiatry: A Comprehensive Update	Boston and virtual
12-15 Nov 2026	RANZCP-SPIDD-GNG Neuropsychiatry Conference	Gold Coast, Australia
26-29 Nov 2026	Biological Psychiatry Congress 2026, with INA	Cape Town, South Africa

Event details may change; please confirm registration, programme and time-zone information with organisers.

CONTINUE THE CONVERSATION

Become a GNG Member

For clinicians, trainees and researchers interested in the interface between brain and mind

GNG is an international professional community built around generous exchange: challenging cases, journal discussions, research updates, academic meetings and connections across disciplines and borders. If this July round-up was useful, we invite you to become part of the conversation.

MEMBER ACCESS

ACADEMIC MEETINGS

Join international academic meetings and journal discussions.

CONTRIBUTIONS

Contribute ideas for future meetings, surveys, handouts and newsletters.

GLOBAL NETWORK

Learn from colleagues working across psychiatry, neurology, neuropsychology, rehabilitation and neuroscience.

ACADEMIC OPPORTUNITIES

Share research, educational resources and carefully de-identified clinical questions.

Our shared purpose: To advance neuropsychiatric education, thoughtful clinical practice and collaboration—while keeping curiosity, humility and patient-centred care at the heart of the work.

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Thank you for reading, contributing and helping GNG connect minds across borders.

Editorial note: This newsletter was curated from July 2026 community discussion and publicly available sources. Patient details were generalised and identifying information omitted. Clinical content is for education only.