How can Traditional Mental Health Care increase the load on Neurodevelopmental and Psychiatric Disorders that involve Hyper and Hypo Connected Brain Networks, Hypo-Metabolism, Endocrine Dysregulation and Mitochondrial Dysregulation?

Traditional mental health care can **increase burden** when it **misses systemic biology** and adds metabolic or network stress.

Traditional mental health care can increase the load on these disorders when it treats them mainly as **neurotransmitter syndromes** or isolated DSM categories, while the evidence increasingly frames many psychiatric and neurodevelopmental conditions as disorders of brain-body networks, energy metabolism, neuroendocrine regulation, inflammation, and mitochondrial function (Nunes et al., 2025; Cirulli & Dickson, 2026; Michelini et al., 2024). That mismatch can worsen burden through three linked pathways: under-recognizing metabolic and endocrine drivers, using treatments that add metabolic load or miss mitochondrial vulnerability, and organizing care in fragmented categories despite transdiagnostic network overlap and multimorbidity (Chokkakula et al., 2026; Cirulli & Dickson, 2026; Ogundele & Morton, 2022; Sha et al., 2019).

Traditional Mental Health Care and Disorder Load

Evidence Strength	Claim
Strong	**Psychiatric disorders are not purely neurotransmitter disorders**; multiple reviews describe them as systemic conditions involving mitochondrial dysfunction, oxidative stress, inflammation, metabolism, and neuroendocrine signaling, so narrow symptom-focused care can miss major disease drivers (Nunes et al., 2025; Kim et al., 2019; Cirulli & Dickson, 2026)
Strong	**Shared hyper- and hypoconnectivity patterns** across DMN, salience, and frontoparietal networks are transdiagnostic, so rigid categorical care can mis-handle disorders that share network-level dysfunction and cognitive burden (Sha et al., 2019; Koban et al., 2021; Michelini et al., 2024)
Strong	**Conventional care can add metabolic burden** because psychopharmacology can obscure assessment and contribute to metabolic complications, while suboptimal management, stigma, and fragmentation delay integrated intervention for multimorbid patients (Chokkakula et al., 2026; Cirulli & Dickson, 2026; Ogundele & Morton, 2022)

FIGURE 1 Evidence strength for pathways increasing disorder burden

Biological Mismatch

When care does not assess **energy metabolism** and mitochondrial status, it can miss mechanisms directly tied to symptoms. Neuroimaging, metabolomics, and transcriptomics show regional brain hypometabolism, altered mitochondrial gene expression, and impaired oxidative phosphorylation across depression, bipolar disorder, and schizophrenia (Nunes et al., 2025; Ni et al., 2022).

High-demand brain regions are especially vulnerable. The prefrontal cortex and hippocampus are sensitive to energy shortages, and chronic stress promotes mitochondrial fission and defective recycling of damaged mitochondria, which can deepen neuronal vulnerability rather than relieve it if care addresses distress without the underlying bioenergetic load (Seo et al., 2025; Tanaka et al., 2022).

- **MDD shows impaired control energy** in default mode and limbic nodes tied to cognition and symptoms (Niu et al., 2026)- ADHD and ASD share **mitochondrial dysfunction** and cerebral hypoperfusion, linking neurodevelopmental symptoms to energy failure (Payares et al., 2024)- Genetic neurodevelopmental disorders also show **mitochondrial bioenergetic dysfunction** affecting neurodevelopment and cognition (Valenti & Vacca, 2023)## Endocrine and Metabolic Burden

Neuroendocrine and metabolic abnormalities often persist beyond medication effects, but clinical management remains suboptimal. Meta-analytic or review-level evidence describes persistent abnormalities in glucose regulation, lipid metabolism, autonomic function, circadian-metabolic coupling, and HPA-axis-related signaling across major psychiatric disorders (Chokkakula et al., 2026; Kim et al., 2019).

Traditional care can therefore increase burden if it overlooks endocrine-metabolic comorbidity or adds treatment-related metabolic stress. This matters because metabolic dysregulation can amplify oxidative stress and neuroinflammation, accelerating psychiatric progression and cognitive decline, while some current treatments have limited efficacy and do not address these mechanisms (Chokkakula et al., 2026; Schwartz et al., 2026).

Fragmented Nosology

Care Problem	Why It Can Increase Load
Categorical diagnosis	High co-occurrence and arbitrary boundaries can hide shared biology across neurodevelopmental and psychiatric conditions (Michelini et al., 2024)
Fragmented services	Multimorbid children and young people often face split services and sub-threshold diagnoses (Ogundele & Morton, 2022)
Brain-only framing	Mental and physical health share vmPFC-DMN systems regulating autonomic, endocrine, and immune function (Koban et al., 2021)
Late-stage response	Recurrent episodes can drive neuroprogression, circuit reorganization, and loss of treatment response (Cyrino et al., 2021)

FIGURE 2 Clinical pathways by which care can increase burden

Traditional mental health care increases the load on these disorders when it remains **brain-only, categorical, and symptom-focused** despite evidence for shared network dysconnectivity, hypometabolism, endocrine disruption, and mitochondrial pathology across diagnoses (Sha et al., 2019; Koban et al., 2021; Chokkakula et al., 2026; Valenti & Vacca, 2023). The literature supports a more integrated, transdiagnostic, brain-body model; evidence for exactly which alternative interventions best reduce that load is growing, but still less mature than the evidence that the burden is systemic (Michelini et al., 2024; Ogundele & Morton, 2022; Nunes et al., 2025; Cirulli & Dickson, 2026).

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