Component	Dopamine Hypothesis	Neurodevelopmental Model	Diathesis-Stress Model	Predictive Coding Model	Immune/Inflammatory Model	Sensitivity Threshold Model (STM)
Positive Symptoms (e.g., hallucinations, delusions)	**Moderate** — Attributed to dopamine hyperactivity in mesolimbic pathway. Explains treatment effects of D2 blockers but not full phenomenology.	**Weak** — Views symptoms as consequences of abnormal development but lacks direct mechanism for psychosis onset.	**Moderate** — Attributes symptoms to stress in vulnerable individuals, but lacks specificity.	**Strong** — Explained as misweighting of sensory priors (prediction errors), leading to false inferences.	**Moderate** — Links cytokine activity to symptom severity; lacks mechanistic clarity.	**Strong** — Positive symptoms emerge from system overload and faulty processing in sensitive systems; aligns with IT analogies and empirical cases (e.g., torture-induced hallucinations).
Negative Symptoms (e.g., apathy, flat affect)	**Weak** — Poorly explained. Dopamine hypothesis focuses on positive symptoms.	**Moderate** — Attributed to prefrontal cortical deficits; doesn't explain variability or treatment resistance.	**Weak** — Stress model lacks mechanism to link to negative symptoms.	**Moderate** — Underexplored; possible result of precision misestimation or low priors.	**Weak** — Inflammation may play a role but causality unclear.	**Strong** — Seen as adaptive downregulation post-overload; fits with chronic stress-induced neuronal exhaustion and supports symptom fluctuation.
Cognitive Impairments	**Weak** — Not explained directly.	**Strong** — Developmental deficits align with early cognitive delays.	**Moderate** — Stress impact on cognition recognized but vague.	**Strong** — Seen as disrupted signal integration; matches task-based evidence.	**Weak** — Cognitive deficits often secondary, not causally explained.	**Strong** — Framed as primary consequence of processing overload; IT analogies (buffer overflow, bandwidth limits) are compelling.
Onset Timing (adolescence/early adulthood)	**Weak** — No specific developmental account.	**Strong** — Tied to brain maturation and synaptic pruning.	**Moderate** — Fits idea that stress increases with life complexity.	**Moderate** — Developmental encoding of priors may explain it, but under-theorized.	**Moderate** — Infections and inflammation in youth linked; timing unclear.	**Strong** — High sensitivity + increasing life load during development = threshold breach; explains delayed onset and progressive nature.
Environmental Influence	**Very weak** — Largely ignored.	**Moderate** — Prenatal exposures and early life stress incorporated.	**Strong** — Central to theory, though non-specific.	**Moderate** — Includes sensory unpredictability and noise as factors.	**Strong** — Pathogen exposure, diet, pollution incorporated.	**Very strong** — Overload by toxins, urban stress, noise; directly modeled as stress-load inputs.
Individual Variability / Sensitivity	**Very weak** — One-size-fits-all model.	**Moderate** — Acknowledges risk strata.	**Moderate** — Emphasizes "vulnerability," but unspecific.	**Strong** — Priors vary between individuals; accounts for idiosyncrasies.	**Weak** — Genetic variation in immune genes included, but not phenotypically refined.	**Very strong** — Individual sensitivity is core; integrates HSP theory and differential susceptibility.
Mechanism Clarity	**Strong but narrow** — Dopamine pathways well studied, limited scope.	**Moderate** — Mechanisms plausible but heterogeneous.	**Weak** — Vague on causal pathways.	**Strong but complex** — Predictive error models rich but hard to apply clinically.	**Moderate** — Cytokine and microglial pathways studied, causal chain unclear.	**Strong and holistic** — Cognitive overload from multisource stress > HPA/cortical dysregulation > psychosis. Elegant but needs empirical testing.
Pharmacological Fit	**Strong** — Antipsychotics work via D2 blockade.	**Weak** — No direct drug targets.	**Weak** — Not predictive of medication response.	**Weak to moderate** — Some relevance to NMDA/glutamate interventions.	**Moderate** — Anti-inflammatory agents under investigation.	**Strong** — Drugs reduce neuronal load (sensitivity), matching STM predictions; explains both effect and side effects.
Empirical Support	**High for mechanism, low for etiology**	**Strong for risk markers, weak for full model**	**Widely accepted, weakly specified**	**Growing evidence in imaging and modeling**	**Moderate support in immune biomarkers**	****Low formal validation, but observational and conceptual support increasing.**
Unexplained Areas	Why dopamine fluctuates; individual variability.	Symptom heterogeneity; resilience.	Why thresholds vary; exact biological stress interfaces.	How priors form and change; subjective experience.	Why symptoms cluster; low specificity.	Needs clearer operational definitions, predictive coding integration, and large-scale testing.