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Beta Oscillations as a Mechanistic Target for Predictive Processing Deficits in Psychosis

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Abstract

Background: Predictive processing abnormalities offer a unifying account of perceptual and expressive disturbances in psychosis, yet classical predictive coding frameworks remain difficult to translate due to limited neurophysiological grounding. Emerging evidence positions beta-band oscillations and their transient burst dynamics as a biologically plausible mechanism for implementing top-down predictions that stabilize internal models. **Study Design:** This narrative review synthesizes evidence from electrophysiology, laminar physiology, computational modelling, language research, and clinical neuroimaging to evaluate beta oscillations as a mechanistic target for predictive processing deficits in psychosis. We integrate data from modified predictive routing frameworks and dendritic computation models to clarify how beta rhythms prepare cortical pathways for predicted inputs. **Study Results:** Across sensory, motor, cognitive, and language domains, schizophrenia features impaired generation, timing, and contextual deployment of beta activity. These include attenuated post-movement beta rebound, reduced or mistimed beta bursts during working memory and inhibition, abnormal beta-gamma interactions during perception, and weakened beta-mediated contextual guidance during language comprehension. Laminar and computational findings indicate that beta bursts arise from the integration of apical (contextual) and basal (sensory) dendritic inputs in layer 5 pyramidal neurons, providing a mechanistic substrate for top-down predictions. Beta disruptions, therefore, offer a parsimonious account of disorganization, psychomotor slowing, and failures of contextual maintenance. Early neuromodulation, pharmacologic, and neurofeedback studies suggest that beta dynamics are modifiable. **Conclusions:** Beta oscillations provide a tractable and mechanistically grounded target for predictive processing deficits in psychosis. Standardizing burst metrics and developing individualized, closed-loop approaches will be critical for advancing beta-based interventions.

Keywords: beta oscillations; burst dynamics; predictive routing; psychosis; thought disorder; schizophrenia; neuromodulation

1. Introduction

We make sense of the world by creating mental models and reconciling them with sensory data. This involves carrying robust expectations about our environment to ensure predictability as we operate in it. Predictability is optimal when our mental models match our current state and redundant sensory processing is suppressed [1]. A disruption in the mechanisms that generate and utilize optimal mental models can affect how we perceive and communicate with others. Understanding this process is crucial in psychosis, where thought and language disruptions are cardinal features. Recent evidence suggests that top-down feedback may act through a preparatory predictive-routing mechanism, rather than purely through selective inhibition as posited in classical

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predictive coding frameworks. This preparatory influence is mediated via spontaneous bursts of neural activity, particularly in the beta frequency band, which are thought to stabilize the cortical network in anticipation of predictable input. Here, we review this evidence and explore the opportunities offered by a beta-oscillation-centred notion of psychosis.

One of the striking features of psychotic disorders, such as schizophrenia, is how events that have low probability come to dominate interpretations of the world (e.g., invasion from Martians, being poisoned by one's parents). Such unlikely events are also evident in expressed behaviour such as disorganized speech (e.g., neologisms, improbable words, tangential responses). A parsimonious account of psychotic symptoms situates them in aberrations of the brain's ability to generate, maintain and utilize internal models of the world [1–3]. These so-called predictive processing deficits are usually formalized as deficits in probabilistic computations [4].

The dominant neural implementation used to explain generative models in the brain is the Predictive Coding framework described by Rao and Ballard [5]. Top-down predictions decrease processing by suppressing predicted activity in lower sensory systems. Unpredictable sensory data, on the other hand, trigger brain activity by causing a prediction error in 'error units'. This signals new evidence that is fed-forward (bottom-up) to update prior predictions or initiate actions to infer causes of the data, revising our mental model of the sensory world. Importantly, this revision not only reduces surprise for the future (error minimization), but also enables one to make sense of the unfolding context ('authentic meaning' via predictive success maximization[6]). In schizophrenia, a disruption in the integration of predictions with evidence is thought to occur: lower-level sensory signals are thought to be assigned excessive weight (aberrant prediction errors), leading to erroneous revisions of the mental model that are insufficient to explain one's environmental context. Clinically, this may present as irrelevant events acquiring outsized significance, contributing to recurrent maladaptive interpretations (delusions and hallucinations). Other than sensory-related symptoms, expressive behaviour such as language aberrations is proposed to reflect a breakdown in the generative circuit of predictive processing [7], especially at the level where higher-order messages influence pre-activation of contextually appropriate lower-level representations (e.g., word choices). This affects the contextual constraints that guide comprehension and production of language and thought (see also [8,9]).

While predictive coding offers a unifying account of psychotic phenomena, it remains abstract and at times inconsistent with neurophysiology. Several testable hypotheses derived from predictive coding have been increasingly challenged. We review evidence that is inconsistent with the classic predictive coding framework and propose a modified predictive routing framework that reconciles these findings by positioning neural oscillations as the main mechanism.

2. Implementation of Predictive Coding

Predictive coding framework's presumed neurophysiological basis is under constant scrutiny.

(1) The predictive coding framework usually assumes that precision (or reliability) weighted cortical feedback cancels expected input by subtracting it via inhibition, discarding the already predicted inputs, and transmitting only those activity 'spikes' that are not predicted. However, electrophysiological and laminar recordings show that feedback and feedforward activity often overlap in time, and feedback is often excitatory; not purely inhibitory or subtractive [10–12].

(2) Predictive coding assumes prediction errors first originate in lower-level cortical areas and propagate upwards. However, empirical evidence does not consistently support this temporal order. Studies using laminar recordings and oscillatory markers infer prediction errors from superficial gamma-band activity and feedforward pathways, but these signals often appear simultaneously across levels or even earlier in higher-order cortices [10,13,14]. Moreover, the timescales required for multiple cycles of iterative feed-forward optimisation to converge are incompatible with most sensory processing timelines [15].

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(3) Predictive coding accounts successfully explain responses to local unpredictability (e.g., oddball sequences with short-range regularities), but they generalize poorly to global violations that unfold over longer timescales [14,16,17].

(4) Predictive coding framework mandates specialized 'error units' that handle the net difference between input and top-down predictions across the entire hierarchy of message passing (two types of these units described more recently by Nour Eddine et al. [18], with self-connections influencing precision computation [19]. Nonetheless, cellular data has not yet found evidence in support of such an architecture to hold residual information [11,20].

The implementational limitations have motivated alternative biological accounts (e.g., [21–23]) and prompted reconceptualizations of what 'prediction' means in cortical computation. For example, the Backward Enhancement of Limited information and Inhibition of Expected information in Feedforward pathways (BELIEF) framework argues that much of the activity traditionally interpreted as predictive suppression may instead arise from feedforward adaptation, while feedback implements capacity-limited, excitatory template selection rather than broad inhibitory cancellation [24]. In parallel, control-based accounts suggest that many so-called predictive effects may reflect perceptual state regulation rather than generative model subtraction [25]. Together, these perspectives maintain the importance of detecting when expectations and sensory input diverge while loosening the assumptions of strict hierarchical directionality and subtractive feedback, re-framing cortical inference as a closed-loop selection and maintenance process rather than a unidirectional prediction-error cascade. Applying predictive coding theories to psychosis presents another challenge: the phenomenological experience of symptoms like delusions does not align with a simple hierarchical predictive coding approach [26]. Recent theories propose hybrid predictive coding [23,27], allowing predictions to travel both top-down and bottom-up. All of these alternatives weaken the inhibition-based account of predictive coding and call for alternative approaches to meet the inherent implementational challenges.

Two promising perspectives have emerged: (1) theories emphasizing oscillatory rhythms as dynamic routing mechanisms that prepare pathways for predicted inputs through alternating functional inhibition and facilitation (i.e., conditional gates that do not mandate supervised precision-weighting) [10,16,28], and (2) models situating 'error' computation within the dendritic compartments of pyramidal neurons, where contextual inputs to apical dendrites modulate feedforward drive at basal dendrites [11,28–30]. Both aspects converge on the idea that predictions are not simply subtracted but rather implemented through rhythmic preparation of the cortex for relevant sensory inputs. When the unprepared cortex encounters the same sensory inputs, 'errors' indexed by cortical activation occur [31]. Importantly, this provides a metric reflecting the integrity of top-down predictions (mediated by beta oscillatory dynamics) that can be measured across tasks in patients with psychosis. Combining these emerging perspectives, we propose a modified predictive routing model (see Table 1).

Table 1. Contrasting Features in Predictive Coding and Modified Predictive Routing Frameworks.

Feature	Predictive Coding	Modified Predictive Routing
Prediction Error (PE) Mechanism	Prediction error is generated by dedicated canonical circuitry.	Prediction error occurs when sensory inputs reach an unprepared (uninhibited) cortex.
Nature of Predictive Signal	Predictions (priors) are thought to be subtractive, generally suppressing overall neuronal activity.	Predictions are sparse, selective preparatory signals issued by higher-order cortex that influence the constraints carried by the lower-order cortex.

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Scope of Predictions	Predictions are widespread and canonical across the entire cortex.	Predictions are mediated by beta rhythms as selective and sparse top-down signals.
Prediction Signal Carrier (Feedback)	Predictions feedback down the hierarchy via deep layers (L5/6).	Predictions feedback via deep layers utilizing alpha/beta rhythms.
Error Signal (Feedforward)	PE signals feed forward up the hierarchy via superficial layers (L2/3).	Enhanced processing at lower levels carried by gamma frequency (40–90 Hz) and associated spiking via superficial layers.
Primary Implementation Mechanism	Canonical microcircuit with dedicated error units; self-connections on error units modulating precision.	Spectrolaminar mechanisms that flexibly route information; no dedicated error units.

3. Oscillatory Rhythms and Predictive Routing

A growing body of work indicates that oscillatory rhythms generated in a layer-specific manner serve to dynamically gate the routes of hierarchical information flow. In macaques, predictable stimuli increase deep-layer alpha/beta (8–30 Hz) power and feedback coupling from higher to lower areas, while unpredictable stimuli enhance superficial-layer gamma (~40–90 Hz) and feedforward spiking, and these are anticorrelated [16,28,32]. Bastos and colleagues (2012, 2020) termed this pattern 'predictive routing'. They argue that low-frequency beta feedback prepares pathways by tuning down excitability (functionally inhibiting) and thus sensory drive in the superficial channels, so inputs that are consistent with the current context (i.e., the expected) are suppressed. When the beta-encoded expectations are not met, gamma activity is released and propagated upward to update internal models [33,34]; thus, there is no explicit error computation but sparsified processing and feedback occurs via laminar-rhythmic mechanisms [35]. This laminar-rhythmic segregation has been interpreted as a mechanistic division of labor between predominantly infragranular beta rhythms carrying top-down predictions and predominantly supragranular gamma rhythms signalling bottom-up sensory evidence [36–39].

Consistent with this framework, [27] proposed that predictive processes in the brain can be divided into two complementary forms: context-independent and context-dependent predictions. Context-independent predictions, or constraints, are embedded within the sensory architecture itself, reflecting long-term regularities of the environment and shaping how incoming sensory signals are processed. These predictions correspond to bottom-up gamma-band processes [40,41], which rapidly convey stimulus-driven evidence shaped by these entrenched regularities. In contrast, context-dependent predictions, or expectations, are flexibly generated based on current goals and situational context, aligning with top-down beta-band feedback that modulates lower cortical areas to pre-tune perceptual channels and suppress predictable inputs [40,42,43]. In this sense, gamma reflects the embedded predictive constraints operating along the feedforward hierarchy, whereas beta reflects dynamic contextual predictions transmitted through feedback pathways, together forming a layered system in which stable, bottom-up constraints and flexible, top-down expectations jointly sculpt perceptual inference.

Complementing this account, Vinck and colleagues [28] emphasize that oscillations exert their influence not by continuously 'carrying' top-down prediction or bottom-up error signals, but through transient bursts that stabilize and sculpt their representations and facilitate inter-areal communication. Bursting dynamics provide flexible, energy-efficient [44,45] and context-sensitive control, amenable to influence across levels of hierarchy via cross-frequency coupling [46–49], offering a biologically plausible mechanism to reconcile predictions with sensory evidence while addressing the limitations of canonical predictive coding models.

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4. Why is Beta Central to Predictions?

Disrupted oscillatory synchrony across several bands is implicated in the pathophysiology of psychosis [50]. We focus on beta band specifically in the context of internal mental models and top-down predictions in psychosis for the following reasons:

(1) *Beta influences cortical excitability:* Top-down mechanisms of resource allocation, such as attention, affect downstream changes in sensory/motor cortex excitability via beta modulation [51,52]. Prefrontal beta increases when we must stop current actions or thoughts to focus on something more important [53], and it dampens exogenous salience in service of internal goals [54,55]. In the motor domain, beta activity exhibits a biphasic pattern, with suppression before and during movement, followed by a rebound after movement [47,56]. This suppression-rebound dynamic, known as the post-movement beta rebound (PMBR), scales with confidence in the internal model [57,58], likely signaling the need to maintain or revise the internal model [59]. PMBR is consistent with feedback-related functional inhibition of redundant activity in line with the current context.

(2) *Beta bursts maintain endogenous representations*: During working memory tasks, beta bursts occur during the maintenance phase, especially at the end of trials when representations need stabilization. They decrease during encoding and retrieval phases when external inputs are processed. This temporal profile suggests bursts reactivate prior content and inhibit distractors, preparing the cortex for relevant and predictable inputs [60,61]. Worth noting for bursting interpretation, recent work cautions that some apparent bursts may arise from momentary fluctuations of a sustained beta rhythm, depending on how bursts are defined (e.g., amplitude threshold vs. oscillatory mode) [62]. Beta may also encode environmental temporal regularities [63–65], which are crucial for predictability.

(3) *Beta dominates resting cortex:* Beta oscillations are strikingly ubiquitous across the cortical surface. In one large intracranial EEG study, only 21 out of 1772 sites failed to exhibit beta activity at rest [66]. Large-scale mapping shows that beta rhythms synchronize activity across widespread cortical networks, forming a dynamic core [42,67]. This core includes default-mode, sensorimotor, and frontoparietal control systems [66,68] that influence cognitive responses (e.g., attentional performance [69], perceptual acuity [70]. Notably, beta synchronizes over long distances in the brain [71], enabling distributed control of excitability. Beta is crucial for task-free brain activity that likely optimizes generative internal models by forming spatiotemporal connectivity scaffolds and low-dimensional priors that can be deployed in novel contexts [72].

Although beta and gamma oscillations often show reciprocal dynamics, they play distinct computational roles within cortical hierarchies (feedforward vs. feedback). Laminar MEG and intracranial recordings show that beta bursts originating in deep (infragranular) layers propagate upward and temporally gate superficial gamma bursts [16,73]. In predictive processing terms, beta conveys *descending predictions* that suppress or sculpt gamma-encoded *ascending errors*. Thus, while gamma amplitude increases when predictions fail, it is the beta burst that sets the gain structure determining whether an error is expected or destabilizing [38]. Moreover, the clinical disturbances (e.g., disorganization, psychomotor slowing, rigidity, and compulsivity) are all characterized by failures of contextual maintenance or over-stabilization, functions most directly indexed by beta rather than gamma. Gamma abnormalities in these disorders likely reflect secondary instability of feedforward signaling rather than its cause [50,74]. Consequently, targeting beta provides a mechanistically specific handle on the *predictive-stability axis*: modulating the rhythm that governs when cortical networks hold steady versus update.

In summary, beta oscillations are the most compelling neural candidate for implementing top-down predictions from well-rehearsed generative internal models to prepare lower-order cortex for selective propagation of bottom-up inputs. Consequently, a beta-oscillation centered notion offers a potent mechanistic model for linking disruptions in predictability to features of disorganization that are characteristic of psychosis.

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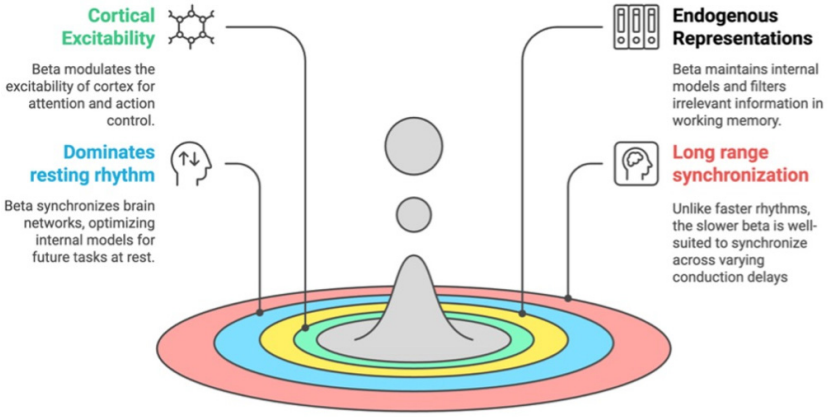

Figure 1. Why is beta central to predictions? (image created using napkin.ai). This schematic highlights the 4 key lines of arguments for placing beta oscillations at the centre of studying predictive routing failures in schizophrenia.

5. Generation of Beta Bursts

Sherman and colleagues (2016) [48] demonstrated that neocortical beta burst generation is best explained by the coincidence of a weak, sustained proximal drive (perisomatic basal dendrites) with a stronger, transient distal drive (apical dendrites) lasting one beta cycle (~50 ms). This interaction of distinct excitatory synaptic inputs to deep and superficial cortical layers, generates current flows in opposing directions (forward and backpropagation), amplified by intracellular dynamics, effectively transforming single axonal inputs into temporally precise bursts.

The above model has since been validated against both primate LFP and human MEG recordings [47,48,73]. Layer 5 pyramidal neurons are central to this process and help reconcile findings between laminar input and output roles. Their basal dendrites, located in infragranular layers (L5/6), receive strong feedforward sensory input from thalamic and lower-level cortical sources, while their apical dendrites, extending to superficial layer 1, receive feedback and contextual inputs from higher-order cortices. This arrangement allows a single neuron to integrate bottom-up evidence with top-down predictions (see [75] for both supra and infragranular participation in beta). Crucially, the output axons of these L5 pyramidal cells constitute the major descending pathways that generate feedback signals to lower cortical areas, and beta-band rhythms are particularly prominent in this infragranular output channel. Thus, while basal dendrites are a site of feedforward drive, the same neurons' outputs provide the substrate for top-down beta-band feedback.

The burst dynamics described above align with a broader class of dendritic computation models, where apical inputs convey contextual or modulatory signals while basal inputs reflect receptive-field information. Furthermore, Phillips' notion of cooperative context sensitivity extends this principle by proposing that contextual inputs at the apical integration zone of pyramidal neurons do not subtract from basal feedforward input (as predictive coding models often suggest) but instead modulate its effective strength, i.e., a form of biasing [76,77]. In this framework, apical inputs amplify, inhibit, or stabilize basal drive depending on contextual demands, thereby shaping whether feedforward information is transmitted or suppressed [77–79]. The computational modeling of Graham et al. [78] shows that in L5 pyramidal cells, coincident apical and basal inputs produce burst firing with probabilities that depend on the relative strength of each input stream (consistent with [48,80], supporting this modulatory view.

As markers of functional inhibition, beta indexes prioritization of current context and suppression of irrelevant responses [47,60,81–84]. This perspective redefines beta as a preparatory

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signal carrying the unique footprint of contextually relevant representations that routes bottom-up evidence by biasing input channels.

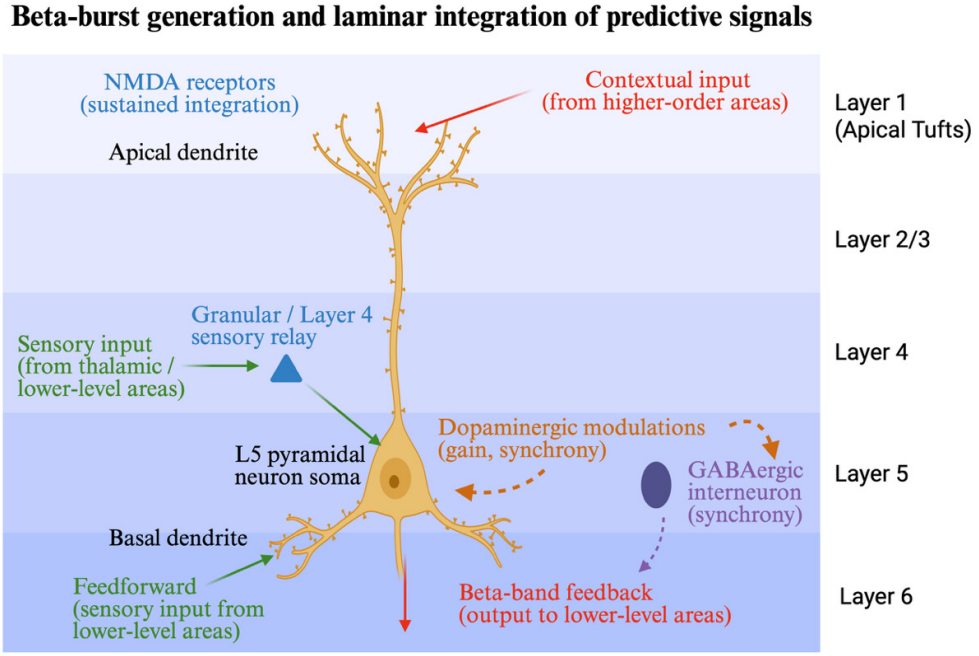

Figure 2. Beta-burst generation and laminar integration of predictive signals. This schematic illustrates how layer 5 (L5) pyramidal neurons integrate bottom-up sensory evidence with top-down contextual predictions to generate beta-band feedback signals. Feedforward sensory input arrives primarily through Layer 4 relay neurons, reflecting thalamo-cortical and lower-order cortical inputs (green). These signals target basal dendrites of L5 pyramidal neurons and are associated with gamma-band activity, conveying sensory evidence and prediction errors. **Additionally**, lower-level cortical areas and some thalamic pathways provide **direct feedforward input to Layer 5**, especially for salient, behaviorally relevant stimuli. In contrast, apical dendrites extend to Layer 1, where they receive contextual feedback (red) from higher-order cortical regions and associative thalamic nuclei. NMDA receptor (blue)-dependent dendritic integration supports ~50 ms temporal summation, allowing contextual inputs to modulate, rather than override, incoming sensory drive. When weak, sustained basal input coincides with transient apical input, the resulting opposing dendritic current flows generate transient beta bursts, providing context-dependent gating of information flow. Thalamic involvement is twofold. First, the sensory thalamus drives the Layer 4 → L5 feedforward pathway. Second, higher-order thalamus regulates cortical excitability via alpha-band inhibitory gating, shaping when sensory information is task-relevant and allowed to influence cortical processing. In this framework, alpha rhythms gate access, while beta rhythms stabilize selected representations once gated. Within L5, dopaminergic modulation (orange) shapes neuronal gain and burst synchrony, enhancing stability in motor cortico-striatal loops and flexibility in frontal executive circuits. Parvalbumin-positive GABAergic interneurons (purple) pace beta rhythms, aligning pyramidal ensemble activity into coherent bursts. Finally, L5 axons serve as the major descending output pathway, meaning beta bursts propagate feedback predictions to lower-level cortical areas. Together, this circuit shows how gamma-band sensory evidence and beta-band contextual predictions converge within individual neurons to implement predictive routing through NMDA-, GABA-, and dopamine-dependent mechanisms.

6. Beta, Language and Higher-Level Meaning

Language comprehension is a quintessential predictive process where lower-level phonemic/lexical signals and global multimodal contextual information iteratively integrate with each other [7,85–89]. Beta oscillations play a critical role in this hierarchical implementation. Evidence

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suggests that beta is influenced by linguistic predictability based on higher-level meaning forming the content of top-down predictions [90,91], with low-gamma reflecting a match between the predicted and observed input [92–94] and high-gamma reflecting a mismatch-related activation of lexical representations (also see [95,96]).

The timing-precise beta bursts that update internal models after movement also stabilize/revise linguistic context in healthy individuals (beta decreases with violations; increases/is maintained with stable predictions) [91,97,98]. Error signals generated in response to deviant lexical stimuli—N400 in lexical processing [99] relate to post-stimulus beta power in task-relevant regions, signalling model revisions (sensory predictions or semantic integration) [100]. Semantically incongruent sentence endings [91,99], or syntactic violations [97,101,102] reliably induce beta suppression, indicating a need to revise the preparatory beta signal (see [103] for a review).

Beyond such constrained paradigms, naturalistic studies point to beta's involvement in higher-order linguistic representations. Zioga et al. [104] used MEG and syntactic dependency parsing to study naturalistic story listening. They found that high-level syntactic features explained unique variance in beta-band power beyond the lower-level lexical/acoustic features. These effects, extending beyond peri-Sylvian language areas to frontal, parietal, and motor-related areas, indicate that beta synchrony reflects broader context-integration processes. Frontocentral beta oscillations encode pragmatic intent (e.g., promises, requests) as shown by experimental manipulation using transcranial alternating current stimulation [105]. Thus, moving beyond word-level predictability circumscribed to left lateralised language network [106], extended beta involving the broader frontoparietal and frontocentral multi-demand system has a role in representing predictions in social communicative contexts in preparation for forthcoming inputs.

Evidently, the dynamic and context-dependent predictions mediated by beta activity [27] are not only about lower-level perceptual or motor anticipation but also extend into the social domain (e.g., communication). When individuals share high-level priors about the environment (for example, norms, roles, or expectations of conspecifics), these priors can serve as context-dependent predictions that modulate processing in lower areas. In this way, beta rhythms have been associated with top-down propagation of predictions from higher to lower cortical levels (e.g., in language comprehension: [91]. Further, studies of social and interpersonal processing (e.g., anticipating social incentives) show that alpha-beta networks engage when individuals process shared social context. Thus, when a high-level prior is shared among individuals (for example, a group expectation or cultural norm), the beta rhythm may carry this shared prediction signal, preparing lower-level sensory or cognitive channels to interpret input in line with that common context. When that shared prediction fails (i.e., the social norm is violated, or an unexpected behaviour by a peer arises), the reduction or reset of beta-mediated feedback may allow bottom-up signals (e.g., via gamma) to assert themselves and update the model. In this framework, beta becomes the conduit for social or inter-agent predictive feedback, encoding not only individual expectations but the collective, socially embedded priors that help align perception and action across agents.

In summary, beta oscillations play a role in the top-down influence of generative internal models that prepare cortical regions involved in lexical processing and thus predictability of spoken language. Both in silico modelling (with GPT-2; [107] and observational data in first episode schizophrenia [108,109] indicate that a generative model with poor top-down influence of context simulates tangentiality, i.e., how quickly meaning drifts away in speech, and presents as reduced lexical predictability and thought disorder in psychosis. Taken together, we can expect beta deficits to relate to disorganization in psychosis.

7. Clinical Implications of Beta Oscillations

Schizophrenia is a quintessential illness of failed predictive processing; several lines of evidence indicate a specific failure to generate and maintain beta rhythms and their underlying burst-like dynamics in this illness.

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Sensorimotor beta disruptions are now well established in schizophrenia. The post-movement beta rebound (PMBR) is consistently reduced or delayed in schizophrenia, and in people with subclinical schizophrenia-like features [110–112], indicating a failure to update the internal model that guides predictions. PMBR impairment relates to the degree of disorganization and psychomotor poverty (termed features of classical schizophrenia by Liddle and colleagues) [113,114]. In response to inhibition paradigms (Go/no-go), beta power is reduced in patients and their relatives in association with higher impulsivity [115], again indicating weak predictions and lack of revision of internal models of the present bodily state.

Working memory (WM) impairments in schizophrenia have been linked to beta-band power deficits during both sensory encoding and maintenance, as demonstrated by vibrotactile stimuli by [116]. Delay-period non-suppression of frontoparietal beta occurs in schizophrenia in relation to poor WM performance [117,118]. This represents a failure to use beta bursts to actively maintain the task-relevant context to prepare for upcoming inputs, allowing internal representations to degrade and be interfered with. Rubinstein and colleagues (2023) [119] utilized an n-back task to demonstrate that, unlike healthy controls who exhibited precise temporal desynchronization of frontal beta power prior to correct responses, patients exhibited attenuated and delayed beta desynchronization. Similarly, the detection of task-relevant stimuli relates to higher beta oscillations compared to task-irrelevant stimuli in healthy people, but patients show an inverse pattern, reflecting a degradation of top-down contextual guidance [120]. Briley *et al.* combined EEG and fMRI and noted that patients exhibited fewer beta bursts than healthy control participants during a verbal n-back task. Beta bursts phasically activate fMRI BOLD signal in task-relevant regions while suppressing tonically active (default) regions, supporting the notion that beta bursts reactivate latent content during delay/maintenance of WM. Patients showed attenuated PMBR that varied with the severity of disorganization, greater task-related reductions in overall beta burst rate, but more extensive burst-related BOLD activation [113].

Uhlhaas and colleagues were the first to link context-dependent beta synchronisation deficits to large-scale synchrony and cognitive dysfunctions in schizophrenia [50,121,122]. They demonstrated that Gestalt perception (the grouping of stimuli aided by the integrative context) is diminished in schizophrenia, particularly in relation to decreased beta band power [121]. These perceptual deficits are concomitant with disorganization [123,124]. The repetition of visual stimuli enhances beta power in the occipital cortex of healthy individuals, thus preparing the cortex for a similar stimulus to follow suit. However, this effect is reduced in individuals with schizophrenia, along with reduced gamma suppression, indicating a predictive routing failure that exposes an unprepared sensory cortex to higher activation [125]. A failure to prepare the cortex for an incoming relevant stimulus in the same modality may also be linked to a failure to increase post-task (evoked) beta power during multi-sensory processing [126] and problems in suppressing task-irrelevant beta activity in other instances of stimulus processing [127,128]. Thus, the latent neural representation of movement plan, sensory context and working memory content appears to be less precisely specified in schizophrenia; this likely contributes to disorganised cognition and behaviour.

In contrast to the lack of task-appropriate generation of beta, increased resting beta power occurs in schizophrenia [129–131]; this may relate to a higher level of tonic inhibition contributing to psychomotor impoverishment. Higher 'baseline' beta-power observed after single-pulse transcranial magnetic stimulation (TMS) in the dorsolateral prefrontal cortex (DLPFC) [132] also relates to reduced working memory (AX-CPT) performance. Left lateralized beta increase occurs in relation to a higher burden of negative symptoms [133] and poor treatment response [134].

In summary, beta deficits are not uniform losses of beta power in schizophrenia. Instead, they represent an imprecise use of predictive content in service of ongoing stimulus processing. Bursts are absent when stimulus is awaited (e.g., delay period / maintenance phase in WM), diffuse when specificity is required (i.e., failing to inhibit irrelevant stimuli), or emerge at the wrong time (e.g., delayed desynchronization). Such patterns are a direct physiological instantiation of aberrant predictive routing that may explain imprecise hierarchical processing [135]. Disrupted beta bursts fail to provide the preparatory signals that are necessary to constrain perception and action.

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Consequently, patients' predictive models do not adequately explain how incoming inputs and motor outputs (verbal and nonverbal) become unpredictable. This manifests clinically as disorganized speech [136], impulsive behaviour [115] and impoverished thought [8,114].

8. Therapeutic Opportunities Targeting Beta

Beta burst dynamics depend on dendritic integration supported by NMDA receptor-mediated glutamatergic signalling, GABAergic interneuron activity, especially somatostatin (SST+) and parvalbumin (PV+) interneurons, and dopaminergic influence, particularly evident in subcortical beta oscillations. Pharmacologically, NMDA receptor antagonists like ketamine, which induce schizophrenia-like thought disorder, reliably reduce beta power in prefrontal and sensorimotor regions in humans and rodents [137–140]. NMDA blockade reduces beta amplitude and disrupts burst precision, fragmenting the temporal structure necessary for predictive processing. In rodent sustained-attention tasks, restoring NMDA signalling rescues oscillatory synchrony and cognitive performance [141,142].

Manipulating the dopaminergic system via l-dopa shortens abnormally long beta bursts in Parkinson's disease (PD), restoring a more flexible temporal structure [143–145]. The effects of manipulating striatal dopamine on beta may be mediated via cholinergic and SST+ interneurons [146]. Acetylcholine enhances beta band oscillations and supports cognitive and motor functions, while anticholinergic medications disrupt beta oscillatory dynamics, impairing attention, learning, and network synchrony [147–154] and worsening cognitive functions [155]. Schizophrenia is associated with midbrain phasic dopamine dysregulation, which leads to a reduced beta-oscillatory response to rewards (gain feedback: [156]. Antipsychotics that block dopamine D2 receptors affect beta band during working memory tasks, suggesting partial restoration of disrupted beta desynchronization [119], and reduce the burden of thought disorder [157]. Clozapine, an effective antipsychotic, reverses the ketamine-induced reduction in low beta power [158,159]. However, the effects of antipsychotics on beta burst dynamics are yet to be experimentally studied.

These observations highlight the importance of dissociating beta changes due to primary pathophysiology from the use of concomitant medications in clinical studies. The complex interactions among the neurochemical pathways also highlight the challenges in harnessing beta via pharmacological manipulation in therapeutic settings.

Unlike the diffuse, regionally non-specific effects of pharmacological agents on beta oscillations, more targeted modulation is feasible using transcranial non-invasive stimulation. Of note, most of the repetitive TMS applied therapeutically in schizophrenia provides a beta-frequency stimulation (20Hz; n=22 studies identified in a recent review [160] to the lateral prefrontal or superior temporal regions. But as TMS is delivered without reference to ongoing brain activity (i.e., open loop approach), using it to modify disrupted beta that shows high intra- and inter-subject variability (as discussed above) is unlikely to benefit people with schizophrenia. A closed-loop strategy, with stimulations being time-locked to beta events, might provide a way forward. There are 2 ways to achieve this: one via EEG-triggered TMS (e.g., [161–163] and the other via inducing neural entrainment.

Transcranial alternating current stimulation (tACS) can bias neural spike timing and phase align brain oscillations with external current (entrainment), enabling direct manipulation of beta rhythms and their cross-frequency coupling [164]. Beta-tACS, applied to motor cortex, reduces bradykinesia [165] in Parkinson Disease, while frontocentral beta-tACS improves psychomotor slowing in depression [166] likely by reducing excessive beta synchrony [167,168]. In schizophrenia, tACS trials have mostly targeted gamma or theta to date [169,170]. Combining tACS with TMS [171] and the emergence of behaviourally driven closed-loop tACS strategies are especially promising to target specific beta events [172].

Another emerging avenue involves behavioral and learning-based interventions, particularly EEG-based neurofeedback. Protocols targeting the beta band (typically 12–22 Hz) have demonstrated the ability to modulate beta activity in both healthy individuals [173] and in certain clinical

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populations (ADHD and autism), where theta-beta ratio training protocols improve sustained attention and inhibitory control linked to changes in frontal beta dynamics [174–177].

It is worth noting that neurofeedback lacks spatial precision; and even for tACS, spatial targeting remains imperfect with no current feasibility to apply laminar resolution. Further, long-term safety, efficacy, and acceptability of beta-targeted interventions remain poorly understood.

9. Limitations and Future Directions

While the therapeutic promise of beta-targeted interventions is compelling, several limitations warrant caution. First, most evidence remains correlational, with few causal demonstrations outside PD [145,178–181]. Second, a singular index to quantify the degree of beta deficits that characterize predictive processing abnormalities in schizophrenia is lacking. Though beta is increasingly understood as a sequence of bursts, there is no consensus on how to define or quantify burst metrics across studies, complicating comparisons and impeding translational progress. Third, oscillatory specificity is a challenge. While we have considered beta as a standalone frequency band for the sake of simplicity, this arbitrarily defined beta band functions as an *oscillome*, in which theta, alpha, beta, and gamma rhythms form an interacting network of frequency-specific computations, rather than independent bands acting in isolation. Addressing these challenges through standardized burst metrics, individualized targeting, and longitudinal designs will be essential for moving beta modulation from conceptual promise to robust clinical practice.

To conclude, beta oscillations and their transient burst dynamics represent the critical, observable link between the theoretical notion of predictive processing deficits and the core symptom of thought disorder in psychosis. An inadequate generation and imprecise deployment of beta is linked to the failure to provide the context that constrains perception and action. This framework redefines disorganized thought and behaviour as a failure of predictive routing, providing a tractable, oscillatory target. Beta rhythms offer a unique and modifiable lever for developing causal, circuit-based interventions, from closed-loop neuromodulation to neurofeedback, to restore the preparatory processes necessary for coherent thought and behaviour.

10. Supplementary

Specificity to psychosis: Beta deficits in schizophrenia appear more often in relation to disorganisation and impoverishment of mental activity and behaviours than other symptoms such as delusions or hallucinations, However, beta deficits are not specific to schizophrenia. In major depressive disorder (MDD), elevated resting beta, reduced beta power related to WM tasks [182,183] as well as reduced PMBR emerge as features that co-occur with psychomotor slowing [184] and relate to treatment responsiveness [185,186]. ADHD often shows reduced or mistimed beta activity during tasks requiring attention and inhibition (see [187] for early task EEG; for more recent WM/attention paradigms with burst measures, see [188], reflecting a failure to stabilize goal-relevant states. Parkinson's Disease (PD) exhibits pathological beta synchrony in motor circuits that directly scales with rigidity and slowness, likely due to persistence of bursts [145,189]. PD in fact provides strong, mechanistically informative evidence that too much beta impairs adaptability. Excessive beta synchrony in cortico-basal ganglia networks correlates with rigidity and bradykinesia; treatments that suppress beta (DBS, dopaminergic therapy) alleviate motor symptoms and restore more flexible dynamics [145,178–181]. OCD shows similarities to PD in beta profile, being characterized by excessive beta synchronization, but predominantly in prefrontal rather than motor circuits, likely contributing to cognitive and behavioral rigidity (e.g., [190]). Thus, there are no specific patterns of beta-oscillatory deficits that can be attributed to the presence of psychotic symptoms. Interventions that restore normal patterns of beta physiology in schizophrenia may have broad benefits such as reducing the constellation of symptoms that are shared with other disorders (e.g., especially depressive features and PD-like psychomotor slowing).

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