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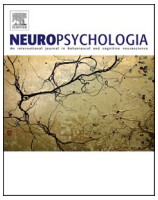

Semantic priming and neurobiology in schizophrenia: A theoretical review

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ABSTRACT

In this theoretical review we bridge the cognitive and neurobiological sciences to shed light on the neurocognitive foundations of the semantic priming effect in schizophrenia. We review and theoretically evaluate the neurotransmitter systems (dopaminergic, GABAergic and glutamatergic) and neurobiological underpinnings of behavioural and electrophysiological (N400) semantic priming in the pathology, and the main hypotheses on their geneses: a disinhibition of the semantic spread of activation, a disorganised semantic storage or noisy lexical-semantic associations, a psychomotor artefact, an artefact of relatedness proportions, or an inability to mobilise contextual information. We further assess the literature on the endophenotype of Formal Thought Disorder from multiple standpoints, ranging from neurophysiology to cognition: considerations are weaved on neuronal (PV basket cell, SST, VIP) and receptor deficits (DRD1, NMDA), neurotransmitter imbalances (dopamine), cortical and dopaminergic lateralisation, inter alia. In conclusion, we put forth novel postulates on the underlying causes of controlled hypopriming, automatic hyperpriming, N400 reversals (larger amplitudes for close associations), indirect *versus* direct hyperpriming, and the endophenotype of lexical-semantic disturbances in schizophrenia.

1. Introduction

Semantic knowledge has been classically conceptualised as an organised network of interlinked nodes (conceptual units), whose proximities are determined as a measure of co-occurrence and shared features (Collins and Loftus, 1975; Kuperberg, 2010a; 2010b). Concretely, nodes could be thought of as corresponding neuronal assemblies, mostly comprised of distributed pyramidal neurons whose evoked firings ultimately elicit a coherent representation, schema, or conceptual unit. Roughly put and as a rule-of-thumb, node proximity could thus follow Hebb's rule: "neurons that fire together, wire together". In cognition, co-occurring nodes gradually strengthen connections so as to facilitate processing of their similars by increasing each other's levels of activation. This automatic, non-conscious pre--activation of proximal nodes is termed *semantic spread of activation*, whereas the measure of this facilitatory effect is referred to as *semantic priming*. Hence, the spread of activation lowers primed nodes' recognition thresholds, insofar as they become more readily available for retrieval in case the need arises shortly thereafter (but swiftly decaying if unattended). Semantic priming is an important means of keeping up with contextual demands in discourse, as it promotes optimal allocation of one's limited cognitive resources towards contextually-congruent information, fine-tuning cognition. It spares time as retrieval is promptly achieved, and cognitive load as retrieval becomes less effortful.

Paradigms used to measure semantic priming are pragmatic and easy to apply. Thus, it is unsurprising that the priming effect has been under research on cognition and language for decades, allowing cognitive psychology and psycholinguistics to thrive a great deal. It is quite regrettable that, in contrast, other niches such as psychiatry and the biological neurosciences have not gleaned nearly as much insight from semantic priming. Arguably, one of the few exceptions can be found in the literature on schizophrenia - a pathology that is notorious for its (profuse) linguistic abnormalities in the domains of semantics and pragmatics (Kuperberg, 2010a; 2010b; Radanovic et al., 2013; Covington et al., 2005; Chaika, 1974). Most of these disturbances are grouped into the *Formal Thought Disorder* (FTD) subsyndrome (schizophrenic language *par excellence*); in broad strokes, the central role of FTD in schizophrenia is so conspicuous that the syndrome was considered pathognomonic for decades (but no longer) (Andreasen and Grove,

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1986).

Importantly, FTD is not simply unidimensional: rather, it is commonly split into Positive (PTD) and Negative (NTD). Whilst NTD is characterised most prominently by poverty of speech, PTD displays abundant and bizarre linguistic semiology, including unclear referencing, peculiar word usage, flights of ideas, verbosity, and most characteristically, "loosening of associations'' (Kuperberg, 2010; Bleuler, 1911). Thus, PTD has been the main focus of research on schizophrenic language for years, to the extent that language production in acute PTD even goes by "schizophasia" (reminiscent of Wernicke's fluent aphasia, e.g., word salad, poor semantic/lexical accuracy or "paraphasia", neologisms, vagueness).

Critically herein, PTD's most outstanding feature is the apparent "enhancement" or "disinhibition" of the semantic spread of activation, which appears to be swifter and broader than average, i.e., greater *facilitation* of semantic nodes, or a larger attenuation of reaction times to related targets relative to unrelated ones (which is not to say that mean reaction times are faster in patients than controls) (Spitzer et al., 1993, 1994bib_Spitzer_et_al_1993bib_Spitzer_et_al_1994; Manschreck et al., 1988; Kiefer et al., 2009; Kreher et al., 2008, 2009bib_Kreher_et_al_2008bib_Kreher_et_al_2009; Pomarol-Clotet et al., 2008; Henik et al., 1995; Weisbrod et al., 1998; Gouzoulis-Mayfrank et al., 2003; Safadi et al., 2013; Wentura et al., 2008). Such semantic disinhibition is generally thought to underlie automatic *hyperpriming* effects, most commonly reported among PTD patients (i.e., increased semantic priming) (e.g., Kreher et al., 2008, 2009; Pomarol-Clotet et al., 2008; Spitzer et al., 1994; Manschreck et al., 1988; Kiefer et al., 2009; Henik et al., 1995; Weisbrod et al., 1998; Kuperberg et al., 2019; Kwapil et al., 1990; Moritz et al., 1999, 2001a, 2001b, 2003; Safadi et al., 2013; Gouzoulis-Mayfrank et al., 2003; Wentura et al., 2008; Salisbury, 2008). Yet, semantic disinhibition does not seem to successfully or integrally explain the phenomenon, because hyperpriming effects are heterogeneous. Specifically, hyperpriming in schizophrenia, especially in PTD, is typically observed with distant semantic relations (Kreher et al., 2008, 2009; Spitzer et al., 1993, 1994; Manschreck et al., 1988; Weisbrod et al., 1998; Moritz et al., 1999, 2001a, 2001b, 2003; Safadi et al., 2013; Gouzoulis-Mayfrank et al., 2003; Wentura et al., 2008; Pomarol-Clotet et al., 2008; Kuperberg et al., 1998, 2006a, 2006b; see also Kwapil et al., 1990), whereas findings on closer associations are more equivocal - ranging from hypo- (Pomarol-Clotet et al., 2008; Kreher et al., 2009; Minzenberg et al., 2003; Aloia et al., 1998; Passerieux et al., 1997; Rossell and Stefanovic, 2007; Henik et al., 1992; Besche et al., 1997; Besche-Richard and Passerieux, 2003; Ober et al., 1995; Barch et al., 1996; Hokama et al., 2003) to normal/no correlation (e.g., Barch et al., 1996; Chapin et al., 1989, 1992; Vinogradov et al., 1992; Tan et al., 2015, Tan and Rossell, 2017; Kreher et al., 2008, 2009; Kuperberg et al., 1997, 2006a, 2006b, 2019; Besche-Richard and Passerieux, 2003; Blum and Freides, 1995; Rossell, 2004; Passerieux et al., 1995) to hyperpriming (e.g., Gouzoulis-Mayfrank et al., 2003; Moritz et al., 1999; Manschreck et al., 1988; Kiefer et al., 2009; Kreher et al., 2008, 2009; Pomarol-Clotet et al., 2008; Rossell and David, 2006; Henik et al., 1995; Gouzoulis-Mayfrank et al., 2003; Spitzer et al., 1994; Weisbrod et al., 1998; Safadi et al., 2013; Neill et al., 2014; Salisbury, 2004, 2008). While this peculiar anomaly may seem compatible with the aforementioned "loosening of associations", it has fueled significant theoretical debate, with alternative views most notably encompassing suggestions that these are artefacts of psychomotor slowing (Pomarol-Clotet et al., 2008; Rossell and Stefanovic, 2007), relatedness proportions (Vinogradov et al., 1992; Rossell and Stefanovic, 2007), caused by looser lexico-semantic networks (Kuperberg et al., 2019) or disorganised semantic storage (Tan et al., 2015, Tan and Rossell, 2017; Rossell and David, 2006), or by an inepsy to mobilise contextual information (e.g., Hardy-Baylé et al., 2003; Brown and Kuperberg, 2015; Titone et al., 2000; Kuperberg, 2010a, 2010b; Sitnikova et al., 2002; Meyer et al., 2021; see also Sharpe et al., 2020) (see section 6). Furthermore, controversies on hyperpriming range even further, seeing that, especially with certain methodologies (controlled, explicit tasks), schizophrenic patients often exhibit a diametrically-opposed effect - *hypopriming* (Pomarol-Clotet et al., 2008; Kreher et al., 2009; Minzenberg et al., 2003; Ober et al., 1995, 1997bib_Ober_et_al_1995bib_Ober_et_al_1997; Aloia et al., 1998; Passerieux et al., 1997; Henik et al., 1992; Barch et al., 1996; Besche et al., 1997; Besche-Richard and Passerieux, 2003; Salisbury, 2008; Hokama et al., 2003). All in all, it is easy to see why debate on these issues remains ongoing.

For all the theoretical debates, however, a very important question is not being asked: what can priming disturbances unveil about the biological endophenotypes of schizophrenia and PTD? Herein, we address this question by critically evaluating the literature on the neurobiology of semantic priming in schizophrenia and PTD from its multifarious levels of approach - which include neuroimaging, dopaminergic/linguistic lateralisation, pharmacological studies, behavioural paradigms, lexical-semantic (N400) evoked-related potentials (ERPs), among others.

Empirical data endorse the idea that the spread of activation is "narrowed" by dopaminergic agents (and possibly GABAergic), restraining priming of distant semantic relations. Inasmuch as dopaminergic (and GABAergic) drugs enhance lateral inhibition in the cortex, they seem to preclude access to spurious "semantic nodes" or neuronal assemblies. Evidence also suggests that a particular type of GABAergic interneuron is of the utmost importance for this effect - namely, parvalbumin-positive (PV) interneurons (a highly prevalent cortical cell type that is well-known for its involvement with schizophrenia, e.g., Lewis et al., 2012; Gonzalez-Burgos et al., 2015; Kaar et al., 2019). Semantic disinhibition may primarily stem from a dysfunction of PVs and Martinotti cells (MCs), which could arise with the contribution of hypofunctioning D1 receptor (DRD1) and/or NMDA receptors (NMDARs). Conjecturally, the very same disinhibition may give rise to controlled processing impairments through semantic interference (an effect typically observed in explicit, controlled tasks) and/or a process of "semantic crowding", whereby top-down suppressive effects over noise/irrelevant nodes are rendered excitatory due to cortical disinhibition in target regions - thereby resulting in semantic indiscrimination and obliteration of contextual nuance in controlled processing. Thus, conforming with Occam's razor, cortical disinhibition might underlie both implicit hyperpriming (automatic) and explicit hypopriming effects (controlled) - with PVs and MCs potentially holding pivotal roles. Multiple putative aberrations that have been previously deemed contradictory with semantic disinhibition (e.g., disorganised semantic storage) will also be shown to be complementary. Finally and in addition to reviewing its literature, hypotheses on the neural substrates of N400 reversals in schizophrenia will be put forth on the basis of extant neurobiological models (Almeida, 2021a; Bornkessel-Schlesewsky and Schlesewsky, 2019; Kotchoubey, 2006).

Altogether, the present theoretical review explicitly favours a neurobiological scope through which it will interpret the lexical-semantic processing literature on schizophrenia. We will objectively review the extensive data on lexical-semantic distortions in the pathology, concomitantly undertaking a more arbitrary process of problem-solving: to articulate it with the neurobiological sciences. Our ultimate goal, therefore, is to paint a cohesive picture on the neurobiological underpinnings of lexical-semantic processing in schizophrenia, and unravel its potential significance for neurolinguistics and psychiatry.

2. Essential framework

2.1. Semantic priming

To start off, let us briefly introduce some fundamentals of semantic priming effects. Priming may be direct or indirect. Direct priming occurs when a prime word is directly related to a preceding word (e.g., lion - *tiger*), whereas indirect links are mediated by another semantic node (e.g., lion - tiger - *stripe*). These phenomena may be probed with multiple

paradigms, which are subdivided into explicit and implicit. Explicit tasks require attention, and generally, behavioural response and decision-making - the canonical example being *lexical decision*. Traditionally, a word flashes on screen for a few milliseconds (prime), after which a new word *or* pseudoword appears (target); participants must judge as quickly as possible whether the target is an existing word, pressing a button to signal their decision. If the target is primed, this decision will be made faster than usual. Conversely, implicit tasks drive attention away from the experimental item and/or its relevant features. For instance, participants may be required to monitor prime-target pairs for filler words in a category (e.g., food) under the recordings of functional MRI, electroencephalograms (EEG), eye-trackers, and so forth. Ideally, decision-making processes are avoided.

The main difference in using explicit tasks is that these necessarily involve some non-negligible degree of controlled processing (Kreher et al., 2009; Neely, 1991), whereas implicit ones are better suited to isolate automatic processing.

2.2. Automatic and controlled processing

Semantic memory access is traditionally subdivided into automatic and controlled: it is thought to be attained through an *automatic spreading activation* or a *limited-capacity attentional mechanism* (Posner and Snyder, 1975). Automatic processing precedes controlled processing, though it does not cease once the latter transpires. Automatic semantic processing takes place almost immediately after the arrival of bottom-up afferences. Accordingly, automatic semantic processes originate in posterior temporal regions that correspond to the stimulus' modality. Acoustic stimuli will be initially processed dorsally, in areas like the Superior Temporal Gyrus (STG), whereas visual stimuli will first engage ventral stream regions like the posterior Fusiform Gyrus (FG) and Inferior Temporal Gyrus (ITG). In a matter of milliseconds, activation will converge forwardly towards (mostly) an anterior cluster of regions that are multimodal/amodal, including the temporal pole and its vicinities (Ralph et al., 2017; Jackson et al., 2018; Binder and Desai, 2011). Thereafter, controlled processing begins to take shape, and a number of other anterior and temporoparietal regions may be recruited, including the Anterior Cingulate (ACC), the posterior middle temporal gyrus (MTG), the inferior parietal lobule (IPL), and notoriously, the Inferior Frontal Gyrus (IFG) or "Broca's area". This is all but a rough panorama, however: purely automatic processing can also employ these regions in parallel (e.g., Kotz et al., 2002; Copland et al., 2003b). In summary, the modulatory influence exerted by controlled processes over semantic processing is referred to as "top-down", as opposed to "bottom-up", which propagates from sensory levels "upwardly" towards higher order cognition.

Finally, semantic priming is an automatic phenomenon (resulting from an automatic spread of activation in semantic memory), but, as previously mentioned, it may be influenced by controlled mechanisms of attention and expectancy. For instance, by paying attention to a developing sentence *Let's head*, one may prime nodes for *downtown* or *to the pub* depending on contextual circumstances, even though the sentence, if stripped from its context, might prime differently - hence, an explicit task like lexical decision may not efficiently isolate semantic priming from kindred top-down influences. A measure that mitigates some of these top-down modulations is the shortening of the *stimulus onset asynchrony* (SOA) - the interval between prime onset and target onset -, supposedly making it too short for significant controlled processes to evolve between prime and target onsets. Still, the same measure may be used for implicit tasks. Furthermore, processes involved in post-lexical decision-making such as semantic matching (an assessment of whether there is a relation between prime and target) (e.g., de Groot, 1984), ensuing between the target's onset and the lexical decision itself, remain largely unaffected by SOA changes. Whilst some of these can be tackled, for example, by reducing the proportion of related word pairs, even this measure does not fully circumvent post-lexical processing interference over explicit behavioural performance.

2.3. Semantic interference

A drawback to "priming" is *semantic interference* (Oppenheim et al., 2010). When multiple alternatives compete with each other, the cost for conflict resolution/selection of a single item increases. This effect is most conspicuous in the face of demands for selection/behavioural response (e.g., Nozari and Hepner, 2019; Oppenheim and Balatsou, 2019). Generally, similarity, rather than unrelatedness, causes semantic interference (Fieder et al., 2019). Hence, "priming" too many nodes could be detrimental, as these nodes may require suppression so as to viabilise the selection of a desired response. This conflict resolution is thought to rely heavily on the left IFG (e.g., Grindrod et al., 2008).

A strong candidate - but not the only one - for promotion of conflict resolution is loosely referred to as *lateral inhibition* (Jung et al., 2017; Oppenheim et al., 2010) - in the brain, this is a way in which interneurons (inhibitory cells that secrete GABA, the main inhibitory neurotransmitter) inhibit neighbouring cells. This idea holds the advantage to be, biologically, quite plausible. Through this prism, interneurons should be crucial to resolving semantic interference by allowing competition to sharpen semantic representations into a coherent whole, suppressing competitors to enhance the appropriate node's salience (Snyder et al., 2010). Accordingly, studies report that GABA levels in the left IFG (Nakai and Okanoya, 2016) and anterior temporal lobe (Jung et al., 2017) strongly mediate semantic processing and interference resolution.

An excellent study and model by Snyder et al. (2010) raised relevant points concerning these nuanced dynamics. They initially outline the difference between *retrieval* demands and *selection* demands. When choosing words, they postulated that high *retrieval* demands would imply that a word is hard to find due to its weak association with a prime (e.g., *giraffe – eat* as opposed to *scissors – cut*), whereas high *selection* demands imply that there is a great number of competing alternatives (e.g., *cat* is profusely associated with other nodes, whereas *scissors* may be more restrictive). Hence, under *high retrieval demands*, activating multiple nodes would be fruitful, as these would create a greater chance that some of them facilitate the activation of a suitable one; on the other hand, under *low retrieval demands*, activating multiple (similar) nodes would be detrimental, as these would unnecessarily *increase selection demands* (i.e., the need to inhibit competing alternatives) for a viable option (Snyder et al., 2010). Accordingly, in a language task where participants were asked to reply with the first verb that came to mind after the presentation of a noun (e.g., *scissors – cut*), the authors showed that participants suffering from anxiety (associated with low GABA function) improved their performance after taking midazolam, a GABA-AR allosteric modulator (i.e., a drug that augments the receptor's affinity, in this case, for GABA), and this enhancement was observed *only* when retrieval demands were *low* (i.e., when selection demands were high). In other words, the role of GABA in attenuating interference through lateral inhibition might have been endorsed, as midazolam improved performance specifically when multiple alternatives were available and interfering with the task.

3. Dopamine modulates semantic priming: the signal-to-noise ratio

The signal-to-noise ratio is reduced in schizophrenia (Winterer and Weinberger, 2004; Winterer et al., 2000; Maher et al., 1983). This ratio is the proportion of coherent information (signal) detectable in the face of irrelevant entropy (noise). Thus, relevant nodes constitute signal, and irrelevant ones constitute noise in the spread of activation. An excessively broad spread would therefore be rated with low signal-to-noise. Likewise, PTD is essentially a disorder in which the spread of activation is very "noisy", unduly eliciting distant, loosely connected nodes - something that likely either underpins or shares a similar

pathophysiology with a feature Bleuler (1911) himself had noticed, namely, the conceptually "loose associations" made by his patients.

An optimal neurochemical balance is crucial for maintaining an optimal signal-to-noise ratio, with dopamine having been shown to play an essential role (Winterer and Weinberger, 2004). Provided levels are at an optimal, dopamine seems to narrow the spread. In semantic priming paradigms, this translates into increases in selectivity for close associations (arguably with an enhancement of direct priming), as per the attenuations of indirect priming effects (and/or other subordinate/irrelevant relations) (Lavigne and Darmon, 2008; Mohr et al., 2006; Copland et al., 2003a, 2009bib_Copland_et_al_2003abib_Copland_et_al_2009; Roesch-Ely et al., 2006; Spitzer et al., 1993; Arnott et al., 2001; Kischka et al., 1996; Chenery et al., 2008; Angwin et al., 2004, 2005bib_Angwin_et_al_2005; Coplanda et al., 2006bib_Angwin_et_al_2004; Mohr et al., 2006, 2006bib_Mohr_et_al_2006). An illustrative randomised, double-blind study with 31 healthy volunteers by Kischka et al. (1996) is one of the early examples to report this. The authors administered either 100 mg of the amino acid L-3,4-dihydroxyphenylalanine (L-Dopa) - immediate precursor to dopamine synthesis in the catecholaminergic sequence - with 25 mg benserazide or a placebo to the participants, who were subsequently asked to engage in a lexical decision task in conditions with different SOAs - 700 ms, and 200 ms (as a control condition). Significant indirect priming effects were not found for either group in the 200 ms SOA condition. The main finding was that indirect priming effects on the 700 ms condition were reduced under L-Dopa; of note, another study also reported increased direct and indirect priming under L-Dopa, but this was strictly due to a prolongation of reaction times to unrelatedness (Mohr et al., 2006), insofar as these findings remain consistent with a dopaminergic role in noise suppression. Although one may question whether the effect in Kischka et al. (1996) actually stems from a norepinephrine (NE) increase (NE synthesis too is posterior to L-Dopa in the catecholamine chain), a study failed to find the same reductions with NE modulation (Cios et al., 2009). Additionally, Gamo et al. (2010) examined the effects of atomoxetine - which raises extracellular dopamine levels and also acts as a NE reuptake inhibitor (i.e., it keeps NE from being cleared away, raising its levels) - on prefrontal enhancement in monkeys, and found that it raised the signal-to-noise ratio by both decreasing firing to non-preferred directions (reducing noise) and increasing persistent firing of relevant neurons (increasing signal), but it was the noise reduction that vanished by application of the selective DRD1 antagonist SCH2 3390, whereas the persistent firing was affected by α2-adrenoreceptor antagonist yohimbine. Evidently, this research implies that the DRD1 subtype, specifically, plays the dominant role in the dopaminergic modulation of signal-to-noise ratios, eliminating noise. Indeed, albeit not invariably, this conclusion is supported by a plethora of further studies on dopaminergic modulation of signal-to-noise ratios (e.g., see Arnsten, 2006; Winterer and Weinberger, 2004; Bensmann et al., 2018, 2020; Roesch-Ely et al., 2006; but see Stalter et al., 2020).

As PTD patients display an overly coarse spread of activation, early authors proposed a prefrontal hypodopaminergia as the endophenotype (Spitzer et al., 1993; see also Cohen and Servan-Schreiber, 1992). Indeed, optimal dopamine concentrations and phasic release have been shown to raise the thresholds for neuronal firing, rendering spontaneous, "spurious" spikes less likely and building up "energy barriers" between neuronal assemblies, enhancing fine cognitive processes (Durstewitz and Seamans, 2008) and fine semantic discrimination (Lavigne and Darmon, 2008; Kischka et al., 1996; Spitzer et al., 1993). The "hypodopaminergia" proposal is also in line with others in the schizophrenia literature. Selective DRD2 antagonists (e.g., haloperidol) attenuate positive symptoms (e.g., delusions, pressured speech, hallucinations) by acting mostly in the limbic system. This observation has led to the hypothesis that positive symptoms arise as a consequence of hypodopaminergic-related "hypofrontality", where mesolimbic dopaminergic activity is rendered hyperactive due to a lack of proper prefrontal modulation (Davis et al., 1991). Dopaminergic-related hypofrontality could help enlighten why FTD often co-occurs with affective disturbances (e.g., Park et al., 2018; Minor et al., 2016; Kemp et al., 2018; Marggraf et al., 2019). Notwithstanding, *hyper*-dopaminergia can also lead to an overall suppression of prefrontal firing, as excessive DRD1 stimulation itself suppresses neuronal activity (Vijayraghavan et al., 2007). Therefore, albeit the literature has focused on hypodopaminergic tones, surmising that neocortical hyperdopaminergia would not be accompanied by at least some measure of PTD semiology is premature.

4. The left hemisphere

At least concerning the spread of activation, the signal-to-noise-enhancing effects of DRD1s may be particularly prominent in the left hemisphere (LH), as noticed by Roesch-Ely et al. (2006). With 40 healthy subjects, the authors compared effects of dopaminergic agonists 2.5 mg bromocriptine (D2), 0.1 mg pergolide (DRD1 and DRD2), and a placebo, in a *lateralised* lexical decision task (SOA = 750 ms). They split the experiment into two conditions, one of which had words presented to the right visual field, and the other to the left. The reason this measure was taken is that there might be an asymmetrical hemispheric contribution to priming effects. Although not unequivocally, the LH seems more selective in processing close relations in priming paradigms, with prime-target relatedness increasing leftward activation, while the right hemisphere (RH) notoriously processes less conventional and novel associations (Weisbrod et al., 1998; Kiefer et al., 1998; Nakagawa, 1991; Burgess and Simpson, 1988; Lavigne and Darmon, 2008; Hutchinson et al., 2003). Indeed, as an *en passant* note and in confluence with these ideas, the RH generally operates on lower-frequency EEG oscillations, especially in the theta band, which is famously tied to semantic distance and non-conventional associations; on the other hand, the gamma band is more typical of the LH, and it is not only more spatially localised than theta (suggestive of a more stringent spread of neuronal activity), but also linked to predictability and coherence (Giraud and Poeppel et al., 2012; Thompson et al., 2016; Morillon et al., 2012; Poeppel, 2003; Spironelli and Angrilli, 2015; Lam et al., 2016; Mellem et al., 2013; van Ackeren et al., 2014; Wang et al., 2012; Hagoort et al., 2004; Herrmann et al., 2004; Obleser and Kotz, 2011; Monsalve et al., 2014; Solomon et al., 2019; Sun and Dan, 2009; Kienitz et al., 2018; Rey et al., 2014; Romei et al., 2011). Thus, the LH debatably partakes more selectively in the processing of closer associations, activating a few nodes and more strongly so, whilst the RH coarsely and less intensely activates both close and distant relations (Lavigne and Darmon, 2008; Weisbrod et al., 1998; Hutchinson et al., 2003; Kiefer et al., 1998; Nakagawa, 1991; Yochim et al., 2005; Beeman et al., 1994; Burgess and Simpson, 1988; Chiarello et al., 2001; but see Coney, 2002). Yet, Roesch-Ely et al.'s experiment did not find increased indirect priming in the RH. Rather, their results pointed to a quantitative reduction of indirect priming effects under pergolide (DRD1 agonist) but not bromocriptine, with these repercussions being found in the right visual field (LH) condition only, which is arguably suggestive of a narrower spread in the LH (so long as dopaminergic function is optimal). Other studies using L-Dopa also reported kindred, lateralisated modulations to the left (De Letter et al., 2012; Mohr et al., 2006).

Hence, on top of DRD1 stimulation reducing the spread's "perimeter" (Roesch-Ely et al., 2006; Arnsten, 2006; Winterer and Weinberger, 2004), we assembled data suggesting these modulations might be hosted preferentially by the LH (at least for behavioural measures). The evidence presented so far, however, only suffices to posit that dopamine focuses the spread of activation during controlled processing or its time windows, as priming experiments that manipulated dopamine were all explicit. This leaves a gap to be filled as to whether or how phasic dopamine release would impinge on purely automatic measures. Suggestively, in lieu of reverberating on early automatic processes (e.g., failing to impact the Mismatch Negativity, N1, P1), to our knowledge, other fields of research mostly demonstrate a DRD1 modulation of

controlled processes (e.g., the P3 late positive component) (e.g., Bensmann et al., 2018, 2020; Leung et al., 2007; Hansenne, 2000). Indeed, we should stress that this selectivity is not implausible. As a brief example, the DRD1 subtype is generally thought to function phasically (e.g., Dreyer et al., 2010). If substantial phasic dopaminergic release from the Ventral Tegmental Area (VTA; midbrain area richly compounded by dopaminergic neurons that supply the cortex) were to be contingent on, say, conscious engagement through frontal feedback, we would only see a DRD1-driven focusing of the spread in explicit tasks. Accordingly, for instance, output from the medial frontal cortex, one of the "seats of consciousness" (e.g., Dehaene and Changeux, 2011), is exceptionally efficacious at triggering massive VTA dopaminergic release (Lodge, 2011).

Finally, it is noteworthy that another study reported that, in a 3-word pronunciation priming task, L-Dopa *accelerated* reaction times to incongruent and subordinate targets of ambiguous words at short SOAs of 150 ms (Andreou et al., 2014). This is at odds with findings discussed so far. One way to conciliate this study with the above literature is to suggest that L-Dopa could, via a non-selective increase in dopamine in multiple brain areas (e.g., the striatum), exert multifaceted influence on semantic priming via mechanisms other than cortical DRD1 stimulation (e.g., altering functional balance between striatal DRD1s and DRD2s, cortical DRD4s, etc.), occasionally confounding results. Further research must be conducted on this matter - particularly because L-Dopa enhanced semantic priming for distant associations in short SOAs, which is precisely what is observed in schizophrenia. All in all, though, dopamine more reliably augments signal-to-noise ratios and squashes activation of distant lexical-semantic associations (Lavigne and Darmon, 2008; Mohr et al., 2006; Gamo et al., 2010; Copland et al., 2003a, 2009bib_Copland_et_al_2003abib_Copland_et_al_2009; Roesch-Ely et al., 2006; Spitzer et al., 1993; Arnott et al., 2001; Chenery et al., 2008; Angwin et al., 2004, 2005bib_Angwin_et_al_2005; Coplanda et al., 2006bib_Angwin_et_al_2004).

5. Positive Thought Disorder: A mechanistic perspective

We may now address PTD and its characteristic semantic phenomena, *tête-à-tête*. The outcome of the research on the spread of activation in PTD translates into empirical conflicts. Reports range from hypopriming, to undifferentiated priming, to hyperpriming effects (e.g., Kreher et al., 2008, 2009; Kuperberg et al., 1998, 2007, 2018, 2019; Kuperberg, 2010a; 2010b; Henik et al., 1995; Spitzer et al., 1994; Pomarol-Clotet et al., 2008; Minzenberg et al., 2002, 2003; Wang et al., 2011; Safadi et al., 2013; Gouzoulis-Mayfrank et al., 2003; Tan et al., 2015, Tan and Rossell, 2017; Rodríguez-Ferreiro et al., 2020; Manschreck et al., 1988; Weisbrod et al., 1998; Maher et al., 1983, 1987; Kwapil et al., 1990; Moritz et al., 1999, 2001a, 2001b, 2001c, 2003; Wentura et al., 2008; Blum and Freides, 1995). As of today, the predominant, most adhered to view postulates there is an enhanced spread in PTD, swifter and broader than normal (Kreher et al., 2008, 2009bib_Kreher_et_al_2008bib_Kreher_et_al_2009; Spitzer et al., 1993, 1994bib_Spitzer_et_al_1993bib_Spitzer_et_al_1994; Manschreck et al., 1988; Weisbrod et al., 1998; Kwapil et al., 1990; Moritz et al., 1999, 2001a, 2001b, 2003bib_Moritz_et_al_1999bib_Moritz_et_al_2001abib_Moritz_et_al_2001bbib_Moritz_et_al_2003; Safadi et al., 2013; Gouzoulis-Mayfrank et al., 2003; Wentura et al., 2008). One of the pioneering works in this area was Spitzer et al. (1993), who stressed that indirectly related concepts elicit hyperpriming more easily than direct ones, and especially in automatic conditions**.** Indeed, in spite of studies reporting direct/close hyperpriming for PTD and schizophrenic cohorts (e.g., Manschreck et al., 1988; Kiefer et al., 2009; Kreher et al., 2008, 2009; Pomarol-Clotet et al., 2008; Henik et al., 1995; Gouzoulis-Mayfrank et al., 2003; Spitzer et al., 1994; Rossell and David, 2006; Weisbrod et al., 1998; Safadi et al., 2013; Moritz et al., 1999; Neill et al., 2014), a large number also reports no such effect, at times even reporting decreased priming, or even when indirect/distant hyperpriming is observed (Chapin et al., 1989, 1992; Kreher et al., 2008, 2009; Vinogradov et al., 1992; Barch et al., 1996; Spitzer et al., 1993; Kuperberg et al., 1997, 2018, 2019; Minzenberg et al., 2002; Wang et al., 2011; Tan et al., 2015, Tan and Rossell, 2017; Henik et al., 1992; Ober et al., 1995, 1997; Chenery et al., 2004; Neill and Rossell, 2013; Rossell et al., 2000; Moritz et al., 2001a; see also Morgan et al., 2006a for a comparison between schizotype groups). Nonetheless, such divaricate in empirical findings may partially owe to the fact that multiple studies did not assess FTD *per se* (not to mention the staggering variety of scales, e.g., TALD, TLC, SPQ, PANSS), or did only mild FTD samples and/or used long SOAs. Yet, notwithstanding the confounders, a meta-analysis by Pomarol-Clotet et al. (2008) mustered evidence supporting generally increased priming in FTD as compared to healthy controls, though this effect was much smaller in comparison to other schizophrenic cohorts. Still, the small subset of studies that also probed indirect associations reported hyperpriming homogeneously (yielding the greatest effect size). In other words, there seems to be a somewhat clear-cut difference between indirect and direct hyperpriming effects in PTD. Critically, primarily short SOAs were linked to increased priming effects of either type (direct or indirect) in this meta-analysis - thereby hinting at a disinhibition of the implicit, automatic spread of activation (it is interesting to note, however, that in offline paradigms such as sentence acceptability, FTD patients have also demonstrated insensitivity to linguistic violations, e.g., Kuperberg et al., 1998, 2006a, 2006b; Dwyer et al., 2014). This, in turn, might suggest that dopamine is not the prime force behind such disinhibition *if* its phasic release mostly sways controlled processes (Bensmann et al., 2018, 2020bib_Bensmann_et_al_2018bib_Bensmann_et_al_2020; Leung et al., 2007; Angwin et al., 2004) (though we cannot rule out homeostatic aberrations arising from hypodopaminergia, for example), although it remains perfectly plausible that hypodopaminergia should interfere with performance in explicit paradigms. Such an issue, again, warrants further scrutiny on whether there is substantial dopaminergic modulation of the automatic semantic spread of activation.

In that vein, another hallmark was a paper issued by Kreher et al. (2009). The authors quite convincingly demonstrated that many equivocal findings in regards to variations between hypo- and hyperpriming were attributable to the employment of explicit tasks, where PTD is prone to show hypopriming due to handicaps in controlled processes (indeed, controlled impairments in schizophrenia are reported in a variety of other fields, Berkovitch et al., 2017). While previous authors mainly focused on shortening the SOA to find hyperpriming, explicit tasks were still used that failed to properly isolate controlled from automatic processes. Kreher et al. maintained a constant SOA of 350 ms and showed that, by simply switching the task from lexical decision to implicit monitoring for filler words in a category (under the recording of an EEG), hypopriming was replaced with hyperpriming in the very same patients. Regrettably, studies so far are yet to join this implicit approach.

Before we move on to discuss data on the endophenotype and electrophysiology, we will provide a mechaninstic take on the above phenomena. Accordingly, herein, it is opportune for us to allude once again to the "semantic interference" effect. As aforedescribed, semantic interference is most conspicuous in the face of demands for behavioural response, in conditions where too many nodes are pre-activated (see Section 2.3). Consequently, one defensible hypothesis is that controlled hypopriming would stem from semantic interference. That is, the same disinhibited spread of activation that "enhances" automatic access to distant associations would also disrupt controlled semantic processing by rendering it overly noisy, protracting the process of word selection. Out of two candidates, this would be the simpler hypothesis we have to offer. The more elaborate one is the following.

The fulcral difference between explicit and implicit tasks lies in attention to relevant features/conscious engagement - i.e., explicit processing presupposes these, above all else (Eysenck and Keane, 2020). In the cortex, attention and overall top-down deployment means feedback projections stemming from higher-order cortices that target

downstream regions, where their most established effect is to amplify target cortical nodes and suppress surrounding noise (e.g., LaBerge, 2005; Störmer and Alvarez, 2014; Boehler et al., 2009; Schwartz and David, 2018) (this should apply to most top-down operations, by and large driven by excitatory corticocortical and transthalamic feedback targeting apical dendrites in superficial layers, LaBerge, 2005). Hence, top-down, attentional engagement would be taken as a perfect remedy for reinstatement of signal-to-noise ratios. Yet, we argue otherwise - in PTD, this is precisely what harms performance.

In visual search paradigms, reaction times typically increase with the number of distracters (Li, 2002). This delay does not happen because the target is missing - its image hits the retina. Rather, it is thought by many that the internal saliency map built upon inputs from the occipital cortex has coordinates - saliency - too muddled to promptly route attention correctly (e.g., Soltani and Koch, 2010; Li, 2002). This retardation reaches its apex when distracters share features with the target - i.e., when distracters and targets are resemblant of one another (Li et al., 2002; Wienrich et al., 2009). For example, Wienrich et al. (2009) administered a visual search feature paradigm to humans; they varied the target-distracter similarity - thereby, the bottom-up saliency map - and found that as distracter-similarity increases, subjects exhibit longer reaction times, accompanied by corresponding behavioural patterns such as longer fixation durations, more reinspections/refixations, etc. In turn, a very interesting neurocomputational study performed by Soltani and Koch (2010) postulated that such saliency is generated by a synergistic interaction between lateral excitation and inhibition, which accentuates the difference between targets and distracters as the signal travels the cortical hierarchy (see also Gong and Theeuwes, 2021). This seems critical, as cortical disinhibition is quite typical of schizophrenia (e.g., Koukouli et al., 2017; Lewis et al., 2012; Alherz et al., 2017). We suggest that in PTD, the disinhibition of the spread of activation in multiple levels of the cortical hierarchy makes it so that the feedforward sweep (i.e., first waves of neuronal activity that cover the entire cortex) conveys unreliable information to the higher-order cortices. In healthy cohorts, this sweep is what carries the "spiking data" that can track feedback to its targets (like a "semantic saliency map", e.g., Li, 2002; Kayser et al., 2005; Treue, 2003; Ptak, 2012), but in PTD, such a "saliency map" is muddled with spurious spikes (noise). The disinhibited (poor lateral inhibition) temporal lobe's map dazes regions like the IFG and frontoparietal network that project back to the temporal lobe to amplify target nodes (e.g., semantic selection is reliant on the posterior MTG, Gottlieb, 2007). Furthermore, cortical feedback is dispatched from layer V, where both DRD1s and PVs (a protagonist driver of lateral inhibition) are pronouncedly expressed (thereby likely working to narrow down output and acuity) (e.g., Gaspar et al., 1995; Gorelova et al., 2002; Wang and O'Donnell, 2001; Anastasiades et al., 2019), both being hypofunctional in schizophrenia. FTD is even correlated with disinhibition of the parietal lobe, as well as white matter alterations in frontoparietotemporal pathways (Cavelti et al., 2018; Horn et al., 2009). In other words, coordinates from the feedforward sweep are misleading, and feedback itself is imprecise.

The outcome is that PTD can "detect" the target "from the distance", in low resolution, but cannot readily compute its semantic features explicitly. In this "*semantic crowding*" scenario, local disinhibition allows the imprecise distribution of attentional drive to further diffuse horizontally, blurring functional contrasts between neuronal assemblies (semantic nodes). A globally stronger (absolute drive), and yet locally weaker (signal) *tabula rasa* is built - attention effectively *multiplies* distracters (see also Winterer et al., 2000). The dynamic range of coding is shortened even at the spatial and populational level, as cortical PV disinhibition renders pyramidal recruitment incredibly easy to saturate (Pouille et al., 2009). Further, as compared to implicit access, explicit access and behavioural response are robustly associated with a nonlinear and long-lasting surge in cortical activity termed *cortical ignition* (in fact, semantic activation tends to be larger with longer SOAs, whereas in automatic processing it is generally thought to be volatile and decay quickly, Kiefer and Spitzer, 2000; Hill et al., 2005; Posner and Snyder, 1975) (e.g., Dehaene and Changeux, 2011; Dehaene and Naccache, 2001) - which should run amok in PTD. Resemblant of all this, visual crowding (which makes cortical ensembles look all the same) has been conceptualised as a result of unfocused, imprecise, coarse and/or low-resolution attentional feedback, which spreads indiscriminately and entails non-selective amplification of network nodes (for more, see Fang and He, 2008). Indeed, in Fang and He (2008), only attended, not unattended stimuli, were associated with crowding. In a similar vein, the hypothesis put forth herein is that only implicit and automatic tasks can preserve PTD's performance (Kreher et al., 2009) - effectively by driving attention (and other top-down projections) away from target representations. Interestingly, these ideas might be endorsed by reports of a correlation between schizophrenia and hyperactive haemodynamic response to semantic associations in long SOAs (Vistoli et al., 2011; Jacob et al., 2017; in PTD, especially with indirect ones in Kuperberg et al., 2007), whereas in short SOAs of 140 ms and early time windows (~400 ms post-stimulus), another study using fMRI and MEG found reductions in activity (Kuperberg et al., 2019).

Also in line with semantic crowding, it is shown that background firing (noise) initially enhances detection of weak stimuli and sensitivity to the unexpected (e.g., as in distant associations in PTD, or even N400 waves evoked by distant and unrelated word pairs in schizophrenia, see section 5.2) by increasing their probabilities of evoking action potentials (floor effect) and reducing spike latency (Shu et al., 2003; Ollerenshaw et al., 2014); however, it subsequently disrupts feature discrimination and categorisation - required for a lexical decision, for example, or Go/No-Go tasks, cue detection, and so forth - by jumbling the spatial sharpening of representations through a failure to constrain excitatory spreads (Ollerenshaw, 2013; Ollerenshaw et al., 2014; Shu et al., 2003). Thus, a potential corollary of the semantic crowding hypothesis is that hyperpriming in short SOAs (harsher disinhibition, more noise) will predict greater semantic crowding later on. Accordingly, the aforementioned meta-analysis by Pomarol-Clotet et al. (2008) *de facto* reported more hypopriming in FTD than in general schizophrenia.

Ketamine is also notorious for producing FTD (e.g., Kircher et al., 2018). This drug consistently impairs discriminability of engrams and exogenous stimuli, and controlled hypopriming accompanies acute intake (Morgan et al., 2006b; Stefanovic et al., 2009). Acute ketamine (and/or phencyclidine) also reportedly greatly dampens the fano factor (mean of variance to baseline activity) of pyramidal firing *relative to their mean response across multiple stimuli/conditions* (i.e., irrespective of context, every response is like the average response), distorts spatial sharpening/selectivity of representations, disrupts feedback, inter alia (van Loon et al., 2016; Ouelhazi et al., 2019; and for phencyclidine, Zick et al., 2018). In regards to the fano factor, for example, this could be observed in PTD behaviourally and electrophysiologically, when behavioural responses as well as ERPs are weakly modulated by semantic distance (Pomarol-Clotet et al., 2008; Wang et al., 2011; see Section 5.1). Finally, albeit semantic crowding should invabilise response selection, the concept is based on attentional and conscious engagement, insofar as it should be calibrated by conscious effort. Hence, effort can be a confounder for an interpretation of hypopriming hinged, for instance, on response selection demands (see Fig. 1).

All in all, before we reviewing the PTD endophenotype and electrophysiology, a contradiction remains: automatic direct/close hyperpriming under those and other settings is still less consistent in PTD and schizophrenia as a whole (e.g., Kuperberg et al., 1997, 2018, 2019; Wang et al., 2011; Pomarol-Clotet et al., 2008; Kreher et al., 2008, 2009; Tan et al., 2015; Rodríguez-Ferreiro et al., 2020). Because the classical connectionist models predict that the facilitatory access to indirect nodes is preceded by that of the direct ones (e.g., Kuperberg et al., 2019; Collins and Loftus, 1975), such a contrast is very puzzling to us and to psycholinguistics in general. We will present two, potentially complementary hypotheses in an attempt to solve this theoretical conundrum, though the second one will only be covered in section 6.1. As for the

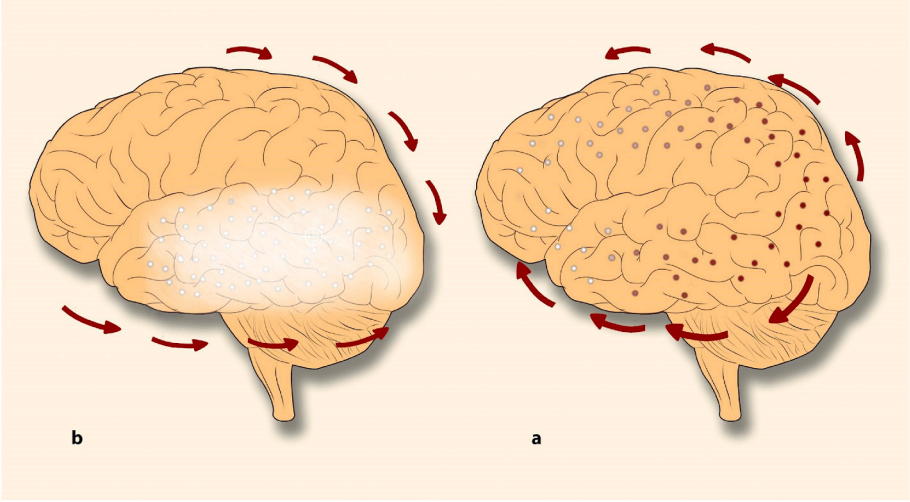

Fig. 1. Semantic crowding. (a) Disinhibition of the spread of activation from PV and/or MC inhibition weakens the signal's fidelity and strength as it covers the feedforward stream. The disintegration of the signal in this horizontal axis creates a frontotemporal disconnection, corrupting harmonious interareal processes. The signal also gradually dies out at the higher-order ends, representing potential hypofrontality. (b) In response to the poor feedforward signal-to-noise ratios, attentional feedback is misled into erroneous targets and contributes to semantic crowding: the already-inaccurate feedback projections arrive and, due to lack of inhibitory constraints, this surplus drive diffuses pervasively upon landing, saturating populational recruitment and promoting salience erasure. In PTD, the target node (drawn in white within the blurred area), whose access would be otherwise achieved normally in implicit tasks, becomes inconspicuous and indiscernible from surrounding activity in explicit conditions. Even though the role of attention is recognised as a suppressive effect on behaviourally-irrelevant nodes and selective amplification of the relevant ensembles, in PTD, attention is a "blind" and disruptive force that actively harms cognition.

first, we suggest that a neurophysiological motif could be at play here - namely, a *semantic surround suppression* in semantic space. Surround suppression is a phenomenon whereby the excitation of an active receptive field's surroundings results in inhibitory interference over the centre typically in sensory cortices. This an effect that has been manifold associated with spatial summation of inputs onto Martinotti cells (MCs), which characteristically respond by spreading "blankets of suppression" over superficial layers (e.g., Adesnik et al., 2012; Karnani et al., 2016). In higher cortices, MCs also drive net suppression in response to spatial summation - only, it is not concentric. This motif may be particularly pronounced in higher-order cortices, which have the densest expression of MCs as well as massive ensemble overlaps (allowing for flexible semantic representations, Wilson and Wilkinson, 2015; Almeida, 2021a, b). Hence, semantic surround suppression is only concentric in an abstract semantic network. In a concrete sense, it arises with spatial summation of inputs. Thus, neuronal ensembles that share greater overlap with the prime words suffer some degree of semantic semantic surround suppression when additional action potentials are fired from their vicinity (hebbian covenants, such as indirect word pairs), as there will be a surplus spatial summation of excitatory inputs onto MCs (Adesnik et al., 2012). In addtion, it should be underscored that ensemble overlap further entails partial "repetition suppression" through temporal summation, i.e., repetitive stimulation of the overlapping parts of the two representations (repetitive stimulation precipitates even stronger MC inhibition due to short-term facilitatory inputs, Natan et al., 2017; Silberberg and Markram, 2007; Berger et al., 2010).

A semantic surround suppression would be consistent with reports that direct hyperpriming is more characteristic of thought-disordered patients (e.g., Kreher et al., 2008, 2009; Spitzer et al., 1994; Weisbrod et al., 1998; Safadi et al., 2013; Pomarol-Clotet et al., 2008), which even display diminished visual surround suppression (Uhlhaas et al., 2004); schizophrenia as a whole also exhibits harshly impoverished MC expression/function (Alherz et al., 2017). Similarly, fMRI population receptive field mapping points to decreased inhibitory surrounds of visual receptive fields (i.e., responsible for surround suppression) in schizophrenia (Anderson et al., 2017). Hence, since there is no sufficient top-down engagement for semantic crowding, automatic direct hyperpriming and PTD severity could inversely correlate with MC function - the more the network is freed from semantic surround suppression, the more disinhibited the overlapping nodes or ensembles (i.e., primed associations), thereby favouring direct automatic hyperpriming as well as subsequent controlled hypopriming. Perhaps in consonance with this differential MC hypofunction in PTD - as well as semantic crowding -, even though surround suppression and overall MC inhibition are enhanced by top-down engagement of predicted and peripheral stimuli in vivo (Sundberg et al., 2009; Liu et al., 2020; Nassi et al., 2014), response enhancements are observed in PTD upon exposure to indirect word pairs (Kuperberg et al., 2007; Wilson et al., 2013); this enhancement even differentiated PTD from other schizophrenic and healthy cohorts in one of the studies (Kuperberg et al., 2007). Of note, semantic surround suppression is not elicited by *unprimed* stimuli (MCs are inhibited by unpredictable stimuli, see Fig. 3), which explains why indirect nodes do not suppress cortical activity in healthy cohorts.

Interestingly but equivocally, it is possible this MC-driven inhibition could be observed preferably in the RH. According to Beeman (1993) and Beeman et al. (1994), in the RH, broader *semantic* fields are fainter, characterised by direct associations being but a bit more salient than others, as opposed to the LH, where the contrast is clear. Burgess and Simpson (1988) reported that, in SOAs 35 and 750 ms with ambiguous words, the LH (right visual field) exhibits facilitation for the frequent meanings in both sides, but decreases left-sided facilitation for infrequent meanings in long SOAs (ambiguity resolution); conversely, the RH (left visual field) increases facilitation for the subordinates while decreasing it for the dominants. Chiarello et al. (2001) also reported that repetition of category-related words (e.g., animals) slightly impairs RH performance, whereas the LH benefits from it (this is interesting because, again, MCs distinctively and nonlinearly amplify their inhibition in the face of temporal summation of excitatory drive, i.e., repetitions, Natan et al., 2017; Silberberg and Markram, 2007; Berger et al., 2010). Thus, one possibility is that a reduced/reversed functional and structural linguistic asymmetry in PTD could contribute to the lack of direct hyperpriming in this way (see section 5.1).

The reasons for this asymmetry are unclear. Provided it is legitimate, the most likely reason for it would be that MCs should be fed with stronger spatial summation of drive in the RH, due to its cytoarchitectural profile - microcolumns are "huddled" more closely together (Chance, 2014). Some even believe this to be the substrate for lateralised cognition (for reviews, see Hutsler and Galuske, 2003; Chance, 2014): widely spaced columns promoting fine-grained high-resolution processes (e.g., left fusiform gyrus for language), and closer arrangements promoting lower resolution, holistic processes (e.g., right fusiform for face processing). Furthermore, one of the main suppressors of MCs - PVs

- is much more poorly expressed in the RH than in the LH (Rosen, 1996; Rosen et al., 1993; Sherwood et al., 2007; Katahira et al., 2018; Butler et al., 2018). Hence, densely packed columns bolster spatial summation of inputs, and lower PV densities further disinhibit MCs - the result being semantic surround suppression of "central" nodes. This would, again, be consistent with the tendency of the RH to operate on lower-frequency oscillations, which are known to be more typically regulated by MCs, stemming from large areas of activation and surround suppression, whereas higher frequencies like gamma are notoriously tied to PV activity and stringent cortical spreads due to fast feedforward (lateral) inhibition (Huang et al., 2020; Hamm and Yuste, 2016; Fanselow et al., 2008; Chung et al., 2020; Javitt et al., 2018; Kienitz et al., 2018; Romei et al., 2011; Sun and Dan, 2009). All in all, the semantic surround suppression hypothesis is very inferential and subject to numerous confounders. Still, it is a possibility that we had to pursue, given the importance of the direct-indirect hyperpriming dichotomy in FTD.

In conclusion, we hope that the above mechanistic interpretations of semantic priming should bring attention to how seldom the spread of activation has been treated as a potential window into PTD's ontology. Moritz et al. (1999) found enhanced spreads in healthy controls who displayed mild FTD symptoms, clearly suggesting a primary role in the subsyndrome. Another example is found in Kreher et al. (2008), who reported an increase in indirect priming for PTD cohorts in comparison to other schizophrenic patients and controls, and increases in direct priming only for severe PTD. There are surprisingly few studies or factor analyses investigating these phenomena as potential roots for broader pathophysiological processes as well. For instance, the pattern change of hyper-to hypopriming could be an indication of hypofrontality in PTD, as simple decision-making severely disrupts performance. In that vein, many authors believe that hyporpiming reflects an inability to integrate contextual information and/or executive function impairments (see also section 6) (for reviews, Kuperberg, 2010a; 2010b). Also interestingly, Sommer et al. (2010) documented that experiencers of auditory-verbal hallucinations (a core symptom of schizophrenia), who were either schizophrenic or otherwise healthy, both displayed PTD symptomatology. As the authors pointed out, this is suggestive of a shared pathophysiology (for auditory deficits in FTD, see also Moschopoulos et al., 2020). Hence, parsing the PTD endophenotype could bring significant prospects for future scientific and medical research.

5.1. Endophenotype

Aside from evoked cortical motifs, what are the concrete neurobiological traits underlying schizophrenic language, and how could they give rise to the aforementioned alterations?

First of all, the LH has long been thought to be lateralised for most language functions. The most notorious lateralised epicentres of linguistic processing are the IFG and the planum temporale, though ultimately it engages widespread networks (Poeppel and Hickok, 2004; Hickok and Poeppel, 2016). Schizophrenia and PTD show robust reductions of the typical functional and structural interhemispheric linguistic asymmetry (or fully-fledged reversions), with an emphatic thinning of left frontal and temporal structures, along with multiple white matter structures and other, mostly less pronounced alterations (e.g., Oertel-Knochel et al., 2012; Kircher et al., 2018; Sumner et al., 2007; Cavelti et al., 2018; Zeev-Wolf et al., 2014). In various paradigms, abnormal perfusion and blood-oxygen-level-dependent patterns, including both hyper- (especially posterior temporal) and hypo-activations (to a lesser extent), are observed in a number of other LH executive and semantic areas like the STG, IFG, ACC, and MTG (Cavelti et al., 2018; Sumner et al., 2018; Assaf et al., 2006; Kircher et al., 2018; Wensing et al., 2017; Weinstein et al., 2007; Wilson et al., 2013). Unsurprisingly, many semantic and executive processes appear to be pronouncedly impaired in FTD (e.g, Nagels et al., 2016; Kerns and Berenbaum, 2002).

As mentioned in earlier sections, priming effects are also lateralised. The LH plays a more selective role in direct priming and stronger associations, while the RH seems to coarsely engage both weaker and stronger associations. The neurobiological reasons for this are unknown. Some suggest it may indicate an asymmetric dopaminergic tone (see Lavigne and Darmon, 2008). There is some evidence, albeit old, that the LH shows substantially greater baseline dopamine levels, including in frontal and subcortical areas (e.g., Slopsema et al., 1982; Glick et al., 1982; Tucker and Williamson, 1984). Similarly, lateralised dopaminergic abnormalities could work in the context of schizophrenia and priming effects.

Accordingly, Weisbrod et al. (1998) found that while both healthy controls and FTD patients show indirect priming to words presented to their left visual field (RH), only FTD patients show indirect priming to their right visual field (LH), with no difference inter-groups on direct priming effects (produced bilaterally). Brugger and Graves (1997) also documented positive correlations between right-hemifield (LH) inattention and the severity of positive symptoms (e.g., magical ideation) in healthy young men (but not women). The authors interpreted those results as reflecting mesocortical left-sided depletion (see also Early et al., 1989). Alternatively or complementary, others have suggested a right-sided mesocortical hyperdopaminergia (Bracha, 1987). Further support for lateralised dopaminergic abnormalities could be derived from studies that observed right hemispatial inattention, as well as left turning biases, in acute psychotic patients and schizotypes (Harvey et al., 1993; Brugger and Graves, 1997; Bracha et al., 1993); turning biases tend to favour the hypodopaminergic side, though this is typically associated with the striatum. These lines of inquiry seem to have waned for some time, however; we are unaware of the reason for the discontinuity. Nonetheless, overall reduced mesocortical function and reduced dopaminergic transmission within the frontal lobes have been linked to schizophrenia. Therefore, they should affect the LH performance in lexical decision tasks anyway. We suggest that even bilateral hypodopaminergia would seem lateralised - at least in respect to behavioural measures - if dopamine/DRD1 modulates the LH preferentially, as in Roesch-Ely et al.'s (2006) study.

Indirect evidence for differential cortical hypodopaminergia in PTD is found in studies based on the Val/Met genotypes, coding for COMT enzyme activity. The COMT enzyme reduces catecholaminergic activity, most prominently in the prefrontal cortex. Winterer et al. (2006a, 2006b) found that Val carriers (higher COMT) - including schizophrenics in Winterer et al. (2006a) - had higher levels of prefrontal noise. In another study, across 98 schizophrenics and 114 controls, lower COMT activity was associated with greater medial temporal lobe volumes (Ehrlich et al., 2010). One study also found dramatically reduced expressions of DARPP-32 in the STG of schizophrenic patients (Kunii et al., 2011); the DARPP-32 protein is encoded by the PPP1R1 B gene, and is found within regions richly innervated with dopamine (Yger and Girault, 2011), regulating both DRD1 and NMDAR functions (Kunii et al., 2011). There is evidence that amphetamines significantly improve certain executive and language-related deficits in schizophrenic-spectrum cohorts as well (e.g., Kirrane et al., 2000; Barch and Carter, 2005). Further, Barch et al. (1996) found that d-amphetamine ministered as an adjuvant with standard neuroleptics had beneficial repercussions for PTD patients, with significant amelioration of PTD symptoms and unclear referencing.

Thus, there is non-negligible evidence to back a (cortical) hypodopaminergic hypothesis of PTD, but additionally, alterations in the glutamatergic system have been speculated to underlie the syndrome. These mainly involve NMDAR hypofunction, and can be supported by psychophysiological alterations (e.g., the mismatch negativity evoked-related potential) as well as reports that acute ketamine intake (a NMDAR antagonist) can induce both PTD and NTD semiology (Kircher et al., 2018). As credible as it sounds, the glutamatergic model of PTD still demands further elucidation, as NMDAR hypofunction has been widely associated with schizophrenia itself, which comprises a whole spectrum of manifestations. Kircher et al. (2018) suggested, specifically,

that PTD would be associated with glutamatergic synaptic impoverishment in the STG and lateral middle temporal gyrus. One way to conciliate the dopaminergic and glutamatergic hypotheses of PTD, we suggest, lies in the fact that DRD1s intimately regulate NMDAR function (e.g., Chen et al., 2004). Thus, hypodopaminergia may contribute to NMDAR hypofunction, insofar as the two may co-exist in PTD. What would be interesting to explore is whether the two are differentially severe in the syndrome.

Furthermore, there are some other ways to support the NMDAR model, generically. Namely, PVs are thought to be acutely dysfunctional in schizophrenia as a whole, and profoundly impaired by NMDAR hypofunction in this pathology, as well as ketamine use (Gorelova et al., 2002; Seamans et al., 2001). We suggest that dopaminergic constraints on the spread of activation are chiefly reliant on PV basket cells, and PTD would be related to differential PV hypofunction in schizophrenic cohorts. For instance, DRD1s′ inhibitory processes - attenuation of noise - are for the most part, or solely, mediated by PV basket cells (Gorelova et al., 2002; Seamans et al., 2001). Like DRD1s ultimately do, but virtually unequivocally, these cells have been shown to reduce firing in non-preferred directions and increase feature selectivity in the cortex (Duan et al., 2017; Glausier et al., 2009; Wood et al., 2017; Yang et al., 2017; Kvitsiani et al., 2013; Sohal et al., 2009; Raghanti et al., 2010). Another interesting point is that acute and chronic ketamine intake release dopamine (e.g., Duan et al., 2013; Homayoun and Moghaddam, 2007; Kokkinou et al., 2018), and yet, the drug produces FTD semiology - indirectly so, this could match the idea that PVs themselves are the central elements in the syndrome, whereas dopamine is a secondary factor. Moreover, if DRD1 influence is truly more restricted to controlled processing windows, this PV hypofunction would explain a hyp odopaminergia-like *automatic* disinhibition of indirect nodes - these cells respond almost instantaneously to the arrival of bottom-up inputs to the cortex (see section 6.1). Additionally, such a hypothesis can also dovetail NMDAR hypofunction and hypodopaminergia. In fact, the latter scenario is where PVs should be most compromised.

Also in line with these considerations, the reported preferential DRD1 modulation of the LH (Roesch-Ely et al., 2006) could owe to the fact that PV density is consistently and substantially greater in the LH (which is, again, in line with gamma-band predominance), even though overall neuronal density is either roughly the same bilaterally or possibly greater in the RH due to the smaller surface (Rosen, 1996; Rosen et al., 1993; Sherwood et al., 2007; Katahira et al., 2018; Butler et al., 2018; Smiley et al., 2011). This would concord with the aforementioned study where only FTD showed indirect priming in the LH (Weisbrod et al., 1998). Moreover, prepulse inhibition (PPI) deficits are quite common in schizophrenic patients. PPI indexes the inhibition of a startle reflex by an immediate weak prepulse; it is a measure of sensorimotor gating, reflecting the ability to safeguard representations from interference, reportedly relying heavily on PVs (Nguyen et al., 2014; Popelář et al., 2013). Therefore, it is critical to highlight that PPI deficits show a trend of disproportionally correlating with the FTD dimension (Perry and Braff, 1994; Perry et al., 1999; Braff et al., 1999; Meincke et al., 2004; Matsuo et al., 2016). Further, shifts to lower gamma band frequencies correlated with the disorganisation dimension (to which FTD pertains) in schizophrenia (Spencer et al., 2003), and hypofrontality for language was also reflected in gamma oscillations in a study (Spironelli and Angrilli, 2015) - gamma frequencies are robustly linked to PV firing and so are their alterations related to PV hypofunction in schizophrenia (e.g., Gonzalez-Burgos et al., 2015). Altogether, in light of their prominent role in schizophrenia, a differential PV deficit in PTD could be a highly significant finding that would further appose PTD to the ontology of schizophrenia. Moreover, PV hypofunction could clearly act as a confounder for studies on dopaminergic modulation of semantic priming.

5.2. Electrophysiology

All in all, we have weaved a number of considerations on the FTD endophenotype. However, one last topic that has not been addressed is that of evoked-related potentials (ERPs). ERPs constitute a fine-grained tool for analysis of the neurophysiology behind semantic priming effects. Of particular relevance herein, priming is widely associated with the N400 waveform (which is usually speculated to index either contextual integration or lexical access) - a broad and monophasic deflection, with an onset latency of 200–250 ms and a canonical peak at 400 ms post-stimulus onset. Among other, less robust correlations, the N400 has been linked consistently to the posterior temporal lobe in functional MRI studies (Lau et al., 2008). More importantly, the N400 can be evoked by means of the lexical decision paradigm: if the target is primed, the N400 is attenuated, and vice-versa (for a review, Kutas and Federmeier, 2011). Hence, direct word pairs tend to evoke smaller deflections than indirect ones. Conversely, and strikingly, multiple studies suggest this relation is to various degrees reversed (at times a fully-fledged reversal) in schizophrenia, whereby the N400 at times suffers a relative attenuation for distant and/or unrelated associations (Kreher et al., 2008, 2009; Mathalon et al., 2002; Kuperberg et al., 2019; Salisbury, 2008; Sharma et al., 2017; Jackson et al., 2014; Ryu et al., 2012; Koyama et al., 1991; shorter latency, Pfeifer et al., 2012; Shin et al., 2008; in schizotypy, Kiang and Kutas, 2005; for amplification of distant associations, Salisbury, 2008, 2010; Salisbury et al., 2000; Niznikiewicz et al., 1997; Siddiqui et al., 2021; Kreher et al., 2009), and much more consistently, a distinctive augmentation for closer ones (most pronouncedly in long SOAs) that can supersede unrelated targets (Kuperberg et al., 2018; Kiang et al., 2008, 2011, 2014; Kostova et al., 2005; Ditman and Kuperberg, 2007; Kreher et al., 2009; Besche-Richard et al., 2014; Wang et al., 2011; Wang et al., 2020; Sharpe et al., 2020; Condray et al., 2010; Salisbury, 2004, 2010; Salisbury et al., 2002; Metzler et al., 2014; Sharma et al., 2017; Olichney et al., 1997; Hokama et al., 2003; Niznikiewicz et al., 1997; Nestor et al., 1997; Bobes et al., 1996; Mathalon et al., 2010; Ohta et al., 1999; Koyama et al., 1991; Boyd et al., 2014; Jacob et al., 2017; Battal Merlet et al., 2018; Condray et al., 1999; in negative mood, Pinheiro et al., 2014; in schizotypy, Kiang and Kutas, 2005; in clinical high risk of psychosis, Lepock et al., 2019, 2020, 2021a, 2021b; literal idioms, Strandburg et al., 1997). In fact, an amplification of close associations was lent clear support from a 2011 meta-analysis (Wang et al., 2011). Due to space constraints, we will not peruse this issue any further, but it should be duly noted that by "close associations" we loosely refer to any closer semantic relations relative to their more distant counterparts in any given study (e.g., identity-related relative to unrelated, directly related relative to unrelated, congruent relative to incongruent, etc).

As for the literature on cognitive disorganisation, such differential correlations with any sort of N400 alterations are qualitatively inconsistent among themselves (smaller N400 effect, Kuperberg et al., 2018; Kostova et al., 2005; Ditman and Kuperberg, 2007; Kreher et al., 2008, 2009; larger N400 effect to congruous scenes, Sitnikova et al., 2009; larger implicit N400 effect, Kreher et al., 2008, 2009; altered mean amplitudes, Andrews et al., 1993; Qiao et al., 2020; larger amplitudes to subordinate-affirmative sentence endings, Salisbury et al., 2000; reduced N400 network activity, Jacob et al., 2019), and have proven highly unreliable (e.g., Mathalon et al., 2010; Jackson et al., 2014; Metzler et al., 2014; Shin et al., 2008; Pfeifer et al., 2012; Kiang and Kutas, 2005; Kiang et al., 2007, 2008, 2010; Ryu et al., 2012;

Besche-Richard et al., 2014; Salisbury et al., 2000, 2002; Boyd et al., 2014; Kostova et al., 2014; Del Goleto et al., 2016; Jacob et al., 2019; in the fMRI, Vistoli et al., 2011). Thus, there is no prima facie correlation between a particular N400 profile in explicit tasks and FTD (Andrews et al., 1993; Kostova et al., 2005; Ditman and Kuperberg, 2007; Kreher et al., 2008, 2009bib_Kreher_et_al_2008bib_Kreher_et_al_2009; Sitnikova et al., 2009; Besche-Richard et al., 2014; Pfeifer et al., 2012; Kiang et al., 2007, 2008, 2010bib_Kiang_et_al_2007bib_Kiang_et_al_2008bib_Kiang_et_al_2010; Kiang and Kutas, 2005; Ryu et al., 2012; Kuperberg et al., 2018; Salisbury et al., 2000, 2002bib_Salisbury_et_al_2000bib_Salisbury_et_al_2002; Mathalon et al., 2010; Boyd et al., 2014), which could owe to the wide variety of different scales (e.g., the widely-used PANSS is only comprised of two items directly relating to PTD) used in these studies. In addition, correlations between larger N400 effects and PTD in implicit proper conditions have not been disproven - to our knowledge, only two studies employed implicit paradigms with PTD patients, and both reported larger N400 effects (Kreher et al., 2008, 2009bib_Kreher_et_al_2008bib_Kreher_et_al_2009). Altogether, whatever the underlying cause of the equivocal offspring of this research, a relative reversal of the N400 - especially a reduced N400 effect - remains a very solid correlation, whether in schizophrenia or schizotypy (Kiang et al., 2007, 2008, 2010, 2011, 2014; Kostova et al., 2005; Ditman and Kuperberg, 2007; Kreher et al., 2009; Sitnikova et al., 2009; Kiang and Kutas, 2005; Besche-Richard et al., 2014; Wang et al., 2011; Koyama et al., 1991; Metzler et al., 2014; Kuperberg et al., 2018, 2019; Condray et al., 2010; Wang et al., 2020; Sharma et al., 2017; Sharpe et al., 2020; Salisbury, 2004, 2008; Salisbury et al., 2002; Ryu et al., 2012; Iakimova et al., 2013; Nestor et al., 1997; Niznikiewicz et al., 1997; Jackson et al., 2014; Jacob et al., 2017; Mathalon et al., 2002, 2010; Condray et al., 1999; Bobes et al., 1996; Battal Merlet et al., 2018; Boyd et al., 2014; Pinheiro et al., 2014; in clinical high risk of psychosis, Lepock et al., 2019, 2020, 2021a, 2021b; in literal idioms, Strandburg et al., 1997). Finally in that vein, we should highlight that there are occasional reports of *increased* N400 effects for close associations in schizophrenia (Kreher et al., 2008, 2009; Grillon et al., 1991; Guerra et al., 2009; Wang et al., 2020; relative to bipolar patients, Raucher-Chéné et al., 2019; high-relative to low-schizotypy patients, Kostova et al., 2014), even with long SOAs (Grillon et al., 1991; Wang et al., 2020; picture sequences, Guerra et al., 2009; high-relative to low-schizotypy patients, Kostova et al., 2014). Concerning this issue, perhaps one point to draw attention to for future studies is epitomised in Wang et al. (2020): both reduced and enhanced N400 effects co-occurred in schizophrenics, varying according to electrode (e.g., it would be curious to inspect whether there are N400 correlations with FTD in some particular electrode).

Unfortunately, it is hard to tell what N400 alterations really index in terms of neurobiology. The only neurophysiological model on this deflection was recently put forth by Almeida (2021a). According to the author, the elicitation of the N400 may owe to an interaction between three very prominent types of interneurons: MCs, vasoactive intestinal polypeptide-positive cells (VIPs), and to a lesser degree, PVs. Cholinergic transients would contribute to these dynamics directly. Grounded on extensive evidence gleaned from phenomena congruent with or within the same time window as the N400 (200–500 post-stimulus onset), the author comes to a series of conclusions. Relevantly herein, and roughly put, PVs would be suppressing somata, and VIPs would be suppressing MCs to disinhibit apical dendrites within the N400 time frame (see Fig. 2). Apical dendrites are contacted by higher-order feedback projections in layer I to elicit sustained plateau potentials (interestingly, plateau potentials even tend to last ~ 300 ms, as does the N400, from 200 to 500 ms, Almeida, 2021a). Essentially, this chain of events builds vertical dipoles out of layer V pyramidal cells, whereby superficial extracellular negativity (positive inward currents) and deep layer positivity (along with capacitive efflux) unleash upward currents of positive ions towards the negative surface (see Fig. 3). These upward ionic currents are recorded by the electrode as upward deflections - i.e., negative. This interpretation may have some merit because each of the three interneuron types in the model has been found to express abnormalities in schizophrenia (Alherz et al., 2017; Koukouli et al., 2017; Lewis et al., 2012). The two other (non-neurophysiological) neurobiological models on the N400 also concord with the wave arising from

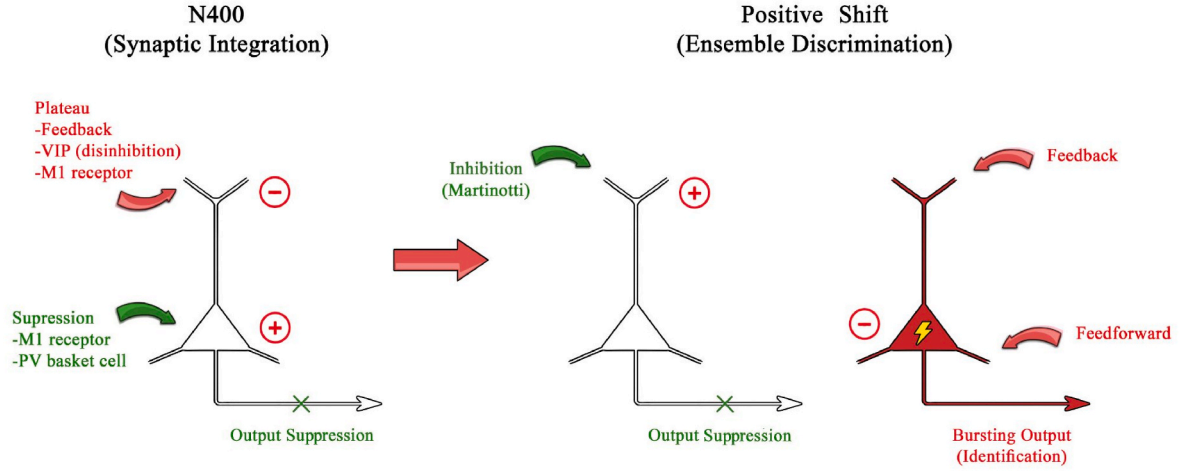

Fig. 2. The N4 model. (N400, synaptic integration, top left): phasic cholinergic input to M1 receptors, nicotinic (not represented) and glutamatergic recruitment of VIPs, and glutamatergic feedback to apical dendrites, trigger dendritic plateaus (superficial negativity) across the network. Plateaus promote synaptic integration as the network "fumbles in the dark" to discriminate the deviant ensemble. (N400, synaptic integration, bottom left): Somatic suppression by cholinergic inputs to M1 receptors and PV basket cells (deep positivity) shuts down outputs to the thalamus. Hence, only the ensembles that receive robust feedforward drive can outstand the silent background by magnifying their plateaus with eventual backpropagation. (Positive shift, ensemble discrimination): once backpropagation and plateaus coincide within a narrow time window, coincidence detection translates into burst firing. (Positive shift, ensemble discrimination, top left): Bursts set in motion a disynaptic motif of MC inhibition in the surroundings (enhanced via rebound spiking), shutting down the interval of synaptic integration by suppressing surrounding plateaus (superficial positivity); complementarily, this inhibition might involve late-spiking layer I interneurons (Carracedo et al., 2013). (Positive shift, ensemble discrimination, right): this way, only the first ensembles to burst (deep negativity) contact the thalamus - the winner takes all. This spatially-coherent information concludes the discrimination period, permitting recurrent communication with the higher-order thalamus to synchronise ensembles for conscious selection/identification of the unexpected higher-order features. The debate on whether to interpret the N400 as an index of lexical access, contextual integration, or both, is quite fierce. This model, on the other hand, is largely concerned with the neurophysiological substrates of the N400 deflection. It is the first careful and detailed account of the mechanistic and neurophysiological underpinnings of the ERP. Reproduced from Almeida (2021a).

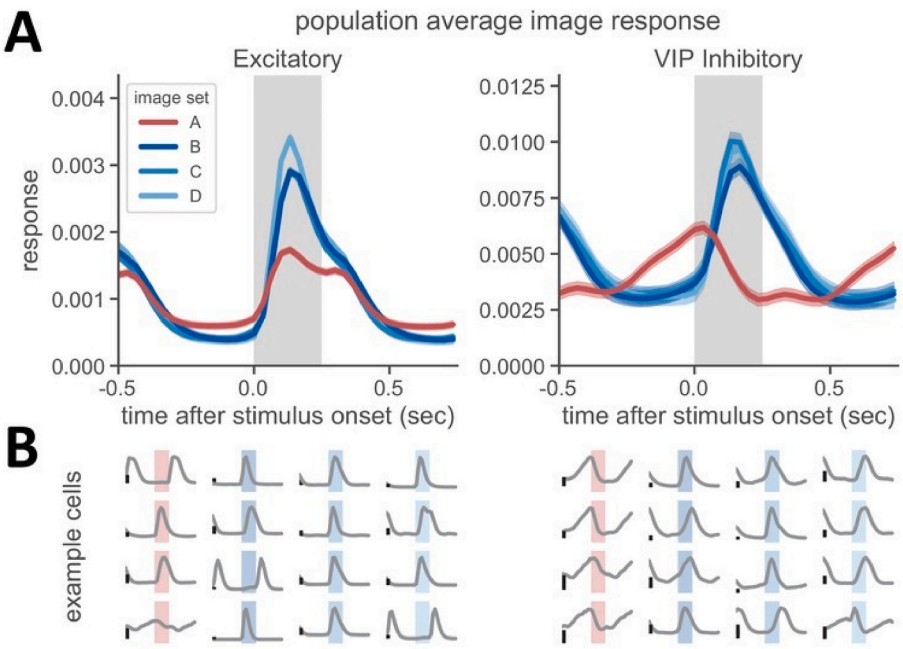

Fig. 3. VIP and pyramidal cell responses to familiarity and unfamiliarity. (A) The two panels display the average response of excitatory and VIP cells to images in either a highly-learned sequence (set A, red) or an unexpected set (sets B to D, blue). Stimulus presentation is represented by the grey band. Notably, VIP response seems to peak just short of 250 msec in response to unfamiliar stimuli (for unexpectd omissions, Garrett et al., 2020; Orlova et al., 2020), whereas it does not simply attenuate its response to predictable relative to unpreditable stimuli - it is effectively *suppressed* (Garrett et al., 2020; Khan et al., 2018; Orlova et al., 2020). This is in accordance with the idea, proposed by Almeida (2021a), that VIPs would be key to eliciting the deflection - predictable stimuli attenuate or abolish the N400 (as in set A), whereas unpredictable ones elicit the deflection (as in sets B-D), relying on VIP-induced disinhibition. In fact, VIPs seem to be the only cell taxon to robustly ramp up their firing preceding predictable stimuli in multiple experiments in striking resemblance to multiple negative ERPs (Khan et al., 2018; Ouelhazi et al., 2019; Garrett et al., 2020; see also Kamigaki and Dan, 2017), That is, there are a number of negative ERPs that behave virtually in the same way, from classical ones such as the Contingent Negative Variation (see Kamigaki and Dan, 2017), to novel ones such as Semantic Prediction Potentials and the Contingent Response (Grisoni et al., 2017; Fishman et al., 2021). This modus operandi thereby further implicates VIPs in driving negativity and, by extension, MC suppression in positivity - such as N400 attenuation. (B) Examples of specific cells' responses to stimuli. Red bands portray familiar sets and blue bands, unfamiliar ones. Adapted from Garrett et al. (2020) with permission. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

superficial layers (also layer I depolarisation and V suppression in Kotchoubey, 2006; supragranular, Bornkessel-Schlesewsky and Schlesewsky, 2019). Hence, any disruption of these mechanisms could have repercussions for the N400. In agreement, in response to acute ketamine intake, the N400 reportedly undergoes some similar alterations as those observed in schizophrenia (Grunwald et al., 1999). Inasmuch as ketamine severely disrupts MC function (Ali et al., 2020), this finding may hint at once more at a direct link between semantic disturbances and MC and/or PV cell hypofunction - and ultimately, NMDAR hypofunction (Kircher et al., 2018).

Indeed, MC hypofunction could conceivably, at least in part, underlie the reversal of the N400 effect in schizophrenia. In line with our discussions so far, Almeida (2021a) postulated that "if we surmise that predictable words are somewhat peripheral to the main ensembles (already-presented words)", "their excitation of a larger area allows MCs to summate horizontal drive", causing net suppression, and again, we would have some contribution of representation overlap. Conversely, aside from potential synaptic depression, repetition proper N400 effects would arise more strictly from MCs' characteristic temporal summation (Almeida, 2021a), which would be in line with reports that N400 repetition effects are hampered by NMDAR blockade (Grunwald et al., 1999), seeing that the drug distinctively disinhibits apical dendrites (i.e., the N400) (Ali et al., 2020) and undermines both representation amplification and suppression (Grunwald et al., 1999; Ouelhazi et al., 2019).

We recently found partial support for these hypotheses in Liu et al. (2020), whose experiment showed that feedback projections can suppress predictable stimuli in a Go/No-Go task by increasing Somatostatin-positive (SST) cell drive, specifically (MCs being, by far, the most prevalent SST cells). Feedback is also found to enhance surround suppression precisely by enlarging the size of the suppressive area (Nassi et al., 2014). Furthermore, Orlova et al. (2020), Garrett et al. (2020) and Khan et al. (2018) all reported that predictable stimuli increase SST drive (Orlova et al., 2020; Khan et al., 2018) and suppress VIPs - thereby, the N400 - (see Fig. 3; Orlova et al., 2020; Garrett et al., 2020; Khan et al., 2018). A host of other studies also demonstrates sui generis VIP responses to unpredictable stimuli (Almeida, 2021a).

In summary, the crux of the argument is that, if suppression of predictability is performed by evoked MC firing (which targets superficial layers) and the N400 is elicited in superficial layers (as per neurobiological models, Almeida, 2021a; Kotchoubey, 2006; Bornkessel-Schlesewsky and Schlesewsky, 2019), it would seem plausible that a larger N400 would denounce a hypofunctional MC-driven suppression of predictable stimuli. Thus, larger amplitudes in response to related words in schizophrenia could be associated with an impaired ability to recruit phasic inhibition. In this scenario, a mutual excitation between prime and target or simply a larger summed excitation in a given area due to ensemble overlap could render amplitudes larger than those of unrelated targets, as there is an absence of the typical MC inhibition that distinctively counters such summation of excitation (Adesnik et al., 2012; and see Löw et al., 2006). In fact, this hypothesis is even in tune with semantic crowding, seeing that the reversal of the N400 effect is most pronounced in long SOAs (Wang et al., 2011; for disinhibition, see also Vistoli et al., 2011), and also translates into semantic indiscrimination/insensitivity to context. Even further, findings from Kreher et al. (Kreher et al., 2008, 2009) and Kuperberg et al. (Kuperberg et al., 2019) are also in alignment with these ideas, i.e., the N400 is abnormally large in explicit (semantic crowding) but abnormally small in implicit tasks .

Hence, N400 reversals could be tied to MC hypofunction, but on top of this, there are reports of impaired VIP excitability in schizophrenia (Koukouli et al., 2017). Such impairments could curtail amplitudes of unrelated targets, for example. In fact, a relatively sturdy body of literature seems to implicate VIPs and MCs in the MMN as well, especially vis-à-vis the abolishments of the wave/neuronal response with certain experimental measures - e.g., pharmacogenetic silencing of MCs preceding deviants, optogenetic silencing of VIPs concurrently with mismatches, etc. (e.g., Almeida, 2021a; Chen et al., 2015; Hamm and Yuste, 2016; Javitt et al., 2018; Lakatos et al., 2020; Halgren et al.,

2018). In regards to biology in particular, there even exists a debate on whether to consider the MMN a lower-order N400 variant (Kotchoubey, 2006; Bornkessel-Schlesewsky and Schlesewsky, 2019; Almeida, 2021a), and a recent study reported a correlation between reduced MMN amplitudes and reduced N400 effects at short but not long SOAs in subjects at high clinical risk of psychosis (Lepock et al., 2020). Shockingly, MMN alterations in schizophrenia have been replicated hundreds of times by 2016 (Michie et al., 2016; Erickson et al., 2015), whilst also being linked to FTD in certain conditions (see Kircher et al., 2018). Finally, VIPs also seem to be deployed for top-down predictions - and/or possibly preparatory attention - manifested in various "predictive negativities" (negativities that precede presentation of predictable targets with regular interstimulus intervals), such as the recently-discovered Semantic Prediction Potentials ((Grisoni et al., 2017) for discussions on VIPs, (Almeida, 2021a)) and the implicit Contingent Response (Fishman et al., 2021). These ERPs could be useful in the context of schizophrenia or FTD research. For instance, the Contingent Response is a wave that evolves (preceding predictable targets) as we implicitly learn contingencies between stimuli presented sequentially, whereas the Prediction Potentials were discovered in an explicit paradigm. It would be very interesting to look into whether these ERPs could discriminate between FTD and other cohorts, after all, both implicit priming and explicit top-down predictions are thought to be abnormal in the syndrome (see section 6.4). The Contingent Response could also be adapted to a lexical-semantic paradigm to help explain Bayesian linguistic knowledge in schizophrenia and healthy cohorts.

6. Alternative views

We have reviewed semantic priming studies in schizophrenia under a neurobiological perspective, mainly focusing on the most prominent, “disinhibited spread of activation” approach. For the upshot, we will break down the main competing hypotheses on priming aberrations in the pathology (for other mediating variables, see also Rossell and Stefanovic, 2007) - which count in disorganised semantic storage, psychomotor slowing, relatedness proportions, and an inability to mobilise contextual information.

6.1. Disorganised semantic storage

Let us begin with disorganised storage. Largely hinged on findings derived from semantic fluency studies (e.g., increased errors, low fluency), some authors proposed that schizophrenia’s semantic disturbances may reflect a disorganised semantic memory (e.g., Tan et al., 2015, Tan and Rossell, 2017; Rossell et al., 1999; Rossell and David, 2006). Whilst there have been only a few priming studies in the literature explicitly backing this hypothesis, resemblant anomalies have also been postulated to underpin hyperpriming (Rossell and David, 2006; Rossell and Stefanovic, 2007; Tan et al., 2015, Tan and Rossell, 2017; Kuperberg et al., 2019; and see Morgan et al., 2006a).

Particularly, the disorganised storage concept was first exhaustively worked on, to our knowledge, by Rossell and David (2006) (though analogous proposals may be found in earlier work, e.g., a potentially less organised or “complete” network was briefly mentioned by Aloia et al., 1998 on the grounds of poor reaction time differentiation between medium/low relatedness; this was observed in low, but not high FTD scorers). The authors administered lexical decision tasks to schizophrenic patients with low- and high-frequency words. They hypothesised that a degraded semantic store, such as putatively observed in Alzheimer’s disease, would promote hypopriming to low-frequency words (degraded), and hyperpriming to high-frequency ones (rendered more accessible). Since their experiment did not yield hypopriming for low-frequency words, Rossell and David (2006) suggested that there is no erosion of semantic nodes in schizophrenia. Rather, seeing that they found hyperpriming effects for high-frequency words, the authors posited that the network portrays an idiosyncratic organisation - nodes could exhibit larger conceptual overlaps. Needless to say, this hypothesis fits quite neatly with the aforedescribed N400 reversals due to hyperactive ensemble overlap.

Yet, findings on word frequency priming are equivocal in the schizophrenia spectrum. For instance, Morgan et al. (2006) administered a lexical decision task to schizotypes with two conditions: short (250 ms) and long SOAs (750 ms), with words of high and low frequencies. Strikingly, not only were there no differences in priming with different word frequencies, their results disclosed that low schizotypal scorers showed greater priming at the short SOAs relative to the long SOAs, whilst high scorers showed the *opposite* pattern (greater at long SOAs). It is hard to explain these results, though herein, the main point is that data on word frequency priming are only scant and contradictory, although there is one caveat to underscore. Namely, scarce data refers only to studies centred on word frequency priming. A more directed surveillance of the priming literature aimed at comparing results from different studies that specifically reported their word frequency could very well reveal some consistent correlations.

Notwithstanding, another study argued in favour of the abnormal semantic storage hypothesis, but from a different standpoint - namely, on the basis of direct and indirect priming effects. Tan et al. (2015) reported reduced percentage direct priming with unchanged indirect priming in schizophrenic patients (low FTD), with the authors proclaiming that their data reflected compromised storage, because overactivation would presumably bring about direct and indirect hyperpriming effects alike (Tan et al., 2015). However, the authors also stated that their results could be related to a decreased search area in a degraded store, which apparently distunes with the idiosyncratic network hypothesis as put forth by Rossell and David (2006) - albeit it seems tenable that larger conceptual overlaps should shrink search areas.

Finally, in some juxtaposition with this disorganised storage idea, a novel viewpoint on hyperpriming was put forth by Kuperberg et al. (2019), grounded on findings within the N400 time window. The authors administered an implicit task with a masked priming paradigm, utilising an SOA of 140 ms as participants monitored for words in a category under the recordings of both fMRI and MEG (with the same participants). They found indirect but not direct priming in the N400 window with both apparata, with the fMRI showing reduced activity for indirect targets. Their interpretation of the results was that the lack of direct in the presence of indirect priming was reflective not of a disinhibited spread - as they too argued that this would necessarily come with direct hyperpriming -, but of “noisy lexical representations”: roughly put, less finely-tuned links (no 1:1 correspondence) between word forms and meaning would allow for an indirect concept to be facilitated if presented shortly after the prime.

Altogether, even though an inhibitory hypofunction in schizophrenia is barely questionable, the above authors stand by a putative disorganised semantic or noisy lexical-semantic store, and have conceptualised it as conflicting with semantic disinhibition. Herein, we suggest that the two may not only be complementary, but causally linked. That is, schizophrenia is thought to be a disorder in which the spatial tuning of neuronal ensembles is disorganised - representations are frequently distorted and both their activation and encoding (thereby, storage) become unreliable due to the variance of noise; critically, this disorganisation is thought to arise from an impairment in lateral inhibition - especially from SST/MCs and PVs - and NMDAR hypofunction (Krystal et al., 2017; Murray et al., 2014; Hamm et al., 2017; Jackson et al., 2004), which disrupts the ability of the network to refine representations through sparse coding (interestingly, this deficit in spatial tuning accords with reports that semantic representations in brain magnetic clusters are poorly defined in schizophrenia, i.e., more diffuse/less clustered than in controls, Löw et al., 2006). Hence, aside from NMDAR alterations themselves, a disinhibition of the network (giving rise to semantic disinhibition) is not only consistent with a disorganisation of the store: it could *cause* it (Krystal et al., 2017).

Noisy links between representations as well as idiosyncratic overlap between concepts can thereby be comfortably articulated with semantic disinhibition - but what of the lack of direct hyperpriming? In an attempt to shed further light on this charade, we suggest the following. PV inhibition works by nearly *instantaneously* - but *transiently* - constraining excitatory spreads upon the arrival of bottom-up input to the cortex (e.g., Bartos and Elgueta, 2012; Yang et al., 2017; see also Zheng et al., 2012). Indeed, the suppression of these cells causes otherwise quiescent surrounding neurons and their columns to rise past suprathreshold depolarisation levels with the arrival of this input - without increases in suprathreshold recruitment in the central columns (Yang et al., 2017) (in turn, bursting pyramidal neurons that are thought to drive conscious access also hold twice the odds of swiftly suppressing their neighbours than actually exciting them, through MC inhibition, Silberberg and Markram, 2007; Berger et al., 2010; Takahashi et al., 2016, 2020bib_Takahashi_et_al_2016bib_Takahashi_et_al_2020)). Hence, we suggest that one could observe indirect hyperpriming (peripheral columns, unduly activated at roughly the same time as direct nodes) - without direct hyperpriming (central columns, activated as usual, or even with a delay) due to defects in lateral inhibition - i.e., semantic disinhibition in the cortex. In controls, with short SOAs, indirect associations are successfully and transiently inhibited (as in studies with dopamine), and as this transient inhibition dissipates in long SOAs, indirect concepts are primed. In schizophrenia, indirect concepts are disinhibited and primed already in short SOAs.

Critically, however, FTD is a syndrome that often remits, and we are aware of a study documenting hyperpriming to be mostly typical of acute FTD in psychotic states (Gouzoulis-Mayfrank et al., 2003; Leeson et al., 2005), as well as others stating that N400 alterations improved within a 1-year interval (whereas behavioural *hypo*priming did not, Besche-Richard et al., 2014; Iakimova et al., 2013; similarly, Jiang et al., 2015; for normal amplitudes in recovered but not non-recovered subjects with psychosis, Jackson et al., 2014). In tandem, these factors conspire to suggest that hyperpriming could be a state to a greater extent than it is a trait (perhaps matching the hypothesis that FTD is related to disorganised *access*, Leeson et al., 2005). They could lend support to the idea that transient semantic hyperactivity, caused by an unbalance in excitatory/inhibitory forces, is ultimately the causal factor in automatic hyperpriming, e.g., network hyperactivity in schizophrenia is most eminent during the early course of the illness (as are positive symptoms), receding at later stages (Krystal et al., 2017; Anticevic et al., 2015).

6.2. Psychomotor artefacts

Semantic priming ensues when a facilitatory effect is observed in the reaction times to a target word, relative to unrelated conditions. As noted by Chapman et al. (1994), there is a larger time gap between related and unrelated word pairs among individuals with slower reaction times (i.e., increased priming), to the extent that hyperpriming might be observed as a result of psychomotor retardation in some cohorts.

For its part, schizophrenia is widely known for its delayed reaction times, which raises questions as to whether PTD's hyperpriming is nothing but a psychomectric artefact (Rossell and Stefanovic, 2007; Pomarol-Clotet et al., 2008). Indeed, while some authors explicitly reported a lack of correlation (e.g., Moritz et al., 2001a; Kreher et al., 2008, 2009), a meta-analysis on behavioural priming has not garnered sufficient evidence to rule out this potential confounder (Pomarol-Clotet et al., 2008). Yet, it is critical to emphasise that, while schizophrenia exhibits such general retardation, this retardation may not be equivalent to that of older individuals (Kuperberg et al., 1997), and more importantly, reaction time gaps are supposed to be increased between related and unrelated word pairs due to psychomotor slowing, as observed in psychomectrics (Chapman et al., 1994). This does not account for the fact that reaction time differences between close and distant associations are often *decreased* in this pathology (e.g., indirect hyperpriming) (Kuperberg et al., 1997; 2006a, 2006b; Aloia et al., 1998). Hence, while there might be some covariation between psychomotor slowing and hyperpriming, it seems that such a link provides no elucidation of semantic indiscrimination whatsoever. Furthermore, it also seems to be challenged by hyperpriming effects found in bona fide implicit tasks (Kreher et al., 2008, 2009bib_Kreher_et_al_2008bib_Kreher_et_al_2009; Kuperberg et al., 2018, 2019bib_Kuperberg_et_al_2018bib_Kuperberg_et_al_2019) and semantic indiscrimination in N400 reversals (Wang et al., 2011).

Nuerobiologically, our take on these issues is the following. It does not seem far-fetched that the relative covariation of hyperpriming and slower reaction times is not a confounding variable, but a confounded variable: semantic disinhibition and motor retardation could stem from shared pathophysiological mechanisms in FTD. For instance, DRD1 hypofunction reportedly slows reaction times (Weed and Gold, 1998), and so does NMDAR blockade on choice reaction time tasks (Micallef et al., 2002) - both being associated with indiscriminate spreads of cortical activity, as discussed previously. Accordingly, the positive/disorganised dimension exhibits differential correlations with slower choice reaction times (Vinogradov et al., 1998). On those grounds, then, we suggest that while *direct* hyperpriming cannot be categorically ruled out as a partial artefact of psychomotor slowing, *indirect* hyperpriming is principally the outcome of suboptimal early lateral inhibition of indirect notes. Thus, direct and indirect hyperpriming and psychomotor slowing could all share similar substrates (e.g., NMDAR hypofunction), but their covariation does not entail full-blown causality.

6.3. Relatedness proportions

Another competing hypothesis to semantic disinhibition is based on relatedness proportions in priming studies. Namely, though these correlations were not unequivocal (e.g., Baving et al., 2001; Spitzer et al., 1994; Rossell et al., 2000; Condray et al., 1999), a review by Rossell and Stefanovic (2007) marshalled data from experiments applying lexical decision tasks with reported relatedness proportions to patients. Strikingly, studies with a low percentage of related word pairs (up to 25%) were prone to disclose normal or decreased priming in schizophrenia (Vinogradov et al., 1992; Henik et al., 1992; Besche-Richard et al., 2014; Besche-Richard and Passerieux, 2003; Besche-Richard et al., 2005; Ober et al., 1995, 1997bib_Ober_et_al_1995bib_Ober_et_al_1997; Passerieux et al., 1995; Chapin et al., 1989, 1992bib_Chapin_et_al_1989bib_Chapin_et_al_1992; Rossell, 2004), whereas higher percentages (>25%) more typically produced hyperpriming (Moritz et al., 2001a, 2001bbib_Moritz_et_al_2001abib_Moritz_et_al_2001b; Spitzer et al., 1993; Surguladze et al., 2002; Weisbrod et al., 1998; Henik et al., 1995; Manschreck et al., 1988; Kwapil et al., 1990). Vinogradov et al. (1992) proclaimed that such correlations with relatedness proportions, rather than the employment of different SOAs, would account for the variability in priming results: schizophrenic cohorts would avail themselves of heavier top-down mechanisms of semantic matching in conditions of higher percentages, whereby this process is more likely to occur.

These findings are certainly valuable, although there is a crucial flaw in this stream of criticism. In one particular sense, the above correlations could be spurious in that, to our knowledge, none but two of those studies reporting normal or decreased priming - which did not sample FTD (category vs. non-category word pairs, Ober et al., 1995, 1997) - with low proportions employed indirect or any other manner of distant associations (Vinogradov et al., 1992; Henik et al., 1992; Besche et al., 1997; Besche-Richard and Passerieux, 2003; Besche-Richard et al., 2005; Passerieux et al., 1995; Chapin et al., 1989, 1992bib_Chapin_et_al_1989bib_Chapin_et_al_1992; Rossell, 2004), whereas, of note, multiple studies with high proportions reporting hyperpriming did so (Moritz et al., 2001a, 2001bbib_Moritz_et_al_2001abib_Moritz_et_al_2001b; Spitzer et al., 1993; Surguladze et al., 2002; Weisbrod et al., 1998). Further, this account again fails to explain evidence of

hyperpriming in fully implicit tasks, where there is little influence of any compensatory top-down machinations (Kreher et al., 2008, 2009bib_Kreher_et_al_2008bib_Kreher_et_al_2009; Kuperberg et al., 2018, 2019bib_Kuperberg_et_al_2018bib_Kuperberg_et_al_2019). Finally, whilst the above findings seem to be independent of SOA (Rossell and Stefanovic, 2007), such an account does not seem to elucidate the preferentiality of hyperpriming for short SOAs in a plethora of studies that did not report word proportions (semantic matching is deployed in lexical decisions irrespective of SOA), or for indirect associations.

All in all, while relatedness proportions do not - at least yet - refute semantic disinhibition/indiscrimination, this is nonetheless an issue worthy of exploration: it might be telling of other, top-down neurocognitive alterations in schizophrenia.

6.4. Use of context

In conclusion, it would be opportune to address a more "cognitive" account of language disturbance in schizophrenia: patients would fail to mobilise top-down resources to inhibit or constrain the activation of irrelevant nodes according to contextual subtleties (e.g., Hardy-Baylé et al., 2003; Brown and Kuperberg, 2015; Titone et al., 2000; Kuperberg, 2010a, 2010b; Sitnikova et al., 2002; Meyer et al., 2021; see also Sharpe et al., 2020). Herein we have covered data on the N400 that provide excellent examples of this, in that they demonstrate how preceding contexts (prime) poorly modulate the amplitude of the following waves (target). As for behavioural priming, for instance, Titone et al. (2000) administered a sentence paradigm biased towards a particular meaning of a homonym to schizophrenics. Whilst controls ceased to prime dominants when the bias was moderate, patients required the latter to be strongly against it. Kindred findings are very common in the literature (e.g., Sitnikova et al., 2002).

Thus, schizophrenics may exhibit shortcomings for suppressing salient but contextually-incongruent information. This hypothesis seems concordant with semantic crowding - only, semantic crowding does not arise because of a lack of top-down influence, but because of its influence. Top-down information cannot be mobilised to sharpen lexical-semantic representations according to contextual nuance because its mobilisation is what obliterates contextual nuance - top-down may no longer be suppressive. This suggests that in an explicit task, if the SOA is short, there could still be priming of nodes that manage to stand out from the "blurred" background (such as in Titone et al., 2000), whereas if the SOA is long, even those nodes will be engulfed in accumulated noise.

Interestingly in that vein, Cohen and Servan-Schreiber (1992) also acknowledged the possibility of noise accrual with the assertion that "When gain is reduced in the context module, the representation of context is degraded; as a consequence, it is more susceptible to the cumulative effects of noise. If a contextual representation is used quickly, this effect is less significant, and the representation is sufficient to overcome a dominant response bias. However, if time passes (as when context is presented first), the effects of noise accumulate, and the representation is no longer strong enough to reliably mediate the weaker of two competing responses"; further, "By maintaining or increasing the gain of neurons in this area, dopamine may help augment contextual representations against a background of noise. This, in turn, would lead to better preservation of contextual information over time and more effective control over dominant response tendencies". Hence, the attenuated gain of a contextual representation in the prefrontal cortex due to hypodopaminergia could also render nodes susceptible to semantic crowding. This interpretation can be coalesced with our hypotheses to fuel future research.

7. Conclusion

Taking on a neurobiological stance, we have reviewed some of the key findings and theoretical postulates on the semantic priming effect in schizophrenia. The studies that were discussed may help us better comprehend linguistic disturbances in this pathology, as well as the neurochemical and mechanistic underpinnings of higher-order cognition. Most notably, we have gathered suggestive evidence that semantic disinhibition could be related to a loss of DRD1 and/or NMDAR function. DRD1, specifically, is known to support inhibitory processes in the cortex quite selectively through depolarisation of parvalbumin-positive (PV) interneurons (e.g., Gorelova et al., 2002). Thus, a plausible deduction is that FTD may be characterised by differential PV cell dysfunction - something that is particularly relevant given the cell's notorious hypofunction and central role in schizophrenia (e.g., Lewis et al., 2012). In addition, and among other things, we have argued that suboptimal Martinotti cell inhibition could underlie N400 reversals in schizophrenia (Almeida, 2021a), whereby related associations would elicit larger amplitudes due to disinhibition of superficial apical dendrites and unconstrained facilitatory excitation. This is observed especially in long SOAs, as is hypopriming. Hence, N400 effects and hypopriming could be instances of "semantic crowding", whereby the effects of feedback are rendered largely excitatory due to inhibitory deficits in target cortices, thereby resulting in unconstrained activation - i.e., apical disinhibition, semantic indiscrimination and insensitivity to context in controlled paradigms. Numerous other considerations on the FTD endophenotype and language deficits in schizophrenia were also put forth. All in all, empirical data seem to bear out a putative relationship between cortical disinhibition and linguistic disturbance in schizophrenia.

So what are our suggestions for future research? Dopamine abnormalities in priming studies have been granted most of the attention for decades, as the neurotransmitter's pharmacological modulation is the most obvious one to impinge on the spread of activation. Notwithstanding, most of these studies have been performed with explicit and coarse behavioural measures. To keep up with state-of-the-art developments in the psychiatric and neuroscience literature, we would provide neurolinguistic research with greater inferential power if we strived for the employment of more refined experimental methods. Specifically, a more exhaustive use of neuroimaging and psychophysiological techniques (such as functional MRI and N400 ERPs) would be helpful: these technologies carry the potential to reveal subtler influences of alternative neurotransmitters and pharmacological agents on the pathophysiology of FTD. Additionally, the effect of antipsychotics in semantic priming should be more thoroughly scrutinised: these drugs could be acting as substantial confounders in the literature. Indeed, this variable may be more important than it seems - dopamine has been shown to even *drive* lateralisation of neural activity in some language tasks (Fuertinger et al., 2018), which, as reviewed herein, is often reduced or reversed in schizophrenia. Moreover, some of these drugs modulate cholinergic systems to which the N400 and MMN seem to be sensitive (Almeida, 2021a). Lastly, factor analyses, meta-analyses and reviews should better parse the symptomatology of hyperpriming in FTD, so as to stave off empirical conflicts (e.g., primarily acute psychosis with FTD could give birth to the effect, Gouzoulis-Mayfrank et al., 2003), home in on an endophenotype, and endow priming measures with potential clinical significance in the future.

In conclusion, it is argued that neurolinguistic research on schizophrenia, as an underexplored field, could unveil a whole new range of completely novel and unexpected findings. As evidence already seems to suggest, these findings may herald clinical and scientific significance for an extremely pragmatic measure in the studies of language: the semantic priming effect.

Declaration of competing interest

None.

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