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Short Communication

Impaired Emotion-Cognition Interactions in Schizophrenia

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Overwhelming evidence suggest that schizophrenia is a highly hereditable disorder with a complex genetic architecture [1]. Available data indicate that schizophrenia is influenced by multiple and interacting genes, each having a small additive effect on the expression of the illness [2]. To deal with this complexity, researchers have used a complementary strategy based on the identification of endophenotypes or intermediate phenotypes (intermediate traits in the chain of causality between genes and diseases) thought to be genetically simpler than the clinical phenotype and, thus, assumed to be linked in more straightforward way to underlying susceptibility genes than the clinical phenotype itself [3]. Among the candidate endophenotypes, the disturbances of emotion-cognition interactions are considered to be the hallmarks of schizophrenia [4-8]. Indeed, considerable evidence linking deficits in emotion-cognitive interactions to possible heritability exists in schizophrenia patients as well as close genetic relatives exhibiting similar deficiencies [9,10]. Although research has clearly demonstrated that emotion-cognition interactions are strongly associated with genetic variations in some candidate genes, identification of susceptibility genes remain elusive [11]. This difficulty is due to the complex and polygenic nature of the disorder as well as to the relative complexity of endophenotypes. Therefore, further refinement of these intermediate phenotypes is needed in order to identify specific deficits and thereby improve their heritability estimates. Since deficits in emotion-cognition interactions reflect fundamental pathophysiological processes that contribute to the emergence of the clinical syndrome, they may also serve as targets for pharmacotherapeutic interventions, as opposed to clinical features. Thus, with the promise from pharmacogenetics of personalized medical treatment through genetically calibrated dosing, radical improvements in the identification and characterization of disturbances of emotion-cognition interactions are imposing in order to improve drug efficacy and reduce toxicity risk.

Emerging evidence suggests that affective information can have both enhancing and impairing effects on various cognitive processes. Moreover, these opposing effects can be identified at different levels, both within the same cognitive process and across different processes. The aim of the studies we are now carrying out is to investigate the mechanisms underlying the enhancement and impairing effects of emotion on different aspects of episodic memory (within the same cognitive process), and across different processes such as working memory *vs* episodic memory. A number of aspects may allow us to disentangling the effects of affective information on cognition, in general, and memory, in particular. Enhancement or impairment in episodic memory may be linked to central *vs* peripheral trade-offs, high *vs* low prioritization of information processing or single item encoding *vs* binding of complex associations. In the same manner, opposing effects across processes can be linked to dissociations between immediate *vs* long-term effects that are mediated by shared and different neural mechanisms, involving bottom-up and top-down processes. Overall, we are carrying out a series of studies that are important for understanding mechanisms of emotion-cognition interactions in healthy as well as in pathological populations such as schizophrenia, in which the opposing effects of affective information processing can be both beneficial and deleterious. In particular, we are currently carrying out studies in the following areas:

Prosody

Emotional prosody (or vocal emotions) refers to the emotional connotation given to a word or a sentence during speech. This connotation, commonly referred to as "the tone of voice", may include a series of non-lexical cues such as intonation, stress, pitch, and volume that together or alone communicate the intention, the emotional reaction or state of the speaker [12,13]. Importantly, being able to process vocal emotions is crucial in social communication since a word (or a sentence) can be interpreted positively or negatively, independently of the valence of the word itself. This makes vocal emotion a powerful contextual modulator of verbal content and depending on the vocal emotion; messages can convey completely different emotional meanings. For example, the statement "We just came back from a trip to Paris" has a general emotionally neutral semantic meaning, but by using a positive or negative tone of voice, the speaker can convey different emotional states about the trip. Interestingly, even though prosody plays a crucial role in comprehension, few studies have addressed the relationship between aging or clinical populations (schizophrenia, depression etc.), emotion and vocal intonation [14,15].

False memories for affective information

Many studies have shown a direct link between memory for emotionally salient experiences and false memories and there is evidence that emotionally arousing positive and negative information enhances cognition compared to neutral material. Forgetting and distortions in memory have therefore received much attention [16,17]. In particular, false memories, or memories about an event never experienced or alterations in the way an event is remembered, are rather common in everyday life. Most importantly, these errors have contributed to our understanding of normal memory functions and the qualitative features involved in the encoding and retrieval of complex memories [18], memory failure in specific brain diseases

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[19] and clinically relevant memory distortions in certain patient populations [20].

False memories seem to interact with the affective content of presented material and numerous studies have shown how the production of false memories is modulated by the affective content of the stimuli [21-23] although effects are complex and contradictory. On the one hand, affective content may reduce false memories due to heightened stimulus control. On the other, it may lead to an increase in false memories due to heightened elaboration processes. In addition, the question is further complicated by the fact that positive and negative content also seem to produce different effects on memory [24,25].

However, despite the growing body of evidence on the influence of emotion on memory distortions, the possible interaction between the emotionality of the to-be-remembered material on false memory occurrence in schizophrenia has not been studied yet. In a recent study [26] we investigated the effect of emotional contents of the to-be-remembered events on true and false memories in patients with schizophrenia relative to normal healthy controls. We used a task that employs photographs depicting events (for example, in the "bike trip" episode a series of colour photographs depict a girl going on a bike trip in a downtown area). For each script, we created photographs depicting cause-effect scenes. The cause-effect scene consisted of a single picture depicting a causal antecedent followed by three different affective outcomes: one positive, one negative, and one neutral. Valence was counterbalanced across participants so that each participant saw three episodes for each of the three affective outcomes. With these scripts, two types of false memories can be studied: gap-filling and inferential causal errors. Gap-filling errors represent the likelihood to remember non-presented photographs that are consistent with the studied scripts. The causal errors refer to the likelihood of remembering the non-presented cause of viewed action effects (for example, viewing an injured girl on the street with a car nearby might lead to the false recognition of the car hitting the girl, even if such scene was not experienced). Emotional episodes yielded a greater production of inferential causal errors overall in patients with schizophrenia than in normal controls. Valence did not affect performance in the patient group. Emotional information reduces the probability of generating causal errors in controls but not in patients suggesting that emotional memory impairments may contribute to deficits in reality monitoring in schizophrenia when emotional information is involved.

Auditory imagery

An advantage of the right ear (REA) in auditory processing (especially for verbal content) has been widely established in decades of behavioral, electrophysiological and neuroimaging literature. The laterality of auditory imagery, however, has received little more attention, despite its potential relevance for understanding auditory hallucinations in clinical populations. Moreover, questions regarding how affective stimuli may modulate lateralization of auditory imagery have yet been investigated.

Regarding anatomical substrates, it is well-known that the most asymmetric cerebral structure of the human brain belongs to the auditory cortex in the posterior portion of the Superior Temporal Gyrus (STG), namely the Planum Temporale (PT), more extended in the left than in the right hemisphere, mainly in right handers [27]. Moreover, this anatomical asymmetry directly leads to functional asymmetries (i.e., speech versus music sounds; spectral versus temporal processing, and soon), confirmed by experimental results [28,29]. In line with this, we may expect modulation of lateralization according to the valence of the affective material.

The REA is so reliable a bias that its deviation is considered a marker of language impairments, such as dyslexia [30], and correlates with psychiatric conditions, such as auditory hallucinations [31,32]. Interestingly, contrasting results have been collected regarding the relationship among PT size, REA effect, and auditory hallucinations: if on one hand a number of morphometric studies have shown the connection between reduced STG area and the severity of schizophrenic symptoms [33] as well as reduced REA , on the other hand an increased activation of the STG has also been shown in schizophrenic patients during the experience of auditory hallucinations [31,34,35].

Future directions

Schizophrenia is associated with disturbances in emotion-cognition interactions likely related to functional abnormalities in neural networks associated with emotion and cognition processes. Further research is required, however, to identify the neural correlates of emotion-cognition regulation. Available data suggest that deficits in emotion-cognition interactions meet several of the criteria of endophenotypes. Understanding the different cognitive deficits is important to improve their heritability estimates and develop targeted treatment for these deficits and associated social interaction problems.

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