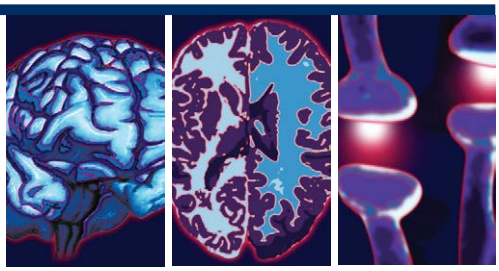
Research



Do People With Depression Always Have Decreased Cognitive Processing Speed? Evidence through Electrophysiological Lens

Daniel Kwasi Ahorsu, Hector WH Tsang†

ABSTRACT

Decreased cognitive processing speed is a well-known deleterious effect of Major Depressive Disorder (MDD). Decreased cognitive processing speed which is closely related to psychomotor retardation is seen as one of the classical cognitive impairment among MDD patients. This, although true in most cases, does not apply to all cognitive processes of MDD patients who are exhibiting symptoms of psychomotor retardation. This review focuses on the cognitive processing speed of MDD patients without comorbidity as compared to their healthy counterparts with respect to emotion perception and intend to argue based on evidence that cognitive processing speed is not always slow in MDD patients. Results indicated that in general, there is longer reaction time (RT) or N170 latency to tasks among MDD patients as compared to healthy controls but MDD patients have shorter RT or N170 latency for sad facial stimuli (relative to happy) as compared to healthy controls. Recurrent episodes of MDD have worse cognitive processing speed which is reflected in the tasks having the shortest RT or N170 latency to sad facial stimuli (relative to neutral and happy) as compared to healthy controls. To conclude, the evidence supports the view that MDD patients may have developed a trait which makes them predisposed towards negative information, especially for sad facial stimuli. This may also be a reason to explain why their cognitive processing speed is observed to be faster in perceiving negative stimuli. Further studies are needed to ascertain the complexities of the information processing pathways in order to shed light on the development of more effective therapeutic strategies to mitigate the effects that are associated with it.

Keywords:

Major Depressive Disorder (MDD), Cognitive processing speed, Emotion perception, Psychomotor retardation

Introduction

Major Depressive Disorder (MDD) is considered as one of the most prominent mental health disorders of public health concern [1] which is also a leading cause of disability [2] worldwide. Its symptoms cause significant clinical distress or impairment in social, occupational, and other important areas of functioning [3,4]. This leads to a loss of over $36.6 billion per year in the United States of America alone through absenteeism and presenteeism [5]. Presenteeism particularly insinuates the effect of depression even after treatment.

Good coordination of cognitive functions in the brain forms a good physiological condition which is the requirement for a well-orchestrated

Neuropsychiatric Rehabilitation Laboratory, Department of Rehabilitation Sciences, The Hong Kong Polytechnic University, Hong Kong

†Author for correspondence: Hector WH Tsang, Department of Rehabilitation Sciences, The Hong Kong Polytechnic University, Hung Hom, Hong Kong, Tel: +852 2766 6750; email: hector.tsang@polyu.edu.hk

personal [6], with normal social [6,7], and occupational life [8,9]. However, impaired ability to think, concentrate, or make decisions (distractibility), memory difficulties, decreased cognitive processing speed, impaired attention, etc. [4,10] constitute major symptoms among MDD patients. These symptoms may impede their coping abilities and thus interfere with their normal functioning in society [11] which includes their social life [10,12,13], and workplace productivity leading to lowered income or unemployment [5]. In this article, we put focus on decreased cognitive processing speed which is often regarded as a hallmark for MDD.

Does decreased cognitive processing speed always happen in MDD? This is a firmly held belief about MDD patients given the fact that psychomotor retardation is often observed in MDD patients. However, this, in fact, may not be necessarily true. This article intends to review existing literature pertaining to cognitive processing speed of MDD patients with respect to emotion perception and argue with evidence that the above belief is not true. Explanations will also be given which support our conclusion.

Faces are key to social communication [14] and easily convey our emotion to the outside world. Due to this, studies have mostly used faces to represent emotional stimuli. Difficulties including slowness in perceiving (detecting and discriminating), recognising, and appropriately interpreting emotions, in particular, facial emotions, will block effective communication which leads to further worsening and or development of emotional disturbance or illness [15]. It is therefore important to thoroughly examine cognitive processing speed which is also involved in emotion perception so as to properly understand the anomalies underlying MDD. A better understanding of this will inform clinicians and pharmacologists of the possible treatment regimen to use in mitigating the effect of depression. With the advent of neuroimaging and electrophysiological techniques, it has now become easier to study the cognitive processing speed involved in emotion perception.

Functional Magnetic Resonance Imaging (fMRI), Electroencephalography (EEG), and its derivative Event-Related Potential (ERP) have deepened our understanding of the mechanism of cognitive dysfunctions of MDD in the past decades. Among these techniques, ERP has the advantages of being non-invasive, relatively inexpensive, and superior temporal resolution [16].

Methods

Online databases, specifically *EBSCO, PubMed, ScienceDirect*, and *Wiley-Blackwell* were searched sequentially using the combination of the keywords; "event-related potential", "cognitive dysfunction", cogniti*, "major depressive disorder", and depress*. The reference lists of the selected articles were also reviewed for relevant studies. The search was limited to English studies published until September 2017, MDD patients without comorbidities, and having at least a comparable healthy control group. In order to make sure that the conclusions drawn by this review is valid, the Joanna Briggs Institute's MAStARI critical appraisal tools for Comparable Cohort / Case-Control Studies [17] was used to assess the methodological quality of the studies used in this review. In this review, only studies with a quality score of 7 or more were included.

Results

Six studies were finally selected which explicitly focused on emotion perception. Electrophysiological data can be streamlined into several processing stages based on the latency but this article puts focus on the early/perceptual (P1, N1, N170) processing stage. A careful examination of ERP studies comparing cognitive processing speed of MDD patients (without comorbidity) and healthy controls on emotion perception using happy, neutral, and sad facial stimuli have produced interesting results. Results from both behavioural (error/accuracy score and reaction time—RT) and electrophysiological (amplitude and latency) data point to the direction that people with depression may have developed a trait which predisposes them to bias towards sad facial stimuli. [18-22].

■ Emotion perception

Data from behavioural perspective indicates that F-MDD and R-MDD patients have higher intensity scores, arousal, or accuracy for sad faces as compared to healthy controls [18-22] and vice versa for happy facial stimuli [18-20,22,23]. F-MDD and R-MDD patients have shorter RT for sad faces but longer RT for happy and neutral faces as compared to healthy controls [18,22] especially among R-MDD patients [20]. No significant difference was noticed between groups on accuracies and RT but within MDD,

RT for sad facial stimuli was shorter compared to neutral facial stimuli whilst no difference existed within healthy controls [24].

Data from electrophysiological perspective indicates that in oddball tasks, R-MDD patients are reported to have the longest N170 latency compared to F-MDD patients and healthy controls in general but have the shortest N170 latency for sad faces as compared to F-MDD and healthy controls [20]. F-MDD patients have lowest N170 amplitude for all faces whilst R-MDD patients have lower N170 amplitudes for only happy and neutral but higher N170 amplitude for sad faces as compared to healthy controls [20]. Similarly, Wu, Chen [22] reported that MDD patients have higher N170 amplitude for sad faces as compared to healthy controls although they have longest N170 latency in general [22]. On the contrary, Wu, Zhong [25] reported no significant difference between MDD patients and healthy controls on N170 amplitude and latency. At the parietal and occipital sites, MDD patients were reported to have lowest P1 amplitude for happy but highest amplitude for sad facial stimuli during intensity evaluation task [18]. In a cued Target-response task, Zhao, Tang [24] reported higher P1 amplitude, in general, among MDD patients as compared to healthy controls at O2 electrode site but not for P1 latency. At P8 electrode, MDD patients had smaller N170 amplitude for only happy and neutral faces compared with healthy controls although no difference was observed for its latency [24]. Summary of the salient findings is presented in **Table 1**.

Discussion

This article examined cognitive processing speed of MDD patients without comorbidity in emotion perception studies using ERP

Table 1: Emotion Perception.

Author(s)	Aim	N (MA ± SD; M/F)	Task	Diagnosis/Type	Results
Chen, Ma [20]	To compare the neural processing of emotional facial expressions	FMDD = (30.6 ± 11.3; 21M/24F) R-MDD = (32.8 ± 13.6; 18M/22F) HC = (31.1 ± 10.8; 22M/24F)	EOP	DSM-V/ First episode MDD (FMDD) & Recurrent MDD (R-MDD)	Electrophysiological processing of emotional facial expressions is altered in patients with depressive disorders. Impaired emotional processing as indexed by N170 amplitude of positive and negative faces may be a useful biomarker for predicting the propagation and recurrence of depression.
Dai and Feng [18]	To investigate the intensity evaluation of social stimuli in depression	MDD = 24(26.58 ± 4.16; 10M/14F) Sub-Clinical = 24(25.96 ± 4.58;9M/15F) HC = 24(25.54 ± 3.41;8M/16F)	EIET	DSM-IV/ Sub-clinical MDD & MDD	MDD patients had higher intensity scores for sad faces compared with the HC group, longer reaction times (RTs) for all faces compared with other groups. The sub-clinically depressed individuals had lower intensity scores for happy and neutral faces compared with other groups, longer RTs for happy faces.
Jaworska, Blier [19]	To assess ERP profiles associated with facial expression processing	MDD = 53(40.7 ± 11.8; 24M/29F) HC = 43 (36.5 ± 9.8; 20M/23F)	FERT	DSM-IV	MDD individuals rated faces as sadder Females had comparatively enhanced pre- and conscious as well as speeded facial processing. Early perceptual and late conscious expression processing did not differ fundamentally between groups.
Wu, Chen [22]	To investigate whether there is an interaction of cognitive bias by gender on emotional processing in minor and major depression	MiD = 33 (31.1 ± 12.55; 16M/17F) MaD = 36 (33 ± 13.65; 16M/20F) HC = 32 (32.1 ± 12.95; 15M/17F)	EOP	DSM-IV/ Minor Depression (MiD) Major Depression (MaD)	Compared with HC, in male, both MiD and MaD had lower N170 amplitudes for happy faces, but MaD had higher N170 amplitudes for sad faces; in female, both MiD and MaD had lower N170 amplitudes for happy and neutral faces, but higher N170 amplitudes for sad faces
Wu, Zhong [25]	To examine the cognitive bias in pre-attentive emotional information processing in remitted late-life depression (r-LLD)	r-LLD = 30 (66.56 ± 7.57; 6/24) HC = 30 (64.75 ± 5.95; 11/19)	EOP	DSM-IV/ remitted late-life depression (r-LLD)	No significant between-group differences were found between r-LLD and HC on N170 amplitude and latency. An impairment with a negative bias in cognition was found among r-LLDs compared to HC during automatic processing of facial expression.
Zhao, Tang [24]	To examine how early perceptual processing is modulated by emotional stimuli in depression	MDD = 22 (31.68 ± 9.75; 12M/10F) HC = 22 (33.91 ± 8.93; 9M/13F)	cTRT	ICD-10	Depressed patients had larger P1 and N170 amplitudes than healthy controls, implying that early perceptual abnormality for sad face but N170 amplitudes for happy faces were larger than for other emotion types in healthy controls.

EOP: Emotional/Expression-related oddball paradigm; **EIET:** Emotional Intensity Evaluation Task; **FERT:** Facial Expression Recognition Task; **cTRT:** cued Target-response Task; **ICD-10:** International Statistical Classification of Diseases and Related Health Problems-10th Revision; **DSM IV/V**: Diagnostic and Statistical Manual of Mental Disorders 4th/5th version; **HC:** Healthy Controls; **FMDD:** First episode Major Depressive Disorder; **RMDD:** Recurrent episode Major Depressive Disorder; **r-LLD:** remitted late-life depression

methodology with both behavioural and electrophysiological data. From the reviewed studies, it is evident that MDD patients become comparatively better at identifying sad facial stimuli as compared to other facial stimuli. More interestingly, most studies report that they identify the sad facial stimuli faster (than neutral and happy) as compared to healthy controls as supported by RT and N170 latency. R-MDD patients are affected by depression more than F-MDD. These results provide the evidence that decreased cognitive processing speed is not always true for MDD patients. This, in fact, depends on the kind of data the MDD patient is processing.

As the results indicate, depression affects cognitive processing speed (reaction time—RT or latency). It has well been documented that MDD patients have decreased cognitive processing speed [3,4,10]. There are few contradictory reports [18,24,25] due to the course of MDD and methodological differences. However, most studies reported MDD patients as having shorter RT [18,20,22,24] or N170 latency [20] for sad (relative to neutral and happy) facial stimuli compared to healthy controls although MDD patients had longer N170 latency in general [20,22]. The interesting finding is the shorter RT/ latency for sad facial stimuli among MDD patients as compared to healthy controls [18,20,22,24] juxtaposing it to the results that MDD patients had longer RT/ N170 latency in general [20,22]. This is very interesting taking into consideration one of the known symptoms of depression—psychomotor retardation [3] also reported among MDD patients [20,22] in general. This emphasises the point that depression leads to the development of schema for processing negative information and the recurrent the episodes of MDD, the more it is ingrained. It is likely that the new neuro-pathway further propagates depression through the reduction in social reinforcement and support but an intensification of negative feedback seeking, reassurance seeking, self-fulfilling prophecies and interpersonal avoidance [20]. Future studies using brain imaging techniques should be done to locate the anatomical sites related to the altered neuro-pathways.

Based on the above, the following evidence further supports the view that MDD patients may have developed a trait which makes them predisposed towards negative information especially for sad facial stimuli [18-22]. This may also be a reason to explain why their cognitive processing speed is observed to be faster in perceiving negative stimuli. They have higher intensity or arousal for sad (compared with happy) facial stimuli as compared to healthy controls [18,24]. This blunts the patient to the outside world making them lose interest in pleasurable external stimuli but focusing on their inner world [3,18]. Closely related to perception or detection of sad faces is energetical resources allotted for cognitively processing the stimuli, amplitude. The amount of mental effort engaged during sad faces among MDD patients is comparatively higher with R-MDD patients having the highest amplitude (N170) for sad facial stimuli compared to happy and neutral facial stimuli [20,22,24]. The higher amplitude for sad facial stimuli is thought to enhance its cognitive processing speed which overshadows the cognitive processing of neutral and happy facial stimuli accounting for the 'seemingly' decreased cognitive processing speed of neutral and happy facial stimuli especially at N170 time window [20,24]. This again supports the point that MDD patients do not necessarily have decreased cognitive processing speed in all areas of their life.

■ Implications

It is now evidently clear that MDD patients have increased cognitive processing speed for sad (relative to happy) facial stimuli as compared to healthy control yet decreased cognitive processing speed in general. More studies need to be conducted to know the anatomical sites of the neuro-pathway for the negative information however, there are some few important points that can benefit clinicians and pharmacologists. Clinicians can explore the use of therapies that help to shift, alternate, and focus attention from negative to positive emotional stimuli like meditation and qigong. It is noteworthy that the effects of depression on emotion perception happen at a very early stage of perception (pre-attention) hence, pharmacologists can capitalise on this to influence perception, attention as well as improve the arousal of positive information processing among MDD patients. Finally, community and family members can also help by being considerate, non-judgemental, and supportive of the healing process so as to facilitate the recovery process and well-being of the MDD patients.

Conclusion

Psychomotor retardation has been well documented to be a symptom of depression,

however, ERP studies on emotion perception report that MDD patients can be faster in reacting to sad (relative to happy) facial stimuli as compared to healthy controls. More investigations need to be done but a probable explanation is that there is a new pathway for processing negative emotional information like sad faces. This new pathway provides a shorter route to processing only negative information which further increases the recurrence of depression. More studies using electrophysiological and or brain imaging techniques should be conducted to identify the exact neuropathway so as to inform the appropriate pharmacological or psychotherapeutic approach to manage people with depression.

References

1. Monroe SM, Anderson SF, Depression: The Shroud of Heterogeneity. *Curr. Dir. Psychol. Sci* 24(3), 227-231 (2015).
2. World Health Organisation. *Depression* 2017 (2018).
3. American Psychiatric Association. Diagnostic and statistical manual of mental disorders : DSM-5. 5th ed. DSM-5. 2013, American Psychiatric Association, © (2013).
4. Schulz EP, EG Arora, Depression: Continuum: Lifelong Learning in Neurology. *Behav Neurol Neuropsychiatry* 21(3), 756-771 (2015).
5. Lépine JP, Briley M. The increasing burden of depression. *Neuropsychiatr. Dis. Treat* **7**(1), 3-7 (2011).
6. Sewall KB. Social Complexity as a Driver of Communication and Cognition. *Integr. Comp. Biol* 55(3), 384-395 (2015).
7. Weightman MJ, Air TM, Baune BT. A review of the role of social cognition in major depressive disorder. *Front. Psychiatry* 5(1), 179 (2014).
8. Then FS, *et al.* Association between mental demands at work and cognitive functioning in the general population - results of the health study of the Leipzig research center for civilization diseases (LIFE). *J. Occup. Med. Toxicol* 9(1), 23 (2014).
9. Hunt E, TM Madhyastha. Cognitive Demands of the Workplace. *J. Neurosci. Psychol. Econ* 5(1), 18-37 (2012).
10. Millan MJ. Cognitive dysfunction in psychiatric disorders: characteristics, causes and the quest for improved therapy. *Nat. Rev. Drug. Discov* 11(2), 141-168 (2012).
11. Sole B. Cognition as a target in major depression: New developments. *Eur. Neuropsychopharmacol* 25(2), 231-247 (2015).
12. Tsang HWH. Psycho-physical and neurophysiological effects of qigong on depressed elders with chronic illness 17(3), 336-348 (2013).
13. Tsang HWH *et al.* The effect of Qigong on general and psychosocial health of depressed elderly with chronic physical illnesses: A randomized clinical trial. in The Eleventh International Congress (2003).
14. Engelmann JB, Pogosyan M. Emotion perception across cultures: the role of cognitive mechanisms. *Front. Psychiatry* 4(1), 118 (2013).
15. Delle-Vigne D. Emotional facial expression processing in depression: Data from behavioral and event-related potential studies. *Clin. Neurophysiol* (2014).
16. Light GA. Electroencephalography (EEG) and Event-Related Potentials (ERP's) with Human Participants. *Curr. Protoc. Neurosci* (2010).
17. The Joanna Briggs Institute, Joanna Briggs Institute Reviewers' Manual: 2014 edition. Australia: The Joanna Briggs Institute (2014).
18. Dai Q, Z lFeng. More excited for negative facial expressions in depression: evidence from an event-related potential study. Clinical neurophysiology : official journal of the International Federation of *Clin. Neurophysiol* 123(11), 2172 (2012).
19. Jaworska N. The temporal electrocortical profile of emotive facial processing in depressed males and females and healthy controls. *J. Affect. Disord* 136(3), 1072-1081 (2012).
20. Chen J. Distinct facial processing related negative cognitive bias in first-episode and recurrent major depression: evidence from the N170 ERP component. *PloS. One* 9(10), e109176 (2014).
21. Bistricky SL. Biased processing of sad faces: An ERP marker candidate for depression susceptibility. *Cognition. Emotion* 1-23 (2013).
22. Wu X. Cognitive Bias by Gender Interaction on N170 Response to Emotional Facial Expressions in Major and Minor Depression. *J. Cereb. Function. Dynamics* 29(2), 232-242 (2016).
23. Vanderhasselt MA. Decreased cognitive control in response to negative information in patients with remitted depression: an event-related potential study. *J. Psychiatry. Neurosci* 37(4), 250 (2012).
24. Zhao Q. Early perceptual anomaly of negative facial expression in depression: An event-related potential study. *Clini. Neurophysiol* 45(6), 435-443 (2015).
25. Wu Z. Negative bias in expression-related mismatch negativity (MMN) in remitted late-life depression: An event-related potential study. *J. Psychiatr. Res* 95(1), 224-230 (2017).