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The mirror neuron system may play a role in the pathogenesis of mass hysteria

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ABSTRACT

Mass hysteria or collective hysteria usually begins when an individual shows a hysteric manifestation in front of others in the same group who later contagiously acquire the same symptoms. The underlying pathogenesis of mass hysteria is still unknown. It has been demonstrated that the mirror neuron system (MNS) provides an important neural substrate for humans' ability to imitate and there is an inhibitive component of MNS keeping us from imitating everything we see. We proposed that the inhibitive component for MNS automatic imitation may not function well in individuals of the group that results in the outbreaks of mass hysteria. We also provide evidences from emotional contagion, gender difference and treatment in mass hysteria to support this hypothesis.

Mass hysteria (also called 'collective hysteria', 'mass psychogenic illness', or 'mass sociogenic illness') typically begins when an individual becomes ill or hysterical during a period of stress, whereby these physical symptoms (e.g. nausea, muscle weakness, fits or extremely erratic behaviors) are exhibited unconsciously and have no corresponding organic etiology [1]. After this initial individual shows these symptoms, others in the same group of the affected individual rapidly manifest similar symptoms [1]. It has been suggested that mass hysteria can be divided into two types: one with predominant features of anxiety (mass anxiety hysteria) and the other with predominant abnormalities of motor behavior (mass motor hysteria) [2]. The pathogenesis underlying mass hysteria is still unknown. Many scientists have tried to look for a predisposition to mass hysteria, but few consistent patterns exist across the studies [1]. Mass hysteria is accepted as unassociated with any psychological or personality disorders and as a behavioral reaction that anyone can show in the right conditions of fear and uncertainty [3]. The recipe for the outbreaks of mass hysteria seems to have been long-standing anxiety, which engendered dissociation and hyper-suggestibility – with the content of their delusions reflecting the dominant sociocultural concerns of the time and the group [1,4]. That is, the cultural belief systems served as a catalyst for the mass hysteria [5].

It is now well established that there exists a neuronal system named mirror neuron system (MNS) in both monkeys and humans. Through the MNS, during action observation, the neural structures involved in the execution of the observed actions are recruited in the observer's brain as if he or she is the agent of the action. Thus, the MNS allows the individual to gain an experiential knowledge of the observed action in the absence of any motor output [6–8]. In humans, it has been established that MNS is involved in a number of higher motor functions including imitation [9,10]. Whenever we observe the behavior of others, our MNS becomes activated and urges us to imitate the observed behavior. However, there is an inhibitive component of MNS keeping us from imitating everything we see because such automatic imitation is not always appropriate for effective social behavior [11]. It has been suggested that echopraxia, a neuropsychological condition that involves the involuntary repetition or imitation of the observed movements of another, is due to the dysfunction of the inhibitive component [12].

We suggest that MNS may play a role in the pathogenesis of mass hysteria. Some evidences to support this hypothesis are suggested below.

Firstly, one of the characteristic features of mass hysteria is the spreading of the symptoms via sight, sound or oral communication [1]. It is likely that the inhibitive component for MNS automatic imitation may not function well in individuals of the group that such spreading results in collective imitation and, then, the outbreaks of mass hysteria.

Secondly, both visual and auditory stimulation have been implicated in MNS [16]. This is in line with the clinical findings that symptoms of mass hysteria were transmitted by sight and sound, and clinical evidence showed that the treatment of mass hysteria consists of separating the participants [2].

Thirdly, in addition of motor imitation, recent evidence suggested that MNS plays a key role in emotional contagion – the tendency to catch and feel emotions that are similar to and influenced by those of others [13]. Mass hysteria may simply demonstrate the phenomenon of emotional contagion, in which the experience of an emotion seems to spread to those around us [14].

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Finally, MNS has been proved to have gender difference. That is, when individuals observe an action done by another individual, motor cortex, an automatic reaction of MNS, in female is more active than in male [15]. Thus, female will be more sensitive to the emotional contagion and, according to our hypothesis, likely to develop mass hysteria than male which is in line with the clinical observation that there is a preponderance of female participants in mass hysteria [1].

The above evidence suggests that MNS may play a role in the pathogenesis of mass hysteria. Further studies using neuroimaging techniques (such as fMRI and mu suppression in the EEG) in subjects with mass hysteria are needed to confirm this hypothesis.

Conflicts of interest statement

The authors declare that there are no conflicts of interest.

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