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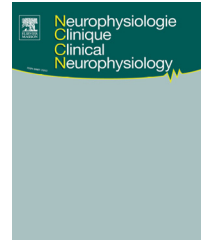


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## LETTERS TO THE EDITOR/LETTRES À LA RÉDACTION

## Heterogeneity in high hypnotic suggestibility and the neurophysiology of hypnosis



### *Hétérogénéité de la haute suggestibilité hypnotique et neurophysiologie de l'hypnose*

#### KEYWORDS

Heterogeneity;  
Hypnosis;  
Hypnotic suggestibility;  
Hypnotizability;  
Typology

Considerable progress has been made regarding the neural basis of hypnosis and a diverse range of studies attest to its utility as an experimental technique for modulating psychological phenomena in the laboratory and to its clinical efficacy. Against this backdrop, we read with great interest Vanhaudenhuyse et al.'s recent review on the neurophysiology of hypnosis [8], in which they succinctly, but comprehensively, review advancements in this nascent research domain. Here, we aim to supplement their review by correcting a mistake concerning heterogeneity in high hypnotic suggestibility and by highlighting the significance of heterogeneity for the neurophysiology of hypnosis.

The extent to which executive cognitive processes are altered in individuals who exhibit high hypnotic suggestibility has important consequences as such alterations may lend insights into atypical frontal or parietal functioning in this population. In reviewing the relationship between hypnotic suggestibility and conversion and dissociative symptoms, Vanhaudenhuyse et al. [8] implied that we found that highly suggestible participants displayed greater responsiveness to hallucination suggestions, greater involuntariness during hypnotic responding, impaired working memory capacity, and greater pathological dissociation and fantasy-proneness than low suggestible participants [5]. Contrary to what they

wrote, these differences were only observed in high dissociative highly suggestible participants, relative to both low dissociative highly suggestible and low suggestible participants. The latter two groups exhibited comparable cognitive profiles despite their differences in hypnotic suggestibility. Evidence of heterogeneity within the population of high suggestible individuals helps to illuminate nuances in hypnotic responding that are of profound importance to the neurophysiology of hypnosis.

Many studies have presented evidence that is consistent with the proposal that highly suggestible individuals are comprised of discrete subtypes. In addition to the differences described above, low and high dissociative highly suggestible participants have been shown to markedly differ in their responsiveness to particular hypnotic suggestions [4,5], the impact of a hypnotic induction on selective and sustained attention [3,6], and the cognitive resources required to respond to hypnotic suggestions [2]. These differences may further help to explain heterogeneous patterns of strategy utilization during responses to hypnotic suggestions [1,9]. Another study found that the two subtypes uniformly displayed lower frontal-parietal EEG phase synchrony in the upper alpha frequency band than low suggestible participants following a hypnotic induction, but did not differ from one another [7]. This suggests that differences between these subtypes may be difficult to observe in resting state studies. An outstanding question is whether highly suggestible individuals are indeed comprised of distinct subtypes or whether dissociative tendencies moderate individual differences in cognition and hypnotic responding in this population [10]. Irrespective of whether heterogeneity in this population is better explained by a typological or an individual differences model, heterogeneity is of critical significance for a nascent neurophysiology of hypnosis because it suggests that highly suggestible participants do not experience hypnotic suggestions through the same mechanisms, and thereby places important constraints on theories of hypnosis that assume homogeneity in this population. Future research and theorizing on the neurophysiology of hypnosis will be greatly strengthened by considering the characteristics of such heterogeneity and the mechanisms that underlie it.



## Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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## Deciphering the death of Jean-François Champollion (1790–1832), the man who decoded ancient Egyptian hieroglyphs

### *Décryptage de la mort de Jean-François Champollion (1790–1832), l'homme qui décrypta les hiéroglyphes*

Jean-François Champollion (1790–1832) is considered amongst the greatest linguists of all time; his decipherment of ancient Egyptian hieroglyphs and the Rosetta stone has led some to consider him the “Father of Egyptology”. His early death at the age of forty-one has typically been ascribed to fatigue and exhaustion from overwork on a yearlong visit to Egypt [1,2]. This journey is reported as having initiated his progressive demise following the return to his native France where he died from a reported stroke 2 years later. The use of the term “stroke” associated with his death is based on the generic application of this term, rather than through formal medical diagnosis and the concept of fatigue-related death remains to be the conventional understanding of his untimely passing.

Evaluating Champollion's life history, a rationalized medical diagnosis of his death can be achieved to identify a neurodegenerative causa mortis. Several factors support this notion:

- his symptoms of malaise primarily began before his journey to Egypt (1828) with upper limb weakness, where he was unable to write long letters due to spasms, cramps and fatigue. On his return to Paris in 1829 aged 39, the severity of his symptoms increased;
- he demonstrated slurred speech (dysarthria) noted during his lectures as professor of Egyptology;
- he subsequently developed a cough from recurrent chest infections.

He had no evidence of loss of cognitive ability and kept high levels of intellectual performance up to his death. There was no clear evidence of inherited family disorders. Although it was mentioned that he suffered from gout, scrutiny of these descriptions reveal the more accurate presence of limb pain and fasciculations of unknown cause. He was not known to be a heavy drinker, did not suffer from obesity, trauma or metabolic disorders [1,2].

Whilst in Egypt, he is not noted to have contracted any infectious diseases and did not suffer from symptoms of pyrexia or lymphadenopathy. In his final years, he did however suffer from muscle weakness, limb paralysis and ultimately was unable to maintain respiratory effort. He died a young man and was not noted for suffering from cardiovascular symptoms of angina, claudication of transient ischemic visual symptomatology. Furthermore, at the moment of decipherment of hieroglyphs (1828), he is noted to have collapsed, through this may be viewed as a vasovagal episode as a result of extreme emotional outpouring. In his final weeks he became emotionally labile consistent with