Title: Neuroscience evidence counters a rape myth

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Standfirst: Victims frequently report immobility during rape and sexual assault (IRSA), often using the term 'freezing'. Neuroscientific evidence suggests fear and threat can block cortical neural circuits for action control, leading to involuntary immobility. Defence arguments blaming victims for freezing are thus inappropriate and unjust.

Consent and rape myths

Rape and sexual assault (collectively, RSA) form a distinctive type of aggressive and criminal human behaviour overwhelmingly committed by men, and directed at women. 30% of women have experienced RSA in their lifetime. The burdens of RSA on individuals and whole societies are extensive, but the subject is often hidden. For example, in England and Wales, police recorded over 70,000 rapes in 2021-22 but only 3% led to a charge. Neuroscientific contributions to public debate over RSA have been limited.

Legal definitions of RSA are based on absence of consent. However, establishing consent or lack thereof is challenging. Victims' reports of non-consent are often questioned in court. Legal actors in RSA cases are susceptible to stereotypes ('rape myths') about how a 'real' victim would behave.

One common rape myth involves a perpetrator claiming that he had assumed consent from the absence of clear attempts by the victim to resist: Why didn't she struggle? Although struggle and violence play no part in formal legal definitions of RSA in many countries, rape myths such as this continue to influence thinking of jurors, lawyers, judges and wider society. Box 1 gives indicative examples of these misogynistic arguments, which seek to transfer the blame for RSA from perpetrator to victim.

The 'Why didn't she struggle?' argument is based on a cognitive model of intentional action that underlies all criminal law. Healthy adult humans are assumed to have voluntary control over their actions, and to carry out their actions intentionally. *Mens rea*, or conscious intention, makes agents responsible for their actions and consequences. Philosophical and legal arguments hold that one may also be responsible for omitting to act – not acting may be intentional in the same way that acting is intentional. In the case of RSA, this argument incorrectly assumes that the victim could have resisted or fled the attacker, yet she intentionally decided not to do so.

The neuroscience of involuntary immobility

We argue that the assumption that victims intentionally choose immobility over resistance is neuroscientifically incorrect. We suggest that RSA victims may remain immobile because of an involuntary neural response to threat[1] which blocks the brain circuits that provide voluntary control over body movement.

Further, legal discussions of the involuntary immobility response may involve an inappropriate double standard. The law has long recognized 'loss of control' defences, and can accord diminished responsibility in specific situations where evidence suggests actions are made outside of voluntary control. These include some medical conditions such as sleep disorders, but also extreme situations of coercive control, and emotional triggering.

To be consistent, legal systems should likewise recognize that omission of action may also sometimes be involuntary. An involuntary immobility response may prevent an RSA victim from responding by escaping or deterring the aggressor. This does not imply that the victim ought to make such actions: obligations and responsibilities in RSA lie with the aggressor, not the victim. Improved legal understanding of neuroscientific evidence regarding involuntary immobility during RSA could prevent inappropriate victim-blaming, and potentially draw wider societal attention to the crucial importance of active consent. Clearly, it is also possible to be voluntarily immobile without actively consenting, but our aim here is to draw attention to the frequency, mechanism and legal implications of involuntary immobility.

How the brain responds to threat

Aggression triggers a defensive cascade of fear/threat responses in the victim. Humans share many of these response patterns with other animals, reflecting evolutionarily-preserved brain circuitry for threat processing.

Neural and behavioural responses to threat depend on the severity and proximity of the threat, and also on perceived ability to escape. Many animal studies describe freezing as brief anticipatory, attentive immobility that occurs before a threat becomes immediate. The animal remains ready to act, so freezing is often considered as fight-or-flight on hold, pending the switch to action.

However, immediate, severe threats, such as physical restraint, may trigger a different kind of response, referred to as tonic immobility (prolonged immobility with a fixed posture) or collapsed immobility (characterised by loss of muscle tone) in animals. Human self-reports of tonic/collapsed immobility make clear that people are unable make voluntary actions during these states. Whether tonic or collapsed immobility is seen appears to vary according to species, and according to the nature of the threat[2].

Immobility behaviours are common during RSA: 70% of women attending an RSA emergency clinic appeared to have experienced tonic immobility during RSA[1]. Victims frequently describe themselves as 'frozen'. Because they consistently report inability to move or cry out, this behaviour more closely corresponds to tonic/collapsed immobility,

rather than the attentive anticipatory 'freezing' described in animal literatures[3]. To avoid terminological confusion, we propose the term immobility during RSA (IRSA) to describe this aspect of victims' behaviour. Victims report a strong desire to escape, together with an inability to do so[4]. Interestingly, paradoxical immobility and suspension of normal voluntary action is also reported in other situations of severe threat, including pilots' 'lockup' states during aviation emergencies.

The neuroscientific literature on threat responses in animals highlights a highly conserved, specialised circuit spanning several sensory and motor structures (see figure 1)[5]. The amygdala receives sensory inputs from evolutionarily-ancient subcortical circuits (blue shading in figure 1), but also from the association cortex, thus explaining how cognitive factors can potentially modulate threat-processing circuitry and thus reduce feelings of fear[5]. Immobility responses depend on specific circuits that the amygdala output targets (green shading in figure 1). The central nucleus of the amygdala projects to the ventrolateral subdivision of the periaqueductal grey (vlPAG)[6]. One recent study shows that attentive freezing in mice involves vlPAG projections to the magnocellular nucleus of the brainstem[6]. Further, specific neurons in the medullary brainstem underlie the switch from locomotion to stopping[7]. Studies in cats clearly showed sudden and generalised motor inhibition after stimulating brainstem motor areas, recalling collapsed immobility[8].

Animal studies can offer only limited insight into how threat-related immobility might impact human voluntary action control. Human neuroimaging studies confirm a similar defensive cascade involving the amygdala and periaqueductal grey[3]. We therefore propose that a circuit linking amygdala, periaqueductal grey and the brainstem motor nuclei may underlie threat-evoked immobility in humans, potentially including IRSA (see figure 1). Evidence from humans suggests that brief, attentive freezing following mild threat facilitates subsequent action[9].

However, human experimental studies are necessarily limited to mild threats, for obvious ethical reasons. The neural responses to severe inescapable threat that characterize RSA cannot therefore be studied experimentally. Instead, questionnaire studies have investigated self-report of past RSA events, using a Tonic Immobility Scale that draws on neurophysiological studies of immobility responses in animals[10]. This research has identified a common factor involving an inability to make voluntary vocalizations and voluntary actions, even in the absence of physical constraint.

We advance two hypotheses that we hope may drive future research. First, the vlPAG drive to the brainstem motor nuclei might lead to inhibition of descending voluntary motor commands (see Figure 1, yellow shading). For example, the brainstem gigantocellular nucleus relays cortical voluntary motor commands to the spinal cord to control context-appropriate action[11]. In cats, ventral reticulospinal axons with cell bodies in the gigantocellular nucleus synapse onto inhibitory spinal interneurons, suggesting a candidate mechanism for inhibiting voluntary action. Inhibitory circuits also exist within the human brainstem itself, but these have not been studied in the context of threat processing. Second, we suggest that two forms of IRSA might exist: one in which muscle tone is preserved, similar to the classical concept of tonic immobility, and a hypotonic "floppy" form of immobility[3]. Further research is required to identify and compare the presentation,

aetiology and sequelae of these two behaviours, including their implications for victim rehabilitation, and for legal outcomes. Importantly, both forms of IRSA would constitute involuntary responses to extreme threat, and both would involve suspension of normal voluntary action control.

Involuntary action and the law

Our interpretation of IRSA as involuntary, threat-induced block of normal voluntary action control raises profound questions about voluntariness, autonomy, and consent. Voluntary action depends on the functioning of specific brain circuits centred on the frontal lobes, and also on the non-engagement of other brain circuits associated with fear and threat, centred on evolutionarily-preserved specialised circuits that also involve subcortical structures.

Legal systems provide normative principles for investigating, explaining and judging human voluntary actions[12]. Legal process often poses "why?" questions about human behaviours, and explains both actions and omissions by reference to agents' intentions. Individuals are held responsible for their actions and omissions when these are intentional and voluntary, but responsibility is qualified or reduced when actions and omissions are unintentional, and individuals are typically not held responsible for actions that are involuntary. Thus, legal concepts of responsibility inevitably implicate the neurophysiological question of the role of cortical voluntary action circuits in control of behaviour.

We suggest that, given the neural mechanisms underlying responses to severe threat, IRSA can be considered as an involuntary omission of voluntary action. As such, legal "why" questions regarding IRSA would not have the same force as for intentional actions or omissions. Legal process should recognise this fact, and ensure it is consistently applied.

We highlight a second issue for legal process surrounding IRSA. Legal case reports show that victims' accounts of immobility during RSA are often disjointed, and lack conventional explanatory terms (see Box 1). The victim may have difficulty answering "why" questions. Defence lawyers often exploit this fact, drawing attention to a victim's inability to articulate and justify their behaviour during RSA. This appears to be straightforward victim-blaming, diverting the court's attention from the aggressive behaviour of the assailant towards the purportedly strange behaviour of the victim. In fact, recent neuroscientific advances can also help in understanding why victims often have difficulty explaining their own behaviour. Victims' accounts of IRSA are likely to share the fragmented, incoherent quality that is characteristic of traumatic memories in general.

In addition, memories of IRSA refer to an unprecedented experience, namely losing voluntary control over one's own body. Voluntary agency is the chief backdrop to mental life in healthy adults, so a sudden loss of ability to act in accordance with one's wishes is likely to seem strange and inexplicable, even in the absence of trauma. Clinical neuroscientific evidence from a range of conditions confirms both the disorganisation of traumatic memory[13] and the bizarreness of losing volitional control[14] that are also present in IRSA. The law already recognizes in evidential guidelines that trauma may affect the ability

to remember and explain events, including one's own behaviour: yet this point seems often ignored in legal discussions of IRSA.

Conclusion

We argue that IRSA is an evolutionarily-conserved involuntary response, characterized by lack of normal voluntary motor control, with further distinctive relations to sexual aggression and fear, and with subsequent memory effects. We hypothesize that IRSA emerges when the aggressor's behaviours activate the victim's brain's threat-defence circuitry, leading to inhibition of the neural pathways underlying the victim's voluntary action control.

This interaction between neural circuits for threat-processing and for voluntary action remains a scientific hypothesis, as we can only have indirect evidence regarding patterns of neural activity during IRSA. Instead, our argument relies on indirect evidence, such as victim testimony and from studies of defence circuitry in animals. This evidence, though indirect, is substantial and convergent. Ethical and moral hazards surround almost all research in this area, making it difficult for neuroscientific research to directly address the legal and societal issues raised by RSA. However, neuroscience should still, in our view, contribute to public debate regarding RSA. For example, mechanistic understanding of IRSA may help to counteract rape myths and ensure justice. Our hypothesis of threat-induced involuntary inhibition of voluntary action pathways may contribute to improving understanding of the facts about RSA crimes, the societal wrongs of gender violence, and the realities of victims' experience and suffering.

Neuroscientists can also make important contributions to justice in this area. For example, a recent intervention study showed that educating police officers to understand the involuntary neural mechanisms of IRSA reduced acceptance of rape myths. Moreover, neuroscientifically-informed training for officers improved victims' willingness to continue with legal proceedings[15] and such training could potentially improve legal outcomes and justice. Finally, increased awareness of IRSA may benefit victims themselves, by reducing victim-blaming including self-blaming and inappropriate feelings of guilt.

A recent Spanish law explicitly requires that consent must be freely and clearly expressed by a person's actions. This progressive and enlightened legislation clearly rules out the rape myth that IRSA could ever be interpreted as consent. Arguments and attitudes implying that immobility might be misinterpreted as consent are neuroscientifically mistaken, and unjustly blame victims. Neuroscience may have a role in helping legal systems and wider society to guard against such rape myths.

Box 1. A persistent rape myth.

Illustrative quotes from case reports show how defence lawyers and judges misrepresent freezing, and immobility during rape and sexual assault (IRSA). Note the victims' difficulty in explaining immobility ("I just…").

Victim testimony is in *italics*. Court reports of defence lawyer and judgement summaries are given in standard font.

R v Dunrobin (2008)

- *I just – I just froze in a way, like I was just scared. And like I didn't know him and I didn't know what he could do to me.*

R v Lennox (2018)

- Did you say anything? *No.* Did you do anything? *No. I just – I didn't do anything*.
- But I suggest, though, that if he was in front of you in the car and he's forcing that to happen, you could have just simply held your legs together? *Yes.* But you didn't do that?

United States v Townsend (1992)

- While there was evidence of the victim saying 'no' on several occasions, there was no evidence that further resistance would have been futile. When asked why she froze, [she] answered *"I don't know, I'd said, 'stop' and he wasn't stopping so I— if he just did what he had to do, then he'd—he—he would just leave."*

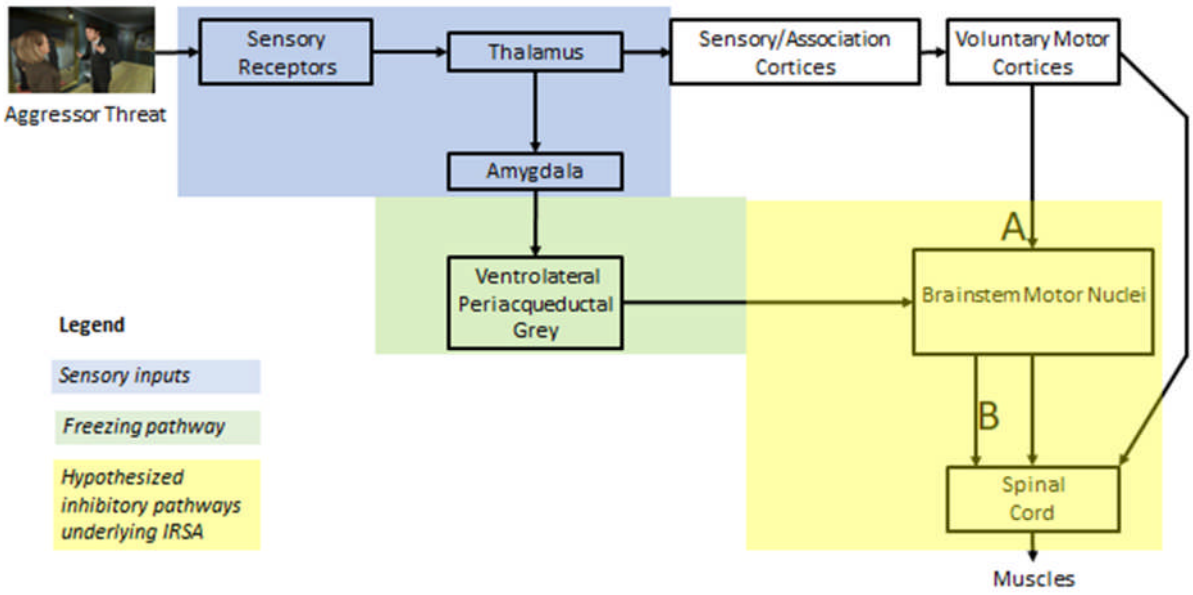

Figure 1.

A hypothesized neural circuit underlying suspension of voluntary action control during IRSA. Blue shading; Threatening sensory stimuli are processed by the amygdala in both humans and other animals. Green shading: freezing responses in animals depend on a ventrolateral periaqueductal grey circuit, that receives amygdala input, and then projects to brainstem motor nuclei, including the magnocellular nucleus[6]. Yellow shading: we hypothesize that circuits within brainstem motor nuclei could suspend normal voluntary action by blocking the relaying of cortical voluntary motor commands (A), or by descending control of inhibitory interneurons in the spinal cord that block cortical commands for voluntary action from accessing motoneuronal projections to muscles (B). Video image taken from with permission from doi: 10.3389/fpsyg.2020.00820.

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Competing interests

The authors declare no competing interests.