Sadistic cruelty and unempathic evil: Psychobiological and evolutionary considerations

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Abstract: Understanding the origins of evil behaviour is one of our most important intellectual tasks. A distinction can perhaps be drawn between overt sadistic cruelty and the lack of empathy to suffering that is a hallmark of evil. There is increasing data available on the prevalence, proximal psychobiological underpinnings, and distal evolutionary basis for these contrasting phenomena.

Understanding the origins of evil behaviour is one of our most important intellectual tasks, and Nell makes a useful contribution by emphasizing the ubiquity of cruelty, its reward value, and its emergence over the course of evolutionary history.

Although the term *evil* has origins in theological rather than scientific literature, it is useful insofar as it emphasizes that cruel behaviour forms only a subset of a larger class of violent behaviour that involves the infliction of physical or psychological pain on others. Cruelty is often associated with delight or with other forms of arousal in the pain of others (as Nell points out), whereas other kinds of violence may simply involve a failure to be sufficiently empathic to the suffering of others (e.g., the evil of bystanders).

If this distinction between overt sadism and a lack of empathy is valid, then immediate questions arise about the relative prevalence of these different kinds of phenomena, about their proximal psychobiological mechanisms, and about the distal evolutionary origins that underlie them. A large body of literature has tackled this area, but at this point in time there are perhaps more questions than answers. Nevertheless, a number of points can be made about the prevalence, psychobiology, and evolutionary underpinnings of overt sadistic cruelty and lack of empathy to suffering.

In an influential review, Baumeister emphasized that absolute cruelty – brutality inflicted on innocent victims for sadistic pleasure – is rare (Baumeister 1999). Instead, he argued that most violence can be understood in terms of emotions such as fear, lust, pride, and idealism. If he is correct, most perpetrators do not enjoy their acts, but nevertheless feel justified in doing them. Certainly, while it is crucial to recognize the overt sadism in the acts of cruelty described by Nell, it is equally important to recognize the banality of evil involved when individuals and societies ignore the suffering caused by their violent acts (Kaminer & Stein 2001).

The neuropsychiatric literature would seem to suggest a distinction in the proximal psychobiology of overt sadism and unempathic evil. Temperolimbic lesions may lead to sadistic behaviour, and more commonly, prefrontal lesions are associated with a lack in empathy and inhibitory dyscontrol (Stein 2000). fMRI studies have indicated that it is not only the occasional patient who takes pleasure in the suffering of others; reward centres are ordinarily activated during altruistic punishment (de Quervain et al. 2004). Similarly, inhibitory dyscontrol is also not uncommon; adolescence and substance use are associated with decreased prefrontal capacity (Chambers et al. 2003).

The evolutionary literature may shed further light on the distinction between overt sadism and unempathic evil. As Nell concludes, there is currently little evidence that cruelty is an adaptation underpinned by a hard-wired model of the brain. In contrast, there is strong evidence that empathy is an adaptation with a specific neurocircuitry and particular adaptive value (Preston & de Waal 2002; Stein 2005). Nevertheless, it is not necessarily adaptive to extend one's empathy to all; there are individual differences in empathic capacity, and in individuals' willingness to extend empathy to unrelated individuals or to other species (Stein 1996).

Nell provides some useful suggestions about the measurement of individual differences in the capacity for cruelty. Here it is relevant to emphasize the possible impact of differences in early environmental adversity on subsequent proneness to sadistic or unempathic behaviour. Prevalence data have emphasized an association between early trauma and adult psychopathology (Paolucci et al. 2001). Psychobiological research has noted that early adversity may disrupt dopaminergic neurocircuitry and reward-related behaviours (Stein et al. 2005). And an evolutionary literature has suggested that in the context of high levels of environmental adversity, impulsivity may be adaptive (Gerard & Higley 2002).

Violence not only presents moral quandaries, but it is a major public health issue. Is it possible to translate gradual insights into the biological and evolutionary psychology of cruelty and evil into the prevention of violence? Arendt, Baumeister, Nell, and many others have emphasized the need to begin by acknowledging the ordinary and universal human capacity for cruelty and evil; these behaviours cannot merely be relegated to those who are "abnormal" or otherwise marginal. Other steps are also needed; individuals and societies need to increase their awareness of violence and to use their empathy and understanding to reduce cruelty and evil (Stein et al. 2002).

Epigenetic effects of child abuse and neglect propagate human cruelty

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Abstract: The nature of children's early environment has profound long-term consequences. We are beginning to understand the underlying molecular programming of the stress-response system, which may mediate the destructive long-term effects of cruelty to children, explain the evolutionary stability of cruelty, and provide opportunities for its reversal of early trauma.

In the target article, Nell tries to demonstrate that cruelty is a historically and cross-culturally stable feature of human behavior. Although the elaborations of cruelty for punishment, amusement, and social control may have arguable evolutionary merits, the problem of explaining cruelty directed against children – child abuse – is profound and perplexing for humans. In fact, recent surveys suggest rates of child abuse to be alarmingly high and unequivocally damaging. For example, child sexual abuse prevalence is at least 20% for women and 5%–10% for men worldwide (Freyd et al. 2005). Further, in clinical (Brown & Anderson 1991), community (Bifulco et al. 1991), and epidemiological samples (Holmes & Robins 1988), experiences of early child maltreatment have been associated with the burden of higher rates of major depression, anxiety, and other psychiatric disorders. More recent studies have begun to examine mechanisms. In one study of 268 adults, retrospective questionnaire responses indicated a significant association of childhood trauma and impulsivity (Roy 2005). Further, Pine et al. (2005) found an association between maltreatment and attention avoidance of threatening faces in 34 children who had been abused. The significant psychiatric sequelae likely result from a plethora of evolutionarily adaptive mechanisms that normally mediate positive influences, which are co-opted by trauma to affect children's sensitive, developing, and adaptive nervous systems (Worthman & Kuzara 2005). Research so far focuses on epigenetic modulation of the stress-response system by the experience of violence and neglect

(Bevans et al. 2005; De Bellis 2005; Heim et al. 1997a; 1997b; Kaufman & Charney 2001; McEwen 2003). Increased stress may lead to chronic cortical dysfunction and activation of the hypothalamic-pituitary-adrenal axis with associated systemic health detriments including hypercortisolemia, immunosuppression, and elevated blood pressure.

Animal studies have examined the impact of trauma on developing brain function. In rat models, infant stress through maternal separation causes increased basal and stress-induced adrenocorticotropic hormone (ACTH) and corticosterone, increased hypothalamic, amygdala and locus ceruleus noradrenaline and corticotropin activity, reduced gamma amino butyric acid (GABA) tone, and hippocampal atrophy (Ladd et al. 1996). However, ACTH response to air-puff startle decreased with handling or foster mothering (Huot et al. 2004). Similarly, in infant macaques, peer rearing rather than maternal rearing (a model of neglect and abuse) is associated with increased ACTH response to separation. However, serotonin gene promoter polymorphism (5-HTTLPR) moderated this effect: Animals with the l/l allele had a lower ACTH response (Barr et al. 2004). Although the molecular details of how childhood experiences shape mental health in humans is unknown, the model of maternal care as the mediator of experience-dependant changes in gene expression has been the subject of numerous recent studies in rats (Meaney & Szyf 2005). Meaney and colleagues describe a fascinating model, in which the environment shapes the expression of glucocorticoid receptors. Tactile maternal stimulation of the rat pup (licking and grooming) stimulates serotonin release in the hippocampus. This, in turn, increases the second messenger cAMP, which activates protein kinase A and stimulates nerve growth factor 1A (NGF-1A), which increases glucocorticoid receptor expression. In addition, NGF-1A causes long-term cytosine demethylation, and histone acetylation that increases NGF-1A binding into the animal's adulthood. This is likely one of several mechanisms by which cruelty towards infants – in the form of physical, sexual, and neglectful traumatic abuse – has long-term consequences.

The possible mechanisms by which early trauma may shape long-term mental health are beginning to be studied (Bevans et al. 2005) in humans as well. In adults, trauma is associated with a range of measurable changes in the stress-response system including increased CRF and noradrenalin in cerebrospinal fluid (Bremner et al. 1996). In one of the first studies of gene–environment interaction in the manifestation of psychiatric problems in children, Kaufman and colleagues have found that the quality and availability of social supports moderated risk for depression associated with a history of maltreatment and the presence of the short (s) allele of the 5-HTTLPR (Kaufman et al. 2004). Maltreated children with the s/s genotype and poor positive supports had the highest depression ratings – scores that were twice as high as the non-maltreated comparison children with the same genotype. However, the presence of positive supports reduced risk associated with maltreatment and the s/s genotype, such that maltreated children with this profile had only minimal increases in their depression scores. These findings are consistent with emerging preclinical and clinical data, suggesting that the negative outcomes associated with early stress are not inevitable. In fact, it appears that the risk for negative psychiatric outcomes is modifiable through both genetic and environmental factors. Specifically, it appears that the quality and availability of social supports are among the most important environmental factors in promoting resiliency in maltreated children, even in the presence of a genotype expected to confer vulnerability for psychiatric disorder.

Given the debatable benefits of cruelty, especially with the knowledge of the negative psychiatric outcomes, and the predisposition to warlike behavior (as described in the target article) in an age of weapons of mass destruction, it appears fortunate that human adaptability through experience-dependant chromatin plasticity may provide a means to reduce human violence. Perhaps cruelty is preventable through interventions at the earliest stages of human development aimed at eliminating child neglect and abuse. Further studies of the molecular mechanisms may also suggest treatments aimed at older children and adults already affected with approaches that combine pharmacological targeting of those molecules along with psychotherapy aimed at reprogramming the stress response system.

Predation versus competition and the importance of manipulable causes

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Abstract: It is difficult to fully account for (1) cruelty in modern society and (2) female cruelty, referring only to a cruelty-satiation association. Instead it seems likely that cruelty acquires its reinforcing value via association with a range of reinforcers. In addition, when one's goal is violence prevention, it is important to identify causes that can be manipulated.

Nell argues that cruelty is a by-product of predation and suggests that signs of pain, blood, and death come to function as positive reinforcers as a result of their association with satiation. However, in modern society few individuals will have the opportunity to acquire such associations, and yet, as Nell points out, examples of cruelty abound. How then can we account for these? One possible explanation is that signs of pain and death have at some point is our evolutionary history become primary (i.e., innate) reinforcers, on a par with food and sex. This seems inconsistent with the vast numbers of men who find the idea of physical cruelty highly aversive. An alternative explanation is that our predatory heritage has left us predisposed to more readily acquire signs of pain and death as *conditioned* reinforcers (see Seligman [1971] for a similar theory of "preparedness" in relation to phobias). Thus, signs of pain and death would very rapidly come to function as reinforcers but *only* after the individual had associated them with existing reinforcers (e.g., approval or submission; see Skinner 1953). It would be possible to test this hypothesis experimentally, though perhaps a challenge to do so ethically.

However, this cannot account for the data on female aggression. Nell states that, because of its association with hunting, active cruelty is likely to be strongly male-gendered. Yet research shows that while males employ more "direct" or "overt" forms of aggression than females (e.g., physical aggression), females employ more "indirect" or "relational" forms of aggression than males (e.g., excluding someone from a group, spreading rumours) (Bjorkqvist et al. 1992; Crick & Grotpeter 1995; Tapper & Boulton 2004). These indirect forms of aggression are consistent with Nell's definition of cruelty as "the deliberate infliction of physical or psychological pain on a living creature" (target article, sect. 1).

What are the implications of female aggression for Nell's theory? If in our evolutionary history cruelty was a marker for male hunting skills, it would have been adaptive for females to be attracted to males who displayed this trait. In a culture that is reliant on hunting, it is also reasonable to assume that females would not be averse to cruelty and may also derive some pleasure from it. However, given that most hunting would have been carried out by males, one would still expect to find higher levels of cruelty among men. How then can we account for the fact that when it comes to psychological cruelty, the evidence suggests that females are more likely to be the perpetrators?

One possible explanation is that much cruelty is a by-product, not of predation, but of competition for resources. (For females