Self-Injurious Behaviours in Autism: A Literature Review

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Abstract

Self-injury is a frequent and serious problem for individuals with autism and developmental disabilities. This paper summarizes the clinical and empirical evidence pertaining to self-injury, and highlights valid theories and treatment options. Unlike most reviews of self-injury, the present one pays particular attention to the impact that research has made within the field of autism treatment. The importance of prospective large-scale research is stressed to support the development of treatments that should alleviate or even prevent the primary causes of self-injurious behaviour. This review aims to impart readers with an unambiguous conceptualization of self-injury and hopes to stimulate further research within the field.

There is little debate over the seriousness of self-injurious behaviour (SIB) in individuals with autism and developmental disabilities. There exists, however, considerable discussion over the etiology, functions, and treatment of SIB. This is problematic since proposed therapies are often shaped by the hypothesized cause of the problem (Matson, 1988), and are inherently linked to issues of human rights (Rothenberger, 1993a). Thus, a literature review examining the association between self-injury and autism is both clinically and academically relevant.

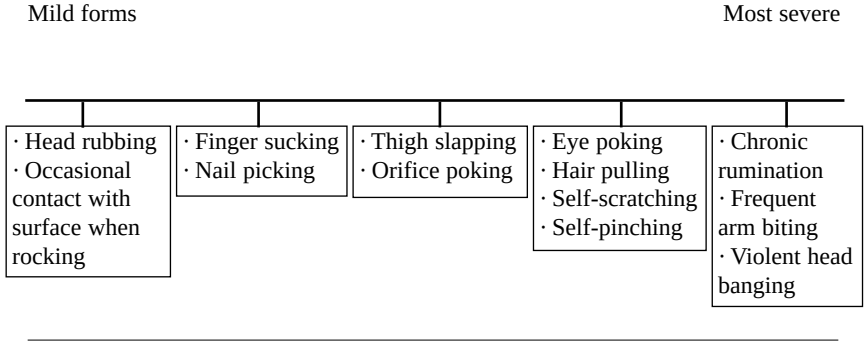
Unfortunately, there has been little theoretical or empirical investigation of self-injury and autism in particular. This is perhaps due to the considerable co-morbidity of autism in developmentally disabled populations, and further, to the fact that self-injurious behaviours in individuals with autism and individuals with developmental disabilities without autism seem to be functionally similar (Oswald, Ellis, Singh & Singh, 1994). Consequently, while this review is meant to highlight SIB in individuals with autism, it also examines studies of SIB in the field of developmental disabilities in general. By summarizing the leading taxonomies, providing current data on the specificity of SIB in autism, and highlighting clinically and empirically validated etiological theories and treatments, this review hopes to provide clinicians and researchers with a practical and clear conceptualization of self-injury.

Definition and Classification

It is important to review and arrive at consensus over the definition and taxonomy of SIB, in order to generalize findings across studies and design appropriate treatments. Self-injurious behaviours can be seen as "a class of behaviours, often highly repetitive and rhythmic, that result in physical harm to the individual displaying the behaviour (Fee & Matson, 1992, p. 4)." Moreover, such behaviours should occur without an apparent intent of wilful self-harm. It is important to distinguish between behaviours, such as suicide, that are associated with intentional desires to cause self-harm, and SIB found in individuals with developmental disabilities, which may occur in association with biological pathologies or are more a result of environmental factors. Intentionally self-harming behaviours are also often referred to as *self-mutilation, self-destructive, or masochistic behaviours* (Fee & Matson, 1992).

Self-injurious behaviours are often placed on a continuum with repetitive sterotypies, and have been suggested to only differ at the moment of injury (Jones, 1987). In fact, recent reviews of repetitive behaviours in autism place SIB within its broad class (Turner, 1999). Self-injurious behaviours can range from severe, life-threatening injuries to less directly damaging cases. In their summary of SIB taxonomies, Fee and Matson (1992) place numerous examples of SIB on a continuum ranging from mild to severe, reproduced in Figure 1. For example, cases of ruminative vomiting (Lang & Melamed, 1969) and uncontrollable climbing (Risley, 1968) are considered SIB since they can indirectly cause harm and are treated with the same behavioural techniques used for other injurious behaviours (Murphy & Wilson, 1985).

Figure 1: Self-injurious behaviours on a continuum from mild to severe



The classification of SIB has occurred on the basis of both conceptual and empirical grounds. In terms of diagnosis, the DSM-IV (American Psychiatric Association [APA], 1994) classifies SIB as Stereotypic Movement Disorder (formerly Stereotypy/Habit Disorder) for individuals with mental retardation, but not for

individuals with a pervasive developmental disorder (PDD). In the latter case, self-injury is thought to be better explained by the PDD diagnosis. As will become evident in the following section on the prevalence of SIB in individuals with PDD and mental retardation, the differential inclusion of one population may be empirically unwarranted.

Jones (1987) has proposed a conceptual taxonomy based on the frequency of SIB, dividing behaviours into two subgroups. The first subgroup, deemed *stereotyped SIB*, is comprised of behaviours that are repetitive, and occur with little variation and high frequency. In contrast, *self-aggressive behaviours* manifest at a much lower rate and may possess adaptive functions. While clinically relevant, there are a number of serious contentions with such a distinction. First, there is little consensus over what quantitative value differentiates 'high' from 'low' frequencies (and 'medium' frequency for that matter). Moreover, SIB frequency and severity may vary depending on particular contexts and environmental demands (Oswald et al., 1994). Thus, a behaviour that may be considered *stereotyped* may also be considered *self-aggressive* in a different context.

It seems that a conceptual taxonomy is only as valid as its empirical basis. For example, Schroeder, Mulick and Rojahn (1980) developed a useful nosology based on epidemiological data (Schroeder, Schroeder, Smith & Dalldorf, 1978) and a review of 75 studies. Findings indicated two main groups of SIB (as outlined in Table 1) which seemed to differ on the basis of whether or not the behaviour was maintained by social reinforcement. On the one hand, *social SIB* seemed to occur more often in a social setting, were directly self-injurious, and were associated with stereotyped behaviours and other behaviour problems. On the other hand, *non-social SIB* seemed to occur less frequently and were all consumatory behaviours.

Table 1: Social and Non-social Self-Injurious Behaviour Classifications (Schroeder, Mulich & Rojahn, 1980)

Social	***Non-social***
Head-banging	Stuffing orifices
Self-biting	Mouthing
Self-scratching	Sucking
Gouging	Rumination
Pinching	Copophagy (ingestion of feces)
Hair-pulling	Aerophagia (ingestion of air)
	Polydipsia (excessive fluid intake)

Prevalence

To date, very few large studies exist that investigate the prevalence of SIB specifically in autism. As mentioned, there is general consensus in the literature that

self-injury is not particular to autism (Turner, 1999). Along with displaying similar types of SIB, individuals with autism seem to have the same prevalence rates of SIB as individuals with developmental disabilities and schizophrenia (Freeman, Ritvo, Schroth, Tonick, Guthrie, & Wake, 1981). It may be that the manifestations and frequency of self-injury is related to other variables such as age, ability level, organic pathology, degree of environmental enrichment, and particular environmental contingencies.

Given that there appears to be a relationship between intellectual level and SIB, a review of the prevalence of SIB in individuals with developmental disabilities seems warranted. A quick search of the literature shows that rates vary from 2.6% to 40% (Griffin, Ricketts, Williams, Locke, Altmeyer, & Stark, 1987), and most probably depend on mediating factors as well as on sample characteristics and on the definition of SIB used. When possible, this review will focus on the mediating effect these variables play with self-injury in individuals with autism, but in the face of a lack of research, it will also highlight findings from the developmental disabilities literature. Although few large-scale studies of SIB prevalence in autism exist, one study of 314 adults with autism found that 20% displayed some form of SIB, a rate slightly higher than the average rate cited in samples with only mental retardation (Janicki & Jacobson, 1983). In young children with autism (4-5 years of age), the prevalence rate of SIB may be as high as 52%, based on parent report (Poustka & Lisch, 1993).

Residence

Whether or not a study's population sample is recruited from institutions or from the community strongly influences prevalence rates, perhaps because individuals who consistently self-injure are more likely to need constant supervision (Lakin, Hill, Hauber, Bruininks & Heal, 1983). Alternatively, institutions that fail to provide appropriate levels of enrichment may also encourage clients' problematic behaviours (APA, 1994). In a community survey of 2,663 students with developmental disabilities (including some with autism), aged between 2.3 and 19.7 years ($M = 10.2$, $SD = 4.3$), only 2.6% of individuals were reported to have self-injured. This is compared to the common findings that 10-17% of institutionalized individuals with mental retardation self-injure (Baumeister & Rollings, 1976; Schroeder et al., 1978). As expected, individuals with severe or profound developmental disabilities self-injure significantly more than higher functioning individuals, a relationship that also exists in individuals with autism (Schroeder et al., 1978).

Ability

In contrast to the paucity of large sample autism-specific research, numerous studies with smaller samples seem to suggest that self-injury is more a function of developmental level than a core dimension of the autistic syndrome. In Bartak and

Rutter's (1976) often-cited study comparing symptoms of 17 developmentally disabled children with autism to 19 intellectually average children with autism, results revealed significant differences in the manifestation of SIB. Based on parental report, approximately 71% of children with autism and intellectual impairment displayed self-injurious head banging or wrist biting, compared to only 32% of children without intellectual impairments. More recent publications add strength to this hypothesis by suggesting a negative relationship between intellectual ability and self-injury in individuals with autism. In an institutional survey of 1300 residents with developmental disabilities, 96% of clients who self-injured had IQ scores of 35 or below, based on staff report (Maisto, Baumeister & Maisto, 1978). In a sample of 61 individuals with autism, with IQ ranging from below 40 to normal and aged between 5 and 33 years, individuals with developmental disability were twice as frequent to display SIB than individuals without (Poustka & Lisch, 1993). It should be noted however that other studies have found only nonsignificant negative relationships between IQ and frequency of SIB, although this may be attributable to low sample size (Volkmar, Hoder & Cohen, 1985).

Autism and developmental disability vs. developmental disability alone

Of interest, children with autism and developmental disability seem to be more prone to SIB than children with only developmental disability. A study comparing maladaptive behaviours in 46 children with autism and mental retardation to behaviours of 128 children with only mental retardation (ages ranged from six to 14 years) revealed that 43% of the former group displayed SIB, compared to only 5% of the latter (Ando & Yoshimura, 1979). As well, groups did not differ in terms of their performance IQ, which remained below 50 for the majority of participants. It has been hypothesized that individuals with autism and mental retardation may display more SIB than individuals with mental retardation without autism due to an inability to process emotion or cope with sensory stimulation (Goodall & Corbett, 1982; Poustka & Lisch, 1993).

Age

A negative relationship may also exist between chronological age and self-injurious displays in individuals with autism and individuals with mental retardation (Maisto, Baumeister & Maisto, 1978; Schroeder et al., 1978), although conclusions are far from clear. It has been suggested that maturation, and consequently increases in adaptive skills most notably in social interaction, would lead to less frustration and consequently less SIB (Poustka & Lisch, 1993). While the aforementioned studies found a negative relationship between age and SIB, other studies investigating age and SIB in individuals with mental retardation found the opposite result (Danford & Huber, 1982; Eyman & Call, 1977). Since both the Danford and Huber (1982) and Eyman and Call (1977) studies compared adults to older adults, these contrasting

results may indicate a bimodal relationship between age and rate of self-injury instead of no relationship. For example, Danford and Huber (1982) found that 12.3% of individuals with mental retardation between the ages of 51 and 60 demonstrated an SIB, compared to 38.5% of adults aged 71 and older. To date no study has compared self-injury in individuals with autism across the lifespan, so the bimodal hypothesis remains to be empirically demonstrated.

The fact that some degree of SIB can be accounted for by age and ability highlights the importance of interpreting problematic behaviour keeping individual variables in mind. At the same time, an unequivocal partitioning of variance will only emerge once large sample sizes are used to enable the covariance of potential mediating variables such as adaptive behaviour skills, settings, symptom severity, age, and ability level. A comprehensive study to attribute the relative importance of the previously reviewed variables is therefore needed.

Etiology and treatment

There are numerous etiological theories attempting to explain SIB in autism and mental retardation. A critical review of such theories is important, since our interpretation of the cause no doubt influences our choice of treatment. While most theories are little more than untested hypotheses, behavioural and neurochemical theories have been empirically validated (Oswald et al., 1994). As such, this review will focus on behavioural and neurochemical theories and their corresponding treatments. It is important to note that attempts to explain the cause of SIB in autism have been profoundly influenced by the practical application of treatments. That is, although there is a difference between cause and treatment, at times the distinction appears ambiguous.[1]

Environmental contingencies

A behavioural interpretation of SIB assumes that an individual's behaviour is shaped by a variety of environmental contingencies. In this respect, the behavioural perspective has proven invaluable in explaining how problem behaviours are maintained and altered, as well as in decreasing self-injury in individuals with autism (Repp, Singh, Olinger & Olson, 1990). Since behaviour is readily observable, such analysis has allowed for the testable hypothesis that reinforcement contingencies play an important role in the development and maintenance of SIB.

Most notably, the functional analysis of behaviour has numerous advantages for understanding problem behaviours in individuals with autism and developmental disabilities. Functional analysis identifies the relations between environmental events and behaviour, and can thus accumulate information to describe the nature of the self-injury (Mace, Lalli & Shea, 1994). There is a strong consensus in the psychological

literature that appropriate treatment interventions, both pharmacological and behavioural, need to be based on a comprehensive functional analysis of the aberrant behaviour and its context (see Mace et al., 1994; Oswald et al. 1994; Thompson & Gray, 1994).[2]

Much of the research investigating behavioural hypotheses is based on a comprehensive review of anecdotal reports and empirical studies by Carr (1977), who summarized the existing literature into three possible motivations for SIB. First, self-injury may be an operant behaviour maintained by positive social reinforcement. In contrast, self-injury may also be motivated by negative reinforcement, in which behaviour is maintained or strengthened by the removal of an aversive stimulus. Finally, self-injury may be reinforced by sensory stimulation. The most frequently used reinforcement-based treatments for self-injury include differential reinforcement of other behaviours, and of incompatible behaviours (Repp et al., 1990).

Positive reinforcement hypothesis

The positive reinforcement explanation can be delineated into two broad classes of reinforcers: attention and increased access to desirables (Mace et al., 1994). Attention refers to social consequences of displaying self-injury, ranging from mild to severe reprimands (i.e., social disapproval), and from sympathetic concern to physical consolation. When self-injury results in increased attention, it is positively reinforced by serving to produce social interactions that may seldom occur otherwise for some individuals with developmental disabilities, given their limited adaptive behaviours and communicative repertoires (Cox & Schopler, 1993; Mace et al., 1994; Picker, Poling & Parker, 1979). Numerous case studies have strengthened the attentional hypothesis by demonstrating that problem behaviours can be diminished by withholding or removing social responses, or by removing self-injurious individuals from the social situation (i.e., time-outs) (e.g., Carr & McDowell, 1980; White, Nielson & Johnson, 1972). As well, self-injury has been noted to decrease when exposed to noncontingent social interaction, in essence undermining the need for the contingency (Burke, Burke & Forehand, 1985; Carr & Durrand, 1985; Mace & Knight, 1986). That is, self-injury fails to serve the once contingent function of increasing attention when the consequences for appropriate behaviours bring about the same result.

Self-injury may also be used to obtain desired tangibles or activities. It has been hypothesized that unresponsive environments and an inability to communicate requests appropriately may promote increasingly problematic behaviours (Carr & Durand, 1985).

Given the profound communication and social deficits inherent within the diagnosis of autism, communication through nonverbal and aberrant means seems highly

probable. As Mace and colleagues (1994) note, while ordinary behaviour may not result in obtaining desirables, and would remain a nonreinforced stimulus, occasional outbursts (i.e., extraordinary behaviour) may bring about reinforcing reactions from the environment. It has further been asserted that teaching individuals who self-injure acceptable means of obtaining desired outcomes would thus reduce the need to use aberrant behaviour (Cox & Schopler, 1993). In fact, many studies suggest that individuals with developmental disabilities who are provided with enriched environments and noncontingent access to reinforcers display lower pica and SIB rates (Favell, McGimsey & Schell, 1982; Madden, Russo & Cataldo, 1980; see also Carr, 1977).

Negative reinforcement hypothesis

According to the negative reinforcement hypothesis, SIB are used as escape or avoidance responses maintained by the delay, removal, or attenuation of an aversive stimulus (Iwata, 1987). More specifically, it has been suggested that difficult tasks (either in the classroom or in aspects of daily living) present opportunities for contingencies to emerge (Edelson, Taubman & Lovaas, 1983). In fact, a consistent finding in the literature has been that the highest rates of SIB are displayed during the most difficult task conditions (Carr, Newsom & Binkoff, 1980; Mace, Bowder & Lin, 1987; Weeks & Gaylord-Ross, 1981).

Self-stimulation hypothesis

The self-stimulation hypothesis is often proposed to account for SIB that seem without observable environmental contingencies (Mace et al., 1994). In this case self-injury is interpreted as being maintained by self-induced stimulation of the senses, and develops into both sensory and social reinforcement (Edelson, 1984). For individuals with mental retardation, SIB may be more common in environments (often institutions) with insufficient stimulation (APA, 1994). Furthermore, anecdotal reports, case studies, and neuropsychological models have long strengthened the notion that individuals with autism are characterized by a dysfunctional modulation of the sensory modalities, resulting in either hypo- or hypersensitivity to stimulation (Ornitz & Ritvo, 1976; O'Neill & Jones, 1997). Self-injury as a form of self-stimulation coincides with the idea that repetitive, stereotyped movements (e.g., body-rocking, hand-flapping) provide under-aroused individuals with stimulation (Maisto et al., 1978). In direct contrast, self-injury has also been suggested to attenuate the effects of over-arousing stimuli (Murphy, 1982).

Although clinically relevant, serious methodological limitations surround the existing experimental research that supports the sensory dysfunction hypothesis in autism, which instead often relies on questionable anecdotal (and autobiographical) reports (O'Neill & Jones, 1997). Unlike the positive and negative reinforcement hypotheses,

internal sensory experiences are not directly observable or measurable. Some studies have attempted to reduce the sensory consequences of self-injury, testing whether an interruption in the hypothesised self-stimulation may decrease the rate of SIB (Lovaas, Newsom & Hickman, 1987). Rincover and colleagues (1977; 78; 79; 82) are often cited as having successfully reduced stereotyped behaviour in children with developmental disabilities by masking auditory, visual, or proprioceptive stimulation.

Another approach, functional equivalence, has been seen as indirectly supporting the previously mentioned hypotheses (Mace, et al., 1994). By allowing individuals to replace the stimulatory effects of an SIB with those of a more adaptive behaviour, reductions in the problem behaviour have been known to occur (Favell, et al. 1982). Unfortunately, masking the sensory consequences of SIB by forcing individuals to wear helmets or hand-guards prevents exploration of the relationship between self-stimulation and SIB (Edelson, 1984).

Functional communication

Given the communication impairments that individuals with autism experience, it is not surprising that another etiological hypothesis has proposed that self-injury is a manifestation of an abnormal and impaired need to communicate. Problematic forms of behaviour that are deemed functional communication, are seen to replace socially acceptable forms of verbal and nonverbal communication that are lacking (Carr & Durand, 1985). While a traditional view may characterize SIB as purely maladaptive, functional communication interprets self-injury as behaviour that results in social and tangible reinforcers, and is adaptive (in a limited way). A similar conceptualization of self-injury has been put forth by Cox and Schopler (1993), who liken the causes of SIB to an iceberg metaphor. Problematic behaviours lie above the surface of the water, whereas self-injury is an easily observable symptom of underlying communicative deficits (as well as other types of deficits). As with functional communication, the iceberg interpretation focusses on replacing the underlying deficit with useful communicative skills.

As Mace and colleagues (1994) note, conceptualizing SIB in terms of adaptive qualities can lead to different treatment philosophies. Instead of merely attempting to decrease the problematic behaviour through direct punishment or pharmacological treatment, attempts to teach socially appropriate forms of communication become paramount. As mentioned, teaching communicative behaviours that result in access to tangibles or escape from aversive situations can replace the functional properties of the SIB, stripping the problematic behaviours of their adaptive qualities. Obviously, determining whether an SIB is replacing a form of communication depends on careful analysis of the antecedents and consequences of the behaviour. As well, the cause and maintenance of the injury are seen more as motivational states, rather than environmental contingencies.

Both the reinforcement and functional communication hypotheses are no doubt important perspectives in determining the causes of SIB. Methodologically sound research is needed however to determine their contributions more reliably and to develop appropriate and effective treatment strategies (O'Neill & Jones, 1997).

Neurochemical hypotheses

Several researchers have proposed that self-injury may be caused by disturbances in neurotransmitter function or by a dysfunction in endogenous opioid systems. While a complete review of all the various psychoactive drug therapies is beyond the scope of this paper, an overview of the most prevalent pharmacotherapies is important to suggest causal links between neurobiology and SIB.[3]

Both dopamine and serotonin have been linked to self-injurious behaviours in autism. It has been shown that lesioning the dopaminergic system, which results in a dopamine deficiency and in sensitization of the dopamine receptors, increases rates of SIB in animals (Goldstein, 1989). Most notably, self-biting was induced in monkeys after unilateral lesioning of part of the midbrain. In terms of the effect that a dysfunction in the dopaminergic system may have on SIB in individuals with autism, many double-blind studies have demonstrated that dopamine antagonists, such as haloperidol, decrease the rate of SIB in people with autism (Campbell,Anderson, Small, Perry, Greer & Caplan, 1982; Campbell, 1989). Some authors suggest however that there exists little support for a strong correlation between self-injury and dopaminergic dysfunction (Minderaa, Anderson, Volkmar, Akkerhuis & Cohen, 1989).

It may also be that that a dysfunction in the serotoninergic system in concert with dopamine sensitivity leads to self-injury. First, the fact that aggression is related to serotonin depletion has led some authors to suggest that a lack of serotonin may be related in the same way to self-aggression (i.e., SIB). Second, atypical antipsychotics such as risperidone, which are potent serotonin and dopamine antagonists, have recently been shown to reduce self-injury in adults with autism (Cohen, Ihrig, Lott, & Kerrick, 1998; Vanden Borre, et al. 1993). Finally, no empirical evidence exists to support the use of only conventional serotonin-reuptake inhibitors, suggesting that the serotonin system alone does not underlie self-injury (Rothenberger, 1993b).

Two neurochemical theories have implicated the endorphinergic system in the development and maintenance of self-injury in autism: The congenital opioid excess hypothesis and the addiction hypothesis (Oswald et al., 1994). Both hypotheses are based on the findings that increased beta-endorphin levels seem to exist in the blood-plasma of individuals with autism who self-injure, and that rates of SIB decrease upon opioid-antagonist administration (Bovier, Gaillard, Widmer, Richard & Knabe, 1992; Winchel & Stanley, 1991). The opioid excess hypothesis (i.e., the pain

hypothesis) states that increased levels of endorphins result in analgesia, most probably brought about by prolonged periods of stress and anxiety, which reduces normal sensitivity to stimulation. Self-injury is therefore used to obtain a critical level of stimulation (Singh et al., 1992). The addiction hypothesis proposes that self-injury stimulates the production of endorphins, which results in positively reinforcing feelings of euphoria. An individual with autism would therefore have to repetitively self-injure to maintain the release of endogenous opioids (Rothenberger, 1993). In terms of treatment, recent reviews have found that approximately 70% of individuals who are treated with an opioid antagonist (i.e., naltrexone or naloxone) show decreases in SIB, although results appear inconclusive for individuals with autism (Willemsen-Swinkels, Buitelaar, Nijhof & van Engeland, 1995).

In sum, no form of pharmacological treatment has been shown to be of general benefit (Rothenberger, 1993a, b). Instead, certain therapies seem to work effectively for certain individuals. Given that the leading neurochemical theories have developed and are supported by the use of psychoactive agents (i.e., very little a priori hypothesis testing), we can arrive at three conclusions given the evidence. If we assume that it is methodologically sound to develop a neurochemical theory a posteriori, then the data seem to suggest that various neurochemical dysfunctions may increase the likelihood that individuals with autism and mental retardation will self-injure. Second, large sample studies are needed to explain the relative roles that each neurotransmitter system plays in SIB. Most studies to date consist of small sample sizes (see Campbell, 1989, for an exception) and thus cannot provide strong support for any one hypothesis (Oswald et al., 1994). Finally, it is important to remember that drug studies asserting to have diminished the rate of self-injury may be merely masking or depressing the behaviour, not addressing the actual cause of the SIB.

Conclusion

To date, very little research has focussed on the self-injurious behaviours in individuals with autism. While there are striking topographic similarities in the SIB of individuals diagnosed with autism and in individuals with particular developmental disabilities associated with autistic features, researchers should be careful not to assume that self-injurious displays in these two groups arise from the same causes or motivations. Future research is needed to outline the course of self-injurious displays in autism throughout the lifespan, in order to develop appropriate treatments that are directed at correcting or even preventing the primary causes of the aberrant behaviour. While it seems obvious that a balance between pharmacotherapy and behavioural intervention is necessary, rationales for such interventions should be founded on knowledge of the primary causes of the aberrant behaviour.

Endnotes

[1] Especially in the area of psychopharmacology.

[2] For a thorough review of behavioural treatment options for SIB, see Oswald, Singh, & Singh, (1994), and Luiselli, Matson, & Singh (1992).

[3] Interested readers are referred to Singh, Singh & Ellis (1992) for a comprehensive review.

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