Available online at www.sciencedirect.com
ScienceDirect
Psychiatry Research 157 (2008) 181–189
PSYCHIATRY RESEARCH
www.elsevier.com/locate/psychres

Temporal patterns of self-injurious behavior correlate with stress hormone levels in the developmentally disabled

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Received 16 March 2006; received in revised form 20 December 2006; accepted 6 April 2007

Abstract

While the origins and developmental course of self-injurious behavior (SIB) remain relatively unknown, recent studies suggest a biological imbalance may potentiate or provoke the contagious recurrence of SIB patterns in individuals with severe developmental disabilities (DD). Evidence from several laboratories indicates that functioning, relations, and processing of a stress-related molecule, proopiomelanocortin (POMC) may be perturbed among certain subgroups of individuals exhibiting SIB. The current investigation employed a unique time-pattern analysis program (THEME) to examine whether recurrent temporal patterns (T-patterns) of SIB were related to morning levels of two POMC-derived hormones: β-endorphin (βE) and adrenocorticotropic hormone (ACTH). THEME was used to quantify highly significant (non-random) T-patterns that included SIB within a dataset of *in situ* observational recordings spanning 8 days (~40 h) in 25 subjects with DD. Pearson's product–moment analyses revealed highly significant correlations between the percentage of T-patterns containing SIB and basal levels of both βE and ACTH, which were not found with any other "control" T-patterns. These findings support the hypothesis that the recurrent temporal patterning of SIB represents a unique behavioral phenotype directly related to perturbed levels of POMC-derived stress hormones in certain individuals with severe DD.

Keywords: Self-injurious behavior; Developmental disabilities; Temporal pattern analysis; Proopiomelanocortin; ACTH; β-Endorphin

1. Introduction

Self-injurious behavior (SIB) is among the most troubling and persistent maladaptive behaviors observed in individuals with severe developmental disabilities (DD) and may be interpreted as a key manifestation of psychiatric co-morbidity. Estimations of the prevalence of SIB have been reported to be as high as 66% among institutionalized individuals with severe DD (Schroeder et al., 1978; Rojahn, 1984; Griffin et al., 1986). Although recent years have seen a dramatic increase in research on the topic, there still exists no clear consensus regarding the transitory causes or 'triggers' (social or biological) or developmental course of SIB in this population. A primary obstacle to understanding and treating SIB is the absence of reliable and effective methods for quantifying

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doi:10.1016/j.psychres.2007.04.003

the complex, recurrent patterns of SIB across varying settings, such that relations with a variety of factors may be empirically assessed. Indeed, the topographies and circumstances surrounding recurrences of SIB vary so considerably from one individual to the next that establishing a metric or analytical technique to reliably assess severity, change, or temporal contingencies is a major methodological impediment to fully understanding the nature of the disorder (Schroeder et al., 2002; Symons et al., 2005).

Sackett (1979) provided a detailed description of the application of *lag sequential analyses* to directly address the complexity and constraints of existing methods for identifying contingent relations across time in multivariate observational data. The conceptual basis for lag analyses derives from the quantitative methods of auto- and cross-correlation. When applied to qualitative behavioral data, the lag principle examines the conditional (or transitional) probability that a criterion event of interest will be sequentially followed by another event of interest (event lag), or that any observed event will fall within a specified temporal window in relation to the criterion event (time lag) (for review, see Bakeman and Gottman, 1997). Of particular relevance to the current investigation are studies that have applied this method of analysis to maladaptive or challenging behaviors such as SIB.

In a cross-validational comparison of time-based lag sequential analyses with traditional, experimental (functional) analyses, Emerson et al. (1995) found a high degree of consistency between the two approaches (86% agreement in the identification of behavioral processes underlying SIB). These results were interpreted as lending support to the external validity and overall viability of time-based lag analyses for exploring the mechanisms and contextual contingencies underlying SIB in DD populations.

In the first reported application of sequential analyses to examine whether contextual contingencies of SIB change with pharmacological treatment, Symons et al. (2001) employed an event-based lag approach to assess the sequential dependencies between SIB and antecedent staff instructional behavior during opiate-antagonist (naltrexone) and placebo administration. Though their conclusions were limited by a low number of subjects ($n=4$), they did report reductions in the rate of SIB and an increase in the sequential dependencies between staff behavior and SIB during naltrexone treatment. They suggest that, because SIB may be maintained by multiple motivating events that are both behaviorally and biologically based, treatment with naltrexone may serve to selectively reduce SIB that is opioidergically mediated, leaving socially mediated SIB unchanged, thereby increasing the relative sequential dependencies between SIB and social environment.

Recently, researchers from our project team (Marion et al., 2003) employed time-based lag sequential analyses to examine whether successive episodes of SIB were sequentially dependent. Sequential dependencies were determined by calculating the conditional probabilities that a match event followed a criterion event within four windows of time: 2, 10, 30, and 60 s. The results indicated that the only, highly significant, sequential predictor of a SIB event was another, antecedent SIB event. There was no evidence that SIB was conditional (sequentially dependent) on environmental events or on other observationally recorded behaviors within these temporal windows. Furthermore, the method of analysis controlled for chance pairings of events and revealed that the sequential patterns of SIB were independent of frequency or rate of occurrence. Additionally, the conclusion that episodes of SIB occur in sequentially related "bouts" was also confirmed using survival analyses to quantify the temporal distribution of SIB patterns (Kroeker et al., 2004). These results suggest that, within some individuals with severe DD, SIB follows "contagious" temporal distribution patterns, which may represent a unique behavioral phenotype that is maintained by biological rather than social or environmental factors.

In a related project, Sandman et al. (2002) found these contagious patterns of SIB reached highest conditional probabilities in subjects who exhibited a disregulation ("uncoupling") of the proopiomelanocortin (POMC) system, as characterized by elevated morning (basal) levels of β-endorphin (βE) relative to basal levels of adrenocorticotropic hormone (ACTH). These two hormones are POMC-derived neuropeptides that are involved in the stress response, as part of the hypothalamic–pituitary–adrenal axis (ACTH), and in the modulation of pain and pleasure because of their affinity for the opiate receptors (βE).

The purpose of the current investigation was to examine whether the relation between the POMC system and recurrent patterns of SIB could be extended by employing a fundamentally different method of analysis on our *in situ* observational data. An inherent limitation of lag sequential analyses is that the temporal windows or variables of interest must be specified *a priori*. Thus, a limiting assumption of this method is that the behavioral patterns of interest will follow a sequential distribution. The consequence of such an assumption is that it may discount the possibility that the behavior may be patterned according to non-sequential, or non-obvious, temporal distributions. Furthermore, Sackett (1979) cautions that lag sequential methods are vulnerable to "capitalization on chance", meaning that as the number of observations collected