available at www.sciencedirect.com

ScienceDirect

www.elsevier.com/locate/brainres

BRAIN RESEARCH

Research Report

Effects of biting on elevation of blood pressure and other physiological responses to stress in rats: Biting may reduce allostatic load

Suzuhito Okada[a], *Norio Hori*[b,e], *Katsuhiko Kimoto*[b,e], *Minoru Onozuka*[c,e], *Sadao Sato*[a,d,e], *Kenichi Sasaguri*[a,d,e,*]

[a]*Department of Craniofacial Growth and Development Dentistry, Kanagawa Dental College, Kanagawa, Japan*
[b]*Department of Oral and Maxillofacial Rehabilitation, Kanagawa Dental College, Kanagawa, Japan*
[c]*Department of Physiology and Neuroscience, Kanagawa Dental College, Kanagawa, Japan*
[d]*Research Institute of Occlusion Medicine, Kanagawa Dental College, Kanagawa, Japan*
[e]*Research Center of Brain and Oral Science, Kanagawa Dental College, Kanagawa, Japan*

ARTICLE INFO

Article history:
Accepted 7 September 2007
Available online 21 September 2007

Keywords:
Allostasis
Allostatic load
Biting
Blood pressure
Body temperature

ABSTRACT

We have investigated how biting modulates some of the physiological changes (blood pressure, core temperature, and chemical mediators in the serum) that are induced by restraint stress. We exposed rats to restraint stress for 60 min. Biting on a wooden stick during restraint significantly suppressed the increase of blood pressure at 30, 45, 60, and 75 min and significantly inhibited the rise in core temperature at 30, 60, 120, and 180 min compared with rats that were restrained but did not bite anything. These differences were visible in infrared thermal images of the restraint-only and restraint-with-biting rats after 60 min. Biochemical analysis revealed that biting significantly suppressed increases of plasma interleukin-1β, interleukin-6, and leptin and that it significantly suppressed a decrease of thyroid-stimulating hormone. These observations suggest that biting produces an anti-stress effect and that para-functional masticatory activity plays an important role in coping with stressful events.

1. Introduction

Psychological stress contributes to the pathophysiology of many inflammatory, autoimmune, and allergic diseases (Eskandari and Sternbergm, 2002). The perception of stress by an individual activates the CNS with two major consequences: (1) the neuroendocrine system responds via the HPA axis and releases adrenal steroid hormones, such as corticosterone, and (2) the autonomic nervous system responds, causing sympathetic nerve termini to locally release catecholamines, especially norepinephrine (Elenkov et al., 2000; Webster et al., 2002).

* *Corresponding author*. 82 Inaoka-cho, Yokosuka 238-8580, Japan. Fax: +81 46 822 8858.
E-mail address: sasaguri@kdcnet.ac.jp (K. Sasaguri).

Abbreviations: HPA, hypothalamic-pituitary-adrenal; ANOVA, analysis of variance; SEM, standard error of the mean; CRH, corticotropin-releasing hormone; IL, interleukin; TNF-α, tumor necrosis factor-α; CNS, central nervous system; GRO/CINC-1, chemokine growth-regulated oncogene/cytokine-induced neutrophil chemoattractant-1; GH, growth hormone; TSH, thyroid stimulating hormone; PLSD, Protected Least Significant Difference; $p<0.05$, probabilities of <5%; nNOS, neuronal nitric oxide synthase; ERK 1/2, extracellular signal-regulated protein kinase 1/2; RIA, radioimmunoassay; C, Celsius; RX, restraint-without biting; RB, restraint-with biting

0006-8993/$ – see front matter © 2007 Elsevier B.V. All rights reserved.
doi:10.1016/j.brainres.2007.09.030

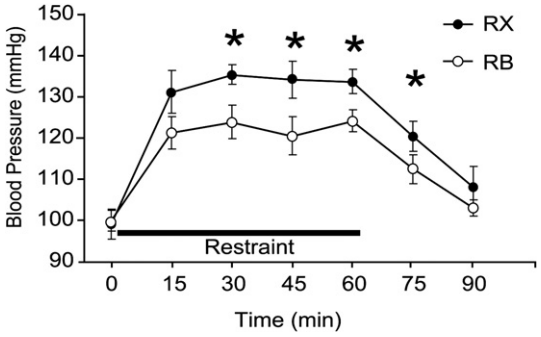

Fig. 1 – The effects of biting on blood pressure rise caused by restraint stress. Black horizontal bar on the abscissa indicates restraint period. Data are expressed as means ($n=10$) ± SEM.

Allostasis is the process that keeps an organism alive and functioning (McEwen and Stellar, 1993), i.e., maintaining homeostasis or "maintaining stability through change" and promoting adaptation and coping, at least in the short run (McEwen, 1998, 2000; McEwen and Seeman, 1999). Each of these adaptive processes has a potential cost to the body when allostasis is either called upon too often or is inefficiently managed; that cost is referred to as "allostatic load."

One of the regulatory systems that may be overworked or inefficiently managed, thereby increasing the organism's allostatic load, is that which controls core temperature, which for humans follows a diurnal cycle centered at 37 °C. Core temperature reaches a minimum during periods of sleep. Recent studies with laboratory animals have shown that psychological stress causes core temperature to increase (Briese, 1995; Marazziti et al., 1992). Endogenous pyrogens, a group that includes IL-1α, IL-1β, IL-6, TNF-α, interferon-α, macrophage inflammatory protein-1, and other substances known as proinflammatory cytokines (Oka et al., 2001), play important roles in the thermoregulatory system.

Stress induced by immobilization (restraint stress) is particularly effective because it combines both physical stress and emotional stress (de Paula Brotto, 2003). It increases blood pressure largely because it activates the sympathoadrenal system (Irving et al., 1997). Para-masticatory activities, such as tooth-clenching, nail-biting, and biting itself, have been shown to influence the general function of the CNS (Nakata, 1998).

Several recent studies of the effect of restraint stress on animals suggest that biting suppresses the stress response, thereby decreasing the allostatic load along with its harmful effects on the organism (Vincent et al., 1984; Tsuda et al., 1988; Tanaka et al., 1998; Gomez et al., 1999; Hori et al., 2004, 2005; Sasaguri et al., 2005; Miyake et al., 2005). In this study, we investigated the effects of biting on three specific manifestations of the allostatic load accumulated during restraint-stress tests: elevated blood pressure, elevated core temperature, and the secretion of chemical mediators into the blood.

2. Results

2.1. *Experiment 1: effect of restraint time and biting on blood pressure*

For both groups RX (restraint-without-biting) and RB (restraint-with-biting), the mean systolic blood pressure rose as a function of restraint time (Fig. 1). At 30, 45, 60, and 75 min, the mean systolic blood pressure of animals in group RB was significantly lower ($^*p<0.05$) than that of animals in group RX. There was no difference between the means of these two groups at 0, 15, and 90 min.

2.2. *Experiment 2: effects of restraint time and biting on core temperature and thermal image*

Subjecting the rats to stress caused their core temperatures to gradually rise and their thermal images to gradually intensify (Fig. 2). At 30, 60, 120, and 180 min, core temperature was significantly lower ($^*p<0.05$) in the RB group than in the RX group (Fig. 2A). There was no difference between these two groups at −30, 0, and 240 min.

Thermal infrared images showed that the mean surface temperature of rats after restraint (Fig. 2B) was higher than that of rats before restraint, both those in the RX group and in the RB group (Fig. 2D). At the beginning of restraint, there was no difference between the mean abdominal surface temperatures of the two groups (Fig. 2C). After 60 min, however, the mean surface temperature of the RB group was significantly lower than that of the RX group (Fig. 2D). In all cases, body temperature had returned to the basal temperature level by 240 min after the end of the 60-min period of restraint stress (Fig. 2E).

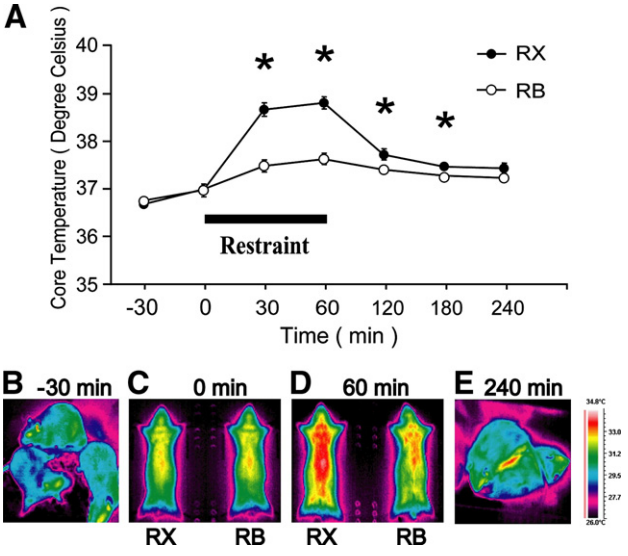

Fig. 2 – The effects of biting on core temperature rise caused by restraint stress. Data are expressed as means ($n=10$) ± SEM (A). Thermal infrared images were compared to visualize the changes in the surface temperature in rats (B–E). In panels C and D, the rat on the left was from group RX and the rat on the right was from group RB. Images of rats before restraint are shown.