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EFFECTS OF EXAM STRESS ON MOOD, CORTISOL, AND IMMUNE FUNCTIONING: INFLUENCES OF NEUROTICISM AND SMOKER–NON-SMOKER STATUS

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Summary—In a number of studies, neuroticism, depression and stress have been reported to be positively correlated with each other, with serum cortisol concentration and with smoking. The same factors are inversely related to measures of immune system functioning. The present study assessed in smokers and non-smokers the effects of the presumed stress of final examinations on moods, cortisol and immune system functioning. Subjects were college students selected because they reported feeling reliable high degrees of stress during examinations. Immune system functioning (natural killer cell cytotoxic activity [NKCA], and ConA and PHA lymphocyte proliferation), serum cortisol concentration and mood were assessed in 19 smokers and 23 non-smokers. The findings indicate that exam stress was associated with large increases in reported tension and slightly increased symptoms of depression. Further, T lymphocyte proliferation in response to ConA, but not to PHA, was suppressed during the exam period, while changes in NKCA during exams were associated with an interaction of smoker status and neuroticism. There was also a neuroticism by stress interaction for negative mood assessed by the Profile of Mood States such that those individuals who scored high on measures of neuroticism were higher in negative affect at baseline and post-exam periods, but not during the exam period. Smokers had higher serum cortisol concentrations than non-smokers across conditions and scored higher in Beck Depression Inventory-assessed symptoms of depression. Cortisol did not vary as a function of stress and was not correlated with changes in immune functioning, with depression or with negative moods. Serum cortisol and beta-endorphin concentrations were not associated with immune functioning or habitual nicotine intake (plasma nicotine and cotinine concentrations). Among smokers, exam stress did not result in elevated plasma nicotine, cotinine or caffeine concentrations. Copyright © 1996 Elsevier Science Ltd.

INTRODUCTION

Evidence suggests that immune system functioning and health are influenced by stress, personality and smoking. However, few studies have controlled for or assessed how stress, personality and smoking interact with each other and other behaviors that may modulate immune functioning and health.

High levels of neuroticism, depression, stress, blood cortisol concentration, a variety of diseases and smoking all tend to be positively correlated with each other (Eysenck, 1991; Gilbert, 1995), while being inversely related to immune system functioning (Geiser, 1989; Herber & Cohen, 1993; Munck & Guyre, 1991). This pattern of findings suggests that elevated blood cortisol concentration may be a common mediator of some of the effects of smoking, personality and stress on immune functioning and disease (Eysenck, 1991; Herbert & Cohen, 1993). Consistent with the possibility that nicotine might increase blood cortisol concentrations that, in turn, might suppress immune functioning, Caggiula, McAllister, Epstein, Antelman, Knopf, Saylor and Perkins (1992) found that high doses of nicotine, acutely administered to rats, elevated plasma corticosterone concentrations and also suppressed immune function.

Over the past decade a substantial body of research evidence has accumulated indicating that both clinical depression and psychological stress compromise immunologic function, possibly influencing an individual's susceptibility to illness and/or altering the course of a disease state (Herbert & Cohen, 1993; O'Leary, 1990; Stein, Miller & Trestman, 1991). Studies such as those of Bartrop, Luckhurst, Lazarus, Kiloh and Penny (1977), Keicolt-Glaser, Ricker, Messick, Speicher, Garner

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and Glaser (1984), and Schleifer, Keller, Meyerson, Raskin, Davis and Stein (1984) indicate that a variety of psychological stressors and emotional states are capable of modulating several measures of immune system functioning. Lymphocyte proliferation in response to phytohemagglutinin (PHA) or conconavalin A (Con A) and changes in natural killer cell cytotoxic activity (NKCA) are frequently used in such studies as operational measures of immune system functioning. Information about the potential effects of smoking, depression-related personality traits and stress on natural killer cell cytotoxic activity (NKCA) would likely be of importance since NKCA suppression has been found in neoplastic diseases (Brenner, Benarrosh & Margolese, 1986). Further, attenuated NKCA may tend to promote the development and spread of malignancies (Barlozzari, Leonhardt, Wiltrout, Heberman & Reynolds, 1985).

A meta-analytic review of the relationship between psychological depression and measures of immune system functioning (Herbert & Cohen, 1993, p. 472) concluded that when "analyzing only methodologically sound studies, reliable immune alterations included lowered proliferative response of lymphocytes to mitogens ... [and] lowered natural killer cell activity ...". However, not all studies report alterations of immune functioning during psychological stress and depression, and it is not clear what differentiates studies showing such effects from those failing to do so (Cover & Irwin, 1994; Stein *et al.*, 1991). Since clinical depression (Stokes & Sikes, 1987) and stress (Henry, 1986) are frequently associated with elevated blood cortisol concentrations, cortisol is considered to be one possible mediator of the associations of depression and stress with lowered immune system function.

In a number of studies cigarette smoking has been reported to alter (most often suppressing) aspects of immune system functioning (Marcy & Merrill, 1987; Mili, Flanders, Boring, Annest & Destefano, 1991; Newman, Kreiss & Campbell, 1991). For example, Phillips, Marshall, Brown and Thompson (1985) reported that light-to-moderate smokers showed NKCA comparable to that of non-smokers whereas heavy smokers showed a significant depression of NKCA. However, findings in this area have not been consistent. For example, Newman *et al.* (1991) found enhanced NKCA in smokers relative to non-smokers and ex-smokers, while Irwin, Patterson, Smith, Caldwell, Brown, Gillin and Grant (1990) found clinical depression and threatening life events, but not smoking to be associated with reductions in NKCA. There are indications that smokers, compared with non-smokers, have reduced functioning of certain immunologic responses (Bahna, Heiner & Myhre, 1980; Miller, Goldstein, Murphy & Ginns, 1982; Petersen, Steimel & Callaghan, 1983; Phillips *et al.*, 1985), but most of this research did not control for the influence of factors such as neuroticism (N) and high levels of caffeine intake that might be common to smokers but not directly related to smoking (Eysenck, 1991).

While cigarette smokers report that they smoke more when stressed or feeling emotional and that smoking helps them reduce stress and negative moods (Spielberger, 1986), the experimental literature indicates that whether or not nicotine attenuates negative affect is dependent on situational and individual-difference factors (reviewed by Gilbert, 1995). Experimental evidence also supports the view that nicotine has stimulant effects that can enhance vigilance and other forms of cognitive performance that can be useful in actively coping with many stressors (Levin, 1994; Sherwood, 1994; Warburton, 1992). Caffeine consumption is higher in smokers than non-smokers (Istvan & Matarazzo, 1984). Caffeine, like nicotine, increases vigilance performance (Silverman, Mumford & Griffiths, 1994) and is used to cope with tasks requiring sustained attention.

Relationships between psychological stress, N, smoking, and immune system functioning are complex, as seen by findings suggesting that smoking intake in some cases increases with stress (Rose, Ananda & Jarvik, 1983), while subjective stress and elevated cortisol concentrations are frequently associated with high scores on measures of N (Eysenck & Eysenck, 1985). Earlier studies of the effects of examination stress on immunocompetence (e.g. Kiecolt-Glaser *et al.*, 1984) did not assess the degree to which exam-stress-induced changes in immunocompetence are influenced by smoking or by individual differences in N, the emotional trait most frequently associated with clinical depression and related negative affect. Finally, smoking results in elevated serum cortisol concentrations (Gilbert, Meliska, Williams & Jensen, 1992; Kirshbaumm, Wurst & Stasburger, 1992; Meliska & Gilbert, 1991), but these effects have been found to vary as a function of N and depression (Gilbert, Meliska, Welser & Estes, 1994).

The overall goal of the present study was to characterize individual differences in immune