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USING BIOLOGICAL MARKERS TO MEASURE STRESS IN LISTENERS OF COMMERCIAL TALK RADIO

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EXECUTIVE SUMMARY

This pilot study established that a methodology that employs biological markers can be useful in determining the physiological and psychological effects of hate speech. The findings show a statistically significant correlation between changes in clinical anxiety and the production of salivary cortisol in listeners who were exposed to hate speech content on commercial talk radio. The research reveals that hate speech could potentially have a deleterious impact on the health of listeners, whether or not they are ideologically aligned with the host.

I. INTRODUCTION

This pilot study is the last in a three-part series conducted by the Chicano Studies Research Center (CSRC) at the University of California, Los Angeles, that uses scientific methodologies to investigate the characteristics and impact of hate speech that is broadcast on commercial talk radio. The first study used qualitative content analysis to examine hate speech in commercial broadcasting that targets vulnerable groups—that is, ethnic, racial, religious, and/or sexual minorities (Noriega and Iribarren 2011). This research revealed a discursive pattern in which talk show hosts employed unsubstantiated claims and other linguistic strategies to directly or indirectly identify vulnerable groups, or those identified in alignment with them, as a threat to their primary radio audience, which likely comprises listeners who are ideologically aligned with the hosts. The significance of ideological alignment was investigated in a second study, which analyzed the social networks that form around the hosts of talk radio shows (Noriega and Iribarren 2012). The findings revealed that the hosts promoted an insular discourse that focused on, for example, anti-immigration, anti-Islam, and pro-Tea Party positions and that this discourse found repetition and amplification through social media.

But what about the impact of hate speech on listeners? For this study, we hypothesized that by defining and tracking the biological markers of stress, we could determine the effect of hate speech broadcast on commercial talk radio or other media. Stress can be described as physical or emotional influences that disturb the homeostatic balance of the body, resulting in psychological and physiological changes (Muir and Pfister 1989). Stress increases activation of the hypothalamic-pituitary-adrenocortical axis (HPAA) (Fisher 1989), a complex set of interactions that control many of the body's processes. One way to assess the impact of stress is to track steroid hormones and cytokines, immunological regulators that are produced by the HPAA in response to a stressful situation. These regulators are present in saliva, where they can be measured by noninvasive means (Arellano-Garcia et al. 2008; Vining et al. 1983; Vining, McGinley, and Symons 1983).

Cytokine regulation may play a role in disease onset and outcomes such as cancer and other inflammatory diseases associated with chronic stress. Among the cytokines that have been linked to stress reactions are T-lymphocyte Helper-Type 1 and 2 (Th1 and Th2) cytokines, which are known to help regulate lymphocyte proliferation and cytotoxicity.[1] Decreases in Th1 (inflammatory) cytokines such as IFN-γ and IL-2 and increases in some Th2 (anti-inflammatory) cytokines such as IL-4 and IL-5 have been found in students undergoing examination stress (Kang and Fox 2001). Exposure to chronic stress can lead to a chronically elevated release of cortisol, a steroid hormone, which in turn may promote a shift from a Th1 to a Th2 cytokine response (DeRijk et al. 1997; DeRijk and Sternberg 1997; Elenkov and Chrousos 1999a, 1999b).

To evaluate changes associated with exposure to hate speech on commercial talk radio, we examined saliva samples collected from volunteer participants before and after they listened to a broadcast segment from a commercial talk radio show that contained a particularly dense amount of language targeting vulnerable groups. Salivary cortisol, testosterone, and a selected set of Th1 and Th2 cytokines were analyzed and correlated against clinical anxiety parameters obtained from the same subjects before and after the experimental intervention.

II. METHODOLOGY

PARTICIPANTS

Volunteers were recruited via advertisement (flyers) and word of mouth. All subjects participated on a voluntary basis. Candidates were screened to identify and exclude those without fluency in English, those with apparent hearing difficulties, and those with a recent history of oral inflammatory disease. Thirteen men aged twenty-nine to fifty participated in the study. The Institutional Review Board Committee of the Los Angeles BioMedical Research Institute at Harbor-UCLA Medical Center approved the investigation (RS-0200880100). Participants were fully informed of the purpose and risks of participating in the study, and they signed informed consent documents prior to testing. Participants were also familiarized with the sampling and survey procedures prior to testing.

SELF-REPORT MEASURES

Participants first completed a questionnaire that collected demographic data. They then completed the clinical anxiety scale (CAS) (Westhuis and Thyer 1989), an instrument designed to rapidly measure an individual's anxiety level. The CAS comprises a

short series of questions to which participants respond using a quintile-based scale (1 = Not at all; 2 = A little bit; 3 = Somewhat; 4 = Very much; 5 = Extremely). The test was repeated after the experimental intervention, and the two sets of scores were compared.

SALIVA SAMPLES

Two saliva samples were collected: the first after the initial data collection and the first administration of the CAS, which provided a baseline, and the second after the experimental intervention and the second administration of the CAS. Participants were instructed to abstain from eating, drinking, smoking, or brushing their teeth for one hour before testing. Saliva samples were collected using oral synthetic swabs (Salivette®-Cortisol). Samples were chilled immediately following collection and then frozen within one hour and held at –20°C until assay. Samples were assayed in duplicate using competitive enzyme immunoassays for testosterone and cortisol levels. A multiplexed bead immunoassay was used to measure levels of two Th1 cytokines (IFN-γ and IL-2) and three Th2 cytokines (IL-4, IL-5, and IL-10).

EXPERIMENTAL INTERVENTION

The broadcast segment selected for the experimental intervention was drawn from the commercial talk radio programs analyzed in the CSRC's pilot study (Noriega and Iribarren 2011). Researchers chose a segment that was particularly dense in indexical terms targeting vulnerable groups. The segment, from *The Savage Nation*, aired on July 24, 2008. Its length, including music and advertisements, was 23 minutes and 30 seconds.

STATISTICAL ANALYSIS

Data were summarized and expressed as a mean and its standard error (SEM). One-way ANOVA was used to compare variances within and among groups. Bartlett's test was used to establish the homogeneity of variance. Whenever needed, post hoc unpaired multiple comparison tests (Bonferroni correction) and Student's t-test were used for comparison between two groups. Findings greater or equal to 0.05 were considered significant. The Pearson correlation coefficient was calculated and is represented by R^2 values.

Table 1. Selected Demographic Characteristics of Study Sample

	Sampled population (*n*=13)
Mean Age (SEM/SD)	37 (1.8/6.7)
Ethnicity	
Caucasian/White	5
Hispanic/Latino	6
African American	1
Asian American	1
Political Views	
Very Liberal	7
Somewhat Liberal	4
Somewhat Conservative	2
English as First Language	
Yes	7
No	6
Born in the United States (Participant)	
Yes	8
No	5
Born in the United States (One or More Parents)	
Yes	7
No	6

III. RESULTS AND DISCUSSION

DEMOGRAPHICS

The study sample was composed of 13 men with a mean age of thirty-seven. Six of the participants identified themselves as Hispanic or Latino, and 7 reported that their primary language was English. Seven of the 13 considered themselves very liberal. In terms of nativity, 8 of the participants reported that they were born in the United States and 5 were born elsewhere. Seven participants stated that one or both parents were born in the United States, and 6 were born elsewhere (table 1). None of the demographic parameters showed a significant statistical correlation with any of the studied variables—anxiety, cortisol, testosterone, cytokines—or their variations.

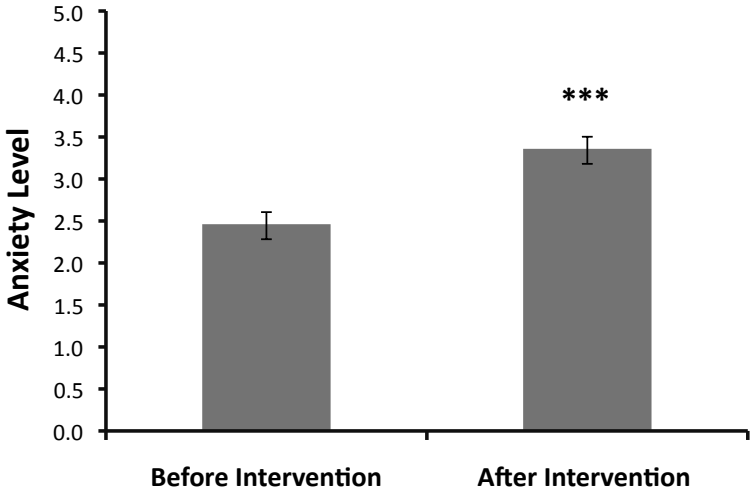
ANXIETY ASSESSMENT

Comparison of the CAS data collected before and after exposure to the broadcast segment showed a significant increase in anxiety, jumping from a mean of 2.5 (± 0.1) to 3.4 (± 0.2). The participants went from being a little or somewhat anxious to somewhat or very anxious (fig. 1).

STEROID HORMONE ASSESSMENT

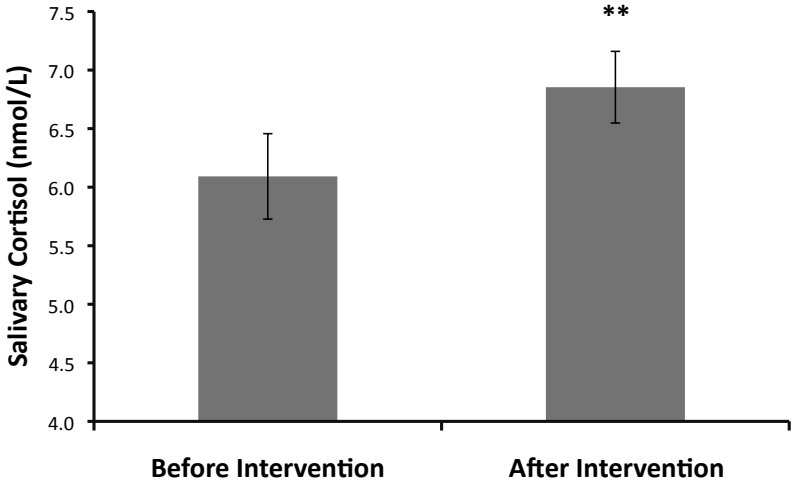
Study participants experienced a moderate increase in cortisol after the intervention, rising from a mean of 6.09 (± 0.36) to 6.85 (± 0.31) (fig. 2). Changes in testosterone were not significant, at 140.85 (± 9.17) compared to 138.02 (± 7.54) (fig. 3). Testosterone is a hormone associated with aggression.

Figure 1. Change in Anxiety Levels after Intervention



Note: *** = p <0.01. Assessed using a five-point Clinical Anxiety Scale, where 1 = "not at all"; 2 = "a little bit"; 3 = "somewhat"; 4 = "very much"; 5 = "extremely."

Figure 2. Change in Salivary Cortisol Levels



Note: ** = p <0.05

Figure 3. Change in Salivary Testosterone Levels

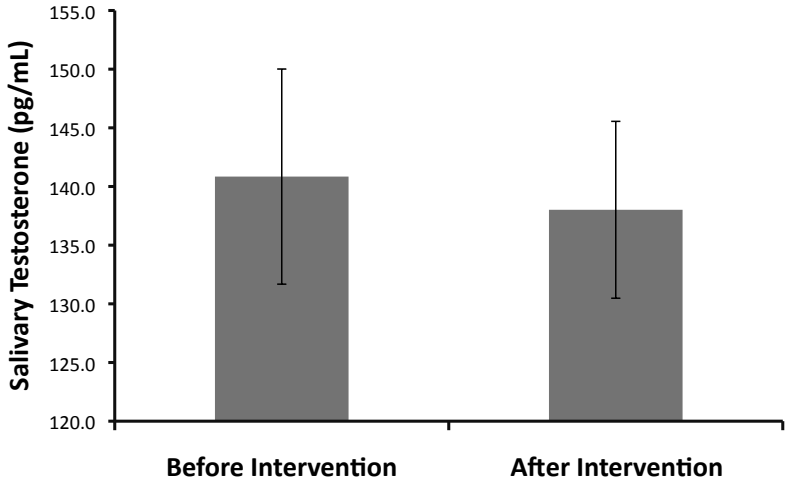


Figure 4. Individual Salivary Cortisol Measurements

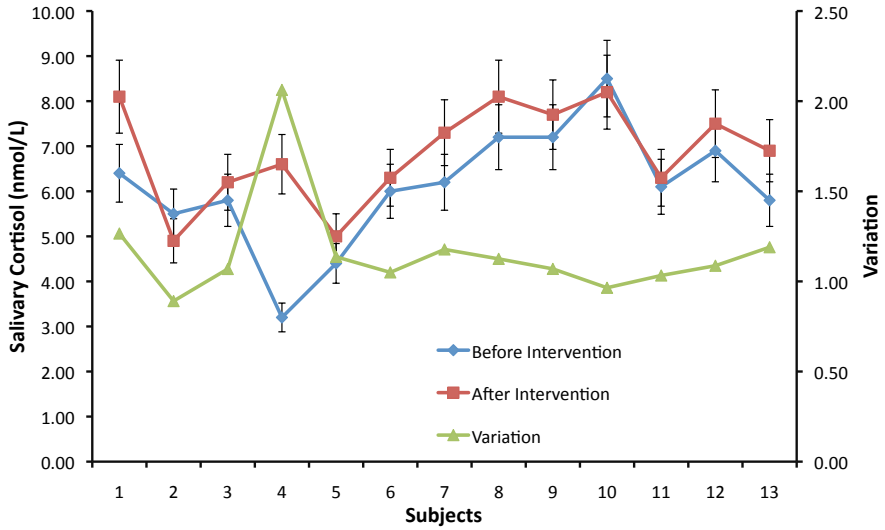


Figure 5. Individual Salivary Testosterone Measurements

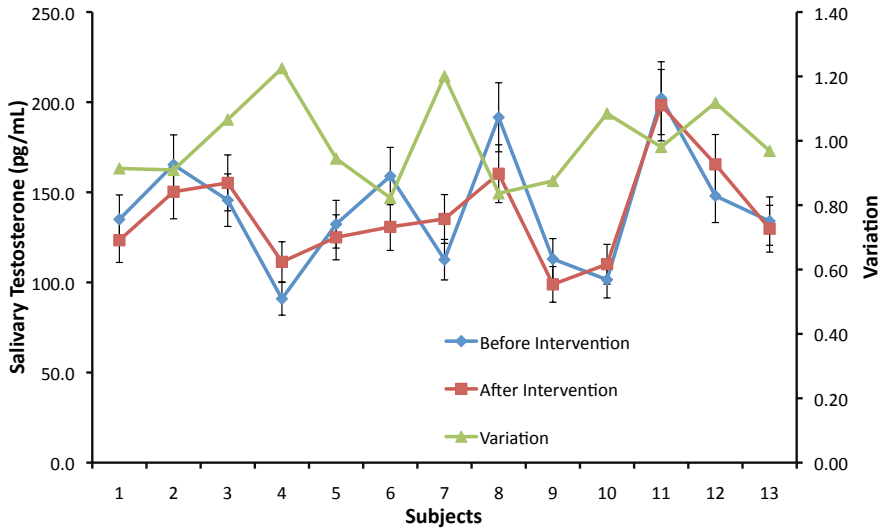
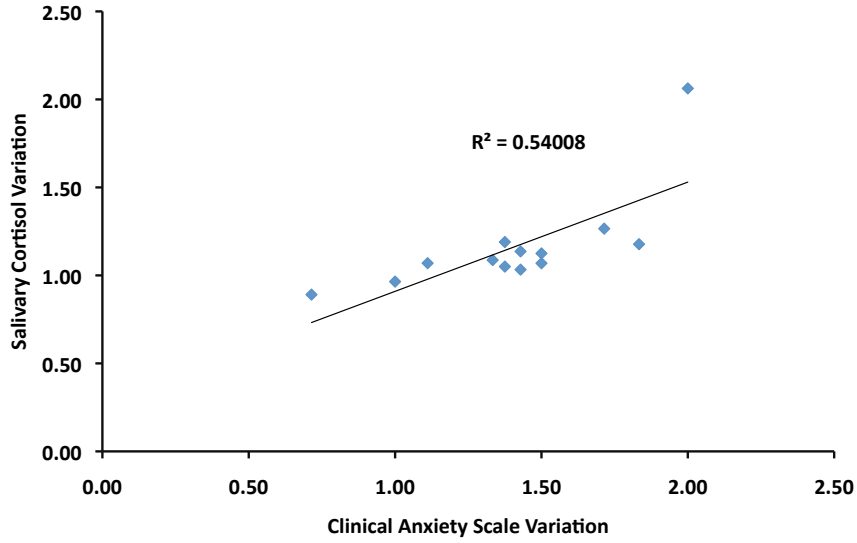


Figure 6. Correlation Between Salivary Cortisol Variations and Anxiety Variations



Changes in cortisol and testosterone showed wide variation among individuals and between sampling periods. Individuals with lower baseline cortisol showed greater reactivity than those with higher baseline cortisol (figs. 4, 5). This pattern may suggest that some individuals were preconditioned to respond to stress stimuli, which affected the production of cortisol during the intervention.

Although a discrete tendency to a positive correlation between changes in cortisol production and clinical anxiety was observed (R^2=0.54), no significant correlation between testosterone and clinical anxiety was found (R^2=0.21) (figs. 6, 7). In addition, no significant correlation was observed between cortisol and testosterone (R^2=0.26).

CYTOKINE ASSESSMENT

Exposure to the broadcast segment significantly increased one of the Th2 cytokines, IL-10, to 1.29 (± 0.15) (fig. 8). Although the correlation between IFN-γ, a Th1 cytokine, and cortisol was not statistically significant (R^2=0.33) (fig. 9), its slope suggests an inverse correlation that corresponds with the direct correlation observed for IL-10 and cortisol (R^2=0.03) (fig. 10). This finding suggests that cortisol influences the regulation of Th2 cytokines. The correlation analysis between IL-10 and CAS yielded a better fit, although it was not statistically significant (R^2=0.33). These results suggest that there is a moderate correlation between stress and the regulation of Th2 cytokines that might contribute to pathophysiological processes or the development of some diseases (fig. 11).

Figure 7. Correlation Between Salivary Testosterone Variations and Anxiety Variations

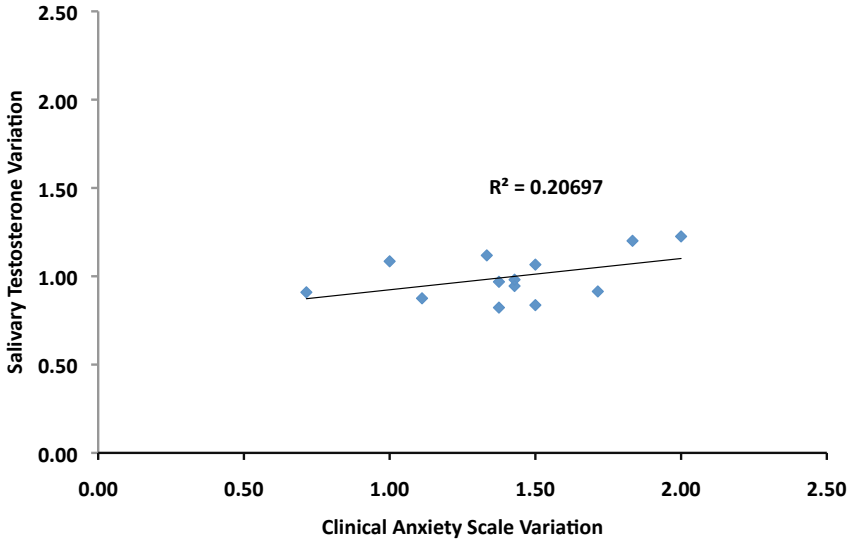
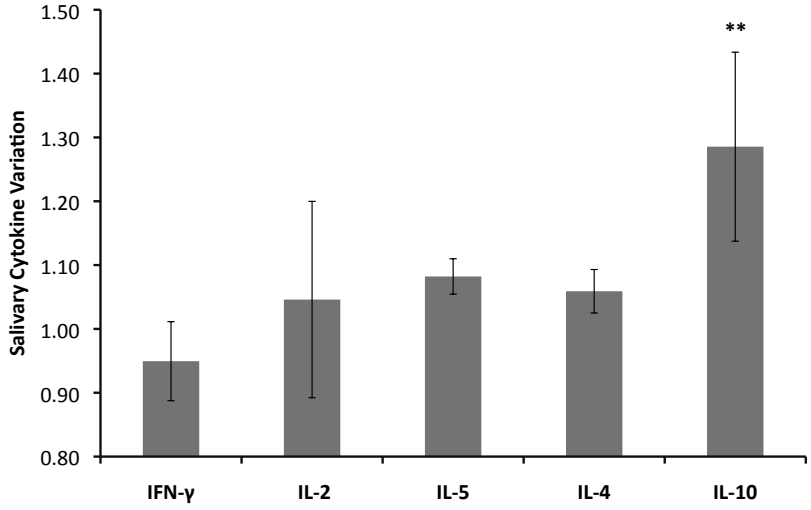


Figure 8. Salivary Cytokine Variations



Note: ** = p <0.05

Figure 9. Correlation Between Salivary IFN-γ Variations and Salivary Cortisol Variations

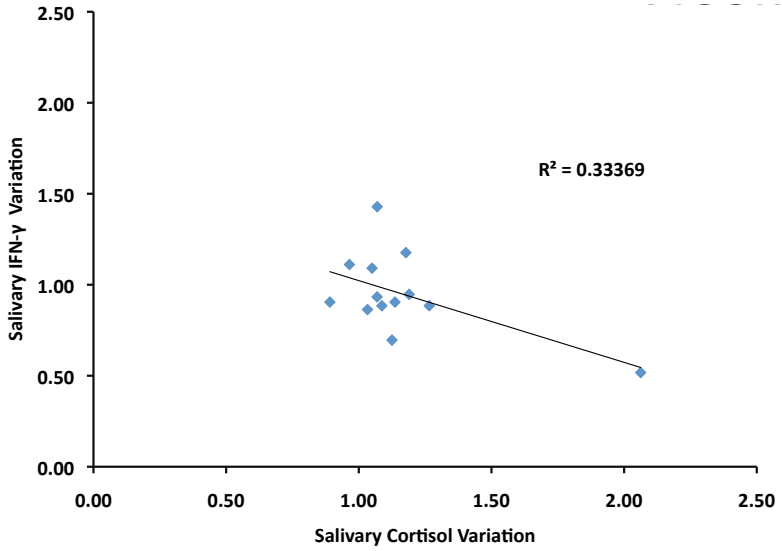


Figure 10. Correlation Between Salivary IL-10 Variations and Salivary Cortisol Variations

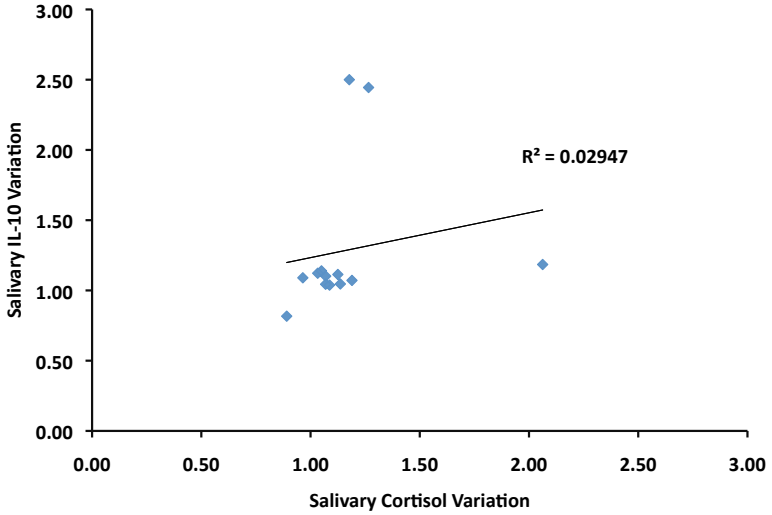
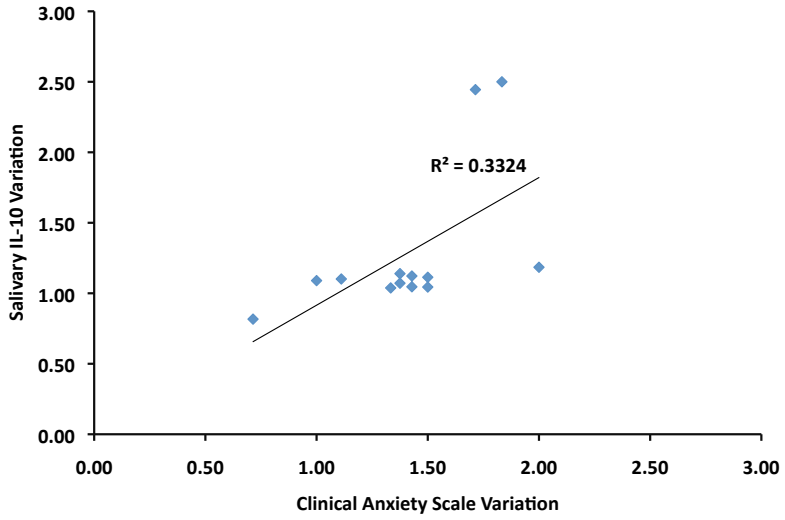


Figure 11. Correlation Between Salivary IL-10 Variations and Salivary Cortisol Variations



IV. CONCLUSION AND RECOMMENDATIONS

In this pilot study we investigated the physiological and psychological effect of commercial talk radio on listeners by assessing changes in biomarkers of stress. Our findings demonstrated a statistically significant correlation between changes in clinical anxiety and the production of salivary cortisol. Although we discovered no significant correlations between the other biomarkers studied, we did find a tendency toward a direct correlation between cortisol and Th2 cytokines, with an inverse correlation between cortisol and Th1 cytokines. This suggests that exposure to selected hate speech content on commercial talk radio programs could potentially influence the onset or development of pathophysiological processes or diseases such as cancer or chronic inflammatory diseases (DeRijk et al. 1997; DeRijk and Sternberg 1997; Elenkov and Chrousos 1999a, 1999b). Lack of significant statistical correlations between the demographic characteristics of the studied population and the experimental variables examined suggests that the correlations and trends that were observed in the data may represent a general effect that is not specific to race/ethnicity, nativity, or ideological alignment with talk radio programs. If further study confirms these findings, the implications are significant with respect to the physiological impact of hate speech on both vulnerable groups and those targeting them.

The pilot study established that the methodology described in this report can be useful in future research on the effects of hate speech. More work needs to be undertaken to determine the correlation between hate speech and the biomarkers studied. Refinements to the research design would include increasing the sample size to enhance the methodology's sensitivity and adding a control group that would be exposed to broadcast content in which vulnerable groups are discussed in comparatively positive terms—for example, with language that promotes their inclusion into U.S. society. Such a comparative approach could shed much-needed light on the effects of the messages generated by mass media.

NOTE

1. Th1 cytokines IFN-γ and IL-2 play a major role in upregulating cytotoxicity and have been associated with a pro-inflammatory response and low levels of cortisol conferring immunological surveillance. Th2 cytokines (IL-4, IL-5, and IL-10) may antagonize these Th1 effects (Antoni et al. 2009). Th2 cytokines such as IL-4 can antagonize the actions of IL-2 and IFN-γ on cell-mediated immune functions such as lymphocyte proliferation and cytotoxicity (Roussel et al. 1996; Saito et al 1996). Several studies have identified a predominance of Th2 cytokines over Th1 cytokines at tumor sites (Elsasser-Beile, von Kleist, and Martin 1992) and have shown suppressed production of IL-2 and IFN-γ across various cancer populations (Elsasser-Beile, von Kleist, and Martin 1992; Fischer et al. 1997).

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