Stressed Power Motivation, Sympathetic Activation, Immune Function, and Illness

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Previous research has reported that individuals high in the need for Power, high in inhibition, and high in power stress (the HHH group) are more likely than other individuals to report more severe illnesses. The present study investigates the possibility that the mechanism underlying this relationship is greater sympathetic activation in the HHH group which has an immunosuppressive effect. College males with the HHH syndrome reported more frequent and more severe illnesses than other individuals, as in previous studies. More of the HHH than other subjects also showed above average epinephrine excretion rates in urine and below average concentrations of immunoglobulin A in saliva (S-IgA). Furthermore, higher rates of epinephrine excretion were significantly associated with lower S-IgA concentrations, and lower S-IgA concentrations were significantly associated with reports of more frequent illnesses. The findings are interpreted as consistent with the hypothesis that a strong need for Power, if it is inhibited and stressed, leads to chronic sympathetic overactivity which has an immunosuppressive effect making individuals characterized by this syndrome more susceptible to illness.

Several recent studies have reported that individuals with a high need for Power, if it is also inhibited, are more likely to become ill. As compared with individuals low in need for Power and low in inhibition they are more likely to develop high blood pressure over time. They also report more affective symptoms, ranging from headaches to depression,[N1] as well as physical illnesses of greater severity.[N2] To seek out the mechanisms underlying these relationships, it is necessary to understand the nature of the need for Power (*n* Power), a personality disposition of major importance which has been the subject of empirical study by psychologists for over 20 years.[2-4] In all of this work *n* Power is measured by coding thought content objectively to avoid response biases which distort self-reports of motivation.[5] Individuals high in *n* Power write imaginative stories about having impact on others by aggression, persuasion, helping others or by arousing emotional responses in others.[3] Men who score high in *n* Power, as compared with those low in *n* Power, are more argumentative in real life, play more competitive sports, accumulate more prestige symbols such as credit cards, and try to act in ways that enhance their

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impact. An important variable which moderates the expression of *n* Power in action is "activity inhibition," also measured in thought content by the frequency with which the individual thinks of inhibiting action by the use of the word "not." Men high in *n* Power and low in inhibition drink more alcohol,[6] lie more, and seek more sexual conquests than other men.[4] In other words, they are impulsive and assertive in actions, but if they are high in inhibition men with a strong need for Power drink less than other men.[6] They also make successful managers[7] since they seek to influence others in a controlled way. In many respects they behave like people who have been characterized as showing Type A behavior[8]—they are assertive and hard driving but also inhibited in direct expression of aggression.

Therefore it was hypothesized that men high in *n* Power and in inhibition might share the cardiovascular problems which characterize Type A individuals. This is indeed the case. Men who were characterized by the inhibited power motive syndrome in their early thirties were much more likely to have high blood pressure 20 years later than men with other motivational syndromes.[1] A possible explanation for this relationship lies in the greater activation of the sympathetic nervous system, which characterizes Type A individuals and which could lead to chronically elevated blood pressures.[8] Further support for this possibility is provided by a study [N3] which showed that increases in power thought content of the type included in the *n* Power score are significantly associated with signs of increased sympathetic nervous system activity—namely, with increases in epinephrine and norepinephrine excreted in urine.

Increased sympathetic activity may have an immunosuppressive effect through release of corticosteroids[9] or epinephrine.[10-12] Therefore the mechanism underlying the relationship between the inhibited power motive syndrome and illness might involve increased sympathetic activity which may impair immune function and make the individual more susceptible to infection. The present study is designed to investigate this possibility directly by obtaining measures of catecholamine excretion levels, of immune function, and of illness in individuals with and without the inhibited power motive syndrome. A measure of life stress related to the power motive will also be included, since prior research has demonstrated that individuals with the inhibited power motive syndrome who are also stressed report more frequent and more severe illnesses than any other group. [N1, N2]

PROCEDURE

The subjects for the experiment were 27 male college sophomores who had taken a version of the picture-story test used to assess motives about a year previously as part of a general testing session for freshmen. Only males were used, to avoid possible complications introduced by sex differences, since Frankenhaeuser reports sex differences in catecholamine excretion to emotional situations.[13] Subjects were given five minutes to write an imaginative story for each of the following pictures: a ship captain explaining something to somebody; a man and a woman at a table in a nightclub; a man at a drafting table with a picture of a family in front of him; and a couple on a bench beside a river. The stories were coded for thoughts relating to power, yielding the *n* Power score,[3] and for thoughts relating to affiliation, yielding the *n* Affiliation score.[14] The coders had demonstrated the ability to code at least at the level of 85 percent agreement with expert coding. The number of times the word "not" appeared in the four stories was also recorded since previous research had demonstrated that it represents the restraint the individual shows particularly in expressing the power motive.[4,6] This characteristic is referred to as Activity Inhibition. Since our primary interest was in subjects high in *n* Power and Activity Inhibition (AI) we recruited half the subjects to be high in *n* Power and AI, defined in the standard way[4] as having at least two "nots" in four stories.

Because thought content codes occur more often in longer stories, the motive scores were corrected for their correlation with length of protocol, using a linear regression transformation which adjusts the score up or down depending on whether it comes from a short or long protocol. The resulting adjusted scores were transformed to standardized T-scores (mean = 50, standard deviation = 10) to make them directly comparable. Thus in the data analysis we follow the practice adopted in other studies of categorizing subjects as high in *n* Power, meaning a T-score of at least 50, which is also higher than their T-score for *n* Affiliation. The reason for this rule is to exclude subjects whose *n* Power score might be high but whose *n* Affiliation score is still higher, since the

evidence is that their desire to be on friendly terms with others undercuts their assertiveness, and the interest here is in those whose drive for power is not ambivalent. Subjects whose *n* Power T-score > 49 ≥ *n* Affiliation T-score will be referred to as high in *n* Power; those in this group who are also high in Activity Inhibition will be referred to as HH subjects.

The experiment was carried out on two days separated by 24-72 hours. On the first day, the subjects filled out a Life Events Schedule adapted from the Social Readjustment Rating Scale of Holmes and Rahe[15] to include events occurring in undergraduate life. They checked whether any of 42 events listed had occurred in their lives since the beginning of the previous summer. Since the subjects were tested individually from December through April, this meant they were reporting events or stresses that had occurred in the previous 6-10 months. Each event was classified by four judges who agreed that it represented either a power/achievement affiliative event, both or neither. Examples of power/achievement events are:

- A major change in your concentration (major) or career plans
- Troubles with the college administration
- A major personal achievement
- Being threatened by a physical attack
- Experienced serious difficulties in meeting your college expenses

There were 22 such events. Subjects were classified as high in power stress if they checked three or more events in this category, which was above the median for the group of subjects as a whole.

Examples of affiliative stresses are:

- A change in family get-togethers
- A new, close personal relationship as in living with someone
- The death of a close friend

There were 11 such events. Subjects were classified as high in affiliative stress if they checked one or more events in this category which was above the median for the group as a whole.

The remaining events had elements of both power and affiliation or neither and so will not be utilized in the data analysis. They included such events as sexual difficulties, a change in religious beliefs or practices, or a major change in eating or sleeping habits.

On the first day the subjects also filled out an Illness Inventory on which they were asked to list all illnesses they had had since school began in September, including colds, flu, hepatitis, sprains, fever, boils, allergies, infections, diarrhea, etc. They rated each illness they listed for severity on a scale of 1-100, "where 100 means you were very, very sick, with high fever, nearly died, etc., and 1 means you hardly noticed the problem." Illnesses were divided into two categories: upper respiratory infections or other types of illness. The two scores analyzed are the number of illnesses reported and the total severity of illnesses listed.

The subject then emptied his bladder completely to adapt him to the procedure of providing urine samples for catecholamine analysis, since Frankenhaeuser[13] has reported that the procedure itself elevates catecholamine excertion in urine. He then made an appointment in the next 48-72 hours to return for a testing session. In preparation for urinary catecholamine analyses carried out on the second day, he was given instructions not to eat chocolate, vanilla, bananas, citrus fruits, cheese or alcohol for two days prior to the testing session. The subject was further instructed to urinate completely about two hours before the experiment (noting the time) and to refrain thereafter from eating, smoking, exercising, or drinking anything except water. Subjects who forgot any of these instructions were rescheduled.

On the second or experimental day, all subjects were run between the hours of 4 and 9 p.m. to minimize known diurnal variations in catecholamine output in urine.[13] On arrival the subject first voided all urine and drank a large glass of water so that urine could be collected again at the end of the 2½-hour testing session. The volume of urine was measured and a sample frozen and sent to a commercial laboratory for assay of the concentrations of epinephrine (E) and norepinephrine (NE) by the trihydroxyindole method.[16] The volume of urine was divided by the time since the last urination to get urine excretion rate. Concentrations of E and NE were multiplied by the urine excretion rate to determine the E and NE excretion rates.

The subject then participated in a number of perceptual and learning tasks for 2½ hours, while his scalp electrical potentials were monitored as part of another experiment. This experience was mildly stressful since it involved working at several tasks under novel circumstances. At the end of this time he again voided all urine, and E and NE excretion rates

were determined for the period of the experiment in the same way as for the period prior to the experiment.

The immunoglobulin A level in saliva was assayed because local immune function within mucous membranes can protect the host via two mechanisms: (1) prevention of pathogen implantation, and (2) elimination of the pathogen following successful colonization of the mucosa.[17] In addition to protection conferred by specific antiviral IgA's, there is evidence that IgA nonspecific for the invading virus is also involved in virus neutralization in the upper respiratory tract. IgA concentration has been monitored for a five-day period following infection with influenza virus.[18] In this study, subjects whose secretory IgA (S-IgA) concentration increased following infection did not become clinically ill although virus particles could be recovered from their nasal secretions. Conversely, subjects with low concentrations of S-IgA developed overt upper respiratory illness. Individual variation in the production of nonspecific S-IgA during upper respiratory infection has been studied prospectively.[19] Those subjects who showed an increase in nonspecific S-IgA during respiratory infection had a lower incidence of such infections throughout a one-year study period than subjects who did not secrete nonspecific S-IgA during respiratory infection. Thus we expected that individual differences in S-IgA concentrations might indicate a general level of resistance to disease, especially to upper respiratory infections. Since it is known that lymphocytes have been demonstrated to have specific membrane surface receptors for β-adrenergic catecholamines[11] which serve to depress lymphocyte reactivity to mitogens, and immune cytolysis,[12] we hypothesized that increased release of epinephrine might result in lower S-IgA concentrations for subjects high in *n* Power, high in Activity Inhibition and high in power stress (the HHH subjects).

Saliva samples were taken on the first day to determine the normal level of S-IgA, and at the end of the experimental session on the second day to determine if the mild stress of the testing had any effect on it. The saliva was frozen until it could be assayed for IgA using the single radial immunodiffusion method. Thawed saliva specimens were deposited in 5 μ cylindrical wells cut in an agar plate impregnated with monospecific goat anti human IgA. As the saliva diffuses in the agar over a 24-hour period, the antiserum forms a disc-shaped immune precipitate with the IgA protein for which it is specific, while other proteins diffuse freely. The

TABLE 1

RELATION OF STRESSED, INHIBITED *n* POWER TO ILLNESS REPORTS

	High Inhibited *n* Power, High Power Stress Subjects[a]	All Other Subjects	Difference	Significance (Predicted Direction)
Number of subjects	7	19*		
Mean number of all illnesses reported[b]	2.08	1.25	.83	t=2.01, p<.05
Standard deviation	.98	.42		
Mean severity of all illnesses reported[b]	10.55	5.86	4.69	t=2.37, P<.05
Standard deviation	4.49	3.18		
Mean severity of upper respiratory infections[b]	6.12	3.03	3.09	t=2.02, p<.05
Standard deviation	3.30	3.09		
Mean severity of other illnesses[b]	6.61	3.62	3.00	t=1.38, p=n.s.
Standard deviation	4.70	3.56		

*Data unavailable on one subject.
[a]T-score *n* Power ≥ 50 and > T-score *n* Affiliation, Activity Inhibition score 2 or more, and three or more instances of power stress reported in the last 6-10 months (=HHH group).
[b]Data have undergone a square root transformation to normalize the distribution.

diameter of the precipitin ring thus formed is proportional to the concentration of IgA in the sample. Concentration values for unknowns are read from a standard curve of precipitin ring diameters vs. known reference concentrations of human serum IgA plotted on semilogarithmic paper. Each agar plate used is checked for possible deterioration with a reference concentration of IgA. The technique used has been described by Mancini, Carbonara and Heremans,[20] modified by Fahey and McKelvey,[21] and made available commercially as the Dade Dataplate immunodiffusion system for quantitation of low levels of serum IgA.

The concentration values obtained must be corrected for the fact that the sedimentation coefficient for serum IgA is lower than that for salivary IgA. Brandtzaeg et al.[22] have demonstrated that there is a linear relation of serum to secretory IgA concentrations and have computed a conversion factor of 3.25 by which secretory levels should be multiplied to give estimates of real concentrations. This conversion factor was applied to the values obtained in this study.

The two extreme reference concentrations provided in this test are 5 and 85 mg/100 ml for serum so that the assay is sensitive between the ranges of 16-273 mg/100 ml for saliva.

RESULTS

Table 1 contrasts the illness reports of the critical group of subjects—the seven subjects high in *n* Power, high in inhibition, and high in reported power stresses (the HHH group)—with the illness reports of the remaining subjects. In all comparisons the raw scores were put through square root transformation to normalize the distributions. One subject, for instance, reported having had 19 illnesses in the previous 6-10 months, while no other subject reported more than four. As expected from other research,[N2] the HHH subjects reported that they had suffered from significantly more illnesses than the other subjects and that they considered these illnesses to have been more severe than the illnesses reported by the other subjects. The HHH subjects reported having had significantly more severe upper respiratory infections (URI's) than other subjects. They also reported more severe non-URI illnesses than other subjects although the difference is not as

TABLE 2

RELATION OF STRESSED, INHIBITED *n* POWER TO CATECHOLAMINE EXCRETION RATES AND CONCENTRATIONS OF IgA IN SALIVA (S-IgA)

	High Inhibited *n* Power, High Power Stress Subjects[a]	All Other Subjects	Significance (Predicted Direction)
Number of subjects	6*	20	Fisher's Exact Test
Percent of subjects above median in epinephrine excretion rates[b]	83%	40%	p = .07[c]
Percent of subjects above median in norepinephrine excretion rates[d]	33%	50%	not sig.
Number of Subjects	7	17*	
Percent of subjects above median in S-IgA concentration on pretest day[e]	14%	65%	p = .03

*Data unavailable on one or more subjects.
[a]T-score *n* Power $\geq$ 50 and $>$ T-score *n* Affiliation, Activity Inhibition score 2 or more, and three or more instances of power stress reported for the last 6-10 months (=HHH group).
[b]Above the median for the average excretion rate before and after the testing = 1 ng/min or more. Mean = 1.94 ng/min, SD = 1.91.
[c]Of the 7 cases low in *n* Power and low in power stress only one showed an above median average epinephrine excretion rate; so the p value in contrast to the HHH group is .016.
[d]Above the median for the average excretion rate before and after the testing = 6.5 ng/min or more. Mean = 5.18, SD = 3.01.
[e]36 mg/100 ml saliva or more. Mean concentration (square root transformation) = 6.21, SD = 3.37.

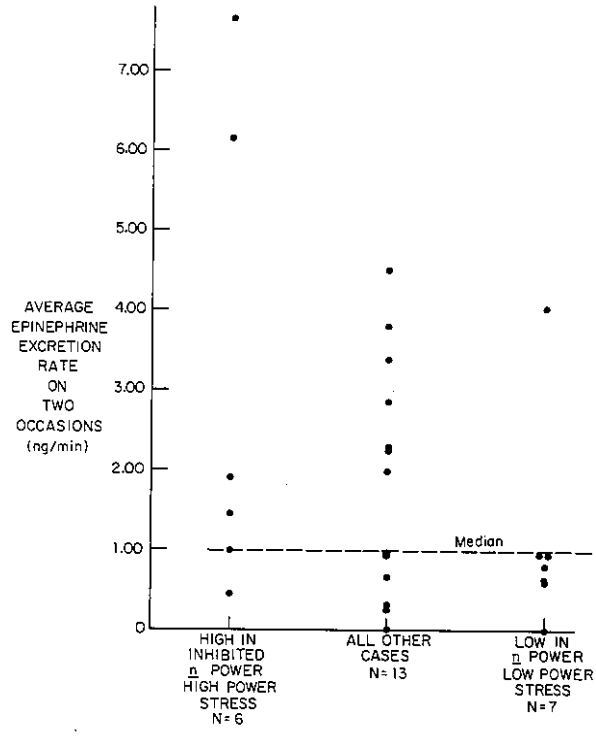

FIGURE 1. RELATION OF INHIBITED AND STRESSED n POWER TO EPINEPHRINE EXCRETION RATE

significant.

In the previous report [N2] the contrast in illness reported was particularly marked between the HHH subjects and those low in power motivation and low in power stress. In this sample the mean number and severity of illnesses reported by the latter group are about the same as for all subjects not in the HHH group. A check was also made to determine whether affiliative stress could act like power stress in being associated with increased reports of illness in subjects high in *n* Power. The six subjects high in *n* Power and in affiliative stress (reporting one or more instances) had a mean illness severity score of 7.95 vs. 7.32 for all other subjects, using square root transformed scores. The difference does not approach significance, nor does it change appreciably if we include subjects also high in inhibition.

To get a reliable index of epinephrine excretion rates, the determinations made before and after the experiment were averaged. As Table 2 indicates, more of the HHH subjects had above average epinephrine excretion rates than other subjects. The contrast is particularly marked, as Fig. 1 shows, with the subjects low in power motivation and in power stress, most of whom had below average epinephrine excretion rates. No significant differences appear for norepinephrine excretion rates.

Finally, as seen in Table 2, more of the HHH subjects had low concentrations of IgA in saliva on the pretest day than subjects with other *n* Power/stress combinations.

Table 3 examines the relationship between epinephrine excretion rates and S-IgA. Subjects who excreted more epinephrine on the experimental day tended to have had lower concentrations of S-IgA on the pretest day. The negative relationship between those two variables is marked (see Fig. 2). Again, the raw scores have been subjected to square root transformations to normalize the distributions.

Table 3 also makes another determination of the relation of epinephrine excretion rate to S-IgA concentrations. Epinephrine excretion rate increased, though not significantly, from before to after the experiment. If epinephrine had a negative effect on immune function, then S-IgA concentrations would be lower after the experiment than on the pretest day. This was the case. The mean S-IgA concentration on the pretest day, after a

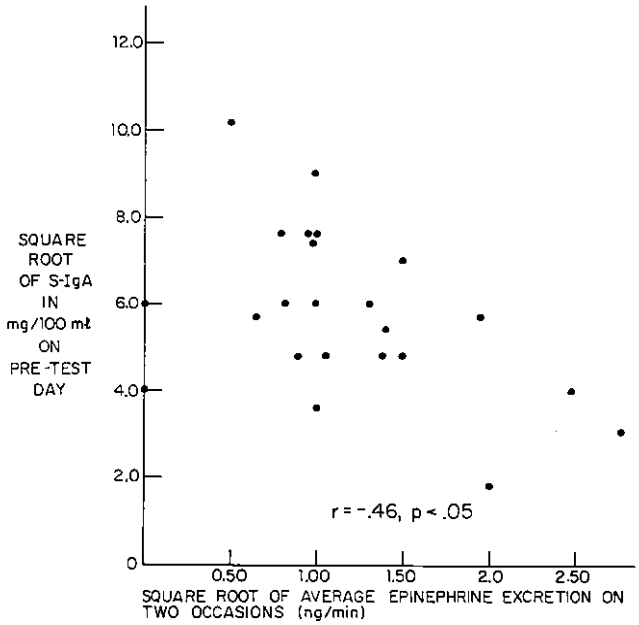

FIGURE 2. RELATION OF EPINEPHRINE EXCRETION RATE TO SALIVARY IgA LEVEL

square root transformation, was 6.16, whereas after the experiment it was 4.19, also after a square root transformation. The difference is highly significant ($t=4.36$, $p<.01$). To pursue this possible relationship further, subjects were categorized into those (N=9) whose epinephrine (E) excretion rate increased from before to after the experiment and those whose E excretion rate decreased, omitting cases which showed zero or near zero E excretion rates both before and after the experiment. Of those with increased E excretion rates, significantly more showed a larger rather than a smaller drop in S-IgA concentrations as Table 3 shows. In other words, increased E excretion rates during the experiment are associated with larger drops in S-IgA concentrations than are decreased E excretion rates, just as overall E excretion rates are associated with lower S-IgA concentrations on the pretest day (Fig. 2).

Finally, there is some evidence that higher S-IgA concentrations on the pretest day are associated with reports of fewer illnesses in the past 6-10 months. Only two out of 11 subjects, or 18 percent with above median S-IgA concentrations (36 mg/100 ml saliva or more), reported having had three or more illnesses in the past 6-10 months, as contrasted with seven out of 12 subjects (or 58 percent) with S-IgA concentrations below the median ($p=.05$ by Fisher's exact test). On the other hand, the correlations between S-IgA concentrations and number of illnesses or their total severity are not significant ($r=-.22$ and $-.13$ respectively), both variables having undergone a square root transformation. However, we had neglected to ask the subjects whether they had been ill at or near the time of the experiment. Such subjects might be expected to have elevated S-IgA concentrations due to specific antibody response to antigens, regardless of the *n* Power/stress group to which they belonged. Such an effort would serve to confound or weaken the tendency for high nonspecific S-IgA concentrations to be associated with fewer illness reported prior to the experiment.

DISCUSSION

The results are consistent with the hypothesis. Subjects high in the need for Power and in inhibition, if they are also under more power stress, (the HHH group) report more severe illness and show signs of higher activity of the sympathetic nervous system under the mildly stressful conditions of this experiment. That is, more of the HHH than the other subjects, particularly those low in *n* Power and power stress, have higher rates of epinephrine

TABLE 3

RELATION OF EPINEPHRINE EXCRETION RATES TO IgA CONCENTRATIONS IN SALIVA (S-IgA)

	S-IgA Concentration on Pretest Day		Significance
	High[a]	Low[a]	Fisher's Exact Test
Number of subjects	11	12	
Percent of subjects above median in epinephrine excretion rates[b]	18%	75%	$p<.01$
	S-IgA Concentration Drop from Pretest to after the Experiment		
	Large 10 mg/100 ml saliva or more	Small (9 mg/100 ml or less)	
Number of subjects	8	8	
Percent of subjects whose epinephrine excretion rate increased from pretest to after the experiment (N=9)	88%	25%	p = .02

[a]Above the median or 36 or more mg/100 ml saliva (high) or less (low).
[b] Average excretion rate before and after the experiment = 1 ng/min or more.

excretion. Furthermore, higher epinephrine excretion rates are associated with a decline in one measure of immune function (S-IgA) whether measured before the experiment or as change in S-IgA concentration from the pretest day to after the experiment.

This relationship does not necessarily mean that higher E release into the bloodstream is directly affecting immune function, though it is known that epinephrine can interfere with lymphocyte function.[10] In the first place, greater E excretion should be considered as a sign of increased sympathetic nervous system activity which probably is also associated with increased release of corticosteroids, which may act as immunosuppressors.[9] Secondly, it is uncertain how E release in the bloodstream could affect concentrations of IgA in saliva over the short course of the experiment (2½ hours) or more generally. One possibility is that the known ability of epinephrine to inhibit salivary secretions[23] may affect IgA concentrations in saliva.

In any case, lowered immune function, however it is produced, should make the individual more susceptible to illness. And significantly more of the subjects with low than with high S-IgA concentrations on the pretest day report having had three or more illnesses in the previous 6-10 months. The relationship is not robust in the sense that it is not significant for correlational analyses, but this may be because some of the subjects had had infections near the time of the experiment. If so, concentrations of IgA in their saliva would tend to rise in specific response to the infection, weakening the general tendency for subjects low in S-IgA to report more illness.

There is some support here for Yodfat and Silvian's finding[19] that higher concentrations of S-IgA are associated with better health (i.e., fewer upper respiratory infections) than lower concentrations of S-IgA. Since in this study higher concentrations of S-IgA are associated with reports of less severe illnesses of all types as well as upper respiratory infections, it seems worth investigating further whether S-IgA concentrations taken when the subject has not been ill may serve as an index of individual differences in general or nonspecific immune function.

This possibility is supported by independent evidence that subjects high in *n* Power and in power stress show other signs of lowered immune function—i.e., lowered leukocyte counts and K-cell function in blood samples.[N1] These signs of depressed

immune function were also associated with reports of more affective symptoms (particularly of depression and anxiety). While the symptomatology involved in that study is more psychic than physiological, as in the present study, in general their findings are consistent with the hypothesis that psychological factors are involved in lowering immune function making the individual more prone to illness.

The results are limited by the small number of cases, by the highly select character of subjects (bright college students), and by the necessity to find life stress events that are particularly distressing to certain types of people. They need cross-validation in other and larger samples, but, so far as they go, they are consistent with the hypothesis that individuals with strong but stressed and inhibited *n* Power show evidence of being more susceptible to illness[1, N1, N2] because they are subject to greater sympathetic activation which, either through the release of epinephrine or other mechanisms, lowers immune function.

INDEX TERMS

power motivation, stress, immune function, catecholamines, illenss.

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