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The Need for Power, Sympathetic Activation, and Illness

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This paper provides a brief summary of a number of recent research reports indicating that n *Power, if it is inhibited or stressed, is associated with high blood pressure and with the frequency and severity of infectious diseases. Other motive/stress or inhibition combinations failed to show these relationships. Blocked* n *Power apparently leads to chronic sympathetic activation, which, over time, results in high blood pressure and which increases epinephrine output that interferes with lymphocyte function, weakening the body's immune defenses against infectious diseases. On the other hand,* n *Power that is expressed successfully and rewarded may lead to better adaptation.*

Men who are high in the need for power (*n* Power), as coded in imaginative thought (Veroff, 1957; Winter, 1973), behave in a number of ways that suggest that they would be characterized by chronic sympathetic nervous activity. As compared with men low in *n* Power they are more argumentative; they anger more easily; they are more apt to engage in competitive sports; they report more difficulty in sleeping at night, which in turn has been associated with higher levels of adrenergic activity (Myager, 1971; and see Winter, 1973, McClelland, 1975). Furthermore, Steele (1973) has shown that sympathetic activation is linked more specifically to the power motive than to the achievement motive. He found that arousing the power motive increased the excretion of epinephrine in urine, a sympathetic activation by-product, more than arousing the achevement motive did (see Table I). And, more significantly, after power motive arousal, the number of power thoughts a subject had was highly correlated with the size of increases in epinephrine and norepinephrine excretion, whereas the same relation between achievement thoughts and signs of sympathetic

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Table I. Relation of Epinephrine Increase to Motivational Content After Different Types of Experimental Arousal

Condition	*N*	Mean gains in epinephrine excretion (ng/min)	Correlations of epinephrine change with motive scores: *n* Power	*n* Achievement
Power arousal (stirring tapes)	16	+ 1.51	.71[a]	.04
Power control (travel tapes)	14	+ .27	.38	.06
Achievement arousal (ego-involving tasks)	19	+ .35	−.02	.25
Achievement control (relaxing tasks)	14	+ .47	.10	.36

[a] After Steele (1973); $p < .05$.

activation did not occur after achievement motive arousal. That is, cognitive signs of increased power motive arousal covaried with signs of increased sympathetic activation.

N POWER AND SYMPATHETIC ACTIVATION

This evidence suggests that men high in *n* Power might be in a chronic state of sympathetic arousal, as indexed by epinephrine excretion, possibly because they are more sensitive to power cues in the environment. And researchers working with Marianne Frankenhaeuser at the University of Stockholm have reported that some individuals do shown chronically higher levels of epinephrine excretion over time than others (cf. Johansson & Post, 1974). Furthermore, the chronically high excreters report significantly more distress (Forsman, 1978) of a type that appears to be mostly related to an inability to act powerfully or so as to exert control over what is happening. For example, they agree more often with statements like "You're working with something that absolutely must be finished within a fixed period of time"; "You have a suspicion—but do not know for certain—that someone in your immediate proximity is irritated with you"; "You are waiting in a long queue and get impatient." These situations all involve power stresses or demands in the sense that the individual wants to exert himself and have control over the situation but cannot. They are certainly of a type that should particularly arouse people high in *n* Power and lead to sympathetic activation and more epinephrine excretion.

N POWER, TYPE A BEHAVIOR, AND CARDIOVASCULAR DISEASE

The Swedish researchers also related their work to the research tradition that distinguishes between Type A (hard-driving) and Type B (more relaxed) individuals (Friedman & Rosenman, 1974). The clinical descriptions of Type A individuals as assertive, often impatient, and angry also are characteristic of individuals high in *n* Power. And Forsman (1978) found that Type A compared to Type B individuals report more stress of the above type (labeled here "power stress"). Type A individuals also show higher levels of catecholamine excretion, particularly when they are prevented from acting or forced into inactivity (Frankenhaeuser, Lurdberg, & Forsman, 1978). Since Type A behavior has been linked to cardiovascular disease, probably through chronic sympathetic activation (Friedman, Byers, Rosenman, & Elevitch, 1970), it seemed reasonable to investigate the possibility that high *n* Power might be involved in susceptibility to cardiovascular disease. That is, it should be if it is a key element in greater responsivity to power stresses of the type the Swedish group was studying and in the assertive, hard-driving behavior characteristic of Type A individuals. A longitudinal study of the blood pressure of male graduates of a prestigious university confirmed this possibility. Men who scored high in *n* Power and also high in inhibition in their early 30s were much more apt to have high blood pressure at a clinically significant level 20 years later than men with other motive characteristics (McClelland, 1979). Furthermore, men who were high in *n* Achievement or *n* Affiliation had significantly lower blood pressure 20 years later than men with low *n* Achievement or low *n* Affliliation. In short, since it was *n* Power alone that was associated with chronic sympathetic activation, it is reasonable to infer that *n* Power might be associated with sympathetic activation.

VARIABLES MODERATING THE EFFECT OF *N* POWER ON ADAPTATION

However, in this study and in others, it turned out that *n* Power is not by itself related to sympathetic activation and illness. For it to be related to disease it must be blocked, as in this case, by an inner inhibition, or by external stressors. In fact, one may presume that the relation between inhibited *n* Power and high blood pressure was obtained in these subjects in part because most of them were also exposed to strong occupational demands for powerful behavior since most of them were in high-prestige occupations. So attention must be given to three variables that interact with

n Power to modify its effects. One such variable is the amount of internal inhibition the person shows, which tends to block the tendency of the person high in *n* Power to act assertively. Another is the degree of power stress or frustration to which the person is subjected, which, if it is extreme enough, may make it impossible to act powerfully. Still a third is the strength of *n* Affiliation, since caring for others tends to moderate the effects of the power drive in individuals (McClelland, 1975). That is, one may think of a high *n* Affiliation as in effect counteracting *n* Power. For this reason, in the blood pressure study and others dealing with the relation of *n* Power to illness reported below, it is stipulated that *n* Power must be at least moderately high (*T* score of 45 or more) and higher than the *n* Affiliation *T* score.

Still another moderating variable related to how *n* Power is related to adaptation is whether it can be expressed in a rewarding way. A clue to the importance of this variable can be found in the fact that the Swedish researchers also report that individuals who chronically excrete more epinephrine are better learners than those who excrete lesser amounts (Johansson, Frankenhaeuser, & Magnusson, 1973; Bergman & Magnusson, 1976). Thus, if we associate high *n* Power with high epinephrine output, it follows that high *n* Power should under some conditions facilitate performance and under others interfere with it through being associated with distress and emotional arousal. The Swedish researchers have argued that the difference in these two effects is connected with whether or not there is a fast return to normal levels of epinephrine excretion after arousal: "An acute rise in adrenaline excretion in a challenging situation and a subsequent fast return to lower levels is associated with emotional stability and well-being, whereas a more constantly high level of physiological and subjective arousal may be indicative of maladjustment" (Forsman, 1978).

In motivational terms, what is suggested by this difference is that in the former instance emotional arousal is associated with reward and in the latter instance it is not. A hungry rat will show signs of physiological and emotional arousal. If it is rewarded by food, it will learn the responses that lead to the food and the signs of emotional arousal will diminish rapidly. If it is not fed, it will not learn and will continue to show signs of distress and maladjustment. To test the applicability of this model to the power motive, we must have a measure of what is rewarding it. There are several reasons for thinking brain norepinephrine (NE) turnover might be an index of what is rewarding to the power motive. For one thing, injection of NE into the ventricle of the rat brain is rewarding in the sense that rats will press a bar repeatedly to get the injections (Olds, 1977). For another, we have already advanced several reasons for thinking that the power motive may be subserved physiologically by the adrenergic or

catecholamine neurotransmitter system. And, finally, studies by Weiss, Stone, and Harrell (1970) have shown that rats that can learn to avoid shock show increases in brain NE whereas rats that cannot learn while being exposed to inescapable shock show decreases in brain NE. It is possible to interpret these findings to mean that learning to control a threat is rewarding to the power drive, that inability to respond successfully to a power threat is frustrating, and that increases and decreases in brain NE reflect the power rewards present or absent in the situation.

However speculative such a line of reasoning may be, it proved possible to test it in humans as follows. Subjects classified as high or low in *n* Power learned to associate power-related and neutral words to power-related and neutral pictures. As expected, those high in *n* Power learned the power-related pairs (as compared to the neutral pairs) more quickly than subjects low in *n* Power. In contrast, subjects high in *n* Achievement did not learn any of the pairs more quickly than subjects low in *n* Achievement. It was also possible to get an estimate of the amount of increase in brain NE turnover during the learning by analyzing the amount of 3-methoxy-4-hydroxyphenylglycol (MHPG) in urine collected before and after the experiment (McClelland, Davidson, Saron, & Floor. It was found that subjects who were high in *n* Power and in increase in MHPG during the experiment learned the power-related pairs most quickly, significantly more quickly than those who were low in both variables. It is possible to interpret these findings as meaning that the subjects high in *n* Power who found the power-related responses rewarding learned them most quickly. By this line of reasoning, the increase in MHPG for the subjects high in *n* Power reflected the extent to which they found the power-related responses rewarding. As would be expected, when the reward for power-related associations is absent (no MHPG increase) for those high in *n* Power or is present for those low in *n* Power, learning is less efficient, but it is least efficient when both the motive to learn power-related responses and their reward value are missing. Other interpretations are possible, the number of subjects was small, and the finding needs to be confirmed, but it is consistent with and suggests a reason for the repeated observation by others that sometimes power stress and power drives lead to better and sometimes to poorer adaptation. If people high in *n* Power can express it in a way that leads to satisfaction, they should learn the relevant behavior more quickly and adapt better generally. The reverse should be true when they are blocked from expressing power-related behavior and can find no satisfaction. In this case, emotional arousal and sympathetic activation should remain high and not return readily to normal levels, leading to the kinds of distress and maladaptation reported by the Swedish researchers.

THE INHIBITED AND STRESSED POWER MOTIVE SYNDROME, IMMUNE FUNCTION, AND ILLNESS

The evidence linking inhibited *n* Power to poorer (as contrasted with better) adaptation has been more fully worked out. Table II shows how three factors related to the power motive combine to increase the severity of illnesses reported by college students. For these data, the variance in severity of illness score among groups is significantly greater than within groups, the motive variable contributes significantly to the variance ($p = .05$), as does the stress variable ($p = .086$), and the interaction between these variables is insignificant, indicating that the two

Table II. Mean Severity of Reported Illnesses Among Groups Differing in *n* Power, Power Stress, and Inhibition[e]

	High activity inhibition[a]			Low activity inhibition		
Subject groups	*N*	*Mean*	*SD*	*N*	*Mean*	*SD*
n Power > *n* Affiliation[b]						
High power stress[c]	10	195.5	195.1	8	79.9	56.6
Low power stress	13	103.7	95.9	10	59.3	49.4
n Affiliation > *n* Power[d]						
High power stress	5	66.4	74.8	11	84.5	64.2
Low power stress	10	51.7	40.9	8	47.4	23.8

Sources of variance among vs. within groups, $df = 7/67$, $F = 2.32$, $p < .04$.

Planned comparisons	*N*	Mean severity of illness	*F* values
High *n* Power, high power stress, high Activity Inhibition	10	195.5	
vs.			
low on all three variables	8	47.4	10.1, $p < .01$
vs.			
all other subjects	65	72.9	13.9, $p < .001$
When Activity Inhibition unknown High on *n* Power and power stress	18	144.1	
vs.			
all other subjects	57	71.9	6.5, $p < .02$
When power stress unknown High on *n* Power, and Activity Inhibition	23	143.6	11.5, $p < .01$
vs.			
all other subjects	52	65.2	

[a] Score of 2 or more.
[b] *T* score *n* Power $\geq$ 45 and > *T* score *n* Affiliation.
[c] Above the median or 4 or more power/achievement life events checked for last year.
[d] *T* score *n* Power $\leq$ 50 and < *T* score *n* Affiliation.
[e] From McClelland and Jemmott, 1980.

variables summate in their effect. The inhibition variable also contributes to the variance, but less significantly ($p = .125$). So, as one would expect, subjects who score high in *n* Power (and higher than *n* Affiliation), high in Activity Inhibition (which controls the expression of *n* Power, McClelland, 1975), and high in power stress report by far the highest level of severity of illnesses of all types, compared to other groups of subjects. Furthermore, any combination of two of these variables is associated with more illness than for other subjects. The same relationships do not hold for other motive-stress combinations, such as high versus low *n* Achievement in combination with high versus low achievement-power stress (McClelland & Jemmott, 1980). The presumed explanation for these findings is that subjects high in *n* Power who cannot obtain power rewards, either because they inhibit assertive actions or cannot deal with power stressors successfully (or both), are likely to be in a state of chronic sympathetic activation, which does not return readily to normal levels. In consequence, they are more likely than other subjects to discharge more epinephrine and/or cortisol into the bloodstream, which can interfere with lymphocyte function, lowering the body's immune defenses against disease. Two studies have shown that subjects with the inhibited or stressed power motive syndrome have impaired immune functions compared to other subjects. In one study, college students high in *n* Power and in self-reports of power stress had significantly lower white cell counts and natural killer cell assays than subjects high in *n* Power and low in power stress, who seemed to be particularly healthy. High or low power stress in combination with low *n* Power (and high *n* Affiliation) was not associated with differences in levels of immune function. Nor were other motive-stress combinations associated with alterations in these indexes of of immune function (McClelland, Locke, Williams, & Hurst, 1980).

In the other study, similar relationships were found for a very different group of subjects—males in prison chiefly for violent crimes—and for a different measure of immune function, namely, salivary immunoglobulin (S-IgA), the body's first line of defense against upper respiratory infections. Prisoners high in *n* Power, and high in the number of stresses reported (all prison stresses were considered power-related), had the highest average level of illnesses reported and the lowest average level of S-IgA as compared to other subjects (McClelland, Alexander, & Marks, 1980). Furthermore, subjects with lower levels of S-IgA more often reported being more sick than other prisoners.

What is still needed is more information on the role of epinephrine and/or cortisol in the presumed causal chain linking the blocked power motive syndrome to disease. Only one pilot study has so far been carried out to check on this connection, but its results do conform to expectation (McClelland, Davidson, Floor, & Saron, 1980). Subjects high in *n* Power,

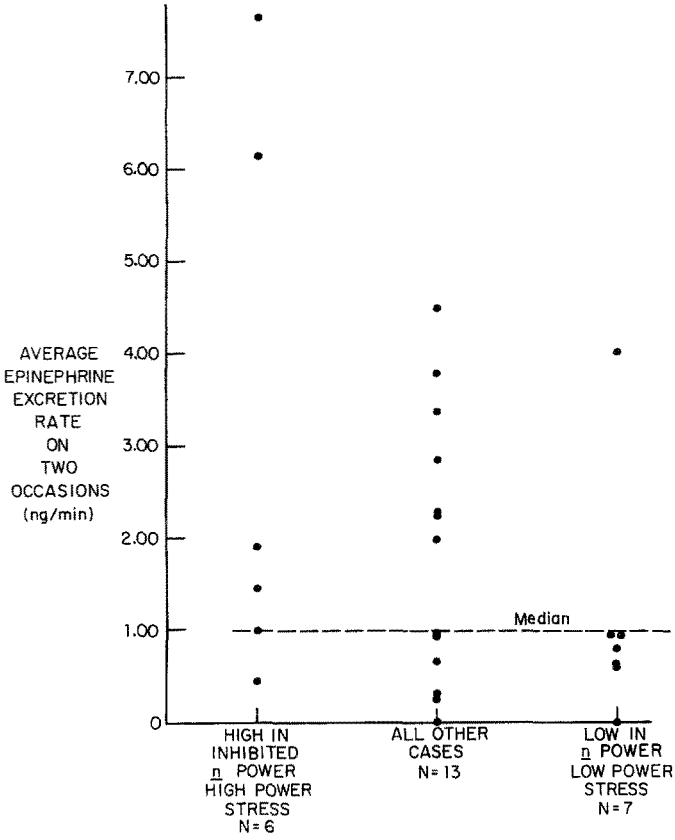

Fig. 1. Relation of inhibited and stressed *n* Power to epinephrine excretion rate (from McClelland, Davidson, Floor, & Saron, 1980).

high in Activity Inhibition, and high in power stress do tend to have higher levels of epinephrine excretion, significantly more often higher than those low in *n* Power and power stress (see Figure 1). And subjects who average high in epinephrine excretion do tend to have lower levels of concentration of S-IgA (see Figure 2). Furthermore, it was found that those whose epinephrine levels increased from before to after the experiment had lower concentrations of S-IgA afterwards than those whose epinephrine levels had decreased. And in this study, as in the one involving prisoners, low concentrations of S-IgA were associated with reports of more severe illnesses in the past 6-10 months.

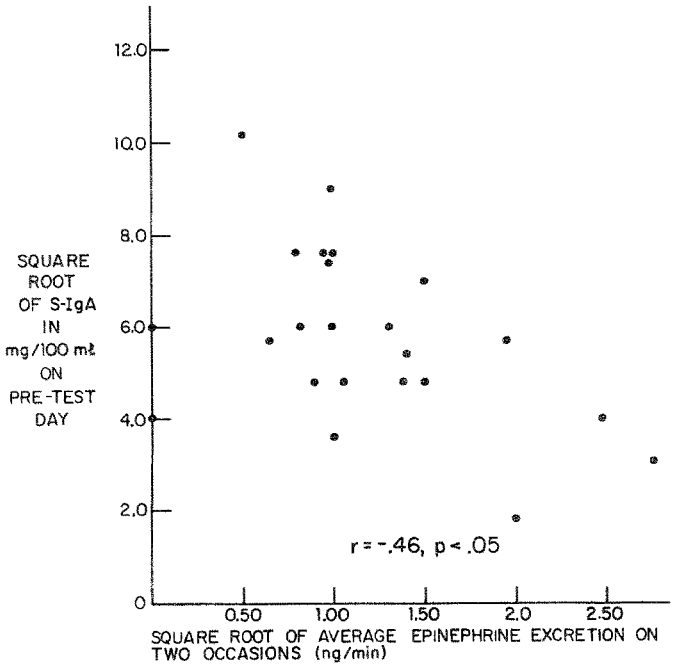

Fig. 2. Relation of epinephrine excretion rate to salivary IgA level (from McClelland, Davidson, Floor, Saron, 1980).

SUMMARY

More research is needed, and some of it is in progress, to work out with confidence the ways in which other factors combine with *n* Power to lead to better adaptation or distress and illness. Certainly *n* Power and power stress appear to be more involved with health and illness than other motive-stress combinations, probably through the connection of *n* Power with sympathetic activation and the catecholamines. And some evidence does exist for all of the connections in the following model, which relates a blocked *n* Power to illness.

A		B		C		D
A high *n* Power that is blocked through internal inhibitions or external stressors (i.e., unrewarded *n* Power)	*leads to*	Signs of chronic sympathetic activation (e.g., more epinephrine excretion)	*leads to*	Lowered immunocompetence (e.g., lower S-IgA)	*leads to*	More physical illness

Significant relationships have been reported between A and D, A and C, and A and B, and also between B and C and C and D. It seems reasonable to assume further that the demonstrated connection between A and chronic high blood pressure is also mediated by the chronic sympathetic activation presumed to be characteristic of individuals with the blocked power motive syndrome.

What is particularly needed is study of the effects on these connections of experimentally induced power stresses or opportunities for power rewards. What is at stake is a much clearer and more detailed knowledge of how particular psychological variables combine with *n* Power to promote health and adaptation or illness.

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