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#### **ABSTRACT**

All chronic illnesses represent assaults on multiple areas of functioning, not just the body. To examine the association between painful chronic illnesses and depression from a cognitive-behavioral perspective, 100 patients of the Pain Management Program at the West Haven, Connecticut Veterans Administration Hospital (784 males) completed a battery of tests. The tests assessed depression, pain severity, life interference attributed to pain, perceived self-control, and social support. An analysis of the results showed that pain was a significant predictor of perceived interference, support, and self-control. In turn, perceived self-control and interference were significant predictors of depression. Pain was not a significant direct predictor of depression. The coefficient of determination indicated that 68% of the variance in depression was accounted for by the cognitive-behavioral model. The results suggest that all of the structural relations predicted by the cognitive-behavioral model of pain and depression were confirmed, with the exception of the relationship between support and self-control, which was found to be nonsignificant. (BL)



Identifying the Links between Chronic Illness and Depression:

Cognitive-Behavioral Mediators (2)

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Running head: CHRONIC ILLNESS AND DEPRESSION

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Identifying the Links between Chronic Illness and Depression: Cognitive-Behavioral Mediators

Among health care providers, the classification "chronic illness" implies a significant degree of irreversability of the pathological process and related disability. Despite the range of conditions and disabilities encompassed by this category, a number of common features are concomitants of each illness. All chronic illnesses represent assaults on multiple areas of functioning, not just the body. Patients with various chronic illnesses may face sepration from family, friends, and other sources of gratification, loss of key roles, disruption of plans for the future, assaults on self-image and self-esteem, uncertain and unpredictable future, as well as such illness related factors as permanent changes in physical appearance or in bodily function.

On the basis of such extensive adjustive demands, one might expect a priori that the presence of chronic illness would result in breakdown of integrated functioning and the development of significant emotional difficulties especially, depression. Because of this face valid assumption, much of the work on the stresses and coping with chronic illness have focused on negative events and dysfunctional patterns of responding. The emphasis has been on the description of adjustment problems. Very little attention has been directed toward understanding how patients and families restructure



their lives, how some develop effective ways of responding to the diversity of adjustive demands that confront them, and how patients and families meet the challenge of chronic disease or disability (Seime & Zimmerman, 1983; Turk, 1979). When research suggests that patients and families are coping in a satisfactory manner, often they are disregarded or belittled with the suggestion that patients are employing the defense mechanism of denial and that true pathology is only masked (e.g., Flannery, 1978; Glassman & Siegal, 1970).

Part of the reason for the emphasis on dysfunction stems from the fact that many of the writings in this area have been conducted by mental health professionals. Because of their training, clinical psychologists and psychiatrists are "prepared" to uncover pathology (Turk & Salovey, in press a, in press b). Moreover, by nature of their work, mental health professionals tend to spend the greatest amount of their time with individuals who are having difficulty adjusting to their plight (i.e., patients and families who are distressed and incapacitated). Relatively small percentages of patients with any specific chronic illness come to the attention of mental health professionals (Turk & Salovey, in press b). As a result, the maladaptive responses of a small minority come to be viewed as representative of all patients with a similar medical problem. It is hazardous to generalize from such a restricted and potentially unrepresentative sample.



Despite the presence of conditions that are clearly traumatic and disruptive, a substantial proportion of individuals and their families appear to make satisfactory adjustments without significant emotional distress (for a review of the evidence to support this conclusion see Turk, 1979). In short, there appears to be no isomorphic relationship between any chronic illness and dysphoric mood.

In this paper we will examine specifically the association between a broad category of chronic illnesses that are characterized by the presense of unremitting pain. The is a good deal of controversy in the pain literature as to whether chronic pain is related to an underlying personality disorder (e.g., Blumer & Heilbronn, 1982) or whether depression, when observed, is a secondary reaction to the presence of a pain problem (Turk & Salovey, 1984). Moreover, there is a great deal of confusion regarding the association between pain and depression with percentages of depression in pain patients ranging from 10% to 90%. One reason for the degree of confusion is that chronic pain and depression have a number of factors that are common to both, for example, sleep problems, reduction in activity, reduction in sources of gratification (i.e., positively reinforcing activities and interactions), loss of interest in sex, feeling of helplessness and hopelessness, and so forth. Additionally, the neurotransmitter, serotonin, appears to play an important role in both pain and depression. Much



research is needed to disentangle the association between pain and depression.

A cognitive-behavioral model of chronic pain (having much in common with cognitive-behavioral models of depression, e.g., Beck, 1967; Abramson, Seligman, & Teasdale, 1978) proposes that the association between pain and depression is mediated by cognitive and behavioral factors. One cognitive factor likely to mediated this association is perceived control (Turk, Meichenbaum, & Genest, 1983). The behavioral factors most likely related to depression are perceived and actual interference with life due to pain and the responses of significant others to expressions of pain by the patient (Fordyce, 1976; Turk et al., 1983). From our cognitive-behvioral perspective, we would predict that the direct association between pain and depression should be quite small. Instead, there should be an indirect relationship mediated by perceived control, perceived interference and satisfaction with life attributed to the pain problem, and environmental contingencies most specifically responses of significant others.

Our cognitive-behavioral model predicts that the presence of pain will be directly related to increased interference with life activities and reduction in perceptions of control and that greater interference with life and reductions in perceived control will be associated with increased depression. In other words, interference and



self-control serve as mediators or intervening variables between pain and depression (see Figure 1). Additionally, we hypothesize that well-meaning and supportive responses of spouses will, paradoxically, actually lead to greater interference with various domains of the patient's life and subsequently greater degrees of depression. The latter prediction stems from an operant model of pain (Fordyce, 1976) that suggests that attention from significant others for "pain behaviors" will lead to reduction in physical activities. Pain behaviors are conceptualized as those behaviors that the patient emits and that communicate the presence of pain even if not directly verbalized (e.g., grimacing, holding the painful part or rubbing the painful area of the body).

Insert Figure 1 about here

#### Method

# Subjects

The participants in this study were selected from 130 consecutive referrals to the Pain Management Program at the West Haven, Connecticut Veterans Administration Medical Center. The inclusion criteria were: (a) duration of pain 6 months or longer, (b) chronic pain other than headaches or related to neoplastic disease, and (c) no evidence of active psychosis or acute suicidal risk.



Of the 130 patients screened, 100 patients, who met the above criteria, provided complete data. The mean duration of pain was 10.7 years (range 6 months to 40.6 years). The distribution of the primary pain syndrome was quite broad (e.g., trigeminal neuralgia, tendenitis, herniated disc, rheumatoid arthritis), with the largest group of patients (36%) reporting lower back pain. The mean age of patients was 50.8 (SD=14.5), 78% were male, 66% were currently married, 56% had at least one pain-related surgery, 52% were receiving some form of disability compensation, and 67% were taking a prescribed analgesic medication.

## Measures

<u>Depression</u>. The patients' levels of depresion were measured by two well established instruments, the Beck Depression Inventory (BDI; Beck, 1967) and the Depression Adjective Checklist (DACL; Lubin, 1965).

Pain Severity. Three measures were used (a) the total score of the Pain Rating Index from the McGill Pain Questionnaire (MPQTOTAL; Melzack, 1975), which is comprised of 20 groups of words reflecting different levels of pain intensity that subjects select to describe their pain; (b) the Pain Severity Scale of the West Haven-Yale Multidimensional Pain Inventory (WHYMPI-PS; Kerns, Turk, & Rudy, 1984), which assesses pain severity by means of multiple 7-point scales that address the patients' pain level at the present moment, during the past week, the amount of



suffering they experience, and so forth; and (c) the mean from 2 weeks of hourly self-monitoring of perceived pain intensity (PAINCARDS; Turk et al., 1983). The latter was based on a self-monitoring procedure developed by Budzynski, Stoyva, Adler, and Mullaney (1973) and adapted by Turk et al. (1983). Patients were taught to keep an hourly record of their level of pain on a 0 to 5-point scale. For each patient, a mean hourly pain intensity rating was derived for all hours coded during a 2 week period.

Interference. The amount of perceived life interference attributerd to pain was assessed with three scales from the WHYMPI (Kerns et al., 1984). These scales were: (a) Social Interference scales (WHYMPI-Social), comprised of questions related to how much patients' feel their pain has affected their ability to participate in social and recreational activities and the amount of satisfaction that they gain from these activities; (b) the Work Interference scale (WHYMPI-Work), also comprised of multiple ability and satisfaction questions in terms of work; and (c) the Family Interference scale (WHYMPI-Family), which is comprised of multiple questions designed to measure how much marital and family disruption patients feel has resulted from their chronic pain problem.

Self-Control. Patients' perceptions of self-control were measures by (a) two items from the WHYMPI related to how much control they felt they had over their life during the



past week (WHYMPI-Life Control) and how much they felt they had been able to solve their problems during the past week (WHYMPI-Problem Solving) and (b) the Internal Subscale from the Multidimensional Health Locus of Control (MHLC-Internal; Wallston, Wallston, & DeVellis, 1978).

Social Support. Supportive responses of significant others to expressions of pain were measures by three subscales from the WHYMPI each designed to assess the patient's perception of the nature of the responses of significant others to their pain. The three subscales included: (a) degree of general suport and attentiveness (WHYMPI-Support), (b) solicitousness including provision of food or medication and asumption of responsibilities (WHYMPI-Solicitiousness), and (c) attemps to distraction the patient (WHYMPI-Distracting).

### Procedure

The scales used in this study were administered as part of a comprehensive assessment and treatment program offered by a hospital-affiliated pain clinic. Although the assessment instruments were self-administeredd, the clinicians conducting the assessment were free to respond to patients' questions.

# Data Analysis

The product moment correlation matrix of the 14 measured variables was used to conduct maximum-likelihood estimation under causal modeling with latent variable



procedures. The mathematical basis for these procedures is discussed by Joreskog (1978) and good discussions of the conceptual and technical issues are provided by Kenny (1979) and Long (1983a, 1983b).

The LISREL-V computer program (Joreskog & Sorbom, 1981) was used to obtain the maximum-liklihood estimates of all model parameters and a chi-square goodness-of-fit statistic. Because the robustness of the chi-square test under non-normality in the data is not known, a normed goodness-of-fit index (A) suggested by Bentler and Bonett (1980) was also computed for each of the structural models tested. This index, which ranges 0 to 1 (the model completely explains the observed data), describes the degree of fit of a specific structural model independent of sample size.

#### Results

The first step in our analysis consisted of computing the Pearson correlations among the 14 measured variables to establish whether the scales that were hypothesized as operationalizing the latent constructs were, in fact, associated with one another. Moreover, we were intersted in examing the relationship between pain and depression. The means, standard deviations, and product-moment correlations for the measured variables are shown in Table 1. As can be seen in Table 1, all correlations among the indicators for each of the four latent constructs were statistically



significant. Interestingly, the correlations among the pain and depression measures were quite low ranging from -.03 to .23. These correlations suggest that the direct assolation between pain and depression is, at best, modest.

# Insert Table 1 about here

Recent developments in the methodology of causal modeling with latent variables permit direct tests of the pain and depression model hypothesized (see Figure 1). In sontrast to the most popular form of causal modeling, path analysis, causal models within the framework suggested by Joreskog (1978) embed factor analysis within the path-analytic tradition.

Structural modeling with latent variables consists of two portions that are estimated simultaneously. One portion, the measurement model, specifies the relationship between the measured or observed variables and the latent (unobserved) variables, the results of which is a set of factor scores.

Thus, although conceptually similar to the factor analytic approach, the measurement model is also confirmatory in that relationships between measures and factors need to be defined a priori based on theory. The correlation matrix in Table 1 was used as the basis for all the structural modeling tests.

The results of the test of the measurement model are displayed in Table 2. Examination of this table reveals that



all of the scales employed loaded significantly on the hypothesized factors and thus confirm that the observable variables are appropriate for assessing the latent constructs of interest, namely, Pain, Depression, Interference, Support, and Self-Control.

# Insert Table 2 about here

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The second portion of causal modeling the structural relations model, specifies the pattern of relationships or influences among the latent variables. If directionality or causal influences are specified among the latent variables, as we did (see Figure 1), then the resulting parameters are regression coefficients. In Figure 1, all of the hypothesized relationships are presented. Thus, as displayed in Figure 1, Pain Severity was hypothesized to cause increases in Life Interference and Social Support, which in turn, based on cognitive-behavioral theories of depression should increase a patient's level of Depression. In terms of the other intervening variable, Self-Control, Pain Severity was predicted to have an inverse affect on a patient's perceived Self-Control which then, as a result of this decline in Self-Control, should increase the patient's level of Depression. In sum, we hypothesized that the regression coefficients between Pain and Interference, Pain and Support, Support and Interference, and Interference and Depression



should all be positive and the the coefficients between Pain and Self-Control, Support and Self-Control, and Self-Control and Depression should be negative. Additionally, although a direct link between Pain and Depression is shown in Figure 1, we hypothesized that this link would not be significantly different from zero and could be dropped from the model without significantly affecting the overall goodness-of-fit of the structural model.

The unstandardized regression coefficients for the latent constructs are presented in Table 3. Observation of Table 3 reveals that, as predicted, pain was a significant predictor of Interference, Support, and Self-Control. In turn, Self-Control and Interference were significant predictors of Depression. Furthermore, as displayed in Table 3, Pain, as hypothesized by our cognitive-behavioral model, was not a significant direct predictor of Depression.

# Insert Table 3 about here

Figure 2 depicits our original theoretical model along with the standardized coefficients for the latent variables that resulted from our structural analysis of the data contained in Table 1. Both the chi-square goodness-of-fit index,  $\chi^2(73) = 88.10$ , p = ns, and Bentler and Bonett's normed index,  $\Delta = .85$ , indicated that our model provided a good accounting of these data. Additionally, the coefficient of



determination indicated that 68% of the variance in Depression was accounted for by this model. In sum, all of the structural relations predicted by our cognitive-behavioral model of Pain and Depression were confirmed with the exception of the relationship between Support and Self-Control, which, contrary to our prediction, was found to be nonsignificant.

#### Discussion

We presented a model describing the relationship between pain and depression that incorporated cognitive and behavioral factors. Specifically we proposed and tested a model that hypothesized that there was no direct relationship between chronic pain and depression. The proposed theoretical model hypothesized that the relationship between pain and depression would be an indirect one with patients' appraisals of the degree to which pain interferes with their life, how much control they have, and how others respond to their pain mediating the association between and depression. Furthermore, following from Fordyce's (1976) operant conditioning model, we hypothesized that as patients' perceived more attention (i.e., positive reinforcement) for their pain behaviors there would be a reduction in activities and consequently increased perception of interference. Structural modelling analytic techniques confirmed the cognitive-behavioral model hypothesized. Moreover, the results of this study revealed that the direct relationship



between pain and depression was quite modest when the intervening variables of interest were excluded from the model.

The results of this study have important implication for the treatment of depressed chronic pain patients as they suggest that treatment should focus on enhancing the patient's perception of self-control. The cognitive-behavioral treatment model for chronic pain proposed by Turk and his colleagues (e.g., Turk et al., 1983) specifically focuses on the patient's perception of control. Thus, the results of the present study provide support for the theoretical rationale of the cognitive-behavioral treatment approach. In addition, some support for Fordyce's (1976) operant conditioning model was provided by the present results as increased positive reinforcement from singnificant others was significantly, although modestly associated with greater interference with various activities and interference was a significant predictor of depression.

More generally, the results of the present study provide some evidence for a buffering hypothesis with high levels of perceived control associated with lower levels of depression. Future research needs to establish the generalizability of these findings with other populations. The results do suggest that the cognitive variable of perceived control appears to be an important mediator of depression in a group of chronic pain patients.



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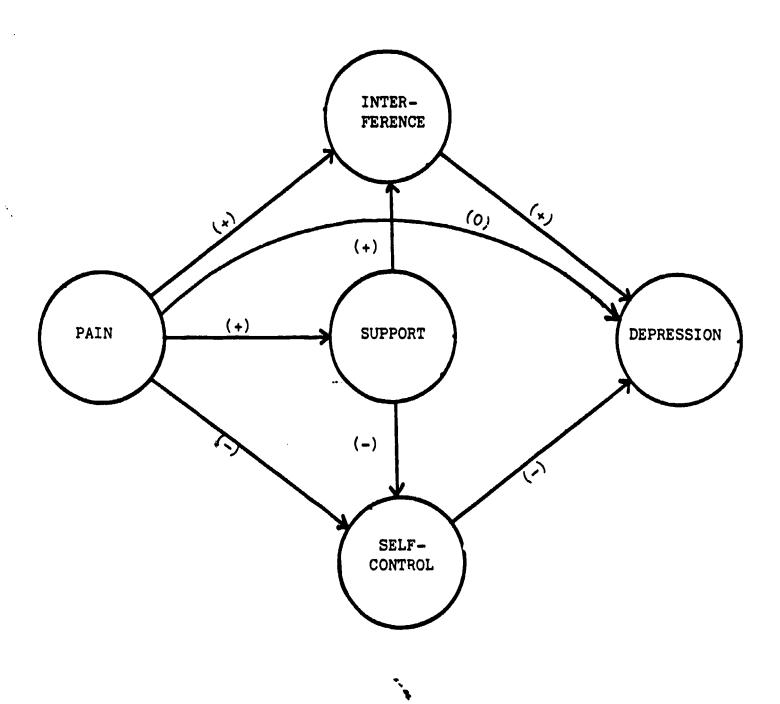
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Figure 1
HYPOTHESIZED LATENT VARIABLE CAUSAL MODEL FOR THE
DEVELOPMENT OF DEPRESSION AMONG CHRONIC PAIN PATIENTS





# PARAMETER ESTIMATES AND STANDARD ERRORS FOR THE STRUCTURAL MODEL

Dependent Latent Variable	I	Inde; II	pendent La II		Variable IV	٧
	l	Instandard	lized Coef	ficie	ents a	
I. Interference			.19	·(.11)	)	.54***(.11)
II. Self-Control			.10(	(.12)		31**(.12)
III. Support						.30**(.13)
IV. Depression	.31*(.15)	75***	(.14)			13(.15)
V. Pain						
	S	itandardiz	ed Coeffi	cient	;sa	
I. Interference		••	.19	)		. 58
II. Self-Control		<b></b>	. 11			35
III. Support	•					. 31
IV. Depression	. 34	77				16
V. Pain						
		Correl	ations		<del></del>	
I. Interference	1.000					
II. Self-Control	181	1.000				
III. Support	. 371	.003	1.00	10		
IV. Depression	. 379	780	. 07	<b>'</b> 6	1.000	
V. Pain	. 637	314	. 30	6	. 302	1.000

Note. Numbers in parentheses are standard errors.



Regression coefficients fixed at zero are not shown.

<sup>\*</sup>p < .05. \*\*p < .01. \*\*\*p < .001.

Table 1
MEANS, STANDARD DEVIATIONS, AND PRODUCT-MOMENT
CORRELATIONS FOR THE MEASURED VARIABLES

15

			_			_				_					
	SCALE	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1.	WHYMPI-WORK														
2.	WHYMPI-SOCIAL	.74					•								
3.	WHYMPI-FAMILY	. 53	.65												
4.	WHYMPI-SELF CONTROL	12	18	30	1										
5.	WHYMPI-PROBLEM SOLVING	11	20	35	.71										
6.	MHLC-INTERNAL	13	10	. 03	. 23	. 31					.•				
7.	WHYMPI-SOLICITIOUS	. 35	. 36	. 23	08	04	02	N							
8.	WHYMPI-SUPPORT	.11	.02	09	. 06	. 09	.09	. 54						,	
9.	WHYMPI-DISTRACTING	.15	.08	05	.09	. 14	. 21	. 49	. 35						
10.	BDI	.425	.29	. 36	54	53	18	. 09	07	. 03					
11.	DACL	. 21	.11	. 29	30	41	06	09	14	. 05	. 42				
12.	WHYMPI-PAIN SEVERITY	. 53	. 46	. 41	15	28	27	. 32	. 01	. 04	. 23	. 18			
13.	PAINCARDS	. 23	. 31	. 26	01	03	09	. 15	05	. 17	03	. 01	. 52		
14.	MPQTOTAL	. 30	. 26	. 26	18	14	10	.12	03	04	. 09	. 11	. 45	. 29	<u>\</u>
	MEAN	3.9	4.3	3.0	3.7	3.6	22.8	2.6	4.3	1.7	14.3	13.4	3.6	2.6	. 4
	STD. DEV.	1.5	1.6	1.5	1.8	1.7	6.3	1.2	1.5	1.1	8.8	5.4	1.2	1.1	.2

Note. Correlations  $\geq$ .19 or  $\leq$ -.19 are significant at or below the .05 level.



# Table 2 PARAMETER ESTIMATES AND STANDARD ERRORS FOR THE MEASUREMENT MODEL

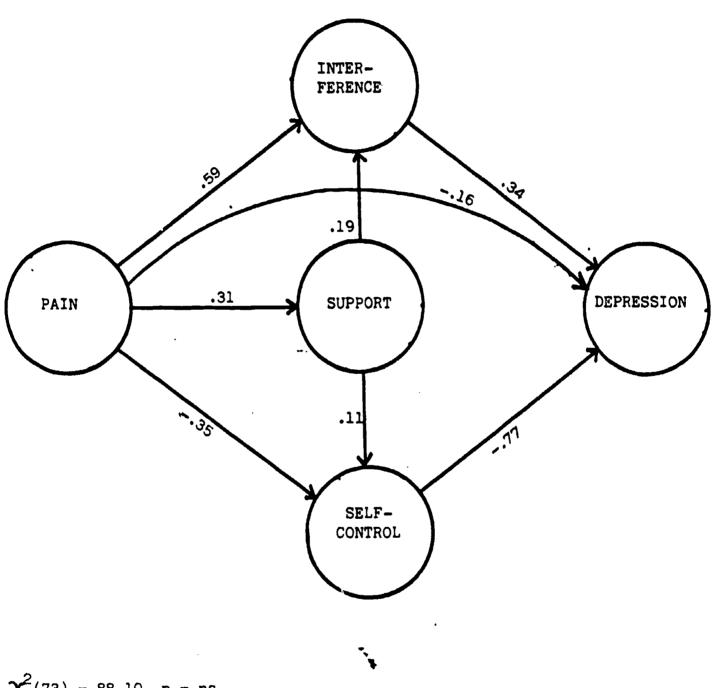
	Unstandardized		Standardized		
Measure	<u>Loading</u>	SE	Loading		
<u>Interference</u>					
WHYMPI-Work WHYMPI-Social WHYMPI-Family	1.000°C 1.062* .839*	.000 .121 .120	. 833 . 884 . 699		
<u>Self-Control</u>					
WHYMPI-Self-Control WHYMPI-Problem Solving MHLC-Internal	1.000 <sup>Q</sup> 1.145 <del>*</del> .418*	.000 .161 .142	.787 .901 .329		
Support					
WHYMPI-Solicitious WHYMPI-Support WHYMPI-Distracting	1.000 .683* .632*	.000 .121 .123	. 874 . 597 . 553		
<u>Depression</u>	••.				
BDI DACL	1.000 <del>a</del> .684*	.000	.770 .527		
<u>Pain</u>					
WHYMPI-Pain Severity PAINCARDS MPQTOTAL	1.000 <del>2</del> .641* .565*	.000 .116 .119	.888 .569 .502		

a Parameter fixed at indicated value during estimation.



<sup>\*</sup> p < .001.

# STANDARDIZED COEFFICIENTS FOR THE LATENT VARIABLES



 $\chi^2$ (73) = 88.10, p = ns Bentler & Bonett's  $\Delta$  = .85 R<sup>2</sup> for Depression = .681

