Hypochondriasis and Somatosensory Amplification

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A self-report questionnaire completed by 177 out-patients showed that hypochondriasis and amplification had a zero-order correlation of 0.56, and in stepwise multiple regression amplification accounted for 31% of the variance in hypochondriasis, after sociodemographic variables had been accounted for. Fears of ageing and death, and a childhood history of illness in the family, increased the R^2 to 0.50. Amplification was more powerful in women than in men and was also a significant (although weaker) correlate of somatisation, explaining 12% of the variance. Somatisation also correlated with being female, the propensity to seek medical care, and a diminished sense of efficacy over one's health. Our findings are consistent with the possibility that somatosensory amplification occurs in hypochondriasis.

While hypochondriasis has often been conceptualised from psychodynamic, interpersonal, and behavioural perspectives, it can also be understood as a disorder of perception and cognition (Barsky & Klerman, 1983). DSM–III–R (American Psychiatric Association, 1987) in fact emphasises the hypochondriac's faulty "interpretation of physical signs and sensations as evidence of physical illness". According to this model, hypochondriacs amplify benign somatic sensations and misattribute them to serious disease. Hypochondriacs, then, may experience a broad range of somatic sensations as more intense, more noxious, more ominous, and more disturbing than non-hypochondriacs. They may be unusually sensitive to, and intolerant of, bodily sensations in general, and be more likely to attribute them to disease.

The hypochondriac may amplify a wide range of sensations: normal physiological functions; the benign symptoms of trivial and self-limited ailments; the somatic concomitants of emotional arousal; as well as the symptoms of serious disease. He or she may thus be characterised as having an amplifying perceptual style entailing a hypervigilance or heightened attentional focus upon bodily sensation, a tendency to select out and attend to certain relatively weak and infrequent bodily sensations, and a propensity for responding to them with affect and cognitions which make them more disturbing and intense.

Because his or her bodily sensations are so intense and so disturbing, the hypochondriac then tends to misinterpret them and incorrectly attribute them to a serious disease rather than to a more benign cause such as overwork, insufficient sleep, inadequate exercise, etc. Thus, for example, a normal irregularity in the consistency of breast tissue is mistaken for a 'lump', or a stomach-ache is attributed to an ulcer rather than to a recent dietary indiscretion. Having hypothesised that he or she is sick, the hypochondriac becomes increasingly apprehensive and hypervigilant. This heightens somatic symptoms further, via two mechanisms. Firstly, people screen their somatic perceptions and selectively attend to those that confirm their explanatory hypotheses, while ignoring sensory input that disconfirms their beliefs (Pennebaker, 1982). Secondly, the individual's mounting anxiety produces its own set of somatic symptoms, which are actually benign, but are incorrectly ascribed to the presumed serious disease. The result is a further heightening of alarm, and a vicious cycle ensues.

In this conceptualisation of hypochondriasis, the other clinical characteristics of the disorder are viewed as secondary phenomena. Given the hypochondriac's somatic experience, he or she quite understandably becomes obsessed with health and preoccupied with his or her body. Given the doctor's inability to explain or treat his or her symptoms, the hypochondriac naturally becomes dissatisfied, feels antagonistic and angry about his or her predicament, and makes multiple medical visits. The hypochondriacal characteristics are, in short, the understandable consequence of the patient's disordered perceptions and cognitions.

Several findings support this conceptualisation of hypochondriasis. Firstly, college students who are more hypochondriacal have a higher level of sensory arousal and are more sensitive to paired flashes of light (Hanback & Revelle, 1978). Secondly, normal subjects who are kinaesthetic augmenters (over-estimating the size of objects placed in their hands while blindfolded), score highest on hypochondriasis scales and have a lower tolerance for experimental pain (Petrie, 1978). In work with patients who had chronic, organic, non-malignant pain, those with higher hypochondriasis scores rated their pain as more intense (Ziesat, 1978). Among patients with

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pain complaints, those with disease conviction and disease phobia have lower thresholds and tolerance to experimental pain (Merskey & Evans, 1975), and psychiatric in-patients with disease phobias were also found to have lower thresholds for, and tolerance of, experimental pain (Bianchi, 1971, 1973). However, there is, on the contrary, at least one study (Miller *et al*, 1981) which failed to find a relationship between hypochondriasis and a heightened awareness of internal bodily sensation – 'private body consciousness'.

Two studies suggest that hypochondriacs are more sensitive to normal physiological sensations. In one, workers who reported respiratory symptoms disproportionate to their pulmonary function tests were found to be more hypochondriacal (Wright *et al*, 1977). Furthermore, among psychiatric out-patients, those who were hypochondriacal were more aware of their cardiac function, determined by actual pulse rate, than those who were phobic (Tyrer *et al*, 1980).

There is also a literature, primarily in experimental and cognitive psychology rather than in clinical medicine, indicating that the cognitive processing of sensation can amplify it: the thoughts, opinions, and beliefs a person holds about his or her physical state powerfully affect his or her level of discomfort. Thus, the experimental manipulation of subjects' beliefs about the causes of their sensations has been shown to reduce distress and arousal (Rodin, 1978; Valins & Nisbett, 1971). Furthermore, misattributions of disease can result in increased symptoms: when apparently healthy individuals receive screening chest X-rays, 8% of those told that the X-ray revealed possible cardiac disease developed new symptoms referable to the heart, although their cardiac status did not change (Wheeler *et al*, 1958). In a more clinical vein, the health concerns of subjects who scored highest on a hypochondriasis scale have been judged to result from the misattribution of bodily sensations (Rodin, 1978).

In previous work (Barsky *et al*, 1986*a*), we found that four core hypochondriacal symptoms (disease fear, disease conviction, bodily preoccupation, and somatic symptoms) tend to cluster together in some ambulatory medical patients in a way consistent with the DSM–III diagnosis. In the present study, we sought evidence that the misinterpretation of benign physical signs and sensations as indicative of serious disease, also included in the DSM–III description, occurs in these patients. The study employed a self-report questionnaire measuring somatosensory amplification to determine whether amplification was related to hypochondriasis and to somatisation. Because of the preliminary nature of this work, it was clear from the outset that while this study could generate hypotheses, it would be unable to test them definitively.

Method

The study was conducted in the general medical out-patient clinic of the Massachusetts General Hospital. The clinic provides primary care to 29 000 patients, accounting for over 50 000 visits annually. It is staffed by 36 physicians and 65 house officers.

The study sample consisted of consecutive patients attending the clinic on randomly chosen days. Fluency in English and the patient's having been followed in the clinic for at least one year prior to the index visit were necessary for inclusion in the survey. The only exclusion criterion was severe organic brain disease. Of 230 patients asked to participate, 185 consented, and complete data were obtained on 177.

Subjects, aided by a research assistant, completed a 93-item self-report questionnaire while at the clinic. A small private area was provided for the purpose. This assured confidentiality and minimised the possibility that subjects seated near each other would discuss their responses.

The battery contained a questionnaire about sociodemographic characteristics; self-report scales measuring hypochondriasis, amplification, and somatisation; and a questionnaire to assess several attitudes toward health and medical care, beliefs about illness, and health practices. All scales were constructed with a five-point Likert response format from one ('strongly agree' or 'very true of me') to five ('strongly disagree' or 'not at all true of me').

As no 'gold standard' exists for the diagnosis of DSM–III–R hypochondriasis, the hypochondriacal syndrome was assessed with the Whiteley Index, a 13-item questionnaire developed by Pilowsky (1967) which focuses upon hypochondriacal attitudes and beliefs. On principal-components analysis, it yields three factors: disease conviction, disease fear, and bodily preoccupation. Its test–retest reliability has been established, it is valid in discriminating psychiatric patients diagnosed as hypochondriacal from those who are not, and there is a high concordance between the Whiteley score and spouses' ratings of hypochondriasis (Pilowsky, 1967). Whiteley Index scores are associated with several clinical characteristics thought to be secondary features of hypochondriasis, such as excessive use of medical care and complaints that are disproportionate to demonstrable pathology (Hanback & Revelle, 1978; Ziesat, 1978; Pilowsky & Spence, 1977; Beaber & Rodney, 1984; Kasteler *et al*, 1976; Wright *et al*, 1977).

Somatisation, generally defined as the tendency to express emotional dysphoria as somatic symptoms, was assessed with 11 somatic symptoms drawn from the Hopkins Symptom Check-list somatisation subscale (Derogatis *et al*, 1974, 1981), and the Minnesota Multiphasic Personality Inventory (MMPI) hypochondriasis subscale. Typical symptoms queried include backache, itching, 'upset stomach', palpitations, numbness, and fatigue. While any of these symptoms can be caused by a serious medical disorder, they are also common 'functional' complaints. Prior work with this scale has disclosed a test–retest reliability of 0.86 in 75 patients over an interval of 1–5 weeks.

Amplification was measured with a five-item self-report instrument which asks about the respondent's sensitivity to a range of normal bodily sensations ("I am quick to sense

the hunger contractions of my stomach'' and "I'm often aware of various things happening in my body") and to neutral and noxious stimuli ("Loud noises really bother me", "I am very aware of changes in my body temperature", and "I cannot stand pain as well as most people can"). These items, scored on the five-point Likert scale, were selected from an item pool composed of unpleasant and uncomfortable sensations volunteered by medical out-patients. A similar item on bodily awareness was eliminated from the Whiteley Index to obviate the problem of covariance between the two scales. Two others are similar to items in Miller's Body Consciousness Questionnaire (Miller *et al*, 1981). Its psychometric properties are the subject of a separate report; in this study, a ten-item version of the scale had a test–retest reliability of 0.85 (coefficient of reproducibility) when administered to 52 medical out-patients over an interval of 28 to 146 days (mean = 72 days), and an internal consistency of 0.70 (Cronbach's alpha).

Attitudes to health, illness and medical care were assessed on a 50-item questionnaire focusing on fears of ageing and death, a feeling of somatic vulnerability to illness and injury, the value placed on health, the propensity to seek medical care, a sense of personal efficacy over one's own health, and a childhood history of illness in the family. These items have face validity and were selected in preliminary work from a 91-item pool composed of common patient responses to questions about each of these areas. This approach was adopted because scales of established reliability and validity do not exist to assess most of these attitudes.

Product moment correlations were performed to determine the association between two variables. Stepwise multiple regression was done to determine the proportion of the variance of a dependent variable accounted for by independent or predictor variables. Whenever a variable enters the regression equation, its incremental contribution to the variance is independent of the variables that have entered in preceding steps.

Results

The mean age of the sample was 54.2 years (s.d. 17.4, range 18–86). Sixty-two per cent (109 patients) were women and 92% were white. Religious affiliation was given as 61% Catholic, 23% Protestant, 6% Jewish, and 10% other. With regard to marital status, 54% were married, 33% were single, divorced or separated, and 13% were widowed. A total of 14% were in social classes I and II, 31% in class III, 27% in class IV, and 28% in class V (Hollingshead & Redlich's (1958) two-factor index of social position).

We first examined the psychometric properties of the instruments used. The intra-scale consistency of the 13-item Whiteley Index was 0.84 (Cronbach's alpha) and that of the 11-item somatisation scale was 0.79. The amplification scale, in part because it was composed of only five items, had a Cronbach's alpha of 0.45. The Cronbach's alpha values of the other subscales were as follows: the propensity to seek medical care (four items) = 0.66; the value placed on health (four items) = 0.53; fear of ageing and death (three items) = 0.43; somatic vulnerability (three items) = 0.36; and sense of efficacy over health (three items) = 0.30.

We then studied the relationship between hypochondriasis and amplification. Their zero-order correlation coefficient was 0.56 ($P = 0.0001$). A stepwise multiple regression, employing hypochondriasis as the dependent variable and including all the study patients, is presented in Table I. The demographic variables, age, sex, and married/not married were forced into the regression model. The stepwise procedure then entered variables in the order of their contribution to the total variance. None of the three demographic variables was significantly associated with hypochondriasis ($R^2 = 0.6\%$). Amplification is the single most powerful predictor of hypochondriasis, increasing R^2 from 0.6% to 31%, followed by fear of ageing and death, and childhood history of illness which raised the R^2 to 50%. The interaction term, sex × married/not married, was significant and further increased R^2 to 53%. The coefficient of this term is negative, indicating a differential effect of marriage on men and women. With the inclusion of the interaction term, sex and married/not married become statistically significant. The next independent variable to enter the equation was the value placed on health, but it did not increase R^2 significantly. Other variables which did not contribute significantly were attitude toward utilisation and the interaction terms sex × childhood history and sex × amplification.

When we examine men and women separately (Table II), the differential influence of married/not married is apparent – among men, not being married (single, widowed or divorced) is positively associated with hypochondriasis, while among women being married is related to hypochondriasis. Further, amplification is somewhat more powerful in women, accounting for 34% of the variance in hypochondriasis.

Separate regression analyses of three of the principal symptoms of DSM–III–R hypochondriasis (there is a Whiteley Index subscale for each) reveal that amplification plays a lesser role in bodily preoccupation than in disease fear or disease conviction (Table III) while fear of ageing and death appears more closely related to bodily preoccupation than to the other two symptoms

We also examined the relationship between amplification and somatisation. The zero-order correlation between hypochondriasis and somatisation was 0.58 ($P < 0.0001$), and between amplification and somatisation was 0.44 ($P < 0.0001$). The results of a stepwise regression against

TABLE I
Correlates of hypochondriasis, in multiple stepwise regression (n = 177)

Step	Variable	b[1]	R²	P[1]
0	Age	0.006		0.78
	Sex	0.39	0.006	0.0027
	Marital status	0.34		0.012
4	Amplification	0.41	0.31	0.0001
5	Fear of ageing/death	0.29	0.45	0.0001
6	Childhood history of illness	0.13	0.50	0.0001
7	Sex × marital status	−0.56	0.53	0.001

1. *P* and *b* values are those obtained at the last step.

TABLE II
Correlates of hypochondriasis by sex, in multiple stepwise regression (n = 177)

	Men (n = 68)					Women (n = 109)			
Step	Variable	b	R^2	P	Step	Variable	b	R^2	P
1	Fear of ageing/death	0.26	0.28	0.0003	1	Amplification	0.43	0.34	0.0001
2	Amplification	0.31	0.44	0.0017	2	Fear of ageing/death	0.30	0.45	0.0001
3	Childhood history	0.18	0.49	0.0018	3	Childhood history	0.11	0.50	0.0016
4	Marital status	0.39	0.55	0.0059	4	Marital status	−0.20	0.52	0.04

TABLE III
Correlates of hypochondriacal symptoms in multiple stepwise regression (n = 172[1])

Step	Variable	b	R^2	P
Disease conviction				
1	Amplification	0.31	0.16	0.0001
2	Childhood history	0.17	0.25	0.0001
3	Fear of ageing/death	0.25	0.32	0.0003
Disease fear				
1	Amplification	0.33	0.18	0.0001
2	Fear of ageing/death	0.23	0.26	0.0003
3	Age	0.01	0.28	0.0054
4	Sex	0.30	0.30	0.0059
5	Childhood history	0.08	0.32	0.0421
Bodily preoccupation				
1	Fear of ageing/death	0.43	0.14	0.0001
2	Childhood history	0.18	0.19	0.0056
3	Amplification	0.34	0.23	0.0062
4	Sex	−0.38	0.24	0.0372

1. Program could not analyse data for 5 of the 177 subjects.

TABLE IV
Correlates of somatisation in multiple stepwise regression

Step	Variable	b[1]	R^2	P[1]
0	Age	0.01		0.0014
	Sex	−0.89	0.11	0.0001
	Marital status	−0.17		0.0662
4	Propensity to seek medical care	0.32	0.31	0.0001
5	Amplification	0.30	0.43	0.0001
6	Sense of efficacy	0.26	0.48	0.0001
7	Sex × childhood history	0.13	0.51	0.0005
8	Fear of ageing/death	0.12	0.53	0.0118

1. b and P values are those obtained at the last step.

somatisation are presented in Table IV. Age, sex and married/not married were forced into the equation; only sex was significantly associated with somatisation – the coefficient is negative, indicating that female gender is associated with somatisation. The R^2 is 11%. The propensity to seek medical care raises R^2 to 31%; with the inclusion of this variable, age becomes a significant predictor of somatisation, indicating that when the tendency to seek medical care is controlled for, age and somatisation are positively correlated. Amplification enters next, raising R^2 to 43%, followed by a diminished sense of efficacy over health. The interaction term, sex × childhood history of illness, is significant with a positive coefficient, indicating an association in women, but not in men, which further raises R^2 to 51%. A small but significant incremental contribution is made by fears of ageing and death. Variables eligible for inclusion which failed to meet the criterion for inclusion ($P \leq 0.05$) were: the interaction term, sex × greater value placed on health (more marked among women than men), physical fitness, and the interaction terms sex × amplification and sex × married/not married.

Discussion

This work suggests that there is a relationship between amplification, as measured by a brief self-report questionnaire, and hypochondriacal attitudes; and between amplification and the bodily complaints characteristic of somatisation. Amplification explains 31% of the variance in hypochondriasis and 12% of the variance in somatisation, after sociodemographic descriptors have been taken into account. Since hypochondriasis was assessed only with a questionnaire, however, these findings cannot necessarily be generalised to DSM–III–R hypochondriasis.

The term hypochondriasis has at least two different meanings. DSM–III–R refers to a distinct and discrete disorder, a primary psychopathological condition which is found in some individuals but not in most. It is thought to be a primary disorder because these symptoms tend to occur together, without other underlying psychiatric disorders of which they are a secondary feature, and to exhibit a characteristic clinical presentation, course, and response to treatment. Conviction of disease, fear of disease, and bodily preoccupation are central features of the DSM–III–R criteria as well as the hypochondriac's misattribution of non-pathological signs and sensations to serious disease. In contrast, hypochondriasis has also been viewed as a non-specific collection of symptoms (a syndrome) which are distributed more continuously throughout a

population and which are secondary features of other more pervasive psychiatric disorders (Kenyon, 1964). In this sense, hypochondriasis is analogous to dyspnoea or jaundice rather than to asthma or hepatitis.

In this paper we have discussed hypochondriasis as a primary disorder. But no criterion standard ('gold standard') exists with which to make that diagnosis. We have therefore employed a self-report questionnaire which measures the degree to which the component symptoms of hypochondriasis are present, but which does not provide a clinical diagnosis. Strictly speaking, then, our data only concern a relationship between amplification and hypochondriacal symptoms, not a relationship between amplification and DSM–III–R hypochondriasis. The two definitions of hypochondriasis, however, appear to be closely related. We previously found (Barsky *et al*, 1986*a,b*) that the component symptoms of hypochondriasis, assessed with the Whiteley Index, do tend to cluster together in a subset of ambulatory medical patients in a way which appears compatible with DSM–III–R hypochondriasis (Barsky *et al*, 1986*a,b*). To the degree that the Whiteley Index corresponds to DSM–III–R hypochondriasis, our data support the validity of DSM–III–R's emphasis upon the misattribution of non-pathological signs and sensations to serious disease. Our findings suggest that hypochondriacs are disturbed not only by the pathological symptoms of disease, but also by bodily sensations which do not suggest disease; the amplifier notices and is bothered by a range of noxious sensations that are not pathological *per se*, such as noise, heat, and hunger. The amplification questionnaire includes items about hunger and pain, and our results are therefore consistent with previous findings that hypochondriacs are more sensitive to some normal physiological sensations, and that they are less tolerant of experimental pain (Petrie, 1978; Tyrer *et al*, 1980; Wright *et al*, 1977.)

These data do not necessarily imply a causal relationship between amplification and hypochondriasis. Since the study is cross-sectional rather than longitudinal, it is not possible to determine the direction of causality. It is as possible that hypochondriacal concerns cause people to amplify bodily sensations as it is that an amplifying style leads to hypochondriasis. Another caution in interpreting the results stems from the nature of the sample. This is a medical population, and the relationship between hypochondriasis and amplification could be confounded by medical morbidity, a variable we did not assess. Zonderman *et al* (1985) pointed out the danger of concluding that a patient's fears and concerns about disease are hypochondriacal without assessing his or her medical status at the same time. It is possible, for example, that medical morbidity itself causes both hypochondriacal symptoms and amplification. Our previous work, however, suggests that hypochondriacal symptoms, as measured by the Whiteley Index, are not significantly related to the number of major medical diagnoses that a patient carries (Barsky *et al*, 1986*a*). A third caveat in interpreting these findings stems from the possible effect of other concurrent psychiatric morbidity (particularly depressive and anxiety disorders) which is prevalent in hypochondriasis, and was not assessed in this study. It is possible that amplification is a function of these other disorders, or even of psychiatric disorder in general, rather than being more specifically related to hypochondriasis. Further work is necessary to elucidate these relationships.

Amplification appears to be somewhat more important in women, and less closely related to bodily preoccupation than to the fear and conviction of disease. The gender difference is compatible with many reports that women report more somatic symptoms and more minor symptoms than men do (Pennebaker, 1982). We did not, however, find amplification directly related to gender: the zero-order correlation between sex and amplification was not statistically significant. Furthermore, no previous work suggests that female hypochondriacs have a markedly different clinical presentation from that of males.

The regression equations also point to a relationship between hypochondriasis and both fears of death and childhood experiences of illness ("a lot of illness in my family when I was growing up"). Both of these findings are consistent with previous work. Kellner (1986), for example, reported a heightened fear of death and dying among hypochondriacs. A childhood history of illness was measured with only a single item in this study, and it is a variable that is highly susceptible to retrospective falsification: the hypochondriac's frequent illness experience in adulthood may colour his or her memory of childhood. One prospective study, however, has linked childhood experiences to subsequent amplification, finding an association between maternal over-attentiveness to benign illness in childhood and bodily hypervigilance in adulthood (Mechanic, 1980). This is also consistent with the general impression in the literature that during their childhoods, hypochondriacs are frequently exposed to medical illnesses and functional symptoms in family members (Kellner, 1986; Barsky, 1983, 1988).

The data also suggest that amplification may be related to the more general process of somatisation. Amplification explained a modest but significant

proportion of the variance in somatisation; therefore, it may not be a unique correlate of hypochondriasis that distinguishes it from somatisation in general. However, it must be reiterated that this is a medical population, and we did not assess the degree to which these bodily complaints reflect medical disease rather than somatised dysphoria.

The concept of amplification deserves further investigation, particularly in studies which control for medical morbidity and other concurrent psycho-pathology. It is important to demonstrate the convergent validity of the self-report questionnaire with perceptual tests such as cortical evoked potentials. In addition, the state versus trait nature of amplification deserves further investigation. Once the concept is on firmer ground, then further studies of its clinical role in somatoform disorders and in somatised depression and anxiety disorders would be in order.

References

AMERICAN PSYCHIATRIC ASSOCIATION (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM-III-R). Washington, DC: APA.

BARSKY, A. J. (1988) Somatoform disorders. In *Comprehensive Textbook of Psychiatry* (5th edn) (eds. H. I. Kaplan & B. J. Sadock). Baltimore: Williams & Wilkins.

—— & KLERMAN, G. L. (1983) Overview: hypochondriasis, bodily complaints, and somatic styles. *American Journal of Psychiatry*, **140**, 273–283.

——, WYSHAK, G. & KLERMAN, G. L. (1986a) Hypochondriasis: an evaluation of the DSM-III criteria in medical out-patients. *Archives of General Psychiatry*, **43**, 493–500.

——, —— & —— (1986b) Medical and psychiatric determinants of out-patient medical utilization. *Medical Care*, **24**, 548–560.

BEABER, R. J. & RODNEY, W. M. (1984) Underdiagnosis of hypochondriasis in family practice. *Psychosomatics*, **24**, 39–45.

BIANCHI, G. N. (1971) Origins of disease phobia. *Australian & New Zealand Journal of Psychiatry*, **5**, 241–257.

BIANCHI, G. N. (1973) Patterns of hypochondriasis: a principal components analyses. *British Journal of Psychiatry*, **122**, 541–548

DEROGATIS, L. R., LIPMAN, R. S., RICKELS, K., et al (1974) The Hopkins symptom checklist (HSCL): a self-report symptom inventory. *Behavioral Science*, **19**, 1–15.

DEROGATIS, L. R., RICKELS, K., UHLENHUTH, E. H., et al (1981) The Hopkins symptom checklist. In *Psychological Measurements in Psychopharmacology* (eds P. Pichot & R. Oliver-Martin), pp. 79–110. Basel: Karger.

HANBACK, J. W. & REVELLE, W. (1978) Arousal and perceptual sensitivity in hypochondriasis. *Journal of Abnormal Psychology*, **87**, 523–530.

HOLLINGSHEAD, A. B. & REDLICH, F. C. (1958) *Social Class & Mental Illness: A Community Study*. New York: Wiley.

KASTELER, J., KANE, R. L., OLSEN, D. M., et al (1967) Issues underlying prevalence of "doctor-shopping" behavior. *Journal of Health and Social Behavior*, **17**, 328–339.

KELLNER, R. (1986) *Somatization and Hypochondriasis*. New York: Praeger.

KENYON, F. E. (1964) Hypochondriasis: a clinical study. *British Journal of Psychiatry*, **100**, 478–488.

MECHANIC, D. (1980) The experience and reporting of common physical complaints. *Journal of Health and Social Behavior*, **21**, 146–155.

MERSKEY, H. & EVANS, P. R. (1975) Variations in pain complaint threshold in psychiatric and neurological patients with pain. *Pain*, **1**, 73–79.

MILLER, L. C., MURPHY, R. & BUSS, A. H. (1981) Consciousness of body: private and public. *Journal of Personality and Social Psychology*, **41**, 397–406.

PENNEBAKER, J. W. (1982) *The Psychology of Physical Symptoms*. New York: Springer-Verlag.

PETRIE, A. (1978) *Individuality in Pain and Suffering* (2nd edn). Chicago: University of Chicago Press.

PILOWSKY, I. (1967) Dimensions of hypochondriasis. *British Journal of Psychiatry*, **113**, 89–93.

PILOWSKY, I. & SPENCE, N. D. (1977) Ethnicity and illness behavior. *Psychological Medicine*, **7**, 447–452.

RODIN, J. (1978) Somatopsychics and attribution. *Personality and Social Psychology Bulletin*, **4**, 531–540.

TYRER, P., LEE, I. & ALEXANDER, J. (1980) Awareness of cardiac function in anxious, phobic and hypochondriacal patients. *Psychological Medicine*, **10**, 171–174.

VALINS, S. & NISBETT, R. E. (1971) Attribution processes in the development and treatment of emotional disorder. In *Attribution: Perceiving the Causes of Behavior* (eds E. E. Jones, D. K. Karouse & H. H. Kelley). Morristown, NJ: General Learning.

WHEELER, E. O., WILLIAMSON, C. R. & COHEN, M. E. (1958) Heart scare, heart surveys, and iatrogenic heart disease. *Journal of the American Medical Association*, **167**, 1096–1102.

WRIGHT, D. D., KANE, R. L., OLSEN, D. M., et al (1977) The effects of selected psychosocial factors on the self-reporting of pulmonary symptoms. *Journal of Chronic Disease*, **30**, 195–206.

ZIESAT, H. A. (1978) Correlates of the tourniquet ischemia pain ratio. *Perceptual and Motor Skills*, **47**, 147–150.

ZONDERMAN, A. B., HEFT, M. W. & COSTA, P. T. (1985) Does the Illness Behavior Questionnaire measure abnormal illness behavior? *Health Psychology*, **4**, 425–436.

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