PSYCHOSOCIAL RISK FACTORS FOR UPPER RESPIRATORY INFECTIONS: EFFECTS OF UPPER RESPIRATORY ILLNESS ON ACADEMIC PERFORMANCE IN U.S. NAVY BASIC TRAINING*

R. R. VICKERS, JR.
L. K. HERVIG

REPORT NO. 89-14

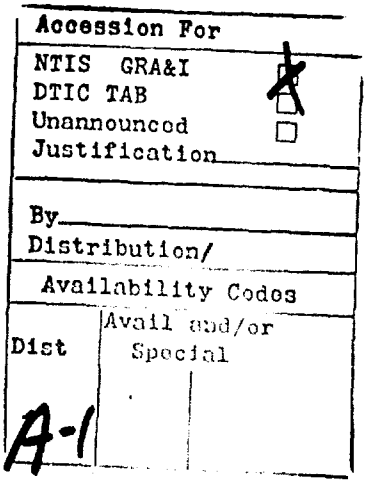
NAVAL HEALTH RESEARCH CENTER
P.O. BOX 85122
SAN DIEGO, CALIFORNIA 92138

NAVAL MEDICAL RESEARCH AND DEVELOPMENT COMMAND
BETHESDA, MARYLAND

Psychosocial Risk Factors for Upper Respiratory Infections: EFFECTS OF UPPER RESPIRATORY ILLNESS ON ACADEMIC PERFORMANCE IN U. S. NAVY BASIC TRAINING*

Ross R. Vickers, Jr.
Linda K. Hervig

Operational Performance Department
Naval Health Research Center
P. O. Box 85122
San Diego, CA 92138-9174



*Report No. 89-14 was supported by the Naval Medical Research and Development Command, Navy Medical Command, Department of the Navy under Research Work Unit MR04101.00A-6004. The views presented are those of the authors and do not reflect the official policy of the Department of the Navy, Department of Defense, nor the U. S. Government.

SUMMARY

Infectious diseases are commonplace in military populations during training and deployment for operational missions. Laboratory studies involving controlled inoculations with viruses indicate that such diseases impair basic mental functions, but little is known about the effects of naturally-ocurring infections on performance of real-world tasks. The present study, therefore, investigated the effects of naturally-ocurring upper respiratory illnesses (URI) on academic performance in U. S. Navy recruit training. It was hypothesized that illness would have a significant negative impact on performance measured a week later.

The study hypothesis was tested in a sample of U. S. Navy recruits (n = 123) who completed a symptom checklist at weekly intervals during basic training. Symptom complaints were scored for URI and for a measure of general tendency to report symptoms (General Symptom Reporting, GSR). Performance measures were provided by scores on academic tests administered as part of the training program. A measure of general intellectual ability was provided by Armed Forces Qualifying Test (AFQT) scores.

A hypothesized causal model was constructed with four focal hypotheses: (1) URI causes poorer performance a week later. (2) General distress, measured by GSR, causes poorer performance a week later. (3) Poor performance causes illness a week later. (4) The effects of URI cannot be explained by associations between URI and GSR or past performance. A structural model to test these hypotheses was developed using LISREL procedures. This model also included effects of AFQT on performance, because it was believed that the effects of illness on performance would be most evident as a deviation from the level of performance expected on the basis of ability.

The analyses confirmed the hypothesis that URI influenced subsequent performance (beta = -.14; t = -2.27). This association was independent of GSR and prior performance. GSR did not influence subsequent performance and poor performance did not predict subsequent illness severity. The estimated effect of URI on performance was half as large as that of general ability on performance.

Respiratory infections caused poorer performance in at least one military training setting. The inference that a true causal effect was

present is supported by the longitudinal character of the study design, the absence of effects of GSR (thereby ruling out physical expressions of psychological distress as an alternative causal explanation), prior evidence that URI symptom reports are valid indicators of infection, and prior experimental research showing that viral infections cause poorer performance on basic cognitive functions. Combined with epidemiological evidence that infections are very common in troops deployed for military training and operations, the cumulative evidence that URI affects mental performance gives good reason to believe that infectious disease should be considered in models designed to predict the performance of military personnel.

INTRODUCTION

The common cold is the most frequent type of illness in the U. S. population, particularly among younger individuals (Verbrugge, 1986). The significance of this fact may be underestimated, because colds commonly are mild, self-limiting illnesses that may not even appear to restrict normal activity. However, the cumulative effects of even mild illness can be significant when estimated in terms of economic losses, because the illness is so widespread. In one estimate, these losses exceed those associated with trauma and coronary disease, despite the more widely accepted economic significance of those illnesses (Harlan, et al., 1986). Even these substantial losses may underestimate the effects of the common cold, because they were based largely on time lost from work. Decrements in performance no doubt occur in individuals who are ill, but chose to continue working. If the performance of these individuals suffers as a result of their illness, the losses in productivity should be included in attempts to estimate the cost of the common cold. The present study was an initial attempt to estimate the effects, if any, of naturally-occurring colds on mental performance in a real-life learning setting.

Prior research provides reason to believe that individuals who are ill, but still working, will perform less well than they would if they were completely healthy. Decrements in mental performance are very likely given the results of studies employing experimentally-induced viral infections (Coates & Kirby, 1982; Smith, et al., 1988; Smith, Tyrrell, Coyle & Willman, 1987); the degree to which these laboratory findings will generalize to other settings is uncertain at this time. One study of naturally-occurring colds indicated that students who developed a cold after a pre-test session showed highly significant decreases in performance on brief tests of reading comprehension, oral comprehension, and visual perception while students who did not develop a cold showed a trend toward improvement on the tests (Heazlett & Whaley, 1976).

The available evidence suggests that viral infections could influence learning, but such effects have not been directly demonstrated. Even Heazlett and Whaley's (1976) study apparently did not measure actual classroom performance, so it remains uncertain whether illness-based impairment of basic mental capacities produces a cumulative effect on actual classroom test performance, the level typically of interest to the

individual and his or her organization. Although an individual's performance on specific mental tests may be impaired by illness, the net effect on performance could be negligible if the effects of illness could be offset by increased effort or other factors. Thus, it is desirable to know whether illness influences performance of socially meaningful tasks in a setting where successful performance is significant for the individual. The present study provided an initial evaluation of these effects by examining the effects of upper respiratory illness (URI) on the academic performance of U. S. Navy recruits.

The study of academic performance in recruits provided a suitable setting and population for the research for several reasons. First, academic performance is emphasized in this setting, and recruits should see it as a means to success in the service, which presumably is a personally important end state. Second, illness occurs at high rates (Arlander, Pierce, Edwards, Peckinpaugh & Miller, 1965; Edwards & Rosenbaum, 1971), but with a wide range of severity (Vickers & Hervig, 1988), thereby providing naturally-occurring differences in the predictor variable of interest. Third, academic tests are scheduled at regular intervals, thereby permitting the application of longitudinal panel designs to assess plausible causal relationships.

Although the effect of URI on performance was the primary focus of the study, several other possibilities had to be considered in developing a model to test for these effects. One possibility was that apparent effects of URI on performance could be obtained because symptom report measures of URI are contaminated by psychological reactions to basic training which take the form of somatisized distress or situational hypochondriasis. If so, URI-performance associations would be spurious. This possibility was a real concern despite a wide range of findings indicating the validity of URI symptom reports, including correlations to clinical ratings (Roden, 1958; Totman, Reed & Craig, 1977), pre-existing antibody levels (Broadbent, Broadbent, Phillpotts & Wallace, 1984; Reed, 1984; Totman, Kiff, Reed & Craig, 1980), biochemical markers of susceptibility to illness (Jemmott, 1987; Lytle & McNamara, 1967; Lytle, Rytel & Edwards, 1966; Rossen, et al., 1970; Yodfat & Silvain, 1977), biochemical markers for pathophysiology of infection (Naclerio, et al., 1988), and response to therapeutic agents designed specifically to eliminate URI symptoms (Howard, et al., 1979).

Doubts remain, because prior studies have shown that URI reports correlate with general symptom reporting tendencies that may indicate a response to stress (Vickers & Hervig, 1988).

A second possibility of interest was that poor performance was a source of psycnological stress. If so, poor performance at one time might increase the likelihood of or severity of subsequent illness by producing immunosuppression (Jemmott & Locke, 1984) and, at the same time, impair subsequent performance by lowering morale and motivation. Here again, there was the potential for spurious associations. Also, demonstrating a stress effect on illness was of interest in its own right, because it would indicate one impact of stress in basic training.

The preceding considerations led to the development of a model which involved causal effects of performance on URI and general symptomatic distress and vice versa as well as effects of general symptomatic distress on URI. The predictive model constructed to test for the associations between URI and performance, therefore, included controls for these competing hypotheses when testing the assumption that URI measured at one point in time predicted poorer performance a week later.

METHOD

Sample

The sample consisted of recruits from two recruit training companies (n = 123) who volunteered to participate in a study of predictors of susceptibility to infectious disease in basic training with the understanding that their performance during training would be obtained from training records. The typical recruit in the sample was 19.2 (S.D. = 2.7, range = 17-33) years of age. The typical recruit had a high school diploma (92%) or Graduate Equivalence Diploma (2%), but a small minority had failed to complete high school (6%). The ethnic composition of the sample was 72% Caucasian, 12% Black, 6% Hispanic; 9% of the recruits gave some other response when asked to indicate ethnic group membership.

Illness Measures

Illness measures were obtained from self-reported symptoms or complaints obtained immediately after the first, second, and third academic tests in training. The URI measure was comprised of 8 symptoms (sore

throat, fever, hoarseness, stuffed-up nose, productive cough, non-productive cough, sinus pain, and sneezing) selected as the best URI indicators of URI in this population and setting on the basis of an extensive evaluation of a wide range of potential URI symptoms (Vickers & Hervig, 1988). The raw score derived by averaging the reported severity of these 8 symptoms was adjusted for the possible effects of other concurrent illnesses to ensure, insofar as possible, that the symptoms were specific to URI that was presumably of viral origin as described elsewhere (Vickers & Hervig, 1988).

A measure of general symptom reporting tendencies also was scored from the symptom reports. Scores on this measure were determined by the severity ratings of four symptoms, skin irritation, vomiting, diarrhea, and trouble hearing. These symptoms are infrequent in the population studied and only moderately correlated relative to symptoms of a well-defined illness syndrome such as URI. For these reasons, General Symptom Reporting (GSR) scores have been interpreted as indications of hypochondriasis or symptom reporting as a form of psychological distress. General Symptom Reporting was used in the present study to test the possibility that any significant effects of URI on academic performance were the product of psychological processes affecting symptom reporting rather than to true illness.

General Ability Measure

Armed Forces Qualifying Test (AFQT) scores were available from training records. This test measures overall mental ability and was used in this study to adjust performance for pre-existing ability differences. This adjustment was judged appropriate, because illness effects should be most evident as deviations from typical performance, and AFQT was the best guide available for estimating typical expected performance.

Academic Performance Measures

Academic performance was measured by scores on standardized tests administered as part of the recruit training curriculum at the end of the second, third, and fourth weeks of training. The first test dealt with the mission and structure of the Navy and the authority and responsibilities of individuals of different ranks. The second test dealt with first aid topics and basic seamanship. The third test dealt with damage control and accident prevention aboard ship. Each test was comprised of 50 multiple-choice items which were administered by projecting the item on a screen for a fixed period of time to control test taking rate. Recruits marked optical

scanning sheets to indicate their choice of an answer from among the options provided.

Analysis Procedures

A general predictive model was constructed which embodied several specific predictions derived from considerations outlined in the introduction. The predictions can be stated in terms of a set of focal hypotheses dealing with the potentially reciprocal relationships between illness and performance and a set of auxiliary hypotheses concerning other relationships between the variables measured in this study. The four focal hypotheses listed below represented the central research concerns:

Hypothesis 1: URI measured at one point in training will predict poorer academic performance the next week (Illness Hypothesis).

Hypothesis 2: General distress measured at one point in training will predict poorer academic performance the next week (Distress Hypothesis).

Hypothesis 3: Although general distress will be associated with higher URI reporting, the hypothesized URI-performance relationship will exist independent of these effects (Contamination Hypothesis).

Hypothesis 4: Poor academic performance at one point in training will increase subsequent illness, including both URI and general distress (Stress Hypothesis).

The four focal hypotheses were augmented by two auxiliary assumptions to produce a complete predictive model capable of reproducing the overall covariation matrix for the study measures. Although these assumptions constitute hypotheses in their own right, they have been labeled "assumptions" to distinguish them from the central issues for this paper. The additional assumptions were:

Assumption 1: General ability will be positively related to academic performance, but will not be related to illness or distress (Ability Assumption).

Assumption 2: Illness and performance scores measured at one time will predict comparable scores the next week (Autocorrelation Assumption).

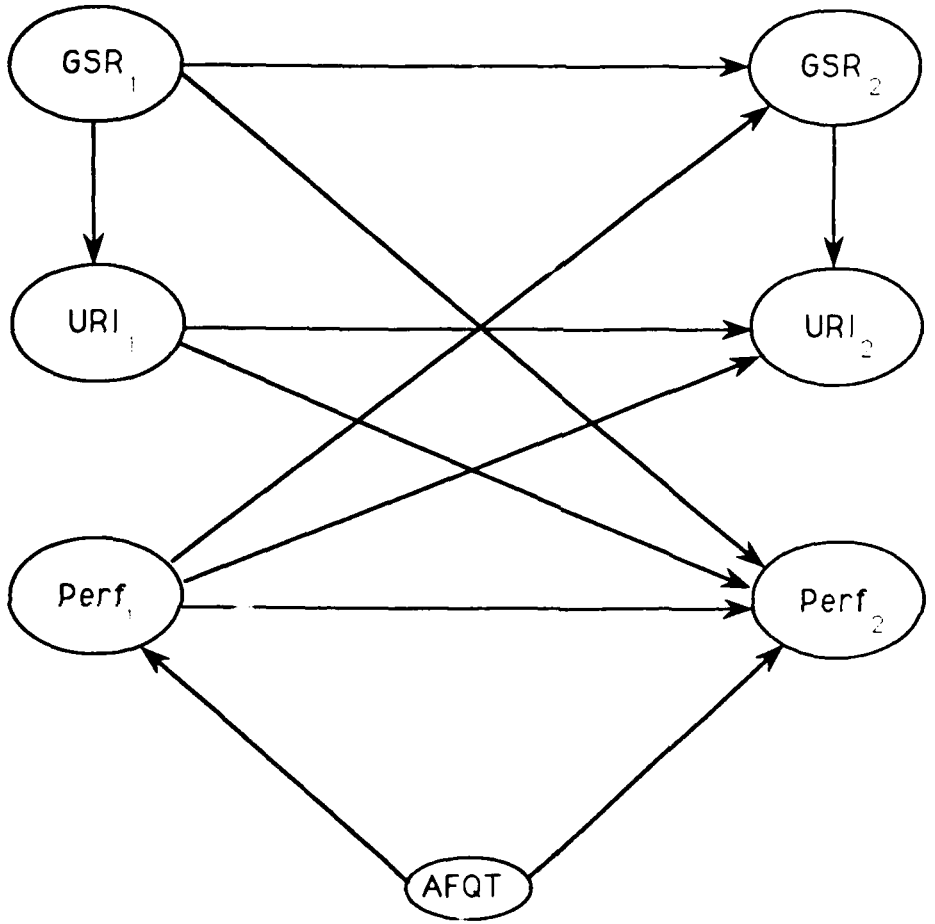


Figure 1

Initial Hypothesized Effects For One Time Interval

Note. GSR=General Symptom Reporting, URI= Upper Respiratory Illness; Perf=Performance, AFQT=Armed Forces Qualifying test. Arrows indicate hypothesized causal effects. Subscript refer to time of measurement.

The foregoing considerations produced the model presented in Figure 1. This figure shows the hypothesized causal effects for the data gathered in conjunction with the first and second test. The full model to be tested extended this base model by assuming the same pattern of associations held for the data obtained in conjunction with the second and third tests. In addition, the model imposed the constraint that the effect of URI on performance was constant for the two time intervals, thereby using one interval as a replication of the other.

This model was evaluated by applying LISREL VI (Joreskog & Sorbom, 1981) to the data and comparing the chi-square value for the model to that obtained under the assumption of independence among the 10 measures. The metric for the latent traits was established by fixing the variance for each of these traits at 1.00. After this overall test for the goodness-of-fit of the model to the data, a revised model was developed by eliminating hypothesized effects with t-values less than 1.65 (absolute), beginning with the smallest t-value and stopping when each remaining effect involved at least one t-value greater than 1.65 (absolute). More than one t-value could be available for a given effect if there were multiple pairs of variables that could be used to test for the effect. For example, the effect of General Symptom Reporting on URI could be tested at three separate points in time and exceeded the criterion at two of these points. One exception to the general rule regarding effects to be retained in the final model was made. Lagged effects of performance on one test on the immediate following test were retained in the model despite the fact that the t-values were less than 1.65. This decision was made, because failure to include these effects produced difficulties in obtaining computational convergence.

An additional simplifying assumption used in constructing the causal model was that the causal effects described in the focal hypotheses were consistent over time. Thus, the causal effect of URI at the time of the first academic test on academic performance a week later was constrained to be equal to the causal effect of URI at the time of the second academic test on academic performance a week later. This assumption simplified the model and provided a test of the temporal generalizability of associations within a single sample. Statistical tests for the legitimacy of this assumed constancy of effect were provided initially by the modification indices for the estimates in the initial model. In addition, the final model was run

with the equality constraint removed to determine the effect of this constraint on the goodness-of-fit of the model.

RESULTS

The hypothesized model provided a good fit to the data. This model produced a chi-square of 39.62 with 29 degrees of freedom ($p < .10$) compared to 327.71 with 45 degrees of freedom for the null model ($p < .001$). The incremental chi-square was highly significant statistically (chi-square = 288.09, 16 df, $p < .001$). The Tucker and Lewis (1973) index of fit for this model, therefore, was .942, an acceptable value.

The initial model contained several hypothesized relationships that were too weak to be statistically significant ($t < .87$ (absolute) for each effect). Academic performance did not affect either illness indicator, so the stress hypothesis was not supported. General Symptom Reporting did not affect academic performance, so the distress hypothesis was not supported. General Symptom Reporting did affect URI, but somatically-expressed distress could not be a cause of spurious URI-performance associations, because General Symptom Reporting did not affect performance. Therefore, the contamination hypothesis to explain URI-performance associations was not supported.

The elimination of the three causal effects implied by the hypotheses that were not supported by the data produced the final model shown in Figure 2. This final model had a chi-square of 40.74 with 32 degrees of freedom ($p < .14$). Thus, the retention of these three effects would have only slightly improved the goodness-of-fit (chi-square = 1.12, 3 df, $p > .77$). Collectively, therefore, these three effects were clearly not required to reproduce the covariation matrix. This point was underscored by the fact that the Tucker and Lewis (1973) index of goodness-of-fit increased to .957 with these three elements eliminated from the model.

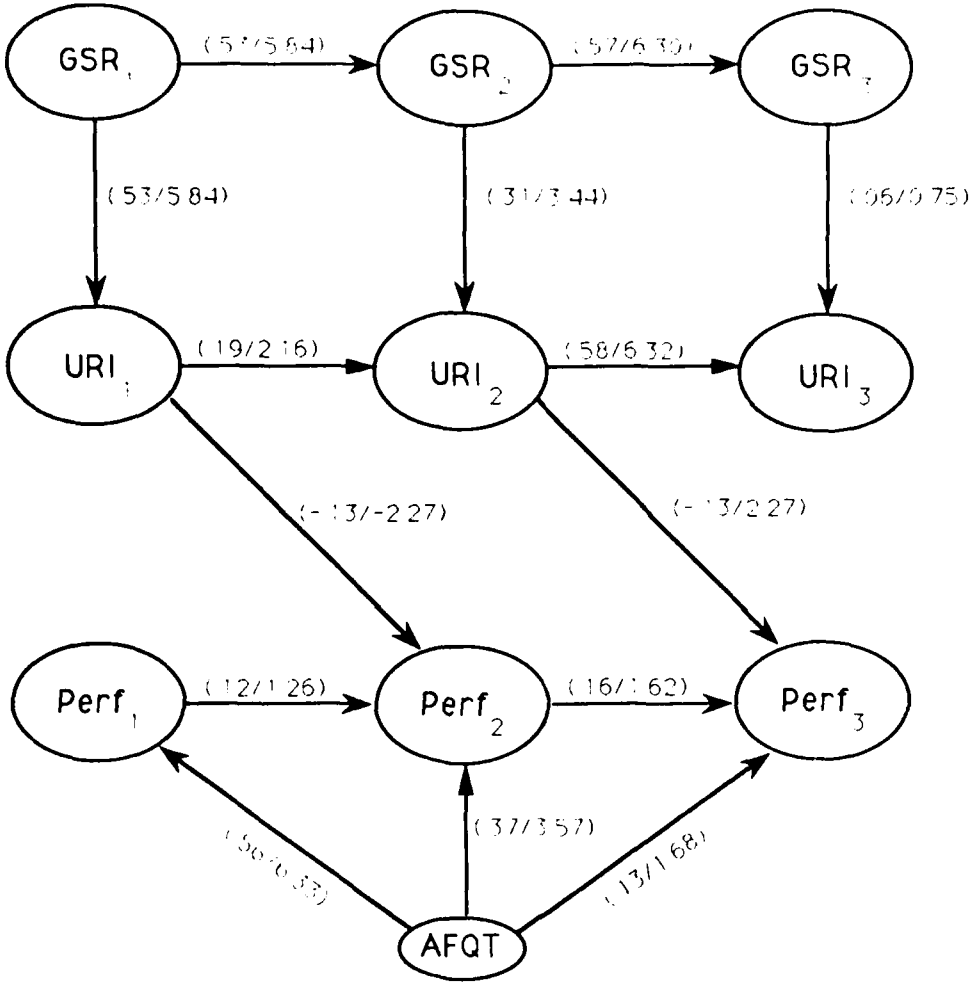


Figure 2

Final Predictive Model

Note. Arrows indicate hypothesized causal effects. Subscripts refer to time of measurement. Figures in parentheses are standardized path coefficients and t values (coeff/t)

Tests of three additional models were conducted to evaluate the appropriateness of decisions incorporated into the initial model. One additional model removed the constraint that the effects of URI on performance be equal from the second to third and third to fourth weeks of training. This model produced a slight, nonsignificant improvement in goodness-of-fit (chi-square = 0.53, 1 df, $p > .53$). The second model imposed the restriction that the effect of AFQT on performance be equal for all three tests. This constraint produced a substantially poorer fit to the data than the model which assumed unequal effects (chi-square = 13.04, 2 df., $p < .002$). The third model examined the possibility that the URI-academic performance would be stronger if the relationship were estimated for performance on the day the symptom reports were obtained rather than for performance a week later. Initial attention focused on the latter possibility to include temporal precedence of the illness as a basis for claiming a causal relationship and to help rule out plausible alternative interpretations (e.g., adverse psychological reactions to poor performance lead to greater symptom reporting). The model linking contemporaneous URI and academic performance did not fit the data as well as the one including lagged effects (chi-square = 45.28 versus 40.74).

A final check on the adequacy of the model in Figure 2 was provided by examination of the modification indices for the omitted effects. These indices indicate the minimum chi-square improvement that would result from adding the effect in question to the model (Joreskog & Sorbom, 1981). None of the modification indices for effects of URI and GSR on performance was greater than 2.08, so none of these effects would have produced a significant improvement in the model. Similarly, the largest modification index for the effects of performance on URI and GST was 3.01, a value well below the critical value for a significant improvement in fit. Thus, none of the omitted causal effects between illness and performance would have produced significant improvements in the model had they been added.

The most important feature of the final model shown in Figure 2 is the fact that URI measured the week prior to an academic test predicted lower performance on the test ($t = -2.27$) following Joreskog and Sorbom's (1981) recommendation that any model parameter with a t-value greater than 2.00 (absolute) be retained as significant. The magnitude of the effect of URI

on performance perhaps can be evaluated best by comparison to AFQT, the other hypothesized causal determinant of performance in this model. This comparison was made from the standardized solution for the causal model, because differences in metric for the AFQT and URI scores otherwise could distort the comparison. In the standardized solution, the estimated effect of AFQT on performance was .37 and .16 for the second and third tests, respectively, an average of .265. The average value for URI effects in the standardized solution was -.135, a value equal to 51% of that derived for AFQT. Thus, the effect of URI was half as large as that of AFQT, on the average.

DISCUSSION

The results were consistent with prior laboratory evidence that viral illness impairs cognitive functioning. The present findings suggest that such impairments cumulatively affect real-life performance. Stronger evidence would have been provided if basic cognitive abilities which have been shown to be affected by viral infections had been included in the study and examined as mechanisms linking illness to performance. However, the available evidence is consistent with the assumption that infectious illness produces acute reductions in basic cognitive abilities which cumulatively impair learning over the duration of the illness and provides a basis for believing more detailed studies could be of value to isolate specific impairments.

The preceding conclusion assumes a causal effect of illness on performance. That inference is justified by the longitudinal design and analysis procedures in the present study which meet two basic criteria for establishing causality, covariation of phenomena and temporal precedence of the hypothesized cause. The causal inference also was supported by the negative findings for plausible competing hypotheses, particularly the evidence that General Symptom Reporting did not affect performance. The initial model included joint effects of General Symptom Reporting on URI and subsequent performance and joint effects of past performance on subsequent URI and subsequent performance. If performance-URI relationships had been mediated by either of these mechanisms, the initial model would have produced clearly nonsignificant URI-performance associations, and this did

not occur. These negative findings help rule out the important competing hypothesis that the effects of URI were the product of purely psychological processes that led to higher symptom reporting as one manifestation of psychological distress in basic training and that the stress of prior poor performance contributed to subsequent illness and related performance deficits. Finally, a causal inference is justified by auxiliary evidence from other research. This evidence includes experimental laboratory studies which have shown that manipulation of illness status induces performance decrements (Coates & Kirby, 1982; Smith, Tyrrell, Al-Nakir, et al., 1988, Smith, Tyrrell, Coyle, et al., 1987) and evidence that symptom reports for URI are valid indicators of disease relative to a wide variety of objective criteria for the presence of infection. Indeed, from a different perspective, the present findings add further support to the claim that URI reports are objective indicators of disease. Taking the present findings in the context of this additional evidence, the effect of illness on performance can be interpreted as evidence that actual disease pathology is affecting performance.

The magnitude of the illness effects was substantial relative to one established performance predictor. This frame-of-reference for the URI effects is important, because the absolute magnitude of the effects was modest. The findings must be replicated before the estimated effects for either AFQT or URI are accepted as accurate, but these preliminary results suggest that the impact of illness on performance is sufficiently strong to merit further attention as a factor in performance prediction models whenever high rates of URI can be anticipated.

REFERENCES

Arlander, T.R., Pierce, W.E., Edwards, E.A., Peckinpaugh, R.O. & Miller, L.F. (1965). IV. An epidemiologic study of respiratory illness patterns in Navy and Marine Corps recruits. American Journal of Public Health, 55, 67-80.

Broadbent, D.E., Broadbent, M.H.P., Phillpotts, R.J. & Wallace, J. (1984). Some further studies on the prediction of experimental colds in volunteers by psychological factors. Journal of Psychosomatic Research, 28, 511-523.

Coates, G.D. & Kirby, R.H. (1982). Organismic factors and individual differences in human performance and productivity. In E.A. Alluisi & E.A. Fleishman (eds.), Human Performance and Productivity: Stress and Performance Effectiveness. Hillsdale, NJ: Erlbaum, pp. 112-125.

Edwards, E.A. & Rosenbaum, M.J. (1971). Surveillance Program: 1964-1970. Great Lakes, IL: Naval Medical Research Unit No. 4, Report RU 71.17.

Harlan, WR., Murt, H.A., Thomas, J.W., Lepkowski, J.M., Guire, K.E., Berki, S.E. & Landis, J.R. (1986). Incidence, utilization, and costs associated with acute respiratory conditions, United States, 1980. National Medical Care Utilization and Expenditure Survey. Series C, Analytical Report No. 4. DHHS Publication No. 86-20404. National Center for Health Statistics, Public Health Service. Washington: U. S. Government Printing Office, Sept., 1986.

Heazlett, M. & Whaley, R.F. (1976). The common cold: its effects on perceptual ability and reading comprehension among pupils of a seventh-grade class. Journal of School Health, 46, 145-147.

Howard, J.C., Kantner, T.R., Lilienfield, L.S., Princiotto, J.V., Krum, R.E., Crutcher, J.E., Belman, M.A. & Danzig, M.R. (1979). Effectiveness of antihistamines in the symptomatic management of the common cold. Journal of the American Medical Association, 242, 2414-2417.

Jemmott, J.B., III. (1987). Social motives and susceptibility to disease: Stalking individual differences in health risks. Journal of Personality, 55, 276-279.

Jemmott, J.B. & Locke, S.E. (1984). Psychosocial factors, immunologic mediation, and human susceptibility to infectious diseases: how much do we know. Psychological Bulletin, 95, 78-108.

Joreskog, K.G. & Sorbom, D. (1981). LISREL User's Guide. Chicago: International Educational Services.

Lytle, R.I. & McNamara, M.J. (1967). Alpha-1 globulins in those susceptible to viral acute respiratory diseases. Great Lakes, IL: Naval Medical Research Unit No. 4, December, 1967.

Lytle, R.I., Rytel, M.W. & Edwards, E.A. (1966). Correlation of immunological and biological factors in the host with susceptibility to respiratory infections. Journal of Infectious Diseases, 116, 67-74.

Naclerio, R.M., Proud, D., Lichtenstein, L.M., Kagey-Sobotka, A.K., Henley, J.O., Sorrentino, J. & Gwaltney, J.M. (1988). Kinins are generated during experimental rhinovirus colds. Journal of Infectious Diseases, 157, 133-142.

Reed, S.E. (1984). The behaviour of recent isolates of human respiratory cornavirus in vitro and involunteers: evidence of heterogeneity among 229E-related strains. Journal of Medical Virology, 13, 179-192.

Roden, A.T. (1958). Clinical assessment of the common cold. Proceedings of the Royal Society for Medicine, 51, 271-273.

Rossen, R.D., Butler, W.T., Waldman, R.H., Alford, R.H., Hornic, R.B., Togo, Y. & Kasel, J.A. (1970). The proteins in nasal secretion. II. A longitudinal study of IgA and neutralizing antibody levels in nasal washing from men infected with influenza virus. Journal of the American Medical Association, 211, 1157-1161.

Smith, A.P., Tyrrell, D.A.J., Al-Nakir, W., Coyle, K.B., Donovan, C.B., Higgins, P.G. & Willman, J.S. (1988). The effects of experimentally induced respiratory virus infections on performance. Psychological Medicine, 18, 65-71.

Smith, A.P., Tyrrell, D.A.J., Coyle, K. & Willman, J.S. (1987). Selective effects of minor illnesses on human performance. British Journal of Psychology, 78, 183-188.

Totman, R., Reed, S.E. & Craig, J.W. (1977). Cognitive dissonance, stress and virus-induced common colds. Journal of Psychosomatic Research, 21, 55-63.

Totman, R., Kiff, J., Reed, S.E. & Craig, J.W. (1980). Predicting experimental colds in volunteers from different measures of recent life stress. Journal of Psychosomatic Research, 24, 155-163.

Tucker, L.R. & Lewis, C. (1973). The reliability coefficient for maximum likelihood factor analysis. Psychometrika, 38, 1-10.

Verbrugge, L.M. (1986). From sneezes to adieux: stages of health for American men and women. Social Science and Medicine, 22, 1195-1212.

Vickers, R.R., Jr. & Hervig, L.K. (1988). Psychosocial risk factors for upper respiratory infections: Assessment of upper respiratory illness during basic training (Report No. 88-23). San Diego, CA: Naval Health Research Center.

Yodfat, Y. & Silvain, I. (1977). A prospective study of acute respiratory tract infections among children in a kibbutz: the role of secretory IgA and serum immunoglobulins. Journal of Infectious Disease, 136, 26-29.

UNCLASSIFIED
SECURITY CLASSIFICATION OF THIS PAGE

REPORT DOCUMENTATION PAGE

1a. REPORT SECURITY CLASSIFICATION (U) UNCLASSIFIED	1b RESTRICTIVE MARKINGS None
2a. SECURITY CLASSIFICATION AUTHORITY N/A	3. DISTRIBUTION / AVAILABILITY OF REPORT Approved for public release; distribution unlimited.
2b. DECLASSIFICATION / DOWNGRADING SCHEDULE N/A	
4. PERFORMING ORGANIZATION REPORT NUMBER(S) NHRC Report No. 89- 14	5. MONITORING ORGANIZATION REPORT NUMBER(S)
6a NAME OF PERFORMING ORGANIZATION Naval Health Research Center 6b OFFICE SYMBOL (If applicable) 10	7a. NAME OF MONITORING ORGANIZATION Commander, Naval Medical Command
6c. ADDRESS (City, State, and ZIP Code) P. O. Box 85122 San Diego, CA 92138-9174	7b. ADDRESS (City, State, and ZIP Code) Department of the Navy Washington, DC 20372
8a. NAME OF FUNDING / SPONSORING ORGANIZATION Naval Medical Research & Development Command 8b OFFICE SYMBOL (If applicable)	9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER
8c. ADDRESS (City, State, and ZIP Code) Naval Medical Command National Capital Region Bethesda, MD 20814-5044	10. SOURCE OF FUNDING NUMBERS PROGRAM ELEMENT NO. [illegible] PROJECT NO MR04101 TASK NO .07 WORK UNIT ACCESSION NO. 6004

11 TITLE (Include Security Classification)
Psychosocial Risk Factors for Upper Respiratory Infections: Effects of Upper Respiratory Illness on Academic Performance in U.S. Navy Basic Training

12 PERSONAL AUTHOR(S)
Vickers, Ross R., Jr, & Hervig, Linda K.

13a TYPE OF REPORT	13b TIME COVERED	14 DATE OF REPORT (Year, Month, Day)	15 PAGE COUNT
Interim	FROM _____ TO _____	1989 March 15	

16 SUPPLEMENTARY NOTATION

17 COSATI CODES		
FIELD	GROUP	SUB-GROUP

18 SUBJECT TERMS (Continue on reverse if necessary and identify by block number)
Performance, Basic training, Anatomy, Medical research, Illness, Upper respiratory infections, Navy men, URI, GSR

19 ABSTRACT (Continue on reverse if necessary and identify by block number)

Significant effects of upper respiratory illness on academic performance in U.S. Navy basic training were demonstrated. These effects were half as large as the effects of general intellectual ability, as measured by the AFQT, on performance. Combined with prior evidence from laboratory studies, the findings provide a strong basis for inferring that naturally-occurring upper respiratory infections significantly impair cognitive performance and learning. These disease effects should be considered in models to predict performance.

20 DISTRIBUTION / AVAILABILITY OF ABSTRACT	21 ABSTRACT SECURITY CLASSIFICATION
☐ UNCLASSIFIED/UNLIMITED ☒ SAME AS RPT ☐ DTIC USERS	UNCLASSIFIED

22a NAME OF RESPONSIBLE INDIVIDUAL	22b TELEPHONE (Include Area Code)	22c OFFICE SYMBOL
Vickers, Ross R., Jr.	(619) 553-8454	10

DD FORM 1473, 84 MAR 83 APR edition may be used until exhausted
All other editions are obsolete

SECURITY CLASSIFICATION OF THIS PAGE

U.S. Government Printing Office 1988-607-047