

Gulf War Syndrome

ABSTRACTS

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Research Group Identifies Immune System Anomaly that Supports Existence of Gulf War Syndrome.
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www.va.gov/health/environ/persgulf.htm.
Antibodies to squalene in Gulf War syndrome.

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Gulf War Syndrome (GWS) is a multisystemic illness afflicting many Gulf War-era veterans. The molecular pathological basis for GWS has not been established. We sought to determine whether the presence of antibodies to squalene correlates with the presence of signs and symptoms of GWS. Participants in this blinded cohort study were individuals immunized for service in Desert Shield/Desert Storm during 1990-1991. They included 144 Gulf War-era veterans or military employees (58 in the blinded study), 48 blood donors, 40 systemic lupus erythematosus patients, 34 silicone breast implant recipients, and 30 chronic fatigue syndrome patients. Serum antibodies to squalene were measured. In our small cohort, the substantial majority (95%) of overtly ill deployed GWS patients had antibodies to squalene. All (100%) GWS patients immunized for service in Desert Shield/Desert Storm who did not deploy, but had the same signs and symptoms as those who did deploy, had antibodies to squalene. In contrast, none (0%) of the deployed Persian Gulf veterans not showing signs and symptoms of GWS have antibodies to squalene. Neither patients with idiopathic autoimmune disease nor healthy controls had detectable serum antibodies to squalene. The majority of symptomatic GWS patients had serum antibodies to squalene. Copyright 2000 Academic Press.

The Importance of Replenishing Phase II Cofactors

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2000 Spring. Functional Medicine Research Center Update. Gig Harbor, WA: HealthComm International (www.healthcomm.com/research/update/back-issues/spring00/index.html).

[Toxic occupational liver diseases. Therapeutic effects of silymarin]. [Article in Italian]

Boari C, Montanari FM, Galletti GP, Rizzoli D, Baldi E, Caudarella R, Gennari P

Minerva Med 1981 Oct 20;72(40):2679-88

We considered two groups, one of 35 and the other of 20 patients, with occupational toxic hepatopathy caused by various toxic substances (mostly solvents, paints and glues). The patients were mostly suffering from chronic or subacute forms. We considered the work conditions in the anamnesis, the period of exposure and most important laboratory parameters of hepatic function checked before and after treatment with Silymarin (420 mg/die/os) for the first group and with "placebo" for the second group. Five patients of the first group were diagnosed by biopsy. The treatment with Silymarin has shown slight variations in some parameters. The therapeutic effect is probably not dependent upon the kind of pathogen noxa; it seems instead to be more evident when the exposure period is shorter. The group "placebo" does not show significant variations.

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Effect of calcium glucarate on beta-glucuronidase activity and glucarate content of certain vegetables and fruits.

Dwivedi C, Heck WJ, Downie AA, Larroya S, Webb TE. College of Pharmacy, South Dakota State University, Brookings 57007.

Biochem Med Metab Biol 1990 Apr;43(2):83-92

Glucarate is normally present in tissues and body fluids and is in equilibrium with D-glucaro-1,4-lactone, a natural inhibitor of beta-glucuronidase activity. Dietary calcium glucarate, a sustained-release form of glucarate, elevates the blood level of D-glucaro-1,4-lactone which suppresses blood and tissue beta-glucuronidase activity. A single dose of CaG (4.5 mmole/kg body weight) inhibited beta-glucuronidase activity in serum and liver, lung, and intestinal microsomes by 57, 44, 37, and 39%, respectively. A chronic administration of calcium glucarate (4% in diet) also decreased beta-glucuronidase activity in intestinal and liver microsomes. Maximal inhibition of beta-glucuronidase activity in serum was observed from 12 noon to 2:00 PM. In contrast, maximum inhibition of beta-glucuronidase activity in intestinal and liver microsomes occurred during mornings, although a secondary depression in intestinal microsomes also occurred around 4 PM. A 4% calcium glucarate supplemented diet also inhibited beta-glucuronidase activity by 70% and 54%, of the bacterial flora obtained from proximal (small intestine) and distal (colon) segments of intestine, respectively. Due to the potential effect of dietary glucarate on net glucuronidation and on other metabolic pathways, glucaric acid levels in various foods were determined. The glucaric acid content varied from a low of 1.12-1.73 mg/100 g for broccoli and potatoes to a high of 4.53 mg/100 g for oranges.

Is Gulf War syndrome due to stress? The evidence reexamined.

Am J Epidemiol 1997 Nov 1;146(9):695-703

Medical policy-makers have concluded that stress from wartime trauma and deployment constitutes an important cause of the chronic physical symptoms observed in US veterans who served in the Persian Gulf War. The author reviewed scientific articles from peer-reviewed journals referenced in the final report of the Presidential Advisory Committee on Gulf War Veterans' illnesses and conducted a MEDLINE literature search. All reported prevalence rates of post-traumatic stress disorder (PTSD) in Gulf War veterans were defined by critical cutpoints on psychometric scales constructed by summing veterans' responses on standardized symptom questionnaires rather than by clinical psychiatric interviews. Observed PTSD rates varied from 0% to 36% (mean, 9%). Correcting for measurement errors with previously determined values of the sensitivity (range 0.77 to 0.96) and specificity (range 0.62 to 0.89) of the psychometric tests yielded estimated true PTSD rates of 0% for 18 of the 20 reported rates. Mean scores on the Mississippi PTSD scale in all subgroups of Gulf War veterans were within the range of values for well-adjusted Vietnam veterans (50-89) and far below that of Vietnam veterans with psychiatrically confirmed PTSD (120-140). Most PTSD and "stress-related symptoms" reported in studies of Gulf War veterans appear to represent false-positive errors of measurement reflecting nonspecific symptoms of other conditions.

Point: Bias from the "healthy warrior effect" and unequal follow-up in three government studies of health effects of the Gulf War

Haley RW

Am. J. Epidemiol., 1998, Aug 15;148(4):315-23

No abstract available.

Is there a Gulf War Syndrome? Searching for syndromes by factor analysis of symptoms.

Haley RW, Kurt TL, Hom J Epidemiology Division, Department of Internal Medicine, University of Texas Southwestern Medical Center at Dallas, 75235-8874, USA.

JAMA 1997 Jan 15;277(3):215-22

OBJECTIVE: To search for syndromes in Persian Gulf War veterans. **PARTICIPANTS:** Two hundred forty-nine (41%) of the 606 Gulf War veterans of the Twenty-fourth Reserve Naval Mobile Construction Battalion living in 5 southeastern states participated; 145 (58%) had retired from service, and the rest were still serving in the battalion. **DESIGN:** Participants completed a standardized survey booklet measuring the anatomical distributions or characteristics of each symptom, a booklet measuring wartime exposures, and a standard psychological personality assessment inventory. Two-stage factor analysis was used to disentangle ambiguous symptoms and identify syndromes. **MAIN OUTCOME MEASURES:** Factor analysis-derived syndromes. **RESULTS:** Of 249 participants, 175 (70%) reported having had serious health problems that most attributed to the war, and 74 (30%) reported no serious health problems. Principal factor analysis yielded 6 syndrome factors, explaining 71% of the variance. Dichotomized syndrome indicators identified the syndromes in 63 veterans (25%). Syndromes 1 ("impaired cognition," characterized by problems with attention, memory, and reasoning, as well as insomnia, depression, daytime sleepiness, and headaches), 2 ("confusion-ataxia," characterized by problems with thinking, disorientation, balance disturbances, vertigo, and impotence), and 3 ("arthro-myoneuropathy," characterized by joint and muscle pains, muscle fatigue, difficulty lifting, and extremity paresthesias) represented strongly clustered symptoms; whereas, syndromes 4 ("phobia-apraxia"), 5 ("fever-adenopathy"), and 6 ("weakness-incontinence") involved weaker clustering and mostly overlapped syndromes 2 and 3. Veterans with syndrome 2 were 12.5 times (95% confidence interval, 3.5-44.8) more likely to be unemployed than those with no health problems. A psychological profile, found in 48.4% of those with the syndromes, differed from posttraumatic stress disorder, depression, somatoform disorder, and malingering. **CONCLUSION:** These findings support the hypothesis that clusters of symptoms of many Gulf War veterans represent discrete factor analysis-derived syndromes that appear to reflect a spectrum of neurologic injury involving the central, peripheral, and autonomic nervous systems.

Evaluation of neurologic function in Gulf War veterans. A blinded case-control study.

Haley RW, Hom J, Roland PS, Bryan WW, Van Ness PC, Bonte FJ, Devous MD Sr, Mathews D, Fleckenstein JL, Wians FH Jr, Wolfe GI, Kurt TL Department of Internal Medicine, University of Texas Southwestern Medical Center at Dallas, 75235-8874, USA.

JAMA 1997 Jan 15;277(3):223-30

OBJECTIVE: To determine whether Gulf War-related illnesses are associated with central or peripheral nervous system dysfunction.

DESIGN: Nested case-control study. PARTICIPANTS: Three veterans with factor analysis-derived syndromes (the cases), 10 well veterans deployed to the Gulf War (the deployed controls), and 10 well veterans not deployed to the Gulf War (the nondeployed controls). METHOD: With investigators blinded to group identities, participants underwent objective neurophysiological, audiovestibular, neuroradiological, neuropsychological, and blood tests. MAIN OUTCOME MEASURES: Evidence of neurologic dysfunction. RESULTS: Compared with the 20 controls, the 23 cases had significantly more neuropsychological evidence of brain dysfunction on the Halstead Impairment Index ($P=.01$), greater interside asymmetry of the wave I to wave III interpeak latency of brain stem auditory evoked potentials ($P=.02$), greater interocular asymmetry of nystagmic velocity on rotational testing, increased asymmetry of saccadic velocity ($P=.04$), more prolonged interpeak latency of the lumbar-to-cerebral peaks on posterior tibial somatosensory evoked potentials (on right side, $P=.03$, and on the left side, $P=.005$), and diminished nystagmic velocity after caloric stimulation bilaterally (P values range from .02 to .04). Cases ($n=5$) with syndrome 1 ("impaired cognition") were the most impaired on brain stem auditory evoked potentials ($P=.005$); those ($n=13$) with syndrome 2 ("confusion-ataxia") were the most impaired on the Halstead Impairment Index ($P=.006$), rotational testing ($P=.01$), asymmetry of saccadic velocity ($P=.03$), and somatosensory evoked potentials ($< \text{ or } =.01$); and those ($n=5$) with syndrome 3 ("arthro-myo-neuropathy") were the most impaired on caloric stimulation ($< \text{ or } =.01$). CONCLUSIONS: The 3 factor-derived syndromes identified among Gulf War veterans appear to represent variants of a generalized injury to the nervous system.

A trial of fasting cure for PCB-poisoned patients in Taiwan.

Imamura M, Tung TC.

Am J Ind Med 1984;5(1-2):147-53

Sixteen patients poisoned by ingestion of a rice oil contaminated with polychlorobiphenyls (PCBs) in Taiwan voluntarily joined a trial of fasting cure for either seven or ten days approximately 26 or 35 months after being poisoned. During fasting, mixed juice made of fresh vegetables and fruits and milk or "tohnyu," that is, boiled soybean juice, were given on a fixed schedule. All these patients showed improvements of their symptoms and signs caused by the poisoning. Some of them enjoyed a dramatic relief of their sufferings such as severe headache, lumbago, arthralgia, pain at the sole, cough, sputa, and/or acneiform eruptions. The eruptions forming abscesses or cysts were, however, hard to cure. Thus, the fasting cure was demonstrated to be effective in the treatment of the patients. PCB concentrations in blood were rather elevated during and after the fasting.

Seminars in medicine of the Beth Israel Hospital, Boston. Mitochondrial DNA and disease.

Johns DR. Department of Neurology, Beth Israel Hospital, Boston, MA, USA.

N Engl J Med 1995 Sep 7;333(10):638-44

No abstract available.

Mortality among U.S. veterans of the Persian Gulf War.

Kang HK, Bullman TA. Department of Veterans Affairs, Environmental Epidemiology Service, Washington, DC 20036-3406, USA.

N Engl J Med. 1996 Nov 14;335(20):1498-504.

BACKGROUND: Since the 1990-1991 Persian Gulf War, there has been persistent concern that U.S. war veterans may have had adverse health consequences, including higher-than-normal mortality. METHODS: We conducted a retrospective cohort study of postwar mortality according to cause among 695,516 Gulf War veterans and 746,291 other veterans. The follow-up continued through September 1993. A stratified, multivariate analysis (with Cox proportional-hazards models) controlled for branch of service, type of unit, age, sex, and race in comparing the two groups. We used standardized mortality ratios to compare the groups of veterans with the general population of the United States. RESULTS: Among the Gulf War veterans, there was a small but significant excess of deaths as compared with the veterans who did not serve in the Persian Gulf (adjusted rate ratio, 1.09; 95 percent confidence interval, 1.01 to 1.16). The excess deaths were mainly caused by accidents (1.25; 1.13 to 1.39) rather than disease (0.88; 0.77 to 1.02). The corresponding rate ratios among 49,919 female veterans of the Gulf War were 1.32 (0.95 to 1.83) for death from all causes, 1.83 (1.02 to 3.28) for accidental death, and 0.89 (0.45 to 1.78) for death from disease. In both groups of veterans the mortality rates were significantly lower overall than those in the general population. The adjusted standardized mortality ratios were 0.44 (95 percent confidence interval, 0.42 to 0.47) for Gulf War veterans and 0.38 (0.36 to 0.40) for other veterans. CONCLUSIONS: Among veterans of the Persian Gulf War, there was a significantly higher mortality rate than among veterans deployed elsewhere, but most of the increase was due to accidents rather than disease, a finding consistent with patterns of postwar mortality among veterans of previous wars.

AI Eskan disease: Desert Storm pneumonitis.

Korenyi-Both AL, Korenyi-Both AL, Molnar AC, Fidelus-Gort R. Office of the Commander, 316th STA. HOSP., Riyadh Saudi Arabia.

Mil Med. 1992 Sep;157(9):452-62.

The authors observed an acute desert-related disease when the mixture of the fine Saudi sand dust and pigeon droppings triggered a hyperergic lung condition. It was further aggravated by various kinds of organic pathogenic components contributing to an opportunistic infection of the lung. These all lead to the recognition of a new clinicopathological entity, Desert Storm pneumonitis or Al Eskan disease. For the first time, the Saudi sand dust's elemental composition was studied by ultrastructural and microanalytical means. The authors concluded that, contrary to previous beliefs, sand particles less than 1 microns (0.1 microns to 0.25 microns) in diameter are present in substantial quantities in the Saudi sand and are pathogenic, causing hyperergia. Pathogenesis of the sand dust, induced hyperergia, and its immunopathologic background are highlighted.

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Roseville, CA: Prima Health.

The Scientific Validation of Herbal Medicine 1986.

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New Canaan, CT: Keats Publishing.

Detoxification: a naturopathic perspective.

Murray, M., Pizzorno, J.

Natural Medicine Journal 1998. Stamford, CT: Fairfax Publications.

Gulf War illnesses: complex medical, scientific and political paradox.

Nicolson GL, Nicolson NL. Institute for Molecular Medicine, Huntington Beach, CA 92649-1041, USA. gnicimm@ix.netcom.com

Med Confl Surviv. 1998 Apr-Jun;14(2):156-65.

Gulf War illnesses are a collection of disorders that for the most part can be diagnosed and treated, if effective programmes exist to assist veterans, and in some cases their immediate family members. Although these illnesses are complex and have multi-organ signs and symptoms, a proportion of these patients can be identified as having Chronic Fatigue Syndrome/Myalgic Encephalomyelitis (CFS/ME) and/or Fibromyalgia Syndrome (FMS). Although there are many possible causes of CFS/ME/FMS, chronic infections can explain, at least in a subset of patients, the apparent transmission of these illnesses to family members and the appearance of chronic, multi-organ and auto-immune signs and symptoms. Unfortunately, many veterans who have been diagnosed with chronic infections, such as mycoplasmal infections, cannot obtain adequate treatment for their condition, resulting in their reliance on private physicians and clinics for assistance. This lack of response may ultimately be responsible for the transmission of the illness to non-veterans.

Physicians and Pharmacist's Guide to the Top 10 Scientifically Proven Natural Products, Second Edition 1997.

Ogletree, R.L., Fischer, R.G.

Brandon, MS: Natural Source Digest.

Effects of Mycoplasma fermentans on the myelomonocytic lineage. Different molecular entities with cytokine-inducing and cytotoxic potential.

Rawadi G, Roman-Roman S, Castedo M, Dutilleul V, Susin S, Marchetti P, Geuskens M, Kroemer G. Roussel Uclaf, Domaine Therapeutique Immunologie, Romainville, France.

J Immunol 1996 Jan 15;156(2):670-8

Mycoplasma fermentans is a mycoplasma species that has been accused of serving as a cofactor of AIDS development. Here, we show that *M. fermentans* affects the function of human monocytes and myelomonocytic cell lines on at least two different levels. Heat-inactivated mycoplasma particles induce inflammatory cytokines such as IL-1, IL-6, and TNF in monocytes, as well as in THP-1 cells. Moreover, *M. fermentans* induces IL-10 (but not IL-12) in freshly isolated human monocytes. The cytokine-inducing effect is mediated by lipid-associated molecules. In addition, we have detected a novel biologic activity that resides in the nonlipid-associated protein fraction of *M. fermentans* (approximate molecular mass: 15 to 30 kDa) and that has a cytotoxic effect on nondifferentiated myelomonocytic cell lines (U937 cells, HL-60 cells), as well as on actinomycin-D-sensitized monocytes. Death is accompanied by oligonucleosomal DNA fragmentation and loss of chromosomal DNA. U937 and HL-60 cells fail to produce cytokines and rather undergo cell death in response to heat-inactivated *M. fermentans*, provided that they are kept in a relatively undifferentiated stage. Whereas the cytokine-inducing activity is a general feature of many mycoplasma species, it appears that only a restricted panel of mycoplasma species exert a cell death-inducing activity. In addition to *M. fermentans* strains, *Mycoplasma penetrans*, another hypothetical cofactor of AIDS, possess a cytotoxic activity. This does not apply to other mycoplasma species, including pathogenic ones such as *Mycoplasma pneumoniae* and *Ureaplasma urealyticum*. The cell death-inducing effect of *M. fermentans* is not mediated by cytokines and obeys different principles than TNF- α -mediated apoptosis. Thus, in contrast to TNF- α -induced death, it is not accompanied by a decrease in the mitochondrial transmembrane potential and is not inhibited by preincubation with the antioxidant drug N-acetylcysteine. In synthesis, it appears that certain AIDS-associated mycoplasma species perturb the function and/or generation of cells from the myelomonocytic lineage via several distinct pathways.

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Natural Healing With Herbs 1984.

Santillo, H.

Prescott, AZ: Hohm Press.

The Chelation Way 1990.

Walker, M.

Garden City Park, NY: Avery Publishing Group.

Suggested Reading

The effect of silibinin (Legalon) on the free radical scavenger mechanisms of human erythrocytes in vitro.

Altorjay I, Dalmi L, Sari B, Imre S, Balla G. 2nd Department of Medicine, University Medical School, Debrecen, Hungary.

Acta Physiol Hung. 1992;80(1-4):375-80.

The effect of Legalon was investigated parallel with that of Adriblastina (doxorubicin) and paracetamol on some parameters characterizing the free radical scavenger mechanisms of human erythrocytes in vitro and on the time of acid hemolysis performed in aggregometer. Observations suggest that Adriblastina enhances the lipid peroxidation of the membrane of red blood cells, while paracetamol causes significant depletion of intracellular glutathione level, thus decreasing the free radical eliminating capacity of the glutathione peroxidase system. Legalon on the other hand, is able to increase the activity of both superoxide dismutase and glutathione peroxidase, which may explain the protective effect of the drug against free radicals and also the stabilizing effect on the red blood cell membrane, shown by the increase of the time of full haemolysis.

Exercise intolerance due to mutations in the cytochrome b gene of mitochondrial DNA.

Andreu AL, Hanna MG, Reichmann H, Bruno C, Penn AS, Tanji K, Pallotti F, Iwata S, Bonilla E, Lach B, Morgan-Hughes J, DiMauro S. H. Houston Merritt Clinical Research Center for Muscular Dystrophy and Related Diseases, Department of Neurology, Columbia College of Physicians and Surgeons, New York, NY 10032, USA.

N Engl J Med. 1999 Sep 30;341(14):1037-44.

BACKGROUND: The mitochondrial myopathies typically affect many organ systems and are associated with mutations in mitochondrial DNA (mtDNA) that are maternally inherited. However, there is also a sporadic form of mitochondrial myopathy in which exercise intolerance is the predominant symptom. We studied the biochemical and molecular characteristics of this sporadic myopathy. **METHODS:** We sequenced the mtDNA cytochrome b gene in blood and muscle specimens from five patients with severe exercise intolerance, lactic acidosis in the resting state (in four patients), and biochemical evidence of complex III deficiency. We compared the clinical and molecular features of these patients with those previously described in four other patients with mutations in the cytochrome b gene. **RESULTS:** We found a total of three different nonsense mutations (G15084A, G15168A, and G15723A), one missense mutation (G14846A), and a 24-bp deletion (from nucleotide 15498 to 15521) in the cytochrome b gene in the five patients. Each of these mutations impairs the enzymatic function of the cytochrome b protein. In these patients and those previously described, the clinical manifestations included progressive exercise intolerance, proximal limb weakness, and in some cases, attacks of myoglobinuria. There was no maternal inheritance and there were no mutations in tissues other than muscle. The absence of these findings suggests that the disorder is due to somatic mutations in myogenic stem cells after germ-layer differentiation. All the point mutations involved the substitution of adenine for guanine, but all were in different locations. **CONCLUSIONS:** The sporadic form of mitochondrial myopathy is associated with somatic mutations in the cytochrome b gene of mtDNA. This myopathy is one cause of the common and often elusive syndrome of exercise intolerance.

Unexplained illness among Persian Gulf War veterans in an Air National Guard Unit: preliminary report--August 1990-March 1995.

[No authors listed]

MMWR Morb Mortal Wkly Rep. 1995 Jun 16;44(23):443-7.

In November 1994, the U.S. Department of Veterans' Affairs (VA), the Department of Defense (DoD), and the Pennsylvania Department of Health requested that CDC investigate a report of unexplained illnesses among members of an Air National Guard (ANG) unit in south-central Pennsylvania (Unit A) who were veterans of the Persian Gulf War (PGW) (August 1990-June 1991). These veterans had been evaluated at a local VA medical center for symptoms that included recurrent rash, diarrhea, and fatigue. A three-stage investigation was planned to 1) verify and characterize signs and symptoms in PGW veterans attending the VA medical center; 2) determine whether the prevalence of symptoms was higher among members of Unit A than among members of other units deployed to the PGW and, if so, whether the increased prevalence was associated with PGW deployment; and 3) characterize the illness and identify associated risk factors. This report presents preliminary findings from stages 1 and 2 (stage 3 is in progress).

Self-reported illness and health status among Gulf War veterans. A population-based study. The Iowa Persian Gulf Study Group.

[No authors listed]

JAMA. 1997 Jan 15;277(3):238-45.

OBJECTIVE: To assess the prevalence of self-reported symptoms and illnesses among military personnel deployed during the Persian Gulf War (PGW) and to compare the prevalence of these conditions with the prevalence among military personnel on active duty at the same time, but not deployed to the Persian Gulf (non-PGW). **DESIGN:** Cross-sectional telephone interview survey of PGW and non-PGW military personnel. The study instrument consisted of validated questions, validated questionnaires, and investigator-derived questions designed to assess relevant medical and psychiatric conditions. **SETTING:** Population-based sample of military personnel from Iowa. **STUDY PARTICIPANTS:** A total of 4886 study subjects were randomly selected from 1 of 4 study domains (PGW regular military, PGW National Guard/Reserve, non-PGW regular military, and non-PGW National Guard/Reserve), stratifying for age, sex, race, rank, and branch of military service. **MAIN OUTCOME MEASURES:** Self-reported symptoms and symptoms of medical illnesses and psychiatric conditions. **RESULTS:** Overall, 3695 eligible study subjects (76%) and 91% of the located subjects completed the telephone interview. Compared with non-PGW military personnel, PGW military personnel reported a significantly higher prevalence of symptoms of depression (17.0% vs 10.9%; Cochran-Mantel-Haenszel test statistic, $< .001$), posttraumatic stress disorder (PTSD) (1.9% vs 0.8%, $P=.007$), chronic fatigue (1.3% vs 0.3%, $< .001$), cognitive dysfunction (18.7% vs 7.6%, $< .001$), bronchitis (3.7% vs 2.7%, $< .001$), asthma (7.2% vs 4.1%, $P=.004$), fibromyalgia (19.2% vs 9.6%, $< .001$), alcohol abuse (17.4% vs 12.6%, $P=.02$), anxiety (4.0% vs 1.8%, $< .001$), and sexual discomfort (respondent, 1.5% vs 1.1%, $P=.009$; respondent's female partner, 5.1% vs 2.4%, $< .001$). Assessment of health-related quality of life demonstrated diminished mental and physical functioning scores for PGW military personnel. In almost all cases, larger differences between PGW and non-PGW military personnel were observed in the National Guard/Reserve comparison. Within the PGW military study population, compared with veterans in the regular military, veterans in the National Guard/Reserve only reported more symptoms of chronic fatigue (2.9% vs 1.0%, $P=.03$) and alcohol abuse (19.4% vs 17.0%, $P=.004$). **CONCLUSIONS:** Military personnel who participated in the PGW have a higher self-reported prevalence of medical and psychiatric conditions than contemporary military personnel who were not deployed to the Persian Gulf. These findings establish the need to further investigate the potential etiologic, clinical, pathogenic, and public health implications of the increased prevalence of multiple medical and psychiatric conditions in populations of military

personnel deployed to the Persian Gulf.

Chronic multisymptom illness affecting Air Force veterans of the Gulf War.

Fukuda K, Nisenbaum R, Stewart G, Thompson WW, Robin L, Washko RM, Noah DL, Barrett DH, Randall B, Herwaldt BL, Mawle AC, Reeves WC. Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, GA 30333, USA.

JAMA. 1998 Sep 16;280(11):981-8.

CONTEXT: Gulf War (GW) veterans report nonspecific symptoms significantly more often than their nondeployed peers. However, no specific disorder has been identified, and the etiologic basis and clinical significance of their symptoms remain unclear. **OBJECTIVES:** To organize symptoms reported by US Air Force GW veterans into a case definition, to characterize clinical features, and to evaluate risk factors. **DESIGN:** Cross-sectional population survey of individual characteristics and symptoms and clinical evaluation (including a structured interview, the Medical Outcomes Study Short Form 36, psychiatric screening, physical examination, clinical laboratory tests, and serologic assays for antibodies against viruses, rickettsia, parasites, and bacteria) conducted in 1995. **PARTICIPANTS AND SETTING:** The cross-sectional questionnaire survey included 3723 currently active volunteers, irrespective of health status or GW participation, from 4 air force populations. The cross-sectional clinical evaluation included 158 GW veterans from one unit, irrespective of health status. **MAIN OUTCOME MEASURES:** Symptom-based case definition; case prevalence rate for GW veterans and nondeployed personnel; clinical and laboratory findings among veterans who met the case definition. **RESULTS:** We defined a case as having 1 or more chronic symptoms from at least 2 of 3 categories (fatigue, mood-cognition, and musculoskeletal). The prevalence of mild-to-moderate and severe cases was 39% and 6%, respectively, among 1155 GW veterans compared with 14% and 0.7% among 2520 nondeployed personnel. Illness was not associated with time or place of deployment or with duties during the war. Fifty-nine clinically evaluated GW veterans (37%) were noncases, 86 (54%) mild-to-moderate cases, and 13 (8%) severe cases. Although no physical examination, laboratory, or serologic findings identified cases, veterans who met the case definition had significantly diminished functioning and well-being. **CONCLUSIONS:** Among currently active members of 4 Air Force populations, a chronic multisymptom condition was significantly associated with deployment to the GW. The condition was not associated with specific GW exposures and also affected nondeployed personnel.

Discourse on Mitochondrial Function.

Bland, J.A.

Functional Medicine Research Center Update 2000 Mar. Gig Harbor, WA: HealthComm International (www.heathcomm.com).

Milk thistle herb provides natural support for the liver.

Brown, D.J.

Drug Store News Pharmacist 1994 Nov 14; 58-60.

No abstract available.

Gulf War symptoms remain puzzling.

Cotton, P.

JAMA 1992; 268: 2619.

No abstract available.

Dermatologic therapies you've probably never heard of.

Ely H. Department of Dermatology, University of California, Davis.

Dermatol Clin. 1989 Jan;7(1):19-35.

This article presents numerous alternative therapies for stubborn dermatologic conditions. Agents include hypertonic saline, mexiletine, alpha-acetoxymandelic acid, deladumone, emulsified steroids, activated charcoal, azelaic acid, silymarin, and

dexamethasone. Several surgical tips are also included for the practitioner's consideration.

Muscle pain, fatigue, and mitochondriopathies.

Griggs, R.C.

N. Engl. J. Med. 1999; 341(14): 177-8.

No abstract available.

Self-reported exposure to neurotoxic chemical combinations in the Gulf War. A cross-sectional epidemiologic study.

Haley RW, Kurt TL. Epidemiology Division, Department of Internal Medicine, University of Texas Southwestern Medical Center at Dallas, 75235-8874, USA.

JAMA. 1997 Jan 15;277(3):231-7.

OBJECTIVE: To identify risk factors of factor analysis-derived Gulf War-related syndromes. **DESIGN:** A cross-sectional survey. **PARTICIPANTS:** A total of 249 Gulf War veterans from the Twenty-fourth Reserve Naval Mobile Construction Battalion. **DATA COLLECTION:** Participants completed standardized booklets measuring self-reported wartime exposures and present symptoms. **MAIN OUTCOME MEASURES:** Associations of factor analysis-derived syndromes with risk factors for chemical interactions that inhibit butyrylcholinesterase and neuropathy target esterase. **RESULTS:** Risk of syndrome 1 ("impaired cognition") was greater in veterans who reported wearing flea collars during the war (5 of 20, 25%) than in those who never wore them (7 of 229, 3%; relative risk [RR], 8.7; 95% confidence interval [CI], 3.0-24.7; < .001). Risk of syndrome 2 ("confusion-ataxia") increased with a scale of advanced adverse effects from pyridostigmine bromide (χ^2 for trend, < .001), was greater among veterans who believed they had been involved in chemical weapons exposure (18 of 108, 17%) than in those who did not (3 of 141, 2%; RR, 7.8; 95% CI, 2.3-25.9; < .001), and was increased in veterans who had been in a sector of far northeastern Saudi Arabia on the fourth day of the air war (6 of 21, 29%) than in those who had not been (15 of 228, 7%; RR, 4.3; 95% CI, 1.9-10.0; P=.004). Effects of perceived chemical weapons exposure and advanced adverse effects from pyridostigmine were synergistic (Rothman S, 5.3; 95% CI, 1.04-26.7). Risk of syndrome 3 ("arthro-myo-neuropathy") increased with an index of frequency and amount of government-issued insect repellent containing 75% DEET (N,N-diethyl-m-toluamide) in ethanol applied during the war (χ^2 for trend, < .001) and with advanced adverse effects from pyridostigmine (χ^2 for trend, < .001). **CONCLUSION:** Some Gulf War veterans may have delayed, chronic neurotoxic syndromes from wartime exposure to combinations of chemicals that inhibit butyrylcholinesterase and neuropathy target esterase.

Association of low PON1 type Q (type A) arylesterase activity with neurologic symptom complexes in Gulf War veterans.

Haley RW, Billecke S, La Du BN. Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas, Texas 75235-8874, USA.

Toxicol Appl Pharmacol. 1999 Jun 15;157(3):227-33.

Previously Haley et al. described six possible syndromes identified by factor analysis of symptoms in Gulf War veterans and demonstrated that veterans with these symptom complexes were more neurologically impaired than age-sex-education-matched well controls. They also uncovered strong associations (relative risks 4-8) suggesting that these symptom complexes were related to wartime exposure to combinations of organophosphate pesticides, chemical nerve agents, high concentration DEET insect repellent, and symptoms of advanced acute toxicity after taking pyridostigmine. Here we have shown that compared to controls, ill veterans with the neurologic symptom complexes were more likely to have the R allele (heterozygous QR or homozygous R) than to be homozygous Q for the paraoxonase/arylesterase 1 (PON1) gene. Moreover, low activity of the PON1 type Q (Gln192, formerly designated type A) arylesterase allozyme distinguished ill veterans from controls better than just the PON1 genotype or the activity levels of the type R (Arg192, formerly designated type B) arylesterase allozyme, total arylesterase, total paraoxonase, or butyrylcholinesterase. A history of advanced acute toxicity after taking pyridostigmine was also correlated with low PON1 type Q arylesterase activity. Type Q is the allozyme of paraoxonase/arylesterase that most efficiently hydrolyzes several organophosphates including sarin, soman, and diazinon. These findings further support the proposal that neurologic symptoms in some Gulf War veterans were caused by environmental chemical exposures. Copyright 1999 Academic Press.

Chronic multisystem illness among Gulf War veterans.

Hunt, S.C., Richardson, R.D.

JAMA 1999; 282(4): 327-8; discussion 328-9. No abstract available.

A comprehensive clinical evaluation of 20,000 Persian Gulf War veterans. Comprehensive Clinical Evaluation Program Evaluation Team.

Joseph SC. Department of Defense, Office of Assistant Secretary of Defense-Health Affairs (Clinical Services), Washington, DC 20301-1200, USA.

Mil Med. 1997 Mar;162(3):149-55.

In response to the health concerns of Gulf War veterans, the Department of Defense instituted the Comprehensive Clinical Evaluation Program (CCEP). Although not designed as a research study, the CCEP provided valuable clinical data. An analysis was conducted of CCEP findings from systematic and comprehensive examinations of 20,000 U.S. Gulf War veterans. Among 20,000 participants, the types of primary and secondary diagnoses varied widely. Also, among veterans with an ICD-9-CM diagnosis of "symptoms, signs, and ill-defined conditions," no single subcategory of illness predominated, and no characteristic physical sign or laboratory abnormality was identified. In-total, there were 74 (0.4%) cases of connective tissue disease; 52 (0.3%) noncutaneous malignancies; 42 (0.2%) peripheral neuropathies; 14 (0.07%) cases of interstitial pulmonary fibrosis; 12 (0.06%) cases of renal insufficiency; and no new cases of viscerotropic leishmaniasis. No clinical indication of a new or unique illness was identified in this self-referred population, and the types of physiologic disease that could result from postulated hazardous wartime exposures were uncommon.

Illness in Gulf War veterans: causes and consequences.

Landrigan, P.J.

JAMA 1997; 277: 259-61.

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Hospitalization for unexplained illnesses among U.S. veterans of the Persian Gulf War,

Leisure, K.M. et al.

Emerg. Infect. Dis. 1998; 4: 707-9.

No abstract available.

Dietary fiber and human health.

Mendeloff AI.

N Engl J Med. 1977 Oct 13;297(15):811-4.

No abstract available.

Is there a Gulf War syndrome?

Milner, I.B. et al.

JAMA 1994; 271: 661.

No abstract available.

Silymarin protects against paracetamol-induced lipid peroxidation and liver damage.

Muriel P, Garciapina T, Perez-Alvarez V, Mourelle M. Departamento de Farmacologia y Toxicologia, Instituto Politecnico Nacional, Mexico D.F.

J Appl Toxicol. 1992 Dec;12(6):439-42.

The effect of silymarin on liver damage induced by acetaminophen (APAP) intoxication was studied. Wistar male rats pretreated (72 h) with 3-methylcholanthrene (3-MC) (20 mg kg⁻¹ body wt. i.p.) were divided into three groups: animals in group 1 were treated with

acetaminophen (APAP) (500 mg kg⁻¹ body wt. p.o.), group 2 consisted of animals that received APAP plus silymarin (200 mg kg⁻¹ body wt. p.o.) 24 h before APAP, and rats in group 3 (control) received the equivalent amount of the vehicles. Animals were sacrificed at different times after APAP administration. Reduced glutathione (GSH), lipid peroxidation and glycogen were measured in liver and alkaline phosphatase (AP), gamma-glutamyl transpeptidase (GGTP) and glutamic pyruvic transaminase (GPT) activities were measured in serum. After APAP intoxication, GSH and glycogen decreased very fast (1 h) and remained low for 6 h. Lipid peroxidation increased three times over the control 4 and 6 h after APAP treatment. Enzyme activities increased 18 h after intoxication. In the group receiving APAP plus silymarin, levels of lipid peroxidation and serum enzyme activities remained within the control values at any time studied. The fall in GSH was not prevented by silymarin, but glycogen was restored at 18 h. It was concluded that silymarin can protect against APAP intoxication through its antioxidant properties, possibly acting as a free-radical scavenger.

Encyclopedia of Nutritional Supplements 1996.

Murray, M.

Rocklin, CA: Prima Publishing.

Considerations when undergoing treatment for Gulf War illness/CFS/FMS/rheumatoid arthritis.

Nicolson, G.

Int. J. Med. 1998; 1: 123-8.

No abstract available.

The role of microorganism infections in chronic illnesses: support for antibiotic regimens.

Nicolson, G.

The CFIDS Chronicle 1999 Sep-Oct.

No abstract available.

The Gulf: war or human laboratory (comments on an article by John Nichols).

Nicholson, G., Nicholson, N.

Med. Conflict Survival 1996; 12(2): 260-2.

No abstract available.

The eight myths of Operation Desert Storm and Gulf War syndrome.

Nicholson, G., Nicholson, N.

Med. Conflict Survival 1997; 13(2): 140-6.

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Doxycycline treatment and Desert Storm.

Nicholson, G.L., Rosenberg-Nicholson, N.L.

JAMA 1995 Feb 22; 273(8): 618-9.

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Chronic fatigue illness and operation Desert Storm.

Nicolson, G.L., Bruton, D.M., Jr., Nicolson, N.L.

J. Occup. Environ. Med. 1995; 38: 14-6.

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Diagnosis and treatment of chronic mycoplasmal infections in fibromyalgia and chronic fatigue syndromes: relationship to Gulf War illness.

Nicolson, G., Nasralla, M., Haier, J., Nicolson, N.L.

Biomed. Ther. 1998; 26(4).

No abstract available.

The Persian Gulf experience and health. NIH Technology Assessment Workshop Panel.

NIH. / [No authors listed]

JAMA. 1994 Aug 3;272(5):391-6.

No abstract available.

A Review of the Scientific Literature as It Pertains to Gulf War Illnesses: Pyridostigmine Bromide 1999;

Rand Corporation.

RAND Rep. MR-1018/2. Santa Monica, CA: Rand Corporation (www.rand.org).

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Rand Corporation.

RAND Rep. MR-1018/4. Santa Monica, CA: Rand Corporation (www.rand.org).

A Review of the Scientific Literature as It Pertains to Gulf War Illnesses: Oil Well Fires 1998;

Rand Corporation.

RAND Rep. MR-1018/6. Santa Monica, CA: Rand Corporation (www.rand.org).

A Review of the Scientific Literature as It Pertains to Gulf War Illnesses: Depleted Uranium 1999;

Rand Corporation.

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Military Use of Drugs Not Yet Approved by FDA for CW/BW Defense: Lessons from the Gulf War 1999;

Rand Corporation.

RAND Rep. MR-1018/9. Santa Monica, CA: Rand Corporation (www.rand.org).

Acetaminophen-induced toxicity to human epidermoid cell line A431 and hepatoblastoma cell line Hep G2, in vitro, is diminished by silymarin.

Shear NH, Malkiewicz IM, Klein D, Koren G, Randor S, Neuman MG. Division of Dermatology, Sunnybrook Health Science Centre, Ont., Canada.

The skin and liver may be targets for cytotoxicity induced by oxidative drug metabolites. We used human epidermoid A431 cells and human hepatoblastoma Hep G2 cells as the experimental model. The aim of the study was to investigate and evaluate the effect of silymarin on acetaminophen (APAP)-induced toxicity under controlled conditions. Silymarin is known to be a potent antioxidant that diminishes toxicity induced by a variety of other hepatotoxins (e.g. Amanita phalloides, algae's toxins, carbon tetrachloride). Glutathione (GSH) depletion was enhanced by adding to the medium buthionine sulfoximine [L-buthionine-(S,R)-sulfoximine, BSO]. Cells were incubated with high-concentration 5-20 mM APAP or alpha-(minimum essential medium for 2-24 h to evaluate the drug's ability to reduce cytotoxicity. Viability was then quantitated by metabolism of the tetrazolium dyes (MTT) and neutral red (NR). Cytotoxicity was 100% for controls. For Hep G2 treated for 24 h with 20 mM, APAP viability was 56.0% by MTT and 62.5% by NR. BSO-treated cells showed an enhanced cytotoxicity, determined by both assays. Administration of 0.5 mM silymarin reduced cytotoxicity significantly. In A431 cells, treatment with 20 mM APAP reduced viability by 57% (MTT) and 69% (NR) versus control (100%). BSO further decreased viability. Since incubation with silymarin showed significant protection against APAP toxicity, it can be considered a cytoprotective agent in this in vitro model of drug toxicity. GSH concentrations in both cell lines decrease significantly after exposure to 20 mM APAP, or 0.5 mM versus control (< 0.05), and increased (< 0.001) if incubated with APAP and silymarin. The protective effect could be through mitochondrial membrane stabilization and/or an increase in available GSH.

Bridging the gulf in war syndromes.

Strauss, S.E.

Lancet 1999 Jan 16; 353(9148): 162-3.

No abstract available.

Physical health symptomatology of Gulf War-era service personnel from the states of Pennsylvania and Hawaii.

Stretch RH, Bliese PD, Marlowe DH, Wright KM, Knudson KH, Hoover CH. Department of Military Psychiatry, Walter Reed Army Institute of Research, Washington, DC 20307-5100, USA.

Mil Med. 1995 Mar;160(3):131-6.

We present data on physical health and possible "Gulf War syndrome" from a Congressionally mandated study of over 4,000 active duty and reserve service members from the states of Hawaii and Pennsylvania who served during Operation Desert Storm. We found that deployed veterans report significantly more physical health symptoms than non-deployed veterans that cannot be explained by reasons other than deployment alone. We also identified a subgroup of 178 deployed veterans at risk for possible Gulf War syndrome. We recommend that services collect baseline information from units likely to deploy in the future and update that information regularly.

Protective effect of Legalon in workers exposed to organic solvents.

Szilard S, Szentgyorgyi D, Demeter I. Tisza Chemical Works Leninvaros, Occupational Health Care Service, Hungary.

Acta Med Hung. 1988;45(2):249-56

Abnormal result of liver function tests (elevated levels of AST, ALT activity) and/or abnormal haematological values (low platelet counts, leucocytosis, relative lymphocytosis) were observed in 49 of 200 workers exposed to toluene and/or xylene vapours for 5-20 years. Thirty of the affected workers were treated per os with Legalon (MADAUS, FRG) t.i.d. for 30 days. The remaining 19 were left without treatment. Under the influence of Legalon the liver function tests and the platelet counts significantly improved. The leukocytosis and relative lymphocytosis showed a nonsignificant tendency of improvement.

The development of the concept of dietary fiber in human nutrition.

Trowell, H.

Am. J. Clin. Nutr. 1987; 31(10): S3-S11.

No abstract available.

DOD and RAND Release Review of the Literature as It Pertains to Gulf War Illnesses and Pyridostigmine Bromide.

U.S. Department of Defense.

News Release, October 19, 1999; Office of the Assistant Secretary of Defense

(www.defenselink.mil/news/).

Fox Tapes Show No Evidence of Chemical Warfare Agent.

U.S. Department of Defense.

News Release, February 24, 2000; DOD/DVA (www.gulflink.osd.mil).

Web Sites

Health Impact of Exposures During the Gulf War.

CDC-sponsored meeting (February 28 - March 2, 1999 in Atlanta).

www.cdc.gov/

Gulf War Illnesses: Role of Chemical, Radiological, and Biological Exposures

War and Health 1999.

www.immed.org

Haley pages refuting U.S. government conclusion that GWS is largely the result of stress.

www.swmed.edu/home_pages/epidemi/gws

New Jersey Gulf War Research Center.

www.umdnj.edu/cfsweb/GW/njgwr_home.html SEE ALSO

The WRIISC site can be found at <http://www.wri.med.va.gov/>

On the Possible Pathogenesis of Gulf War Illnesses.

www.cfsaudio.4biz.net/ccf/simpson.htm

Research Group Identifies Immune System Anomaly that Supports Existence of Gulf War Syndrome

www.chronicillnet.org

Senate Committee Determines No Single Cause for GWS.

www.news.bbc.co.uk

Study Can't Rule Out Possible Link Between Drug and Gulf War Illnesses.

www.abcnews.com

Veterans Administration (copies of Rand Reports may be accessed from this site).

www.gulflink.osd.mil

www.cnn.com/HEALTH/9903/01/gulf.syndrome.01/index.html

www.gulfwarvets.com anthrax.htm

www.gulfweb.org

www.healthlinkusa.com

www.mcsurvivors.com

www.stayhealthy.com

www.va.gov/health/environ/persgulf.htm

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