MYALGIA

A MEDICAL DICTIONARY, BIBLIOGRAPHY,
AND ANNOTATED RESEARCH GUIDE TO
INTERNET REFERENCES

JAMES N. PARKER, M.D.
AND PHILIP M. PARKER, PH.D., EDITORS

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FORWARD

In March 2001, the National Institutes of Health issued the following warning: "The number of Web sites offering health-related resources grows every day. Many sites provide valuable information, while others may have information that is unreliable or misleading."[1] Furthermore, because of the rapid increase in Internet-based information, many hours can be wasted searching, selecting, and printing. Since only the smallest fraction of information dealing with myalgia is indexed in search engines, such as **www.google.com** or others, a non-systematic approach to Internet research can be not only time consuming, but also incomplete. This book was created for medical professionals, students, and members of the general public who want to know as much as possible about myalgia, using the most advanced research tools available and spending the least amount of time doing so.

In addition to offering a structured and comprehensive bibliography, the pages that follow will tell you where and how to find reliable information covering virtually all topics related to myalgia, from the essentials to the most advanced areas of research. Public, academic, government, and peer-reviewed research studies are emphasized. Various abstracts are reproduced to give you some of the latest official information available to date on myalgia. Abundant guidance is given on how to obtain free-of-charge primary research results via the Internet. **While this book focuses on the field of medicine, when some sources provide access to non-medical information relating to myalgia, these are noted in the text.**

E-book and electronic versions of this book are fully interactive with each of the Internet sites mentioned (clicking on a hyperlink automatically opens your browser to the site indicated). If you are using the hard copy version of this book, you can access a cited Web site by typing the provided Web address directly into your Internet browser. You may find it useful to refer to synonyms or related terms when accessing these Internet databases. **NOTE:** At the time of publication, the Web addresses were functional. However, some links may fail due to URL address changes, which is a common occurrence on the Internet.

For readers unfamiliar with the Internet, detailed instructions are offered on how to access electronic resources. For readers unfamiliar with medical terminology, a comprehensive glossary is provided. For readers without access to Internet resources, a directory of medical libraries, that have or can locate references cited here, is given. We hope these resources will prove useful to the widest possible audience seeking information on myalgia.

The Editors

[1] From the NIH, National Cancer Institute (NCI): **http://www.cancer.gov/cancerinfo/ten-things-to-know**.

CHAPTER 1. STUDIES ON MYALGIA

Overview

In this chapter, we will show you how to locate peer-reviewed references and studies on myalgia.

The Combined Health Information Database

The Combined Health Information Database summarizes studies across numerous federal agencies. To limit your investigation to research studies and myalgia, you will need to use the advanced search options. First, go to **http://chid.nih.gov/index.html**. From there, select the "Detailed Search" option (or go directly to that page with the following hyperlink: **http://chid.nih.gov/detail/detail.html**). The trick in extracting studies is found in the drop boxes at the bottom of the search page where "You may refine your search by." Select the dates and language you prefer, and the format option "Journal Article." At the top of the search form, select the number of records you would like to see (we recommend 100) and check the box to display "whole records." We recommend that you type "myalgia" (or synonyms) into the "For these words:" box. Consider using the option "anywhere in record" to make your search as broad as possible. If you want to limit the search to only a particular field, such as the title of the journal, then select this option in the "Search in these fields" drop box. The following is what you can expect from this type of search:

- **Differential Diagnosis of Masticatory Muscle Pain and Dysfunction**

  Source: Oral and Maxillofacial Surgery Clinics of North America. 7(1): 29-49. February 1995.

  Summary: This article on the differential diagnosis of masticatory **muscle pain** and dysfunction is from an issue of Oral and Maxillofacial Clinics on the medical management of temporomandibular disorders (TMD). The author notes that there are many local and systemic conditions besides TMD that can cause similar signs and symptoms and that need to be considered in the differential diagnosis. The author describes many of these conditions, their causes, and their distinguishing characteristics. Conditions discussed include masticatory **myalgia;** myositis; fibromyalgia syndrome (FS); direct muscle injury; acute cervical strain (whiplash); tendonitis; secondary pains of dental origin; complications of middle ear infections; maxillary sinus disease; paranasal

sinus neoplasia; salivary gland disease; lymphadenitis; skeletal disease; neurogenic disturbances; vascular disturbances; psychogenic disturbances; and systemic disorders. 3 tables. 72 references. (AA-M).

- **Fibromyalgia and Chronic Fatigue Syndrome: Similarities and Differences**

  Source: Rheumatic Disease Clinics of North America. 22(2):219-243; May 1996.

  Summary: This journal article for health professionals examines the similarities and differences between fibromyalgia (FM) and chronic fatigue syndrome (CFS). CFS and FM are clinical conditions characterized by a variety of nonspecific symptoms, including prominent fatigue, **myalgia,** and sleep disturbances. There are no diagnostic studies or widely accepted, pathogenic explanatory models for either illness. Despite remarkably different diagnostic criteria, CFS and FM have many demographic and clinical similarities. Similarities and differences in the epidemiologic, clinical, laboratory, and psychiatric features of FM ad CFS are discussed, as are the prognosis and treatment of these conditions. This discussion reveals that few differences exist in the domains of symptoms, examination findings, laboratory tests, functional status, psychosocial features, and psychiatric disorders. FM appears to represent an additional burden of suffering among those with CFS. Further clarification of the similarities and differences between CFS and FM may be useful in studies of the prognosis and help define subsets of patients who may benefit from specific therapeutic interventions. 178 references and 5 tables. (AA-M).

- **Eosinophilia-myalgia Syndrome: Review and Reappraisal of Clinical, Epidemiologic and Animal Studies Symposium**

  Source: Journal of Rheumatology. 23(Supp. 46):1-110; October 1996.

  Summary: This journal for health professionals includes papers that were presented at a conference on eosinophilia **myalgia** syndrome (EMS) in December 1994. Papers presented the criteria for the definition of EMS, a diagnostic algorithm for differentiating EMS from fibromyalgia and chronic myofascial pain, a study on the clinical status of EMS patients 2 to 4 years after onset, a critique of epidemiologic studies on the association of L-tryptophan (LT) with EMS, a review of epidemiologic studies that assessed the association between LT and EMS, and an analysis of Centers for Disease Control and Prevention criteria for EMS in a geographically defined population, animal models of EMS, and the results of a comparative histopathologic evaluation of animal studies of EMS. Other topics included the pathophysiology of EMS, the way in which fibromyalgia and psychiatric disorders may complicate the assessment of patients with possible EMS, and the causal relationship between tryptophan produced by Showa Denko and epidemic EMS. Discussions follow many of the papers. Appendices list conference participants and data collection variables used in the development of a diagnostic algorithm for EMS. Numerous references, 25 figures, and 30 tables.

Federally Funded Research on Myalgia

The U.S. Government supports a variety of research studies relating to myalgia. These studies are tracked by the Office of Extramural Research at the National Institutes of Health.[2] CRISP (Computerized Retrieval of Information on Scientific Projects) is a searchable

[2] Healthcare projects are funded by the National Institutes of Health (NIH), Substance Abuse and Mental Health Services (SAMHSA), Health Resources and Services Administration (HRSA), Food and Drug Administration

database of federally funded biomedical research projects conducted at universities, hospitals, and other institutions.

Search the CRISP Web site at **http://crisp.cit.nih.gov/crisp/crisp_query.generate_screen**. You will have the option to perform targeted searches by various criteria, including geography, date, and topics related to myalgia.

For most of the studies, the agencies reporting into CRISP provide summaries or abstracts. As opposed to clinical trial research using patients, many federally funded studies use animals or simulated models to explore myalgia. The following is typical of the type of information found when searching the CRISP database for myalgia:

- **Project Title: HANTAVIRUS: HEMORRHAGIC FEVER IMMUNOPATHOGENESIS**

  Principal Investigator & Institution: Libraty, Daniel H.; Assistant Professor; Univ of Massachusetts Med Sch Worcester Office of Research Funding Worcester, Ma 01655

  Timing: Fiscal Year 2003; Project Start 01-OCT-2003; Project End 30-SEP-2008

  Summary: Hantaviruses are RNA viruses that cause hantavirus pulmonary syndrome (HPS) and hemorrhagic fever with renal syndrome (HFRS). HPS and HFRS are characterized by fever, **myalgia,** rapid onset of a vascular leak syndrome, hemoconcentration, and thrombocytopenia. In HPS, the lung is the prominent target organ; while, in HFRS, the kidney is the prominent target organ. Hantaviruses are NIAID category A priority pathogens with regards to biodefense, as they can produce severe, potentially fatal, diseases, are transmitted by aerosol, and do not have effective vaccines or specific therapeutics. The goal of this project is to understand the immunologic mechanisms that lead to HFRS. Several lines of evidence suggest that HFRS is not caused by direct cytopathic effects of hantaviruses, but rather by exuberant host immunopathological responses. This project will rely on samples provided from a prospective cohort study of Puumala (PUU) virus infections, a HFRS-associated hantavirus in Finland. The first aim will be to characterize dendritic cell functions and humoral immune responses that affect the PUU virus burden, using flow cytometry, antibody detection assays, and quantitative viral RT-PCR. The second aim will be to analyze the patterns and temporal regulation of cellular immune responses throughout acute PUU virus infection. ELISAs, multiplex immunoassays, quantitative RT-PCR, and genomic screening techniques will be used to examine immune response mediators in a comprehensive fashion, along with virus levels and disease severity. The third aim will be to characterize the antigen specificity and behavior of T lymphocyte responses during and after PUU virus infection. CD8+ and CD4+ T cell epitopes from PUU virus proteins will be identified using cell cloning techniques, ELISPOTs, cytotoxic T lymphocyte (CTL) assays, and mapping with overlapping synthetic peptides. Effector mechanisms of vascular leakage will be studied by examining interactions between endothelial cells and PUU virus-specific T cell clones. Peptide stimulation with intracellular cytokine staining and peptide-HLA Class I and II tetramers will be used to identify and quantify antigen-specific T cell responses across a spectrum of PUU virus disease. Elucidation of the immunopathogenetic mechanisms in PUU virus infection will contribute to the development of effective vaccine strategies and immune-based therapies of HFRS and HPS.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

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(FDA), Centers for Disease Control and Prevention (CDCP), Agency for Healthcare Research and Quality (AHRQ), and Office of Assistant Secretary of Health (OASH).

- **Project Title: MECHANISMS OF PAIN CAUSED BY DISRUPTION OF MICROTUBLES**

  Principal Investigator & Institution: Levine, Jon D.; Professor of Medicine; University of California San Francisco 500 Parnassus Ave San Francisco, Ca 941222747

  Timing: Fiscal Year 2002

  Summary: The chemotherapeutic agent paclitaxel (Taxol) is widely used for the treatment of many different types of carcinomas. At present, the dose of paclitaxel that can be tolerated by patients is limited primarily by the development of a painful peripheral neuropathy characterized by paresthesias, **myalgia** and arthralgia. Similar dose-limiting painful neuropathies are produced by other microtubule-disrupting chemotherapeutic drugs, dose-limiting painful neuropathies are produced by other microtubule-disrupting chemotherapeutic drugs, including vincristine therapy, but also increase the effectiveness of their treatment by permitting the use of higher doses of the drugs. We propose a series of experiments to elucidate the cellular mechanisms of paclitaxel- and vincristine-induced painful neuropathy. Specifically, we will establish a model of paclitaxel-induced painful peripheral neuropathy in the rat, and then analyze paclitaxel-induced changes in the excitability of nociceptive nerve fibers in this model. Our interpretation of those data will be greatly enhanced by our extensive knowledge of the effects of vincristine on nerve fiber excitability. Because taxol stabilizes microtubules while vincristine stimulates microtubule depolymerization, comparison of similarities and differences in the effects of the two agents may provide valuable insights into the mechanisms by which microtuble disruption causes nociceptor hyperexcitability. Guided by these in vivo electrophysiological analyses, we will employ patch clamp recording of cultured sensory neurons to study the effects of both drugs on specific transduction molecules and ion channels. Finally, we will investigate intracellular second messenger pathways that participate in the production of hyperalgesia and nociceptor hyperexcitability induced by paclitaxel and vincristine. The proposed in vitro studies may identify specific molecules to be targeted by new pharmacological strategies to treat chemotherapy-induced neuropathic pain caused by chemotherapy. By improving our understanding of the cellular mechanisms of neuropathic pain, these studies can potentially provide important insights into the pathophysiology and treatment of orofacial neuropathies.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: MOLECULAR ETIOLOGY OF FAMILIAL MEDITERRANEAN FEVER**

  Principal Investigator & Institution: Gumucio, Deborah L.; Associate Professor; Cell and Developmental Biology; University of Michigan at Ann Arbor 3003 South State, Room 1040 Ann Arbor, Mi 481091274

  Timing: Fiscal Year 2003; Project Start 01-JUL-2003; Project End 31-DEC-2007

  Summary: (provided by applicant): Patients with the autosomal recessive disease, Familial Mediterranean fever (FMF), suffer periodic, unpredictable attacks of fever associated with severe pain; the pain is localized most commonly in joints (arthritis), abdomen (peritonitis) or chest (pleuritis). Occasionally, this disease presents with skin manifestations (erysipeloid erythema), pericarditis, vasculitis, or **myalgia.** In many patients, amyloidosis is a complication, and if untreated, this can be life-threatening. FMF is caused by missense mutations in pyrin, a protein of unknown function expressed in neutrophils, monocytes, eosinophils, dendritic cells, synovial cells and skin and peritoneal fibroblasts. Pyrin expression in these cells is induced by pro-inflammatory cytokines and by LPS. Thus, it has been speculated that pyrin modulates

the inflammatory response. Evolutionary studies of the pyrin molecule indicate that it has been under positive Darwinian selection during evolution of the primates. Moreover, the high frequency of mutant pyrin alleles in several human ethnic groups supports a heterozygote (selective) advantage for the mutant allele. Mutant forms of pyrin may enhance the body's ability to clear important pathogen(s). Indeed, acute phase reactants, important agents of innate immunity, are up-regulated not only in patients but in carriers of mutant alleles. Structural analysis of the pyrin molecule revealed that exon 1 encodes a death-domain related structural motif (known as the pyrin domain or PyD) that is found in a growing family of proteins involved in inflammation and innate immunity. Identification of pyrin-interacting proteins as well as additional functional studies reveal that pyrin is linked directly to apoptotic and cytoskeletal signaling cascades, and that it modulates cytokine secretion. Experiments described in this proposal are designed to further explore these functions of pyrin and determine the effects of pyrin mutations on apoptosis (Aim 1); cytoskeletal signaling (Aim 2); and cytokine production (Aim 3). Recently identified pyrin isoforms will also be examined in these functional assays, since preliminary studies indicate that the various isoforms may function differently. Such studies could provide clues to understanding of the molecular pathogenesis of FMF, and may reveal new information about inflammatory pathways in general. In fact, pyrin-interacting proteins and pyrin domain-containing family members have already been connected to several human diseases, including inflammatory bowel disease, PAPA syndrome, Muckle-Wells syndrome, familial cold urticaria, Blau syndrome and Bechet's disease.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: NON-TOXIC HUMAN INTERFERON-ALPHA ANALOG**

  Principal Investigator & Institution: Villarete, Lorelie H.; Pepgen Corporation 1255 Harbor Bay Pky, Ste B Alameda, Ca 94502

  Timing: Fiscal Year 2003; Project Start 01-AUG-2003; Project End 31-JUL-2005

  Summary: (provided by applicant): The clinically available forms of human interferon (IFN) alpha-IFNalpha2alpha (Roferon-A), IFNalpha2b (Intron). Consensus IFN and pegylated IFNs (PEG Intron and Pegasys) - are useful in the treatment of several viral diseases and cancers. However, when used at therapeutic doses they produce frequent and sometimes serious side effects, including fever, **myalgia,** CNS effects and leukopenia, which limit their use. IFNinterferon, a structurally related interferon in ruminants, has similar antiviral and antitumor properties as the IFNalpha's but little or no toxicity. However, as a xenoprotein IFNinterferon is not a suitable candidate for development as a parenteral drug for humans. We have synthesized an analog of human IFNalpha2b, NLVgalpha2b, which contains five amino acid substitutions at positions19, 20, 22, 24 and 27 using residues from the corresponding positions in the IFNinterferon molecule. The in vitro and in vivo data from our SBIR phase I study demonstrated that these substitutions conferred markedly reduced cellular toxicity on the resulting molecule without diminishing its antiviral and antitumor activities. In this phase II project we will advance NLVgalpha2b into preclinical development by optimizing expression of this recombinant IFN in yeast, producing pegylated as well as unpegylated preparations and subjecting them to rigorous evaluation in well established animal models. The antiviral, anticancer, immunogenicity and toxicity profiles of NLVgalpha2b will be compared with those of commercially available IFNalpha2b. If this project is successful, it should be possible to administer NLVgalpha2b to patients in higher doses than can be achieved with current IFNalpha's, resulting in improved clinical outcomes.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: PILOT--DNA AND LIPID INDUCE TH1 MEDIATED PULMONARY INFLAMMATION**

  Principal Investigator & Institution: Van Ginkel, Frits; University of Alabama at Birmingham Uab Station Birmingham, Al 35294

  Timing: Fiscal Year 2002

  Summary: Description (adapted from the application): This pilot project deals with elucidation of mechanisms involved in the DNA/lipid complex-induced toxicity in CF patient during cationic liposome-mediated gene therapy. The symptoms observed include fever, **myalgia** and arthralgia, occurring at six hours with full recovery between 24-48 hours after the administration of DNA/lipid 67 complexes. The applicant believes that the toxic effects of DNA/lipid complexes are caused by unmethylated CpG motif in plasmid DNA. It is hypothesized that CpG motif activates T lymphocytes and macrophages to release inflammatory cytokines (TNF-a and IL-1), Th1-inducing cytokines (IL-12 and IL-18), Th-1 cytokine (IFN-r), leading to fever, **myalgia,** arthralgia and activation of NK and B cells. The applicant has proposed two Specific Aims to test his hypothesis. Aim 1 is to explore the effect of methylated and unmethylated CpG motifs of plasmid DNA in the presence or absence of lipid 67 on the production of specific cytokines in human peripheral blood cells (PBMCs) in vitro, and to test the effect of anti-inflammatory cytokines IL-10 and IL-4 on the production of the specific cytokines. Aim 2 deals with similar experiments in vivo using IL-12 or IFN-r knock out mice.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: RECOMBINANT & LIVE ORAL SALMONELLA TYPHI HYBRID VACCINES**

  Principal Investigator & Institution: Levine, Myron Max.; Director; Medicine; University of Maryland Balt Prof School Baltimore, Md 21201

  Timing: Fiscal Year 2004; Project Start 01-APR-1990; Project End 28-FEB-2009

  Summary: (provided by applicant): In November 2002 in China, an outbreak of atypical pneumonia occurred in which a proportion of cases were very severe or fatal, and a high lethality was seen among elderly patients. The clinical syndrome began with fever, dry cough, **myalgia** and sore throat and progressed to atypical pneumonia. Outbreaks followed thereafter in 2003 in Vietnam, Hong Kong, Singapore, Canada, and Taiwan. Extraordinary characteristics of this global epidemic of "Severe Acute Respiratory Syndrome" (SARS) include the rapid isolation of the etiologic agent (a novel coronavirus; SARS-CoV), elucidation of the complete sequence of the viral genome, accelerated development of diagnostic tests, and rapid global exchange of clinical, epidemiologic and microbiologic information via the Internet by scientists and health officials in many countries. Investigators in the USA and Hong Kong were first to isolate from patients the novel coronavirus that is distinct from previously recognized groups of coronavirus. The underlying hypothesis of this research plan is that by appropriate manipulation of attenuated Salmonella enterica serovar Typhi (S. Typhi) and Shigella live vectors it will be possible to develop a mucosally-administered "prime-boost" vaccination strategy to prevent SARS. We will utilize attenuated S. Typhi or Shigella flexneri 2a live vector vaccine strains to deliver (via mucosal immunization) a Sindbis eukaryotic DNA replicon encoding the S (spike) and M (membrane) glycoproteins and the N nucleocapsid protein of the Urbani strain of the SARS-CoV to prime the immune

system to recognize these coronavirus antigens. We will then boost the immune response by mucosally administering proteosomes (meningococcal outer membrane protein vesicles) to which the same SARS proteins are adsorbed (along with a lipopolysaccharide adjuvant). Virus-like Particles and attenuated S. Typhi expressing SARS peptide epitopes will serve as back-up boosting strategies. We will study whether these constructs can elicit the relevant immune responses, first in mice, then in cynomolgus monkeys, and finally in clinical trials in humans (the latter under separate funding). The induction of B and T cell memory pools will also be examined in monkeys. This approach aims to mimic the strong and broad immunity elicited by live virus vaccines with the inherent safety factor of not having to use putative attenuated live SARS virus derivatives. If the proposed vaccination strategy can indeed elicit broad, balanced and long-lasting immune responses in cynomolgus monkeys, these studies can be followed by a challenge (under respiratory pathogen biosafety level 3 containment) to assess the efficacy of the vaccine against wild type SARS-CoV.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

E-Journals: PubMed Central[3]

PubMed Central (PMC) is a digital archive of life sciences journal literature developed and managed by the National Center for Biotechnology Information (NCBI) at the U.S. National Library of Medicine (NLM).[4] Access to this growing archive of e-journals is free and unrestricted.[5] To search, go to **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Pmc**, and type "myalgia" (or synonyms) into the search box. This search gives you access to full-text articles. The following is a sample of items found for myalgia in the PubMed Central database:

- **Hypodense eosinophils and interleukin 5 activity in the blood of patients with the eosinophilia-myalgia syndrome.** by Owen WF Jr, Petersen J, Sheff DM, Folkerth RD, Anderson RJ, Corson JM, Sheffer AL, Austen KF.; 1990 Nov; http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=55014
- **Severe myalgia from an interaction between treatments with pantoprazole and methotrexate.** by Troger U.; 2002 Jun 22; http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=116448

The National Library of Medicine: PubMed

One of the quickest and most comprehensive ways to find academic studies in both English and other languages is to use PubMed, maintained by the National Library of Medicine.[6]

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[3] Adapted from the National Library of Medicine: **http://www.pubmedcentral.nih.gov/about/intro.html**.

[4] With PubMed Central, NCBI is taking the lead in preservation and maintenance of open access to electronic literature, just as NLM has done for decades with printed biomedical literature. PubMed Central aims to become a world-class library of the digital age.

[5] The value of PubMed Central, in addition to its role as an archive, lies in the availability of data from diverse sources stored in a common format in a single repository. Many journals already have online publishing operations, and there is a growing tendency to publish material online only, to the exclusion of print.

[6] PubMed was developed by the National Center for Biotechnology Information (NCBI) at the National Library of Medicine (NLM) at the National Institutes of Health (NIH). The PubMed database was developed in conjunction with publishers of biomedical literature as a search tool for accessing literature citations and linking to full-text

The advantage of PubMed over previously mentioned sources is that it covers a greater number of domestic and foreign references. It is also free to use. If the publisher has a Web site that offers full text of its journals, PubMed will provide links to that site, as well as to sites offering other related data. User registration, a subscription fee, or some other type of fee may be required to access the full text of articles in some journals.

To generate your own bibliography of studies dealing with myalgia, simply go to the PubMed Web site at **http://www.ncbi.nlm.nih.gov/pubmed**. Type "myalgia" (or synonyms) into the search box, and click "Go." The following is the type of output you can expect from PubMed for myalgia (hyperlinks lead to article summaries):

- **A 38 year old military pilot referred with complaints of profound malaise, fever, rigors, myalgia, intermittent headaches and nausea of 2 days duration.**
  Author(s): Murdock MC.
  Source: Aviation, Space, and Environmental Medicine. 1991 February; 62(2): 185-6.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=2001222

- **A 59-year-old female with increasing dyspnoea, an unusual rash and myalgia. Diagnosis: dermatomyositis with associated interstitial lung disease.**
  Author(s): Botha JA, Carney IK.
  Source: Respiration; International Review of Thoracic Diseases. 1999; 66(4): 377-9.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=10461091

- **A Belgian case of the eosinophilia-myalgia syndrome.**
  Author(s): Naeyaert JM, Cuelenaere C, De Bersaques J, Platevoet D, Kint A.
  Source: The British Journal of Dermatology. 1991 March; 124(3): 303-5.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=2018743

- **A case of L-tryptophan-induced eosinophilia-myalgia resulting in death.**
  Author(s): Marks DR.
  Source: Conn Med. 1990 October; 54(10): 552-4.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=2265542

- **A case of Zieve's syndrome presenting with myalgia: not to be confused with polymyalgia rheumatica.**
  Author(s): Martin JC, Ross A, Watson D, O'Sullivan MM.
  Source: British Journal of Rheumatology. 1996 May; 35(5): 495-6.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=8646447

journal articles at Web sites of participating publishers. Publishers that participate in PubMed supply NLM with their citations electronically prior to or at the time of publication.

- **A comparative study of tissue distribution and excretion among three substances implicated in eosinophilia-myalgia syndrome.**
  Author(s): Adachi J, Ueno Y, Tatsuno Y, Gomez M, Smith CC, Sternberg EM.
  Source: Advances in Experimental Medicine and Biology. 1996; 398: 365-70.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=8906290

- **A comparative study on antibodies to nucleoli and 5-hydroxytryptamine in patients with fibromyalgia syndrome and tryptophan-induced eosinophilia-myalgia syndrome.**
  Author(s): Klein R, Berg PA.
  Source: Clin Investig. 1994 July; 72(7): 541-9.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=7981584

- **A comparison of behavioral and educational interventions for fibromyalgia.**
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