AMERICAN ASSOCIATION FOR
CHRONIC FATIGUE SYNDROME

FOURTH INTERNATIONAL
RESEARCH, CLINICAL AND PATIENT
CONFERENCE

PROCEEDINGS
Saturday - Monday, October 10-12, 1998

CHRONIC FATIGUE SYNDROME

Hyatt Regency Hotel
Cambridge, Massachusetts, USA

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AACFS RESEARCH CONFERENCE

9:45 AM -11:15 AM

CLINICAL

Saturday October 10, 1998

State of the Art in Clinical Studies:

THE MEASUREMENT OF 5-HIAA URINARY CONCENTRATIONS AS A PREDICTIVE MARKER OF EFFICACY OF NADH IN CHRONIC FATIGUE SYNDROME

A PROSPECTIVE COHORT STUDY OF POST-INFECTIVE FATIGUE: ILLNESS CHARACTERISTICS AND RELATIONSHIP TO IMMUNOLOGICAL AND PSYCHOLOGICAL DISTURBANCE
Andrew Lloyd, Ian Hickie, Ute Vollmer-Coana, Michael Douglas, Catherine Brennan, Sandy Beynon, and Denis Wakefield.

EFFECT OF GROWTH HORMONE TREATMENT IN PATIENTS WITH CHRONIC FATIGUE SYNDROME: A PRELIMINARY STUDY
G. Moorkei, H. Wijnants, R. Abs

DURABILITY OF THERAPEUTIC BENEFIT WITH AMPLIGEN® TREATMENT OF CHRONIC FATIGUE SYNDROME (CFS) AS MEASURED BY THE KARNOFSKY PERFORMANCE SCORE (KPS)
David R. Stayer, William A. Carter, Thomas J. McCarron.

A PHASE I TRIAL OF AUTOLOGOUS EX VIVO EXPANDED LYMPH NODE DERIVED CELLS AS IMMUNOMODULATORY THERAPY IN CFS
Nancy Klimas, Mary Ann Fletcher, Kevin Maher, Roberto Pina, Jean Walling, Mack Smith, Lauren Vlack, and Herb Brester

REHABILITATION FROM WORK DISABILITY DUE TO CHRONIC FATIGUE SYNDROME
David C. Klonoff, M.D.
Chronic fatigue syndrome (CFS) is a disorder characterized by prolonged, severe fatigue that persists six months or greater in duration and a multitude of symptoms including neurocognitive dysfunction, flu-like symptoms, myalgia, muscle weakness, arthralgia, low-grade fever, sore throat, headache, sleep disturbances and swelling and tenderness of the lymph nodes. The overall goal of the present study was to evaluate the efficacy of reduced nicotinamide adenine dinucleotide (NADH), a natural substance known to trigger energy production through ATP generation and to alleviate symptoms of depression, in a group of 26 patients with CDC-defined CFS using a randomized double-blind, placebo-control crossover design. Medical history, physical examination and laboratory tests were performed at baseline, 4, 8 and 12 weeks. Subjects were randomly assigned to receive either 10 mg of the oral stable form of NADH (ENADA) or placebo at week 0 for a 4-week period, followed by a 4-week wash-out period, followed by a final 4-week period in which subjects were crossed over to an alternate regimen. No significant adverse effects were observed related to the oral form of NADH. The efficacy was measured by an arbitrary scoring system reflecting the symptoms of the patients as well as by laboratory tests, including plasma neurotransmitter concentrations and the urinary concentrations of serotonin metabolites i.e. 5-hydroxy indole acetic acid (5-BIA). There were 8 patients who showed >10% improvement while on the drug in contrast to 2 while on placebo. Assuming that these improvements came from two independent samples of 26 patients the success rate for the drug is 31% and for the placebo 8% (p < 0.05). The urinary concentrations of 5-HIAA were elevated prior to treatment in 50% of the patients. Following NADH treatment, these elevated 5-HIAA concentrations returned to the normal range, whereas with placebo they remained elevated or increased further above the upper normal range. Further, 18 (72%) study patients thus far enrolled in a longer open label follow-up study reported significant improvement in clinical symptomatology and energy levels. The results of the present study not only suggest that NADH is a safe, naturally-occurring biologic substance which may be a useful therapeutic adjunct in the management of chronic fatigue syndrome, but also that the measurement of urinary 5-HIAA may serve as an important predictive marker of neurocognitive dysfunction, as well as an objective measure of improvement following therapy.