FOCUS ON INFLUENZA
With this issue, we begin a two-issue series focusing on pandemic influenza. In this issue, see Influenza: Lessons for the clinic from 1918, below, and Cytokines and Herbal Therapeutics in Influenza on page 8. In our next issue, we will feature three clinical articles: Influenza prevention, Therapeutics for acute febrile illness, and Therapeutics for the acute cough.

Influenza: Lesson for the clinic from 1918
by Paul Bergner

Abstract: Avian influenza and the potential threat of a major influenza pandemic have been among the top news stories over the past year. Public health concern is high because of the high human mortality rate associated with bird flu and fears of the potential for emerging strains to spread from human to human. Coupled with the news stories are accounts of the deadly 1918 influenza pandemic. The “superbug” theory of pandemic influenza generally attributes all excess mortality to the strength of the pathogen and ignores all other factors involved in host resistance. This theoretical stance does not withstand close critical examination, particularly in light of what is historically known about the 1918 influenza pandemic. Factors such as urbanization, micronutrient malnutrition, poor diet, vitamin D deficiency, and host-weakening iatrogenic disease may have contributed significantly to mortality in the 1918 pandemic, and these factors may have been as important as the pathogenic potential of the virus. An examination of these factors from 1918 leads to recommendations for prevention or treatment during future pandemics.

By their very nature, viruses constantly mutate and evolve, partly in response to host resistance in general, but also in response to the specific host carrier. Influenza viruses mutate and evolve in human, bird, and pig hosts, and spread between these hosts in agricultural areas. Viral strains may evolve characteristics that make them

Echinacea in autoimmunity revisited
by Paul Bergner

Abstract: Case studies of aggravation of several auto-immune conditions by echinacea are presented, and a physiological mechanism is proposed.

A German regulatory commission lists several contraindications for echinacea, including “progressive systemic diseases like tuberculosis, leukosis, collagen disorders, or multiple sclerosis.” (Blumenthal) This class of diseases would include auto immune conditions such as systemic lupus and other autoimmune connective tissue conditions. This contraindication has been criticized by some medical herbalists, citing a lack of case-based evidence in the scientific literature. Ten years ago, I researched the question of echinacea contraindication in autoimmunity thoroughly, and published the opinion in the book The Healing Power of Echinacea and Goldenseal (Prima Publishing, 1997) that echinacea was not so contraindicated. Honest clinicians are always ready to eat their words or their previous opinions, when clinical facts get in the way, and especially when a patient may be hurt, and I now have to do the same.

In ten years of searching scientific literature and interviewing both physicians and scientists on the question, I had been unable to find a single case or even a rumor where echinacea in oral doses made any auto immune disease worse. Within a month of the book publication, however, I met my first patient with a severe exacerbation of an autoimmune disease possibly due to taking echinacea. She ended up hospitalized with autoimmune nephritis after taking the tincture for 3 days. A month later, one of my long term lupus patients told me that echinacea had caused a worsening of flare-ups before she became my patient. And then a third reported on intake that she had an aggravation of ulcerative colitis within twenty-four hours of beginning to take echinacea for the first time. Subsequently I have collected 17 case studies where echinacea caused an exacerbation of several autoimmune conditions, with 15 of those cases reported by professional medical herbalists or found on history among my own clients. Two of these cases – one in systemic lupus

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and the other in multiple sclerosis occurred at least twice on re-challenge, constituting the highest level of reliability in adverse event reporting. I have also received reports of multiple patients who were not aggravated by echinacea, despite taking it regularly, so this must be regarded as a potential problem but not a universal one.

CASE #1

The first was a case of unspecified auto immune disease with damage to the kidney. The kidney component of the illness was being controlled by unidentified immuno-suppressive drugs, and the dose had recently been reduced by half. About a week later, the individual began to get a sore throat, and took echinacea for it in unspecified doses. The pathogen affecting the throat was also unidentified. Very soon the kidney inflammation returned, and the patient was hospitalized to receive dialysis. The flare-up could have been caused by the reduction of the medication, or by the organism that provoked the sore throat, but echinacea must still be held suspect. The patient, a professional herbalist, thought that echinacea was in fact responsible.

Case #2

The patient had severe systemic lupus with kidney inflammation, poorly controlled by steroids, methotrexate, Plaquenil, and other drugs. She reported that when she took echinacea during an active lupus attack, many of the symptoms would get worse. She repeated this several times with the same results, and the symptoms would subside when she stopped the echinacea. She is now completely off drugs and managing the lupus with diet, herbs, and other lifestyle factors, but at least one rechallenge after achieving stability on this regimen with echinacea had the same effect. This patient is a professional herbalist.

CASE #3

The third patient had ulcerative colitis reasonably well-controlled with steroids. She had never taken echinacea before. When she heard about it, she thought it would be good for her. Within twenty-four hours of beginning to take a dropper three times a day, the ulcerative colitis flared up with a severe attack of bloody diarrhea. This dose of echinacea can elevate with white blood count within twenty-four hours, and could reasonably be the culprit in this case.

CASE #4

A middle-aged woman with multiple sclerosis predominantly used natural methods and diet to control the disease, with occasional exacerbations during the course of a year. Over a period of several years, she used echinacea tincture to treat a transitory respiratory infection. On three occasions, the echinacea caused a flare-up of her condition, in each case on the third day of three or four times daily dosing of the tincture.

POSSIBLE MECHANISM

Research into factors that balance the immune system in the past decade provide a likely mechanism for the above cases. The T-helper cells that activate the specific immune response are divided into two types. The TH-1 helper cells promote the production of more TH-cells, macrophages, and T-killer cells, and is ultimately responsible for removal of antigens. TH-2 helper cells promote the proliferation of B-cells, and trigger their promotion into antibody-producing plasma cells. The plasma cells migrate throughout the body, settling in the connective tissue. Their antibodies are responsible for tagging antigens, initiation the inflammatory and immune responses, but do not directly remove the antigens challenging the system. These two systems work in balance, one arm tagging the antigen for removal, and the other removing it. Various factors in modern life, such as stress or immunizations, may cause this system to become imbalanced, with the pro-inflammatory TH-2 cells predominant, and the antigen-clearing TH-1 cells deficient. This imbalance is common (though by no means universal) in some conditions of autoimmunity and allergy and is also strongly associated with chronic fatigue syndrome. For a full discussion of the pathology of TH imbalances, See Bergner 2004.

Echinacea rapidly stimulates white blood cell production, with a peak at about 24 hours (Bergner 1994; Zwickey), and also increases antibody production from the TH2, B-cell, and plasma cell system. This is beneficial in a balanced system, but increasing production of TH-2 white blood cells in an already imbalanced system, and thus preferentially promoting inflammation-stimulating antibodies can reasonably be expected to produce an exacerbation in the individual with such an imbalance. Many cases of autoimmunity involve the generation of cross-reactive auto-antibodies to various food antigens or microorganisms and echinacea may stimulate the generation of such auto-antibodies. In an unbalanced system, this effect might outweigh any potential benefit of removing the antigen via the TH-1 system. Not all autoimmune patients have an imbalance of the TH system, and even those with a condition such as systemic lupus may have it only intermittently. This may explain why some patients have an adverse reaction to echinacea and others may not at any one time.

The above model for echinacea’s aggravation of a TH balance is supported by frequent complaints of exacerbation of chronic fatigue or fibromyalgia by echinacea. These conditions have a high correlation to TH-2 dominance in the scientific literature (Bergner). While echinacea may benefit some individuals with chronic fatigue syndrome, those with such

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imbalance may be most likely to have the adverse event.

Of the seventeen case reports I've received, eight included exacerbations on removal and later rechallenge. Cases 3 and 4 reported here included multiple rechallenges. Thus we have a reasonably large number of case reports, nearly all from professional herbalists, several of these reporting on their own health or on the health of family members, reported by multiple individuals. Many of the cases reach the standard of re-aggravation on rechallenge, and we have a reasonably possible mechanism. The above constitutes the highest standard of evidence in the study of potential adverse effects of a therapeutic agent.

REFERENCES


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