Clinical ecology, whilst not a recognised medical specialty, has some interesting observations about the effects of toxic substances on the human body.

Clinical ecologists such as Dr. William Rea, founder of the Environmental Health Center in Texas, have followed the ideas that originated in the 1940s with Dr. Theron Randolph, who asserted that allergies to foods and common substances could cause non-specific symptoms such as fatigue and confusion.

They hypothesise that repeated small exposures (or a single high exposure) to environmental agents can sensitise individuals and cause a malfunction in the immune system.

Rea defines chemical sensitivity as "an adverse reaction to ambient doses of toxic chemicals in our air, food and water at levels which are generally accepted as subtoxic."([i])

He contends that the reaction will depend on the organ affected, the nature of the toxin, individual susceptibility (genetic, general state of health etc.), length of time of exposure, other stressors and the derangement of metabolism resulting from the original insult.

Looking at Rea's theories about Multiple Chemical Sensitivity ([ii]), chemically-induced arachnoiditis seems to fit well into his proposed scenario.

For instance, in people who have undergone one or more oil-based myelograms, (a high initial insult), there seems to be a higher than normal incidence of the development of multiple allergies (and indeed to autoimmune conditions).

Multiple Chemical Sensitivity (MCS) manifests itself in a broad manner, with non-specific symptoms such as: skin: sores, rashes etc.; eyes: redness, burning, blurred vision; ears: dizziness, balance problems, tinnitus; nose: congested, nosebleeds; throat: dry, hoarse voice; chest: pain, shortness of breath etc.; gastrointestinal: nausea, vomiting, cramps, diarrhoea; menstrual: irregular periods; musculoskeletal: muscle and joint pain; nervous system: fatigue, headaches, memory lapses, depression, etc.

Rainville et al. ([iii]) suggested that

"The study of pain may be relevant to the study of chemical intolerance (CI) in many ways".

They noted that pain is often reported as a symptom of CI, and that CNS plastic changes in persistent pain states "may share some similarities" with those seen in sensitisation to environmental chemicals.

They pointed out that functional brain studies have shown that acute pain is accompanied by activation of a wide network of cerebral regions including the thalamus, which has been shown to be involved in neuropathic pain.

Rea has described phenomena ([iv]) such as total body load (total toxic load) which is the amount the body can tolerate (the immune system is like a barrel filled with water, once it is full, it starts to overflow); adaptation (the body adjusts to acute toxic exposure, which depletes its resources, at which point the system can no longer cope, adaptation may mask sensitivity).

Bipolarity (exposure to pollutants may not cause immediate reaction, but effects may be demonstrated by ?withdrawal symptoms' after the exposure ends); spreading: sensitisation to one chemical leads to reaction to other chemicals; switch phenomenon: transient symptoms migrate from system to system, e.g. joint pain followed by diarrhoea, followed by palpitations.

Assumedly, chemical injection into the spinal area causes the Total body load to be exceeded.

As this is directly into the epicentre of the body's neuroimmunomodulatory system, the effect may be more dramatic than in chronic insidious environmental exposure and therefore arachnoiditis might serve as a very useful scientific model for the effects of toxins on the body.

Whilst MCS has yet to be accepted in mainstream medicine, the effects of toxins on the body (e.g. the Camelford incident, silicone implants) are now being recognised and to some extent the explanations offered by doctors such as Rea may help to explain why arachnoiditis sufferers tend to reach a plateau but may then experience a minor trauma which triggers rapid deterioration.

One possible explanation is deadaptation: the ?barrel' is full and overflowing and the system is unable to continue to compensate. (In effect, this may be akin to the straw that breaks the camel's back, so to speak).

Chemically induced arachnoiditis (CIA) seems to involve a chronically-hypersensitised CNS, with substantial autonomic effects and centrally-originating pain. This chronic "red-alert" situation then seems to trigger autoimmune problems, presumably via neuroimmunomodulation.

Cruse et al ([v]) discuss dysregulation of the sympathetic nervous system seen in spinal cord injury patients (which is similar to that seen in diffuse arachnoiditis) and the consequent effects on the immune system.

The sympathetic nervous system is known to be linked with the immune system ([vi]), via the hypothalamic-pituitary-adrenal axis, with cytokines being seen as the immune mediators involved. Sciatic denervation in mice ([vii]) has been shown to cause an increase in cell-mediated immunity.

Perhaps another explanation can be advanced: General Adaptation Syndrome
Hans Selye looked beyond the body's immediate response to stress and observed that:

(a) Long term exposure to stressful situations can deplete the organism's ability to maintain the stress response, and

(b) The pattern of these deleterious effects is independent of the source of stress.

In 1956, he outlined a three-stage progression of responses to stress termed the General Adaptation Syndrome : Alarm, Resistance and Exhaustion.

Stage of Alarm . When a stressor is first encountered, the initial series of responses depends upon the autonomic nervous system, the immune system and other defences to cope with the emotional, behavioural and physiological aspects of the stressor.

Stage of Resistance . Involves maintenance of this reaction to the stressor, which includes reparative processes such as fever regulation, tissue repair, control of inflammation, etc.

Stage of Exhaustion . The defences fail, metabolic reserves are depleted, physiological functions undergo a general decline, and serious illness (or even death) ensues.

Note that this general response is independent of the initial trigger event, being more closely related to the interpretation of the environment than to the physical intensity of the aversive stimuli.

In animal experiments, exposure to shock (even if unpredictable and uncontrollable) will not cause physical illness such as stomach ulcers unless the frequency of occurrence is fairly high: an occasional brief shock does not cause this problem.

However, acute trauma such as surgery can lead to the ?shock syndrome' a diffuse outpouring of the entire autonomic nervous system.

I suspect this is even more likely if post-operative pain control is sub optimal. In animals, a lack of coping response for acute, profound stressors can cause sudden death through hyperactivity of the parasympathetic nervous system (part of the ANS).

Relatively mild stressors, if not controllable by the individual, can lead to suppression of the immune system, which in turn can increase the vulnerability to diseases, trigger allergies, or lead to autoimmune disorders.

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[i] Rea WR et al Considerations for the Diagnosis of Chemical Sensitivity IN Talamge DW et al. *Biologic Markers in Immunotoxicology* Washington DC National Academy Press, 1992, p.169

[ii] Rea et al, Considerations for the Diagnosis of Chemical Sensitivity , From ATSDR publication Multiple Chemical Sensitivity: A Scientific Overview.

[iii] Rainville P, Bushnell MC, Duncan GH. *Ann N Y Acad Sci.* 2001 Mar;933:130-41. Representation of acute and persistent pain in the human CNS: potential implications for chemical intolerance.

[iv] Rea WJ *Chemical Sensitivity: Principles and mechanisms* Lewis Publishers, Inc., Boca Raton, FL, 1992

[v] Cruse JM, Keith JC, Bryant ML, Lewis RE Jr, *Immunol Res* 1996;15(4):306-14 Immune system-neuroendocrine dysregulation in spinal cord injury.

[vi] Friedman EM, Irwin MR *Pharmacol Ther* 1997; 74(1):27-38 Modulation of immune cell function by the autonomic nervous system.

[vii] Kubera et al *Int J Immunopharmacol* 1997 Jan; 19(1):25-9 Effect of sciatic denervation on cell-mediated immunity .