

A PRIMER IN SICK BUILDING SYNDROME

LESSONS
FROM THE
SOMERSET COUNTY DISTRICT COURT

By
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There are tremendous variations in the kinds of buildings that can become home to toxin-forming species of fungi. Any building that provides the proper mix of food and water can potentially be at-risk. When the building has air circulation that is closed, with little outside air input and windows that don't open, any intrusion of water can become the source of fungal blooms. The variations on how the building became sick (water intrusion through leaky roofs, windows or doors; wicking of water along a concrete slab or saturation of carpets; and pooling of surface water in basements come to mind) are relatively few. The Somerset County District Court Building in Princess Anne, Md., provides a great example of the most common problems seen in typical sick building syndrome (SBS) site investigations. This is a true story; it has been presented on TV, discussed in the newspaper and on radio.

It was the judge who called me, "Ritchie, I think I have a sick building here. The entrance foyer smells like a four day old wet sock, you can see the black mold on the ceiling tiles and all of us are sick." It seemed ironic; the debate about whether or not a building makes people sick usually ends up in court (or is settled before trial). This one already was in court! I agreed to do a site visit and test everyone who worked there using visual contrast sensitivity (VCS). Contrast testing is such an elegant diagnostic device-portable, non-invasive, reproducibly reliable, fast and low cost. A neurotoxin history, an essential part of the case definition

of a SBS patient, is so easy to do once you learn how (it takes less time than asking a good cardiac history!), so I was ready to go.

The case definition of SBS isn't too complicated. You need to show exposure, a distinctive grouping of symptoms, presence of biomarkers, especially VCS, have no confounding exposures, respond to cholestyramine (CSM), proven to be effective when prescribed properly, as in our time-tested protocol, show relapse with re-exposure off medication, and again respond to CSM treatment. Seeing the biomarkers, blood tests, neurotoxicologic tests and physiologic measures of blood flow in the neural rim of the optic nerve, change in step with improvement or worsening of symptoms was important confirmation of the neurotoxic basis of SBS.

"Don't forget your CSM, Ritch," I could almost hear my wife say. No problem there. Over the past 4 years, I have sampled molds and tested patients in so many buildings that have subsequently made me sick that I don't forget to take my mycotoxin (fungal toxin) binding medication before exposure. I can just about tell within 20 minutes after entry when mycotoxins are present. There is nothing else that gives me that distinctive hot taste on the sides of my tongue, queasy stomach, headache and sensitivity to the fluorescent lights found in nearly all office buildings. After being in the buildings with the worst mold contamination, I end up being extra-sensitive to smells, too. Visitors to my office after I have been testing in such a building of-

ten ask why a fan by my desk blows upward next to the computer. Get those fumes away from me! Fortunately, CSM, taken as a preventive measure, blocks the group of symptoms that define the acquisition phase of illness caused by toxin-forming fungal species. Maybe this time I won't again be forced to have the papers on my desk weighted down while my sensitivity to many chemicals is again being treated successfully.

So armed with my standardized light source, symptoms lists, VCS equipment and score sheets, I'm off to the jewel of the judicial system in rural Somerset County, Md., about 15 miles from home. The single-level, colonial style building is only about 5 years old. Built on a concrete slab and surrounded by a paved parking lot, with marginal drainage at best, it has several suspect angles in the roof, and moisture-retaining carpet covering all the concrete. If the roof doesn't leak, just the run-off from the pavement could provide the moisture necessary to provide favorable habitat for growth of any number of genera of fungi. The dry-wall and composition ceiling tiles provide a welcome source of nutrients for fungi too. With the water (possibly) entering through the roof or from the outside, the mold could grow freely, hidden from view on the out-of-sight side of the cellulose construction materials. To be a fungus finder, one has to look where the sun doesn't shine!

The building was built to satisfy all codes (we might use the "Nuremberg Trial" argument here that the real culprit in the explosion of the number of sick build-

ings, like this one, is a lack of understanding of fungal adaptation by those who write the building codes - does that mean those who know the codes are inadequate have an ethical obligation to surpass them, thereby increasing building costs? Quality at the start can save suffering and lots of money spent on remediation in the long run). It really isn't anyone's fault that the sewer pipes backed up shortly after the building opened. The indoor "flood" from the malfunctioning exhaust side of the plumbing might have started the fungus ball rolling. We will never know if that first water intrusion created the sanctuary that later nourished impressive numbers of *Stachybotrys*, *Aspergillus* and *Penicillium*. We can't dump all the blame on the plumbers because the front entry way lets in water whenever the wind comes from the West, as it usually does, except for the occasional Nor'easter that buffets this small Chesapeake Bay town. And the framing carpenters who are responsible for moisture seals around the doorway are off the hook too, because the concrete slab, poured as a foundation to save money, was like a fungal roller rink due to water draining off the parking lot. Why didn't someone recognize that concrete slabs are portals of entry, especially in low-lying, high water-table areas? Sure, when the roof leaked, everyone noticed it. Just imagine the scene when an indoor trial was postponed because of rain!

Most sick buildings don't have this many sources of water intrusion. It usually is easy to isolate one source of water entry, like a roof with inadequate flashing or a basement with inadequate ventilation. Being able to pinpoint negligence gives attorneys an open invitation to get to work. Lawyers often pick out a responsible party, usually one with deep insurance pockets, as a target of negligence suits. Yet all the litigation, settled claims, abandoned buildings (how about the burned buildings!), and expensive retrofits won't do what our treatment protocols do: restore health of affected patients. We hope that we can identify and treat the patients in time, before added adverse downstream health effects caused by the pro-inflammatory cytokines, released in response to mycotoxin exposure in susceptible patients, either damage critical immunomodulatory hormone pathways in the hypothalamus or alter the normal defense mechanisms in mucus membranes, or (even worse) both. Once the exposure has gone on too long or with too much intensity, we have to do a lot more to help SBS victims regain energy, cognitive function and quality of life. Don't think for a minute that ongoing mycotoxin exposure is benign. When I hear someone (especially someone with political power) say that a little mold isn't harmful, I wonder if they realize how dangerous their misinformation can become.

Just imagine if the sick building is a school. When will we know if it is too late? Low scores on standardized tests? An increase in learning disorders? A rise in Ritalin prescriptions? An increase in absenteeism? Low SAT scores years later? The answers to these questions will only arrive when VCS testing becomes a mandatory part of the yearly school health

evaluation, just like a hearing test and a visual acuity. And that will happen only when groups like the PTA demand action and no longer put up with empty statements, like "mold isn't harmful."

So who was negligent in the construction of the courthouse? Who do the victims sue? Which water intrusion allowed the mold to grow? Who should pay for clean up and remediation? If everyone can blame someone else, who lets the bill stop on his or her desk? Why were so many fungi found? Which fungus made the judge sick?

When patients ask me why we rarely see "monocultures" of fungi in sick buildings, the answer is simple. A sick building simply lets fungi survive, reproduce and release mycotoxins and other compounds into the air the inhabitants breathe. With many organisms able to grow, and given food, water, cover and a chance to reproduce (the building amply provided for all of those), it is a fact of biology that multiple species will compete for small niches well enough to survive. Every time the front door opened, for example, or when the judge came in on Saturdays to do some paper work, wearing his gardening shoes, a new opportunistic invader could have been introduced.

Just look at a drop of pond water or the rich sediments of the Pocomoke (Md) River in a microscope. If there hasn't been some catastrophe of natural selection (see related articles on this website on *Cylindrospermopsis* and *Pfiesteria* for examples of that kind of catastrophe), we will see a rich diversity of species. One may reasonably ask, why isn't a diversity of species of fungi always identified in sick buildings? Do we know why some buildings have a dominance of one kind of toxin-forming species over another? While these questions have merit, and do need to be answered, we have to remember that treating the illness begins with recognition. The current practice of "no testing, no recognition of illness, therefore the building isn't sick" has to stop.

What isn't any surprise to users of www.chronicneurotoxins.com is how easy it is to find the distinctive grouping of symptoms presented by those who work in a sick building. In the Somerset County courthouse, nearly everyone had symptoms: fatigue, weakness, aches, cramp, unusual stabbing pains, sensitivity to bright light, tearing, blurred vision, headaches, sinus congestion, cough, shortness of breath, abdominal cramping, rashes, skin sensitivity, memory impairment, confusion, difficulty in concentration, impaired ability to concentrate or find words in conversation, metallic taste, numbness, tingling, vertigo and mood changes too. All were found as a distinctive grouping. No, this wasn't depression or mass hysteria. This was a classic presentation of chronic, neurotoxin-mediated illness. Not all patients had all symptoms and the symptoms they had changed from day to day, but there were no days without any symptoms for the affected employees.

The VCS scores were predictably abnormal. They all showed the typical inverted "U" shape, with the VCS deficit greatest

in the mid-frequencies. This curve is routinely recognized in patients with neurotoxin-mediated illnesses, but it isn't specific for one type of neurotoxin compared to another. It simply tells the experienced investigator that the presence of health problems are clearly linked to the effects of biologically produced toxins. Eliminating confounding exposures, an essential part of the SBS case definition, took some additional time. In the end, we had 6 sick workers on the judge's side of the courthouse, each of which had prolonged, close exposure to the greatest concentrations of mold. Four other workers, on the "other side" of the partitioned offices, agreed to be tested, resulting in identification of 2 more cases.

Blood tests showed the typical normal values for all the standard parameters we use in medicine, except for the elevated levels of particular pro-inflammatory cytokines that until now have not been measured by other investigators. I didn't waste any money or time testing for antibodies to the fungal species: a positive antibody test tells me nothing about time of exposure, location of exposure, duration of exposure to fungi and certainly nothing about exposure to mycotoxins. And I didn't waste any time or money giving patients anti-fungal medications for treatment either: the illness is neurotoxin-mediated. It isn't an infection. Every time I see a SBS patient taking fungus killers, I cringe. The drugs are not benign. They provide marginal benefit, likely by stabilizing production of inflammatory cytokines, which is short lived. As long as the mycotoxin load is present, taking antibiotics is a prescription for failure.

The attitudes of the employees regarding my findings were mixed. Some were genuinely relieved to know that there really was a problem and that they had reason to hope that they would feel normal again someday. Some were mad at the building owner who (apparently) had cleaned up the mess, installed new tiles when the roof leaked and fixed the carpet after the flood, and said everything was fine. Some were scared; if they spoke out and said they were sick, they might lose their jobs. Some were downright devious, confabulating absence of symptoms (one red-eyed employee from the "other side" of the contaminated areas adamantly stated that she never had any redness of her conjunctival membranes). Another said his ability to assimilate new knowledge was fine, but it took 4 separate explanations for him to learn how to do the VCS test. His ploy appeared to be a transparent attempt to sabotage the investigation. Still, despite the subterfuge, we had not just one sick patient (a complainer) or two (a conspiracy); we had at least three and three makes a cohort deserving of a careful health investigation. No cover-ups allowed.

The judge and several others were real sick. They started on my treatment protocol right away, with prompt improvement both in VCS scores and abatement of symptoms. When the medication was stopped and they continued to work in the building, relapse began within 36 hours. Fortunately, the site investigation came at a time when I had the Heidelberg Reti-

nal Flowmeter in my office, on loan from the manufacturer, Heidelberg Engineering. I was able to show a clear deficit in velocity of flow of red blood cells in capillaries of the neural rim of the optic nerve head and the lamina cribosa (the tissue that separates the optic nerve from the retina). Flow was maintained in the retina, a finding that separates victims of SBS from patients with the Post-Lyme Syndrome. With treatment, flow rates increased, beginning in 12-24 hours. With re-exposure, flow rates fell within 12 hours.

So, we had biomarkers to spare. The VCS deficits matched the elevated cytokine levels and both matched the diminished Heidelberg flow rates, confirming worsening with illness acquisition. All parameters improved with treatment. To secure the diagnosis of Sick Building Syndrome beyond any doubt, I still had to show that there were no possible variables of alternative exposures that might cause the neurotoxic syndrome in these exposed patients. In SBS screening studies (case detection studies), all we really do is document the presence of the neurotoxic effects and correlated symptoms and confirm exposure to toxin-forming mold species. But those entities won't rule out Lyme disease or chronic fatigue syndrome, for example. Beyond that, some patients might have multiple sources of fungal exposures or multiple contacts with neurotoxins.

We take the patient as we find him! If he had sinus congestion unrelated to mold exposure, he will still have it following treatment. But having sinus congestion from another source does not rule out the significance of mycotoxins in genesis of the chronic, neurotoxic illness.

So what we have to do are repetitive exposure trials. These trials essentially use the patients as markers that pinpoint the exact exposure that makes them sick. After the patients were clinically cured with the CSM protocol (used exactly as prescribed, with no alterations because some attending physician hadn't "heard" of the protocol before), and remember, they still were working in the building, they went back to work. Call them a blank slate now, if you will. Only this time, they were in the building without the protection of CSM. All confounding exposures were recorded.

The instructions were clear: if the medical symptoms recur, come in for repeat testing immediately. Sure enough, within a week, the full-blown syndrome was back, with VCS deficits, reduced capillary flow and return of symptoms, in *all* of the patients. There were no confounding exposures. Most patients reported that their symptoms were noticeable within 2-3 days. The CSM protocol was re-instituted, with the same statistically significant rate of recovery, accepting the fact that people are biologic creatures and biological creatures always show some variation in response. Only this time, patients didn't have any unknown consumption of Ciguatera-laden reef fish for supper, or tick bites in the back yard, or trips to algae-infested lakes around Orlando, Florida, or fishing expeditions to the Pfiesteria attack zones in the Chicamacomico (Md)

River to confound our analysis. The only exposure to toxins causing the illness: the building.

The next step was intriguing. If we could treat the illness so easily, could we prevent it as well? If so, then who cared if the building dripped *Stachybotrys* over the inside edge of the wallpaper? Who cared if the mycotoxins entered our lungs with each breath, then circulated to nerve, muscle, brain, eye, sinus, lung, gastrointestinal tract, skin, joints and around the loop again, endlessly repeating the process, making patients feel like they were living a life of pain, fatigue and cognitive impairment without hope of ever feeling like they did once before? It turns out that our prophylaxis CSM protocol worked to prevent reacquisition. Of course, even better than prevention, was removal of the affected patient from the affected environment.

CSM isn't an easy medication to take. Bloating, reflux, and constipation are predictable side effects that accompany CSM use. Sure, we can find clever ways to get around most of the gastrointestinal complaints and we have some pharmacists who can make up special preparations of CSM that won't have sugar, aspartame or unwanted additives thrown in the mix. Human nature being what it is, however, taking the time to drink a mix of CSM four times a day, on an empty stomach, 30 minutes before eating or taking any other medications, just won't happen for long unless the patient is truly motivated. We use less frequent dosing of CSM and some additional medications for maintenance, but even then, some patients stop their preventive doses. And here comes the real answer to why *Aspergillus*, growing like Spanish moss on live oaks alongside Mobile Bay, isn't conducive to quality of life, or long life for that matter.

Repeated cytokine responses and prolonged cytokine responses can take their toll on target organs. We have some patients who don't recover all their mental capacity. Some don't have all their joint pains go away. Some acquire new organisms, growing opportunistically in newly altered niches in the nose, for example, that can create their own additional cytokine responses. Even worse, some patients suffer damage to integrative neuro-endocrine pathways in the hypothalamus. These patients can go on to develop intractable pain, chronic, non-restorative sleep and unexplained weight gain.

They also usually develop increased amounts of compounds (plasminogen activator inhibitor-1 and matrix metalloproteinases) that actually *deliver* oxidized LDL cholesterol from the bloodstream to the sites of developing atherosclerotic plaques, under the lining cells of the blood vessels. Heart attacks from fungi?? We are preparing our data for publication that will demonstrate that the levels of these pro-thrombotic and atherogenic compounds increase with exposure and fall with treatment. It's true. Just when you thought that the explosion of heart disease, diabetes and obesity was due to sedentary lifestyle and increased fat consumption, now you have to fac-

tor in a huge variable: inflammatory cytokines from neurotoxin exposure. You may be interested in reading Chapter 14 of my new book, *Lose the Weight You Hate*, entitled, *Environmental Acquisition of Diabetes and Obesity*.

Many SBS patients also begin to notice that they become more sensitive to fumes, smells and chemicals. With repeated exposures, the sensitivity becomes more pronounced for some, though (Thank goodness!) not for all. In the full-blown sensitive (Multiple Chemical Sensitivity, MCS) patients, fumes coming off computers and phones, or from freshly printed reading material, or even just a ream of copy paper, can make patients sick for weeks. Our treatment protocols for MCS may bring order to this difficult-to-confirm diagnosis.

One particular patient nearly died from his/her (I can't tell you who it was) repeated exposures to the courthouse, which caused acute jaundice and near liver failure. The liver is an attack site for pro-inflammatory cytokines. Normally, the liver uses a wonderful "detox" system in which cells that line the tiny bile ducts use a transport system (organic anion transport system) to preferentially secrete negatively charged organic toxins, including mycotoxins, against a gradient into the bile. From there, the toxins go into the intestine, and were it not for the reabsorption of the toxins further downstream, the toxins would go into the toilet. CSM grabs the toxins, preventing reabsorption and escorts them into the commode. No toxin, no cytokine response, no illness.

But the transport system stops working when it is overwhelmed by cytokines. Moreover, excess levels of cytokines can stop bile flow as well. Acute cholestatic jaundice, we call it in the trade. Not good for long life, you might call it. Sure enough, this patient had a stair-stepping cytokine response to repetitive mycotoxin exposure. It was not surprising to find the markers of acute cholestatic jaundice, as the liver functions tests went through the roof. Fortunately, we have a medication that revs up the toxin transport system, defeats the pro-inflammatory cytokine effects and in this case, saved the patient's liver.

Just one more thing - why didn't everybody in the building get the same illness? Most of them did, but some were worse than others. The answer likely lies within our genetic make-up. If we look at a group of genes (immune response genes, if you will), part of the "HLA system," minor changes in order of those genes can direct minor changes in amino acids in proteins that line a cleft on the cell surface of an immune cell (T-cell), receives (processes) an antigen presented by a different immune cell. Without a normal T-cell response to a mycotoxin, there likely would be an impaired subsequent immune response. In this way, we have reason to believe that minor changes in amino acid linkages can create major changes in three-dimensional structures of proteins and, therefore, changes in the immune response. Sound complicated? It is, and I don't have answers yet that will satisfy the skeptical scientist.

So far, though, the patients with the worst SBS-associated health problems all have a particular HLA subtype, an unusual type, not found in patients with exposure who don't have symptoms. Perhaps as few as 7 amino acids are altered, but like a lock and key, one change in a tumbler makes all the difference in how the same key works in the altered lock. Does this mean that something in antigen presentation is a risk factor for enhanced damage from cytokines generated by exposure to neurotoxins? How is that possible if the mycotoxins move through fatty tissue, acting as "stealth invaders," cloaked to the immune system? Perhaps it is the defective cleft structure that prevents a normal, health-restoring antigen response when the mycotoxins happen to enter the blood stream on their way to a different fat cell. Time and additional research will tell us if we have an additional biomarker, a genetic marker, which will enable us to *recognize ahead of time* patients at-risk for more serious forms of chronic illness from exposure to mycotoxins.

While I am not ready to start testing school kids with learning disability to see if they have a particular genetic make-up, possibly creating susceptibility to the molds found so frequently in our schools (especially flat-roofed schools, where risk for water intrusion may be greatest), I am doing routine HLA testing on all my mycotoxin exposed patients, building a data base to try to answer the questions that underlie old favorites, like, "Why Johnny can't read?" and why Johnny's Mom shouldn't work in a sick building. If we find out that many kids have learning disability related to fungal toxin exposure in schools, I suspect we will find the unusual HLA subtype over-represented in the most affected children compared to those with exposure who learn normally.

This case has a happy ending. Most do not. The State of Maryland decided to move the District Court out of the building, even in the face of the expense and inconvenience involved.

We now have multiple requests to do "prevalence studies" in local buildings, where the employees feel the building makes them sick. As you might expect, those responsible for safeguarding the work place in several of these buildings aren't too happy with the prospect of health screening being done. I simply respond to them that if the building has no contamination with toxin-forming fungi, they have nothing to worry about. Simply proceed with the testing and allay the fears and concerns of the employees. Like the Pfiesteria example of several years ago, where water quality tests had nothing to do

with patients sickened by exposure to estuaries with endemic toxin-forming dinoflagellates, in sick buildings, air quality tests often end up being irrelevant to the presence of human illness. If the staffers who say they are sick show the typical mycotoxic symptoms and VCS deficits, the correct response is to figure out where the problem is, mitigate it, fix the health problems and get on with the day-to-day operation of the building. The problem arises, however, when someone (usually in management) decides to deny responsibility for maintaining a safe work place in the face of unequivocal association of building exposure to human illness. In this regard, you might find reading Chapter 10 of *Desperation Medicine* (available on the website), *The Appearance of Good Science*, enlightening. There is a repetitively observed pattern used by public officials to deny the existence of environmentally acquired illness. I see it every day.

The State of Maryland has been forced, by clear evidence of human illness following exposure to toxin-forming indoor fungi, backed up by a series of biomarkers, to take the national lead in dealing with the problem of SBS by acting in the patients' best interest at the Somerset County District Court. I hope that other states will follow Maryland's example, soon, because the fungal species that we have good reason to fear are growing in thousands of buildings. We need an organized, patient-friendly approach to diagnose and treat the many actual and potential victims of SBS. We should begin with careful symptom recording and organized neurotoxin histories, VCS testing and our patented treatment protocols. Ideally, all people would record their own baseline levels of symptoms and VCS by taking our tests before biotoxin exposure, helping to establish the diagnosis of neurotoxin-induced illness when, for example, flawed construction gives rise to fungal contamination. The lessons of the Somerset County District Court are there for all of us to use.

"Sick Building Syndrome", Part 1, was published in Filtration News, May/June 2001; "Getting Behind Sick Building Syndrome", Part 2, was published July/August 2001, and "A Primer in Sick Building Syndrome" was published May/June 2002, with permission of the author, Dr. Ritch C. Shoemaker. For more information, contact Dr. Ritch C. Shoemaker, 1604 Market St., Pocomoke City, MD 21851.

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