Owls Under the Beaver Moon
And
Lessons on the Activity of Water

Note: This essay might upset those who feel that looking for fungi in nasal rinses is an acceptable way to diagnose health problems associated with exposure to wet buildings. That idea is not supported by any science.

Note: For those who feel that mycotoxins possibly identified in urine actually indicate illness, this essay will raise major issues. See the references on normal amounts of mycotoxins in urine (see ochratoxin bibliography and trichothecene/DON bibliography) included for realization that the issue of mycotoxins in urine following dietary ingestion has been studied by reliable methods for years. The annotated bibliographies will provide evidence that mycotoxins in food we eat every day will result in measurable levels of ochratoxins and trichothecenes in just about all of us. Mycotoxins in air in wet buildings, however, are the enemy, together will all the rest of the elements found in the chemical mixtures that are found in the air and dust of water-damaged buildings that sets off similar inflammatory responses.

Note: For the rest of us, this essay is presented on several levels. This essay is the third of an ongoing series on Integrity in Science sponsored by www.survivingmold.com. The first of the series is a science-based building inspection report done by Greg Weatherman followed by the research guidelines of our non-profit research group, CRBAI.

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Words can have a life all their own. For others who love words, especially those derived from a good myth, the names of full moons are a welcome example. Moon names come to us largely from tribal Indian lore, primarily from the Algonquin Nation. Worm Moon; Pink Moon; Harvest Moon; Hunter Moon; and the Beaver Moon of November are just a few of my favorites. The forests and wetlands on Maryland's Eastern Shore were long-time Algonquin tribal grounds with the Beach to Bay Trail tracing the movement of local tribes in summer and fall. In nearby Snow Hill, the "Algonquin Trail" is the current name for the North to South (and vice-versa) trading routes of many Native American tribes. Native artifacts, from arrowheads to grinding stones are commonly found here.

We aren't the first to walk in these wooded wetlands.

It is November; we still have beaver activity in Worcester County. They live here. When you visit our lovely Nature Trail, you can see the results of beaver work adjacent to the new floating boardwalk that extends around Stevenson's Pond. We tend to think of beavers as a long-ago part of the history of our country. Beaver hats, beaver coats: lots of dead beavers. Jim Bridger and Kit Carson (the list of frontiersmen goes on) began their

careers as beaver trappers. Let us not forget to mention the monopoly on the beaver trade created by Oregon's John Jacob Astor. His fur legacy includes the name for the state university football team (no, not asters: beavers!). Yet, despite the ravaging of beaver populations caused by the odd fad of wearing a dead animal pelt on one's head as some obscure symbol, the water-dwelling rodents survived to live amidst our new world.

This past week has given full evidence that as the cold tones of the failing light of winter replace the bright contrasts of October, it is the time for beavers to shore up their dams and stock up on submerged branches secured near their lodges before winter's freeze sets in. Beavers won't budge much from their lodges as the water chills, though they won't hibernate. Inside the warmer lodge, winter is breeding time. Beavers will be busy.

This is the season we always hear the night-time calling of the great horned owls. Demanding, haunting; no wonder owls were a symbol of wisdom--or death--in so many cultures, not just the Algonquin. The owl calls in the distance are penetrating in the otherwise quiet cold night of the Beaver Moon. The answers are equally insistent. I pay attention. The owls rule this time of year from the heights of our windrow of loblollies.

The timing of mating rituals of owls coincides with the time that older owls chase away mature juveniles. I guess it makes sense; with winter coming, aggressive carnivores like owls don't need competition for fresh meat from Great Horned, Junior. Wise decision.

So it was not surprising to hear a chorus of great horned owls the other night and then for the full week that followed. Now that our forest has grown up in the East, our loblollies are a preferred vantage point for crows in the morning, eagles in the afternoon and under the moonlight of the Beaver Moon, owls.

Collectives are another source of entertainment for word lovers. Groups of owls have been called many things. Here are a few: A bazaar of owls; a wisdom of owls; and my favorite, a parliament of owls.

Big Daddy Great calls, forcefully. Junior calls nearby, then in the distance, further away and then gone as the minutes go by. Parliament is dissolving. I didn't hear Junior fly.

Owls feature prominently in countless cultures of North and South America. The great horned owl range and habitat is incredibly diverse so nightwalkers from Manitoba to Argentina have probably been entranced in fall by owl calls just as I am. The constancy of Nature is such that next year, in this time and in these similar temperatures and light conditions, I will again marvel at the horned owls. Behavior of owls remains dependent on the ecology of their environment. Next year it might be Junior Great in the loblolly chasing away the III. But owl behavior and habitat won't change. If we want to know about great horned owls, and beavers for that matter, then we need to know all we can about where they live and what they do to stay alive. The moral is both familiar and simple: "one should never be where one does not belong." If greats end up where they don't belong, the greats are long-gone.

Note: Since I am writing for a website that focuses on treating patients sickened by exposure to the interior environment of water-damaged buildings, I bet you think I am going to talk about molds and other organisms that flourish inappropriately in wet buildings. You would be right.

Beavers do what they do. We can count on their behavior to be similar in Colorado and Pocomoke. Owls do what they do. We can predict their behavior down to the moon calling. Microbes might be different in many ways from owls, but the idea of habitat, life requirements and behavior don't change. If we try to ascribe new behaviors to ancient creatures we must have proof, real hard scientific proof that supersedes the tests of time, as mere speculation does not outweigh the forces of owls, beavers and moons. We can't accept junk science and we won't accept anecdotes as proof of causation of illness. If any idea is at variance with the laws of nature, it will wither and disappear under the full glare of the light of science.

In spring and summer we hear the barred owl most commonly. "Who, who, who cooks for you," it seems to call. The greats simply demand "who, who, who, who" though there is some modulation of later whos in the sequence and the timing of the phrasing changes from beginning of the call to the end. I have no idea if the modulation reflects chasing away Junior or chasing Mama Owl. At midnight on this full moon, walking with Celia, the Dudley Labrador, the owls sound like they have bullhorns. They are just ahead!

When the owls are so close, we make sure our cat stays in. A 12-pound cat is a bed-time snack for Poppa Owl after a night of hooting, carousing and hollering. Many people know that owls are often rousted by crows during the day. The pursuit by a posse of crows, collectively called a murder of crows (no kidding), seems so unfair. Ten against one. "Poor owl. Let him sleep."

Poor owls indeed: eating crow is a favorite pastime for greats. There isn't much meat that predator won't eat! But they really love to eat crow babies. So in return, a murder of crows taunts the murderer of crows until the curtain of dusk changes the taunters into the pursued. Murder by owl tonight could befall the taunters.

We used to have mute swans and Australian black swans on our ponds. No more. They didn't belong here. One died of Aspergillus fumigatus (confirmed on autopsy; this fungus is common killer of larger birds). Another died of microcystin (from a bloom of cyanobacteria) poisoning. A third and a fourth vanished just as the owls starting calling. Could a great horned carry away a 15 pound swan? You bet. How about a 45 pound beaver? Not likely but a cat or a beaver kit weigh much less. Young'uns and little-uns must stay in at night during the Beaver Moon!

We can only wonder what is so attractive to owls about these tall loblollies with their views over the stubble of the harvested fields. One answer is easy. Food.

Night time is rabbit eating time for owls (no wonder owl pellets are called hare balls). Ever since any one with a gun around here was allowed to shoot red and grey foxes

without limit, we have bunnies and more bunnies. Owls say yum. Why aren't the owls here all year long like the bunnies are? Actually, they are. But they aren't in these loblollies making a racket.

Like the owls and the beavers, we have woodpeckers all around us. Downies will be on the same trees as the owls, year round, though they like hardwoods better. Pileateds feed on standing dead wood more than live wood. Seeing their distinctive oblong nest cavities in dead trees is a delightful find in the woods. Red-bellieds work on living hardwoods. Sapsuckers prefer conifers yet leave their rune-like signatures chiseled on elms, autumn olives and maples (among others) as well. Woodpeckers show us a lot about what makes organisms unique by how they live and where they live. We just need to pay attention to detail.

The collective for woodpeckers is a descent. No kidding. I have heard of the descent of Man, but woodpeckers ascend! This morning I watched a downy and then a pileated each move upwards searching for bark dwelling insects. Only the nuthatch was working his way down the tree. **Note:** I couldn't find a collective for a group of nuthatches. Everything has a name. Maybe a cashew of nuthatches? Or how about a filbert of nuthatches?

Ecology is all around us. Birds are wonderful indicators of ecology but in the end, habitat includes food, water, breeding sites and cover. Habitat is specialized. Just imagine what a spice bush swallowtail uses for its food and cover. Soils are complex habitats for living things. In just a few feet soil make-up can vary incredibly. Think water presence and as a delivery mechanism when pondering differences in soils. I look down; there must be three kinds of mosses in a ten foot circle around me. Those living things tell us about these soils. We can tell the water-saturation of swamp soils, for example, by the mosses and ferns that grow in one area but not just ten feet away. If we want to get technical we can further sort mosses and ferns by water needs and tolerances found in clays, loams and silts (**Note:** not today).

Forest edge plants don't grow under the canopy of cypress and gums, but thrive just a few feet away from the shadow line with slightly lower water saturation in the soils. More importantly, the edge plants must have the right activity of water in their substrate or they will die. The activity of water, "a(w)" doesn't get much publicity but is vital to life and death of loblollies and cypress just like it is for all creatures great, like owls; and small, like fungi.

Note: what is the a(w) in nasal epithelium? What is the minimum a(w) in any substrate for fungi need to manufacture mycotoxins? Hint: it is much higher than you think. Can we say anything about the conditions in the nose required for fungi to make mycotoxins? Put the question in a different way. What are the permissive requirements in sinuses in patients needed to become a fungal habitat? The answer is nothing to sneeze at.

My November morning walk with Celia is so peaceful and quiet. The kingfisher calls rattle over the ponds as the wood ducks wing-whistle off the still waters. Our pace is slow

as the virtues of various sticks and the time since the last fox marked a fence line requires discussion. And we have plenty of foxes marking our fence rows. We don't shoot them, preferring instead to enjoy their antics in fall as they attempt to feast on our persimmons hanging just out of reach on upper branches. Fox calls in their mating season is for another essay.

But then whoosh! I am thrilled even as I am startled by a woodcock flushed out of the moist bottom of a down-sloping thicket of laurels and sweet pepper bush. I marvel at the hardy mountain laurels living here as if the laws of ecology involving cold weather somehow don't apply to these odd denizens of dense groves of spiral trunks that will burst forth with blooms.

I must be missing something about these swamps: what are the laurels doing here? How can they thrive in wetlands swamps?

Winter temps, summer temps and more? Unlikely. The soil holds the answer: find the right acidity and the right a(w). We have it here and there are the laurels. Believe in a(w). Laurels do and so do the woodcocks (a fall of woodcocks is the appropriate collective but I only saw one on this fall day).

Woodcocks have long slender bills that they use to probe for and eat insects, worms and grubs in soils of a given acidity and water activity. Makes sense that the woodcocks would be living next to the laurels in our forests. We don't find woodcocks in lower a(w) soils but we might find them (rarely) in higher a(w) soils. Not so for the laurels. The idea of woodcock habitat being related to a(w) makes sense. Imagine sticking a straw in concrete and trying to suck up worms. Now put the worms and the woodcock bill in a high a(w) soil. Dinner is served.

No wonder the woodcocks are here and not on nearby Lakeland or Othello soils. The same idea applies to microbes living in and on us. If the a(w) is wrong for fungi to manufacture secondary metabolites, don't tell me about mycotoxin production in sinuses. And a(w) in respiratory mucous membranes can be far less than 0.5.

The proponents who tell us fungi in sinuses are creating mycotoxins there left a(w) out of their theories. That absence is fatal to the idea, just as feeding on concrete is fatal for woodcocks. And, even worse, what is in the nose has nothing to do with what is in the sinuses! Yet there are physicians teaching the "endogenous mycotoxin production" idea as if there were data to support the concept.

The activity of water (or water activity) is a term that comes from thermodynamics. Call it a measure of escapability of water if you will; it is the ratio of the partial pressure of water vapor from the substance compared to the water vapor pressure of pure water at the same temperature. Can water escape into the area around a substance? If so, the water is active. If not, it isn't. Microbes need water in their habitat. They won't waste energy making secondary metabolites in an arid environment. They won't turn on the genes that are involved with manufacturing mycotoxins without environmental signals indicating

the moisture around the fungus is adequate to make it worthwhile to invade and then digest plant life.

A(w) can be confusing, for sure. This term seems to be understood by food scientists far better than anyone else. I read the comments of Dr. Angela Fraser at Clemson at foodsafetysite.com with interest. Reducing a(w) is the best way to retard spoilage. Spoilage means microbial growth. No water, no growth. Water activity can be altered by temperature (freezing); saltiness; or sweetness (why doesn't honey or drawn butter (ghee) spoil?). Osmolality counts too. Add to the list chemical binding of water, like mixing water into the viscoelastic gel called mucus. That water isn't free: it is chemically trapped.

Paradoxically, nasal rinses that have an a(w) over 0.8 foment fungal production of mycotoxins. Push the a(w) over 0.85 and then watch bacteria flourish. But who uses plain water with no additives in nasal rinses? We all put our nasal solutions in saline. Don't gargle with tap water, use salt water. Why? Reducing a(w) on tonsillar tissue helps retard microbial growth, not to mention washing away inflammation compounds that are found on the surface of tonsils like bradykinins and kallikreins.

Penn Sate also has an excellent site (http://agsci.psu.edu) on water activity. Take a look. Any a(w) below 0.75 just won't grow toxin-forming Aspergilli. So where is all the putative mycotoxin growth coming from? Not the nose or sinus. Could the mycotoxins found in urine simply be coming from the food we eat safely? That idea is well-supported in many peer reviewed studies as shown by the bibliographies for this essay, understanding those references are just a small sampling of relevant literature.

The missing point in all the new-found "discussion" about fungi in noses and sinuses is inflammation. Imagine seeing that a nasal rinse showed fungi and concluding that the fungi were making people sick. Where is the inflammation that typifies patients sickened by fungal growth? It isn't there! The idea that mere presence means disease is about as illogical as saying, "Look, we found E. coli in stool. Therefore people are sick from that germ because it might make an endotoxin." Just look at the studies done on bacterial flora in noses of nurses beginning to work in an ICU. In a week they have colonization with gram negative rod bacteria that they didn't before. Are they ill?

Do the fungi found in nasal rinses in so may control patients tell about inflammation? NO! Does finding E. coli in stool tell us about inflammation? NO! Illness? NO!

If there is evidence of breach of the blood/mucus/epithelial layers in upper respiratory mucosa, there can be an inflammatory response directed against non-toxin forming fungi, creating chronic rhinosinusitis (CRS), but this response is from specific interleukins (cytokines) that are driven by TGF beta-1. Mycotoxins aren't the culprit.

But our friend mucus (unless it is too viscous as in cystic fibrosis; or not viscous enough as in bacterial vaginosis) has multiple other mechanisms to defeat the ubiquitous fungi found on mucus membranes (see Lai, S. in Adv Drug Deliv Rev 2009; 61(2): 86-100).

The high sialic acid content in mucin decreases a(w) even if the water content of mucus that approaches 90%. Water, water, everywhere but not a drop to drink. Indeed.

The lipids in mucus also enhance its hydrophobic aspects, essentially trapping microbes in a mesh of viscoelastic mucopolysaccharides and fatty acids. No water there at all despite all that water in the mucus! It is the activity of water that counts! The list goes on to include ionic characteristics, salt aspects, DNA content, protein content, cellular content, immunoglobulins, T-regulatory cells and extracellular neutrophil nets collecting hyphae as well.

Perhaps someday I will ask the red-shouldered hawks found in these swamps to show me more about mucus in sputum.

The size of invading compound matters too. Small viruses move through mucus as if it were water but larger viruses slow way down (Note: small molecules, like the 28 amino acid long structure of VIP, zip line through mucus membranes). Compare their transit time to structures like intact fungi which are a 1000 fold larger. Mucus acts like molasses for fungi! (Sugary enough, yet with such a low a(w) that no fungus grows or makes toxins in it).

Let us not forget that there are people with inflammation induced by colonization of non-toxin-forming fungi in sinuses (chronic rhinosinusitis; CRS). These were the people discussed by Drs. Eugene Kern, David Sherris and Jens Ponikau while working at Mayo and then SUNY-Buffalo. Those patients had evidence of abnormalities on CT and some of those patients improved with long term anti-fungals. But the CRS patients I have seen (just over 100) just don't have a positive visual contrast sensitivity (VCS) test. What does that mean?

Simple: it is highly unlikely that these patients are biotoxin patients! We know that 8% of patients with confirmed illness will have a normal VCS. The likelihood that normal VCS results would occur in all 100 patients is 0.08 to the 100^{th} power. That is a small number, so small that a statistician would tell us to ignore the idea that CRS patients have mycotoxins causing their illness.

But if patients with positive tests for fungi in nasal rinses are now being reported to be toxin formers as shown (only) by a non-specific test done on urine, one that hasn't been shown to separate mycotoxins from food (especially ochratoxin A and all 18 of its metabolites; and a trichothecene, deoxynivalenol, DON) from mycotoxins in indoor air, does that make us wonder about the diagnosis? You bet!

I am unsettled today by these unnatural claims made by some in the mold world but not by the glorious display of the woodcock. I would rather be visiting woodcock habitat than worrying about what I perceive to be junk science (**Note:** line by line review of several mold papers will be included later in this series. For an example of what this kind of review can do, please see ACOEM: Ploys and Lies, 2011 on the survivingmold

website). Claims about toxin production by fungi in sinuses and noses just ignore the ecology of fungi.

And then I read comments attributed to a friend from the CFS community that fungi make toxins simply for defense mechanisms to be directed against bacteria and other fungi. Nope. Not true. Funny, in a way, for years none of the physician players in the CFS community would give much credibility to my work with patients sickened by exposure to the interior environment of water-damaged buildings; now, they talk like they knew it all along. To paraphrase axioms about acceptance of new ideas: First they ignore you; then they attack you; and then they knew it all along.

Still, all any health care provider or patient needs to do is see the results of the labs our group uses; and see the results of use of our peer-reviewed treatment protocols. And then see the same things twenty more times. The world just got bigger and better for them!

The best known mycotoxin found on food (and therefore in us and then in our urine when we eat the food), the trichothecene deoxynivalenol, DON, uses its very expensive secondary product of metabolism to defeat the chitinases and peroxidases made by plants as they try to defend themselves from being digested by fungi. Call it counter-terrorism, if you will. Not a primary offensive weapon. Let's not forget the habitat for fungi includes food from plant life!

But, the truth is that I haven't interviewed a toxin to ask what its purpose in life is. And biology, being ever inscrutable, predictably is far more complex than what I think I know.

Will it matter if I challenge my friend's ideas? I did ask him for his references and none ever arrived. Will my disagreement matter to the owl or the beaver? No, and not to the woodpeckers, laurel or woodcock either. So what difference does it make?

In the world where the ends justify the means and money is the usual end sought by so many, the process of truth-seeking can get overlooked. How we get from point A to B does matter. Teaching wrong ideas does matter. Let me have you agree that 1+1=3; I can then prove just about anything illogical.

Truth, like Mother Nature, isn't for sale. As physicians, we swore oaths to be honest, truthful people. We said we would not let bad things happen to our patients if we could prevent those occurrences. I am concerned that some have lost our lessons of integrity. I worry that what we see in this era of quick and sloppy review of on-line papers actually means a more rapid approach to cashing in. I do worry that some have lost the lessons of the ecology of the woods and the wild, especially the lessons of a(w). Or maybe those lessons never were learned.

The process of science matters. There is right and there is wrong. Portraying speculation as fact in published papers and using "publication" as a means to convince an unknowing public to accept speculation as science matters. A lot.

I listen to the owls and ask as they do: who in the water-damaged building world will stand up for integrity in science? Who will stand up for integrity of patient care?

WHO? WHO? WHO? WHO?

This series on Integrity in Science will continue with a Round Table discussion on qualities of good and bad science in academic papers with guest contributors including Drs. Judy Mikovits, Russell Jaffe and Frank Crusetti. I have invited several prominent authors from the CFS world to participate as well.