

At the heart of the issue is the LaSalle Park Marina Association's call to build a permanent 400-metre breakwater — made from 10,000 tonnes of rock — to protect boats from the waves of Lake Ontario.

The problem is that wave action also keeps the water at LaSalle Park ice-free in winter, which gives trumpeter swans access to aquatic plants that are essential to their diet. If the water freezes, the swans will have to move on. But where? Kingdon and Benneian say that's one of the biggest questions when you consider that the level of human development on Lake Ontario's shoreline leaves few options.

The relationship between the swans, their supporters and the marina association — which also wants to increase its number of slips by 130 to 349 — has not always been contentious. In fact, it's been mostly peaceful since the marina was constructed in the early 1980s.

The reason is that, until the current proposal, the marina association has used a floating breakwater to protect its boats. Each year, the breakwater is pulled from the water at the end of the boating season, just before the swans arrive. It goes back in during the spring, after the swans leave for summer breeding.

"It's like a waltz," says Kingdon, who was among the original volunteers to help breed the trumpeter swan cygnets that became the foundation of today's population in Ontario. "It's so smooth.... It's total compatibility and it is beautiful."

For its part, the marina association, a local non-profit, continues to favour a permanent breakwater — the most costly of options explored for improvements to its operations — citing superior protection for boats.

The association, however, may not have the final word on its proposal. In September, the City of Burlington voted to take over the project. Its concern: the financial risk to local taxpayers should the association, which operates the marina on behalf of the city, default on loans to build the breakwater and additional slips.

Kingdon and Benneian are happy about the city's decision, saying that the process around the proposal has been flawed from the outset. But there's still work ahead for the coalition in public education, community outreach, monitoring the marina proposal process — and speaking up on behalf of the swans. 🌊

Discoveries From the Deepest Sleep

Unusual changes take place inside the brains of hibernating animals. Understanding them may shed light on the mysteries of Alzheimer's disease.

By Jay Ingram



This is the season where some of our native species are sound asleep. Some, like Arctic ground squirrels, are in a deep slumber that takes them to the very edge of death. Others, like bears, are asleep too but have not shut down their metabolisms quite so thoroughly.

No matter what the version, this sleep — hibernation — fascinates because it takes normal mammalian physiology and turns it upside-down. And there is one little-explored aspect that might provide clues to the nature of a human scourge: Alzheimer's disease.

The parallel between Alzheimer's patients and hibernators is that in the brains of both, things are slowing. The animals are able to reverse this process when necessary. Humans are not.

Alzheimer's is frustratingly complex, which is one of the reasons that we have no truly effective treatment for it. Its course—loss of memory, changes in behaviour, inability to communicate—is the outward signs of a cascade of disruptions of the brain inside. The first changes occur years, perhaps even decades, before symptoms first appear. And while there is controversy at every turn, most scientists agree that there are two particularly bad actors in the development of the disease—deposits called plaques and tangles that litter the Alzheimer's brain.

Plaques seem to kick things off. They begin life as tiny, short-chain molecules that steadily aggregate, hundreds of thousands of them, into clumps of twisted and misfolded proteins. At some point, they trigger the appearance of tangles.

Tangles are derived from a protein called *tau*, which stabilizes the elongated skeletal elements in nerves called "microtubules." Under the microscope, a single nerve cell in the brain, a neuron, looks like a tall, leafless, multi-limbed tree in a very dense forest. Each tree—the individual neurons—is superficially similar but uniquely branched, right down to the twigs. The microtubules inside the neurons stabilize and support these structures.

But neurons, at times, also extend and grow. They become plastic. Wherever that is happening in a neuron, the molecules of *tau* holding the microtubules together become plastered with phosphates (we're down in the world of chemistry now) and lose their grip. The microtubular scaffold begins to collapse.

There are two times in a human's life when that breakdown happens: in utero, when the brain is frantically building and needs its neurons to be as flexible as possible, and in Alzheimer's. Two sides of the same coin. Except that as the fetus develops, the process slows and then stops; in the Alzheimer's patient it does not.

Thomas Arendt of the University of Leipzig has led the way in investigating a similar phenomenon in hibernating animals. Working with two species with very different approaches to hibernation—Arctic ground squirrels and black bears—Arendt has shown that the same sort of brain changes occur in both.

That's surprising given the differences between the animals. When an Arctic ground squirrel enters the torpor that precedes full hibernation, its body chemistry slows and its temperature falls—dramatically. The

squirrels spend five to six months with body temperatures hovering around the freezing point. The only exceptions are occasional "wakings," the term for returns to normal metabolism that last only a few hours.

Much less startling is the back bear, whose body temperature drops by only about 7 degrees C from its normal of 37 degrees. (Being able to maintain that relatively high body temperature surprised scientists, who had expected the drastic reductions in metabolism—heart rate halved or more, one breath every 45 seconds—would have forced a lower temperature.)

Despite these differences in body temperature, both species show the same phenomenon in their brains: the microtubules in their neurons are in disarray. When hibernation ends, the neurons' structural integrity returns, and the animals waken with no appreciable mental deficits—at least in the long run. (It's clear from experiments with a variety of species that the waking-from-hibernation brain is extraordinarily active and in some cases slow to recover its pre-hibernation state.)

The parallel between Alzheimer's patients and hibernators is that in the brains of both, things are slowing. The animals are able to reverse this process when necessary. Humans are not. In people, the loss of *tau* and the disaggregation of microtubules is a foreshadowing of neuronal death and cognitive loss, a symptom of decline. In hibernators, it appears to be a protective response to the dramatic shutdown of metabolism that allows them to survive the hostile conditions of the Canadian winter. 🐾




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