

OPINION

Everyone has early cancer (they just don't know it yet)

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Nobody wants to hear the words, “You have cancer.” And yet, [40 per cent](#) of us will be diagnosed with cancer in our lifetime, with [nine out of 10](#) of those diagnoses coming after the age of 50.

Although these statistics are scary, cancer is frequently curable if found early (up to [90 per cent](#) of some common diagnoses can be cured). Many cancer types that cannot be completely eradicated can at least be controlled over extended periods. Unfortunately, 50 per cent of cancers are still detected at [later stages](#), at which point cure rates plummet.

An obvious conclusion is that finding more cancers early should be a good thing, and not nearly as upsetting or anxiety-inducing. More frequent early detection and better outcomes could, over time, help destigmatize a cancer diagnosis.

In that light, recent discoveries across many scientific publications now demonstrate that as we age, each of us develops multiple genetic mutations in our cells, some of which are commonly associated with cancers: The seeds have been planted and are slowly germinating, we just don't know it yet.

The risks of developing many types of cancer are lower with lifestyle modification – for example, by avoiding carcinogenic toxins, limiting excess UV exposure, controlling your weight and getting antiviral vaccines.

The latest science emphasizes, and in part reveals, a previously underappreciated interplay of environmental exposure and biology across a person's lifespan. As the body adapts to continuous environmental assaults, whether from sunlight, tobacco, chemicals, obesity or viral infection, our biology responds to both protect and defend us.

As in all evolution, survival of the fittest is at work, and it starts from birth, accelerates as a function of age, and culminates in the seeds of cancer being present in all of us. We all have early cancer, we just don't know it yet.

Let us explain.

After mapping the first complete sequence of a human genome in 2003, the cost of genetic analysis dropped astonishingly fast. Technology improved, the speed of analysis accelerated and the ability to look deep into the body at much greater depth became a reality. From looking at the tip of the iceberg in one individual – to the tune of billions of dollars – tens of thousands of people could now be studied at great depth, at an affordable cost.

What have we learned from this scientific progress?

An early clue came from [examining the blood DNA](#) of expectant mothers for signs of genetic differences in the fetus. In 2015, scientists were surprised to discover that these very sensitive blood tests also revealed, albeit rarely, mutations associated with cancer in the maternal blood, even though the mothers were young and outwardly healthy. Using the same technology, a [2014 study](#) of more than 17,000 “healthy” individuals had also found traces

of cancer mutations in the bloodstream of an astonishing 10 per cent of people older than 70. These individuals had a higher likelihood of a subsequent diagnosis of [blood cancers](#), and an unexpectedly increased risk of other age-associated diseases, such as atherosclerosis and its complications (stroke and heart attack).

In parallel, other large population studies have also demonstrated that about 5 per cent of adults [over the age of 70](#) will have a detectable, precancer protein marker (a monoclonal protein) in the blood. Using recent, more sensitive protein-detection technology, this [number rises](#) even further to a surprising 13 to 18 per cent of tested older adults.

So, in combining these studies, if you look hard enough at the blood alone, approximately 25 per cent of adults over the age of 70 harbour genetic or protein signatures associated with the earliest, precursor stages of cancer.

What about other parts of the human body? Being harder to access than blood, data are available on a much smaller number of individuals in this regard. To compensate, in the study of tissues such as [skin](#), the [esophagus](#) and the liver, scientists have probed at ever-greater depth, identifying a shocking minefield of genetic mutations present in all studied tissues of the body, growing more prominent every decade. It seems every cell in our body is [mutating](#) 10 to 30 times a year – or acquiring a new mutation about every two weeks. Every square millimetre of skin harbours a [genetic mutation](#). By middle age, all adult tissues explored are [studded with colonies](#) of genetically mutated cells.

It is increasingly clear then, that naturally occurring genetic mutations, including those linked to future cancers, appear in all studied organs of the body and appear to be expanding continuously over time. Given the enormous number of mutations and the high frequency of detection in adult populations, it follows that most acquired mutations are likely harmless and of no clinical consequence. Why, then, are some mutations later found to be the dominant mutation of a cancer?

This is where classical models of evolution operate. As we adapt to our environment, including where we live, what we eat, drink and breathe, our cells learn to resist toxins or stressors. Survival of the fittest takes over. A random genetic mutation that helps a cell resist will emerge dominant. DNA mutations, which offer protection, create new and stronger cells that survive and flourish.

As an example, imagine that lung bronchial tissue is damaged by cigarette smoke. A mutation that protects against the inflammation and toxicity of that smoke, and keeps the cell in better condition, will enable “survival of the fittest” amongst all other cells in that tissue. Over time, the genetically selected superfit cells become the dominant lining of the lung. Importantly, it seems that many of the same gene mutations that allow adaptation to injury may also result in cells with faster growth or the ability to survive longer. Hence the seeds of future cancer have been sown.

As further toxin damage (wrought by the smoker continuing to smoke) continues to pile up, the drive to survive continues in the lung bronchial cells. New protective mutations emerge and accumulate, eventually necessitating escape from the body’s normal growth-control guardrails. As these unconstrained colonies of cells grow without restraint, the clones of multiply-mutated cells reach a detectable size large enough to form a “tumour” and cause symptoms, resulting in what we would term, on a clinical biopsy, a lung cancer.

The good news is that, for some exposures to certain toxins such as cigarette smoke, science shows that the process is [somewhat reversible](#) when the toxin is removed – so it is never too late to stop smoking.

The “bad” news, and the now-recognized reality, is that as we age, we all carry the seeds of a cancer, in fact probably multiple cancers in multiple organs.

Fortunately, a majority of these cancer seeds will never propagate in such large numbers as to cause a recognizable clinical disease – at least not during our natural lifespan. We know this because, for example, up to 50 per cent of men have an [undiagnosed prostate cancer](#) found at autopsy. In women, approximately 10 per cent have an undiagnosed ductal carcinoma in-situ (DCIS) [of the breast at autopsy](#) – a step on the way to full-blown malignancy. Both studies are highly suggestive of a natural reservoir of early cancers in large numbers of us, unsuspected during our lifetime.

We now know the seeds of cancer exist in us all. Since most of these seeds will never result in clinically detectable cancer, more research is required to better define the critical junctures, and timing, for potential intervention during the precancer phase. These interventions might include lifestyle changes, vaccines or benign therapeutic innovations that stem or slow the tide of cancer’s progress.

The promise of finding cancer at its very earliest origins means successful intervention becomes increasingly plausible, but could be offset by the very real potential for overtreatment and increased mental stress related to a cancer diagnosis. There is also, of course, the potential impact of increased expenses for mass screening in an already burdened health care system. While advances in detection technology will increase the rate of [early cancer diagnoses](#), cures will also become more common and late cancer diagnoses should decrease. Hearing the words “You have cancer” may no longer carry the same stigma and trigger so much fear.

So yes, we all have early cancer and we don’t know it yet. But the science should inspire hope, and not fear.

<https://www.theglobeandmail.com/opinion/article-everyone-has-early-cancer-they-just-dont-know-it-yet/>

Let me pose a certain “thought experiment.” Once you get cancer of any kind, you know what you will die from. It is only a question of when, and will the fatal variety be the same as the original, or, likely, worse. Chemotherapy, radiation, and surgery can quell the initial outbreak, but at a great toll. From watching others go through chemo, we know how debilitating it can be, the nausea, depression, diminishment, and enervation. As we get older, even the fall-out from anesthetic in surgery can leave a lasting mark beyond scarring, catheters, and mastectomy.

So, is it a decision to be made whether to submit to treatment after the first diagnosis? When you know that, even if you survive the first phase of cancer, the suffering even during that treatment may well prove daunting, only to be followed by worse suffering likely after harsher treatment in a later phase that still proves unsuccessful? If someone persists with treatment anyway—in that ballyhooed “brave battle with cancer”—it may well be a desperate hanging on to life in mortal fear. It may be submitting to pressure from loved ones. If it is decided by whether “anyone will miss me—Is there anyone who cares (enough) that I care about (enough) to justify submitting to this pain?”—then we need to let the afflicted know that, indeed, we love them and dearly want them with us, but we do not ask them to suffer more for our sakes. We view life, and—if fitting, the afterlife—on broader and deeper, more enduring terms than this. It is a kind of MAiD (Medical Assistance in Dying) decision: How much choice, and what kind of choice, will I exercise in my death? The terrible fact is that we all make “choices” all life long that prove “fatal” to us, and these go beyond smoking and playing only Wi bowling ensconced on the couch. “Suicide” is something we face and flirt with, or avoid, all our lives. Sigmund said so. Countless other saner “lay psychoanalysts” who have battled with Thanatos to varying degrees and outcomes would agree. TJB