'I smoked right up until the day of the operation.'

KEN ORMEROD
RETIRED TRUCK DRIVER, LUNG CANCER SURVIVOR

WHAT IS CANCER?

STEVE BUIST
The Hamilton Spectator

THE TRAGIC PARADOX of cancer is that the disease kills people because cancer cells are programmed to be immortal.

Defining cancer is straightforward: Cancer is the uncontrolled growth and spread of abnormal cells anywhere in the body.

Incredibly, cancer starts when one rogue mutated cell out of the 50 trillion cells that make up the human body slips away from the normal controls that regulate how cells divide and how they're killed off when necessary.

Each cell carries inside its nucleus a complete copy of a person's genes — the individual blueprint that makes up each person. There are about 100,000 genes in that human blueprint, divided onto 46 X-shaped strands of DNA called chromosomes. The genes are the same in each one of your cells — what differentiates cells for specific tasks is which genes are switched on and which are switched off.

Those strands of DNA — deoxyribonucleic acid — are made up of two long sequences of compounds, called nucleotides, spiralled around each other. Think of DNA as a long string of four different-coloured beads in varying combinations. These beads would represent the four nucleotides — adenine, guanine, cytosine and thymine — and each pairs up with only one of the other four. That means the two long intertwined ribbons are mirror images of each other.

The length of this sequence is staggering. The DNA of each cell's chromosomes contains about three billion pairs of nucleotides. Genes are simply short segments of these beads in the long sequence of the strand.

In an adult, cells can be grouped into three broad categories.

The cells of some tissues and organs stop dividing at maturity, such as nerve cells and those of the heart. When one of those cells dies, it is lost forever and not replaced. But the good news is that because those cells don't divide, they can't become cancerous.

Some cells, such as those in the liver or lung or cells that make up the outer shell of most tissues, divide throughout life, but only occasionally. When one cell dies, that's the signal for a new one to be generated and the cell population of that tissue stays constant.

And there are some cells that are constantly dividing to produce the massive amount of cells needed to replace those lost from daily wear and tear — blood-forming cells, for example, or the cells that line the intestinal tract. These tissues require the constant division of relatively undifferentiated precursors called stem cells.

When a normal healthy cell divides, the 46 chromosomes are duplicated, then separated before the cell splits in two so that each cell will have the full complement of genetic material. That means all three billion pairs of nucleotides have to be encoded, decoded then reproduced — with no mistakes in the order of the pairs.

Since genes are short, specific sequences of nucleotide pairs, any change in their order can mean that a gene is altered, disabled or, in some cases, switched on.

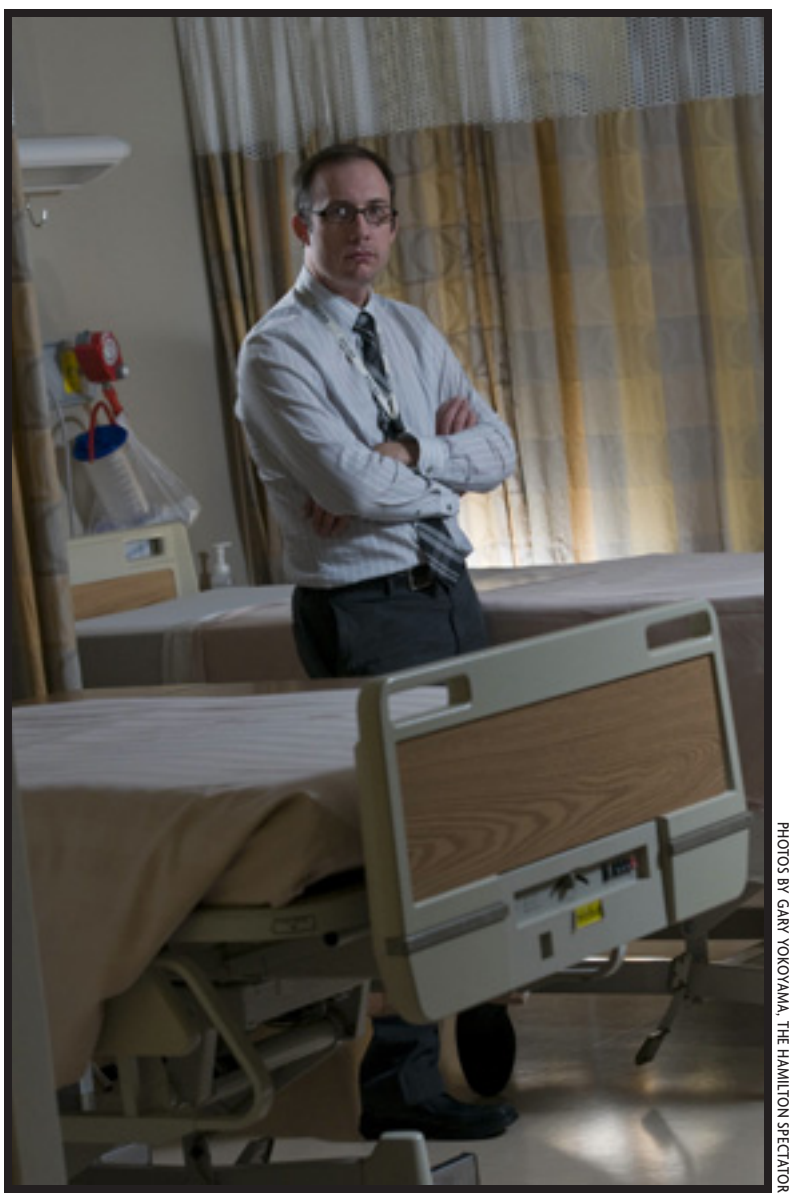
When the DNA of a cell is altered like this, it is called a mutation. If a cell's DNA suffers the right combination of mutations, it can become cancerous.

Things such as the sun's ultraviolet rays or asbestos fibres or certain chemicals, such as those found in cigarette smoke, for example, are known to cause mutations in the DNA of a cell, even though the actual mechanisms might not be yet well understood.

Luckily for us, cancer is not a one-step process but one that can take many years of trauma to a cell. Cancer is a complex, multistep process that may require from four to six progressive alterations of a cell's DNA.

Unluckily, these alterations make a cancer cell different from a normal, healthy cell but not so different that the body recognizes the cancer cell as being a foreign object. If that were the case, it would be relatively simple to figure out a way for the body's own immune system to attack a cancer cell, like it would with a virus.

Understanding exactly the right combination of mutations is part of the challenge facing oncologists, or cancer specialists.



PHOTOS BY GARY YOKOYAMA, THE HAMILTON SPECTATOR

Colorectal cancer surgeon Dr. Shawn Forbes: 'holding death in your hands.'

What is it like to hold a tumour in your hands?

Dr. Shawn Forbes, a colorectal cancer surgeon at the Juravinski Cancer Centre: "You realize the incredible responsibility you have to your patient. You're holding death in your hands.
"Treating colorectal cancer is very much a multidisciplinary activity, but surgery is still the mainstay of therapy. You breathe a little sigh of relief once it's out, then move on to putting people back together.
"Literally and figuratively, people's lives get turned upside down by a diagnosis of cancer. This is the start of a new, and hopefully, healthier chapter in their lives."

It's been discovered that our genes include a group called oncogenes. Dozens of oncogenes have been identified and, normally, these genes are part of the everyday growth and functioning of a cell. But a trigger, such as ultraviolet rays or chemicals in cigarette smoke, can switch on an oncogene so that it is no longer helpful to a cell but harmful.

One oncogene by itself may not be enough to turn a cell cancerous.

In fact, it may take more than one hit to turn on an oncogene, or it may take several switched-on oncogenes acting in unison. Oncogenes are like the gas pedal that will accelerate cancerous growth.

DNA also comes equipped with a brake, a group of genes called tumour-suppressors which help control disorderly cell growth. But a mutation can turn off the tumour-suppressor genes, disabling that control.

During normal cell division, tumour-suppressor genes act as an editor when the three billion pairs of DNA nucleotides are being copied.

If there are spelling mistakes, so to speak, or faulty stop and start signals between genes, the tumour-suppressor gene would shut down the cell-division process. The damage would either be repaired and the process resumes or, if the damage is too severe, the cell dies.

But when a mutation affects the tumour-suppressor gene, it's as if the editor has fallen asleep at the switch. The cancer cell can carry on dividing, even though the new copy of DNA might have a significant number of mistakes.

It's the combination of having the gas pedal pushed down and the brake no longer working that leads to the formation of a tumour.

A tumour begins life much as an embryo does — one cancer cell becomes two, two become four, then eight and so on.

Early progress of the tumour is microscopic and easily escapes detection. By some estimates and depending on the type of cancer, almost two years from that first cancer cell division the mass of cancer cells is still not much bigger than the head of a pin. By two and a half years, the tumour is the size of a small grape.

Cancerous cells in a tumour have also figured out an ingenious way to stay alive as the tumour grows and becomes more dense.

Because of their rapid, uncontrolled growth, cancer cells require a lot of energy and nutrients. Tumour cells secrete substances called growth factors, which entice the development of blood vessels into and around the tumour that supply the necessary fuel.

If cancer was just a matter of uncontrolled growth of abnormal cells, it could be attacked in a straightforward fashion: catch the offending lump before it interferes with the function of the tissue and cut it out. And, for good measure, zap the affected area with some radiation or drugs just to make sure that any stragglers are killed off.

But cancer is a devious foe and its deadliest weapon is the ability of a cancer cell to break away from the original tumour, slip through the wall of a blood vessel, migrate to another spot in the body, slip back out of the blood vessel, then lodge itself inside another organ or tissue. Thus, the whole process of tumour development begins anew at a secondary site.

No other cell can do this. A normal, healthy liver cell can't break away, enter the bloodstream and travel to the lung, for example, and begin growing. But a cancerous liver cell can.

It's this ability to migrate, or metastasize, that has made cancer such an efficient killer.