

Evading growth suppressors: Hallmark #4

Many people think of cancer as a disease in which our own cells essentially go ‘rogue’-growing and dividing to form tumors. A long-standing question for scientists has been to understand how this happens. In particular, cancer researchers have described a number of characteristics of cancer cells that distinguish them from the normal cells that populate our bodies. These characteristics are often referred to as the ‘hallmarks’ of cancer, and the ability of cancer cells to ignore simple instructions to stop growing is one of these hallmarks. In other words, cancer cells are like a car that has no brakes!

Perhaps the easiest way to explain what these ‘negative’ growth signals are is to offer a few examples. All of us as humans grow in size in the early part of our lives. We get taller and our shoulders broaden, to name just a few examples. This is fuelled by cell division. Cells grow in size, divide to give rise to two new cells, and repeat the process billions upon billions of times until we all reach our own growth potential. The signals that instruct our cells to stop growing are complex, but many of these signals come from interactions between neighbouring cells. So when an organ or body part reaches a certain size all of the cells that are touching each other are essentially telling one another it is time to stop dividing. Cancer

cells lose the ability to stop when touching their neighbours. In some cases they don’t receive the ‘stop’ signal anymore, and in other types of cancer they receive this signal but don’t understand it. Regardless of the reason, cancer cells universally lose the ability to put on the brakes and stop dividing when they are told.

Another example of cell growth gone rogue, is in the case of cells that maintain the ability to grow and divide throughout our lifetime. Many of these cells are found on our skin, but they also line our lungs, stomachs, and other internal organs. New cells



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located in these places are continually produced through cell division as older ones die and need to be replaced. For this reason these are cells that normally divide regularly, but they also need to stop when necessary to do their job in places like our skin. In this case cancer that arises in these locations can be thought of as a speeding car that doesn’t stop for a red light. Cell growth and division is already well underway, but the specific problem is that cancer arises develops in this situation when the dividing cells can’t put on the brake. Perhaps not surprisingly, most cancers come from cells that normally divide regularly but eventually aren’t able to stop when they should.

Since cancer cells are often missing their brakes, fixing this problem should be a way to treat cancer. Work in my lab is focused on this goal. After much research in this area, we now know of a few ways that cancer cells can be stopped. In particular, we have identified and are beginning to understand the genes that function as the brakes for stopping cell division. Encouragingly, drugs are emerging that are capable of turning on the brakes in cancer cells. In particular, our research is focused on how to identify patients that will benefit most from these new drugs. By no means is the battle won, but we are starting to find ways to fix the faulty brakes in cancer cells.