Cancer is a disease that scientists and physicians have been attempting to tackle for decades. In order to make progress in the vast field of oncology, one must first reach an understanding as to what cancer is, how it is caused, and how it varies in different species. Put simply, cancer is a disease that occurs when a significant amount of cells in the body go awry, causing uncontrolled cell growth. The cells that are rapidly dividing destroy body tissue and disrupt vital bodily functions. A number of forces can inflict this dreadful disease, including gene mutations, smoking, radiation, viruses, carcinogens, and a lack of exercise (Mayo Clinic Staff, 2018). Being the global and widespread disease that it is, cancer has impacted human health, the ability of society to pay and care for its citizens, and the quality of life of those affected. Thus, providing the public with a general understanding of its causes and available treatments is essential to ensure that we’re well-equipped to fight this disease in all its forms.

Interactions with the Human Body

To get to know cancer better, we must first learn more about the organisms in which it occurs: complex, multicellular organisms. These organisms are all made up of bodily systems
that are necessary for survival. Within these systems are organs which must work together to maintain the body. The cells that make up these organs are specialized to properly do their job. For example, the cells that line the intestine have longer membranes for an increased “amount of surface area that is available to absorb food” (AAAS, 2019).

The purpose of the human body’s immune system is to provide defense against infectious organisms and disease-causing substances. Cancer can manipulate certain cells within the immune system, making it easier for it to infiltrate the body. Macrophages, the white blood cells responsible for destroying foreign bodies and cells, are tricked by cancer cells. The cancer cells put out what is commonly referred to as “don’t eat me signals”, which allow them to evade macrophages and ultimately the immune system itself (Newman, 2017). For example, one of the “don’t eat me signals” comes from cancer cells’ high levels of protein CD47, which binds to the protein SIRPalpha on macrophages. This binding interaction weakens the macrophages, reducing their ability to kill the cancer cells. Essentially, cancer’s manipulation of the immune system enables it to evade the body’s defense system and continue uncontrolled cell growth.

The Impact of Metastasis

Cancer becomes difficult to treat is due to its ability to spread to different locations within the body. Cancer cells break away from their primary site and travel to another part of the body, where they continue to develop and disrupt the body’s regular function (Cancer Research UK, 2017). This process, called metastasis, occurs when cancerous cells enter the bloodstream or lymphatic system and take up residency in distant tissues (ASCO, 2019). The circulatory system thus has a large role in the spread of cancer, as it is responsible for blood circulation throughout
the body. Circulating Tumor Cells (CTCs) are cancer cells that have successfully entered small blood vessels and the bloodstream (Cancer Research UK, 2017). These cells usually get stuck in extremely small blood vessels, such as capillaries, allowing the cancer to move through capillary walls and into the tissue of nearby organs where they can potentially form a new tumor (Cancer Research UK, 2017). Out of the many thousand cancer cells that enter the bloodstream, only a few will be able to survive and cause secondary cancer. But these few cells are all it takes for metastatic cancer to develop, putting the patient at a higher risk for cancer’s fatal effects. Metastasis generally complicates the original treatment pathway, thus rendering it a challenging obstacle for the medical community to design effective treatment plans and therapies.

The Advantage that Plants have

Metastasis, a process that’s all too familiar to those studying and treating cancer in humans, does not occur in plants. Plant tumors do not metastasize, due to the major differences in the circulatory system of plants versus that of vertebrates. Even though plants can get cancer, specifically through infection, they are significantly less vulnerable to the fatal effects seen in humans with cancer. This can be attributed to the rigid cell walls of plant cells. Unlike the constant motion of cells in the human circulatory system, plant cells are locked in place by a matrix of cell walls, making it difficult for cells to migrate (Engber, 2014). So even when a plant cell divides uncontrollably, the tumor stays in the same place: the cancer is trapped. Since they are stationary, plant tumors are rarely fatal; the plant finds ways to work around cancerous cells. Regardless of the source of the damage—radiation, physical attack, etc. (Thompson,
—plants can replace dead cells or tissue more easily, making them highly resistant to uncontrolled metastatic cancer.

**Variation of Cancer Across Species**

The ways in which animals develop and deal with cancer is similar to humans, however there are several animal species that are naturally cancer resistant. The common misconception is that since large animals, say elephants, have more body mass and thus more cells, they are at higher risk for cancer. But elephants rarely get cancer. Peto’s Paradox states that there is no relationship between body size and cancer incidence across species (Wikipedia, 2019). Elephants’ cancer resistance may be related to their 20 or so copies of the tumor-suppressing gene called P53, a gene that humans only have a single copy of (Wei-Haas, 2018). Most animals rely on their cells to do some self-repairing when there is damage in their DNA, whereas elephant cells with DNA damage are killed by P53. Having an excess amount of the gene that specifically kills cancerous cells is clearly advantageous over the less efficient repair process in humans.

Along with elephant’s extra copies of P53, their extra LIF (Leukemia Inhibitory Factor) gene, called LIF6, may be another reason for their cancer resistance, by additionally tipping the scales towards the self death of damaged cells (Wei-Haas, 2018). LIF6, activated by p53, creates a protein that travels to the mitochondria, where it triggers apoptosis (Science Daily, 2018). Though there is ample evidence that suggests that this process is an important reason why elephants are much less likely to get cancer than other species, there could be a bigger, more important piece to this complex puzzle that we just haven’t figured out yet. In particular, why
haven’t humans evolved to have multiple copies of P53, or LIF6? And what are the costs of having such multiple copies? Like all of biology, the deeper one digs, the more mysteries appear.

**Impacts on Aging Individuals**

In the struggle to improve the quality of life of human beings, researchers view cancer as one of the most significant obstacles. It is sometimes referred to as a “disease of the aging”, and for good reason (Ershler, 2019):. As we age, our cells are constantly dividing to replace old ones, and with this turnover, genetic errors are virtually inevitable. For this reason, cancer is more common in older people. According to the National Cancer Institute, 25.4% of new cancer diagnoses are in people from ages 65-74. The concept of aging itself is truly perplexing: do we have to age, or is aging a problem that can be solved with enough technology and biological insights? One common theory for increased cancer risk as we age is that as time goes by we experience “prolonged exposure to carcinogens such as sunlight, radiation, environmental chemicals, and substances in food” (Dana-Farber Cancer Institute, 2016). But why do many people live beyond 80 years with no detectable cancer? In the constant human effort to achieve immortality, cancer has proven to be a real bump in the road. Trying to articulate the process of aging raises many important questions that are constantly being asked. If we’re able to cure all cancer sometime in the future, what would kill us? How much longer would we live? How would society as a whole be different?
Conclusion

Cancer has troubled patients, doctors, families, and friends for generations. It has taken the lives of many, which has caused tremendous amounts of time and money to be put into research. We have come a long way as far as treatment, but there is still a long road ahead. The many existing question marks of cancer and its treatment can be discouraging for some, but the sheer deadliness of the disease and its impact on human health and well-being should be enough motivation to invest in research that produces more effective treatments. Currently, we diagnose cancer, analyze many aspects of the patient and their condition, develop a treatment plan, and administer it in the best way possible, even though it is rarely a perfect therapy for the patient. Striving for perfection when dealing with cancer is -- at this point -- far-fetched; the best we can do is strive for progress. Although it is still the second largest cause of death worldwide, just behind heart disease, we have managed to significantly lessen cancer’s deadly impacts. However, it is going to take significantly more time, money, resources, and original research to reduce the impact of cancer on human health and well-being.

Bibliography


https://www.cancerresearchuk.org/about-cancer/what-is-cancer/how-cancer-can-spread


https://www.medicalnewstoday.com/articles/320177.php


https://en.wikipedia.org/wiki/Peto%27s_paradox


http://sciencenetlinks.com/student-teacher-sheets/cells-your-body/


What is Metastasis?. (2019). Retrieved 18 July 2019, from

https://www.cancer.net/navigating-cancer-care/cancer-basics/what-metastasis


Zombie gene protects against cancer -- in elephants: Dead gene reborn helps destroy cells with damaged DNA. (2018). Retrieved 18 July 2019, from