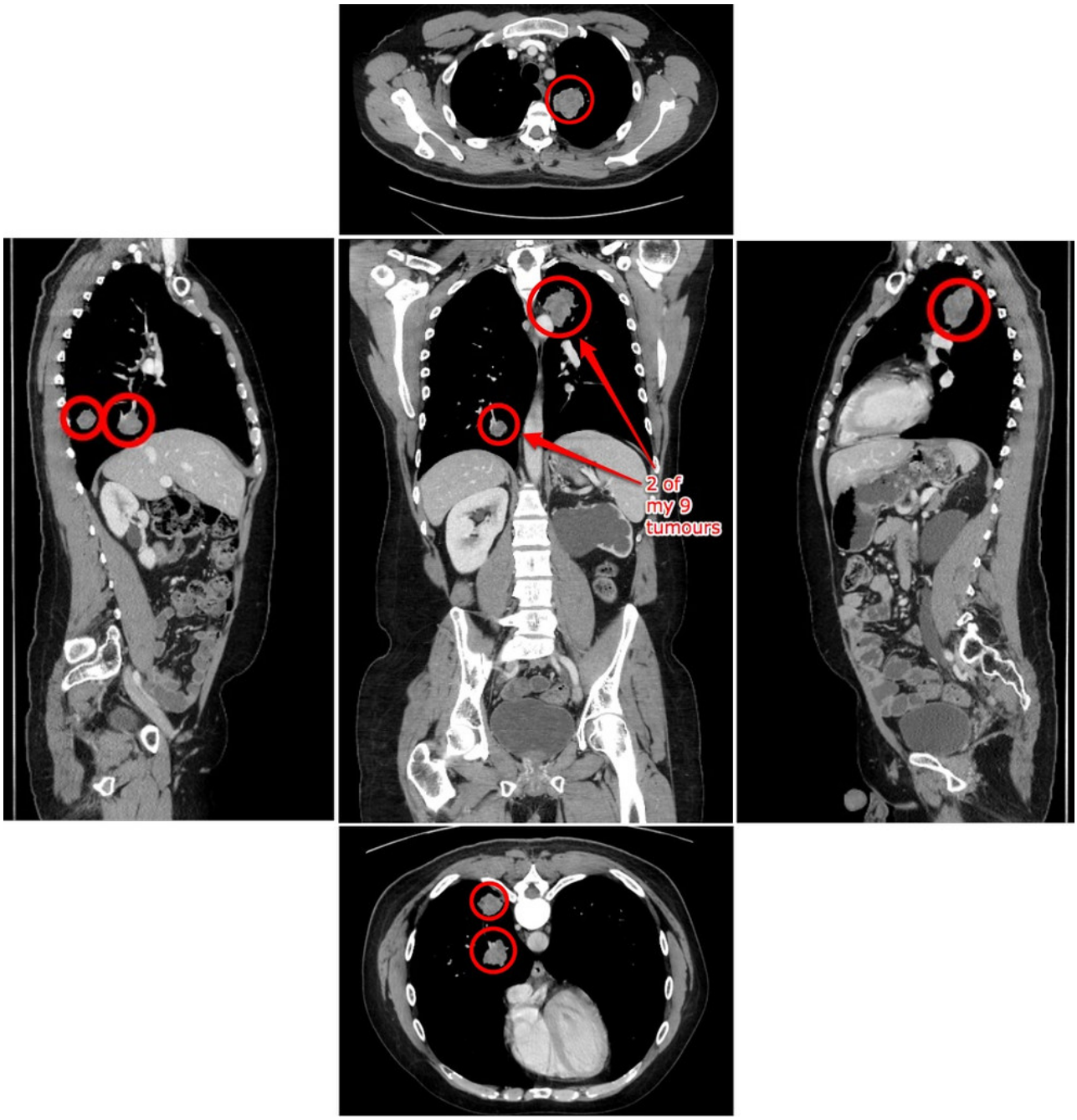
HOW CANCER CAN FURTHER DEVELOP LIFE.

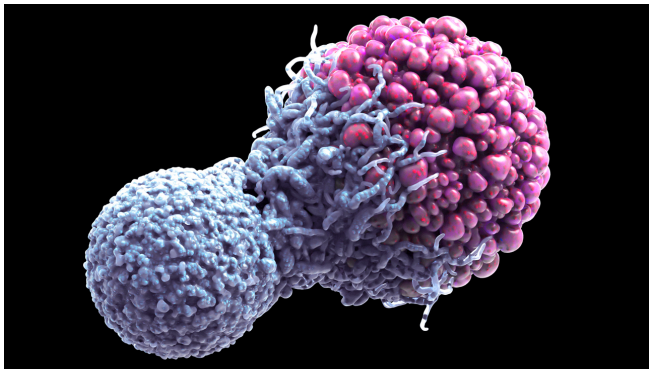


[1] Figure 1: Tumors visualized through various CT scans of abdomens; a common way cancer is diagnosed.

How Cancer Can Further Develop Life.

What if there were benefits from cancer? Cancer is a medical tragedy that can be a death sentence in its latest stages. According to the World Health Organization, there were around 10 million deaths relating to cancer alone in 2021. However, cancer cells may hold evolutionary and developmental properties that we have used since birth.

One researcher, A. Kozlov (2022) hypothesizes that there may be a link between hereditary tumors and the evolution of multicellular organisms. While this sounds far-fetched, he found that there is a possibility that tumors might aid multicellular organisms with extra cell mass and other resources. This extra mass is similar to a boost that complex organs need to grow. Think of it as a supplement for growing tissues. This sprouted a new theory titled “the carcino-evo-devo theory” (Kozlov A.P. 2022). By investigating genes in both new tumors and new organs, Kozlov found that they were identical. Organs like the eutherian placenta, prostate, mammary gland, and newborn human brain in humans all have tumor-like features. These complex tissues have intensely complex structures. He concluded that after development in babies, these “tumor-like organs" lose their features and their high possibility of becoming cancerous (Kozlov A.P. 2022). From there, the cells become normal organs with no tumor features. These complicated tissues found in babies and other forms of life need this extra push to grow. The brain for instance is an extremely important part of any organism’s life. By having a “tumor-like shift” the brain can get the nutrients and mass it needs to grow, kind of like being pushed on a swing set for that added momentum. The evidence of “tumor-like shifts" is just one instance where cancer can assist in developing life.

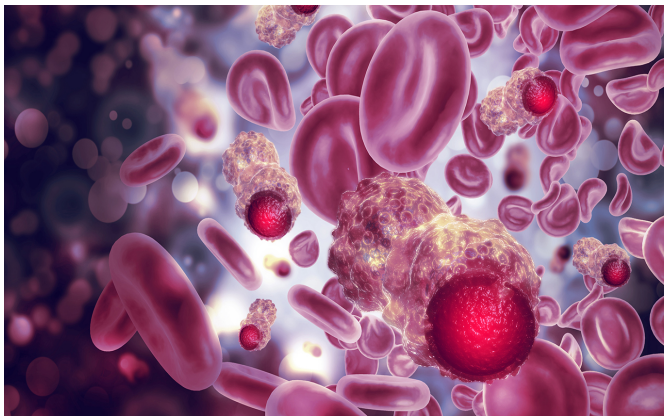


Cancer’s evolutionary properties are not only found in newborns. Further research done by Liu R in 2021 shows a co-evolution between tumor cells and immune cells in Multiple Myeloma patients (Liu R 2021). Multiple Myeloma is a type of skin cancer. To this day, almost all cancers including MM are incurable as we do not fully understand the disease progression. As is the same with almost all cancer diagnoses, a broad database is absent. So, in order to combat cancer and MM, we have a vast unknown to explore. In their research, the study collected around 17,267 plasma cells along with 57,719 immune cells for comparison of their identity. They found that patients with MM seem to have both their plasma cells and immune cells grouped together.

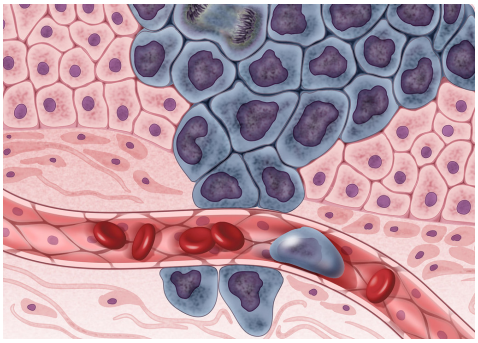
The group was able to visualize plasma and immune cell populations across MM’s disease progression. Amongst their findings, patterns with cell stability and gain or loss in body mass occurred. Using these patterns, we can diagnose a patient with MM or precancer by using the stability of the

[2] Figure 2: Cancer cells invading a normal cell visualizing invasions amongst cells.

immune cell populations. What was most interesting is that in multiple patients, a link between their many tumor cells was found grouped with a B type cell (white blood cells). This matters because we can diagnose a patient by looking into the gain or loss of the white blood cell populations and the same gain or loss in body mass. They report that as MM disease progresses, the white blood cells do as well. The immune system grows in mass and number to strengthen and fight. They found as one went up, so did the other when they were clustered like this. Both instances show a possibility where positive evolution can occur due to cancer progression in patients. Tumors can sometimes inhibit the immune system or drain bodies making certain parts of their immune system shut down. However, this example shows us that certain types of cancers can boost your white blood cell count. An increased white blood cell count only strengthens the immune system. Another example could be seen when you have an infection. As your body fights the infection, your immune system responds and thus, your white blood cell count increases. The same thing is occurring in these tumors. The body is actively evolving and changing its white blood cells to have an "arms race" against the enemy tumor. Every new paper on oncology shrinks the vast void of unknown knowledge, deepening the human understanding on cancer.



There is still so much not known about cancer and its progression. In the Network of Cancer Genes (NCG) there are massive gaps and incomplete databases in the drivers of cancer (Dressler L. 2022). An outside research group has been investigating the genetic change in certain cancerous cells. Specifically, they are looking between somatic (body) tissues and what is causing non-cancerous (benign) tumor formation. They found the alterations in 7,953 cancerous samples and discovered that since the database of known drivers is so incomplete, they have not been able to fully understand the link. Through using different methods of manual genetic modification, researchers have begun to play around with this "cancerous genome." The team has been able to confirm that in the 7,953 genes, somatic change in just one of the genes seems able enough to drive a cell towards harmless expansion, but not cancerous (Dressler L. 2022). Some interesting applications for this research could be wound closure, scar revision, and cell production by forcing these somatic (body) cells into a state of harmless growth for cell reproduction.



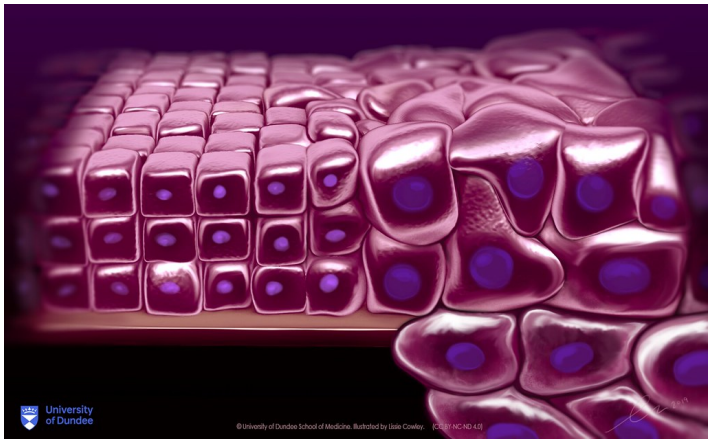
Other research uses models to further deepen our oncology understanding. Recently, Genetically Engineered Mouse Models (GEMMS) have been more popularly used to visualize cancer progression. These GEMMs have been used before, but they have all lacked the genetic diversity that is found in

[3] Figure 3: Cancer cells surrounding and invading blood vessels. Resembles how lymph nodes are invaded.

[4] Figure 4: Tumor cells migrating through the endothelial wall. This is the beginning of how cancer spreads throughout the body through metastasis.

humans. Without this genetic diversity, human tumors could not be visualized in these models. However, the recent progression in sequencing technology and the further investigation in drivers for tumors found above have given lead-way into generating new GEMMs for exploration in tumor evolution (Hill W. 2021). These GEMMs hold a wonderful opportunity to safely investigate DNA damage and chronic carcinogen exposure to see the malignant effects firsthand. We can also use these models to apply new treatments.

Along with the use of models to visualize cancer progression, studies on actual tumors and their genome sequencing hold promising information as well. In one study, tumors were found to have gone through a massive shift in their appearance into unicellularity (Anatskaya O.V. 2022). They found that even in aggressive tumors, changes in the genetic code can cause the cancerous cells to cluster into one big cell formation before reproducing to a dangerous size. Think of it like during a football game when there is a fumble. One player will jump on top of the ball and then another, and another, over and over to get the ball. The very same thing happens here except the number of players jumping on the ball never stops. As new ideas inspire new research, more analysis has been done on studying these cancerous genomes. Hepatitis-B was investigated between its link to cancer recurrence and evolutionary path (Zhou S. 2022). They have found that patients with liver cancer have a very low chance of long-term survival due to a high rate of cancer recurrence. Little is known about the trajectory of tumor recurrence in patients after the primary tumor. By performing a whole-genome sequencing on 40 pairs of primary and early recurrent Hepatitis-B virus related tumors, they identified two patterns. The first is a de novo recurrence which occurs genetically separate from the primary tumor. The other is an ancestral recurrence, which is a clone that is related to the primary tumor and has a much faster disease progression than the initial recurrence (Zhou S. 2022). By investigating the two separate patterns, they found that tumors found farther away from the first tumor were more linked to the initial pattern and localized tumors that were close to the primary followed the ancestral recurrence. This data allowed a deeper understanding with more clarity on the genomic evolution during Hepatitis-B virus related hepatocellular cancer in patients. The understanding allows a more focused edge in finding better treatment and therapies for patients (Zhou S. 2022).



While we have not slayed that old great dragon cancer just yet, our swords are sharpening and our numbers are rallying. New research sprouting up every day gives us a ray of hope in discovering evolutionary treatment options. Even today, research is being performed inside of Webster University's Interdisciplinary Science Building on the effects of different drugs such as nicotine and CBD on different bladder and breast cancer cells. While cancer may grow, our understanding and fight against it also grows. The exciting work being done above is only a miniscule level of what we can discover for further treatments.

[5] Figure 5: Normal body cells mutating into a cancerous tumor. This is an example of the “football fumble” of cells.

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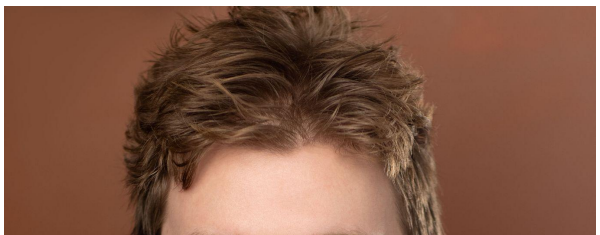

BIOGRAPHY: COPPER PETERMEIER is a 21-year-old junior at Webster University double majoring in a B.F.A. in Acting with the Conservatory and a B.A. in Biology with the Biological department. He also works as an emergency department scribe overnight on the weekdays. He is the current president of Webster University's Pre-Medical Society and plans on taking the MCAT next spring where he will hopefully go into medical school to study emergency medicine.