



# Effects of Transfection on Metastatic Potential of Mouse 3T3 Cells



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## INTRODUCTION

About 1,500 people die every day from cancer, thus emphasizing the importance of further cancer research and in-depth explorations of cancer treatment plans (Seyfried and Hysentrut, 2014).

### What is metastasis and how does it relate to this project?

Metastasis is the "process by which cancer cells spread to other parts of the body" (National Cancer Institution). By transfecting the cells (knocking down the p53 and Myosin Heavy Chain 9 genes in Mouse 3T3 cells), I hope to decrease the metastatic potential, the likelihood of the cancer cells to spread, of the cells.

### What are the p53 and Myosin Heavy Chain 9 genes?

The p53 gene behaves as a tumor suppressor gene; the Myosin Heavy Chain 9 controls cell growth, especially during the cytokinesis stage.

**Landmark Experiment:** Scientists wanted to see how mutations in proto-oncogenes cause cancer in the lymphatic system. This research showed that "somatic hypermutations in tumor suppressor genes involved in major human malignancies offer a novel insight in cancer development, progression and spread" (Kashuba, 2009). This landmark case revealed that many cancer or metastasis cases involve mutations in the proto-oncogenes or tumor suppressors genes, like p53.

## DISCUSSION, ANALYSIS, AND EVALUATION

**Research Question:** How does the transfection of the p53 and MYH9 genes in Mouse 3T3 cells affect the metastatic potential of the cells?

### Significance of Results

Figure 3 implies that if p53 and MYH9 are knocked down, more cells migrate toward the other chamber. Thus, this emphasizes the importance of the p53 and MYH9 genes in controlling cell growth and cell migration.

The lower the ratio between serum-present and serum-absent plates signifies that cell migration is lower, and that metastatic potential is lower. Therefore, when the cells were transfected and p53 and MYH9 were knocked down, the metastatic potential increased, as 2.463 and 2.751 is significantly larger than the Wild Type's 2.342 ratio.

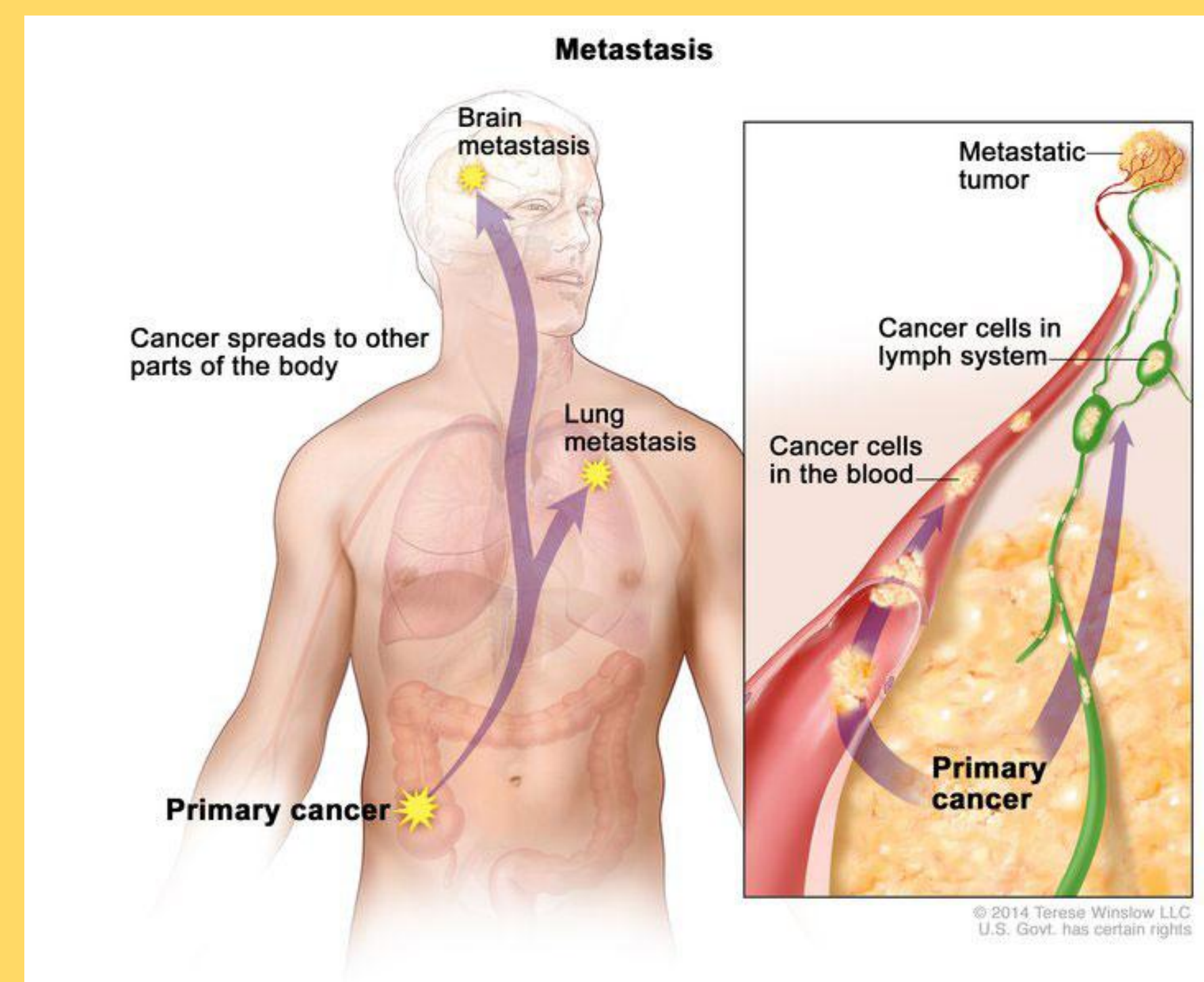
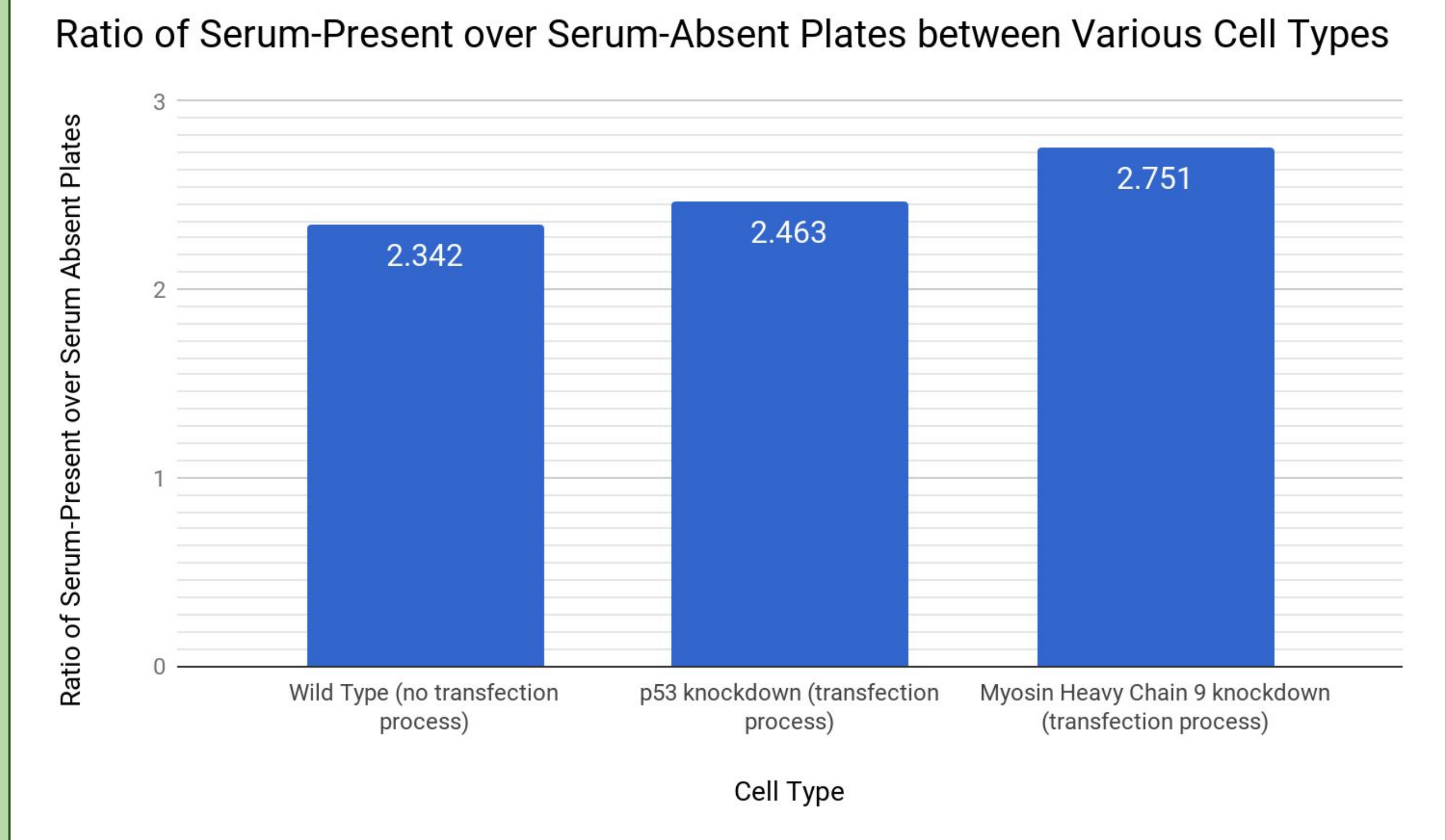


Figure 1: The image above shows how the cancer in the primary site spreads among different body parts through the flow of blood. This eventually leads to cancers being formed in places like the lungs and brain. This is an example of metastases.

## DATA AND FINDINGS

Figure 3: The graph shows the ratios of serum-present over serum-absent trials for each of the different plates (WT, p53 knockdown, and MYH9 knockdown). Each of the plates had two trials, and the serum-present and absent values were found by taking the average of the two values.\*



## RESEARCH METHODOLOGIES

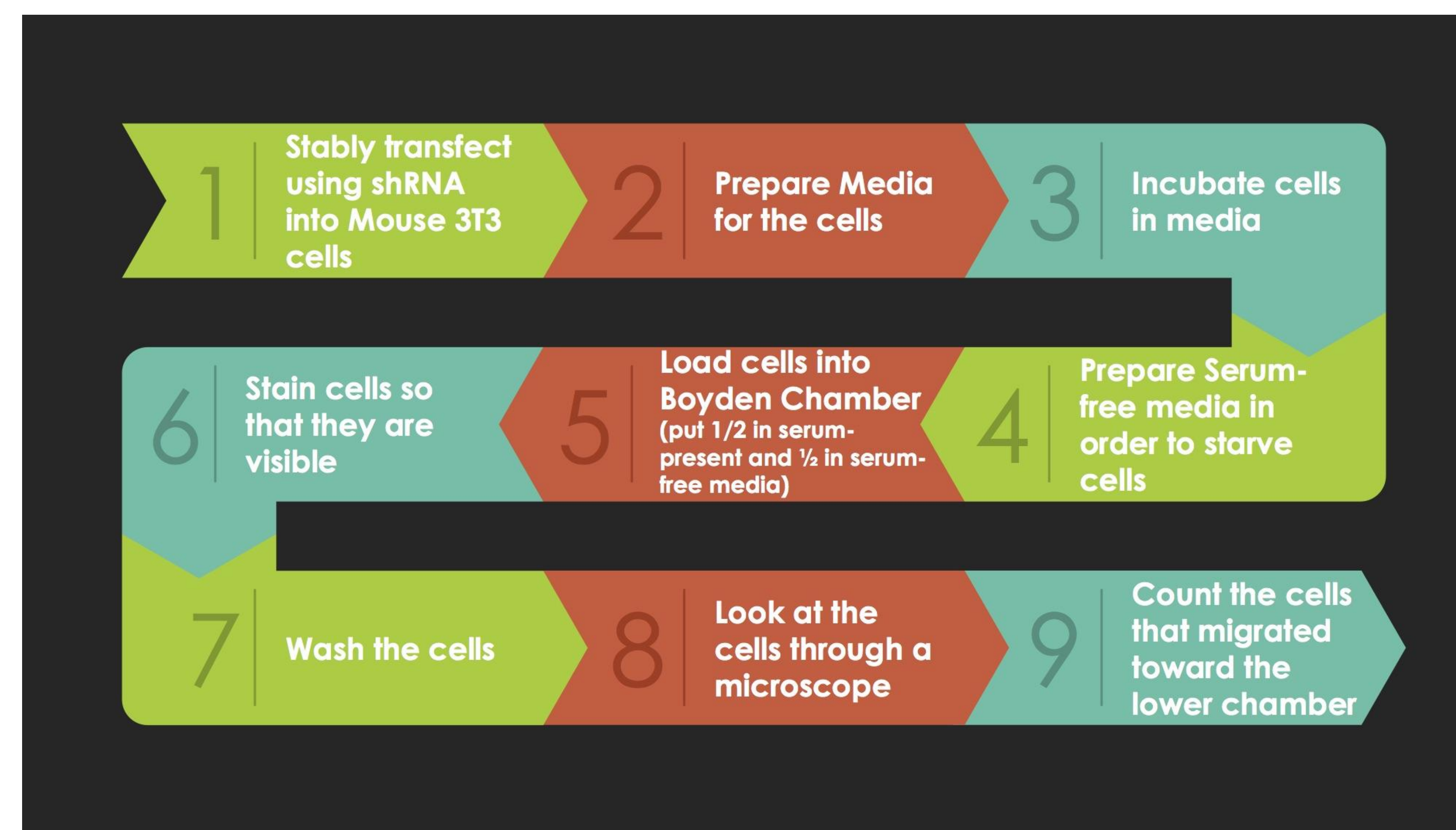


Figure 2: This diagram shows the overall process of the experiment.

- ❖ Equipment used:
  - Serum-free media and Serum-present media
  - Mouse 3T3 cells (standard fibroblast cell line)
- ❖ Types of Mouse 3T3 cells:
  - Wild Type cells (no transfection)
  - Myosin Heavy Chain 9 knockdown cells (transfected cells)
  - p53 knockdown cells (transfected cells)

\*The different plates were WT serum-present, WT serum-absent, p53 knockdown serum-present, p53 knockdown serum-absent, MYH9 knockdown serum-present, and MYH9 serum-absent.

## CONCLUSIONS, IMPLICATIONS, AND NEXT STEPS

Noticing that the p53 and MYH9 genes have an effect on metastatic potential can emphasize that they are necessary in controlling cell growth. In fact, physicians can possibly enhance these genes to terminate metastases in future patients.

Figure 3 supports the fact that knocking down MYH9 (ratio of 2.751) has a much bigger impact on cell migration than knocking down p53 (mere 2.463 ratio). So, specifically working with enhancing the MYH9 protein may be beneficial, as it seems to have more of an influence on cell migration.

## ACKNOWLEDGEMENTS / REFERENCES

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### Works Cited:

Metastatic Cancer. (n.d.). Retrieved January 11, 2018, from <https://www.cancer.gov/types/metastatic-cancer>

Seyfried, T. N., & Hysentrut, L. C. (2013). On the Origin of Cancer Metastasis. *Critical Reviews™ in Oncogenesis*, 18(1 - 2), 43-73. doi:10.1615/critrevoncog.v18.i1-2.40

Vladimir Kashuba-Tatiana Pavlova-Elvira Grigorieva-Alexey Kutsenko-Surya Yenamandra-Jingfeng Li-Fuli Wang-Alexei Protopopov-Veronika Zabarovska-Vera Senchenko-Klas Haraldson-Tatiana Eshchenko-Julia Kobliakova-Olga Vorontsova-Igor Kuzmin-Eleonora Braga-Vladimir Blinov-Lev Kisselev-Yi-Xin Zeng-Ingemar Erberg-Michael Lerman-George Klein-Eugene Zabarovsky - <http://journals.plos.org/plosone/article?id=10.1371%2Fjournal.pone.0005231>

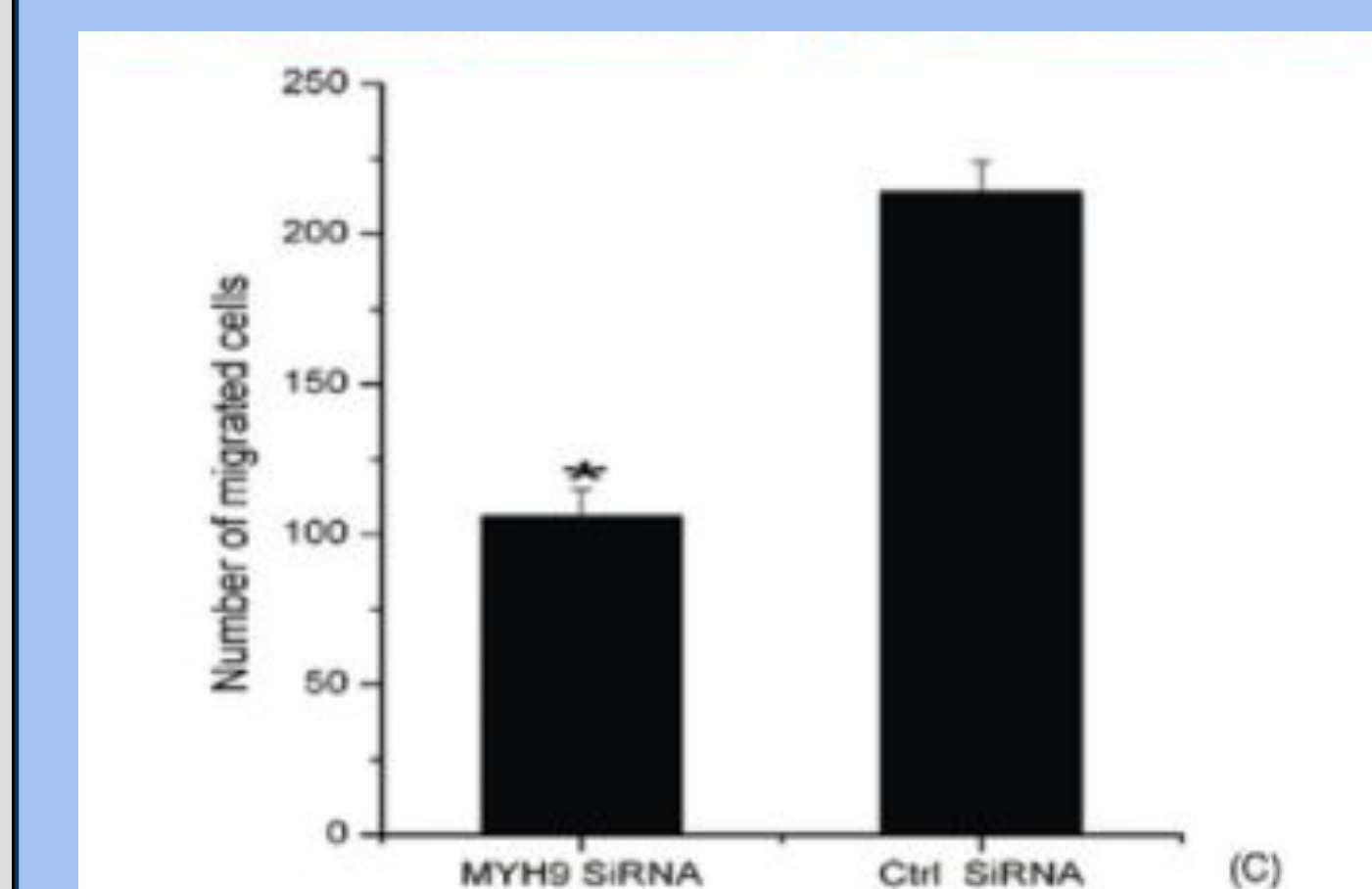


Figure 4A: This figure shows that when Myosin IIA is depleted by shMYH9, the cell migration is significantly decreased, showing the vitality of MYH9 gene in controlling cell migration.

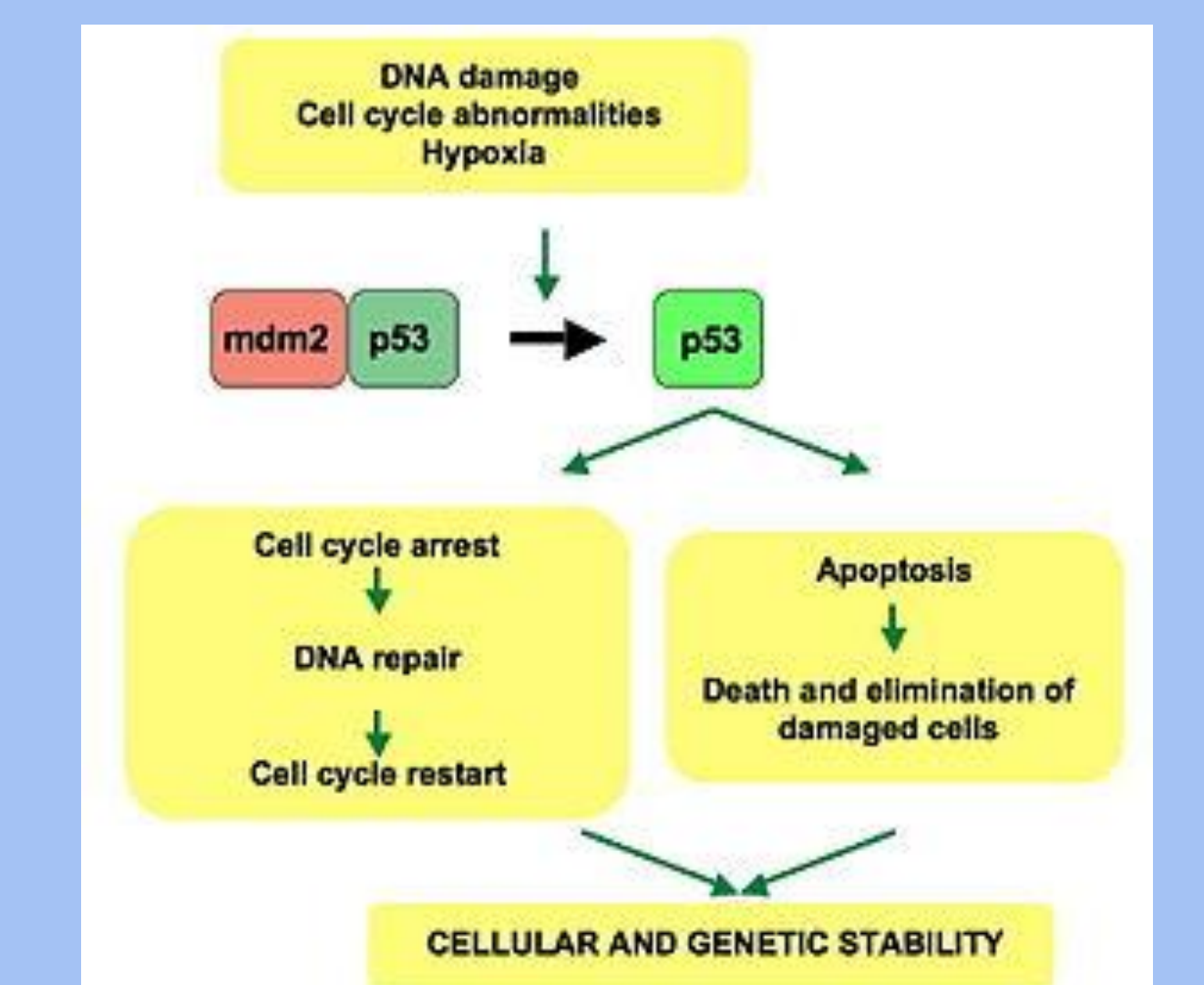


Figure 4B: The figure shows the function of p53 and its role in tumor suppression. If the gene is mutated or removed, it can cause massive cell growth, which can lead to cancer and/or metastasis.