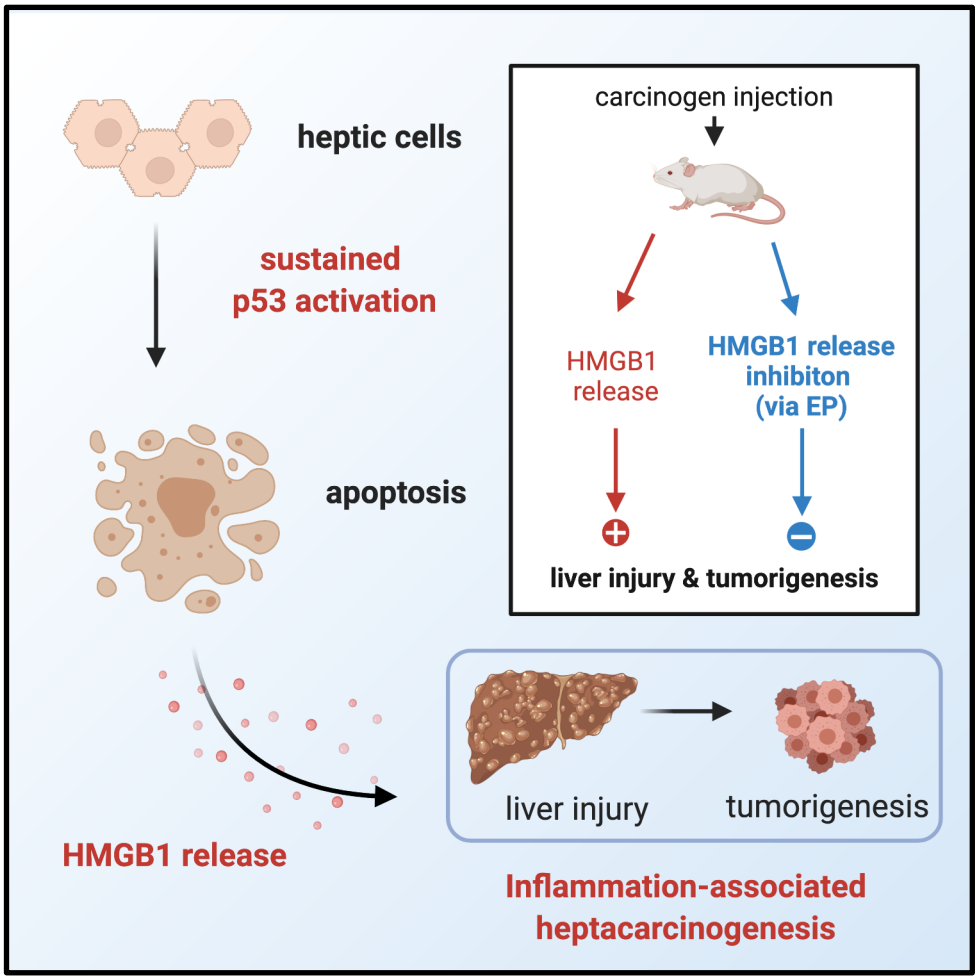
IID Graphical Abstract ICA

Student No. 530

Yan HX, Wu HP, Zhang HL, Ashton C, Tong C, Wu H, Qian QJ, Wang HY, Ying QL. p53 promotes inflammation-associated hepatocarcinogenesis by inducing HMGB1 release. *Journal of hepatology*. 2013 1 October;59(4):762-8.

1. Graphical Abstract



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2. Reflective Report

Introduction

As I was following He-Xin Yan's research on inflammation-associated tumorigenesis, I chose one of his studies to make a graphical abstract (GA), regarding HMGB1 that accelerates inflammation-associated hepatocarcinogenesis. To do this ICA, it was crucial to **select only the most important findings**. The second challenge was **how to use various visual objects to present the findings logically**. Besides, many GAs about inflammation-associated hepatocarcinogenesis only includes molecular mechanisms, which made me unsure about **whether and how I should include therapeutic implication in the GA**.

Depiction

Good examples are the best guideline that helped me tackle most of the challenges. For example, the paper of Nomura *et al.* (2010) makes a good examplar GA for cancer research with the therapeutic implication (Figure 1), where the modification adds a box indicating the therapeutic significance to the original GA only containing molecular pathways, which hints for the importance of therapeutic implications in similar papers like mine. This was why I also included the therapeutic implication with a similar box in my GA.

Reading previous research helped me identify the critical points in the paper. At first, I was unclear about whether I should include how the authors constructed p53 heterozygotic mouse model until I found that they published an article regarding this before the research, which makes the model relatively unimportant (Tong *et al.*, 2010). This also reminded me of the importance of comparisons while reading papers and trained my literature reading skills.

Feedback

Peer feedback was a good platform for learning from each other; however, since each student worked on different paper and few would make detailed comments about the GA without enough important background information, **effective communication** was crucial. I finally developed a strategy where I first ask them a few questions in advance. In this way, to comment on my questions, they would normally often ask me for more details, thus providing more specific feedback. Also, by explaining the GA to them, I even realised that I made a mistake about the role of apoptosis because one peer reviewer asked whether HMGB1 could be released from apoptosis (Figure 2).

Concluding remark

In summary, the ICA, as training of example-based learning, comparative reading and effective communication, should contribute to my career development. Furthermore, **my worries about oversimplification drive me to be cautious about the research and GA.** Notably, I may need to find better ways to indicate the time of EP treatment next time. Though EP treatment does take effect after carcinogen treatment activates p53-mediated apoptosis as in the GA, it happens before carcinogen treatment, which could be misleading. **The caution about oversimplification in GA fuels my critical thinking about research results**, making me curious about how each experiment contributes to the. This also makes me sensitive to flaws in the research, driving me to learn whether they have been solved elsewhere or could be solved in future studies, which drives me to be a better researcher.

Word Count: 486

3. Figures

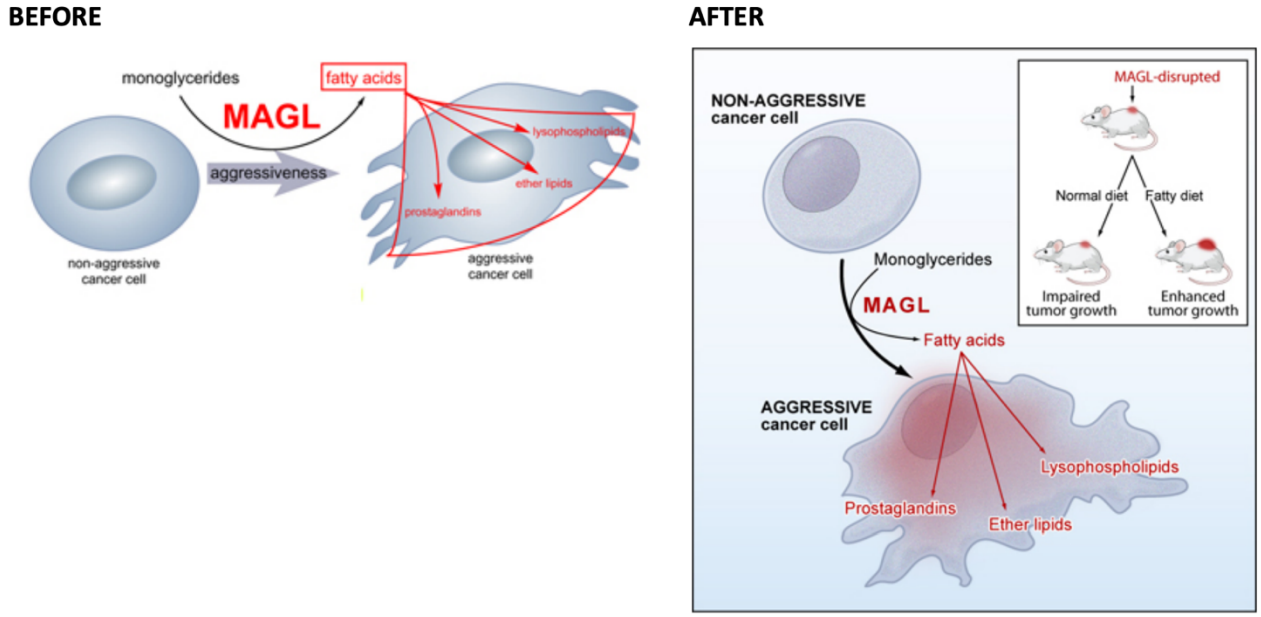

Figure 1. The Cell Press modification of the graphical abstract of Nomura *et al.* (2010). This picture provides an excellent example of how to make GA for basic research with therapeutic implications (Cell Press, 2019).

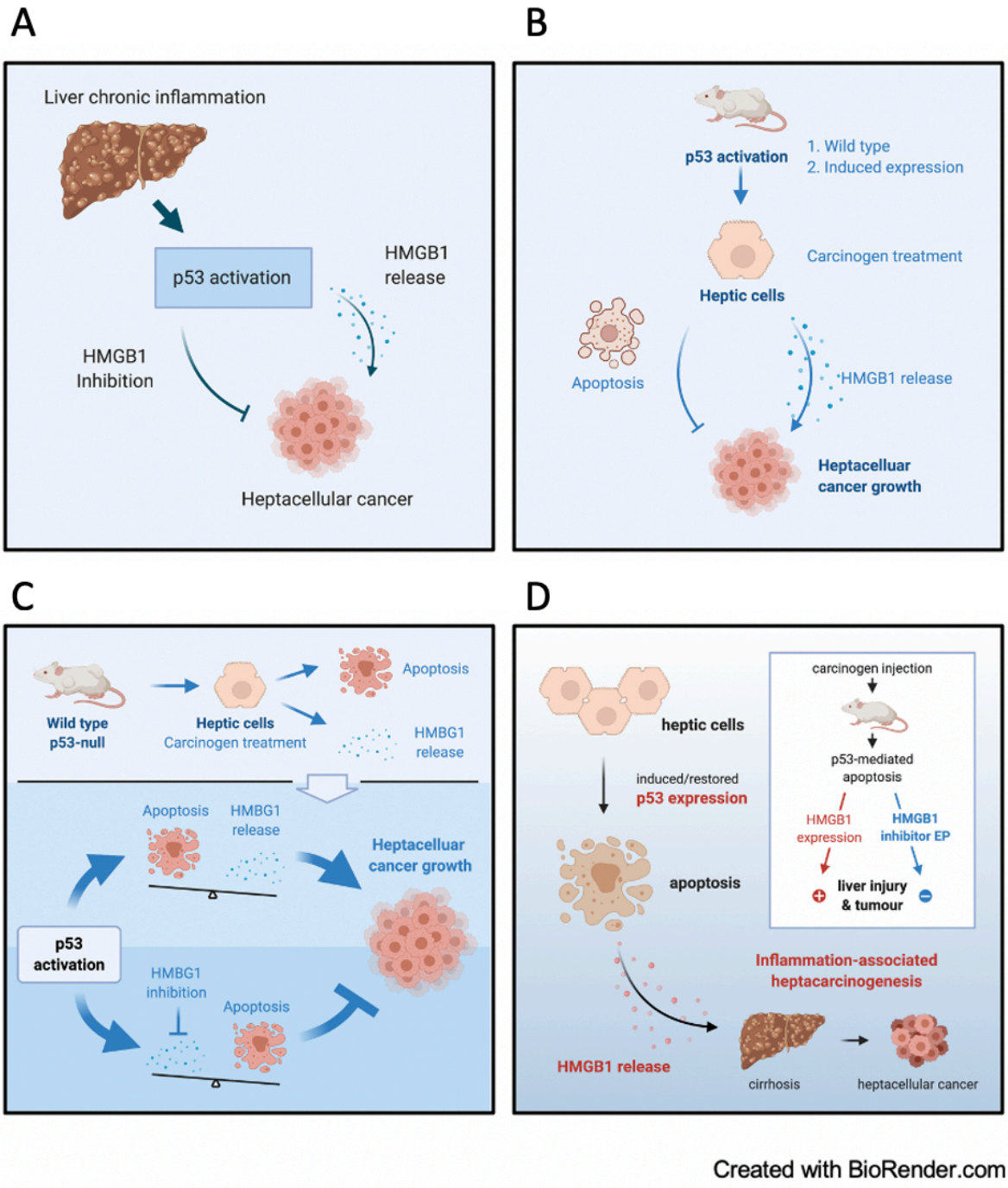

Figure 2. Evolution of my GA

- **(A) 1st draft:** I only depicted the molecular pathways at first.
- **(B) 2nd draft**: I added some essential experimental approaches, with some misunderstandings regarding the role of apoptosis. Apoptosis actually has dual roles, i.e. tumour-suppressing and tumour-promoting, but here the authors only studied the latter.

- **(C) 3rd draft:** I detailed the role of HMBG1 and apoptosis, but still misunderstood the role of apoptosis.
- **(D) 4th draft:** I corrected the misunderstandings and structured the GA like the GA in Nomura *et al.* (2010).

4. References

CELL PRESS (2019) *Cell Press Graphical Abstract Guidelines*. Available at: https://www.cell.com/pb/assets/raw/shared/figureguidelines/GA_guide.pdf (Accessed: 25 November 2020).

NOMURA, D. K., LONG, J. Z., NIESSEN, S., HOOVER, H. S., NG, S.-W. AND CRAVATT, B. F. (2010) Monoacylglycerol Lipase Regulates a Fatty Acid Network that Promotes Cancer Pathogenesis, *Cell*. Elsevier, 140(1), pp. 49–61. doi: 10.1016/j.cell.2009.11.027.

TONG, C., LI, P., WU, N. L., YAN, Y. AND YING, Q.-L. (2010) Production of p53 gene knockout rats by homologous recombination in embryonic stem cells, *Nature*. 2010/08/11, 467(7312), pp. 211–213. doi: 10.1038/nature09368.