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| **Paper** | **Questions** | **Responses** |
| Spoerri L, Beaumont KA, Anfosso A, **Haass NK\*** (in press)  Real-time cell cycle imaging in a 3D cell culture model of melanoma.  ***Methods Mol Biol*** accepted for publication April 2nd 2016 | 1. What does this technique allow insights in to? 2. Spheroids mimic physiological tumour behaviour in term of what? |  |
| **Haass NK\*** (2015)  Dynamic tumor heterogeneity in melanoma therapy: how do we address this in a novel model system?  ***Melanoma Manag 2: 93-95***, [*http://www.futuremedicine.com/doi/pdf/10.2217/mmt.15.1*](http://www.futuremedicine.com/doi/pdf/10.2217/mmt.15.1)  ***This one is just a brief summary discussing why we do this kind of work.*** | Can you summarise why Haass’s team do this work? It is the most straight forward of all papers |  |
| Beaumont KA, Hill DS, **Daignault SM**, Lui GY, Sharp DM, Gabrielli B, Weninger W, **Haass NK\*** (2016)  [Cell Cycle Phase-Specific Drug Resistance as an Escape Mechanism of Melanoma Cells.](http://www.ncbi.nlm.nih.gov/pubmed/26970356)  ***J Invest Dermatol 136: 1479-1489***  ***This one has online movies – one of our key papers for this project.*** | 1. What is required to understand to develop effective therapeutic strategies for patients? 2. Hypoxia induces phenotype switching- what does this lead to? |  |
| Beaumont KA, Anfosso A, Ahmed F, Weninger W, **Haass NK** (2015)  [Imaging- and Flow Cytometry-based Analysis of Cell Position and the Cell Cycle in 3D Melanoma Spheroids.](http://www.ncbi.nlm.nih.gov/pubmed/26779761)  ***J Vis Exp 106: e53486***  ***This one has a 10 min video presentation.*** | 1. What does cellular heterogeneity contribute to and why? 2. What are the advantages of the 3D spheroid model? 3. What influences the choice of imaging strategy? 4. What two techniques does the FUCCI spheroid system combine with? 5. What did this study show when the order of drug delivery was changed? |  |
| **Haass NK**\*, Beaumont KA, Hill DS, Anfosso A, Mrass P, Munoz MA, Kinjyo I, Weninger W (2014)  [Real-time cell cycle imaging during melanoma growth, invasion, and drug response.](http://www.ncbi.nlm.nih.gov/pubmed/24902993)  ***Pigment Cell Melanoma Res 27: 764-776***  ***This one has online movies – one of our key papers for this project.*** | 1. Drug resistance is thought to be due to heterogeneity- what are new therapies targeting and why? 2. What extrinsic factors regulate tumour cells? 3. What did a combination of FUCCI and time lapse microscopy show? 4. What colours do the cell cycle phases appear to progress in? 5. What is the likely explanation for the segregation of cycling melanoma cells within spheroids? 6. Is the reaction to microenvironment stress reversible? |  |
| Beaumont KA, Mohana-Kumaran N, **Haass NK**\* (2014; Epub 2013 Dec 23)  [Modeling Melanoma In Vitro and In Vivo.](http://www.ncbi.nlm.nih.gov/pubmed/27429258)  Special Issue "Melanoma and Neoplasms of Skin"  ***Healthcare (Basel) 2: 27-46***  ***This one explains the different model types.*** | Briefly describe each of the models and list their advantages and any disadvantages. |  |
| **Extension papers (more complex)** | | |
| Tonnessen CA, **Haass NK**\* (2015)  Melanoma: From tumor specific mutations to a new molecular taxonomy and innovative therapeutics.  In: Bieber T & Nestle F (Eds.): Personalized Treatment Options in Dermatology  *Springer-Verlag Berlin Heidelberg, pp. 7-27*, DOI: 10.1007/978-3-662-45840-2\_2 | Where are melanocytes found in the body?  What is the most common form of melanoma?  What is the problem with treatments before 2011?  What new therapies changed melanoma treatment in 2011?  What two pathways are known to regulate cell proliferation?  What is the most common familial genetic alteration?  What amino acid is substituted at position 600?  What therapies are on the horizon? |  |
| **Haass NK**, Sproesser K, Nguyen TK, Contractor R, Medina CA, Nathanson KL, Herlyn M, Smalley KS (2008)  [The mitogen-activated protein/extracellular signal-regulated kinase kinase inhibitor AZD6244 (ARRY-142886) induces growth arrest in melanoma cells and tumor regression when combined with docetaxel](http://www.ncbi.nlm.nih.gov/pubmed/18172275).  ***Clin Cancer Res 14: 230-239***  ***Example for MEK inhibition.*** | Read the discussion the answer the following questions-   1. Which signalling pathway is research centering on? 2. What three things does MEK1/2 cause? 3. Why is it important to inhibit growth? |  |