During the first year of an honours Immunology degree at the University of Glasgow, I expressed an interest in a career in academic research. I had enjoyed the practical aspects of the degree course spent in the laboratory and had developed an interest in cell signalling from lectures I had received during the year. After discussions with lecturers at the University of Glasgow, I was put in touch with Dr David Waugh of the Centre for Cancer Research and Cell biology (CCRCB), in my home city of Belfast, who in turn introduced me to his colleague Dr James Murray, also of the CCRCB. It was decided that as Dr Murray's lab was involved in investigating signalling molecules, that during my summer studentship my time would be split between both Dr Waugh's and Dr Murray's labs.

I began my eight-week Biochemical Society studentship in July. The project undertaken was entitled "Does the inhibition of chemokine signalling in prostate cancer cells re-activate autophagy, and lead to cell death?". To attempt to answer this question I investigated the effect of the chemokine antagonist CXCR2 on levels of key regulators of autophagy, ULK1 and hVps34 in 2 different prostate cancer cell lines, PC3 and LNCaP. The process of autophagy contributes to the routine degradation and turnover of cells. It is activated under stressful conditions in cells, when nutrient levels are low. Autophagy promotes cell survival by the breakdown of sequestered cytoplasmic components, such a proteins, organelles, and cytoplasm, which can be used by the cell as nutrient sources.

ULK1 has protein kinase activity which is required for initiation of autophagy in mammalian cells and it has been shown that knockdown of ULK1 inhibits autophagosome formation. hVps34 is part of the Beclin1 complex which stimulates autophagy and is involved in early stages of autophagosome formation and it's synthesis of PtdIns3P is essential for autophagosome formation. Investigating these kinases should have allowed me to determine if blocking the IL-8 signalling pathway reactivated autophagy. mThis could prove detrimental to cancer cells under specific conditions, leaving them susceptible to hormone and chemotherapy treatments. Dr Waugh's lab had previously demonstrated that under stressful conditions, such as hypoxia, expression of the CXC chemokine IL-8 and it's signalling receptors CXCR1 and CXCR2 were induced. Therefore under stressful conditions IL-8 signalling is linked to maintenance of cell proliferation and survival, and confers a survival advantage. The levels of ULK1 and hVps34 were investigated in hypoxic prostate cancer cells along with control cells.

Although, on first reading, the experimental outline appeared to be straightforward I soon found that the techniques needed, were going to take time to master. Firstly, I had to learn mammalian tissue culture techniques, which I found difficult technically difficult, in the beginning. I quickly discovered that science can sometimes be slow as my LNCaP cell line grew slowly in culture and therefore the majority of my experiments were carried out on the PC3 cell line. The experiments involved taking cells that had reached confluency and placing them in a hypoxia chamber for 72 hours. This mean't that for the first few weeks I had a lot of free time, but I used this time to learn additional techniques, including how to assay protein and lipid kinase activities in vitro. After harvesting cell extracts, following hypoxia treatment, I found that there was not enough protein extract to allow me to immunoprecipitate the kinases to do the activity assays. This was a

persistent problem throughout the experiments and was very frustrating, but it demonstrated to me the reality of science; that sometimes experiments don't go right first time, or even the first four times! Once the experiment was scaled up to larger cell culture dishes I was then able to assay kinase activities of ULK1 from cellular extracts and after that I was very busy.

The studentship was a learning experience and therefore not long enough to gain conclusive results. However at the end of the eight weeks I had found that expression of ULK1 is higher in PC3 cells than other cell lines that I tested and that in PC3 cells CXCR2 and/or hypoxia caused a decrease in ULK1 protein expression, but an increase in hVps34 protein levels, in contrast to LNCaP cells where there appeared to be no difference in the protein levels of ULK1 or hVps34 following treatments. As far as I could determine, there was no differences in the activities of these kinases following CXCR2 and/or hypoxia in PC3 cells. Although my project did not answer the original research questions completely, I've been told that the data I generated will stimulate further research on this project in the laboratory, which was nice to hear.

Over the course of my Biochemical Society studentship I learned how to perform a number of techniques, including mammalian cell culture, SDS-PAGE, siRNA transfection, Western immunoblotting, immunoprecipitation and *in vitro* radiometric protein & lipid kinase assays. Working in the lab over the summer was completely different from what I expected, and entirely unlike the lab course at University. However, it gave me an insight into the reality of a research career and I would recommend undertaking a summer studentship to any student studying for a degree in which the last year contains a lab based honours project, as it helps to prepare for the reality of lab work. I now expect the first few weeks of my honours project to be a short learning experience as by the end of my Biochemical Society studentship I really felt like I had found my feet in a laboratory and I feel much better prepared and ready for starting my honours project.

I am extremely grateful for the opportunity given to me by the Biochemical Society and the help given to me on a day-to-day basis by Drs Murray and Waugh, as well as the postdoctoral researchers and postgraduate students in both labs. It has definitely been a positive experience, which I hope will be reinforced by my final year of study.