The contribution of immune cells to cancer progression and metastasis is now well established. Our lab is focused on a particular type of immune cell, called a gamma delta (γδ) T cell. We are exploring the involvement of γδ T cells in breast, colon, and pancreatic cancers. γδ T cell is actually an umbrella term that encompasses a variety of cell subsets with distinct properties and anatomical locations. There are γδ T cell subsets that kill cancer cells and other subsets that promote cancer progression. Our lab has ongoing projects aimed at understanding when and where these diverse γδ T cell subsets are important. We also study the role of immune cells during anti-cancer therapy response in ovarian cancer models.

At the beginning of 2020, we were excited to have Rob Wiesheu start as a PhD student after we secured funding from Breast Cancer Now. Soon after, the COVID-19 pandemic forced a major disruption to our work and dramatically slowed our progress. The experimental work was limited, and we started working from home. Despite the upheaval, our lab accomplished many things in 2020. We were proud that two of our lab members made a huge contribution to controlling the pandemic by volunteering at the Lighthouse COVID-19 testing facility. Our lab wrote and contributed to six scientific papers. Mark Lawrence was featured in a media article on the impact of lockdown on his PhD studies (https://www.glasgowlive.co.uk/news/glasgow-news/glasgow-lives-lockdown-mark-25-19285775). Mark was supervised by Patricia Ridsough. Mark was supervised by Joanne Edwards and Antonia Rosasver. Pancreatic Cancer UK Future Leader. Mark Lawrence was featured in a media article on the impact of lockdown on his PhD studies (https://www.glasgowlive.co.uk/news/glasgow-news/glasgow-lives-lockdown-mark-25-19285775). Mark was supervised by Patricia Ridsough. Mark was supervised by Joanne Edwards and Antonia Rosasver. Pancreatic Cancer UK Future Leader.

In previous years, we had generated a single cell RNA sequencing (scRNAseq) dataset of γδ T cells isolated from the lungs of tumour-free and tumour-bearing mice. This analysis has yielded a number of new targets for pro-metastatic γδ T cells, including co-inhibitory and co-stimulatory molecules expressed on the surface of these cells. We have found that a subset of lung γδ T cells expressed constitutively levels of PD-1 and ICOS. Manipulation of these molecules on lung γδ T cells in vivo and in mice has shown that PD-1 regulated IL-17A expression, while the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1). Excitingly, γδ T cell tumours regulated expression of another co-inhibitory molecule on lung γδ T cells, which is the receptor TIM-3. Blocking TIM-3 on lung γδ T cells increased IL-17A. This finding suggests that γδ T cells expressed constitutively levels of PD-1 and ICOS, and the function of ICOS remained unknown (Figure 1).