The Pursuit of Science: Reflections on Research, Leadership, and Discovery

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Introduction and Research Aims

Type 2 diabetes mellitus (T2DM) is a major global health challenge characterised by insulin resistance, chronic inflammation, and progressive metabolic dysfunction (Chandrasekaran and Weiskirchen, 2024). Cardiovascular disease (CVD) represents a major source of morbidity and mortality associated with T2DM (British Heart Foundation, 2025), highlighting the importance of understanding the cellular mechanisms that connect early metabolic dysfunction with later vascular disease.

My Laidlaw research project focused on investigating these mechanisms at a cellular level by exploring how insulin-resistant conditions influence protein expression and cellular behaviour in human umbilical vein endothelial cells (HUVECs) and adipocytes, with an overarching interest in understanding the intercellular communication between adipose tissue and the vascular endothelium.

Prior studies strongly suggest that adipose tissue and endothelial cells engage in intercellular communication mediated by signalling molecules including adipokines, cytokines, and microRNAs (miRNAs) (Viswambharan et al., 2021). When this communication becomes dysregulated during metabolic disease, it may contribute to endothelial dysfunction and ultimately CVD.

I was particularly interested in Meteorin-like (METRNL) and inflammatory miRNAs as potential molecular mediators of this process. METRNL has been implicated in metabolic regulation, inflammation, mitochondrial function, and insulin sensitivity (Wang et al., 2026), meanwhile miRNAs can regulate gene expression and act as intercellular signalling molecules. Together, these molecules serve as likely catalysts of metabolic dysfunction and could influence endothelial behaviour.

The primary aim of my project was to investigate cellular and molecular changes occurring under insulin-resistant conditions, using HUVECs as an endothelial model. Through experimental protein analysis, microscopy, flow cytometry, bioinformatics, and literature review, I examined potential mechanisms connecting metabolic dysfunction with CVD, seeking to identify a novel pathway or potential biomarker for earlier diagnosis and intervention.

Within six weeks of research, I was able to generate preliminary evidence and develop a roadmap for future studies investigating protein localisation, protein-protein interactions, and intercellular signalling using advanced imaging technologies.

Research Background

Insulin Resistance and Cardiovascular Disease

Insulin resistance is a defining feature of T2DM and occurs when cells become less responsive to insulin, resulting in impaired glucose regulation and lipid metabolism (Galicia-Garcia et al., 2020). However, insulin resistance is not confined to classical metabolic tissues. Vascular endothelial cells are also responsive to insulin, meaning that disruption of endothelial insulin signalling can result in harmful consequences to vascular homeostasis.

Therefore, measurement of blood glucose alone is not sufficient to examine the cardiovascular consequences of T2DM. Chronic inflammation, oxidative stress, altered lipid metabolism, and endothelial dysfunction can interact to create a systemic pathological environment in which CVD develops and progresses.

Endothelial Dysfunction as an Early Feature of Insulin Resistance

The vascular endothelium is essential for maintaining vascular homeostasis. Endothelial cells respond directly to insulin through signalling pathways involving the insulin receptor, insulin receptor substrates, phosphoinositide 3-kinase (PI3K), and protein kinase B (Akt).

Under physiological conditions, insulin signalling promotes endothelial nitric oxide production through Akt-mediated activation of endothelial nitric oxide synthase (eNOS). Nitric oxide contributes to vasodilation and helps maintain vascular function, most notably glucose uptake (Cyr et al., 2020).

However, this signalling pathway is disrupted during insulin resistance. Chronic exposure to inflammatory cytokines, free fatty acids, hyperglycaemia, and oxidative stress activates pathological pathways that interfere with insulin signalling. Consequently, insulin-resistant states may selectively impair pathways responsible for nitric oxide production while leaving other insulin-responsive pathways relatively active.

Oxidative stress further contributes to this dysfunction as the excessive production of reactive oxygen species (ROS) can reduce nitric oxide bioavailability, hence promoting inflammatory and vascular changes (Cyr et al., 2020). Mitochondrial dysfunction and altered cellular redox signalling can therefore reinforce endothelial dysfunction.

These changes often occur early in the development of metabolic disease. Understanding endothelial responses to insulin-resistant conditions could therefore provide insight into the mechanisms preceding more severe cardiovascular complications.

Adipose-Endothelial Crosstalk

Adipose tissue is far more dynamic than a means for energy storage. It serves as an active endocrine organ that communicates with other tissues through adipokines, cytokines, extracellular vesicles, and other signalling molecules (Viswambharan et al., 2021).

During obesity and insulin resistance, adipose tissue undergoes substantial metabolic and inflammatory changes. This consequently alters the molecules they release into the surrounding environment and circulation. Because endothelial cells are continuously exposed to circulating signals, they become at risk of experiencing metabolic dysfunction.

Meanwhile, endothelial cells can themselves influence adipocyte differentiation, metabolism, and tissue vascularisation. Disruption of this bidirectional communication creates a feedback loop wherein metabolic dysfunction and vascular dysfunction reinforce one another.

This concept provided an important framework for my experimental studies. Rather than viewing altered endothelial protein expression as an isolated cellular response, I considered it as a component of a larger network of communication between metabolic and vascular tissues.

Potential Molecular Mediators: METRNL and Inflammatory microRNAs

Two molecular areas became particularly relevant to my investigation: METRNL and inflammatory miRNAs.

METRNL is an adipokine that has been associated with metabolic regulation, insulin sensitivity, inflammation, and mitochondrial function (Wang et al., 2026). Its potential involvement in endothelial and metabolic processes makes it a strong candidate for investigating how adipose-derived signals may influence vascular cells.

MicroRNAs are another potential candidate involved in this intercellular communication. These small, non-coding RNAs regulate gene expression post-transcriptionally and can influence pathways involved in inflammation, oxidative stress, insulin signalling, and cellular metabolism. Moreover, they can be transported between cells through extracellular vesicles, further positioning them as a likely component of adipose-endothelial intercellular communication.

Together, these mechanisms raise the possibility that dysregulated molecular communication could contribute to the transition from metabolic dysfunction to CVD.

Methodology

HUVEC Culture and Metabolic Treatment

My experimental work used human umbilical vein endothelial cells (HUVECs) as an in vitro model of vascular endothelial cells.

This developed my skills in working within a tissue culture laboratory, including aseptic technique to maintain ultra-sterile experimental conditions. This involved careful preparation of the apparatus, appropriate handling of cultures, and consistent awareness of potential sources of contamination.

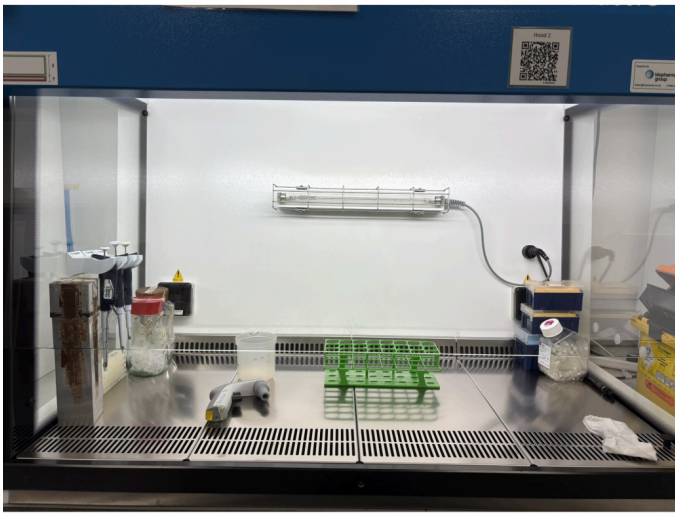

Fig. 1: Aseptic technique experimental setup for tissue culture work.

Following induction training, HUVECs were exposed to glucose and insulin treatment to model metabolic conditions associated with insulin resistance. Cells were subsequently incubated before being harvested for downstream analysis.

Microscopy and Cell Phenotyping

I observed HUVECs, undifferentiated human pre-adipocytes, and differentiated white adipocytes under an inverted phase-contrast microscope. Comparing their morphologies and phenotypes helped me connect the theoretical concepts I studied in the literature with my experiment.

I subsequently visited the Cheney Biomedical Accelerator, where I was introduced to advanced imaging technologies, including confocal and widefield fluorescence microscopy. These technologies are particularly relevant to the future direction of my project because they could allow investigation of protein localisation and translocation within cells.

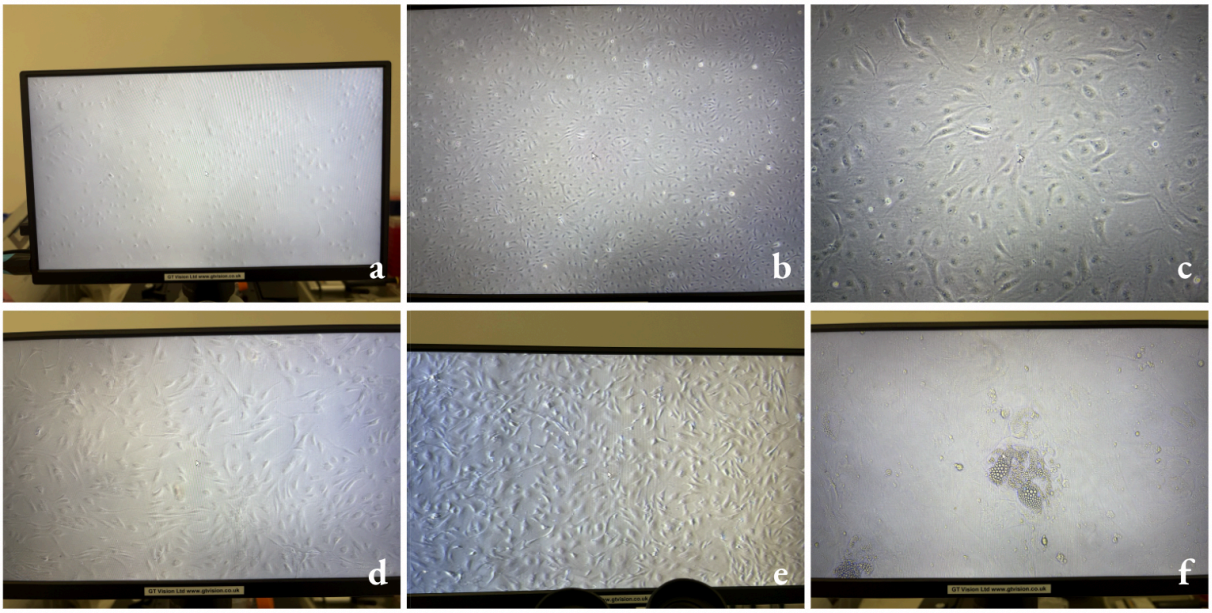

Fig. 2: (a) Human umbilical vein endothelial cells (HUVECs). **(b)** Undifferentiated human adipocytes (24 hours). **(c)** Undifferentiated human adipocytes (48 hours). **(d)** Undifferentiated human adipocytes (5 days). **(e)** Undifferentiated human adipocytes (14 days). **(f)** Differentiated white human adipocytes.

Protein Extraction and Western Blotting

The main component of my experimental work involved analysing protein expression using SDS-PAGE electrophoresis and Western blotting.

These procedures involved preparing cellular protein samples, separating proteins according to molecular weight using SDS-PAGE, transferring the separated proteins onto a membrane and subsequently detecting proteins of interest through antibody-based staining and densitometric imaging.

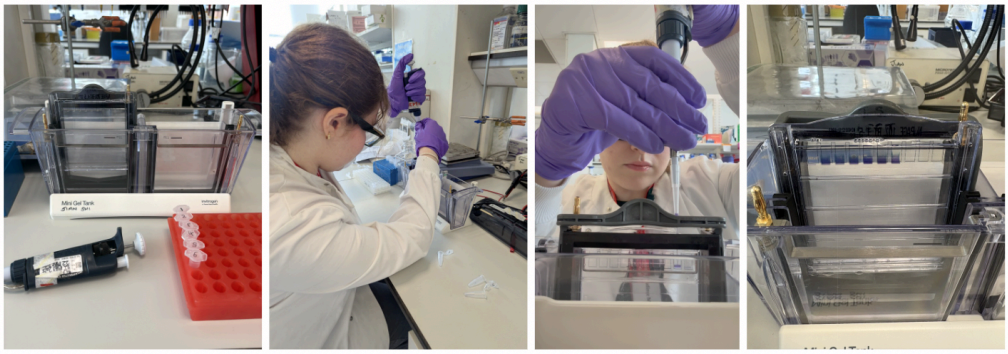

Fig. 3: Sample loading to separate proteins via SDS-PAGE electrophoresis.

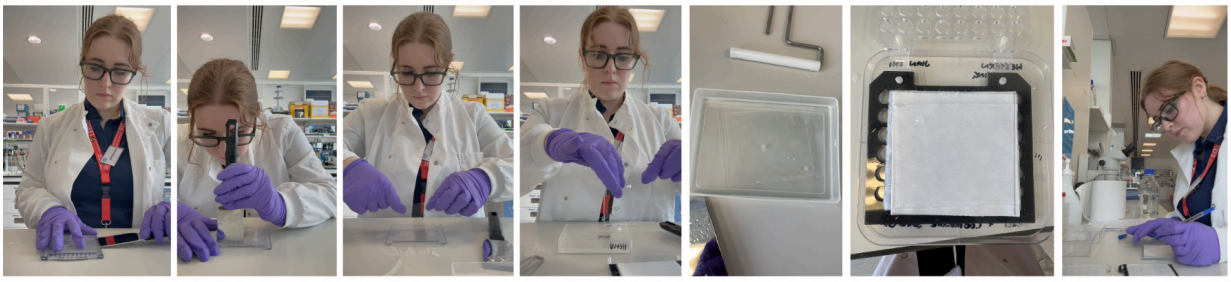

Fig. 4: Assembly of Western blot cassette for membrane transfer.

Flow Cytometry

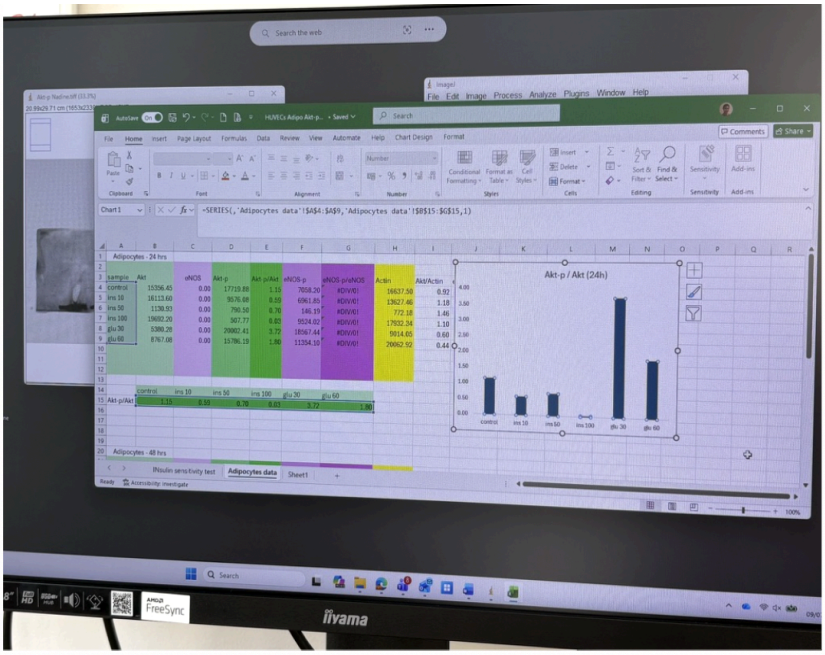

Fig. 5: Protein quantification and single-cell analysis.

As part of an inter-laboratory collaboration, I visited the Northern Cell Metrology Hub at Nexus, the University of Leeds' research and innovation hub. I had the opportunity to shadow researchers working with flow cytometry and learn how this technique can provide information at the single-cell level. This complemented my experience with Western blotting, which provides information about protein abundance across a population of cells. The experience reinforced the importance of using complementary techniques to answer complex biological questions and combine varying approaches to produce a more comprehensive understanding of disease mechanisms.

Bioinformatics and Literature Review

I used secondary data and bioinformatics tools to investigate inflammatory miRNA signatures relevant to cardiovascular pathology. I conducted an integrative network analysis of inflammatory microRNAs, using publicly available datasets and bioinformatics resources to explore shared regulatory mechanisms.

Although this work did not generate novel biological discoveries, it provided a valuable roadmap for prioritising future research directions and expansions of my project.

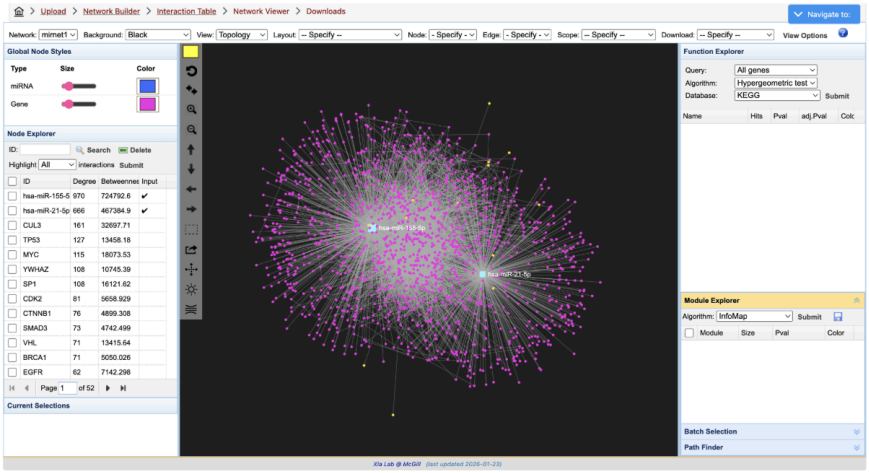

Fig. 6: Integrative network analysis of key inflammatory miRNAs using bioinformatics platform miRNet.

Findings and Research Progress

At the experimental level, I successfully mastered the skills of human tissue culture and Western blotting, completing the full protocols independently, from harvesting cell and tissue samples, protein separation, transfer, and imaging.

During my first independent transfer, the molecular weight ladder failed to appear on the membrane. Rather than immediately relying on my supervisor to identify the problem, I reflected on the protocol and considered which stages could have produced the outcome. I identified insufficient transfer buffer within the tank as the likely cause, repeated the procedure, and successfully obtained the expected transfer.

Similarly, when my initial imaging produced unclear bands, reviewing the protocol revealed that membrane washing and incubation had not been performed for sufficient durations. Repeating these stages resulted in clearer bands.

These experiences demonstrated that successful experimental research involves more than following protocols accurately. It requires understanding the underlying logic of the protocol so I can be adaptable to unexpected results and have an agile mindset to allow for effective troubleshooting.

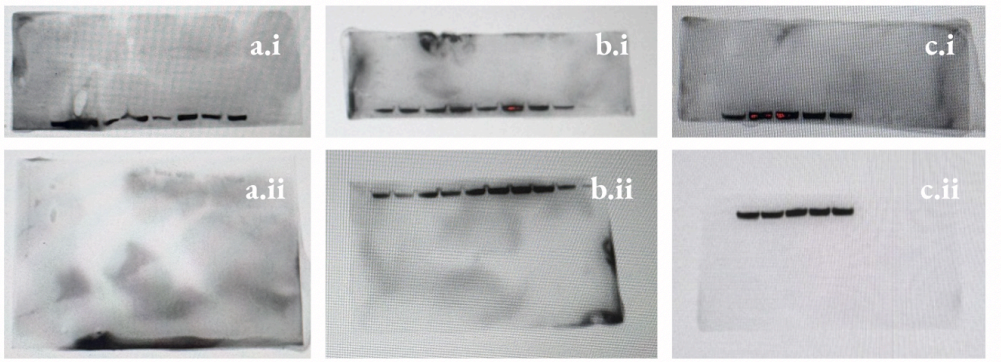

Fig. 7: (a) Western blot imaging before troubleshooting; bands distorted and unclear, background is patchy. **(b)** Western blot imaging after troubleshooting; bands are clearer but background is still patchy. **(c)** Western blot imaging after troubleshooting; bands and background are both clear.

As the project progressed, I began compiling the Western blot data I generated and examined trends in protein expression. This allowed me to move beyond considering each experiment individually and begin asking what the combined findings infer about the cellular response to insulin-resistant conditions.

Because the six-week timeframe limited the number of experimental replicates and downstream analyses that could be completed, these preliminary observations cannot be considered sufficient to establish a complete molecular pathway. Instead, they serve as pilot data setting a foundation for further investigation.

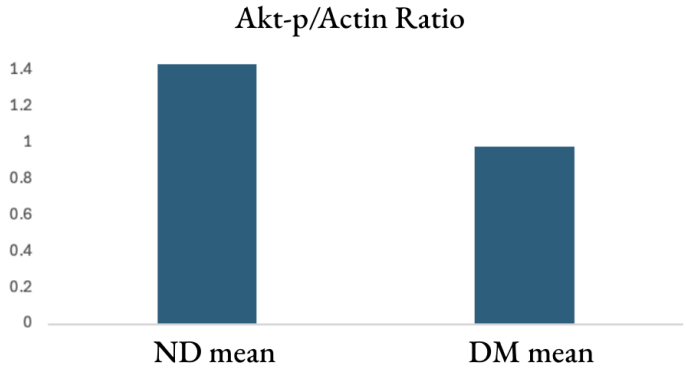

Fig. 8: Western blot analysis of Akt phosphorylation (Akt-p) normalized to Actin in non-diabetic (ND) versus diabetic (DM) SVECs.

The experimental and computational work undertaken during my project produced several findings that helped refine my understanding of the molecular mechanisms associated with insulin resistance and endothelial-adipocyte communication.

One of the clearest findings came from the analysis of Akt phosphorylation, an important component of the canonical insulin signalling pathway. In diabetic SVECs, Akt phosphorylation was 32% lower than in non-diabetic SVECs, with mean Akt-p/Actin ratios of 0.98 and 1.44 respectively (Fig. 8). This

reduction suggests decreased activity of the Akt signalling pathway in diabetic endothelial cells and is consistent with impaired insulin signalling under diabetic conditions.

This finding was particularly valuable because it provided an experimental link between the established insulin-Akt signalling pathway and the endothelial dysfunction associated with metabolic disease. The canonical pathway involves insulin receptor activation and downstream PI3K-Akt signalling, which contributes to processes including glucose uptake and endothelial nitric oxide production. Reduced Akt activity could therefore have consequences for endothelial function and vascular homeostasis.

However, the response of adipocytes to metabolic treatment produced a more unexpected result. High-glucose exposure increased Akt phosphorylation in adipocytes, despite the insulin-resistant experimental conditions. Rather than simply indicating improved insulin signalling, this observation may represent a compensatory metabolic response to the stress stimulus.

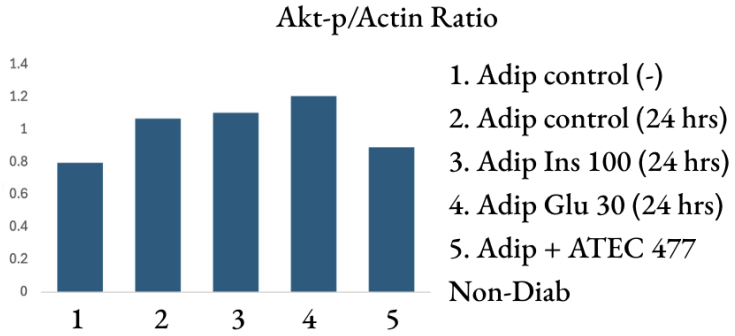

Fig. 9: Western blot analysis of Akt phosphorylation (Akt-p) normalized to Actin in adipocytes following metabolic and endothelial-derived treatments.

An even more interesting observation arose when adipocytes were exposed to the endothelial-derived factor ATEC 477. Treatment reduced Akt phosphorylation relative to the 24-hour control (Fig. 9). This suggests that endothelial-derived signals can influence adipocyte metabolic signalling and may act through mechanisms extending beyond the canonical insulin-Akt pathway.

These findings support the concept that endothelial and adipocyte signalling may contribute to the regulation of metabolic homeostasis. The contrasting effects of glucose exposure and endothelial-derived treatment suggest that cellular responses to metabolic stress are more complex than a simple increase or decrease in insulin signalling. Instead, endothelial-derived factors may influence adipocyte function through alternative or compensatory signalling mechanisms.

The bioinformatics component of the project provided further evidence for the involvement of interconnected metabolic and inflammatory pathways. KEGG enrichment analysis identified significant enrichment of pathways including TNF signalling, insulin resistance, insulin signalling, PI3K-Akt signalling, AMPK signalling, adipocytokine metabolism, and TGF-β signalling (Table 1). The strongest enrichment was observed for TNF signalling (adjusted p = 1.4×10^{-12}), followed by insulin resistance (7.8×10^{-9}), insulin signalling (1.45×10^{-8}), and PI3K-Akt signalling (1.48×10^{-8}).

Pathway	Adjusted P-value
TNF signalling	1.4e-12
Insulin resistance	7.8e-9
Insulin signalling	1.45e-8
PI3K-Akt signalling	1.48e-8
AMPK signalling	8.02e-8
Actin cytoskeleton regulation	0.0000121
TGF-β signalling	0.0000831
Toll-like receptor signalling	0.000143
Adipocytokine metabolism	0.000171
Fatty acid metabolism	0.000781
Glycolysis/ gluconeogenesis	0.00194
Type 2 diabetes mellitus	0.00293
Vascular smooth muscle contraction	0.00736
Adipocyte lipolysis	0.0113
Tight junction	0.0113
JAK–STAT signalling	0.0208

Table 1: Significant inflammatory and metabolic pathways identified by KEGG enrichment analysis via miRNet.

The convergence of these pathways was particularly informative because it reinforced the idea that insulin resistance cannot be understood through a single signalling pathway in isolation, but rather is highly systemic. Inflammatory, metabolic, and vascular pathways appear to intersect, providing potential molecular routes through which metabolic dysfunction could catalyse cardiovascular consequences.

However, the lack of repeated trials limits the reliability and concordance of the experimental findings, while endothelial cells and adipose tissue were cultured separately, meaning that the proposed intercellular crosstalk was not experimentally validated.

Seeing that altered Akt phosphorylation represents part of a broader signalling response, the next step is to determine where these signalling changes occur within the cell and how they interact with other molecular components. This led me towards exploring the subcellular localisation of phosphorylated Akt using confocal microscopy, its potential co-localisation with eNOS and METRNL, and the structural organisation of endothelial junctions using VE-cadherin staining in a future project.

A further long-term direction would be to directly visualise endothelial-adipocyte communication using fluorescent labelling and live-cell imaging, allowing uptake of endothelial-derived factors by adipocytes to be investigated rather than inferred from changes in protein expression alone.

Discussion and Future Research Directions

One of the most significant outcomes of the project was the development of a clearer research roadmap. At the start of my research timeline, the potential scope of the project was extremely broad.

Discussions with my supervisor introduced me to numerous possible avenues, including protein-protein interactions, intracellular localisation, adipocyte-endothelial communication, and bioinformatics.

Although each of these research areas are equally important in combating the global health crisis of T2DM and CVD, they could not all be addressed within six weeks. This required me to distinguish between what was important in the long term and what was achievable within the immediate project timeframe. Using project-management principles I had learned during the leadership development residential, including SMART goal setting and the Eisenhower matrix, I effectively narrowed the scope of my research (Fig. 10).

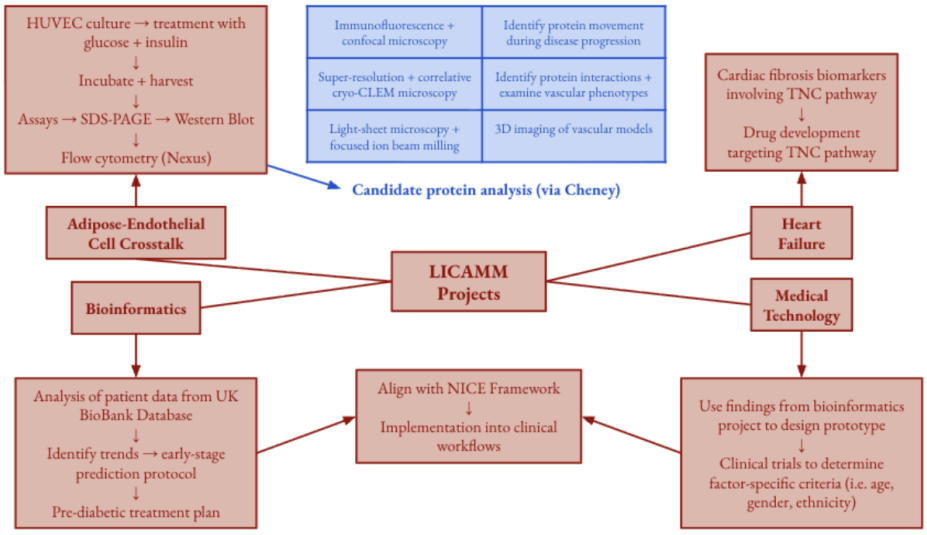

Fig. 10: Current and future project planning.

This resulted in a more focused experimental plan centred on understanding endothelial responses to insulin-resistant conditions while establishing the foundation for more detailed investigations in the future.

One particularly promising direction is the use of confocal and widefield fluorescence microscopy to investigate protein localisation and translocation. Western blotting can demonstrate that protein abundance changes, but it cannot tell us where within the cell that protein is located or whether its spatial distribution changes during disease.

More advanced technologies available through the University of Leeds' Cheney Biomedical Accelerator could subsequently enable investigation of these questions at increasingly high spatial resolution. Super-resolution microscopy and cryo-correlative light and electron microscopy (cryo-CLEM) could

eventually provide information about protein organisation and interactions that cannot be obtained through conventional Western blotting alone.

The ultimate significance of this work lies in its potential contribution to understanding the earliest stages of metabolic dysfunction and CVD. Improving our understanding of the cellular mechanisms underlying insulin resistance may help identify molecular biomarkers or therapeutic targets that could eventually support earlier detection and intervention.

The research conducted this summer therefore contributes to a wider pool of scientific knowledge surrounding the cellular mechanisms driving early-stage T2DM. While my work does not directly produce a diagnostic or therapeutic intervention, understanding these mechanisms is an important step towards identifying opportunities for more effective early-stage diagnosis and prevention of the severe cardiovascular and metabolic complications associated with late-onset T2DM.

Impact of Conducting Research

Starting in a professional research environment can be intimidating, particularly when you are one of the least experienced people in the room. During my first few days, I was hesitant to take initiative because I was worried about making mistakes, damaging equipment, or wasting expensive laboratory materials.

These concerns did not disappear immediately. However, as I became more familiar with the laboratory environment, they gradually became easier to manage.

A particularly meaningful turning point occurred when my supervisor began allowing me to work independently. She would remain available if I had questions, but for the most part, I was encouraged to work through problems myself.

Initially, this was overwhelming. There were moments when I felt completely clueless about what I was supposed to do next. However, research does not always provide clear instructions. Even experienced researchers encounter failed experiments and unexpected results. Learning to operate in that environment taught me to rely more confidently on my own knowledge and develop my critical thinking skills.

The mistakes I made during Western blotting were particularly valuable in this regard. Rather than viewing an unsuccessful transfer simply as a failed experiment, I learned to ask why it had failed.

Understanding the relationship between the protocol and the resulting outcome gave me a much stronger conceptual understanding of the technique and the underlying scientific theory.

This changed my perception of mistakes. Previously, I associated making a mistake with not knowing enough. Through this project, I began to understand that mistakes can instead be opportunities to identify gaps in understanding and develop better problem-solving strategies. Not only does this further the process of scientific discovery, it has also helped me come into my own as a researcher so that I may offer a unique perspective and subsequently, meaningful contributions to science.

The project was also different from how I initially imagined research would be. I had expected most of my time to be spent physically carrying out experiments. In reality, research involves a much broader range of activities: reading literature, planning experiments, troubleshooting, analysing data, writing, creating figures, attending seminars, collaborating with colleagues, and synthesising ideas for future research directions that bridge the gap between what is known and what has yet to be discovered.

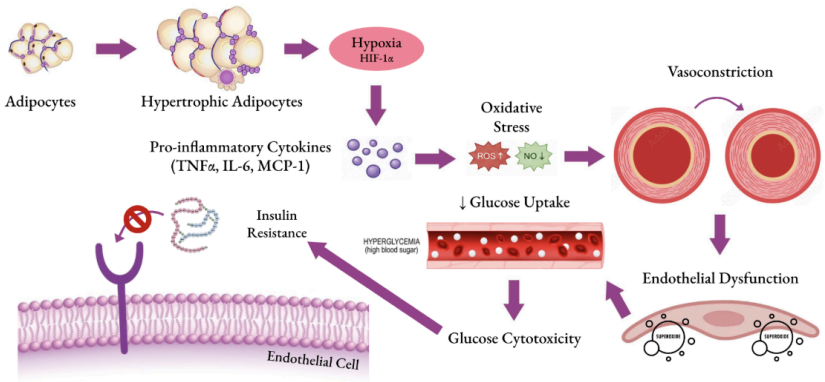

Fig. 11: One of the scientific figures I created for my literature review, outlining the pathological disease response under insulin-resistant conditions.

Leadership Development

Although the project was primarily a research experience, it also significantly developed my understanding of leadership. I developed many skills and attributes that are characteristic of an effective and ethical leader, one of the most important ones being confidence. Initially, I relied heavily on my supervisor for reassurance. As the project progressed, she deliberately gave me more responsibility. By the midpoint of the project, I was completing Western blots independently and using my own judgement to troubleshoot problems.

This taught me that effective leadership does not necessarily mean knowing the answer immediately. It can mean having the confidence to navigate uncertainty, seek information when necessary, and make informed decisions with incomplete information.

The project also strengthened my adaptability. My original ideas for the project were broader than what could realistically be achieved in six weeks. Rather than viewing this as a limitation, I learned to restructure the project around achievable milestones while preserving the longer-term research vision. Moreover, with the constant need to troubleshoot and redirect my efforts, I learned that no amount of scheduling and planning can compensate for the everchanging nature of any industry, and a leader should be able to thrive in spite of those obstacles.

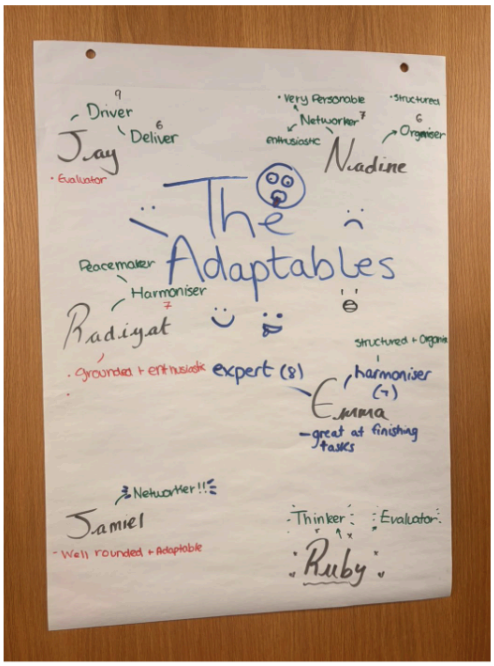

Fig. 12: Group task from leadership development residential; evaluating our Quintax profiles.

This also helped me develop my critical thinking skills. Instead of following protocols mechanically, I became increasingly interested in understanding the reasoning behind them. When an experiment produced an unexpected result, I learned to examine the method, identify possible sources of error, and consider how the outcome related to the underlying biology.

Furthermore, I became a more effective communicator and strengthened my abilities across a range of different modes of communication. I practised communicating through my literature review, research report, conference poster, and scientific figures (Fig. 11). Designing figures for my literature review and research poster was particularly useful because it required me to distill complex molecular pathways into visual representations without losing their scientific meaning.

I also gained experience communicating science verbally by attending master's student presentations and engaging in discussions with my supervisor, facility managers, and researchers at Nexus and the Cheney Biomedical Accelerator.

The most important skill I developed, however, was the ability to synthesise and conceptualise ideas and see the bigger picture. Throughout the project, I learned to look beyond individual experiments and ask how each finding contributed to a wider understanding of disease. This meant constantly

considering not only what my results showed, but why they mattered and how they could eventually translate beyond the laboratory. Discussions with my supervisor encouraged me to think about the practical applications of our research and its potential to contribute to earlier diagnosis, improved treatment, and better patient outcomes. This shift in perspective taught me that impactful scientific research requires more than technical competence, but also the ability to connect individual findings to wider clinical and societal challenges.

Most importantly, this research experience reinforced my belief that the foundation of effective and ethical leadership is genuine and authentic compassion. Scientific progress ultimately has little meaning if it is disconnected from the people it is intended to serve. Throughout this project, I became increasingly conscious of the responsibility scientists have to use their knowledge to address unmet needs, challenge inequities, and improve the lives of others. For me, leadership means far more than achieving personal or professional success. Leadership is a practice, not a position. A true leader has the compassion to recognise injustice, the conviction to challenge it, and the willingness to use their skills and the opportunities available to them in service of the wider public, especially in defense of underrepresented communities and those without a voice. This is a principle I hope to carry with me throughout my career as a scientist and leader.

Fig. 13: The Laidlaw 3C's.

Dissemination of Research

Before beginning the laboratory component of my Laidlaw project, I designed a bioinformatics practical using publicly available secondary data and applied several bioinformatics tools to investigate inflammatory miRNAs and their potential regulatory relationships with cardiovascular pathology. Upon completing this research, I decided to challenge myself and put my communication skills into practice by preparing an abstract for the Genetics Society RNA in Physiology & Disease conference.

Although the analysis did not generate a novel discovery, it provided valuable experience in scientific writing, data analysis, and interpretation. More importantly, the findings of the research support the use of integrative miRNA network analysis to identify candidate regulatory molecules and molecular pathways that may contribute to the development of cardiovascular complications in T2D. Bioinformatics approaches such as this offer a valuable framework for identifying and prioritising candidate biomarkers and therapeutic targets, providing a foundation for future experimental validation.

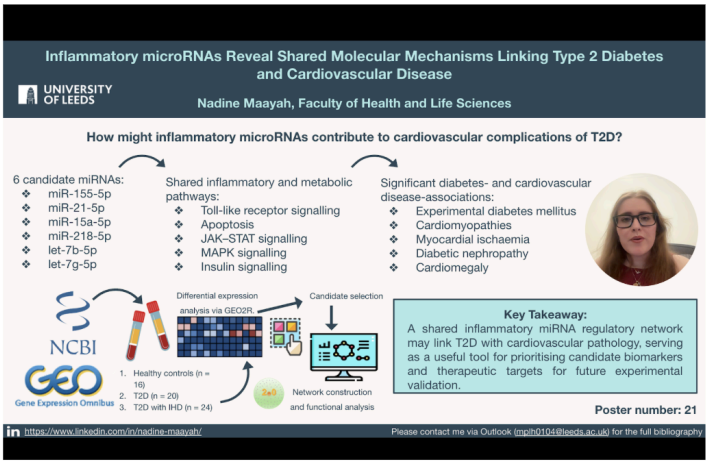

Fig. 14: Flash talk presentation of my research delivered virtually at The Genetics Society RNA in Physiology & Disease Conference.

I was subsequently delighted to have my abstract selected for presentation at the Genetics Society RNA in Physiology & Disease conference in Newcastle upon Tyne in September 2026.

Preparing this poster alongside my Laidlaw project has allowed me to practise communicating research visually and concisely. This experience has also prepared me for the upcoming Laidlaw Conference at Imperial College London, where I will have the opportunity to present and discuss my research with other scholars.

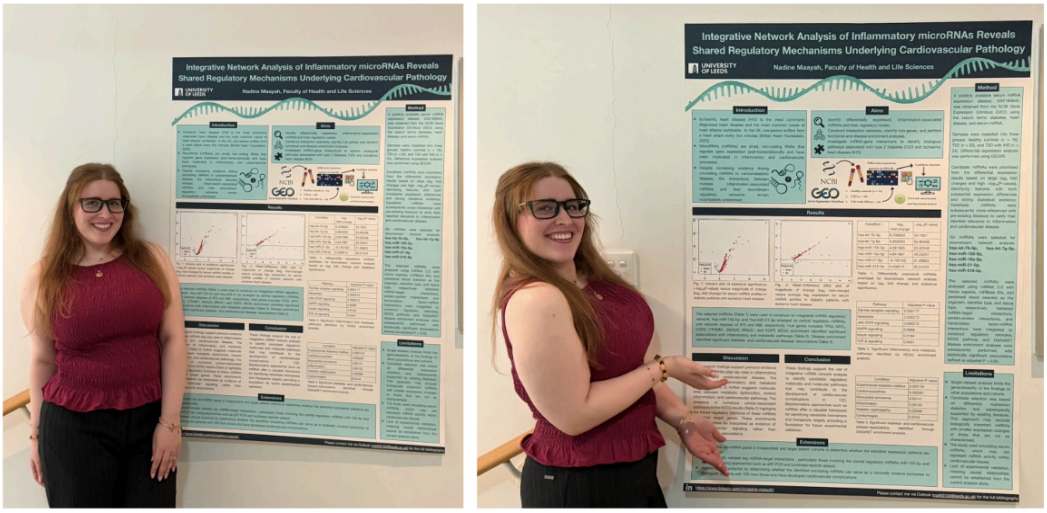

Fig. 15: Presenting my research poster for The Genetics Society RNA in Physiology & Disease Conference.

Future Career and Research Plans

Before beginning the Laidlaw programme, I was already interested in genetics, disease mechanisms, and translational research. However, I primarily viewed research through the lens of laboratory experimentation.

After completing my research project, I now understand the multidisciplinary nature of scientific research. This has strengthened my interest in pursuing future research opportunities where I can combine these approaches and skills.

The project has also given me several potential directions for continued research. The ideas that could not be addressed during the six-week project now form a roadmap for future projects. These could potentially be explored during a placement year, my third-year dissertation, or postgraduate research.

In particular, I am interested in continuing to investigate the relationship between metabolic dysfunction, inflammation, and cardiovascular disease, potentially using advanced imaging and bioinformatics approaches to investigate cellular signalling at greater resolution.

The possibility of working with the Cheney Biomedical Accelerator in a future project is particularly exciting. My exposure to its imaging technologies has shown me how advanced microscopy could complement conventional molecular biology techniques and help bridge the gap between protein expression and cellular function.

Ultimately, I hope to pursue a career in biomedical research that combines molecular understanding with translational impact. My long-term ambition is to contribute to research that can eventually improve how disease is detected, prevented, and treated. Furthermore, I aspire to use my platform as a biomedical researcher to enter the public health sector and leverage my skills and experience to lobby for change in global health policies that make treatment and patient care more accessible, effective, and affordable across all demographics.

Conclusion

Six weeks in the laboratory have given me considerably more than the opportunity to learn new experimental techniques.

I began the project with limited experience in tissue culture and Western blotting and some apprehension about working independently in a professional research environment. I leave with

greater technical competence, confidence in my ability to troubleshoot experiments, a stronger understanding of cardiovascular and metabolic research, and a clearer vision for the research questions and career path I want to pursue in the future.

The most important lesson I have learned is that research is rarely linear. Experiments fail, hypotheses change, timelines evolve, and new questions emerge from unexpected results. Learning to navigate this uncertainty has been one of the most valuable aspects of the project.

My research has also changed how I think about scientific impact. The work I conducted this summer represents a small contribution to a much larger effort to understand the cellular mechanisms underlying insulin resistance and cardiovascular complications. While it is unlikely that any single six-week project can resolve such a complex clinical problem, research progresses through the accumulation of evidence, each study representing a piece of the larger picture.

I am extremely grateful to my supervisor for creating an environment in which I was encouraged to become increasingly independent while knowing that support was always available. Her willingness to let me troubleshoot problems rather than immediately solve them for me played a significant role in developing my confidence and critical thinking.

I am equally grateful to the Laidlaw Foundation for providing me with the opportunity to undertake this project. The programme has allowed me to experience research not simply as an academic exercise, but as a process of questioning, experimentation, collaboration, and discovery.

Most importantly, I leave the project with more questions than I started with. Rather than seeing this as a sign that I have not found enough answers, I see it as a gateway to new avenues of discovery, deeper questions, and opportunities to continue building upon what I have learned.

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