



Propuesta de Trabajo Fin de Máster

Año académico 2026-2027

MÁSTER EN CIENCIA DE DATOS PARA CIENCIAS EXPERIMENTALES

<b>Proyecto Nº 18</b>
<b>Título:</b> Decoding Epigenetic Mechanisms Driving Cardiac Fibrosis
<b>Departamento/ Laboratorio:</b> Bioquímica y Genética
<b>Director:</b> Beatriz Pelacho Samper <b>Correo electrónico:</b> bpelacho@unav.es <b>Codirector:</b> <b>Correo electrónico:</b>
<b>Resumen:</b> <p>Cardiovascular disease, primarily driven by adverse remodeling and fibrotic scar formation, remains the leading cause of mortality and morbidity worldwide. Despite significant research progress, effective anti-fibrotic therapies have not yet reached clinical practice. However, promising advances are emerging through the exploration of novel epigenetic approaches.</p> <p>Our group has employed both <b>bulk and single-cell CRISPR perturbation screens</b> in primary cardiac fibroblasts (CFs), together with ex vivo functional assays, to identify key chromatin regulators involved in fibrotic processes (<a href="https://doi.org/10.1038/s41467-025-66597-9">doi: 10.1038/s41467-025-66597-9</a>). Subsequent ATAC-seq analyses have revealed the roles of multiple transcription factors regulated by these chromatin regulators, highlighting their critical involvement in fibrosis.</p> <p>Building on these findings, we aim to further investigate the underlying molecular mechanisms by first conducting <b>in silico analyses</b> of the signaling pathways governed by these factors in both experimental models and clinical samples from patients with cardiac disease. This initial approach will leverage both public and proprietary single-cell datasets to gain deeper insight into the regulatory networks involved.</p> <p>In parallel, we will <b>experimentally</b> examine the regulation and interactions of these factors using primary murine fibroblasts and established cell lines stimulated with fibrotic cues and treated with <b>candidate regulatory drugs</b>. <b>Functional in vitro assays</b> will be used to elucidate the molecular mechanisms underlying the regulatory effects of these inhibitors.</p> <p>Our major innovative outcome will be the identification and development of novel molecules with significant potential to reduce cardiac fibrosis, and possibly other fibrotic diseases, with future clinical applications. The successful development of a new anti-fibrotic therapy would address a major global health challenge and could reduce healthcare costs, shorten hospital stays, and significantly improve patient outcomes.</p>

<b>OPTATIVAS RECOMENDADAS</b> <ol style="list-style-type: none"><li>1.</li><li>2.</li><li>3.</li><li>4.</li></ol>
--