Research Statement

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WHERE MY WORK MEETS THIS LABORATORY

Your oncology programme targets three compartments — tumour cells, tumour-associated macrophages and the lymphatics. My doctoral work addressed the first and my contributing work addressed the second. The thread joining them, and the reason I am writing, is that **the tumour's escape routes are multiple and a carrier is the instrument for closing more than one at a time**.

WHAT I ESTABLISHED

I designed a **mesoporous polydopamine-based Pickering emulsion** carrying two agents with incompatible solubility and release requirements: shikonin, which inhibits PKM2 and suppresses tumour glycolysis, and JQ1, a BET bromodomain inhibitor. Co-delivery in one particle held the ratio fixed to the point of release, which separate administration cannot do.

In triple-negative breast cancer models the system suppressed primary tumour growth and lung metastasis. The mechanism ran through three independent routes acting together: **apoptosis**, blockade of **epithelial–mesenchymal transition**, and blockade of **vasculogenic mimicry**, in which tumour cells build their own perfusion channels and so evade anti-angiogenic therapy. Published in *Acta Pharmacologica Sinica* 46(12):3314-3326, **joint first author, equal contribution formally recorded**.

Separately, as a contributing co-author within my institute's macrophage programme, I worked on lactoferrin-modified carriers targeting LRP-1 on activated macrophages (*Acta Pharmaceutica Sinica B*) and on a review of macrophage-based nanotherapeutics (*Journal of Controlled Release*). I also wrote a first-author review in **Drug Delivery and Translational Research** on how nanocarriers can be used to modulate the tumour immune microenvironment.

WHAT I WOULD PROPOSE HERE

1. Myeloid re-education as a delivery problem rather than a drug problem. Repolarising tumour-associated macrophages usually fails not because the agents are weak but because they reach the wrong cells. I would work on carriers whose biodistribution is validated at the level of the myeloid compartment rather than the tumour as a whole — which requires the flow cytometry and immunofluorescence I use routinely.

2. Vasculogenic mimicry alongside macrophage targeting. These are treated as separate literatures. My doctoral result suggests they need not be: a carrier that reaches both the tumour cell and the myeloid compartment could close a structural escape route and an immunological one together.

3. The metabolic axis. Shikonin acts on glycolysis, and the tumour microenvironment's metabolic state governs macrophage phenotype. Whether a glycolysis-directed payload delivered to macrophages shifts polarisation is an open, testable question that sits inside your existing programme.

WHAT I BRING, AND WHAT I DO NOT

Bring: carrier design and optimisation; full physicochemical characterisation by DLS, zeta potential, TEM, SEM, HPLC and UV–Vis; the molecular and cellular biology that establishes a mechanism, including macrophage culture and polarisation; and the complete in vivo programme run independently, from ethics approval through biodistribution to histopathology.

Do not: I have not formulated RNA, proteins, peptides or antigens. Your laboratory's biologics platforms would be new to me, and I would be learning them.

FUNDING AND TIMING

I am **not yet eligible for the "la Caixa" Junior Leader Incoming call**, which requires a PhD obtained two to seven years before the 23 September 2026 deadline; mine was awarded in 2025. I would be eligible for the following round. I am eligible now for an **MSCA Postdoctoral Fellowship** (2026 call closes 9 September) and for **Juan de la Cierva – Formación**, and I would write either proposal myself given a host.