Research Statement

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RESEARCH FOCUS

My research asks how changes in gene and protein expression translate into a change in what a diseased tissue actually does — and how to prove that link rigorously rather than correlatively. I have pursued that question in cancer, in a setting where the answer had to survive both peer review and an animal model.

DOCTORAL WORK AND PRINCIPAL FINDING

At the Shanghai Institute of Materia Medica, Chinese Academy of Sciences (2017–2025), I investigated why combining two mechanistically distinct agents suppresses metastatic triple-negative breast cancer when neither does so alone. Shikonin inhibits PKM2 and suppresses tumour glycolysis; JQ1 is a BET bromodomain inhibitor acting on transcriptional programmes. Delivered together from a single mesoporous polydopamine carrier, they suppressed both primary tumour growth and lung metastasis.

The mechanism is the part I consider my contribution. Using Western blot, RT-qPCR, ELISA, flow cytometry, immunofluorescence and immunohistochemistry, I established that the effect ran through three independent routes acting simultaneously: induction of **apoptosis**; blockade of **epithelial–mesenchymal transition**, the transcriptional programme by which carcinoma cells acquire the motility to leave a primary site; and blockade of **vasculogenic mimicry**, in which tumour cells form their own perfusion channels and thereby evade therapies directed at endothelial angiogenesis. Closing all three is what produced the anti-metastatic result. Published in *Acta Pharmacologica Sinica* 46(12):3314-3326, **joint first author, equal contribution formally recorded**.

I designed and executed the accompanying in vivo programme independently — protocol and endpoint definition, institutional animal ethics approval, dosing and route selection, longitudinal monitoring, biodistribution, terminal tissue collection, histopathology and organ-toxicity assessment.

PROPOSED RESEARCH PROGRAMME

1. Vasculogenic mimicry as a tractable target. It remains under-studied relative to angiogenesis, and it explains part of why anti-angiogenic therapy underperforms in aggressive tumours. I would characterise the transcriptional and signalling requirements for channel formation in patient-relevant models, and test whether they can be interrupted pharmacologically without the toxicity that limits current agents.

2. Metabolic–epigenetic coupling. My two agents act on glycolysis and on transcription respectively, and the combination was more than additive. Why is an open question. Establishing whether glycolytic inhibition alters chromatin accessibility, and therefore sensitises cells to BET inhibition, would be a genuinely mechanistic programme and a fundable one.

3. Local and sustained intervention after surgery. The post-resection window is where residual disease seeds recurrence and where systemic dosing is least suited. My thermosensitive hydrogel work begins this; the biology of that window deserves proper study rather than an assumption.

TEACHING AND MENTORING

I hold D.Pharm, B.Pharm and M.Pharm qualifications alongside the doctorate and roughly three years of hospital pharmacy practice, so I can teach biochemistry and molecular biology with clinical illustrations drawn from my own experience rather than from a textbook. I mentored Master's and undergraduate researchers throughout my doctorate in experimental design, cell-based assay technique and data analysis. I have not yet delivered formal lecture courses and would welcome structured support in a first year.

POSITION AND RECORD

Seven peer-reviewed publications, 324 citations, h-index 3 (Google Scholar, July 2026). One filed Indian patent (No. 202421042057). Funded throughout the doctorate by the ANSO Young Talent and Belt and Road Government Fellowships. I should note plainly that **protein purification is not among my techniques** and that I have approximately one year of post-doctoral experience against a stated preference for five.