INTERCEPT

Karim Brohi, December 2025

"What if we could give first responders a rapid-dose treatment that extends the window of human resilience when organs lose their blood supply."

Summary

INTERCEPT will transform outcomes for conditions including trauma, post-partum bleeding, heart attack and stroke, by extending the prehospital survival window with field interventions that intercept the early self-amplifying cascades of cell injury. Modern medicine is very good at stopping bleeding and reopening blocked arteries, but these interventions are time-critical. Minutes decide between life and death, recovery and disability. There is currently nothing deliverable in the home, on the street, or on the battlefield that can extend those minutes into hours. INTERCEPT will create a human-anchored translation engine using organ-on-chip and digital twin models, to develop new therapies that can be tested through emerging pre-hospital trial networks. Novel treatments will save lives, preserve organ function, and lessen the long-term burden of disability on families, health systems, and economies. INTERCEPT will birth a new age of survival therapeutics with endotype-targeted treatments, companion diagnostics and AI decision support tools that emergency teams, combat medics and even bystanders can deploy within minutes**.**

What we are trying to do.

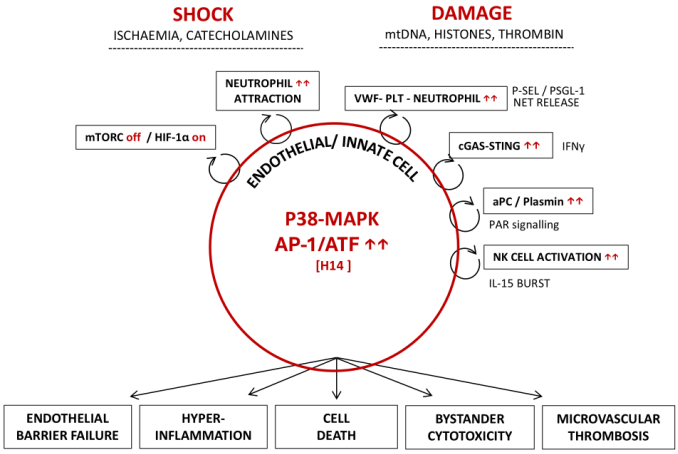
INTERCEPT aims to develop rapid, safe treatments that interrupt the early damage cascades triggered by inadequate perfusion, thereby prolonging cell and organ tolerance until bleeding control or reperfusion is achieved. These cascades are the final common pathway to avoidable death and disability across major trauma haemorrhage, myocardial infarction and ischaemic stroke. INTERCEPT creates a new category of medicine - survival therapeutics - that extend the window of human resilience when blood supply is cut off. It would transform trauma care, cardiac care, stroke care, combat casualty care and disaster response.

Why this is needed.

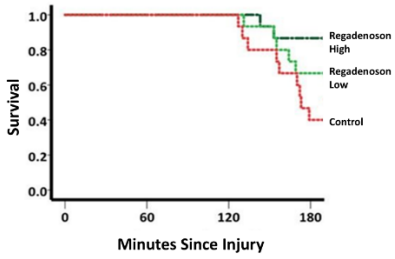
Today we focus on fixing the cause (stop bleeding, unblock vessels, restore flow). We have become extraordinarily effective, but time to definitive care remains the limiting factor. Even when sufficient blood flow is restored, many patients die or suffer organ failure because hearts, livers, kidneys and brains cannot tolerate the period of low-flow that has passed. Urban/rural delays, underdeveloped healthcare systems and modern conflict zones widen this gap. We lack field deployable minutes-to-dose targeted therapeutics that extend biological resilience.

What's new in INTERCEPT

INTERCEPT focuses on the injury cascades that result in endothelial damage, immune overreaction, microscopic clotting and dysregulated cell death that begins the moment blood supply is cut off. Previous attempts in this space have focused on attempts to reduce the amount of oxygen that cells need to survive, or to reduce post-reperfusion injury. These spaces have proved too constrained by human biology, delivery context, and fundamentally by incomplete pathway knowledge. New research instead suggests that there are multiple innate pathways activated the moment blood supply is reduced that result in endothelial damage, immune overreaction, microscopic clotting and dysregulated cell death. Even modest upstream control of these can yield outsized survival benefits when applied early to these cascades.



Proof-of-possible: In preclinical trauma-haemorrhage work, an upstream stress-signalling modulator (regadenoson) has reduced 3-hour mortality and maintained cardiac function consistent with extending the survival window (QMUL C4TS, unpublished).



Only recently have multi-omic tools, vascularised human organ-on-chip, ex-vivo perfused organ systems, and digital twins matured enough to be calibrated to human samples. In parallel, prehospital clinical platforms can now support mechanism linked, minutes-to-dose studies. ***INTERCEPT will orchestrate these into a human-anchored discovery engine for survival science.***

Who cares and what difference will it make.

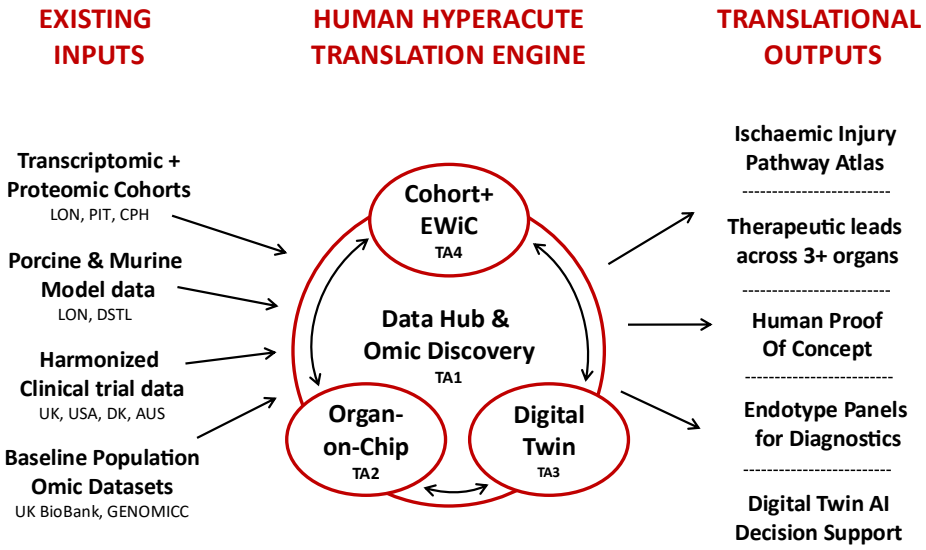
This matters to those whose lives hinge on minutes they do not have: a trauma patient on the street, a soldier far from surgical care, a parent with a heart attack at home, a stroke patient awaiting transfer. Worldwide, cardiovascular disease kills around 20 million people a year; injuries and violence kill a further 4.4 million.

Extending the survival window by just four hours could prevent over 100,000 deaths each year across the UK and US and avert a further 120,000 cases of permanent disability. Globally, this equates to millions of QALYs gained annually, The benefit is greater the farther the patient is from a hospital, thus massively improving equitable access to healthcare worldwide.

PROGRAMME OVERVIEW

A five-year programme to build a validated human discovery engine and deliver first-in-human proof-of-concept. Major trauma haemorrhage is the proving ground because it offers multi-organ readouts and established clinical platforms for rapid translation. The engine links:

(TA1) A human ischaemic injury pathway atlas from omics profiling with confirmed mechanistic underpinnings; **(TA2)** vascularised organ-on-chip models (skeletal muscle, myocardium, liver; universal media; animal-free matrices); **(TA3)** Digital-twin models calibrated to patient data; and **(TA4)** An Experiment-within-Cohort (EWiC) clinical platform that connects prehospital dosing to in-hospital biomarker/organ-injury effects.



Timeline and Milestones

Years 1–3 (Map & Model): Integrate existing omics with novel human-platform derived samples and underpinning mechanistic validation. Stand-up first-generation OoC and digital-twin platforms. Deliver the trauma haemorrhage response atlas with mechanistic response endotypes and candidate biomarker panels; identify ≥5 conserved injury nodes across ≥3 organs; down-select to 3 priority mechanism families with cross-organ evidence.

Year 4 (Human proof of concept, novel therapeutic leads): high-throughput, human-anchored screening to generate and triage modulators; advance one repurposed/low-risk asset into a prehospital minutes-to-dose mechanistic (Phase IIa equivalent) study in trauma; Validate and deliver companion diagnostic-ready biomarker panels.

Year 5 (Readiness & ecosystem): deliver the validated discovery engine (atlas, calibrated models, biomarker panels, SOPs, operational playbooks), 5+ qualified leads across mechanism families, and companion diagnostic dossiers with field-feasibility; formation of 2+ spinouts and UK prehospital trial playbooks; and a repurposing playbook enabling external performers to tune the engine to other organs/disease states (heart attack, stroke) under shared standards.

Scale-up and Off-ramps (ecosystem opportunities)

Enable a £100M+ Phase II to drive multiple therapeutic candidates through IND and first-in-human in trauma (with extension potential to heart attack and stroke via shared mechanisms), while partnered diagnostics (built from identified trauma-shock endotypes and panels) and decision-support modules progress on off-ramps via spinouts, industry and Catapult/CPI partners. Stand up a UK Survival Consortium with shared standards, data and manufacturing, and provide a repurposing playbook so external performers can tune the discovery engine to other organs/disease states under the same framework. Pre-build within INTERCEPT the buyer path transition lane to NHS/Defence/international buyers.

Programme Cost

The five-year programme is budgeted at ~£40 million. This level of investment is sufficient to deliver, at speed, the core discovery engine: a validated injury-pathway atlas, first-generation organ-on-chip and digital twin platforms across multiple organ systems, and operational playbooks, while testing a repurposed therapeutic into a Phase IIa mechanistic trial. Roughly £20m supports the development and testing of the discovery engine (Years 1-3), the remainder funds therapeutic discovery, translational testing, and field-readiness feasibility.

Risks and Mitigation

Scientific: No conserved or druggable nodes emerge. *Mitigation:* build the atlas across multiple organs and validate in human models before committing to leads. *Go/No-Go:* by Year 2, ≥5 conserved nodes identified across ≥3 tissues; if not, narrow to organ-specific development.

Technical: Models and formulations fail to translate. *Mitigation:* calibrate OoC/digital twins prospectively against patient samples; *Go/No-Go:* by Year 3, OoC/digital twins must predict human biomarkers within predefined thresholds.

Clinical: Repurposed therapeutic shows no effect in patients. *Mitigation:* Prioritise mechanistically justified with dominant effect in OoC. *Go/No-Go*: by end Year 4, trial demonstrates improvement;;else shift emphasis to other leads, plan for new Phase IIa in Year 5.

Regulatory: Agreement on appropriate endpoints & consent process for field-delivery trials. *Mitigation:* co-design a surrogate endpoint set with MHRA/FDA and ethics from Y1; legal emergency consent frameworks. *Go/No-Go*: written concurrence with regulators (end Y3).

Organisational: Fragmentation across creators. *Mitigation:*Discovery engine driven by data commons, quarterly portfolio reviews, and independent oversight. *Go/No-Go:* all performers on data commons within 6 months.