

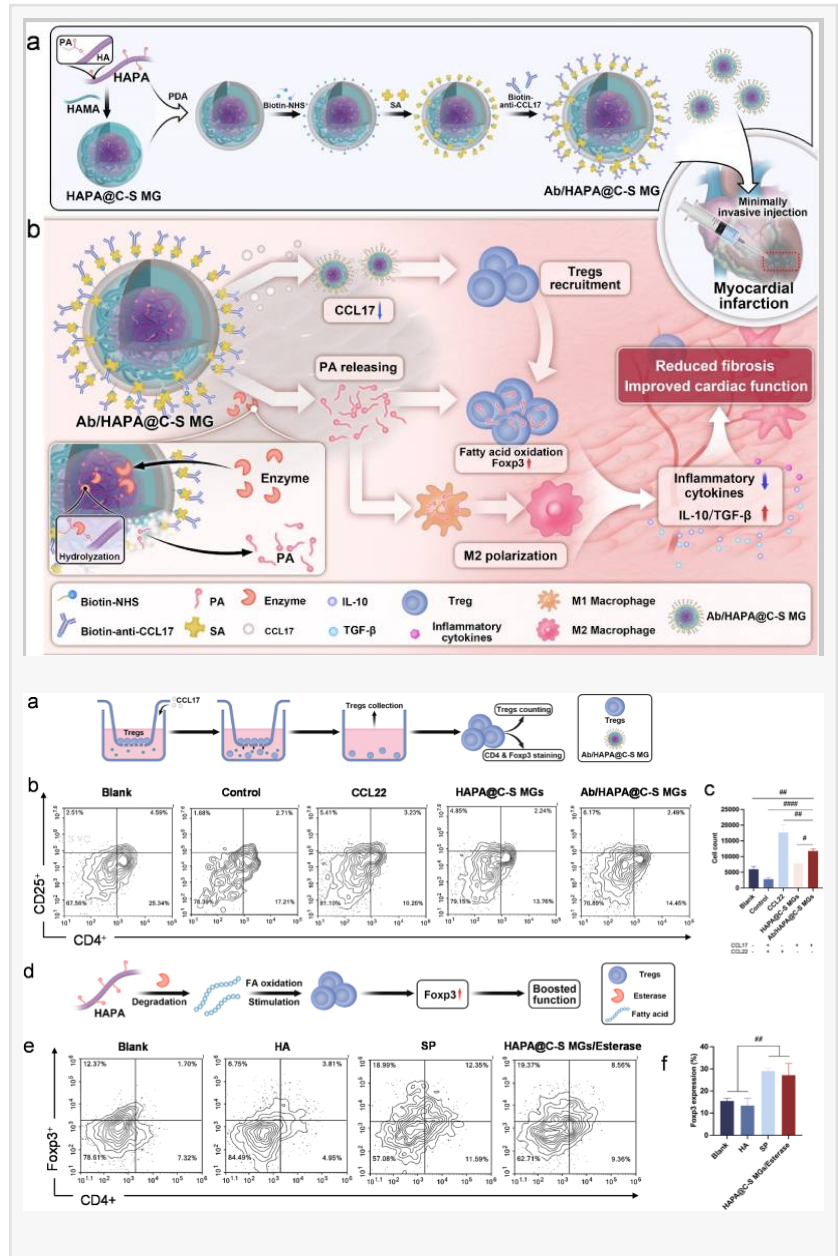
Microgel Paired with Cloud-Clone Antibody: Precisely Reset Post-Infarction Immunological Microenvironment

A core-shell microgel platform achieves sequential Treg recruitment and functional potentiation to drive post-infarction cardiac tissue repair

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EINPresswire.com/ -- Myocardial infarction triggers persistent inflammatory responses within infarcted cardiac tissue, which severely hinders endogenous tissue repair. Suppressed recruitment and functional impairment of regulatory T cells (Tregs) constitute a major barrier to restoring immune homeostasis. A research team from Zhejiang University has developed an esterase-responsive core-shell microgel system to solve this unmet challenge, where a biotin-labelled [Cloud-Clone antibody](#) serves as a critical building block of the therapeutic construct. The published work describes a synergistic strategy to recruit endogenous Tregs and boost their suppressive capacity, opening new avenues for immunomodulatory therapy following myocardial infarction.

After myocardial infarction, the infarct zone falls into a chronically inflamed state that blocks tissue-repair progression. Impaired recruitment and functional exhaustion of regulatory T cells (Tregs) are core obstacles preventing immune-homeostasis restoration. Therefore, synchronizing targeted Treg homing and enhancement of their suppressive function represents a key



unresolved scientific question for post-myocardial-infarction immune-repair strategies. The Zhejiang University research team delivers a promising solution in their publication titled CCL17-neutralizing and esterase-responsive core-shell microgels for endogenous Tregs recruitment and functional enhancement in myocardial infarction.

Figure 1. Design of Ab/HAPA@C-S MGs: Synergistic shell-mediated CCL17 neutralization and core-driven PA release to restore immune homeostasis post-myocardial infarction.

The Dilemma: Tregs “Lost” and Functionally Compromised Under Inflammatory Storm
Regulatory T cells are central mediators of immune homeostasis. Following myocardial infarction, Tregs undergo dual-layer functional suppression. On one hand, overexpressed CCL17 within infarcted tissue disturbs directional Treg migration toward injury sites. On the other hand, the inflamed microenvironment blunts Treg suppressive activity. Even when Tregs successfully arrive at target lesions, they cannot exert sufficient reparative effects. Coexisting impaired recruitment and defective function drastically weaken the immunomodulatory performance of endogenous Tregs. The research group concludes that restoring immune homeostasis requires resolving both constraints simultaneously, calling for an intervention strategy combining targeted delivery and sustained functional augmentation.

Breakthrough: Dual-action Core-Shell Microgel Design

The rationally engineered core-shell microgel system Ab/HAPA@C-S MGs enables dual-mode immunomodulation. Its well-organized structure features compartmentalized, coordinated functions for shell and core compartments.

The outer shell removes chemokine barriers: Anti-CCL17 neutralizing antibodies are covalently conjugated onto microgel surfaces, acting as a “neutralization mesh” within inflamed tissue to sequester excess CCL17 and remove barriers for Treg homing. In vitro Transwell assays confirm that surface-tethered anti-CCL17 antibodies effectively neutralize surplus CCL17, re-establish chemokine gradients and markedly improve Treg migration.

The inner core executes payload release: Prodrug precursor HAPA is encapsulated inside microgels. Catalyzed by high-level esterase (CES1) present in infarcted tissue, HAPA undergoes gradual hydrolysis and liberates propionic acid (PA). Via fatty-acid oxidation pathways, PA strengthens Treg immunosuppressive activity, upregulates the key transcription factor Foxp3, and promotes macrophage polarization toward the reparative M2 phenotype.

Shell-based neutralization and core-based payload release operate in tandem, with release kinetics well-aligned with Treg-recruitment recovery, enabling spatiotemporally specific modulation of the post-infarction microenvironment.

In-vitro Validation: Demonstrating the Power of Synergistic Effects

The research team conjugated biotin-labelled anti-CCL17 antibody from Cloud-Clone (Catalog No. LAB571Mu71) onto microgel surfaces. ELISA results verify that anti-CCL17 antibodies on Ab/HAPA@C-S MGs efficiently neutralize CCL17 in culture medium. Transwell migration assays reveal that simulated inflammatory conditions with co-existing CCL17 and CCL22 strongly inhibit Treg migration. Treatment with Ab/HAPA@C-S MGs reverses this inhibitory phenotype. Surface-bound anti-CCL17 antibodies deplete excessive CCL17 and restore Treg migration rates close to

positive-control levels.

Under esterase-mimicking conditions, microgels sustain continuous PA release. Tregs treated with the system exhibit significantly elevated Foxp3 expression and robust IL-10 secretion. Macrophage conversion to the M2 reparative phenotype is also greatly increased. Collectively, these cellular-level data confirm the dual capability of this platform for Treg recruitment and functional enhancement.

Figure 2. Dual regulation of Treg recruitment and function achieved by Ab/HAPA@C-S MGs via CCL17 neutralization and PA release.

In-vivo Therapeutic Efficacy: From Immune Remodelling to Cardiac Functional Recovery

Promising in-vitro performance of Ab/HAPA@C-S MGs is further validated in mouse myocardial-infarction models, showing encouraging therapeutic outcomes. Ab/HAPA@C-S MGs exert early immunomodulatory effects after myocardial infarction through a two-phase “recruitment-then-enhancement” mechanism. During the recruitment phase, microgel-conjugated anti-CCL17 neutralizing antibodies eliminate excessive inflammatory chemokine signals to facilitate Treg infiltration and build local immunosuppressive signalling. In the subsequent enhancement phase, esterase-triggered HAPA degradation releases PA to amplify Treg suppressive functions. Transcriptomic analysis further demonstrates that this therapy remodels metabolic networks of infarct-infiltrating immune cells and shifts global immune responses from pro-inflammatory toward pro-reparative profiles.

This coordinated immunomodulatory cascade accelerates inflammation resolution and re-establishes immune homeostasis, generating a favourable microenvironment for myocardial repair and functional restoration. In-vivo experimental evidence proves that the sequential “recruitment-followed-by-potential” strategy of Ab/HAPA@C-S MGs successfully redirects pathological hyper-inflammation toward regenerative repair.

Reliable Research Tools Underpin Innovative Immunotherapy Research

Scientific exploration moves forward with consistent, high-quality reagent support. This Zhejiang University study not only presents an elegantly designed microgel delivery platform, but also establishes a valuable paradigm for spatiotemporally coordinated, functionally synergistic immune modulation. Pathological signal clearance by neutralizing antibodies and small-molecule metabolite release from prodrugs work in precise spatial-temporal coordination to fine-tune complex immune networks.

Rigorous antibody selection underpins the reliability of key experimental datasets in this work. The Cloud-Clone anti-CCL17 antibody (LAB571Mu71) serves as a critical research tool for verifying the therapeutic potential of CCL17 neutralization. When researchers dissect complex disease microenvironments, the quality of fundamental reagents directly impacts research depth and conclusion credibility.

Backed by ISO 9001 and ISO 13485 dual-quality-system certifications, Cloud-Clone implements a three-level closed-loop quality-control framework covering raw materials, semi-finished products and final finished goods. The biotin-conjugated anti-CCL17 antibody (LAB571Mu71) adopted in this study is a multi-dimensionally validated reagent, delivering solid technical

support for investigators exploring sophisticated disease models.

Translational progress for cardiac immunotherapy relies on both innovative material-system design and dependable biological reagents. This study showcases how carefully selected Cloud-Clone antibodies can be integrated into sophisticated biomaterial constructs to unlock novel therapeutic insights for myocardial infarction. As global researchers continue to tackle intricate immune-related disease challenges, rigorously validated antibody reagents will remain indispensable for turning innovative hypotheses into publishable, reproducible scientific discoveries.

About Cloud-Clone Corp.

Cloud-Clone Corp. is dedicated to the development and production of high-quality immunoassay reagents and detection solutions. With a focus on antibody engineering, multiplex assay development, and cross-platform compatibility, the company provides research tools designed to support precision medicine and advanced biomedical investigation globally. Our core products and services include the research and development of proteins, antibodies, ELISA kits, primary cells, and multiplex cytokine assay kits, as well as professional CRO services to fully meet the diverse needs of biomedical research and related fields.

For more information about Cloud-Clone Corp, visit www.cloud-clone.com.

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