

An injured heart remains scarred and deformed (right) when it is unable to recruit a clean-up crew of inflammatory monocytes.

Monocyte diversity heals hearts

It takes two to heal a wounded heart. Nahrendorf et al. (page 3037) now find that two sets of monocytes are involved in the healing: one type mops up injury debris and the other then helps repair the damage.

Circulating monocytes recruited to an injured heart break down dead cells and scoop up their toxic products. This inflammation is followed by reconstruction, when monocyte cytokines promote blood vessel growth and recruit healing fibroblasts.

Previous studies suggested that a single monocyte population accomplishes both these functions by switching from clean-up mode to repair mode. But the recent identification of two monocyte subsets in the circulation, only one of which is inflammatory, implies otherwise. Each subset has its own unique receptors that respond to different chemokine signals.

Nahrendorf et al. now find that damaged hearts in mice recruit the two subsets in turn by secreting only one chemokine at a time. The first chemokine drew in inflammatory monocytes, which accumulated in the injured heart for three days and then disappeared. The pro-repair subset arrived later to help rebuild the heart.

Mice suffering from atherosclerosis—the main cause of human heart failure—have many inflammatory monocytes. Their injured hearts thus remained scarred and deformed. It is possible that a similar imbalance in humans predisposes these individuals to heart failure. [JEM](#)

recruitment and rapidly reduced the bacterial load. Normally, immunity-promoting cytokines are turned on by the NF- κ B pathway when Toll-like receptors recognize bacteria. But vasopressin suppressed their production in collecting duct cells by increasing the levels of phosphatases that inhibit NF- κ B activation.

These findings may help explain why drinking more water speeds up recovery from UTIs and kidney infections. The resulting drop in vasopressin levels probably allows a strong antibacterial immune response to be unleashed. But why this antidiuretic hormone has evolved an antiinflammatory function is still a mystery. [JEM](#)

Improved mucosal vaccines

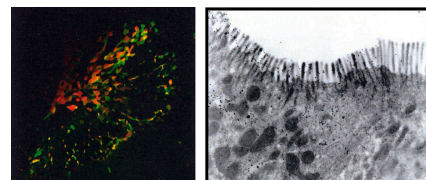
Pathogens that invade the mucosa are better repelled if the vaccines against them are delivered to the right target cells. Nochi et al. (page 2789) now identify an antibody that delivers vaccines to super-absorbent cells lining mucosal surfaces.

In mucosal tissues such as the airways and digestive tract, immune responses are only weakly stimulated by injected vaccines, which are taken up by circulating cells that boost systemic immunity. Local immunity is more effectively generated by ingested or inhaled vaccines that can be taken up directly by lymphoid tissues in the gut and airway. These tissues are lined by microvilli-bearing M cells that soak up the vaccines and pass them along to the immunity-inducing dendritic cells and macrophages lurking in the tissue below.

In past attempts to get vaccines to M cells, vaccine antigens were hitched to lectins that bind to carbohydrates on the M cells. But since these carbohydrates are also present on nonabsorbent mucosal cells, the antigen becomes diluted.

Nochi et al. now identify a mouse antibody that gets around this problem. The new antibody recognizes part of a carbohydrate that is found only on M cells. Mice fed this antibody coupled to a bacterial toxin remained healthy even when later given a 10^4 higher dose of the toxin than would normally cause disease.

Whether the human antibody will be just as effective remains to be seen. [JEM](#)



Vaccine antigens coupled to an antibody (green) that binds to carbohydrates on M cells (right) induce a stronger immune response than when coupled to lectins (red).

Rehydrating hormone smothers inflammation

Avoiding dehydration comes with a price, according to Chassin et al. (page 2837). The body's efforts to stay hydrated reduce its ability to fight kidney-invading bacteria.

The body responds to dehydration by producing the hormone vasopressin, which instructs the kidneys to absorb more water. Vasopressin tells cells that line the collecting ducts linking the kidneys to the urinary tract to make more membrane water channels and thus absorb more water. These collecting duct cells are also the targets of *Escherichia coli* and other intestinal bacteria, which can then enter the kidney and trigger inflammation.

The resulting kidney and urinary tract infections (UTIs) are particularly common in dehydrated individuals. Chassin et al. now find that high levels of vasopressin may be to blame.

Mice that were given vasopressin and later inoculated with bacteria in the urinary tract failed to clear the pathogens. Treatment with a vasopressin antagonist stimulated inflammatory cytokine production and neutrophil