NEWS & VIEWS

I. BERRY/MAGNUM PHOTOS

Arsenic alert. The red paint on this well-head in Jhikargachha, Bangladesh, is a warning that the water is impure. But there is often no choice of water source.

ENVIRONMENTAL SCIENCE

Poisoned waters traced to source

Charles F. Harvey

South Asia's well-water is widely polluted with arsenic, but no one has located the source. A study on the Mekong River finds that contamination begins in pond sediments, and is spread by groundwater flow to wells.

Millions of people living in the Ganges delta of Bangladesh and West Bengal drink groundwater contaminated by arsenic. Many more ingest arsenic-contaminated water drawn from wells along the Mekong and Red rivers of Cambodia and Vietnam, and probably also along the Irrawaddy River in Myanmar. Arsenic dissolves in groundwater from naturally deposited sediments, but its localization within sedimentary aquifers is puzzling — wells from which severely contaminated water is drawn can be found a mere ten metres away from safe wells. On page 505 of this issue, Polizzotto *et al.*[1] describe the first study that traces the contamination from its source. The authors show that, at a site near the Mekong River, arsenic-laden groundwater originates from ponds. This water flows horizontally through the aquifer, beneath groundwater that percolates through soils, to contaminate downstream wells.

To understand how contaminated water may be traced back to its source, one must consider the physics of solute transport by groundwater flowing through aquifers. Groundwater flow-paths through typical undeveloped aquifers are arranged in a largely horizontal pattern (Fig. 1, overleaf), in which successively deeper layers originate from sources farther away[2]. Little mixing occurs across these flow-paths[3], so solute concentrations within an aquifer are also layered, with each deeper layer representing the biogeochemical outcome of water inputs from more distant sources.

By applying an understanding of these physical processes, Polizzotto *et al.*[1] arrived at two crucial insights. First, they realized that if they selected a location where groundwater flow is primarily perpendicular to the Mekong River, a single transect of wells of different depths aligned with that flow would sample water from the various layers of the subsurface system, fully mapping the local three-dimensional system. Second, they recognized that the chemical composition of groundwater at different depths reflects the different origins of the water — thus avoiding the common mistake of assuming that the composition is purely determined by a gradual process of change as solutes react with aquifer sediments over time. Although it is true that deeper groundwater is usually older, the water found at shallow depths does not flow straight down to deeper levels. By analysing the arsenic content of samples from different depths and locations at their site, Polizzotto *et al.* conclude that the local arsenic contamination originates from nearby pond sediments. So how can this source be explained?

The arsenic originally comes from eroded Himalayan sediments that have washed down into low-lying regions. It is widely believed that this arsenic dissolves and enters the groundwater under anaerobic conditions. It is therefore unsurprising to find that highly contaminated groundwater originates from pond sediments: the steady settling and decomposition of organic material at the bottom of tropical ponds takes up all the oxygen that diffuses, or is carried by downward flow, into the sediment.

Water passing through pond sediments could

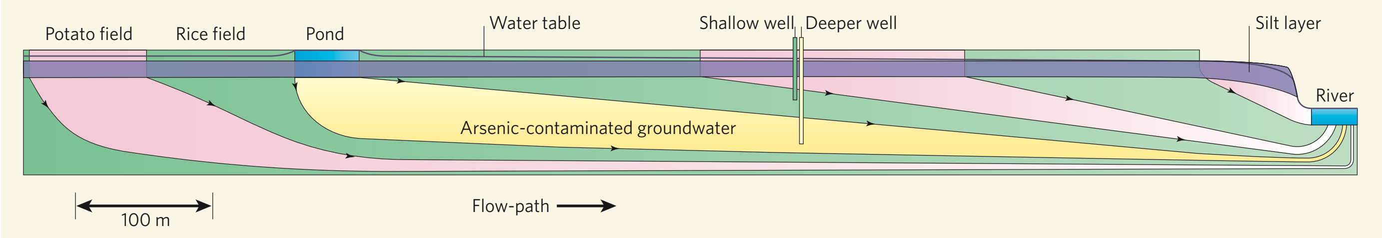

Figure 1 | Arsenic contamination in south Asian aquifers. Polizzotto *et al.*[1] report that arsenic contamination of groundwater at a site on the Mekong delta can be traced to ponds that supply the aquifer with water. The diagram shows groundwater flow through the cross-section of a system similar to that studied by the authors. Groundwater from different sources (potato fields, rice fields and a pond) flows in layers towards the river. A shallow well 'downstream' from the pond accesses water that started out from a rice field. A deeper well draws contaminated water that originated from the pond.

also contain organic carbon that, on decomposition, might help liberate arsenic from deeper sediments, adding to the contamination. But any organic carbon that is already contained in deeper aquifer sediments probably contributes less to biogeochemical processes because it is not replenished, and what remains is typically of low reactivity. Polizzotto and colleagues used carbon dating to show that the inorganic carbon dissolved in contaminated water at their site is young, as would be expected if it comes from pond sediments.

The authors' results raise the question of whether similar processes are responsible for the arsenic contamination observed in other aquifers throughout south Asia. If so, wells could be placed so as to avoid drawing groundwater that originates from ponds or similar bodies. Answering this question requires fieldwork at other sites, but several observations support the idea that the proposed mechanism[1] for arsenic contamination occurs elsewhere in south Asia. For example, natural and man-made ponds are ubiquitous in the region. Because the pond sediments are always saturated with water, they are more likely to be anoxic than soils — which, even under rice cultivation, are exposed to air several times a year.

Another clue is that arsenic concentrations in groundwater often increase with depth. This might also be indicative of surface-water inputs. Wells are never installed in ponds, so well-water drawn from the top of the aquifer will have passed only through the surrounding soil, rather than through anoxic sediments that would cause arsenic contamination. Deeper wells access groundwater that might have originated from ponds or similar bodies, and would therefore have been contaminated by arsenic.

The challenge in the Ganges delta, where the largest population of people is exposed to arsenic contamination, is that groundwater flow is exceedingly complex, depending on both geographical and temporal factors. Humans complicate the picture further — irrigation pumping drives groundwater in three-dimensional patterns that shift with the seasonal monsoon cycle. Furthermore, the widespread excavation of ponds and the explosive growth of irrigation pumping are changing subsurface solute concentrations over the course of decades[4].

In the United States and Europe, groundwater contamination sites are often first studied by characterizing groundwater flow. But in south Asia, research has focused on the biogeochemistry of arsenic transfer from aquifer substrate materials. This difference is understandable, because the source of dissolved arsenic in south Asia is naturally occurring sediments, rather than contaminant spills as in the United States and Europe. But Polizzotto and colleagues' work[1] clearly demonstrates that groundwater flow can control the localization of arsenic that originates from sediments. Understanding groundwater movement at more complex sites than the Mekong will require in-depth characterization of physical hydrogeology, such as that routinely used for small contamination sites in the United States and Europe. Although costly, this would surely be a small price to pay for the benefit of securing safe, clean drinking water for millions. ■

Charles F. Harvey is in the Department of Civil and Environmental Engineering, Massachusetts Institute of Technology, 77 Massachusetts Avenue, Cambridge, Massachusetts 02139, USA.
e-mail: charvey@mit.edu

1. Polizzotto, M. L., Kocar, B. D., Benner, S. G., Sampson, M. & Fendorf, S. *Nature* **454,** 505–508 (2008).
2. Freeze, R. A. & Cherry, J. A. *Groundwater* (Prentice Hall, 1979).
3. Gelhar, L. W. *Stochastic Subsurface Hydrology* (Prentice Hall, 1993).
4. Harvey, C. F. *et al. Chem. Geol.* **228,** 112–136 (2006).

PHYSIOLOGY

Myoglobin's new clothes

Andrew Cossins and Michael Berenbrink

Nitric oxide generated from the nitrite ion limits the tissue damage caused by restricted blood flow. Gene knockout experiments in mice now reveal that myoglobin is the mediator of this effect.

All students of biology encounter the richly pigmented protein myoglobin early in their education, where it provides the first and most famous example of a revealed protein structure combined with a straightforward physiological role. Its restricted distribution to endurance muscle and heart cells throughout the vertebrates, and its notable expression in diving mammals, are a reflection of its widely accepted function in cellular oxygen transport and oxygen buffering. That function is to support high levels of aerobic muscular activity, and to deal out stored oxygen during extended periods of hypoxic — low oxygen — physiological conditions.

Given that all mammals have large quantities of myoglobin, experimental deletion of the genes concerned should have severe effects. Imagine, then, the surprise when the first myoglobin-knockout mouse[1] seemed to be perfectly normal, with no obvious ill effects during exercise and hypoxia. Unease about the accepted dogma has grown into debate about alternative functions for myoglobin[2–5], and has culminated in work by Hendgen-Cotta *et al.*, just published in *Proceedings of the National Academy of Sciences*[6]. Their paper shows that myoglobin knockout in mice has consequences that are both surprising and medically important.

This story has its origins in the apparently ordinary inorganic anion, nitrite (NO_2^-). Sodium nitrite is an ancient means of curing meat, but it is also used to maintain the desirable cherry-red colour of meat while it is on supermarket shelves. It has been used medicinally in high concentrations as a vaso- and bronchodilator (that is, for dilating blood vessels and airways), and as a treatment for cyanide poisoning, and the organic derivative amyl nitrite is used recreationally as a psychoactive drug. Environmental nitrite has been identified as an aquatic pollutant[7], and nitrite derived from contaminated drinking water is the cause of blue-baby syndrome[8]. This is an ailment in which human infants suffer from nitrite-induced formation of methaemoglobin,