

# Degassing of Lake Nyos

**SIR** — Lakes with CO<sub>2</sub>-rich waters are distinguished by their extraordinary chemical evolution, their lethal nature and their uniqueness among natural hazards in potential for mitigation. Three possible endpoints of chemical evolution exist for these systems: equilibrium, where gas input is balanced by gas loss; gas bursts, where recharge surpasses loss; and controlled degassing, where the cycle of recharge and release is short-circuited. Here we report the use of direct measurements of CO<sub>2</sub> recharge rates and introduce a new parameter of lake stability to evaluate the likelihood of equilibrium or of further gas bursts, and to analyse the potential of controlled degassing.

The changing inventory of CO<sub>2</sub> in these lakes is the difference between inputs of dissolved gas issuing from bottom or side vents<sup>1-4</sup>, and losses including leakage, surface flushing and ventilation to the atmosphere. Since the 1986 gas burst in Lake Nyos, Cameroon, a total of  $19 \times 10^8$  mol CO<sub>2</sub> have been lost through flushing and ventilation as the surface mixing layer deepened to 50 m, whereas gas inputs resulted in an accumulation of  $11 \times 10^8$  mol CO<sub>2</sub> below 50 m ( $2 \times 10^8$  mol yr<sup>-1</sup>;

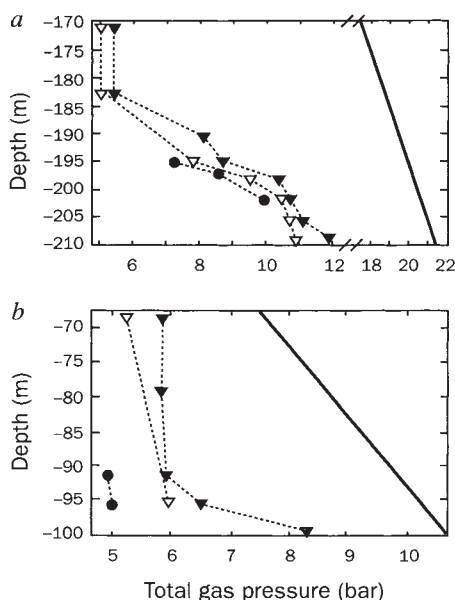


FIG. 1 Total gas pressure measured *in situ* by probe in Lakes Nyos (a) and Monoun (b) (total probe error,  $\pm 0.15$  bar<sup>2</sup>).  $P_{\text{gas}}$  at 171 and 183 m in Nyos was calculated as the sum of  $P_{\text{CO}_2}$ ,  $P_{\text{CH}_4}$  and  $P_{\text{N}_2}$  (99% of total  $P_{\text{gas}}$ ) measured in samples from pressurized cylinders. Average changes in  $P_{\text{gas}}$  were calculated for each depth and between each date, and then compared to the 1992 gas pressures to derive the time required for  $P_{\text{gas}}$  to reach  $P_{\text{amb}}$  (solid line). All rates and times were then statistically averaged; there was no statistically significant difference in buildup rate in 1989–90 compared to 1990–92. Filled circles, Dec 1989; open triangles, Sep 1990; filled triangles, Mar 92.

table). It is unlikely that losses from surface waters will continue to be a significant counterbalance for gas recharge. The surface layer is now chemically similar to the surface layer before the event<sup>2</sup>, and data on controls of mixing depth in tropical lakes<sup>5</sup> suggest that strong density gradients at 50 m in Nyos will inhibit further substantial deepening. Diffusional loss of CO<sub>2</sub> from below 50 m into the surface layer will continue; the current rate is about  $0.17 \times 10^8$  mol yr<sup>-1</sup>, or 8% of recharge rate.

In Lake Monoun, Cameroon, the surface mixing layer is shallow (5 m), and has changed little since 1987. Consequently, gas inputs were weakly compensated by surface losses, and the total CO<sub>2</sub> inventory increased from  $5.41 \times 10^8$  to  $6.24 \times 10^8$  mol ( $0.17 \times 10^8$  mol yr<sup>-1</sup>). Direct measurements of gas recharge in both lakes confirm the hypothesis of slow accumulation and gas storage in bottom waters<sup>1,6</sup> rather than rapid injection of gas by phreatic explosions<sup>7</sup>, and highlight the continuing danger of violent degassing.

Gas release is controlled by local stability and the ratio of total gas pressure ( $P_{\text{gas}}$ ) to ambient hydrostatic pressure ( $P_{\text{amb}}$ ) (Fig. 1). Dissolved CO<sub>2</sub> has a non-linear effect on stability, increasing water density and stability until the ratio  $P_{\text{gas}}/P_{\text{amb}}$  exceeds 1, at which point turbulence from exsolving gas destroys local stability, and possibly triggers a gas burst. The maximum  $P_{\text{gas}}/P_{\text{amb}}$  is 0.55 in Nyos (at 209 m) and 0.78 in Monoun (99 m) (Fig. 1). If current rates of pressure buildup continue,  $P_{\text{gas}}/P_{\text{amb}}$  will reach 1 in  $30 \pm 8$  yr in Nyos and in only  $7 \pm 2$  yr in Monoun.

Knowledge of local stability is required to model the process of gas release, to predict hazard potential, and to determine the optimal procedure for controlled degassing. Because standard measures of local stability fail to account for the effects of dissolved gas, we introduce a modified stability parameter

$$E^* = [(1/\rho)(d\rho/dZ)] \times [(P_{\text{amb}}/P_{\text{gas}}) - 1] \times (1/P_{\text{gas}})$$

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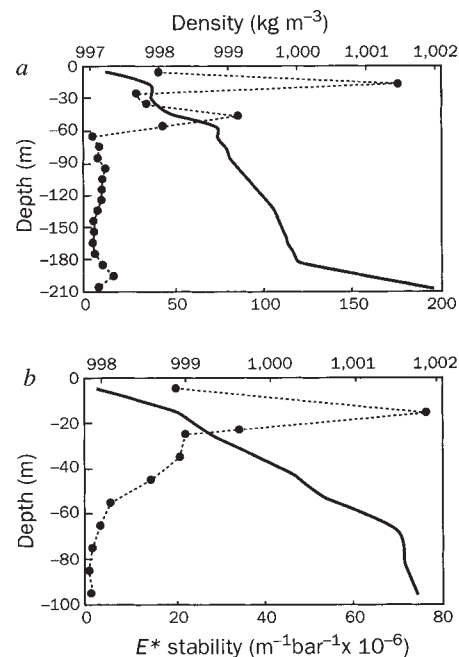


FIG. 2 Potential density ( $\rho$ ; solid line) and local water column stability including the effects of dissolved gas ( $E^*$ ; dotted line) in Lakes Nyos (a) and Monoun (b). Final potential density due to temperature, dissolved salts, and gases was calculated as in ref. 4. Bathymetric and dissolved salt data are from refs 2,4; dissolved salt data from 1992 are unpublished, but are similar to September 1990 data<sup>2</sup>. The first term of  $E^*$ ,  $(1/\rho)(d\rho/dZ)$ , is a standard measure of stability; the second term  $(P_{\text{amb}}/P_{\text{gas}}) - 1$ , describes the pressure ratio such that as  $P_{\text{gas}}$  approaches  $P_{\text{amb}}$  the stability  $E^*$  goes to zero; the final term,  $(1/P_{\text{gas}})$ , is required to distinguish situations where local saturation is reached in a layer with low absolute gas pressure, such as near the lake surface, and thus a low potential for triggering a gas burst. Negative values of  $E^*$  indicate unstable conditions due to denser water overlying less dense water (first term of  $E^*$ ) or to gas oversaturation (second term of  $E^*$ ).

where  $\rho$  is potential density of the fluid and  $Z$  is depth (Fig. 2). Accordingly, as  $P_{\text{gas}}$  rises the local stability  $E^*$  is reduced.

It has been suggested that degassing the uppermost CO<sub>2</sub>-rich layers is a priority during mitigation<sup>8</sup>; we disagree. During controlled degassing, water pumped from a specified depth and degassed at the surface may be released in the lake, or it may be removed from the basin so that lake level is lowered. In the first case,  $E^*$  increases everywhere above the pumping depth and remains constant elsewhere. In the second,  $E^*$  decreases at all points below the degassing depth and remains constant elsewhere; stability decreases because lowering lake level decreases  $P_{\text{amb}}$  while  $P_{\text{gas}}$  remains constant. Therefore, regardless of the degassing procedure, the safest and most stable conditions will be attained when pumping occurs from the deepest zones of low  $E^*$ . Zones of low  $E^*$  are also the most likely initiation points in

Depth CO <sub>2</sub> (m) (μmol kg <sup>-1</sup> )	Depth CO <sub>2</sub>
<b>LAKE MONOUN</b> Dec 1989	<b>LAKE NYOS</b> Apr 1992
23 17,900	53 55,500
46 54,300	76 71,800
69 145,000	99 92,500
95 120,000	137 130,000
79 138,000	171 144,000
95 140,000	183 155,000
Sep 1990	190 209,000
23 20,400	198 289,000
46 64,600	206 318,000
61 119,000	206 319,000
69 140,000	209 315,000
Mar 1992	
23 21,100	
69 145,000	
79 14,800	
95 150,000	

Concentrations of dissolved gas in Lakes Nyos and Monoun measured in pre-evacuated cylinders<sup>2</sup>. Recharge rates are calculated using earlier data for Nyos<sup>2,4</sup> and Monoun<sup>13</sup>.

triggering a gas burst.

Dangerous gas-rich lakes can be identified before gas bursts occur<sup>3</sup>, and alleviation of the hazards is possible through

controlled pumping in pipes<sup>1,10,11</sup>. Preliminary calculations based on field tests<sup>11</sup> and our measured inventory and recharge indicate that Monoun could be fully degassed in less than 2 yr using three pipes of 141-mm diameter. The 550,000 tonnes of CO<sub>2</sub> in Lake Nyos will require more or larger pipes and a longer period of time, and complications arise due to the geologically weak spillway of the lake<sup>12</sup>. Permanent installation of the pipes would short-circuit the natural cycle of gas recharge followed by gas burst in these lakes. Controlled degassing of the lakes is needed urgently.

**George W. Kling**  
**William C. Evans**  
**Michele L. Tuttle**  
**Greg Tanyileke**  
Department of Biology,  
University of Michigan,  
Ann Arbor,  
Michigan 48109, USA

Other addresses: US Geological Survey, Menlo Park, California 94025, USA (W.C.E.); US Geological Survey, Denver Federal Center, Denver, Colorado 80225, USA (M.L.T.); Institute for Geological and Mining Research, Mesres, Yaounde, Cameroon (G.T.).

## Migration of muscle cells

SIR — Immature muscle cells (myoblasts) can deliver gene products to mature muscle fibres following intramuscular or systemic delivery<sup>1-4</sup> and thus could have a therapeutic role in inherited muscle diseases<sup>1,5</sup>. Successful therapy would re-

quire migration of introduced cells throughout the muscle so that gene products are disseminated diffusely. Myoblast migration is controversial, many reports showing movement within muscles, and few movement between them<sup>6-10</sup>.

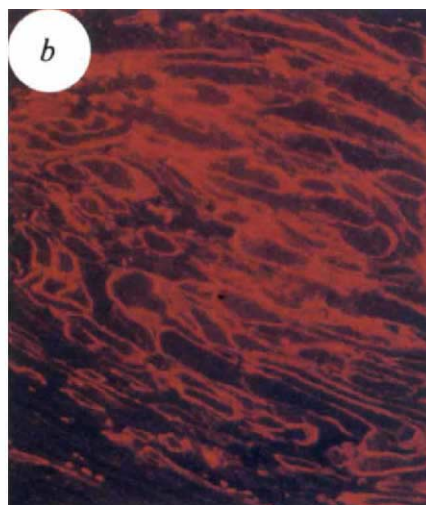
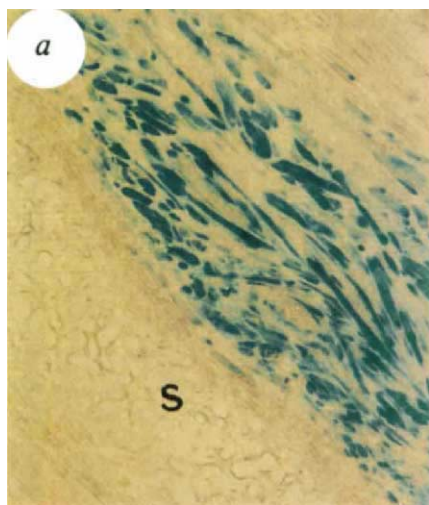


FIG. 1 Micrographs showing the EDL isograft 17 days after implantation. Most EDL fibres are positive for (a) β-gal (blue) X 180, and (b) dystrophin, X 270, S; Spongostan.

**METHODS:** Isografting was performed by removal of C2C12-implanted EDL from the *mdx nu/nu* host and transplanted into the EDL muscle bed of a second *mdx* recipient from which the autologous EDL had been removed. Using a PCR pipette flamed to a fine point, the EDL to be isografted was injected with 3 X 10<sup>5</sup> C2C12-*lacZ* cells 3–5 days before their removal from the initial host. Muscle complexes removed 15, 17 or 22 days following isograft insertion and crush injury, were frozen in isopentane maintained at -165 °C in liquid nitrogen. Cryostat sections (8 μm) were cut at three levels throughout the complex for examination by light and electron microscopy. Analysis for β-gal activity was carried out as described<sup>16</sup>, but leaving the reaction product to develop overnight. For dystrophin analysis<sup>12</sup>, sections were immunocytochemically stained using an anti-dystrophin antibody of M<sub>r</sub>, 60,000, a gift from E.P. Hoffman.

Here we demonstrate the preferential migration of implanted mononuclear myoblasts from isografts to regions of muscle injury and regeneration in adjacent muscles.

We injected mouse C2C12 myoblasts carrying the *lac Z* gene<sup>11</sup> encoding β-galactosidase (β-gal) into a mouse extensor digitorum longus muscle (EDL) subsequently isografted into a second host. All recipients were athymic nude mice bearing the mouse X-linked muscular dystrophic (*mdx*) gene and therefore lacking dystrophin<sup>12</sup>. Spongostan provides a penetrable support medium<sup>13</sup>, and we inserted it between the isograft and surrounding muscles. We injured tibialis anterior (TA) muscles and/or peroneal muscles using forceps.

The presence of β-gal- and dystrophin-positive fibres in the EDL isografts confirmed the incorporation of implanted cells (Fig. 1a, b). Injury to one or more neighbouring host muscles resulted in β-gal- and dystrophin-positive myotubes and fibres in the Spongostan adjacent to the injured muscle (Fig. 2a, b). In the case of single muscle injury (9 cases), we saw positive fibres in the adjacent injured muscle but not in the neighbouring uninjured muscle (Fig. 2c, d). Where two host muscles were injured (2 cases), implanted cells migrated to both (Fig. 2e). These results indicate the preferential migration of implanted cells from the isograft, through the Spongostan to a site of muscle injury. Where host muscles were uninjured, there was no evidence of migration of implanted cells, either in the Spongostan or adjacent muscles in two of the three cases. But in the third case, we saw β-gal/dystrophin-positive fibres in both TA and peroneus (Fig. 2f). This finding

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