MERCURY CONCENTRATION IN BLOOD AND URINE BY RISK GROUP (ARITHMETIC MEAN)

	No. in sample	Hg in blood ($\mu g\ l^{-1}$)	Hg in urine ($\mu g\ l^{-1}$)
Trader	42	30.4	78.9
Miner	18	34.3	18.5
Pregnant female	8	14.1	10.0
Fisheater: adult	14	96.7	13.9
Fisheater: child	5	70.4	6.4
Resident: adult	4	20.0	18.5
Resident: child	16	14.5	18.6

sure are the inhalation of inorganic Hg vapour when mercury/gold amalgam is burnt, both at the site of extraction and when it is subsequently traded, and the ingestion of methylated (organic) Hg in fish tissue. Mercury in urine is an appropriate indicator of the former. At urinary Hg concentrations of 100 μg creatine[4] (approximately 100 $\mu g\ l^{-1}$) there is a high probability of developing the classic signs of mercurialism, erethism and tremor. At Crepurí, six individuals exceeded 100 $\mu g\ Hg\ l^{-1}$, all of them traders, three of whom showed levels above 200 $\mu g\ l^{-1}$ Hg, the highest level recorded being 843 $\mu g\ l^{-1}$ Hg. In all, 22 of the 106 individuals sampled exceeded or approached 50 $\mu g\ l^{-1}$ Hg, a level at which industrial workers have been recommended to be removed from further exposure[4].

Mercury in blood is an appropriate indicator of recent exposure to ingestion of organic Hg in fish. In a population with high fish consumption, a blood Hg level of the order of 200 $\mu g\ l^{-1}$ has been shown to be associated with neurological change characterized by paraesthesia[5]. Four individuals in Jacareacanga, all fisheaters, either exceeded or approached this threshold, from a total sample of 25. Blood Hg levels in Jacareacanga were on average several times higher than in the other sites.

Environmental samples were also taken at each site. Mercury data showed good overall agreement between the Tapajós valley and the results previously recorded for the Madeira basin[2]. Concentrations of Hg in floordust samples taken from gold trading houses in Itaituba and Crepurí showed massive pollution of the workplace; several samples exceeded 1% Hg and there was visible Hg in floor dust at two locations. There were elevated Hg levels in fish samples from Itaituba, Jacareacanga and Crepurí, ranging up to 2.6 mg kg^{-1} fresh weight. The WHO provisional tolerable weekly intake for methyl mercury is 0.2 mg, which would be provided by a weekly intake of only 1 kg of about half the 52 samples taken.

There is an urgent need to establish and quantify exposure pathways and the health impact of mercury in fisheating and gold mining/processing populations. It is also imperative that cleaner technologies for gold mining and processing are developed. A three-year project was started in January to address these issues, financed by the European Commission.

David Cleary
Centre of Latin American Studies, Cambridge University, West Road, Cambridge CB3 9EF, UK
Iain Thornton*
Nigel Brown
George Kazantzis
Global Environment Research Centre, Imperial College, 56 Queen's Gate, London SW7 5JR, UK
Trevor Delves
Department of Clinical Biochemistry, Southampton General Hospital, Southampton SO9 4XY, UK
Simon Worthington
SOL-3, 3961 Vissoie, Valais, Switzerland

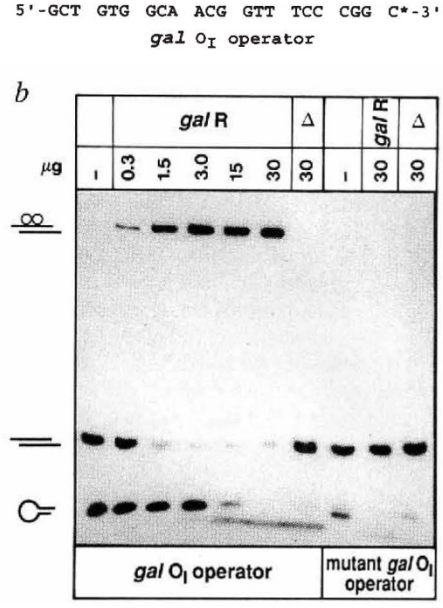
*To whom correspondence should be addressed.

1. Nriagu, J. *et al. Nature* **356**, 389 (1992).
2. Pfeiffer, W. & Lacerda, L. *Environ. Techn. Lett.* **9**, 325–330 (1988).
3. Malm, O., Pfeiffer, W., Souza, C. & Reuther, R. *Ambio* **9**, 11–15 (1990).
4. WHO *Recommended Health-Based Limits in Occupational Exposure to Heavy Metals* Tech. Rep. Ser. No. 647 (Geneva, 1980).
5. WHO *Environmental Health Criteria 101: Methylmercury* (Geneva, 1990).

DNA recognition and the code

SIR — The *gal*S repressor[1] belongs to a large family of proteins that contain a helix–turn–helix motif. Weickert and Adhya[1] found that this repressor binds to an operator within its own coding sequence. We show here that this operator is identical to the DNA sequence coding for residues 1–6 of the recognition helix of this repressor. Our *in vitro* gel retardation experiments confirm the *in vivo* results of Weickert and Adhya, and show that *gal* repressor specifically retards a double-stranded 22-residue oligonucleotide encoding residues 1–6 of its recognition helix (see figure).

Does this phenomenon reflect a general property of the genetic code? Hickok and collaborators[2] have recently performed molecular modelling experiments to show that amino acids of the recognition helix of the glucocorticoid receptor can bind to their coding sequences. We have analysed six other real or model recognition helices in gel-shift experiments, but could not find



a, Sequences of the recognition helices of *gal*S or *gal*R repressor and gal operator O_1. *b*, Gel retardation assay[3] with crude extract of *gal* repressor and *gal* O_1 operator or mutant *gal* O_1 operator. The mutant *gal* O_1 operator carries an A → C and a G → C exchange in codon 2 of the recognition helix. The triangle indicates crude extract prepared from the same *gal*R host containing no *gal*R-overproducing plasmid; symbols on the left indicate hairpin structure, free DNA and the protein–DNA complex (from bottom to top).

another example where there is any specific binding to the coding region (a list of sequences is available on request from the authors). The likelihood that the symmetry axis of *gal* O_1 is positioned as it is, is about 1 in 600, assuming it could be positioned anywhere upstream or downstream between base pairs 92 and 401 (the positions of O2 and O3 of the homologous *lac* system). The likelihood that the relevant base pairs of *gal* O_1 can encode residues 2–5 of the recognition helix of *gal*R is less than 1 in 256 if we make the minimal assumption that the first position of the codons cannot be replaced by other base pairs. Thus the combined likelihood is about 1 in 10,000.

It remains to be seen whether our failure to find other examples indicates that the actual positioning of the *gal*S operator is an accident of evolution.

Benno Müller-Hill
Peter Kolkhof
Institut für Genetik, Universität zu Köln, D-50931 Köln, Germany

1. Weickert, M. J. & Adhya, S. *J. Bact.* **175**, 251–258 (1993).
2. Harris, L. F. *et al. Proc. natn. Acad. Sci. U.S.A.* **90**, 5534–5538 (1993).
3. Lehming, N. *et al. EMBO J.* **9**, 615–621 (1990).