amino acids, uric acid or glucose in the urine) are also found in some patients with X-linked recessive nephrolithiasis[9].

About 70–80 per cent of the filtered calcium is reabsorbed in the proximal tubule in a largely passive process that is dependent on solvent drag associated with bulk fluid reabsorption[10], which in turn is dependent on reabsorption of active solute, including chloride. One possibility is that CLC-5 channels in the proximal tubule are involved in transepithelial chloride transport; loss of this channel would result in diminished reabsorption of chloride and fluid and consequently of calcium. But how such a mechanism could account for the presence of low-molecular-weight proteins in the urine is not clear.

CLC-5 chloride channels, like a number of other types of ion channels, appear to form multimeric complexes[6,11] which might not only influence the biophysical properties of CLC-5-containing complexes, as suggested by Thakker and colleagues, but could also permit the association of CLC-5 with other subunits, expanding the repertoire of possible physiological roles for this channel. For example, low-molecular-weight proteins that are specifically absorbed in the proximal tubule are taken up by endocytosis into a vacuolar-lysosomal system with an acidic interior provided by the action of an electrogenic H^+-ATPase pump[12]. Chloride channels are important in such vesicular systems because they provide a mechanism for dissipating the charge that results from proton pumping.

Perhaps CLC-5 channels play some part in this process. Could the disrupted movement of these vesicles within the cell affect the function of the transporters for calcium (and maybe those for phosphate or amino acids) in renal tubule plasma membranes, and therefore the absorption of these solutes? Or could CLC-5 channels also be involved in the control of calcium transport in more distal segments of the human nephron? Regulated calcium absorption occurs in these more distal nephron segments and divalent mineral ion absorption is strongly dependent upon, or influenced by, chloride transport processes[10]. The possible participation of CLC-5 in these processes needs to be explored.

Another big question to emerge from the new work[5] concerns the differences found among these three hereditary hypercalciuric disorders in bone pathology (rickets) and in the amount of deposition of calcium salts in the kidney. There is no obvious correlation between the phenotypic differences and the type of *CLCN5* mutation, but the limited number of mutations studied may have obscured such an association. Could *CLCN5* actually be expressed in bone cells and thus contribute to bone abnormalities? Perhaps other genetic or environmental factors, such as calcium or phosphate intake, dietary vitamin D, or circulating levels of parathyroid hormone, may influence the severity of bone and renal pathology in these patients.

Once the localization, function and regulation of CLC-5 are known, we should have a clearer picture of how this chloride channel functions in the kidney. But there may be more surprises in store. □

Steven C. Hebert is in the Laboratory of Molecular Physiology and Biophysics, Renal Division, Department of Medicine, Brigham and Women's Hospital, 75 Francis Street, Boston, Massachusetts 02115, USA.

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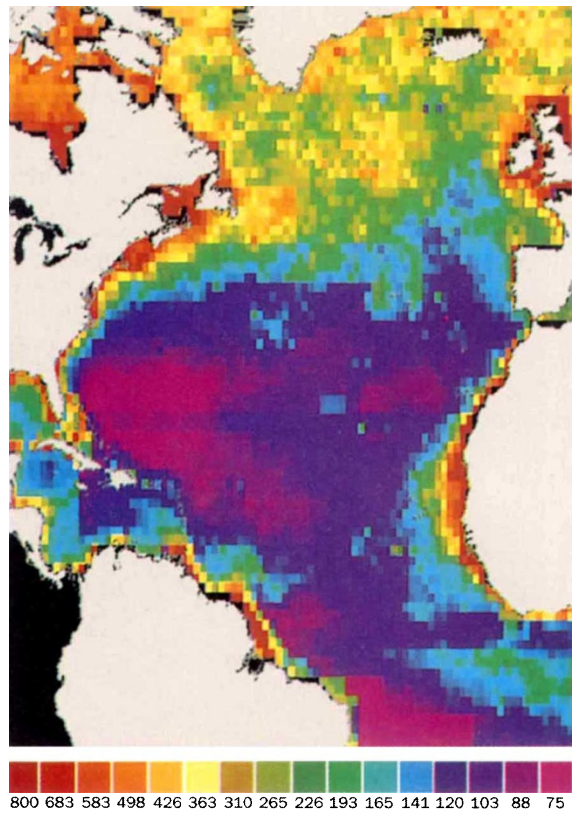
BIOLOGICAL OCEANOGRAPHY

Provincial primary production



Is this picture just another ocean-colour satellite image of the North Atlantic? Not at all. S. Sathyendranath *et al.* have exploited the remote-sensing revolution — which transformed marine science with ocean-colour measurements made using a trial sensor between 1978 and 1986 — by calculating the biological productivity of the entire North Atlantic at a regional resolution and by season (*Deep-Sea Res. I*, 42, 1773–1802; 1995). The picture shows the estimated annually averaged primary production in 1979, the year with the best total data set (units are g carbon m^{-2}).

The authors went about their task by first devising a scheme for dividing the ocean up into 18 different 'biogeochemical provinces' according to observed algal ecology and physical processes such as vertical mixing — these provinces are akin to different terrestrial biomes, boreal forest, savanna, desert and tundra for instance. They then used thousands of *in situ* measurements of photosynthetic parameters and vertical distributions of chlorophyll, combined with phytoplankton pigment data derived from the satellite observations, to compute primary production for different seasons for each biogeochemical province. Despite the patchiness of the satellite data, these are the most robust regionally resolved estimates yet made of marine primary productivity on a basin-wide scale. Such estimates are of great value not only to ecophysiologists, but — given the involvement of marine ecosystems in the global carbon cycle — also to climate researchers and oceanographers.

Plans for the deployment of advanced purpose-built sensors have been frustrated for the past few years. But eleven such sensors are scheduled for launch before the year 2000, several of them by the end of 1996. They will carry with them hopes that the ensuing data will ultimately provide estimates of the rates of other key processes within the global carbon cycle, such as new production and air–sea exchange of carbon dioxide.

Philip Newton