Isotope geochemistry

Contamination or source-region heterogeneity?

from Alex Halliday

OVER the past thirty years considerable effort has been invested in using the isotope compositions of certain elements (principally Sr, Pb, Nd and O) to reveal information about the chemistry and history of the source regions of magmas. There are two causes of variation in isotope ratios — radioactive decay (for example, the production of ^{87}Sr from ^{87}Rb) and mass-dependent isotope fractionation during reactions. In the first case, the isotope compositions tell us about the chemical history (that is, the age and parent/daughter elemental ratios) of the source. In the second case we can, in addition, learn about some specific processes that operated in the magma itself. Such data have been fundamental in solving major petrological problems such as the origin of granites and basalts. This has led to a relatively detailed knowledge of the chemical structure and evolution of the Earth's mantle and of the growth of the continental crust.

But the case is not always as clear cut. There is still debate over the interpretation of the isotopic characteristics of rocks believed to have formed in the mantle but to have risen through the continental crust. The argument centres on the relative roles played by contamination by the continental crust and inheritance of features from the sub-continental mantle. The problem is fundamental to the origin of igneous rocks and is made worse by the range and complexity of potential contamination mechanisms, for example, by fluids or partial melts, which produce non-diagnostic effects. One school of thought discounts crustal contamination on the basis of banal assumptions about the probable effects, whereas those from the other school simply ignore the possibility that anomalous mantle is important. Attempts to resolve these views have often resorted to lengthy convoluted arguments.

Fortunately there are some studies which have shed light on this confusion and have provided clear evidence for both open (contaminated) system behaviour and the existence of anomalous mantle. The recent work of Cortini and van Calsteren[1] is refreshing in that they have studied the genesis of lavas from southern Italy by determining the Pb isotope composition in separated phenocrysts (thought to be crystals precipitated from the magma) as well as in whole rocks. The observation of a systematic isotope disequilibrium led to the conclusion that some open system process has operated, which clearly could not have been demonstrated so unambigously if the authors had simply analysed the whole rock samples. Remarkably few such detailed dissections of lavas have been performed previously, although studies of nodule suites[2,3] have provided incontrovertible evidence for anomalous sub-continental mantle and others have proved the presence of crustal components in plutonic rocks[4,5]. Having said this, studying minerals separated from volcanic rocks for signs of open systems behaviour is not new[6-8], and the evidence acquired for significant isotope disequilibrium has been ascribed to a number of possible causes.

These kinds of studies highlight two important features that could provide the key to understanding the genesis of young volcanic suites. First, detailed studies that include mineral separation and analysis of individual mineral grains and glass, although tedious, are of immense potential value in providing clear evidence of open system processes. One might further envisage that with the development of ion-probe techniques detailed spatial resolution of trace-element concentration and oxygen-isotope composition will also be an exceedingly powerful way to proceed. Second, much more information on petrogenetic processes can be obtained by using various isotope techniques to study the samples. The elements in silicate systems vary in their mass-transfer response to different conditions and processes. Hence, it is now simply inadequate to study contamination with a single isotope method. It is also a less efficient use of resources to use different samples when using different techniques. Combined isotope studies optimize information with which to define uniquely a signature for a component or process. As there are many igneous provinces for which the crustal contamination versus enriched mantle debate is still unsettled, it is to be hoped that fresh approaches will provide further rigorous testing for the two hypotheses. □

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Tumour immunology

Molecular basis of tumour spread

from Ian R. Hart

METASTASIS, the spread of tumours throughout the body, is a process of obvious clinical significance. Despite the fundamental importance of the phenomenon, relatively little is known about the molecular and cellular mechanisms of its regulation and control. One way in which disseminating malignant cells may evade host defences is suggested in a report on page 301 of this issue, where transfection of neoplastic murine cells with *H-2* genes is accompanied by nullification of their metastatic capacity[1].

Major histocompatibility complex (MHC) class 1 antigens (termed H-2 K,D and L in mice) are distributed widely on nearly all cell types and play an obligatory role in immunoregulation; cytotoxic T lymphocytes only recognize 'foreign' antigens when they are associated with these class I molecules. Immune regulation of tumour growth and development, then, is not solely a consequence of tumour-cell antigens but also depends on MHC class I antigen expression, as demonstrated experimentally in recent studies using DNA transfection techniques[2,3]. If solid tumour growth can be affected so profoundly by class I antigen expression, how much more likely are metastasizing cells, travelling through the body singly or in small clumps, to be affected by a similarly regulated immune response? The answer, provided by Hammerling and colleagues[1], seems to be that such effects are just as dramatic.

Two clones from the murine T10 sarcoma, one metastatic and one non-metastatic but both lacking expression of H-2 molecules encoded by the K-end of the H-2 complex, were transfected with $H\text{-}2K^b$ or $H\text{-}2K^k$ genes. Expression of these H-2K antigens in the metastatic clone was found to be associated with a profound decrease in metastatic capacity as determined in immunocompetent recipient mice. Metastatic capacity in immunologically depleted animals remained unimpaired, thus demonstrating the central involvement of the immune response in this phenomenon.

Limitation of metastatic spread by an immune response regulated through MHC class 1 antigens not only offers hope for potential therapeutic application (interferons, for example, induce a marked increase in the surface expression of these molecules), but also suggests that an inverse relationship exists between malignancy and expression of MHC class 1 molecules[4].

This phenomenon may account for the marked heterogeneity that exists for the metastatic phenotype in tumour cell populations[5]. Some findings are consistent