Crystallography

Defects in reduced oxides

from C.J. Humphreys

CRYSTALS are like people: it is the defects in them which tend to make them interesting. In recent years, solid-state physicists and chemists have become increasingly aware that real crystals are usually not perfectly periodic arrays of atoms. Instead they contain a variety of isolated defects, which are important because they can strongly influence the physical and chemical properties of the crystals. An article on page 319 of this issue, by Bursill and Smith, reports the use of high-resolution electron microscopy to provide new information on the isolated defects which form when oxide crystals are reduced. This is the latest development in understanding reduced oxides, which have long puzzled chemists and physicists.

The basic question is the following: when oxide crystals are reduced (for example, $MoO_3 \rightarrow MoO_{3-x}$), are the resulting oxygen vacancies in the structure distributed homogeneously or are there isolated extended defects, and if so how do they form? Until 1950 it was believed that the oxygen vacancies were distributed homogeneously. Then A. Magnéli (*Ark. Kemi* **1**, 513; 1950) observed extra diffraction spots in X-ray diffraction patterns from reduced oxides which he correctly attributed to an ordered array of defects called crystallographic shear planes (CSPs). Five years later A. D. Wadsley postulated the existence of isolated CSPs (*Rev. pure appl. Chem.* **5**, 165; 1955). But the then available techniques of X-ray and neutron diffraction gave diffraction patterns which yielded atomic positions statistically averaged over a relatively large diffracting volume (typically 1 mm^3) of the crystal and so were incapable of detecting individual crystal defects. It took another fourteen years before Wadsley's predictions were confirmed by the observation of isolated CSPs by electron microscopy (Bursill, L.A. & Hyde, B.G. *Phil. Mag.* **20**, 657; 1969).

We can think of CSP formation as follows. If it is energetically favourable for them to do so, oxygen vacancies will cluster together on a crystal plane rather than exist as isolated vacancies. The crystal on each side of this cluster of vacancies will then collapse into the missing layer of oxygen atoms and also shear sideways to reform chemical bonds. The defect so formed is a CSP. In MoO_3 the local chemical composition at a CSP is MoO_2. Hence a reduced crystal of overall composition MoO_{3-x} can be thought of as a matrix of MoO_3 plus the appropriate number of CSPs of composition MoO_2. We thus have a beautifully simple concept of how oxygen vacancies are accommodated in reduced oxides. If there are a number of CSPs in a crystal they will form an ordered array at a sufficiently high temperature, as Magnéli originally observed.

The picture I have just given is a simplified one. In particular, for a very small degree of reduction (corresponding to $x<0.005$) CSPs are not usually observed. Are the oxygen vacancies at these low concentrations uniformly distributed or are they concentrated into isolated small defects, and how do the CSPs form and grow? These are the questions that Bursill and Smith can address in their paper because of the availability of the latest generation of very high-resolution electron microscopes, with a resolution of less than 0.2 nm. They show, for the first time, that there are small localized defects which precede the CSPs and that these defects may be characterized through careful analysis of very high-resolution electron micrographs. Thus we gain new insight into the earliest stages of structural changes that accompany reduction. This work is not only important for chemists and physicists studying oxides but also illustrates the power of high-resolution electron microscopy for studying, at the atomic level, the structure of defects in a wide range of materials. □

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Immunology

More on the T-cell receptor

from Philippa Marrack

NOT surprisingly, one of the main subjects of interest at a recent symposium* on 'Regulation of the immune system' was the receptor on T cells which allows them to respond clonally to antigen in association with products of the major histocompatibility complex (MHC). A year or two ago, when the molecules responsible for this feat were identified on the surfaces of T-cell clones, using clone-specific antibodies, they were shown to be disulphide-linked proteins made up of two different chains each of molecular weight 40,000–50,000 and containing variable and constant peptides. The T-cell receptor was therefore recognized to be strikingly similar in overall design to immunoglobulins. Recent papers in *Nature* (**308**, 145 & 149) reported the isolation of cDNA clones which probably encode one of the two polypeptide chains.

Since nearly all the protagonists involved in these discoveries were at the meeting, immunologists were given an opportunity to be brought up to date on all aspects of this work. Steven Hedrick (University of California, San Diego) and Tak Mak (Ontario Cancer Center) reviewed the data on the cDNA clones. The mRNAs from these clones are T-cell specific, come from genes which rearrange between the germ line and mature T cell, and are similar but not identical to immunoglobulins in nucleotide sequence. Moreover, the proteins they encode are very immunoglobulin-like in sequence and, in particular, include cysteines which could mark the domain-like structures so characteristic of this type of molecule. Hedrick also pointed out that the cysteine near the carboxy-terminal end of the protein is in a position analogous to the cysteine on immunoglobulin light chains. Since the latter is involved in bridging light chains to heavy chains in most immunoglobulins, the former may have a similar function in the T-cell receptor, being used to bridge the two chains of the heterodimer.

Mark Davis and Nicholas Gascoigne (Stanford University) described the structure of mouse genomic clones encoding these sequences; their data are published on page 322 of this issue. Again there are striking similarities with immunoglobulin, since several segments of DNA, which occupy separate loci in the germ-line genome but are brought together by rearrangement in T cells, contribute to T-cell receptor cDNA (see the figure). One segment contains a cluster of variable-region (V_T) genes, mapping an undetermined distance from a proposed segment containing several diversity-region (D_T) genes (DNA encoding this segment has not yet been found). A third segment contains a cluster of seven joining-region (J_T) genes and a constant-region (C_T) gene. The last contains four exons, of which three were expected: the exons for the constant-region immunoglobulin-like domain, which is extracellular, a transmembrane sequence, and a cytoplasmic domain. But the fourth, encoding four to six amino acids between the extracellular domain and the transmembrane sequence, came as a surprise. This extra domain has some sequence homology with the hinge region of immunoglobulins, the part thought to confer flexibility on antibodies. A lack of prolines in the hinge region of the T-cell receptor suggests it might not be quite so flexible. The cysteine equivalent to that thought to be involved in interchain disulphide bonds in antibody molecules is found in this exon.

Davis and his co-workers have found

*The Ortho-UCLA symposium on 'Regulation of the immune system' was held in Park City, Utah, 18–25 March.