Antistructure and Point Defect Response in the Recovery of Ion-Irradiated Cu_3Au

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The response of the point defect and antistructure systems to ion beam irradiation is investigated using methods of linear response on thin single crystals of ordered Cu_3Au grown by molecular beam epitaxy. We demonstrate that antisite evolution, as measured by electrical resistance, quantitatively determines both the defect populations and diffusion in the irradiation field, and we explore new linear and nonlinear response processes as the antistructure system is driven from equilibrium.

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Irradiation effects and damage in crystals are critical factors in energy research but for decades their detailed understanding has remained an elusive goal. Defect processes have been treated by mean field theory and by computer simulations but both fail to describe defect interactions and the defect life cycle with sufficient accuracy, mainly because the defect sink strengths are hard to predict [1,2]. In the present research we control the sinks by thin film methods using Cu_3Au crystals grown by molecular beam epitaxy. These are sufficiently perfect that the free surface alone provides the dominant sink at which defects equilibrate, typically after random walks of $\sim 10^7$ jumps [3]. By varying the irradiation flux we control whether point defects annihilate each other or at the sink. Methods of linear response are employed to track the evolution of irradiation-induced antistructure in response to damage pulses. We establish that this evolution provides a quantitative measure of radiation enhanced diffusion that agrees with earlier measurements of mean square displacements. Close antistructure pairs created in the initial state of damage can be identified explicitly in the recovery, as well as differences of damage caused by different irradiation sources. Furthermore, the measurements afford a quantitative determination of both the thermal and irradiation-induced concentrations of vacancies as functions of ion flux.

Specimens were prepared lithographically from thin single crystal films of Nb and Cu_3Au grown successively on sapphire substrates by methods that are described elsewhere [3,4]. With the entire sample restricted to a square $0.25 \times 0.25\ mm^2$, large beam currents could be used without significant sample heating. The lower inset of Fig. 1 shows the resistance increases caused by irradiations for 8 s with 50 nA of 0.6 MeV He^+ ions at two temperatures, together with the subsequent recovery. The recoveries fit well to simple exponentials that define time constants τ for the process. The amplitudes of the transients and their characteristic times τ both contain important information.

Figure 1 shows τ for Cu_3Au as a function of temperature and irradiating ion. Data for Kr, Ne, ^{4}He, and ^{2}H are included here; further results for Ar, ^{3}He, and ^{1}H follow precisely the same pattern and will be reported elsewhere. The results are highly systematic: different ions give very similar temperature dependences of τ, merely offset to larger τ the larger the mass of the irradiating ion. The solid lines in Fig. 1 indicate the same T dependence, with an offset, fitted to the data for each ion. The model that underlies the fit is explained in what follows.

Irradiation creates point defects in the form of vacancies and interstitial atoms, i.e., Frenkel defects, and also antisite defects in which Cu and Au occur on the wrong sublattices of the Cu_3Au L_{12} structure [5]. The resistance changes observed here are too large by a factor of 10^2 for the expected number of Frenkel defects [6,7] and they evolve over time intervals too long by a factor of 10^3 for vacancy or interstitial migration to the known free-surface sink [3,8]. Sufficiently few ions are employed here that each creates its separate region of damage in the lattice. Such regions are

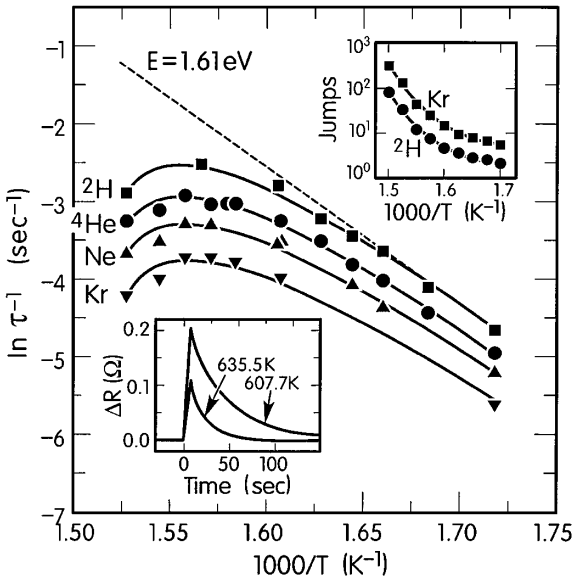

FIG. 1. The main figure shows time constants τ^{-1} for resistance decay after pulses of ^{2}H, ^{4}He, Ne, and Kr irradiation at various temperatures. Lines through the data are predictions described in the text. The lower inset shows for two temperatures the resistance transient following 8 s pulses of 1 MeV He^+ ions, which fit well to exponentials. The upper inset shows the number of jumps that occur during this relaxation time for Kr and ^{2}H ions, as deduced from the known diffusion coefficient. At low temperatures ^{2}H damage is seen to heal after only two jumps.

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known to vary in form with projectile mass and energy, with compact disordered (fcc) volumes (thermal spikes) containing 10^4 atoms created by the present Kr irradiation, while the lighter bombarding ions mainly cause chains of isolated antistructure pairs [1,9]. The two critical inferences here are, first, that virtually all excess point defects are eliminated before the observed recovery begins and, second, that excess resistance is due almost entirely to the retained antisite disorder. Therefore the decay of resistance with time during recovery must measure the annihilation of antisite disorder by steady state diffusion in the crystal. This deduction, further confirmed below, provides the basis for all the results that follow.

A remarkable feature of the damage is revealed when the number n of jumps that occur in the recovery time τ is calculated from the known diffusion coefficient [3,4], using $n = (12D\tau/a_0^2)$, with $a_0^2/2$ the squared jump length and a_0 the lattice parameter. Results for ^{2}H and Kr are shown in the upper inset of Fig. 1; those for other ions fall systematically in between. It is a fact, established by this graph, that the decay of antisite disorder from ^{2}H irradiation at low temperature requires only $\sim$2 jumps per atom. This demonstrates unambiguously that the disorder is restricted almost entirely to antisite pairs with the defects nearest neighbors. The increase of n at high temperatures reveals that the ordering energy is less effective there in biasing jumps, so that fewer direct annihilation sequences now take place. The recovery of damage from heavier ions is similar to that for light ions, but with the larger n caused by the greater number of jumps per atom in the thermal spike, and the consequent loss of close pair correlations in the antistructure.

We model the behavior of the recovery following the ion pulses by noting first that the ordering energy decreases towards the critical temperature T_c (= 663 K) so the energetic bias of jumps towards recovery therefore weakens. Suppose that the bias is an energy ε_a, with $\varepsilon_a/2$ decrease of hopping energy for decay of antistructure, and $\varepsilon/2$ increase for jumps that make antistructure persist, so that the resulting flow has a net relaxation time

$$\tau^{-1} \propto D \tanh\left[\frac{\varepsilon_a}{2T}\left(\frac{T_0 - T}{T_0}\right)\right]. \qquad (1)$$

Here, ε_a has been written as $\varepsilon_a(T_0 - T)/T_0$ so that the bias vanishes at some temperature T_0 where the crystal is disordered. Fits to this heuristic form are shown in Fig. 1. They are reasonably satisfactory for all ions with $T_0 = T_c \pm 2$ K and $\varepsilon_a \approx 1000$ K.

The validity of this interpretation has been verified by further experiments that drive the antistructure system from equilibrium using transient fluxes of irradiation. First, Fig. 2 shows the transients observed at two temperatures when steady irradiation with fluxes of 1 MeV $^4He^+$ is switched off. The resistance decays are almost independent of flux. This verifies that the decay relates to the *equilibrium* vacancy levels and diffusion, rather than to irradiation-induced processes. Second, when the transient response occurs *during* irradiation, using small changes of an ambient flux, the resulting transients now occur in the presence of irradiation-induced point defects.

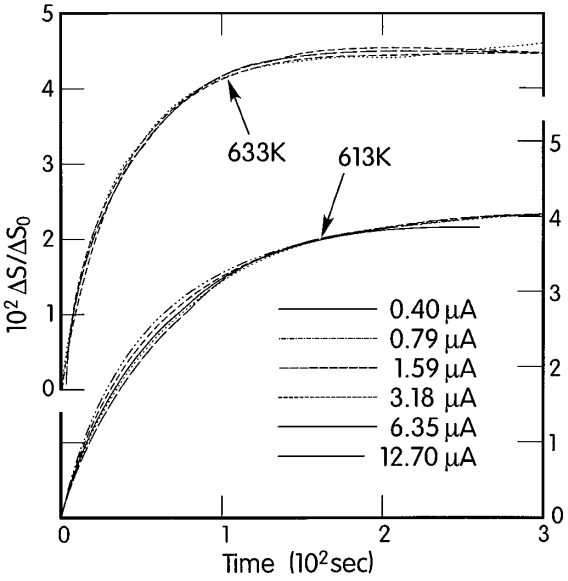

FIG. 2. Following steady states established at various irradiation levels, the transient decays in zero flux follow almost identical time evolutions. It is inferred that the decays are driven by the equilibrium point defect levels present after the irradiation is switched off, which do not depend on the flux in the earlier steady state.

Correspondingly, the measured time constants $\tau(\phi)$ now depend strongly on irradiation flux, and differ as functions of temperature from those, $\tau(0)$, the zero flux. Figure 3 shows the dependences on T first of $\tau(0)$, the recovery at zero flux, and second of $\tau(\phi_m)$, the recovery at flux

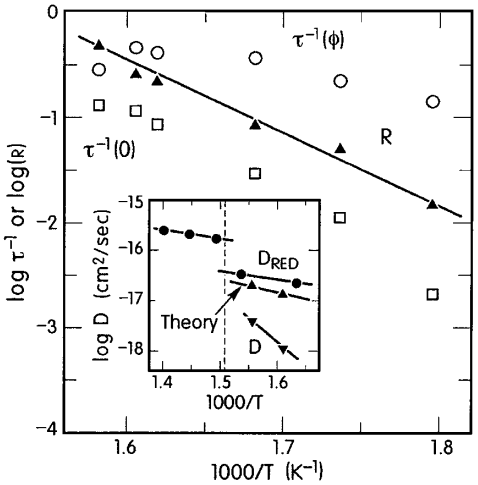

FIG. 3. The antistructure decay rates for zero flux (squares) and for flux ϕ (circles) shown as functions of T^{-1}, together with their ratio R. Inset: diffusion coefficients D and irradiation enhanced diffusion D_{RED}, measured from mean square displacements [3], and also the prediction of D_{RED} obtained from D using R.

$\phi_m = 6.5\ \mu\text{A/cm}^2$ of 1 MeV $^4\text{He}^+$ ions; also shown is the ratio R of the first to the second data sets. Note that the deviation from activated behavior in Fig. 1 cancels from the ratio R in Fig. 3. The line through the data for R has an activation energy of $\sim$0.5 eV, which agrees well with the expected magnitude of half the vacancy migration energy, $E_m/2 \sim 0.4$ eV, predicted by mean field theories of vacancy migration on the Cu sublattice [3,8].

A convincing test of this discussion can now be made. The ratio R in Fig. 3 is the ratio of diffusion in equilibrium ($\phi = 0$) to the diffusion in the irradiation field ϕ_m, as determined from antistructure evolution. Therefore it should equal the ratio of D to D_{RED}, as measured under the same two conditions in earlier, time-consuming determinations of mean square displacements. The value of D_{RED} predicted from D, using the ratio R measured here, is compared with the actual measured values in the inset of Fig. 3, where the two agree as functions of temperature to within a factor of 2. We judge this agreement to be very satisfactory. Details of the separate contributions to diffusion from the two sublattices that are neglected in the preceding discussion can undoubtedly affect the results by a small numerical factor.

One main goal of irradiation research is to understand the dependence on flux ϕ of quantities like D_{RED} and defect populations. Here we report the first measurements using the present techniques that reveal processes nonlinear in ϕ. A normal expectation [2,10] is that diffusion should increase at first linearly with ϕ, and then as $\phi^{1/2}$ in the regime of second order point defect kinetics. As the amplitude A of resistance change is more easily determined than the kinetics, we phrase the following discussion in terms of A. Suppose then that to the equilibrium vacancy diffusion D, irradiation adds a term $a(\phi)$ dependent on ϕ.

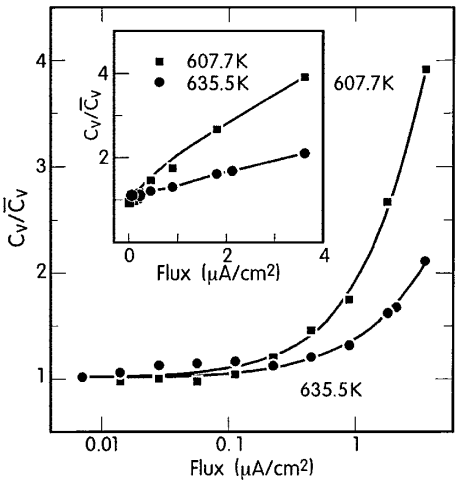

FIG. 4. Measured fractional changes of $A(\phi)/\phi$ as functions of irradiation flux, at two temperatures, shown both on a log scale and on a linear scale (inset). The line through the data for 607 K is a fit to mean field theory that yields $\overline{c}_v = 1 \times 10^{-8}$. At 635 K the behavior is almost linear so no value of c_v can be determined.

The time-dependent contribution to the resistance caused by any particular event must then take the general form $\Gamma\{[D + a(\phi)]t\}$. By summing over events that occur at rate $k\phi$, beginning at $t = 0$, we find the steady state resistance change for linear response as

$$\Delta\rho = \int_0^\infty k\phi \cdot \Gamma\{[D + a(\phi)]t\}\, dt = \frac{k\phi}{D + a(\phi)}\, G(\infty), \tag{2}$$

in which G is the integral of Γ [11]. Thus the amplitude factor $A(\phi) = k\phi/[D + a(\phi)]$ that occurs on the right of Eq. (2) determines the ϕ dependence of

$$D_{\text{RED}} = D + a(\phi) = k\phi/A(\phi) \propto \tau(\phi)^{-1}, \tag{3}$$

in which $\tau(\phi) \propto [D + a(\phi)]^{-1}$ is the time constant of the response $\Gamma(t/\tau)$. Equation (3) shows how either the time constant $\tau(\phi)$ of the response or the amplitude $A(\phi)$ of the response to an irradiation level ϕ allows the net diffusion $D + a(\phi)$ and hence the vacancy concentration c_v to be tracked with changing ϕ.

Figure 4 presents measured values of $\phi/A(\phi)$ for two temperatures, shown as functions of ϕ, and normalized by values for zero flux; these are more easily determined than values of τ. It is known [3,12] that the decay of antistructure occurs almost entirely by vacancy diffusion. Then D_{RED}/D from Eq. (3) also represent values of $c_v/\overline{c}_v$, with the denominator the vacancy concentration for thermal equilibrium. As expected, c_v depends linearly on ϕ for small flux, but this dependence slows for $c_v \gg \overline{c}_v$, in the regime of second order kinetics (see Fig. 4, inset). The precise criterion for the transition is that interstitials annihilate more with vacancies than sinks. This occurs roughly where the interstitial loss rate to the surface, $D_i\pi^2/(2L)^2$, with L the film thickness, equals the annihilation rate with vacancies, namely $4\pi D_i c_v d/\Omega$, with Ω the atomic volume and d the distance at which vacancies and interstitials recombine [3]. Because the vacancy behavior is balanced against the known sink loss, it allows the absolute vacancy concentrations to be determined whenever c_v is small enough that vacancies annihilate only a small fraction of interstitials. The quantity d has not been measured by experiment but is known to be a_0 well within a factor of 2. For simplicity above we do not distinguish between thermal and induced vacancies, but this distinction can be included exactly (in mean field theory) to give [3]

$$c_v/\overline{c}_v = A_1 + [A_1^2 + (1 - 2A_1)(1 + A_3)]^{1/2}, \tag{4}$$

in which

$$A_1 = \tfrac{1}{2}[1 - (\pi/2L)^2/4\pi l c_v/\Omega], \qquad A_3 = \sigma\phi/(\pi/2L)^2 D_v, \tag{5}$$

D_v is the vacancy diffusion coefficient, and σ is the ion cross section for creation of vacancy-interstitial pairs [3]. The lines fitted to the data points in Fig. 4 yield $\overline{c}_v = (1 \pm 0.5) \times 10^{-8}$ for $T = 607.7$ K, while for

$T = 635.5$ K the dependence of c_v on flux is linear within the uncertainties, and so no value of c_v can be obtained.

Vacancy populations for metals are believed to vary systematically so that they may be estimated with reasonable accuracy. An earlier estimate [3] of this type for Cu_3Au is $\overline{c}_v = 7\exp(-12\,000/T) = 2 \times 10^{-8}$ at 607 K, which is within a factor of ~ 2 of the values fitted in Fig. 4. This is an important demonstration that absolute vacancy populations can be determined by the present methods. It also provides one more check that the comprehensive description of defect kinetics provided by the present framework is self-consistent, within the precision of a numerical factor of order 1. In addition, the defect production rate can be obtained from A_3, if an independent measure of D is available [13].

In summary, we have employed thin film methods to investigate the response of thermal defects and antistructure of Cu_3Au to irradiation fields of diverse ions. Owing to the control provided by tailoring samples, it has been possible to quantify the effects of irradiation well beyond the limits of any previous investigation. The effects of bombarding ion mass on the recovery process and the important contribution of close antisite pairs are both clearly revealed in the transient electrical resistance. We demonstrate that antistructure recovery accurately monitors the ambient diffusion, and have been able to predict quantitatively from these observations the radiation enhanced diffusion measured in earlier studies of rms atomic displacements. The dependence of radiation enhanced diffusion and vacancy concentration on irradiation flux have also been determined. For the future it is clear from the results described above that resistive measurements of transient antistructure evolution, together with thin film growth, offer a powerful new means for determining irradiation-induced diffusion, herein accurately linked to the rms displacement of atoms.

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