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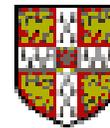


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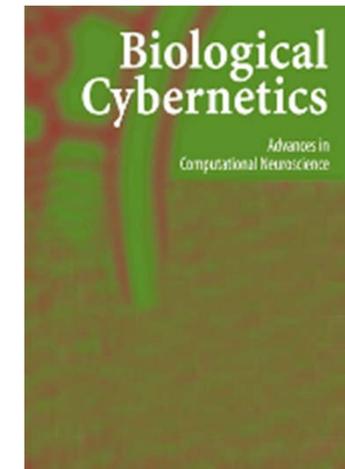
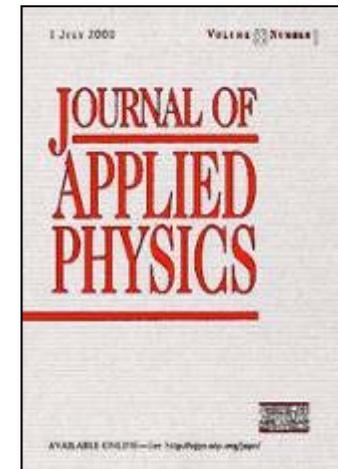
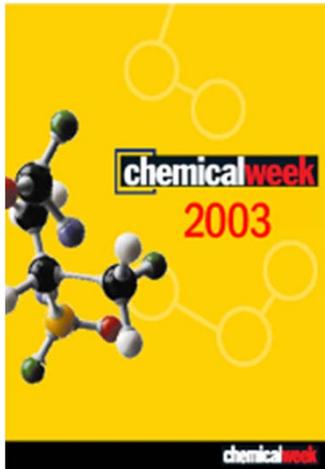




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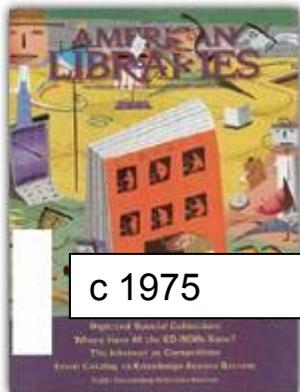
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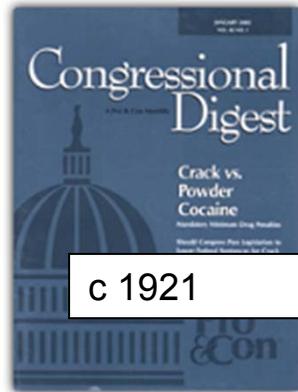


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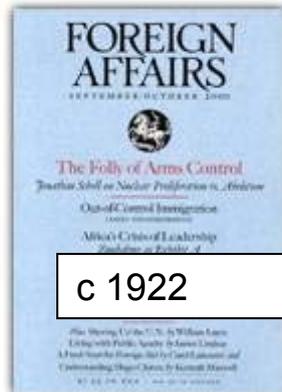
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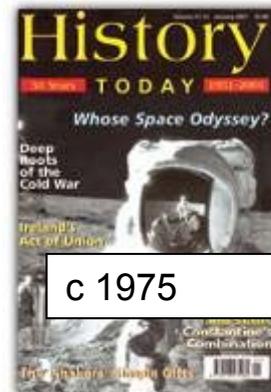
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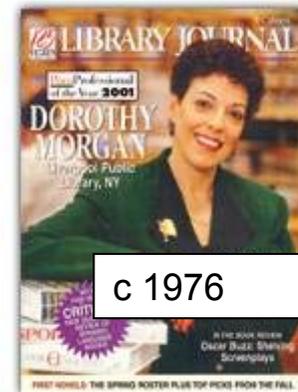
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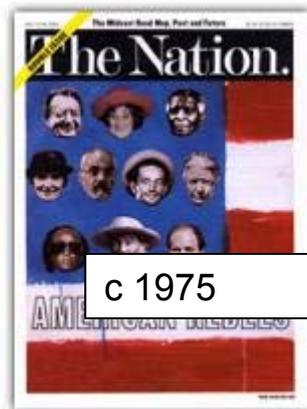
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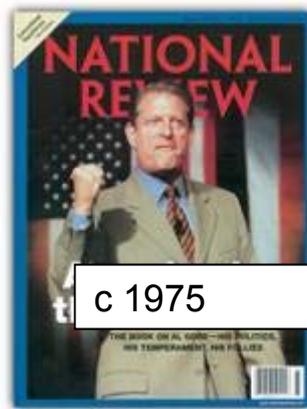
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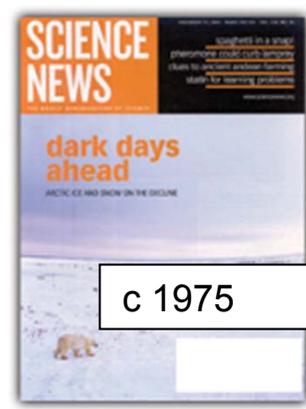
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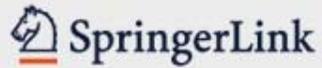


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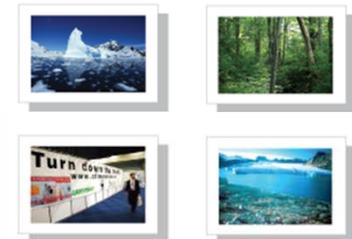
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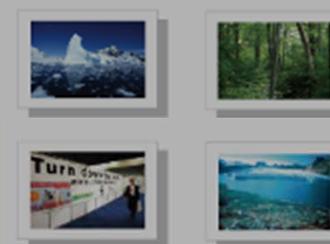


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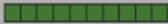
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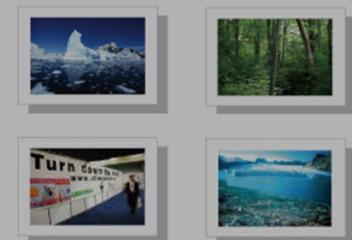
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Source: [Bulletin of the Atomic Scientists](#); Sep/Oct2007, Vol. 63 Issue 5, p26-63, 38p, 1 chart, 12 graphs, 2 maps, 2c

Document Type: Article

Subject Terms:
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Author Affiliations: ¹The New York Times

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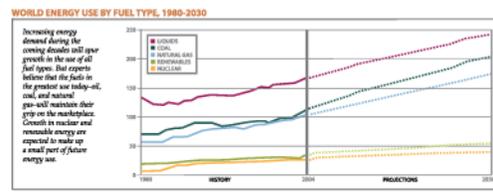


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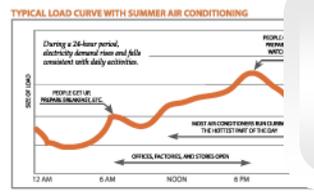
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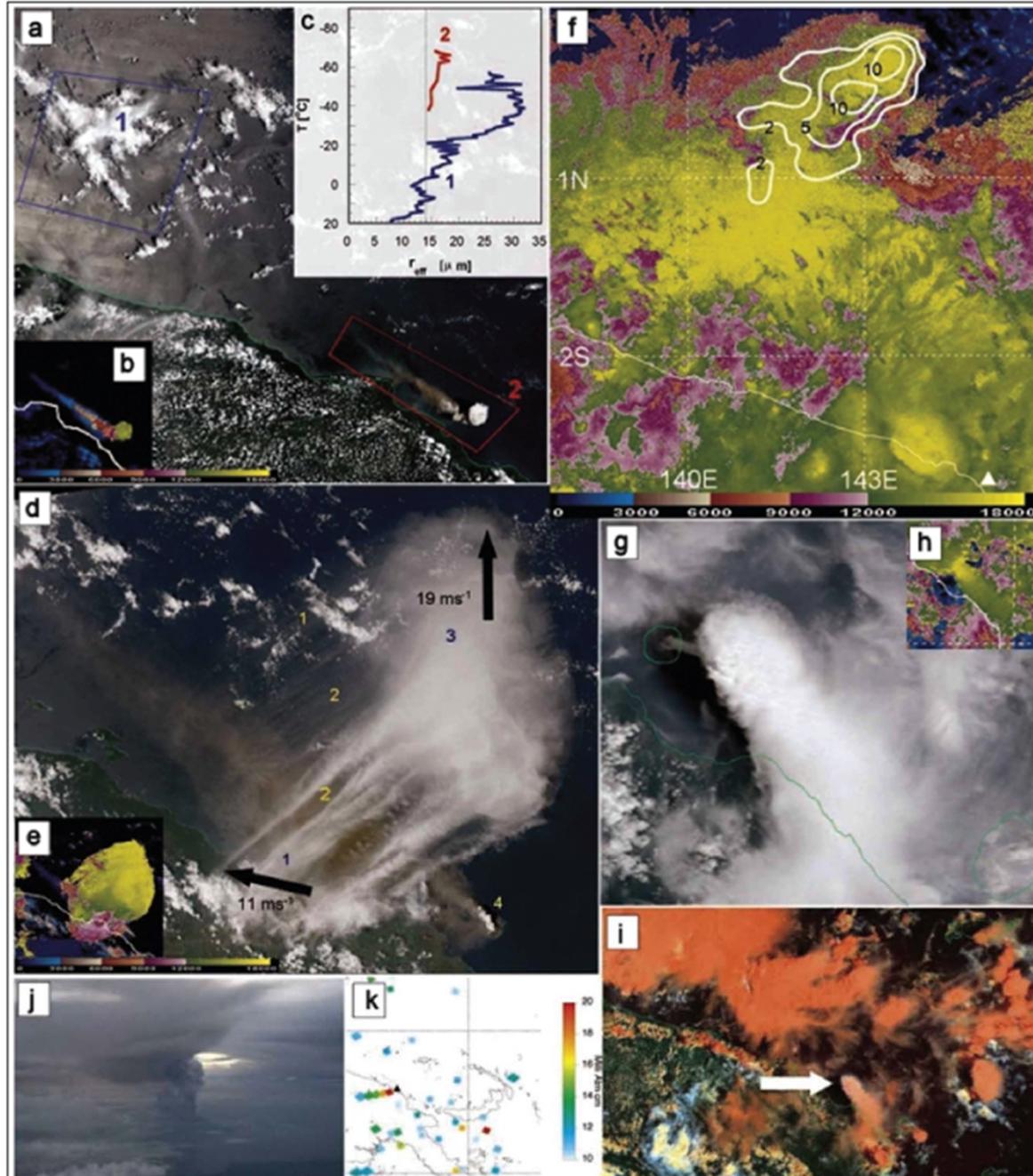
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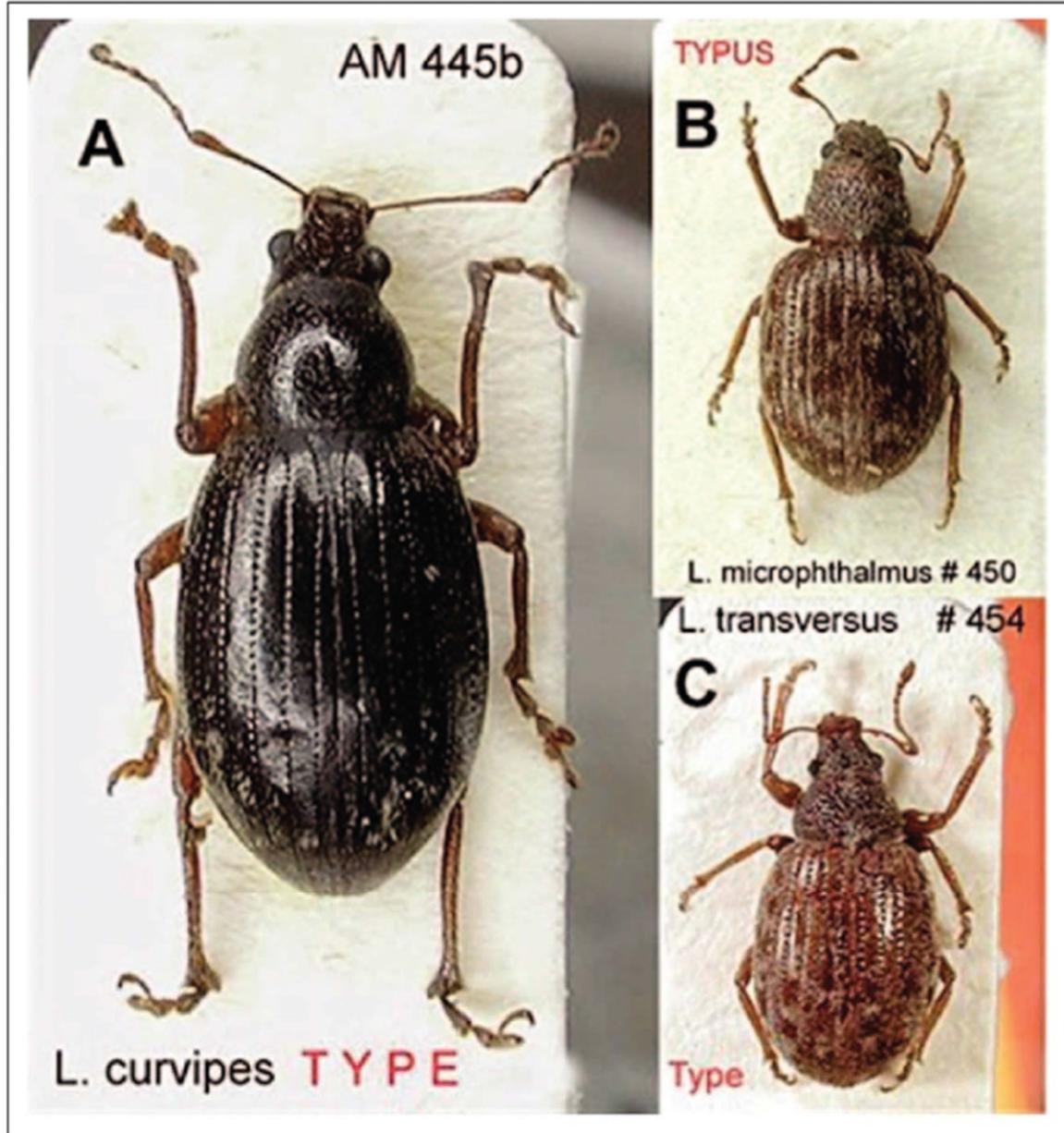
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Caption: (A) Paratype of *Laparocerus curvipes* Lindberg, 1950; (B) paratype of *Laparocerus microphthalmus*.

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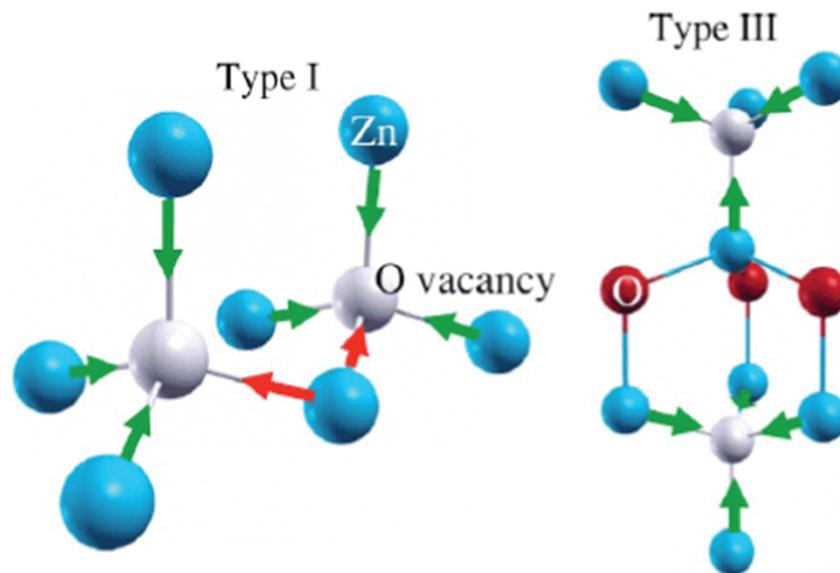
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Caption: FIG. 7. Geometrical rearrangements induced by the presence of divacancies types I and III in ZnO (shown as arrows), the light (blue online) spheres represent Zn atoms, the dark (red online) spheres oxygen atoms, and the gray spheres oxygen vacancies.

Article Title: On the convergence of isolated neutral oxygen vacancy and divacancy properties in metal oxides using supercell models.

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People: [ROBERTSON, Charles](#)
[MEMMOTT, Jane](#)

Abstract: The article considers the potential effects of global warming on the schedule of animal visitors in exchange for pollination. Jane Memmott of the University of Bristol's 1929 book, "Flowers and Insects," to study pollination. Its author, Charles Robertson, observed the timing of pollination between 429 plant species and their 1,420 pollinators. On the basis of timing shifts that have been observed in several plants and pollinators, Memmott and her colleagues figure out how the activities of plants and pollinators will advance by one to three weeks.

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insight overview

The future for stem cell research

Robin Lovell-Badge

Division of Developmental Genetics, MRC National Institute for Medical Research, The Ridgeway, Mill Hill, London NW7 1AA, UK
(e-mail: rlb1@nimr.mrc.ac.uk)

Stem cells have offered much hope by promising to greatly extend the numbers and range of patients who could benefit from transplants, and to provide cell replacement therapy to treat debilitating diseases such as diabetes, Parkinson's and Huntington's disease. The issue of stem cell research is politically charged, prompting biologists to begin engaging in ethical debates, and generating in the general public an unusually high level of interest in this aspect of biology. But excitement notwithstanding, there is a long way to go in basic research before new therapies will be established, and now the pressure is on for scientists and clinicians to deliver.

Scientists and clinicians have been beavering away in the background on stem cell research for years, but it took two breakthroughs — animal cloning by Ian Wilmut, Keith Campbell and colleagues¹ and the derivation of pluripotent human embryonic stem (ES) cells by Ian Thomson and co-workers² — to really shake things up. The first showed that an adult cell nucleus can be reprogrammed to produce an entire animal, a dramatic demonstration of hidden potential, and the second provided a possible source of cells for cell-based therapies for many human diseases. Even more intriguing was the notion of combining the two in the process of 'therapeutic cloning', whereby ES cells could be (in theory anyway) derived from a patient's cell, thus avoiding problems of immune rejection, and then coaxed into forming large quantities of the cell types needed for a cure. The derivation of human embryonic germ (EG) cell lines from aborted fetuses, with a similar potential to ES cells, by

John Gearhart's group³, provided an alternative route to obtain cells for treating patients, avoiding the need to use early embryos. But then, more recently, adult stem cells have leapt into prominence with many claims that they are more plastic than previously thought, and can in some circumstances contribute to cell types very different from those in their tissue of origin. This has suggested that patient-specific stem cell therapy can be carried out without cloning.

As is evident from reading the articles comprising this Insight, stem cell research has opened for exploration a vast new terrain of basic biology. Fundamental questions remain, such as what defines a stem cell in molecular terms, what signalling events control stem cell differentiation, and what does it mean to reprogramme a cell? Scientists are now reconsidering the very definition of a stem cell, with the attendant notions of lineage restriction and the permanency of the differentiated cell state. Although this uncertainty is unnerving, in the end we will profit from a better

Figure 1 Pluripotency of mouse embryonic stem (ES) cells. **a**, Aggregates of mouse ES cells form embryoid bodies. The dark staining shows expression of Sox2 in the less differentiated cells, whereas the rim of differentiated endodermis is unstained. (Image courtesy of D. Adixic). **b**, Histological section of a testis carcinoma derived from mouse ES cells. Many different cell types are found, all derived from the ES cells, including representatives of all three germ layers. (Image courtesy of M. Perales). **c**, The ultimate test of pluripotency is a chimera made by injecting ES cells into a blastocyst. The pigmented areas reveal the contribution of ES cell derivatives to the skin, but all tissues are composed of a mixture of ES and host embryo derivatives. Even a single ES cell can give a chimera host.

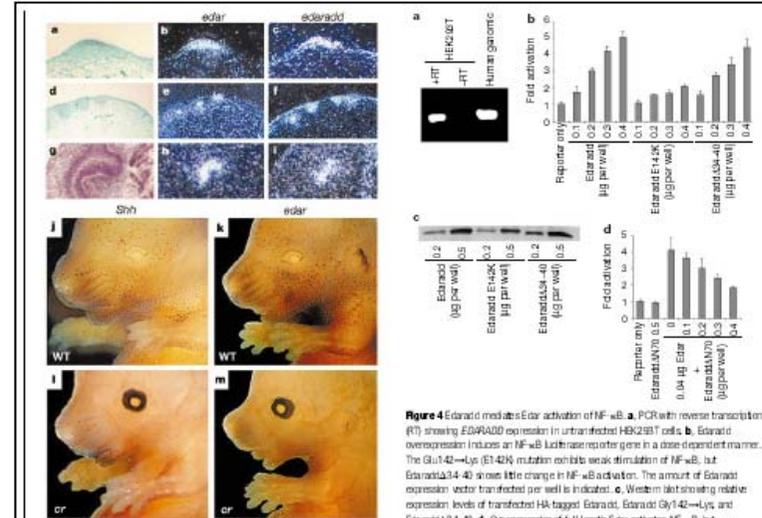
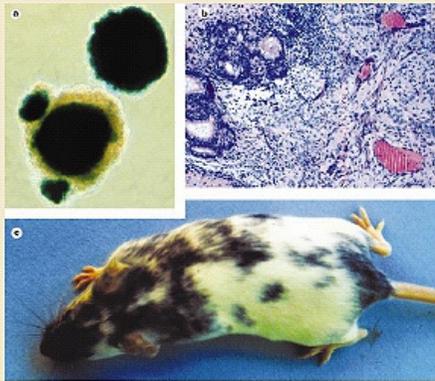


Figure 3 Co-expression of *edar* and *edaradd*. **a–f**, Serial sections of E12.5 skin (**a–c**), E14.5 skin (**d–f**) and E19.5 tooth (**g–i**) either stained with malachite green (**a, d**) or haematoxylin (**g**), or hybridized to *edar* (**b, e, h**) or *edaradd* (**c, f, i**) riboprobes. *edar* and *edaradd* are co-expressed in epithelial cells in developing whisker hair follicles (**a–f**) and in the enamel knot of the developing tooth (**g–i**). **j–m**, Whole mount *In situ* hybridization to E19.5 embryos. *Shh* (**j, l**) and *edar* (**k, m**) are expressed in whisker follicles on the snout and in hair follicle precursors on the head and trunk of wild-type embryos (**k, m**). In hair follicles but not in enamel knot (**l, m**), the *edar* mutant has black eyes as it is on a pigmented genetic background.

(Fig. 4a), and that its overexpression activates an NF- κ B reporter gene in a dose-dependent manner (Fig. 4b, c). The Glu142→Lys mutation reduces the activation of NF- κ B by *Edaradd* (Fig. 4b); however, removing the Trf-binding motif has little effect on NF- κ B activation (Fig. 4b), suggesting that Trf recruitment is not crucial for this function. *Edaradd* signalling was abolished by deletion of its N-terminal 70 amino acids (*Edaradd* Δ N70) (Fig. 4d). When co-expressed with full-length *Edar*, *Edaradd* Δ N70 interferes with the receptor's ability to activate NF- κ B (Fig. 4d), presumably acting as a dominant negative inhibitor. That *Edaradd* Δ N70 can block the activation of NF- κ B by *Edar* illustrates *Edaradd*'s central role in *Edar* signalling, and also suggests that it has a modular structure¹⁶ in which the C-terminal death domain is required for receptor engagement and the N-terminal region is responsible for signal transduction.

Our results identify a pathway in which *Edar* is activated by *Eda* and uses *Edaradd* as an adapter to build an intracellular signal-transducing complex. This linear pathway explains the identical phenotypes of the *Ta*, *d* and *cr* mutants, and also the genetic heterogeneity of human hypohidrotic ectodermal dysplasia. The adapter molecules *Tradd* and *Fadd* are central to death receptor

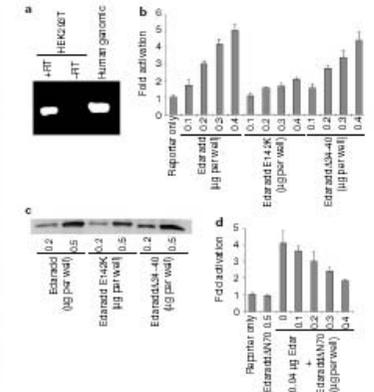


Figure 4 *Edaradd* mediates *Edar* activation of NF- κ B. **a**, PCR with reverse transcription (RT) showing *EDARADD* expression in transfected HEK293T cells. **b**, *Edaradd* overexpression induces an NF- κ B luciferase reporter gene in a dose-dependent manner. The Glu142→Lys (E142K) mutation exhibits weak stimulation of NF- κ B, but *Edaradd* Δ 34–40 shows little change in NF- κ B activation. The amount of *Edaradd* expression vector transfected per well is indicated. **c**, Western blot showing relative expression levels of transfected HA-tagged *Edar*, *Edaradd* Gly142→Lys, and *Edaradd* Δ 34–40. **d**, Overexpression of full length *Edar* activates NF- κ B, but on co-expression of *Edaradd* Δ N70 blocks this activation. For each co-transfection, 0.04 μ g of *Edar* expression vector was transfected per well. The amount of *Edaradd* Δ N70 expression vector (wt) is indicated. *Edaradd* Δ N70 alone does not activate NF- κ B (second bar).

signalling¹⁷, and the molecular identity of *Edaradd* illustrates the conservation of the archetypal death receptor pathway in a developmental context.

Methods

edaradd mapping and identification

We identified *edar* on a C57/BL6J background from The Jackson Labs. *Card6* mice were a gift from A. Bradley. Mouse *edaradd* specific oligonucleotides: *D12M104*, *D12M107*, *D12M112* and *D12M121A*, and *YACs* were obtained from Research Genetics. We carried out cDNA solution *in situ* hybridization.

The sequences of cDNA clones were checked against the high throughput genomic sequence (http://database.uchicago.edu) at the National Center for Biotechnology Information (http://www.ncbi.nlm.nih.gov/blast). Matching human RAC sequences were input into the *RG/ENSEP* gene prediction program at the Sanjour Centre website (http://genomics.sanger.ac.uk/phylo.html). The amino acid sequences of predicted genes were searched for protein domains using Pfam v5.2 at the San Diego Supercomputer Center website (http://opm.scripps.edu).

We carried out rapid amplification of cDNA ends using the GeneAmp procedure (Invitrogen) with E17.5 mouse skin cDNA as the template. Oligonucleotide sequences and PCR conditions are available on request.

Mutation analysis

Familial with no history of sensitive hypohidrotic ectodermal dysplasia were recruited in a research study approved by the institutional review board of the Oregon Health Sciences University. Consent was obtained for the use of clinical information, relevant family history and DNA samples. We isolated genomic DNA from patients by described methods¹⁸. PCR fragments from each of the alleles of *EDARADD* were amplified and sequenced. Genomic DNA from relevant family members, as well as from control individuals, was analysed for the presence or absence of mutations by ASO analysis¹⁹ or by



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<p>4. A General Disequilibrium Model of Income and Employment. By: Barro, Robert J.; Grossman, Herschel I.. American Economic Review, Mar71, Vol. 61 Issue 1, p82, 12p, 8 graphs; (AN 4515123) Cited References (18) Times Cited in this Database (96)  PDF Full Text (824K)</p>	 Add



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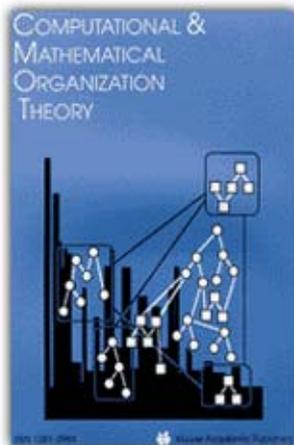
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