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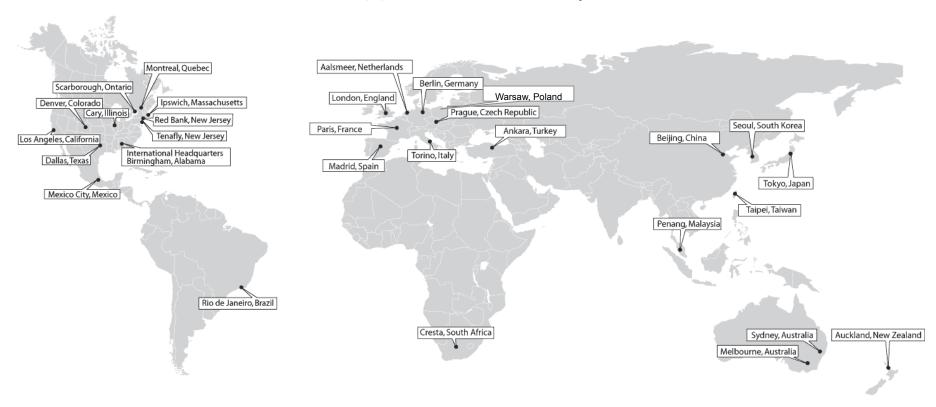
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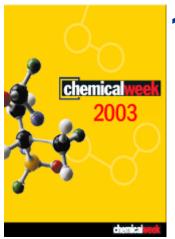
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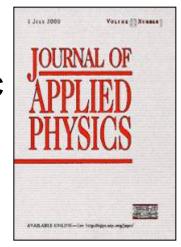


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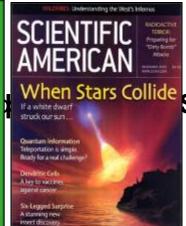


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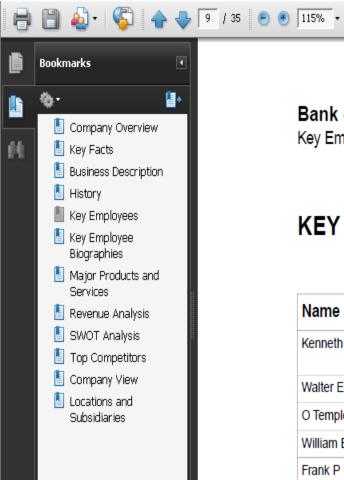
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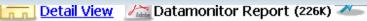


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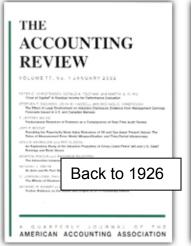
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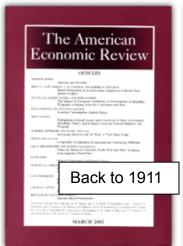
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Leading market position further strengthened by the acquisition of Countrywide and Merrill Lynch Ability to withstand financial crisis and economic slowdown Prudent capital management and dividend policy – strengthening solvency	Sub-optimal geographic asset allocation impacting return on assets Inability to curb deteriorating asset quality impacting returns and profitability		
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Credit card operations and affinity marketing likely to provide long term growth Wealth management business likely to be benefited from Merrill Lynch acquisition	CDO and sub prime exposure likely to be significant threat Increased regulatory pressures on interchange rates likely to impact credit card		

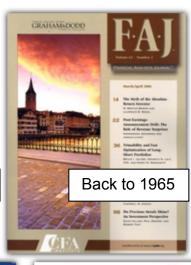


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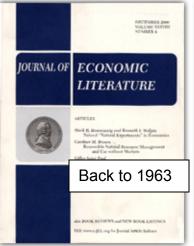




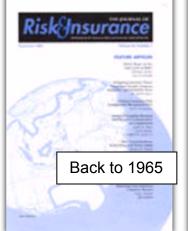
















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BRAND POSITIONING



Youngme E. Moon



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Positioning (0:26)

- Classic definition of positioning (0:22)
- Category positioning map (0:39)
- Classic approach to positioning (0:41)

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Positioning

Classic definition:

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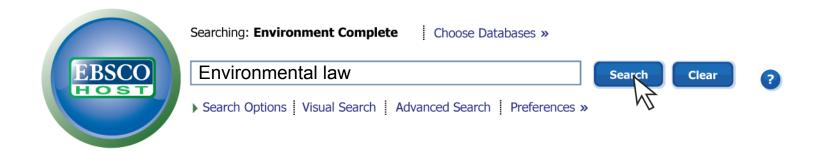




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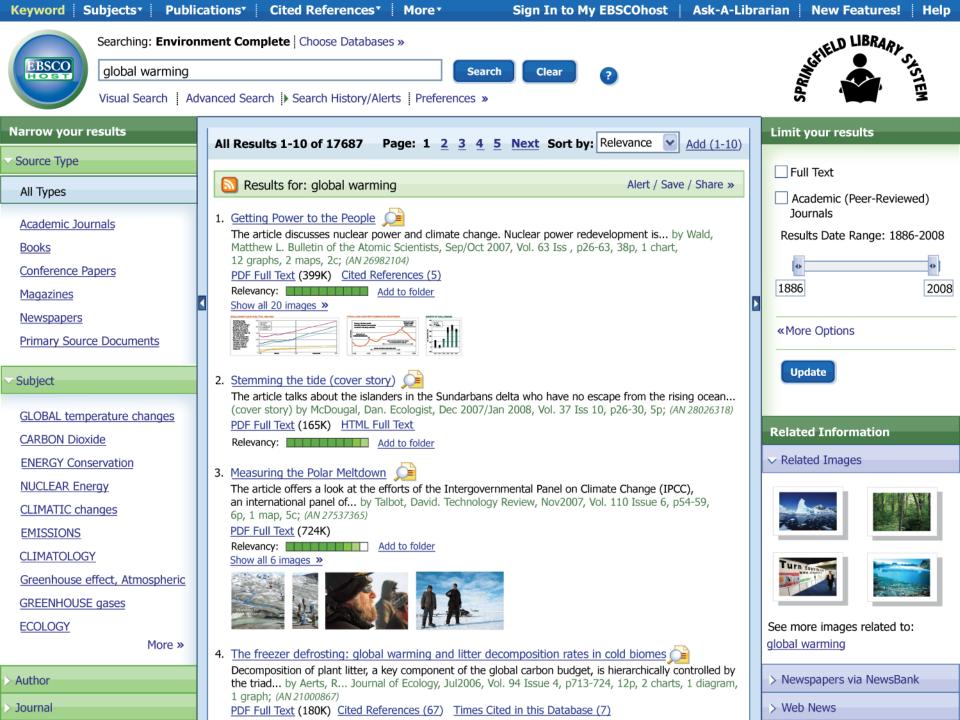
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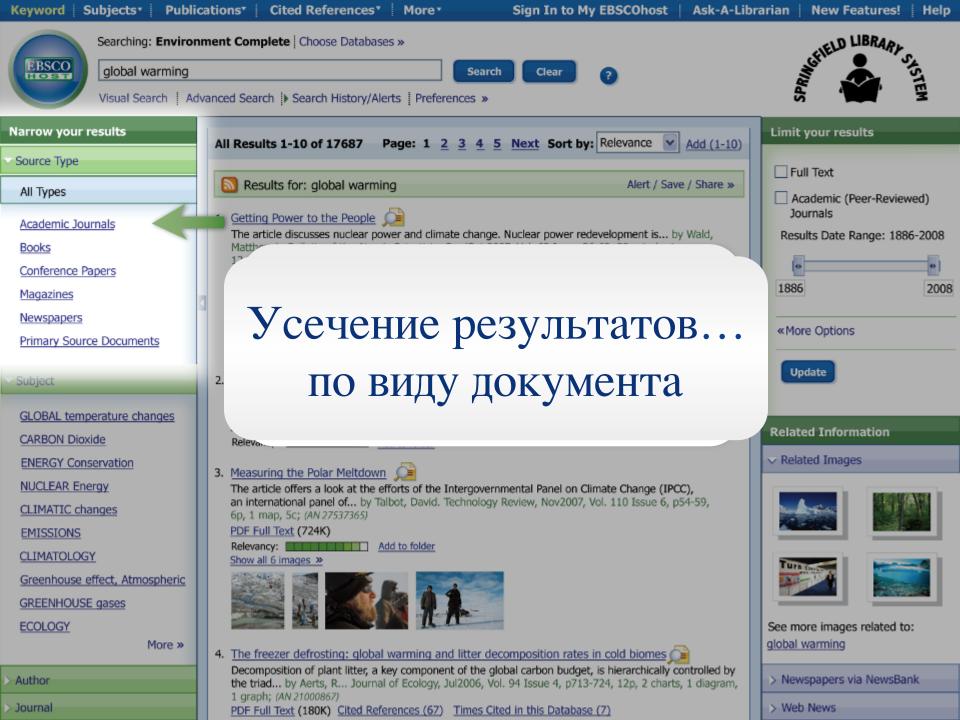


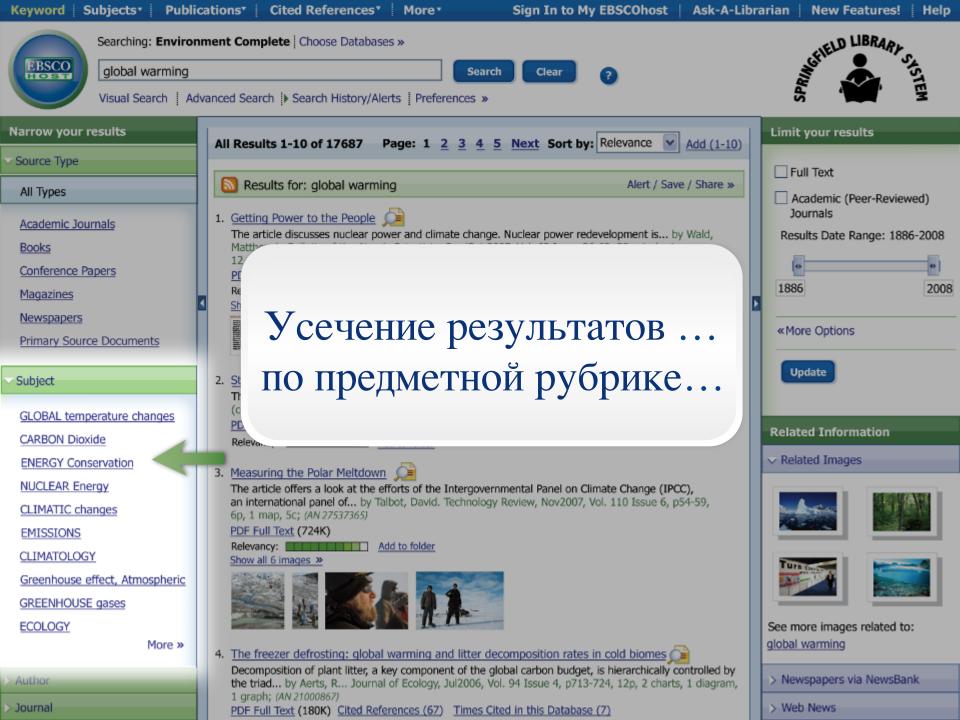
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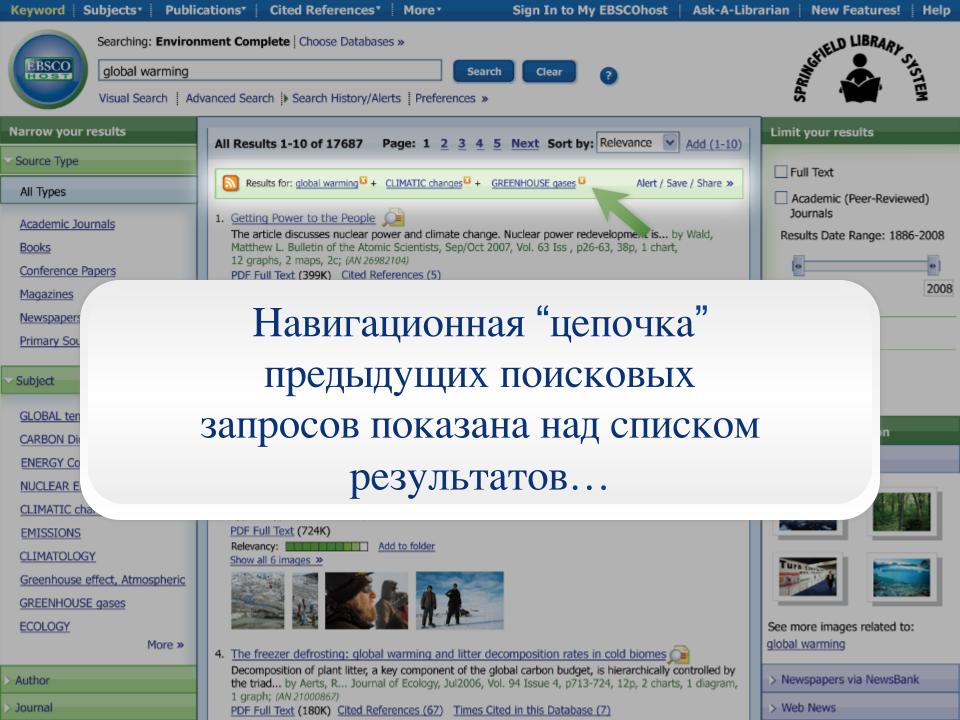
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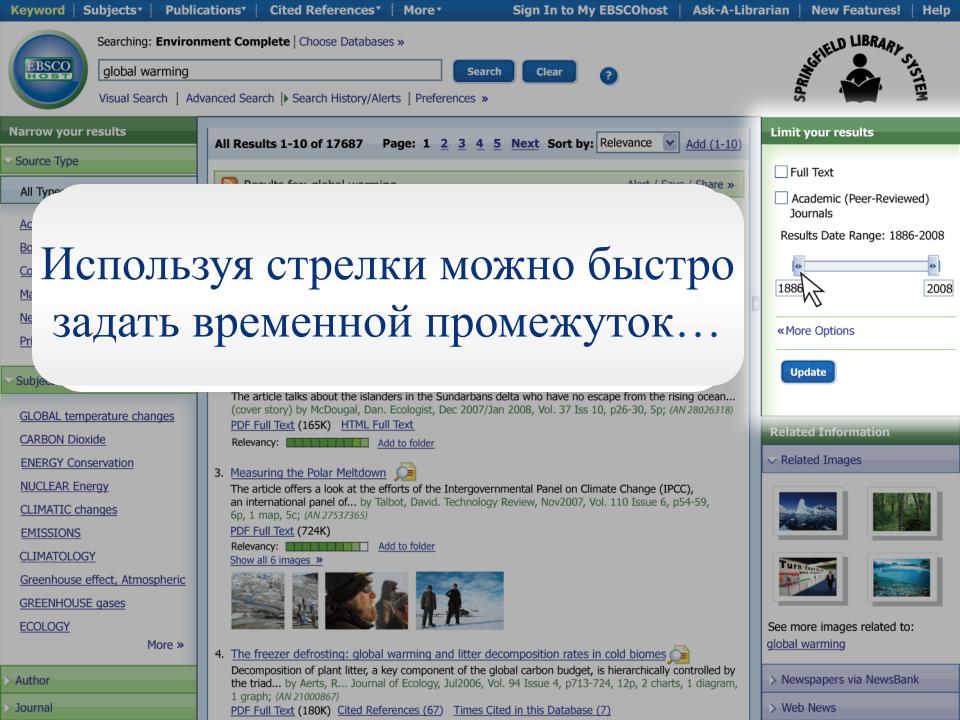


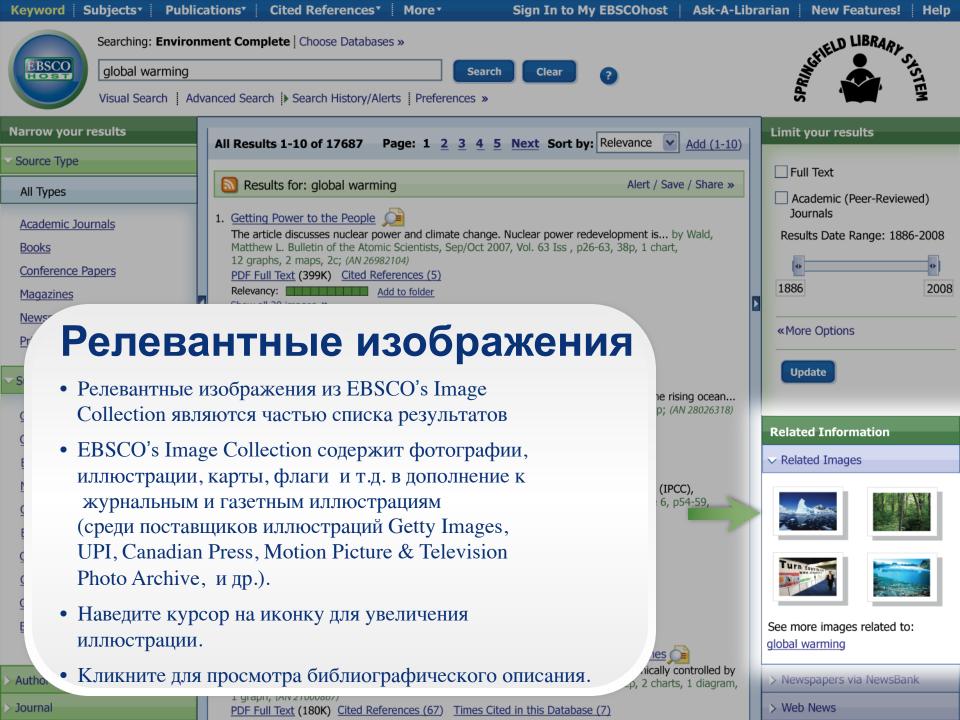
















PDF- изображения полнотекстовых статей

insight overview

The future for stem cell research

Robin Lovell-Badge

Division of Developmental Generics, MRC National Institute for Medical Research, The Rulgewey, Mill Hill, London NW7 1AA, 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

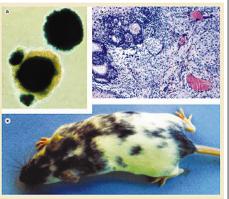
away in the background on stem cell research for years, but it took two breakthroughs animal cloning by Ian Wilmut, Keith Campbell and colleagues¹ and the derivation of phiripotent human embryonic stem (ES) cells by Jamie Thomson and co-workers - to really shake things up. The first showed that an adult cell nucleus can be reprogrammed to produce an entire animal, a dramatic demonstration of hicklen 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

cientists and clinicians have been beavering. 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 chims 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 signal ling events control stem cell differentiation, and what does it mean to reprogramme acell? 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

NATURE | VOL414 | 1 NOVEMBER 2001 | www.nature.com

Figure 1 Plurinotence of mouse embryonic stem ES cells. a, Aggregates of mouse ES cells forming embryoid bodies. The dark staining shows expression of Sor2 in the less differentiated cells, whereas the rind of differentiated encloderm is unstained. (Image courtesy of A. Aution), b. Histological section of a tesatocarcinoma derived from mouse ES cells. Many different cell types are found, all formed from the ES calls, including representatives of all frace gamlayers. Image courtesy of M. Parsons J c. The ultimate test of pluripotentiality: a chimaera made by injecting ES cells into a blastocyst. The pigmentoclarens reveal the contribution of ES cell derivatives to the skin, but all tissues are composed of a minture of ES and host embryo derivatives. Even a single ES cell can give a chimacra like this.



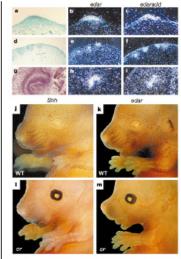
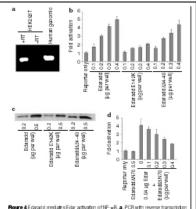


Figure 3 Co-expression of exter and exteredd, a-ii. Sorial sections of E12.5 skin (a-c). E14.5 skin (d-fl) and E15.5 tooth (g-fl) either stained with malachite green (a, d) or haemotrovin (a), or hybridized to edar (b, e, b) or edapadd (c, f, ii) ripporobes, edar and educated are coressed in epithelial cells in developing whisker hair to likes (a-fiand in the enamel knot of the developing tooth (g-i), j-m, Whole-mount Joseph hybridization to E15.5 embryos. Str (1.1) and edut (k, m) are expressed in whister follows on the shout and in hair follide precursors on the head and trunk of wild type embryos ii. kii. No hair follicle induction is detected at this age in the or mutant (ii. mi). The or mutant has black eye sas it is on a pigmented genetic background.

(Fig. 4a), and that its overexpression activates an NF-κB reporter gene in a dose-dependent manner (Fig. 4h, c). The Glu142→Lys mutation reduces the activation of NF-xB by Edaradd (Fig. 4b); however, removing the Traf-binding motif has little effect on NF-KB activation (Fig. 4b), suggesting that Traf recruitment is not crucial for this function. Edaradd signalling was abolished by deletion of its N-terminal 70 amino acids (Edaradd N70) (Fig. 4d). When coexpressed with full-length Edar, Edar add N70 interferes with the receptor's ability to activate NF-xB (Fig. 4d), presumably acting as a dominant-negative inhibitor. That Edaradd AN70 can block the activation of NF-KB by Edar illustrates Edaradd's central role in Edar signalling, and also suggests that it has a modular structure10 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 signaltransducing complex. This linear pathway explains the identical phenotypes of the Ta, dl and cr mutants, and also the genetic heterogeneity of human hypohidrotic ectodermal dysplasia. The adapter molecules Tradd and Fadd are central to death receptor



RT) showing EDARADD expression in untransfected HEX293T cells, b. Edaradd overecoression includes an NF-xB lucife rase reporter giene in a dose dependent marme The Glu142→Lvs (E142M mutation exhibits weak stimulation of NE-vB. but Edaradd∆34-40 shows little change in NF-xB activation. The amount of Edaradd expression vector transfected per well is indicated, q. Western blot showing relative expression levels of transfected HA-tagged Edaradd, Edaradd Gly142--- Lys, and Edaradd∆34-40.d, Overexpression of full-length Edar activates NF-x-B, but co-expression of Edaradd ΔNYO blocks this activation. For each co-transfection, 0.04 μ.g. of Edar expression vector was transfected per well. The amount of EdaradidAN70 expression vector used is indicated. Edaradd ANTO alone does not activate NF-xB

signalling^{1,1}, and the molecular identity of Edaradd illustrates the conservation of the archetypal death receptor pathway in a developmental context.

edarated mapping and identification

We obtain ed crunice on a C57/C5H background from The Jackson Lab. GastEi mice were a eif from A. Bradley. Microsstellite specific olisonschotides DTSM644. DTSM657. DESMitT14 and DTSMit216, and YACs were obtained from Research Genetics. We c out cDNA relation as described?

The sequences of cDNA clones were searched against the high throughput genomic ecusing (http://database.using BLAST at the National Center for Biotechnolog mation (http://www.cn.chi.nkm.nih.gov/blast). Matchinghuman BAC sequences wen input into the PGENESH gove prediction program at the Sanger Centre versite (http://goversica.augerac.uk/g//glahtml). The amino-acid sequences of predicted govers were nearth of the protein domains using PFAM v3.2 at the San Diago. Supercomp ster vehilte (http://fps.idiscedu).

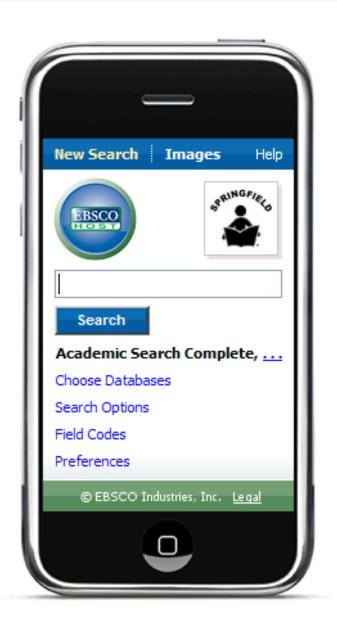
We carried out rapid amplification of cDNA ends using the Generacer procedure (In vitragen) with E17.5 mouse skin cENA as the template Oligonuclantide sequences and PCR conditions are available on request.

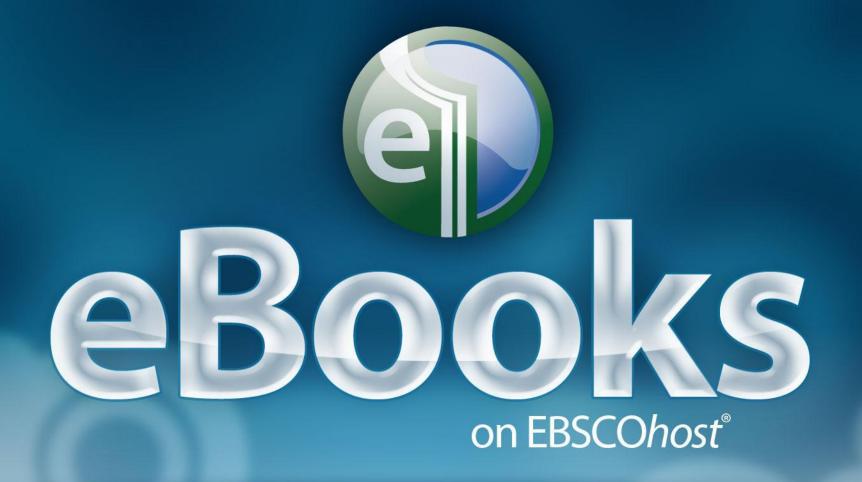
Families with autonomal recenive hypolaidrotic ectoder real dysplasia were recruited into a sewarch study approved by the institutional sevice board of the Oregon Health Sciences University. Coment was obtained for the use of dinical information, relevant family history and DNA amples. We isolated genomic DNA from patients by described methoda". PCR fragments from each of the six exons of EDARADD were amplified and sequenced. Genomic DNA from selevant family members, as well as from control individuals, was analysed for the presence or absence of variants by ASO analysis²⁶ or b



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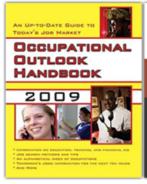


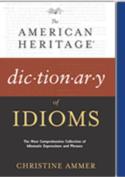
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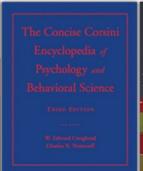
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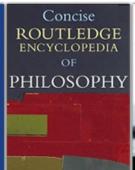
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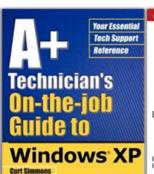


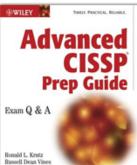










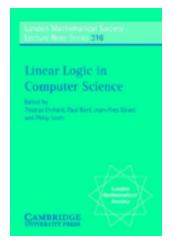


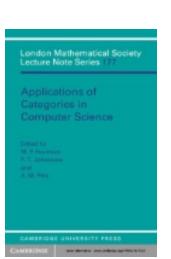
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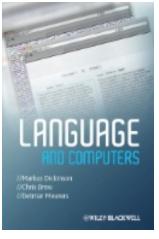
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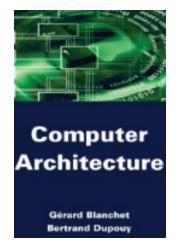


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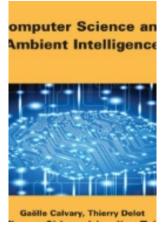






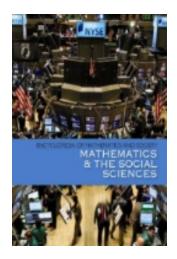


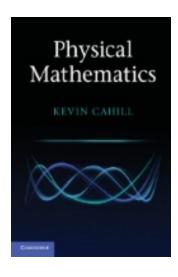


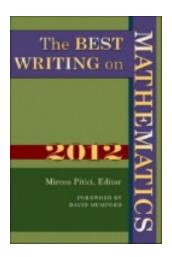


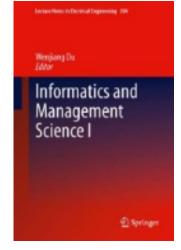


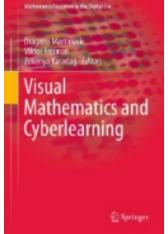
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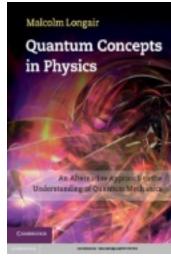




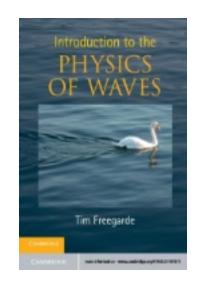


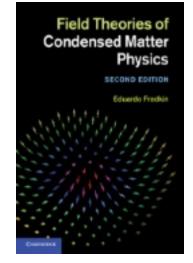


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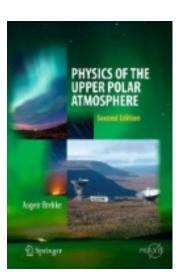






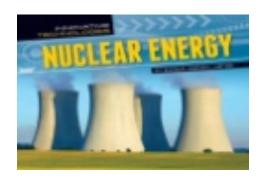


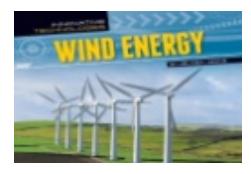




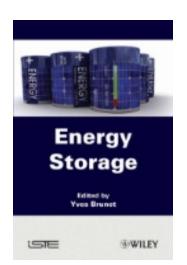


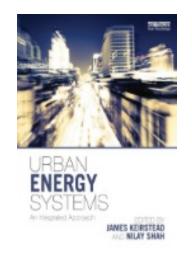
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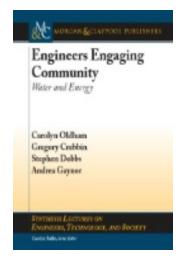






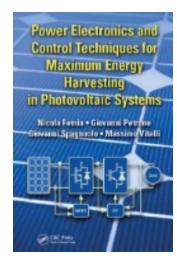


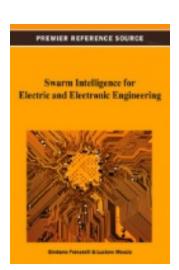


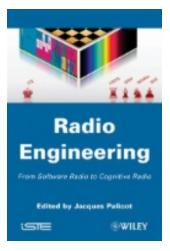




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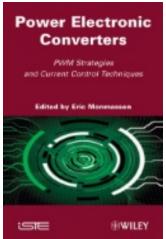












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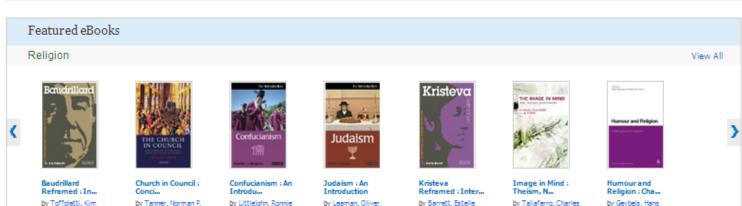
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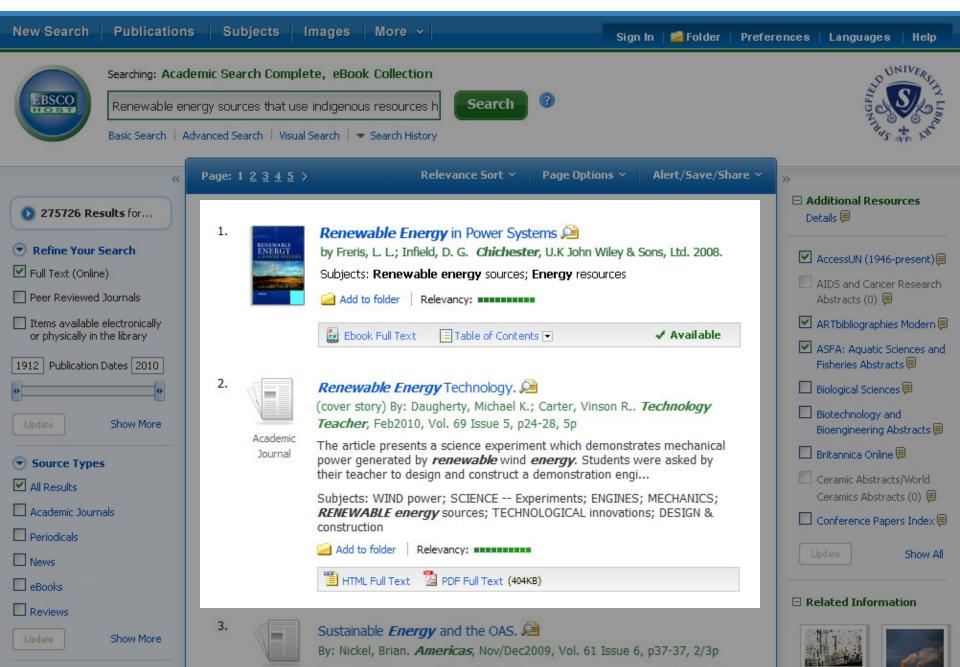
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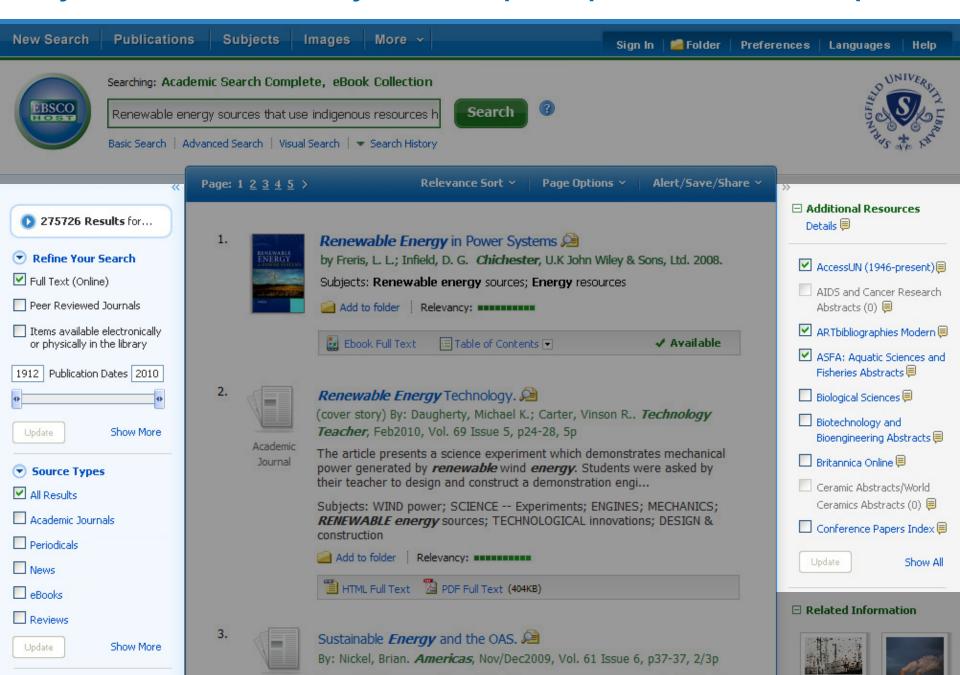




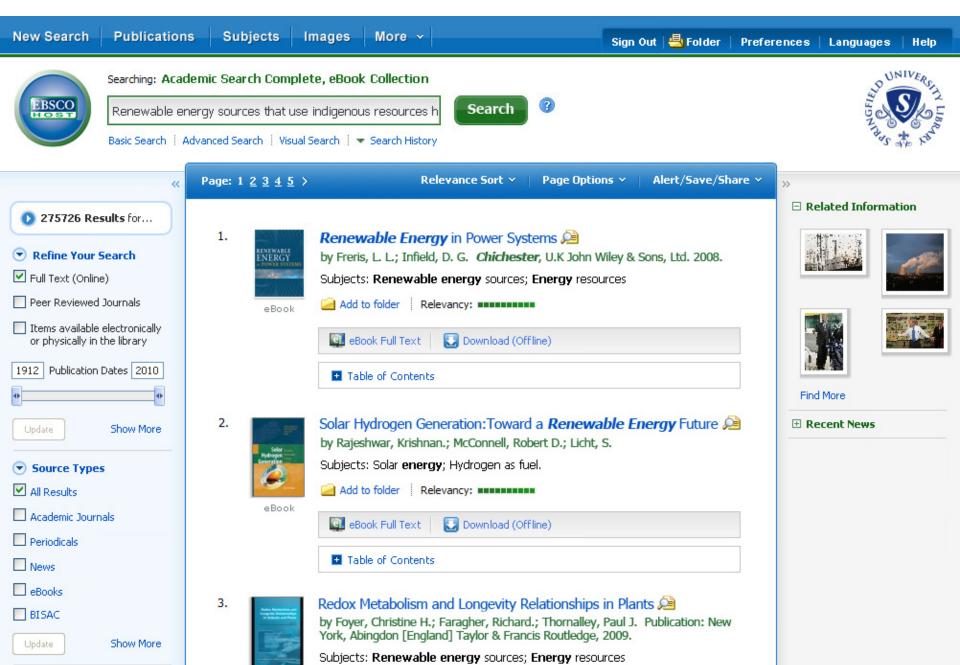
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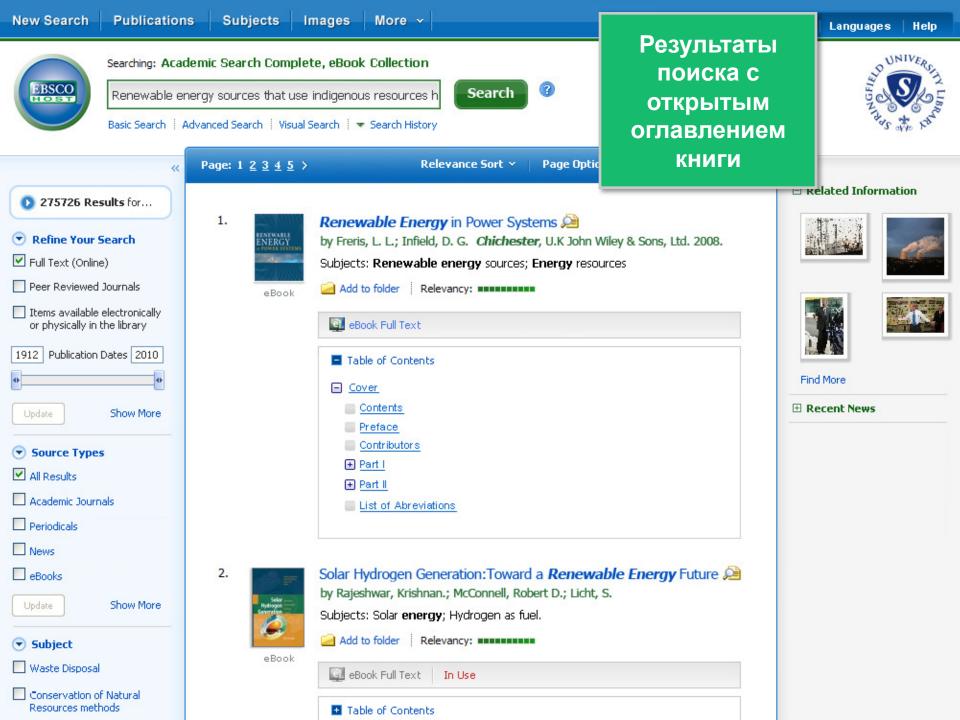


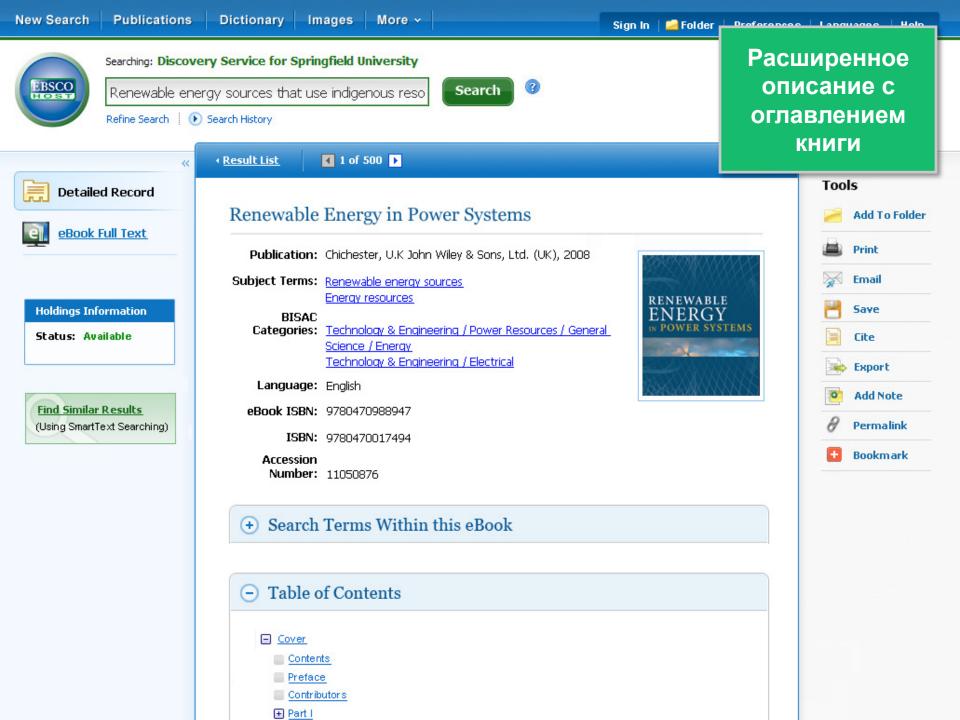
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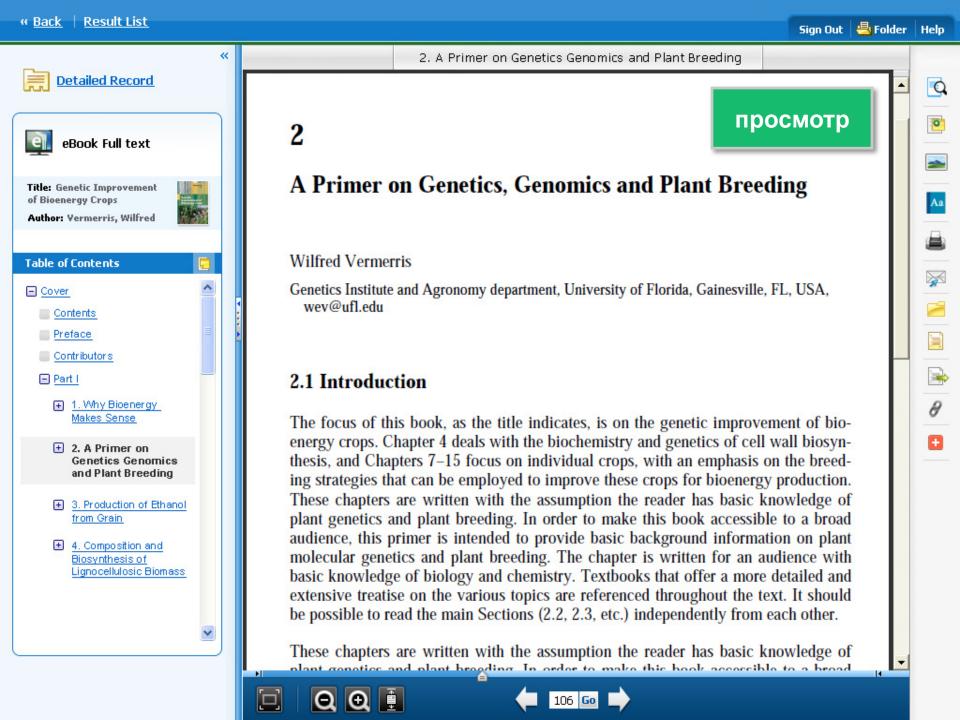


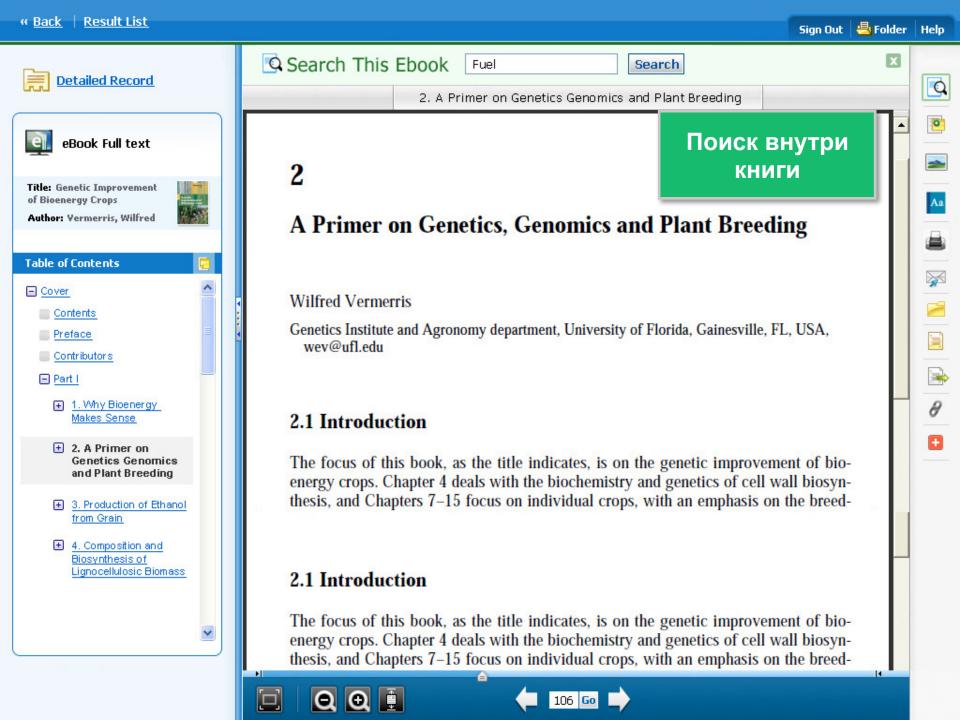
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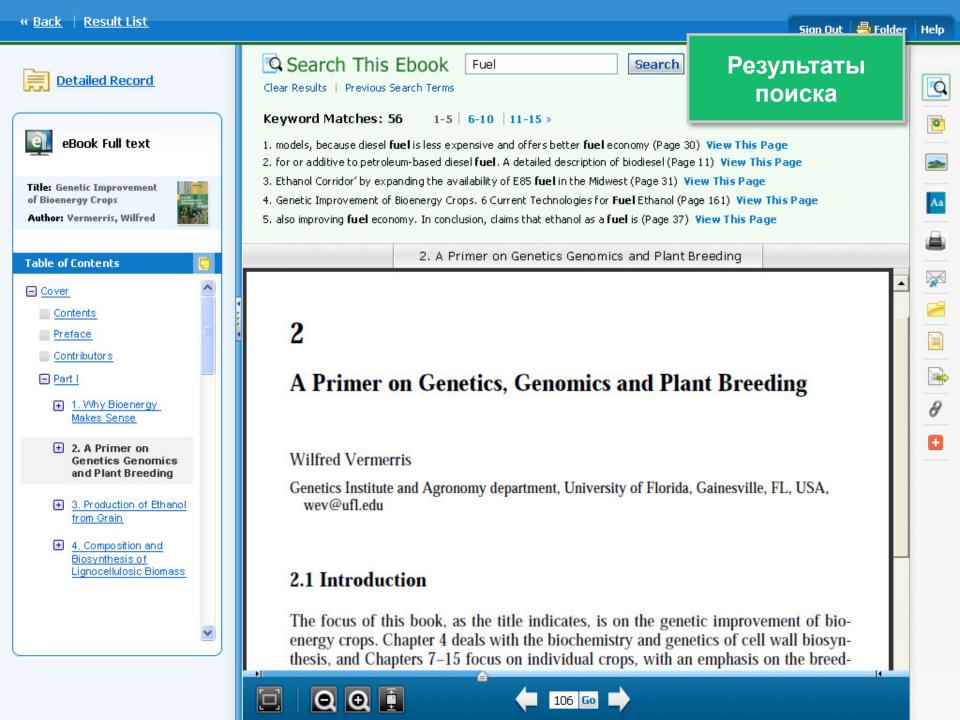


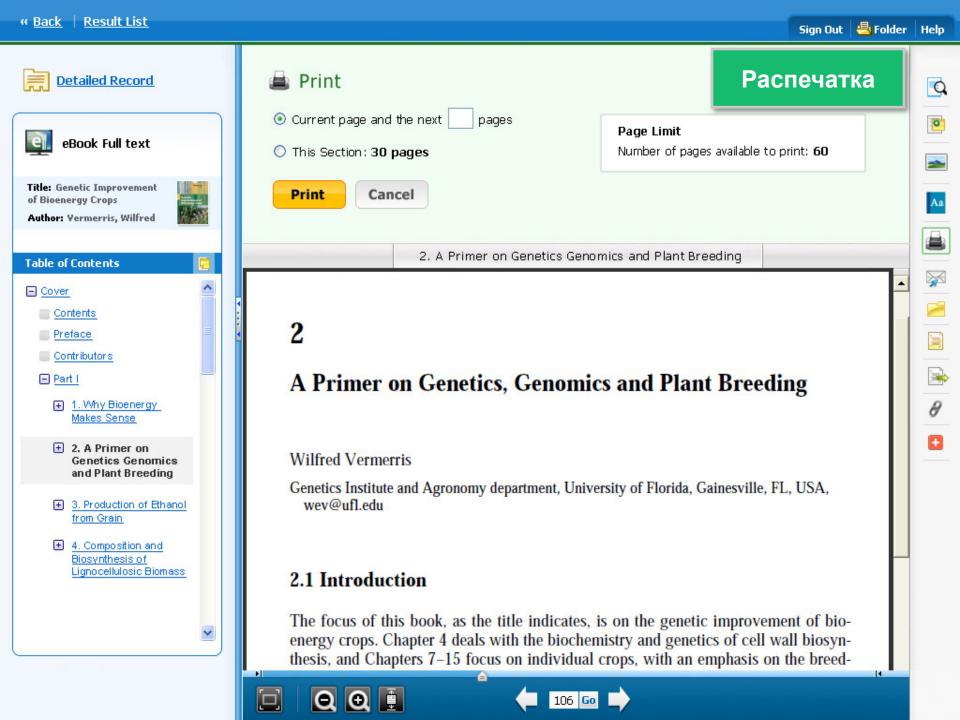


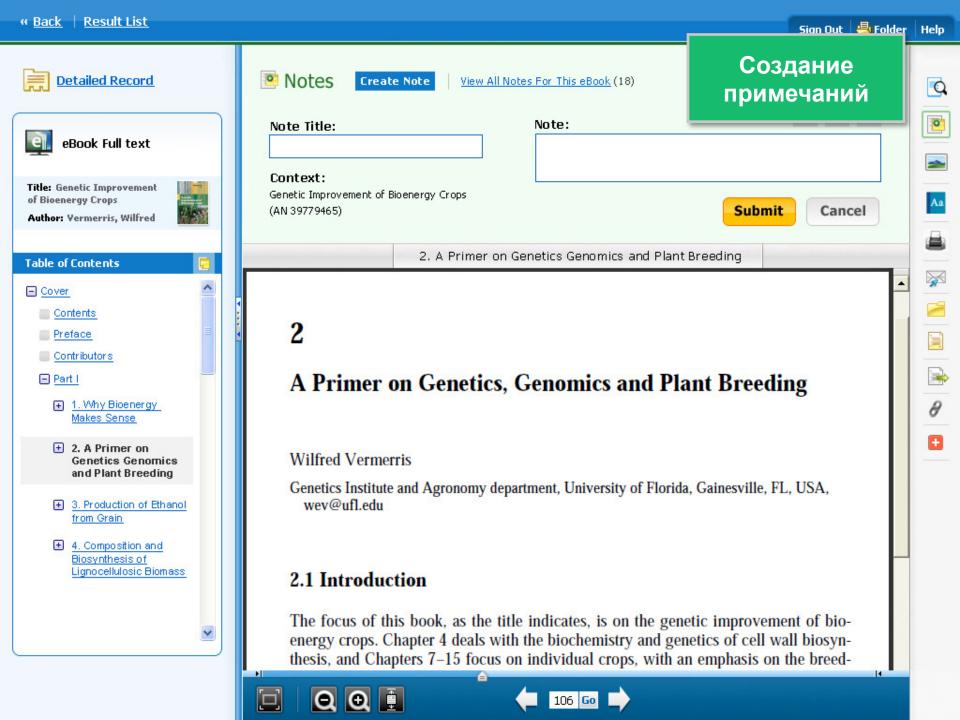


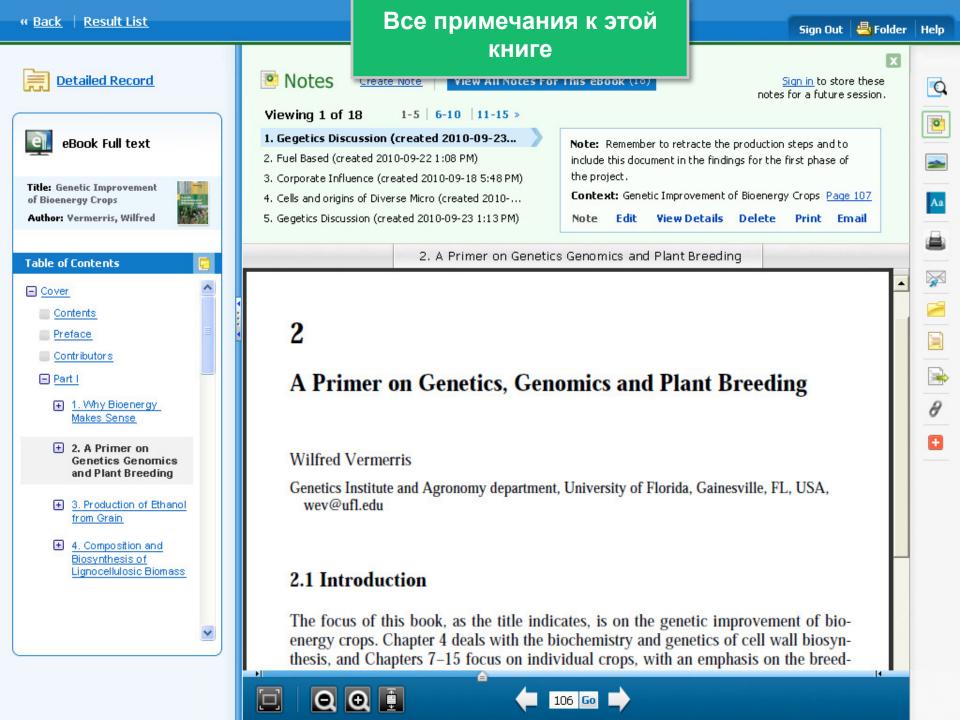


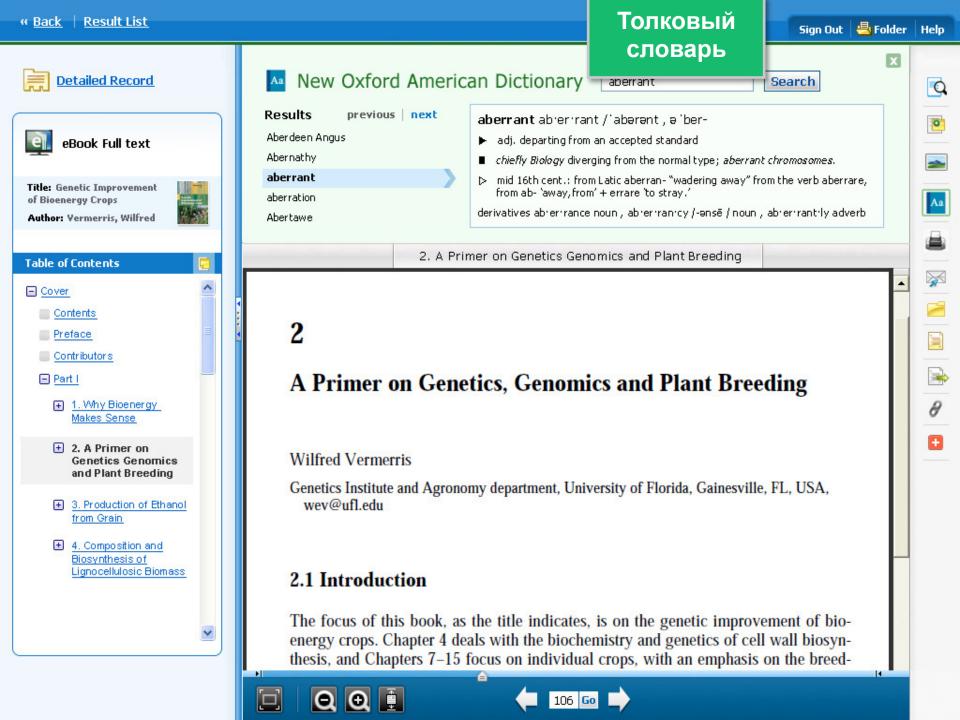












A Primer on Genetics, Genomics and Plant Breeding

Wilfred Vermerris

Genetics Institute and Agronomy department, University of Florida, Gainesville, FL, USA, wev@ufl.edu



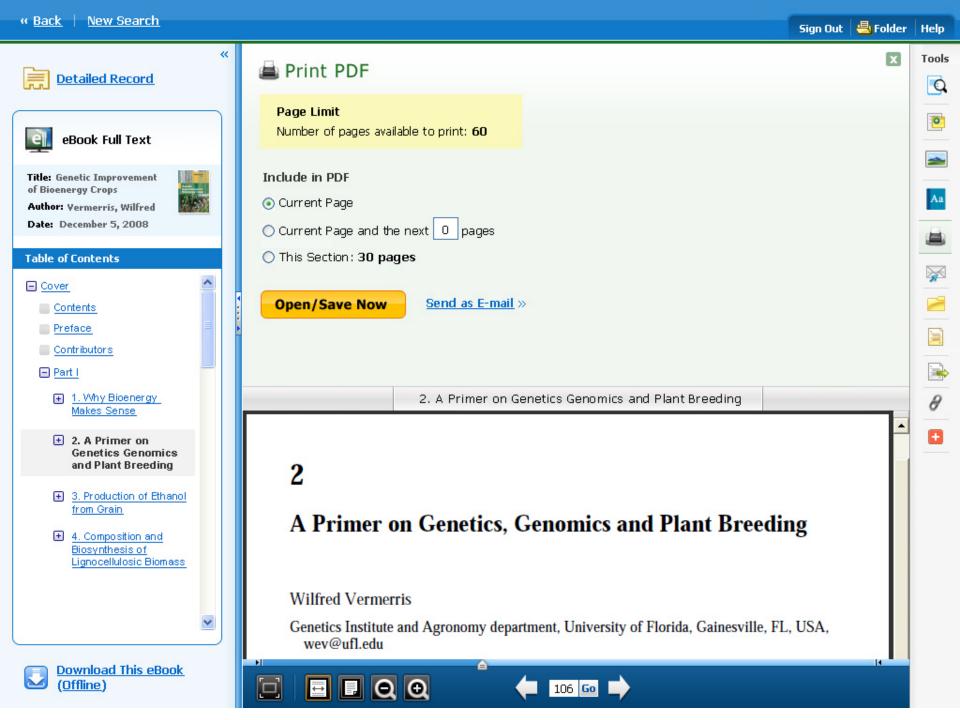












with and emphasis on the breeding strategies that can be employed to improve these crops for bioengineering.

2.1 Introduction

The focus of this book, as the title indicates, is on the genetic improvement of bioenergy crops. Chpater 4 deals with the biochemistry and genetics of cell wall biosynthesis, and chapters 7-15 focus on individual crops, with and emphasis on the breeding strategies that can be employed to improve these crops for bioengineering production. These chapter are written with the assuption the reader has basic knowledge of plant gentetics and plant production.

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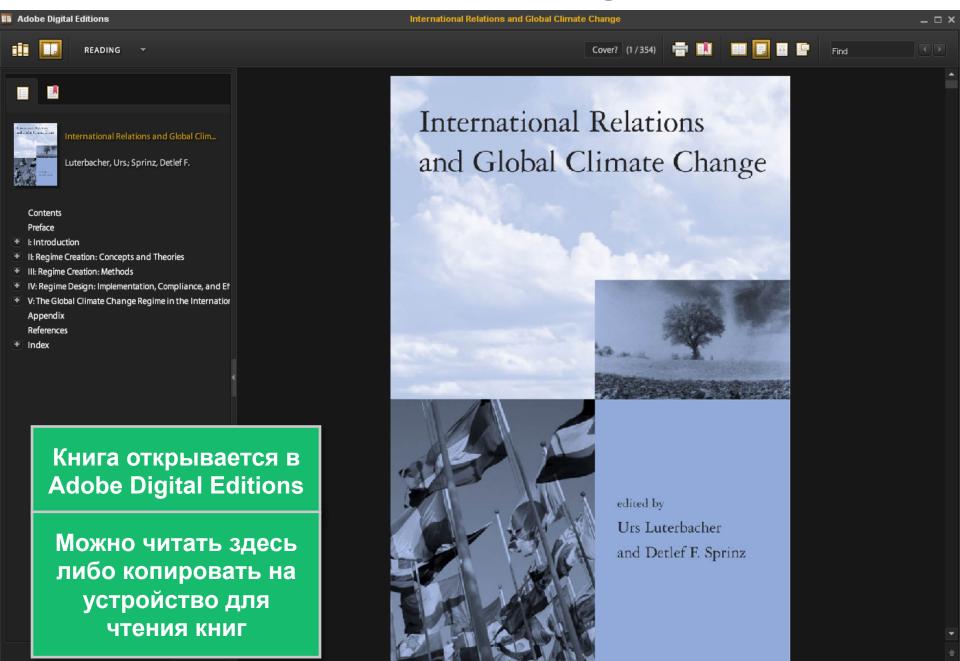


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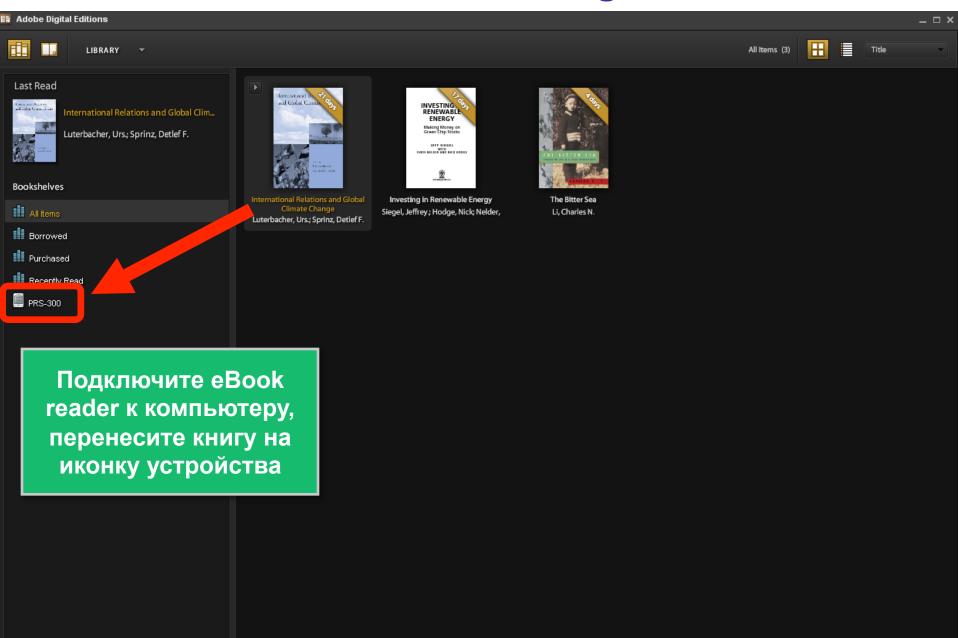


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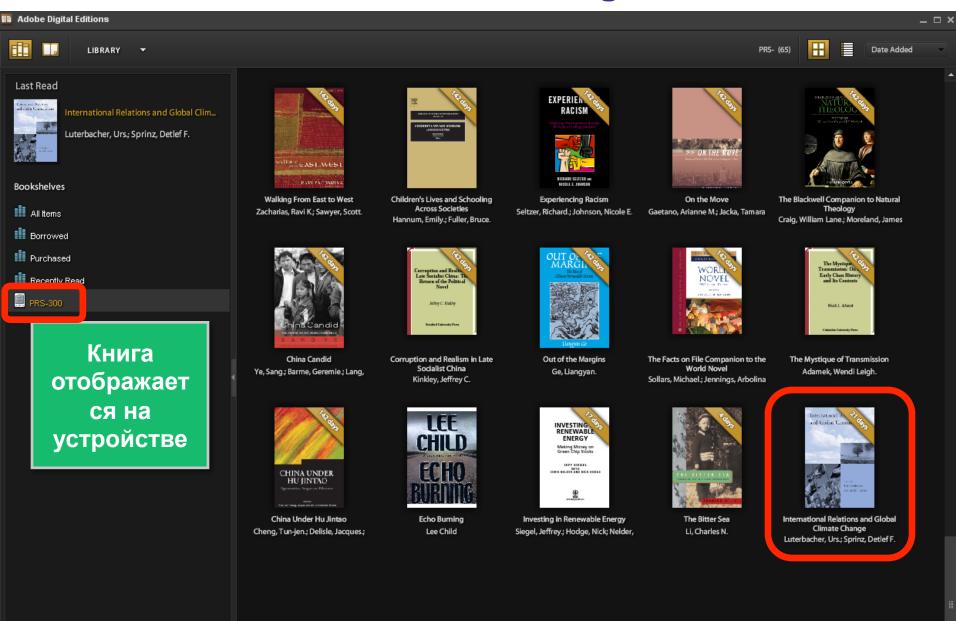
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