spheres. No other differences were observed between samples imaged on aluminum or carbon surfaces.
20. AFM images were obtained with a Digital Instruments Nanoscope IIIa AFM in tapping mode. Samples were deposited onto quartz slides.
21. Monomeric molecules with dimensions of 1 to 2 nm can readily permeate multilayer films [see F. Caruso, E. Donath, H. Möhwald, *J. Phys. Chem. B* **102**, 2011 (1998); R. v. Klitzing and H. Möhwald, *Macromolecules* **29**, 6901 (1996)].
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Auxin-Dependent Cell Expansion Mediated by Overexpressed Auxin-Binding Protein 1

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To test the hypothesis that auxin-binding protein 1 (ABP1) is a receptor controlling auxin-mediated plant cell expansion, *ABP1* complementary DNAs were expressed in a controllable fashion in tobacco plants and constitutively in maize cell lines. Induction of *Arabidopsis ABP1* expression in tobacco leaf strips resulted in an increased capacity for auxin-mediated cell expansion, whereas induction of ABP1 in intact plants resulted in leaves with a normal morphology, but larger cells. Similarly, constitutive expression of maize *ABP1* in maize cell lines conferred on them the capacity to respond to auxin by increasing cell size. These results support a role of ABP1 as an auxin receptor controlling plant growth.

Auxins are plant growth hormones that cause rapid increases in plant cell wall extensibility, alter ion flux at the plasma membrane, and cause specific changes in gene expression (*1*, *2*). A receptor mediating these effects has not been unequivocally identified, although ABP1 is a leading candidate for at least the plasma membrane and wall effects (*1*). ABP1 has been carefully characterized with regard to its auxin-binding properties (*3*). Moreover, it has been shown to fit the criteria of a receptor mediating auxin-induced cell expansion, on the basis of its expected tissue distribution, its plasma membrane location, and on results from experiments designed to antagonize or mimic auxin action using antibodies to ABP1 and an ABP1 peptide mimetic (*4*, *5*). These results, while consistent with ABP1 receptor function, are nonetheless indirect. Therefore, we took a molecular genetic approach by examining the effect of inducible overexpression of *ABP1* in plants and constitutive expression in cell lines.

Tobacco plants expressing the tetracycline repressor were transformed with the full-length, *Arabidopsis ABP1* cDNA placed under the control of a tetracycline promoter (*6*). The inducible expression of *ABP1* in these plants is shown in Fig. 1A. One transformant designated MJ10B was used to further characterize the induction kinetics and dose dependence of induction (Fig. 1, B and C). Steady-state transcript is detectable in uninduced transformants and is shown to greatly increase within 6 hours after application of anhydrotetracycline (AhTet). *Arabidopsis* ABP1 protein (*7*) is detected by immunoblot analysis in uninduced MJ10B transformants (homozygous for ABP transgene) and increases significantly during the 48 hours after induction (Fig. 1D).

Having established the transgene induction kinetics and expression characteristics, we began our analysis of the physiological effect of ectopic expression of ABP1 in leaves, because previous work found that ABP1 mediates auxin-induced hyperpolarization in mesophyll protoplasts. The capacity of leaf cells to respond to auxin is developmentally acquired (*8*). Cells in the tips of young leaves respond to auxin by ethylene-independent expansion, whereas cells in the base of the lamina do not acquire this capacity until much later as the basal lamina expands. We therefore chose to examine the effect of controlled expression of *ABP1* in cells of the basal region of the lamina at a time point when they are not normally auxin responsive. Interveinal lamina tissue taken from the basal region of ABP1 transformants exhibited AhTet-

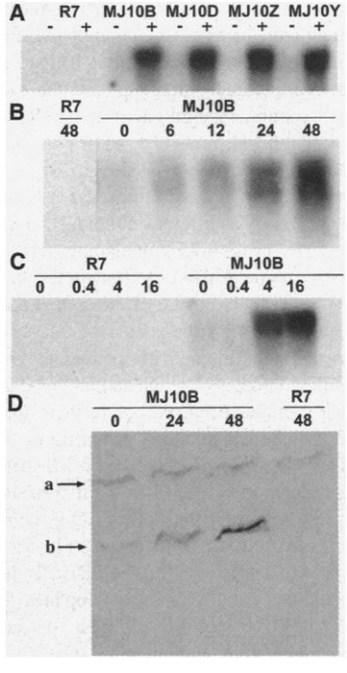

Fig. 1. Anhydrotetracycline-inducible expression of *ABP1* in tobacco. (**A**) Northern blot analysis of *Arabidopsis ABP1* transcript level in total RNA (15 μg) isolated from all leaves of 40-day-old (3 to 4 leaf stage) control plants (R7, expressing the tetracycline repressor) and four independently transformed tobacco plants (MJ10B, MJ10D, MJ10Z, MJ10Y) expressing both the tetracycline repressor and the tet-inducible *Arabidopsis ABP1* construct. Plants were hydroponically fed 12 μg/ml of the nontoxic tetracycline analog AhTet via the roots for 48 hours (+) or left untreated (–) on the lab bench under 10/14 light/dark cycles at 25°C. (**B**) Northern blot analysis of the *Arabidopsis ABP1* transcript level in total RNA from R7 and MJ10B tobacco plants after the indicated number of hours in the presence of 12 μg/ml of AhTet. (**C**) Northern blot analysis of the *Arabidopsis ABP1* transcript level in total RNA from R7 and MJ10B tobacco plants after 48 hours treatment with AhTet at the indicated concentrations (micrograms per milliliter). (**D**) Protein immunoblot analysis of *Arabidopsis* ABP1 in crude microsomal fractions from R7 and MJ10B tobacco plants after the indicated hours of treatment with 4 μg/ml of AhTet. MJ10B plants are homozygous for the *Arabidopsis ABP1* transgene. Polyclonal antisera directed against recombinant *Arabidopsis* ABP1 were used. Note that this serum does not recognize tobacco ABP1 (lane R7, 48 hours). Band "b" represents the *Arabidopsis* ABP1. The nonspecific recognition of band "a" shows equal loading between the samples.

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inducible epinastic growth (reported as leaf curvature) that is significantly higher than the basal growth of control tissues from R7 leaves (Fig. 2). This inducible growth is strictly dependent on the presence of auxin, as expected for any receptor mediating the ligand response. The structurally similar nonauxin, 2-naphthaleneacetic acid (2-NAA), was ineffective, indicating structural specificity is required for inducible growth. The increased auxin-regulated growth in *ABP1* transgenic plants without induction with AhTet (Fig. 2A; MJ10B and MJ10Y, gray bars) is most likely due to leaky basal expression of *ABP1* (Fig. 1D, lane 0). The consistency of the effect of *ABP1* overexpression is seen in Fig. 2B, which shows AhTet-inducible growth in an additional five independent transformants.

To determine if overexpression of *ABP1* alters the spatial pattern of auxin-regulated growth in young leaves described above, we measured growth of the leaf at different positions within lamina of control and *ABP1*-overexpressing leaves (Fig. 2C). Auxin-induced growth of the control leaf occurs in the tip only, whereas AhTet-treated MJ10B leaves exhibit growth at all three positions of the young leaf (Fig. 2D). This effect was dependent on induction of *ABP1* expression with AhTet. Cross sections of the strips show that this increase in growth is due to increased cell expansion, most notably by the palisade mesophyll cells (Fig. 2E).

Plants transformed with the *ABP1* transgene did not have an altered phenotype when treated with AhTet (Fig. 3, A and B). Furthermore, the rate of expansion of leaves on plants treated with AhTet did not differ from the control R7, AhTet-treated plants. However, despite normal morphology, cells from mature leaves of MJ10B plants treated with AhTet are larger. Plants were fed 4 μg/ml AhTet via roots for a period greater than a plastochron, then protoplasts of interveinal cells located in the midsection of the youngest, fully expanded leaves were prepared and analyzed (*9*). The distribution of protoplast volumes of cells from control plants and MJ10B plants not treated with AhTet were similar. In contrast, the protoplast volume of cells in AhTet-treated MJ10B plants was greater (Fig. 3), evident both in a shift toward the largest class of protoplasts and the average protoplast volume. These results indicate that ABP1 mediates auxin-dependent cell expansion in the intact plant. Furthermore, these results are consistent with the intriguing observation that the basic unit of morphology is not the cell, because leaves having the same morphology and size can be produced by fewer but larger cells (*10*).

Successful studies on receptor function in animal cells have used ectopic expression of the receptor in cell lines that otherwise do not express the receptor. In such an approach, the expressed receptor often confers to the cells a hormone-mediated response. Therefore, as a second test of the hypothesis that ABP1 is a functional auxin receptor, we ectopically expressed maize *ABP1* in a clonal maize endosperm cell line that does not contain detectable levels of ABP1 (Fig. 4) and does not have a strict requirement for auxin to proliferate. This enabled us to examine the effect of ABP1 on cell expansion without the complication of high endogenous ABP1 levels; by this approach, we have shown that ectopic expression of *ABP1* confers on these cells an auxin-dependent response.

Maize cells were cotransformed with a CaMV 35*S*:maize *ABP1* construct and the *bar* gene (*11*). Immunoblot analysis using several different antibodies to ABP1 (*12*) showed that cell lines F652 and F631 overexpress *ABP1* (Fig. 4), whereas ABP1 in control cells (C101) was not detectable.

F652 and F631 cells grown in the presence of auxin and expressing detectable levels of ABP1 are significantly larger than the C101 control cells of the same age (Table 1). Ploidy differences between F652, F631, and C101 cannot account for the observed size differences (*13*). The increased cell size of the *ABP1*-overexpressing lines (designated cell size class "c") is not neomorphic, because this phenotype is observed in the control cells, albeit at low frequency (Fig. 5A). This means that these clonal endosperm cells are competent to expand to the class "c" cell size, but only do so infrequently. In contrast, F652 and F631 lines are predominantly larger class "c" cells, suggesting that ABP1, through its normal action, shifts the growth capacity of endosperm cells toward the formation of larger cells.

The most important observation supporting auxin receptor function is that expression of *ABP1* in maize endosperm cells confers on these cells an auxin-dependent response. Figure 5B illustrates that while auxin had no effect on cell size in the control cell line (cell

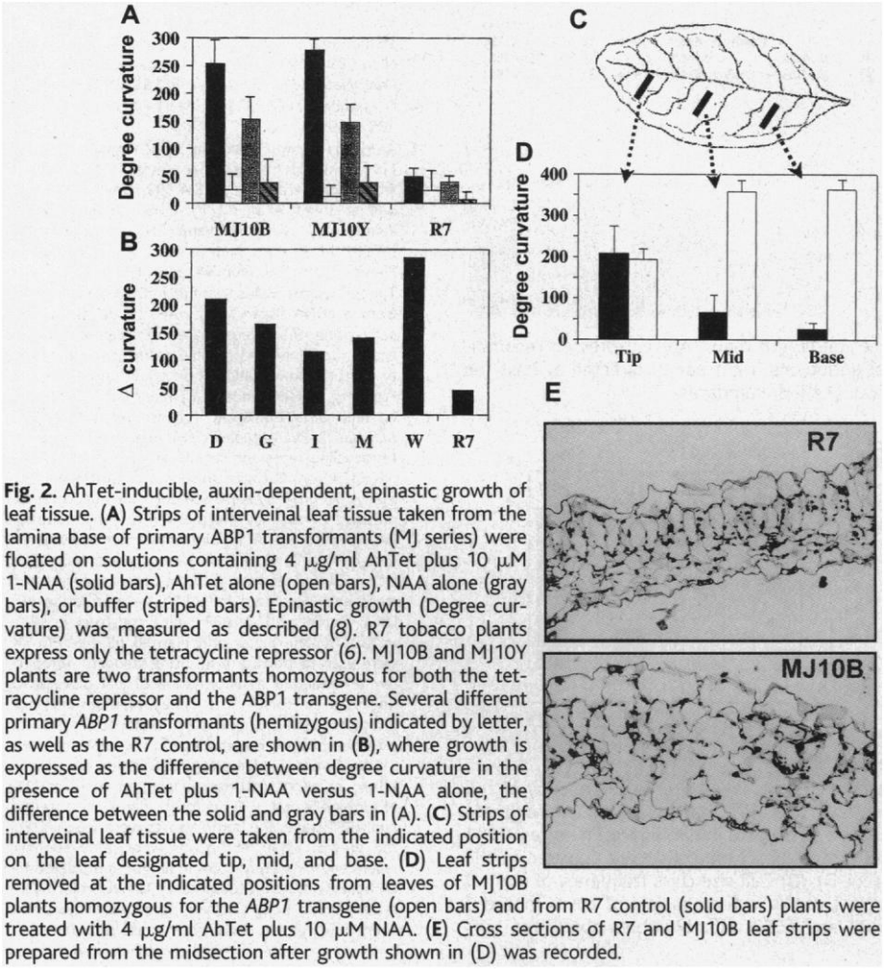

Fig. 2. AhTet-inducible, auxin-dependent, epinastic growth of leaf tissue. (**A**) Strips of interveinal leaf tissue taken from the lamina base of primary ABP1 transformants (MJ series) were floated on solutions containing 4 μg/ml AhTet plus 10 μM 1-NAA (solid bars), AhTet alone (open bars), NAA alone (gray bars), or buffer (striped bars). Epinastic growth (Degree curvature) was measured as described (*8*). R7 tobacco plants express only the tetracycline repressor (*6*). MJ10B and MJ10Y plants are two transformants homozygous for both the tetracycline repressor and the ABP1 transgene. Several different primary *ABP1* transformants (hemizygous) indicated by letter, as well as the R7 control, are shown in (**B**), where growth is expressed as the difference between degree curvature in the presence of AhTet plus 1-NAA versus 1-NAA alone, the difference between the solid and gray bars in (A). (**C**) Strips of interveinal leaf tissue were taken from the indicated position on the leaf designated tip, mid, and base. (**D**) Leaf strips removed at the indicated positions from leaves of MJ10B plants homozygous for the *ABP1* transgene (open bars) and from R7 control (solid bars) plants were treated with 4 μg/ml AhTet plus 10 μM NAA. (**E**) Cross sections of R7 and MJ10B leaf strips were prepared from the midsection after growth shown in (D) was recorded.

Table 1. Maize cells overexpressing ABP1 are larger and contain more cell wall material.

Parameter	C101	F652	F631
Cell diameter (μm)	32	44	54
Cell volume (μm³)	17, 148	44, 549	82, 406
Cell wall (μg/cell)	0.6	1.5	–

size classes "b+c"), the large cell size of cells overexpressing *ABP1* was strongly auxin-dependent. When auxin was removed from the medium, the frequency of cell size class "a" (Fig. 5B) approximated that seen in control lines with or without auxin in the medium. At auxin concentrations twice the normal culture levels, the frequency of cell types "b+c" increased, indicating dose dependency.

Our data demonstrate that an auxin-inducible growth phenotype is conferred on tobacco cells in planta by the AhTet-inducible *ABP1* construct, and in cultured maize cells by expression of a *35S:ABP1* construct. This molecular genetic evidence, taken together with the extensive characterization of ABP1 auxin-binding properties, unequivocally demonstrates that ABP1 is an auxin receptor that controls cell expansion. Considering the complexity of auxin action even in a single response such as cell expansion and considering the evidence supporting multiple auxin pathways, it is likely that additional auxin receptors yet to be identified are involved in auxin perception and cell expansion.

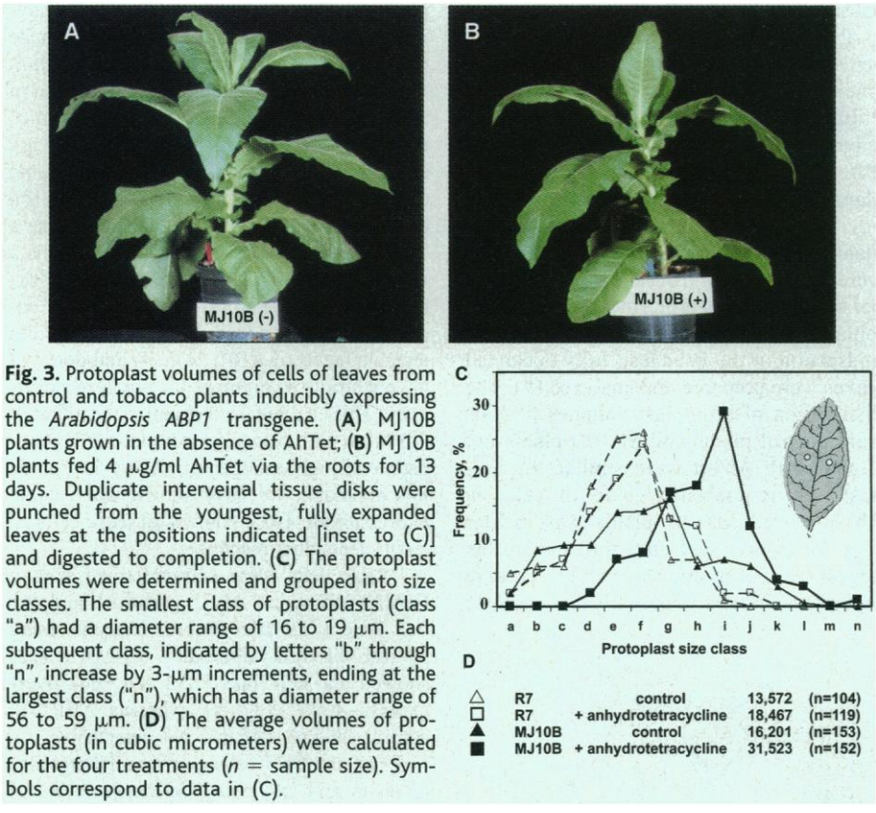

Fig. 3. Protoplast volumes of cells of leaves from control and tobacco plants inducibly expressing the *Arabidopsis ABP1* transgene. (**A**) MJ10B plants grown in the absence of AhTet; (**B**) MJ10B plants fed 4 μg/ml AhTet via the roots for 13 days. Duplicate interveinal tissue disks were punched from the youngest, fully expanded leaves at the positions indicated [inset to (C)] and digested to completion. (**C**) The protoplast volumes were determined and grouped into size classes. The smallest class of protoplasts (class "a") had a diameter range of 16 to 19 μm. Each subsequent class, indicated by letters "b" through "n", increase by 3-μm increments, ending at the largest class ("n"), which has a diameter range of 56 to 59 μm. (**D**) The average volumes of protoplasts (in cubic micrometers) were calculated for the four treatments (n = sample size). Symbols correspond to data in (C).

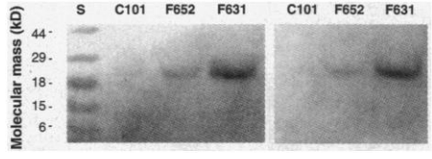

Fig. 4. ABP1 expression and growth of transformed maize cells. Immunoblot analysis of crude extracts of equal fresh weight amount of maize cells transformed with CaMV *35S:ABP1* and CaMV 35*S:Bar* (Cell lines F652 and F631) compared to a control line transformed with *35S:Bar* alone (C101). Immunoblots were probed with a polyclonal serum (NC13) directed against the full-length maize ABP1 expressed recombinantly in *Escherichia coli* (left panel) or monoclonal antibodies (right panel) directed against the purified maize ABP1 [pooled MAC 256, 257, 259, 260; (*14*)]. S, standards.

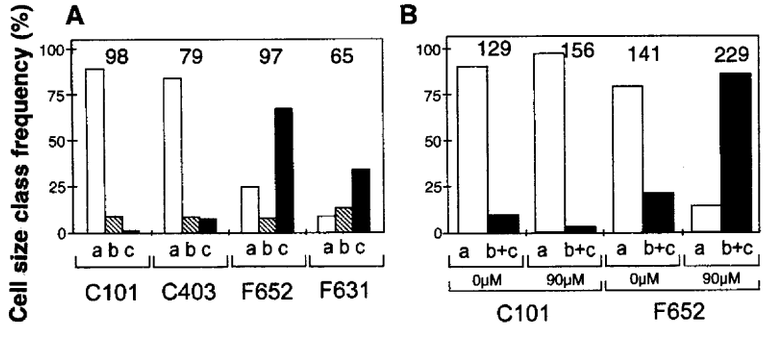

Fig. 5. Auxin-dependent phenotype conferred on cells overexpressing *ABP1*. (**A**) Frequency of cell size classes "a" (open bars; approximate diameter, 20 μm), "b" (hatched bars; approximate diameter, 30 μm), and "c" (solid bars; approximate diameter, 50 μm) in two control cultures, C101 and C403, and two ABP1-overexpressing lines, F652 and F631. These cells were cultured in the presence of 45 μM 2,4-dichlorophenoxyacetic acid (2,4-D). (**B**) Cell size class frequency of control (C101) and ABP1-overexpressing (F652) cells cultured 4 weeks in the absence of auxin (noted 0 μM) and in the presence of 2,4-D (90 μM) for C101 and F652 cells. Numbers above the bars indicate the cell sample size.

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6. The full-length *Arabidopsis ABP1* cDNA was amplified from a cDNA library, confirmed to be genuine by sequencing, then placed in transcriptional frame to the tetracycline-derepressible promoter on the binary vector pBin-HYGRO-TX described by C. Gatz, C. Frohberg, and R. Wendenburg [*Plant J.* **2**, 397 (1992)]. F_1 seed of a transgenic tobacco plant (*Nicotiana tabacum* L. cv. Wisconsin 38) that overexpress the tetracycline repressor structural gene (tet^{R+}) were obtained from C. Gatz (University of Gottingen, Germany). F_1 progeny were screened for high-level expression of tet^R by Northern (RNA) hybridization (M. A. Savka and A. N. Binns, data not shown); one designated R7, which had the highest levels of Tet^R transcript and was shown to be homozygous for this transgene, was used for ABP1 transformations. T-DNA vectors pMJ10 (ABP1 gene construct in pBin-HYGO-TX) and vector-only plasmid, pBin-HYGO-TX, were used to transform R7 using standard *Agrobacterium*-mediated transformation of leaf disks [S. G. Rogers, R. B. Horsch, R. T. Fraley, *Methods Enzymol.* **118**, 627 (1988)]. Shoots that developed roots in the presence of both kanamycin and hygromycin within 14 days were considered transgenic and were maintained as shoot cuttings on plantlet medium (1/2 Murashige-Skoog, 1% sucrose). Plants were selfed, and ABP1 transgene homozygotes were selected. For the growth assay, leaf strips were removed from leaves approximately 5 cm in length and treated for 24 hours with AhTet followed by 18 hours of 1-naphthaleneacetic acid (1-NAA), then were measured. Plants were grown under greenhouse conditions.
7. Antiserum directed against recombinant ABP1 was

generously provided by K. Palme, Max Planck Institute, Cologne, Germany.

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9. Leaf cell volume was indirectly measured as described by A. Hemerley *et al.* (*10*). R7 and MJ10B plants having approximately four fully expanded leaves were grown under normal greenhouse conditions and watered daily with 0.1% Peter's solution (Peter's Professional, Marysville, OH) plus or minus 4 μg/ml AhTet for 13 days, a period in excess of one plastochron. The youngest, fully expanded leaf from each plant was harvested, and a 0.5-mm disk of interveinal leaf tissue was punched from the midsection of the leaf (see inset of Fig. 3) using a #2 cork borer. The cell walls of these tissues were digested for 10 hours in 25 mM MES (pH 5.6), 1/2 strength MS and Gamborg vitamins, 0.4 M sucrose, 1% cellulase (RS), and 0.5% macerase (R10). Just at this point, 75% of cells were released from the tissue and assumed a spherical shape. Yields for protoplasts were the same for all treatments. Protoplasts were photographed and the diameters determined.
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11. The maize *ABP1* cDNA described by U. Tillmann *et al.* [*EMBO J.* **8**, 2463 (1989)] was placed in transcriptional frame with the 35*S* Cauliflower Mosaic viral (35*S* CaMV) promoter to form vector pAJ15. Linear DNA from pAJ15 containing only the 35*S*: ABP1 construction was cotransformed with linear DNA containing the same promoter driving the bar gene of *Streptomyces hygroscopicus* encoding phosphothricin acetyltransferase into maize protoplasts as described by R. Shillito *et al.* [*Biotechnology* **7**, 581 (1989)]. Primary selection and screens of transformants was as described by C. Kramer, J. DiMao, G. K. Carswell, and R. D. Shillito [*Planta* **190**, 454 (1993)]. Calli surviving on plates containing phosphothricin were then subjected to a polymerase chain reaction–based screen using ABP1-specific primers. Cells confirmed to be transformed were then subjected to immunoblot analyses using antibodies to maize ABP1. Cells were maintained on solid and in liquid 2N6 medium [C. C. Chu *et al.*, *Sci. Sin.* **18**, 659 (1975)].
12. Cells (0.2 g) were collected by filtration and extracted using a 3% SDS-containing buffer. Extraction and immunoblot analysis was performed as described (*4*).
13. Cells were digested overnight in 5% cellulase (RS) and macerase (R10) at 25°C with constant agitation to produce individual cells and cells in small aggregates. The nuclei were stained with 4′6′-diamidino-2-phenylindole, and the fluorescence intensity of individual nuclei was measured using a microphotometer.
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15. Supported, in part, by NSF (MCB-9514306) and USDA NCRICGO (9602846) to A.M.J., and by NIH (GM47369-06) to A.N.B. Special thanks to R. Napier, AFRC in UK, for providing monoclonal antibodies to maize ABP1 and S. Whitfield for assistance in preparing the illustrations.

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Large-Scale Sprouting of Cortical Connections After Peripheral Injury in Adult Macaque Monkeys

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Distributions of thalamic and cortical connections were investigated in four macaque monkeys with long-standing, accidental trauma to a forelimb, to determine whether the growth of new connections plays a role in the reorganization of somatosensory cortex that occurs after major alterations in peripheral somatosensory inputs. In each monkey, microelectrode recordings of cortical areas 3b and 1 demonstrated massive reorganizations of the cortex related to the affected limb. Injections of tracers in area 1 of these monkeys revealed normal patterns of thalamocortical connections, but markedly expanded lateral connections in areas 3b and 1. Thus, the growth of intracortical but not thalamocortical connections could account for much of the reorganization of the sensory maps in cortex.

The reorganization of somatosensory cortex that has been observed in monkeys with forelimb amputation (*1*) or sensory deafferentation (*2*), and in human amputees (*3, 4*), is presumed to be the basis for the sensation of phantom limbs (*5*) and perhaps phantom pain (*4*). A critical issue is how such large-scale changes are mediated in the adult brain. We previously showed that sprouting of peripheral nerve axons in the brainstem could account for some of the changes in cortical organization after forearm amputation (*1*), but additional mechanisms might be necessary for complete reactivation of deprived cortex. To investigate the possibility that new growth at other levels of the pathway contributes to the cortical reorganization, we studied thalamocortical and corticocortical connections in monkeys that had long-standing injury to the forearm, including arm amputation and wrist fracture. Electrophysiological maps of the cortical forelimb representation in the same monkeys allowed us to relate the patterns of connections to the functional changes produced by the injury.

Small injections of a bidirectional tracer, either wheat germ agglutinin conjugated to horseradish peroxidase (WGA-HRP) or fluoro ruby, were made into somatosensory cortical area 1 of three normal macaque monkeys and four monkeys that had suffered accidental forelimb injuries (*6*) at other primate facilities 1 to 10 years before the terminal experiments were performed (*7*). The injuries resulted in amputation of all the digits on one hand in one monkey, arm amputations in two monkeys and, in a fourth monkey, a wrist fracture that healed with the hand in a ventrally flexed position that rendered it useless (*6*). Although the injuries differed in each of the monkeys, they greatly altered the nature of the effective sensory input to cortex. Thus, these monkeys presented a valuable opportunity to learn about mechanisms of large-scale cortical reorganization that follow major changes in afferent drive.

Injections of similar size and location were placed in the hand representation of the control monkeys and in the reorganized representation of the experimental monkeys (*7*). The locations for the injections were determined from surface landmarks in the normal and injured monkeys and were confirmed by electrophysiological recordings in one of the controls and in all experimental monkeys. Subsequently, extensive electrophysiological recordings were made throughout the deprived zone in cortical areas 3b and 1 of the experimental monkeys. All procedures were performed in accordance with both National Institutes of Health and university guidelines for the care and use of animals in research. This report describes the patterns of label in cortical area 3b (primary somatosensory cortex) and area 1 because we have evidence for reorganization after peripheral injury in these fields. (Labeled neurons and processes also were apparent in other cortical somatosensory areas, including areas 2, 3a, 5, SII, and PV, but it remains uncertain whether or how these connections differ from those in normal animals.)

The distributions of label in areas 3b and 1 in the normal monkeys were similar across animals (Fig. 1). Both in area 1 and in the adjoining somatosensory field, area 3b, clusters of labeled cells and processes were separated from other clusters by zones of little or no labeling (Fig. 1). The label extended across much of the anteroposterior widths of areas 1 and 3b, but this distribution involved limited shifts in representation across the hand, from the palm or the proximal portion of a digit onto the distal digit tips. However, in the mediolateral dimension, where large topographic shifts can occur across the fore-

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