for higher level psychological explanations when lower level ones will do (*20*). A theory of human phonetic categorization may need to be no more (and no less) complex than that required to explain the behavior of these quail.

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4. Blackbirds and pigeons can make the relatively easy discrimination of steady-state vowels [R. D. Heinz, M. B. Sachs, J. M. Sinnott, *J. Acoust. Soc. Am.* **70**, 699 (1981)].
5. Japanese quail are terrestrial birds considerably smaller than the bobwhite quail (adults weigh only about 150 g). Although hearing in *Coturnix* has not been studied, audiometric functions for the three ground-foraging birds for which data exist (see below) indicate close similarities between bird and human audiograms for frequencies below 5 kHz. Vocalizations do not appear to play a large role in Japanese quail social behavior; consequently, one reason to use *Coturnix* is that they presumably lack rigidly specialized responses to acoustic stimuli. Turkey (*Meleagris gallopavo*): V. A. Maiorana and W. M. Schleit, *J. Aud. Res.* **12**, 203 (1972); bobwhite quail (*Colinus virginianus*): L. Barton, E. D. Bailey, R. W. Gatehouse, *ibid.* **24**, 87 (1984); pigeon (*Columba livia*): R. D. Hienz, J. M. Sinnott, J. M. Sachs, *J. Comp. Physiol. Psychol.* **91**, 1365 (1977).
6. Two tokens of each syllable were recorded from a male speaker, low-pass filtered at 4.9 kHz, and digitized at a 10-kHz sampling rate. Amplitude and duration varied across vowels but did not vary systematically among consonants.
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9. Phonetic symbols represent these English vowel sounds:

[i] "heat"	[ʊ] "hood"	[oʸ] "hoist"
[ɪ] "hit"	[oʷ] "hope"	[a] "hot"
[eʸ] "hate"	[æ] "hat"	[ʌ] "hut"
[u] "hoot"	[ɛ] "head"	[ɜ˙] "hurt"

10. Quail were deprived of food 5 to 10 hours before experimental sessions. Each was trained and tested in a standard pigeon operant chamber lined with sound-attenuating foam. Birds pecked a single lighted 4-cm round key 15 cm above the floor and centered between two 9-cm speakers. Stimuli were presented and reinforcement was controlled by a DEC PDP 11/34 computer. On each trial, a single syllable was presented repeatedly once per second at a peak level of 75-dB sound pressure level. The average duration of each trial was 30 seconds, varying geometrically from 10 to 76 seconds. The interval between trials was 15 seconds. Responses to stimuli beginning with [d] were reinforced on a variable interval schedule by a 2-second access to food from a hopper beneath the peck key. The average interval of reinforcement was also 30 seconds (10 to 76 seconds), so that positive stimuli were reinforced on an average of once per trial. When a positive trial was relatively long (56 or 76 seconds) and times to reinforcement were short (10 to 16 seconds), reinforcement was available more than once. Likewise, on shorter positive trials reinforcement was not available at all if time to reinforcement was longer than the trial. Such intermittent reinforcement encouraged consistent peck rates during nonreinforced test trials. Training sessions lasted about 70 to 80 minutes.
11. Asymptotic performance was defined as the largest ratio of pecks per minute to positive versus negative stimuli over a sustained period of at least 1 week. This criterion was reached for bird 716 after 4,240 trials, bird 730 after 8,176 trials, and bird 768 after 12,304 trials.
12. Statistical analyses of peck rates to novel stimuli were carried out separately for each of three birds across eight test sessions. The mean peck rate for 96 presentations of novel positive stimuli (three trials by eight sessions by one consonant [d] by four vowels) was compared with the rate for 96 presentations of novel negative stimuli (three trials by four sessions by two consonants [b,g] by four vowels). Comparisons are significant at $P < 0.001$ (two-tailed t tests) for each of the three birds.
13. Bird 768 began training 2 months later than birds 716 and 730 and finished testing on the first four novel vowels after those birds had completed testing on the second four novel vowels. Given the clarity of the results from birds 716 and 730, bird 768 was not tested on the second set of vowel contexts.
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Chemical Conversion of a DNA-Binding Protein into a Site-Specific Nuclease

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The tryptophan gene (*trp*) repressor of *Escherichia coli* has been converted into a site-specific nuclease by covalently attaching it to the 1,10-phenanthroline–copper complex. In its cuprous form, the coordination complex with hydrogen peroxide as a coreactant cleaves DNA by oxidatively attacking the deoxyribose moiety. The chemistry for the attachment of 1,10-phenanthroline to the *trp* repressor involves modification of lysyl residues with iminothiolane followed by alkylation of the resulting sulfhydryl groups with 5-iodoacetamido-1,10-phenanthroline. The modified *trp* repressor cleaves the operators of *aroH* and *trpEDCBA* upon the addition of cupric ion and thiol in a reaction dependent on the corepressor L-tryptophan. Scission was restricted to the binding site for the repressor, defined by deoxyribonuclease I footprinting. Since DNA-binding proteins have recognition sequences approximately 20 base pairs long, the nucleolytic activities derived from them could be used to isolate long DNA fragments for sequencing or chromosomal mapping.

THE TRANSFORMATION OF DNA-binding proteins into site-specific nucleases provides an approach to the construction of a new family of endonucleases with recognition sequences about three to four times as long as naturally occurring restriction enzymes. Since fewer binding sites for these semisynthetic nucleolytic agents would be present in any genome, they might be useful in chromosomal mapping and the isolation of large DNA fragments for sequencing. In this report, the chemical conversion of the *Escherichia coli trp* repressor (*1*) into a site-specific nuclease is outlined. This was achieved by

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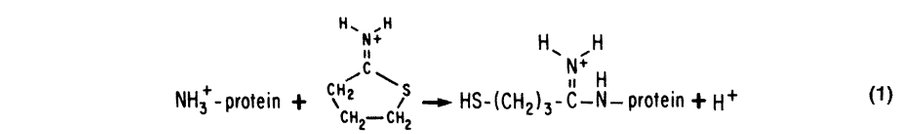

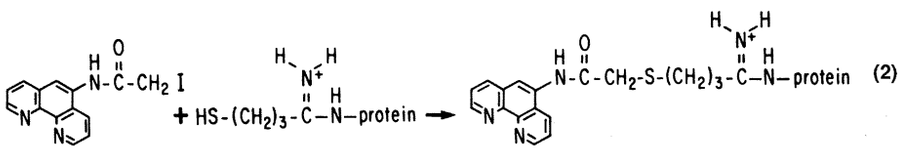

covalently attaching the protein to a 1,10-phenanthroline–copper complex that cleaves DNA by oxidatively degrading the deoxyribose moiety (*2, 3*). Sequence-specific cutting of single-stranded DNA after hybridization to an oligonucleotide linked to the 1,10-phenanthroline–copper complex (*4*) or ferrous EDTA, a hydroxyl radical generator (*5*), has been reported (*6, 7*). An advantage of nucleolytic agents designed with DNA-binding proteins as carriers is that they will recognize double-stranded DNA and not require the denaturation of the substrate to achieve site-specific cutting.

The chemistry of the attachment of the 1,10-phenanthroline (OP) moiety to the protein is summarized in Eqs. 1 and 2. The protein is first modified with 2-iminothiolane hydrochloride (*8*) in the presence of the corepressor L-tryptophan (*9, 10*) (Eq. 1). The sulfhydryl groups generated by the modification of primary amino groups are then alkylated with [^{3}H]5-iodoacetamido-1,10-phenanthroline (*4, 10*) (Eq. 2). The

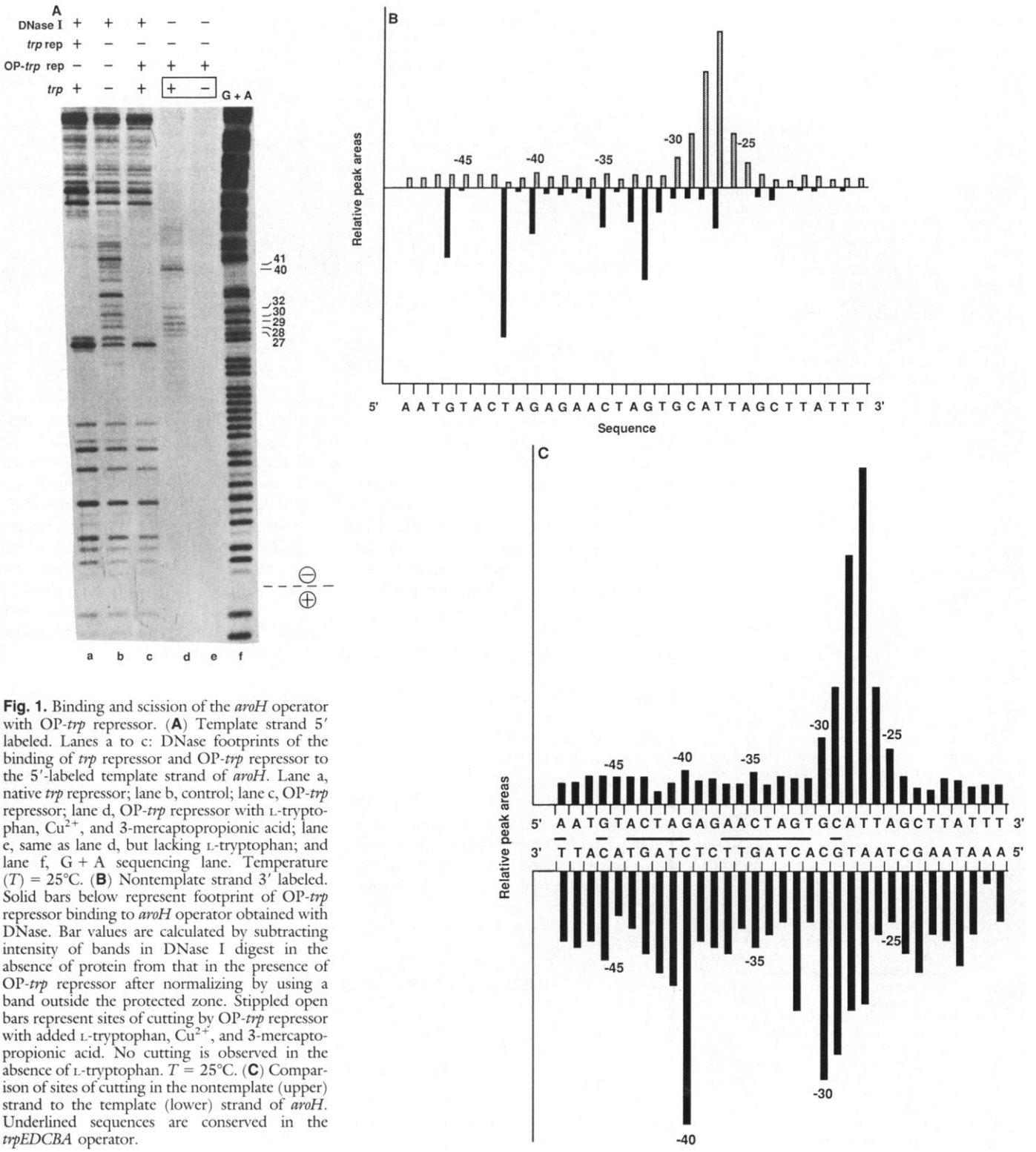

Fig. 1. Binding and scission of the *aroH* operator with OP-*trp* repressor. (**A**) Template strand 5′ labeled. Lanes a to c: DNase footprints of the binding of *trp* repressor and OP-*trp* repressor to the 5′-labeled template strand of *aroH*. Lane a, native *trp* repressor; lane b, control; lane c, OP-*trp* repressor; lane d, OP-*trp* repressor with L-tryptophan, Cu^{2+}, and 3-mercaptopropionic acid; lane e, same as lane d, but lacking L-tryptophan; and lane f, G + A sequencing lane. Temperature (T) = 25°C. (**B**) Nontemplate strand 3′ labeled. Solid bars below represent footprint of OP-*trp* repressor binding to *aroH* operator obtained with DNase. Bar values are calculated by subtracting intensity of bands in DNase I digest in the absence of protein from that in the presence of OP-*trp* repressor after normalizing by using a band outside the protected zone. Stippled open bars represent sites of cutting by OP-*trp* repressor with added L-tryptophan, Cu^{2+}, and 3-mercaptopropionic acid. No cutting is observed in the absence of L-tryptophan. T = 25°C. (**C**) Comparison of sites of cutting in the nontemplate (upper) strand to the template (lower) strand of *aroH*. Underlined sequences are conserved in the *trpEDCBA* operator.

radiolabel allows the quantification of the extent of phenanthroline derivatization after purification with a G-25 spin column. For the *trp* repressor, incorporation of the phenanthroline was completely dependent on prior reaction with iminothiolane, since the protein lacks free cysteine. In the presence of an excess of chemical modification reagent (*10*), 4 mol of 1,10-phenanthroline are incorporated per protein subunit with four lysine residues. For proteins with cysteine residues, prior reaction of the protein with iminothiolane may not be essential for generating active nucleolytic species. The modification strategy presented was adopted for two reasons: (i) iminothiolane reacts readily with primary amino groups while preserving their positive charge, and (ii) 5-iodoacetamido-1,10-phenanthroline can be readily synthesized in radioactive form from available precursors (*4*).

In order to test the binding and nucleolytic activity of the 1,10-phenanthroline–derivatized *trp* repressor (OP-*trp* repressor), a restriction fragment derived from the control region of the *aroH* transcription unit was used as substrate. This gene encodes one of the isozymes of phospho-2-keto-3-deoxyheptonate aldolase, which catalyzes the first step in the common pathway of aromatic amino acid biosynthesis (*1*). The binding of the unmodified and OP-*trp* repressor to the *aroH* operator was compared under identical conditions by deoxyribonuclease (DNase) I footprinting with either the template or nontemplate strand-labeled (Fig. 1) (*10, 11*). No difference between the modified and native protein was observed in this assay when we used 100 n*M* DNA and 100 n*M* protein, but these concentrations may be too high for the detection of changes in the binding affinity. Both proteins required L-tryptophan as a corepressor for sequence-specific, high-affinity binding. To activate the nucleolytic activity, we added cupric sulfate followed by 3-mercaptopropionic acid to the solution containing the modified *trp* repressor, the DNA fragment, and the corepressor (L-tryptophan) (*12*). The reaction mixture was incubated for 0.5 hour at 25°C and quenched with 2,9-dimethyl-1,10-phenanthroline. The digestion products of the reaction were then separated on a sequencing gel. L-Tryptophan–dependent scission of both DNA strands was observed (Fig. 1). For the template strand, cutting was observed at positions −27 to −30, but position −40 was also a major site of attack (Fig. 1A); for the nontemplate strand, the major sites of attack were clustered about thymidine −27 (Fig. 1B). For both strands, all points of scission were within the DNA-binding domain defined by DNase I footprinting. The sites of cutting on the two strands are compared in Fig. 1C.

By denaturing sequencing gels we detected single-stranded nicks on both strands, but these results do not necessarily imply that DNA-bound OP-*trp* repressor can act catalytically and can cut both strands prior to dissociation. In order to assay for double-stranded breaks, the pBR327 plasmid that contains the *aroH* operator linearized at a unique Sph-1 site was used as a substrate (Fig. 2), and the products were analyzed on nondenaturing agarose gels. Ater 5′ labeling with polynucleotide kinase, the DNA (5 n*M*) was incubated for 20 hours at 37°C with tris HCl (*p*H 8) in the presence of 50 n*M* OP-*trp* repressor, 2.0 μ*M* Cu^{2+}, 5.8 m*M* 3-mercaptopropionic acid, and 10 m*M* L-tryptophan. The reaction was quenched with 2.8 m*M* 2,9-dimethyl-1,10-phenanthroline. As predicted from the map, two products, a 0.4-kb fragment and a 2.75-kb fragment, were obtained. The yield of the 2.75-kb fragment was 14%. The yield of the shorter piece was lower (8%), probably as a result of diffusional broadening during electrophoresis. The nuclease activity of OP-*trp* repressor therefore can accomplish site-specific, L-tryptophan–dependent, double-stranded cleavage. No cleavage was observed in the absence of L-tryptophan.

The *trpEDCBA* transcription unit, which encodes the enzymes responsible for the synthesis of L-tryptophan from chorismic acid, is also negatively regulated by the *trp* repressor in the presence of L-tryptophan (*1, 9*). An important test for the specificity of the OP-*trp* repressor would be its ability to cut this operator whose sequence is different from the *aroH* operator. The OP-*trp* repressor cuts the operator of *trpEDCBA* in an L-tryptophan–dependent reaction under conditions comparable to those when the *aroH* operator is cut (Fig. 3). As in the case of the *aroH* operator, no nicking was observed outside of the sequence domain defined by the DNase footprinting. At 37°C, the half-life for the disappearance of the parent band was 6 ± 0.3 hours (Fig. 3).

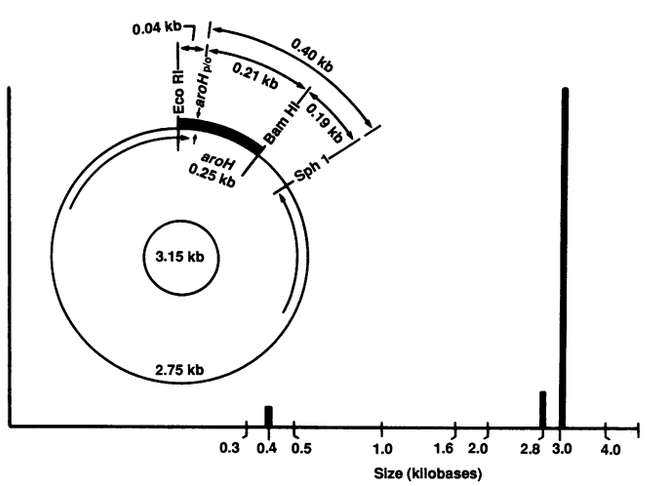

Fig. 2. Double-stranded cutting of pBR327 containing the *aroH* insert. Double-stranded products of 0.40 and 2.75 kb obtained after a 20-hour incubation at 37°C. Yield of 2.75-kb fragment is 14%. Reaction conditions: plasmid (5 n*M*), OP-*trp* repressor (50 n*M*), 2.0 μ*M* Cu^{2+}, 5.8 m*M* 3-mercaptopropionic acid, and 10 m*M* L-tryptophan. Intensities have been corrected for two-fold higher specific activity of the parent peak. Plasmid map shows these are the products anticipated after linearization with Sph-1 and cutting with OP-*trp* repressor. Abbreviation: p/o, promoter/operator.

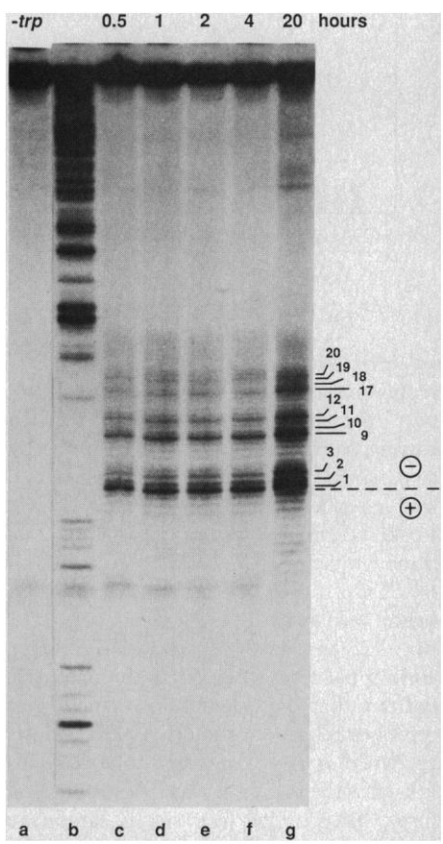

Fig. 3. Kinetics of cutting of *trpEDCBA* with OP-*trp* repressor. 3′-labeled nontemplate strand. Lane a: OP-*trp* repressor with Cu^{2+} and 3-mercaptopropionic acid, but no L-tryptophan, incubated for 4 hours; lane b: DNase I footprint of OP-*trp* repressor with L-tryptophan; lanes c to g: OP-*trp* repressor with L-tryptophan, Cu^{2+}, and 3-mercaptopropionic acid incubated for 0.5, 1, 2, 4, and 20 hours, respectively, at 37°C (*12*).

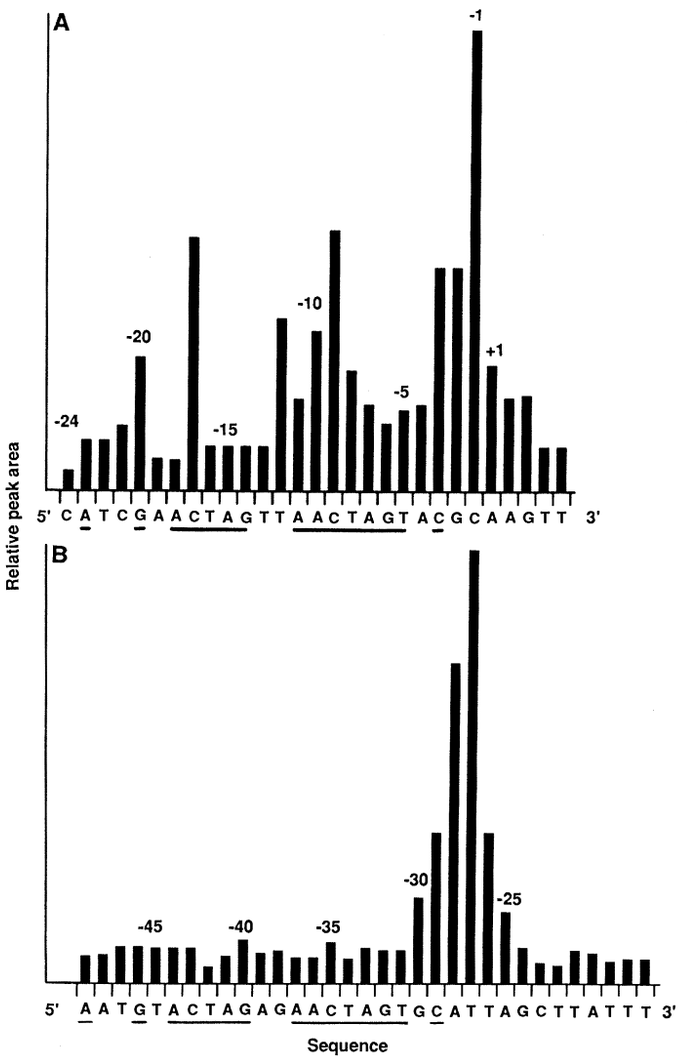

Fig. 4. Comparison of sites of attack on nontemplate strand of the (**A**) *trpEDCBA* and (**B**) *AroH* operators. Operator sequences are aligned according to underlined identities. Numbering system in each cistron is defined with +1 as the start of transcription.

When the sites of attack by the OP-*trp* repressor on the nontemplate strand of these two operators are compared (Fig. 4), the cutting patterns appear distinct. In contrast to the *aroH* operator, where cutting is localized in sequence positions −26 to −31 with weaker cutting at positions −44 to −46, three families of bands are observed for the *trp EDCBA*, each of which are 10 bp or one helical turn apart at positions −1 to −4, −9 to −12, and −17 to −20. The different cutting patterns observed in the complexes indicate that the orientation of the OP-*trp* repressor relative to the DNA helix is different when *aroH* as compared to *trp EDCBA* is used as the recognition sequence. Interestingly, three of the four strong families of bands are centered four sequence positions downstream from the sequence 5′-AACTAGT-3′.

On the basis of our studies of the nuclease activity of the 1,10-phenanthroline–copper complex (*3*), the chemistry responsible for strand scission involves multiple steps after the initial binding of the OP-*trp* repressor–L-tryptophan complex. First, cupric ion is coordinated by the protein-bound OP. Then, it is reduced by 3-mercaptopropionic acid and the resulting cuprous complex reoxidized by molecular oxygen to produce hydrogen peroxide. The in situ generated hydrogen peroxide reacts with the cuprous complex to form the copper-oxo species directly responsible for nucleolytic cleavage. Calibration of digestion patterns with the Maxam-Gilbert sequencing reaction indicates that 5′ and 3′ phosphomonoester termini are the major stable products formed under the reaction conditions. The major reaction pathway to these products for the free 1,10-phenanthroline–copper complex involves oxidative attack on the deoxyribose at C-1. In the case of the protein-associated nuclease, these products might also result from attack of the oxidative species at C-4 and C-3 of the deoxyribose. Two important characteristics of the site-specific nucleases made with 1,10-phenanthroline will be a direct consequence of the chemistry of scission. (i) Cutting can occur at any of the four bases because no feature of the base structure dictates the deoxyribose scission chemistry. (ii) Scission will occur at more than one sequence position because of the flexibility of the linker arm and the possible diffusion of hydroxyl radical from the copper ion.

The generation of a site-specific nuclease activity by means of the chemical modification procedures outlined requires that the derivatized lysyl residues be unimportant for high-affinity binding, yet proximal to the deoxyribose backbone in order for cleavage to proceed. The likelihood of transforming any given DNA-binding protein into a nucleolytic agent is therefore difficult to predict and must be established by experiment. For the *trp* repressor, despite the availability of the primary sequence and a 2.8 Å structure of the *trp* repressor (*13*), the 1,10-phenanthroline–modified lysyl residues most responsible for the nucleolytic activity cannot be unambiguously identified. This question could be addressed by altering the chemical modification conditions to selectively modify particular lysyl residues or by the deletion of selected lysyl residues with site-specific mutagenesis.

The chemical conversion of the *trp* repressor into an endonuclease provides a new reagent for the analysis of DNA structure. Alteration of the DNA-binding domain of the *trp* repressor by mutagenesis should allow the engineering of a family of nucleolytic agents with recognition sequences of about 20 bp. Since many DNA-binding proteins, including the *trp* repressor, coordinately regulate different transcription units, one potential application of nucleolytic agents derived from them would be the identification of additional binding sites for these proteins dispersed within a genome. For example, the *trp* repressor regulates its own synthesis (the *trpR* locus) as well as *trpEDCBA* and *aroH* (*14*). Digestion of *Escherichia coli* DNA by the OP-*trp* repressor might reveal binding sites for the *trp* repressor not previously identified.

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10. *Escherichia coli trp* repressor was reacted with 2-iminothiolane (Pierce Chemical Company) accord-

ing to a procedure adapted from a method first applied to the modification of ribosomal subunits (*8*). From a freshly prepared 2-iminothiolane hydrochloride solution (0.5*M* in 0.1*M*, *p*H 8.0, phosphate buffer) 2.4 μl was added to 0.021 μmol of *trp* repressor in 250 μl of phosphate buffer, containing 1% 2-mercaptoethanol and 1 mg of L-tryptophan at 4°C. After overnight incubation at 4°C, the mixture was passed through a G-25 (coarse) spin column [H. S. Penefsky, *Methods Enzymol.* **56**, 527 (1979)] to remove the unreacted 2-iminothiolane. The spin column was equilibrated in 0.1*M* phosphate buffer, *p*H 8.0, prior to use. Then, 1.6 μmol of [^{3}H]5-iodoacetamido-OP (specific activity, 3 mCi/mmol) in *N,N*-dimethylformamide (4 μl) was added to the eluant collected from the spin column. The alkylation reaction proceeded at 4°C overnight. The product was isolated by passing the mixture through a G-25 spin column.

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12. The *aroH* operator region was part of a 257-bp Bam HI–Eco RI fragment, which had been cloned into pBR327 (*11*). The control region of *trpEDCBA* was contained in a Bam HI–Eco RI restriction fragment which had also been cloned into pBR327. We carried out 5′ and 3′ labeling of these fragments as described [T. Maniatis, E. F. Fritsch, J. Sambrook, *Molecular Cloning* (Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, 1982)]. For the footprinting and cleavage reactions, a solution of labeled *aroH* operator fragment (100 n*M*, 10,000 count/min), *trp* repressor or OP-*trp* repressor (100 n*M* to 200 n*M*), and L-tryptophan (10 m*M*) was incubated at room temperature for 5 minutes in 10 μl of tris-HCl (30 m*M*, *p*H 8.0), KCl (100 m*M*), and $MgCl_2$ (3 m*M*). Footprinting reactions were carried out with 0.04 unit of DNase I for 5 minutes at room temperature. The reactions were quenched by the addition of "stop" solution [transfer RNA (200 μg/ml), 2*M* sodium acetate, and 10 m*M* EDTA]. The DNA was then precipitated with ethanol, redissolved in Maxam-Gilbert loading buffer, and analyzed by the use of a 10% polyacrylamide gel that contained 8.34*M* urea and was cross-linked at a ratio of 19:1. The chemical cleavage reaction was initiated by adding 1 μl of 20 μ*M* cupric sulfate and 1 μl of 58 m*M* mercaptopropionic acid. After 30 minutes at room temperature or at 37°C, the reaction was quenched by adding 2,9-dimethyl-1,10-phenanthroline followed by 1 μl of stop solution. The DNA was then precipitated with ethanol, redissolved in Maxam-Gilbert loading buffer, and analyzed by 10% polyacrylamide 8.34*M* urea, 19:1 cross-linked gel.
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15. We thank R. Gunsalus, A. Kumamoto, and D. Arvidson of the Department of Microbiology and the Molecular Bilogy Institute, UCLA, for frequent and useful discussions. They also generously provided the *trp* repressor and the plasmids that bear the *aroH* and *trpEDCBA* operators. We thank our colleagues T. W. Bruice, J.-F. Constant, M. Kuwabara, and T. Thederahn for valuable comments. This research was supported by USPHS GM 21199 and Office of Naval Research grant 14-86K-0524.

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Seismomagnetic Observation During the 8 July 1986 Magnitude 5.9 North Palm Springs Earthquake

M. J. S. JOHNSTON AND R. J. MUELLER

A differentially connected array of 24 proton magnetometers has operated along the San Andreas fault since 1976. Seismomagnetic offsets of 1.2 and 0.3 nanotesla were observed at epicentral distances of 3 and 9 kilometers, respectively, after the 8 July 1986 magnitude 5.9 North Palm Springs earthquake. These seismomagnetic observations are the first obtained of this elusive but long-anticipated effect. The data are consistent with a seismomagnetic model of the earthquake for which right-lateral rupture of 20 centimeters is assumed on a 16-kilometer segment of the Banning fault between the depths of 3 and 10 kilometers in a region with average magnetization of 1 ampere per meter. Alternative explanations in terms of electrokinetic effects and earthquake-generated electrostatic charge redistribution seem unlikely because the changes are permanent and complete within a 20-minute period.

STRESS CHANGES THAT ACCOMPANY seismic failure are expected to cause piezomagnetic effects and consequent time-dependent local magnetic anomalies (*1*). Local magnetic field changes accompanying, and perhaps preceding, moderate to large earthquakes have therefore been actively sought in countries subject to earthquake hazards (*2*). Observations of local magnetic field transients apparently related to aseismic crustal activity (tectonomagnetic effects) have been well recorded (*3, 4*), but the most easily identifiable tectonomagnetic effect—the coseismic change or seismomagnetic effect expected to accompany rupture—has not yet been unambiguously detected near any moderate to large earthquake. Stable data in this search have been obtained only since about 1960 when modern drift-free and vibration-insensitive absolute magnetometers were first introduced (*5*).

A moderate earthquake (M_L 5.9, where M_L is the local Richter magnitude) occurred at a depth of 11.3 km on the Banning fault, approximately 12 km northwest of North Palm Springs, California, at 0921 UT on 8 July 1986. Preliminary determination of the focal mechanism indicates strike-slip motion in a direction N60°W on the Banning fault with a dip of 45° down to the north (*6*). This earthquake provided a rare opportunity to verify the reality of the elusive seismomagnetic effect; two proton magnetometers had been installed in 1979 at distances of 3 km and 9 km from the subsequent epicenter and have been sampling and transmitting data every 10 minutes since then through a 16-bit digital telemetry system to Menlo Park, California (*7*). Unambiguous observations of a seismomagnetic effect were obtained on these instruments and are reported here.

Figure 1 (left) shows locations of magnetometer sites in southern California at the time of the North Palm Springs earthquake. Because of discontinued maintenance, only ABLM, CHUM, and the two magnetometers LSBM and OCHM in the epicentral area (all shown as closed circles) were operating. The location of the earthquake (star) in relation to the two nearby magnetometers, the aftershock zone, and the primary faults is shown in the upper right section of Fig. 1.

Magnetic field differences between OCHM and LSBM for the 38 days before, and a few days after, the earthquake are shown in Fig. 2A. Close inspection of the data near the time of the earthquake indicates that the offsets were possibly complete within one sample interval, and certainly complete within two (< 20 minutes). The net field offset generated by the earthquake between these two sites is 1.0 ± 0.2 nT. To isolate the relative offsets at each site, simultaneously recorded data from the nearest operating magnetometer, CHUM, about 260-km distant, were subtracted from each time series. Figure 2, B and C, shows the plots of these differences during the same time as the OCHM-LSBM difference. The local magnetic field at OCHM and LSBM apparently decreased by 1.3 ± 0.2 and 0.3 ± 0.2 nT, respectively, at the time of the earthquake. Although these changes are quite small, they are still quite evident in 13-month (Fig. 2D) and 7.5-year plots (Fig. 2E) of OCHM-LSBM difference data, and have remained since the earthquake.

It might be argued that the offsets result from earthquake-induced physical displacement of both of the sensors. Each sensor holder is 2 m above ground and its 15 cm by 15 cm wooden support is set vertically in 1 m of concrete in a borehole. Even though both LSBM and OCHM are within a substantial regional magnetic anomaly derived from metamorphic rocks with measured surface magnetizations between 0.1 and 1 A/m, both sites were chosen in areas with low local gradients (< 2 nT/m). Sensor displacements of between 15 and 65 cm, which are

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