Molecular mechanisms of anesthetic action on neurotransmitter receptors are poorly understood. The major excitatory neurotransmitter in the central nervous system is glutamate, and recent studies found that volatile anesthetics inhibit the function of the a-aminoo-3-hydroxyisoxazolopropionic acid subtype of glutamate receptors (e.g. glutamate receptor 3 (GluR3)), but enhance kainate (GluR6) receptor function. We used this dissimilar pharmacology to identify sites of anesthetic action on the kainate GluR6 receptor by constructing chimeric GluR3/GluR6 receptors. Results with chimeric receptors implicated a transmembrane region (TM4) of GluR6 in the action of halothane. Site-directed mutagenesis subsequently showed that a specific amino acid, glycine 819 in TM4, is important for enhancement of receptor function by halothane (0.2–2 mM). Mutations of Gly-819 also markedly decreased the response to isoflurane (0.2–2 mM), enfurane (0.2–2 mM), and 1-chloro-1,2,2-trifluorocyclobutane (0.2–2 mM). The nonanesthetics 1,2-dichloroehexafluorocyclobutane and 2,3-dichlorooctafluorobutane had no effect on the functions of either wild-type GluR6 or receptors mutated at Gly-819. Ethanol and pentobarbital inhibited the function of both wild-type and mutant receptors. These results suggest that a specific amino acid, Gly-819, is critical for the action of volatile anesthetics, but not of ethanol or pentobarbital, on the GluR6 receptor.

There has been a shift of research focus in the field of mechanisms of anesthetic action from nonspecific lipid perturbation to specific protein sites of action (1). Anesthetics inhibit the activity of the lipid-free enzyme luciferase (2) and produce stereoselective actions on ion channel function (3, 4), suggesting receptor specificity in anesthetic action. Recently, Michie et al. (5) used site-directed mutation of glycine and GABA_A receptors to identify specific amino acids in transmembrane domains 2 and 3 that are critical for the allosteric modulation of these receptors by alcohols and volatile anesthetics. We employed a similar strategy in this study.

The neurotransmitter glutamate plays a major role in synaptic excitation in the central nervous system and is critical for information storage in memory and learning (6). There has been an explosion in understanding the biochemistry and molecular biology of all the glutamate receptor types (N-methyl-D-aspartate, AMPA (a-aminoo-3-hydroxyisoxazolopropionic acid), kainate, and metabotropic) (7). Molecular cloning studies have identified many different glutamate receptors (GluRs), with GluR1–4 (AMPA), GluR5–7 (kainate), and KA1–2 (kainate-binding proteins) representing three distinct families of non-N-methyl-D-aspartate glutamate receptors based on sequence homology and pharmacological properties (7). Kainate receptors are widely distributed throughout the central nervous system (8). GluR6 is found in high levels in most regions of the hippocampus and also in the granule cells of the cerebellum, and an understanding of its roles in brain function is emerging. Previous studies have suggested a presynaptic function on unmethylated moves (C-fibers) in the spinal cord (9) and on excitatory synapses within the CA3 region of the hippocampus (10). Recent studies have found that GABA and glutamate release is inhibited by presynaptic kainate receptors in rat hippocampus (11, 12). A role for kainate receptors in synaptic transmission in the CA3 region of the hippocampus was demonstrated in other recent studies (13, 14). In addition, a genetic linkage study suggests that GluR6 is associated with the pathogenesis of Huntington’s disease (15).

With regard to anesthesia, AMPA receptor antagonists decrease the minimum alveolar concentration (MAC) for halothane or isoflurane (16–19), but there are no reports of the effects of kainate receptor antagonists on MAC. There are few studies of the effects of volatile anesthetics on AMPA and kainate receptors, but Dildy-Mayfield et al. (20) found that enfurane, halothane, and isoflurane (at concentrations achieved during anesthesia) inhibited the function of GluR3 receptors, but enhanced GluR6 receptor function.

One goal of this study was to investigate the mechanisms of anesthetic action on GluR6 receptors. For this purpose, we constructed GluR3/GluR6 chimeras and used these to determine a region of GluR6 that is essential for anesthetic action. We then constructed a series of mutant GluR6 receptors to define specific sites of anesthetic action in GluR6.

**EXPERIMENTAL PROCEDURES**

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†Te whon correspondences should be addressed: Dept. of Pharmacology, University of Colorado Health Sciences Center, Denver, Colorado 80262, the Veterans Affairs Medical Center, Denver, Colorado 80220, the Program for Molecular and Genetic Medicine, Stanford University, Stanford, California 94305.

‡The abbreviations used are: GABA_A, y-aminobutyric acid type A; AMPA, a-aminoo-3-hydroxyisoxalopropionic acid; GluR, glutamate receptor; MAC, minimum alveolar concentration; F3, 1-chloro-1,2,2-trifluorocyclobutane; F6, 1,2-dichloroethoxfluorocyclobutane; F8, 2,3-dichlorooctafluorobutane; TM, transmembrane domain.

††To whom correspondence should be addressed: Dept. of Pharmacology, University of Colorado Health Sciences Center, 4200 E. 9th Ave., P. O. Box C236, Denver, CO 80262. Tel.: 303-315-8609; Fax: 303-315-7499; E-mail: adron.harris@uchsc.edu.

‡‡This paper is available on line at http://www.jbc.org.
Ethanol was purchased from Aaper Alcohol and Chemical Co. (Shelbyville, KY). Halothane was from Halocarbons Laboratories (River Edge, NJ). Enflurane and isoflurane were from Ohmeda Pharmaceutical Products (Liberty Corner, NJ). 1-Chloro-1,2,2-trifluorocyclobutane (F3), 1,2-dichlorohexafluorocyclobutane (F6), and 2,3-chlorooctafluorobutane (F8) were obtained from PCR Inc. (Gainesville, FL). The Ultra-compet Escherichia coli transformation kit was from Stratagene (La Jolla, CA). A QIAGEN plasmid kit was used for purification of plasmid cDNA. GluR3, GluR5, and GluR6 cRNAs were prepared using the mCAP mRNA capping kit (Stratagene). The QuikChange site-directed mutagenesis kit (Stratagene) was used for preparation of the single amino acid mutations.

**GluR cRNA Preparation**—The cDNAs for the GluRs were inserted into the pGEM-HE vector. The cDNAs were linearized with NheI, phenol/chloroform-extracted, and ethanol-precipitated with sodium acetate, and cRNA was prepared using the Stratagene mCAP mRNA capping kit. The cRNAs were extracted using phenol/chloroform and precipitated with ethanol and sodium acetate.

**Whole Cell Voltage Clamp of Injected Oocytes**—Isolation and microinjection of Xenopus oocytes were performed as described by Dildy-Mayfield et al. (22). Xenopus oocytes were injected with 50 ng of cRNA coding for the GluRs. Oocytes were placed in a 100-ml recording chamber and perfused with modified Barth's solution containing 88 mM NaCl, 1 mM KCl, 2.4 mM NaHCO3, 10 mM HEPES, 0.82 mM MgSO4, 0.33 mM Ca(NO3)2, and 0.91 mM CaCl2, pH 7.5, at a rate of 1.8 ml/min at room temperature. Recording and clamping electrodes (1–5 megaohms) were pulled from 1.2-mm outside diameter capillary tubing and filled with 3M KCl. A recording electrode was impaled into the animal pole, and once the resting membrane potential stabilized, a clamping electrode was inserted, and the resting membrane potential was allowed to restabilize. A Warner OC 725-B oocyte clamp was used for voltage-clamping each oocyte at 270 or 290 mV. For oocytes expressing wild-type GluR5 or GluR6, chimeras from GluR6, or mutants of GluR6, 10 mM concanavalin A in modified Barth's solution was preapplied for 2 min to prevent desensitization (23–25). The anesthetics (halothane, isoflurane, enflurane, and F3), nonanesthetics (F6 and F8) (26), ethanol, and pentobarbital were preapplied for 2 min to allow for complete equilibration in the bath. Solutions of volatile compounds were prepared immediately before use. The anesthetic, nonanesthetic, and ethanol concentrations in the figures represent bath concentrations, measured as described previously (20, 27).

In most experiments, we used maximally effective concentrations of kainate to study anesthetic modulation; specifically, 1, 10, or 400 μM kainate.
Fig. 3. Halothane actions on mutants derived from GluR6 subunits.

The effects of 2 mM halothane on kainate (100 nM) responses were tested in oocytes expressing wild-type and mutant GluR6 receptors. Values represent percent change in kainate response produced by halothane and are the means ± S.E. from four to eight oocytes. Statistical analyses were performed using an unpaired *t* test and Dunnett’s correction. * and ***p < 0.05 and p < 0.001 versus wild-type GluR6, respectively.

| M1 (G814S) | SALGQVQGIGFIVL |
| M2 (V815L) | SALGQVQGIGFIVL |
| M3 (G816S) | SALGQVQGIGFIVL |
| M4 (I818V) | SALGQVQGIGFIVL |
| M5 (G819A) | SALGQVQGIGFIVL |
| M6 (I821V) | SALGQVQGIGFIVL |
| M7 (I823Y) | SALGQVQGIGFIVL |
| Wild-type  | SALGQVQGIGFIVL |

TABLE I

EC$_{50}$ values for kainate activation of wild-type and mutant GluRs and GluR chimeras

<table>
<thead>
<tr>
<th></th>
<th>EC$_{50}$ (μM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wild-type</td>
<td>24.8 ± 4.4</td>
</tr>
<tr>
<td>Wild-type</td>
<td>0.2 ± 0.1</td>
</tr>
<tr>
<td>M1 (G814S)</td>
<td>0.3 ± 0.1</td>
</tr>
<tr>
<td>M2 (V815L)</td>
<td>0.3 ± 0.1</td>
</tr>
<tr>
<td>M3 (G816S)</td>
<td>0.3 ± 0.1</td>
</tr>
<tr>
<td>M4 (I818V)</td>
<td>0.3 ± 0.1</td>
</tr>
<tr>
<td>M5 (G819A)</td>
<td>0.4 ± 0.2</td>
</tr>
<tr>
<td>M6 (I821V)</td>
<td>0.6 ± 0.2</td>
</tr>
<tr>
<td>M7 (I823Y)</td>
<td>0.4 ± 0.2</td>
</tr>
<tr>
<td>GluR6 (G819Q)</td>
<td>0.6 ± 0.1</td>
</tr>
<tr>
<td>GluR6 (G819L)</td>
<td>0.6 ± 0.1</td>
</tr>
<tr>
<td>GluR6 (G819S)</td>
<td>0.8 ± 0.2</td>
</tr>
<tr>
<td>GluR6 (G819H)</td>
<td>0.5 ± 0.1</td>
</tr>
<tr>
<td>GluR6 (G819F)</td>
<td>0.6 ± 0.1</td>
</tr>
<tr>
<td>GluR6 (G819W)</td>
<td>0.7 ± 0.2</td>
</tr>
<tr>
<td>GluR6 (G819C)</td>
<td>0.4 ± 0.1</td>
</tr>
<tr>
<td>T1</td>
<td>0.4 ± 0.2</td>
</tr>
<tr>
<td>T2</td>
<td>0.3 ± 0.2</td>
</tr>
<tr>
<td>T3</td>
<td>0.4 ± 0.1</td>
</tr>
<tr>
<td>C1</td>
<td>0.8 ± 0.1</td>
</tr>
<tr>
<td>C2</td>
<td>0.7 ± 0.3</td>
</tr>
<tr>
<td>C3</td>
<td>0.4 ± 0.2</td>
</tr>
<tr>
<td>C4</td>
<td>3.8 ± 0.6</td>
</tr>
<tr>
<td>C5</td>
<td>1.5 ± 0.5</td>
</tr>
<tr>
<td>C6</td>
<td>50 ± 8</td>
</tr>
<tr>
<td>Wild-type</td>
<td>60 ± 10</td>
</tr>
</tbody>
</table>

RESULTS

Effects of Halothane on Chimeric GluR3/GluR6 Receptors—We studied effects of 2 mM halothane on chimeras between GluR3 and GluR6 (Fig. 1). The enhancement produced by halothane on wild-type GluR6 was eliminated in C1 and C2 by replacement of TM4 and some of the sequence N-terminal to TM4 with the same region from GluR3. Results with these chimeras indicated that TM4 was important for halothane potentiation. We also replaced the N- and C-terminal regions of GluR3 with GluR6 (C5 and C6). These chimeras were similar to GluR3, and halothane inhibited the kainate-induced current, indicating that neither the C- nor N-terminal region was the site of enhancement in GluR6 or the site of inhibition in GluR3. Likewise, 2 mM halothane inhibited the kainate-induced current by 47% on C4, suggesting that the N terminus, TM1, TM2, and C terminus were not very important in either enhancement or inhibition by halothane. It is notable that the inhibitory effect of halothane on C4 was converted to a stimulatory effect in C3 by changing the TM4 region.

To further define the site of halothane enhancement in TM4 of GluR6, we made a series of mutants of C1 (Fig. 2). We replaced the 4 amino acids just before TM4 (T2) and in the middle of TM4 (T1 and T3). Halothane had little effect on T2 and T3; however, it enhanced the function of T1 by 72 ± 6%. These results indicate that the sequence GGIF1 in TM4 is...
critical for the enhancing effects of halothane on GluR6.

Effects of Single Amino Acid Mutations in TM4—Next, we made single amino acid mutations of the GGIFI sequence as well as of the flanking amino acids, selecting residues that were different between GluR3 and GluR6 and changing the GluR6 residue to GluR3 (Fig. 3). Halothane enhanced the M1 (G814S), M2 (V815L), and M3 (Q816S) mutant receptor function to an extent similar to that seen with the wild-type receptor (158 ± 6%). However, halothane enhancement was reduced in the M4 (I818V), M5 (G819A), M6 (I821V), and M7 (I823Y) mutant receptors to 80 ± 6, 34 ± 10, 92 ± 24, and 68 ± 9% of control, respectively. The reduction was greatest in GluR6(G819A), and these results suggested that position 819 was particularly important for halothane action, but the amino acids at positions 818, 821, and 823 may also have a role in the enhancement. It is important to note that mutations in the TM4 region did not alter the EC₅₀ for kainate responses (Table I).

Effects of Anesthetics, Ethanol, and Nonanesthetics on Gly-
819 Mutants—We compared the effects of halothane, isoflu-
rane, enflurane, ethanol, the novel halogenated compound F3 (26), and the nonanesthetics F6 and F8 (26) on wild-type GluR6 and mutant GluR6(G819A) receptors. Halothane, isoflurane, enflurane, and F3 enhanced the actions of kainate on the wild-type GluR6 receptor in a concentration-dependent manner, as reported earlier (20). However, these compounds had no significant effects on GluR6(G819A) (Figs. 4 and 5A). The non-
anesthetics F6 and F8 had little effect on either wild-type
GluR6 or GluR6(G819A) (Fig. 5, B and C). At concentrations corresponding to anesthetia (MAC), halothane (0.25 mM) pro-
duced 30% enhancement of wild-type GluR6 function, enflu-
rane (0.6 mM) produced 45% enhancement, isoflurane (0.28 mM) produced 20% enhancement, and F3 (0.8 mM) produced 60% enhancement (Fig. 4).

We also measured the effects of ethanol and pentobarbital on
GluR6(G819A) function. Ethanol inhibited the kainate-induced
currents in a concentration-dependent manner with both wild-
greater than the anesthetic (MAC) concentrations, whereas rane were tested at concentrations eight and seven times the concentrations used for these drugs in Fig. 7. Halothane and isoflurane showed a significant effect of ethanol concentration (Fig. 8). It should be noted that the modest enhancement produced by halothane (and the even smaller enhancement produced by isoflurane) on glutamate receptors may be due to the relatively high concentrations used for these drugs in Fig. 7. Halothane and isoflurane were tested at concentrations eight and seven times greater than the anesthetic (MAC) concentrations, whereas enfurane and F3 were tested at concentrations corresponding to three and one times MAC, respectively. In contrast to their effects on the actions of the volatile anesthetics, none of the mutations affected the action of ethanol or pentobarbital (Fig. 8).

Effects of Ethanol and Anesthetics on GluR5—Because the GluR5 subunit is homologous in the Gly-819 region to GluR6 but not to GluR3 (GluR6 and GluR5 have identical amino acid sequence from Ala-810 to Ala-836) (6), we tested the effects of ethanol and anesthetics on the function of homomeric GluR5 receptors. Modulation of GluR5 was quite similar to that of GluR6, with volatile anesthetics enhancing function and ethanol and pentobarbital inhibiting function (Fig. 9 and Table I). Comparison of drug action on GluR3, GluR5, and GluR6 supported the idea that Gly-819 in GluR6 is a critical determinant of the effects of volatile anesthetics on AMPA/kainate receptors. Again, it should be noted that halothane and isoflurane were tested at eight and seven times MAC and enfurane and F3 at three and one times MAC, respectively, in Fig. 9.

Molecular Modeling of the G819A Mutation—The model of TM4 shows that Gly-819 provides a small cavity that may not be filled by side chains of adjoining helices (Fig. 10A). This cavity can be filled by interaction with a halothane molecule (Fig. 10B). The halothane molecule not only occupies the space provided by the small glycine side chain, it has favorable interactions with adjoining amino acids. Substitution of alanine for Gly-819 reduces the cavity at that position, as shown in the profile of the α-helix in Fig. 10C. The difference in molecular volume caused by the mutation of glycine to alanine is 99.2–78.9 Å3. Mutation of glycine to tryptophan decreases the molecular volume by 224.0–78.9 Å3. These decreases in volume can be compared with the 121.7 Å3 volume of halothane itself.

DISCUSSION

In this study, we identified specific amino acids in the TM4 region of GluR6 that determine actions of volatile anesthetic action on this receptor. In particular, mutations of Gly-819 markedly decreased halothane, isoflurane, enfurane, and F3 enhancement. Recently, Mihic et al. (5) used mutagenesis to implicate several residues of the glycine α1-receptor subunit and of the GABA_α or β-subunits as important sites for ethanol and enfurane actions on these receptors, suggesting that alcohol and volatile anesthetics act on a specific region of these proteins. Our results are consistent with findings in the GABA_α and glycine receptors in that specific mutations markedly affect anesthetic action on all these receptors.

Glutamate receptors have been proposed to contain a large extracellular N-terminal domain, four hydrophobic domains, and an intracellular C-terminal domain (6, 21, 28, 29). It has been hypothesized that TM2 is inserted into the ion channel pore, based on comparison of the amino acid sequence between the N-methyl-D-aspartate receptor and K⁺ channels (28). These models indicate that the region of TM4 implicated in anesthetic action on GluR6 in our study is close to the extracellular membrane surface. The site of enfurane action on GABA_α and glycine receptors also appears to be near the extracellular surface (5). The binding site for local anesthetics recently established on Na⁺ channels is also near the membrane surface, but in this case, it is the cytoplasmic surface (30).

Our results raise the question of why anesthetics had little effect on the kainate-induced currents for the GluR6 Gly-819 mutants. Previously, it was reported that domains in the extracellular loop between TM3 and TM4 are part of the kainate-binding site based on chimeric receptors (21). In the present study, mutations in the TM4 region did not alter the EC₅₀ for kainate responses, suggesting that this Gly-819 site is not close...
to the kainate-binding site. Halothane (0.2–2 mM), in contrast to its affect on wild-type GluR6, did not enhance the kainate-induced currents for GluR6(G819A); however, it is interesting to note that the hydrophilicity, hydrophaticity, and polarity of alanine are similar to those of glycine (31–33). In addition, a variety of amino acid substitutions differing in physical properties all reduced anesthetic action. These results suggest that the hydrophilicity, hydrophaticity, and polarity of amino acids at the site of action of anesthetics are not important factors for their effects. However, mutation of glycine to alanine may produce sufficient increase in volume to prevent halothane from binding within a cavity formed (in part) by the Gly-819 region of TM4. Modeling of TM4 shows that Gly-819 resides between more bulky amino acids and that a small cavity is created at this position that can be filled by halothane. However, the decrease in molecular volume of 20 Å³ created by the G819A mutation is small compared with the 122 Å³ of halothane, whereas the 145-Å³ decrease in volume caused by the G819W mutation exceeds that of halothane. In that both the G819A and G819W mutants are fully functional ion channels with EC₅₀ values for glutamate similar to that of wild-type GluR6, it is clear that halothane must have some effect other than filling a cavity. It is tempting to suggest that halothane enhances receptor function by entering into a cavity and interacting with residues one turn above or below Gly-819.

Ethanol and pentobarbital inhibit GluR6 function (25). Because mutations of GABAₐ and glycine receptors that affect volatile anesthetic actions also affect alcohol (but not barbitu-

---

**Fig. 7.** Effects of halothane (2 mM), enflurane (2 mM), isoflurane (2 mM), and F3 (0.8 mM) on the kainate-induced currents for the wild-type GluR6, GluR6(G819A), GluR6(G819C), GluR6(G819F), GluR6(G819H), GluR6(G819L), GluR6(G819Q), GluR6(G819S), and GluR6(G819W) receptors. Anesthetics or ethanol was preapplied for 2 min before being coapplied with kainate (1 mM) for 20 s. Values are the means ± S.E. from 4 to 20 oocytes. All results with the GluR6 mutants are significantly different from those with wild-type GluR6.

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**Fig. 8.** Effects of ethanol (200 mM) and pentobarbital (100 μM) on the kainate-induced currents for the wild-type GluR6, GluR6(G819A), GluR6(G819C), GluR6(G819F), GluR6(G819H), GluR6(G819L), GluR6(G819Q), GluR6(G819S), and GluR6(G819W) receptors. Anesthetics or ethanol was preapplied for 2 min before being coapplied with kainate (1 mM) for 20 s. Values are the means ± S.E. from 4–20 oocytes.
rate) actions (5), we asked if mutation of Gly-819 would change ethanol and pentobarbital inhibition of GluR6. We found that a range of mutations did not alter the action of ethanol or pentobarbital. This suggests a different mechanism of action, and perhaps different sites of action, for these drugs as compared with those of the volatile anesthetics on GluR6. Indeed, mutation of the TM2 region of AMPA receptors has been shown to alter the action of pentobarbital (34). A distinct site and mechanism of action could explain why alcohols and barbiturates inhibit the function of GluR6 receptors, but volatile anesthetics have the opposite effect.

In conclusion, we identified amino acid residues of the GluR6 receptor that are important for volatile anesthetic potentiation of receptor function. These results support the view that volatile anesthetics interact with specific regions of membrane proteins rather than nonspecifically with multiple regions of lipids or proteins. By analogy to glycine and GABA<sub>A</sub> receptors (5), it is tempting to speculate that Gly-819 is part of a hydrophobic pocket and provides an anesthetic-binding site on GluR6 receptors. Unfortunately, it is not currently possible to carry out the direct measurement of binding required to test this hypothesis. Although there is little information regarding the role of GluR6 in anesthesia, the creation of Gly-819 mutants <i>in vivo</i> using transgenic animal technology (35) should be useful for answering this question.

Acknowledgments—We thank Drs. S. J. Mihic, C. F. Valenzuela, M. P. Mascia, R. Cordoso, and V. Bleck for helpful discussions and technical suggestions.

REFERENCES

![Fig. 9](https://example.com/fig9.jpg)

**Fig. 9.** Effects of halothane (2 mM), isoflurane (2 mM), and enfurane (2 mM) (<i>A</i>) and of ethanol (200 mM), pentobarbital (100 μM), and F3 (0.8 mM) (<i>B</i>) on the kainate-induced currents for GluR5. The drug concentrations were the same as tested on GluR6 in Figs. 7 and 8. Anesthetics or ethanol was preapplied for 2 min before being coapplied with kainate (100 μM) for 20 s. Values are the means ± S.E. from four to eight oocytes. <i>Hal.</i>, halothane; <i>Iso.</i>, isoflurane; <i>Enf.</i>, enfurane; <i>Pento.</i>, pentobarbital.

![Fig. 10](https://example.com/fig10.jpg)

**Fig. 10.** The amino acid sequence of TM4 for GluR6 was modeled as an α-helix, with the pore of the ion channel toward the left (<i>A</i>). A molecule of halothane was modeled, positioned near the pocket formed by Gly-819 in GluR6, and then docked onto TM4 using Discover 97 (see "Experimental Procedures" for details) (<i>B</i>). The effect of the G819A mutation was modeled by replacing Gly-819 with alanine and then re-optimizing the side chain packing (<i>C</i>).
Sites of Volatile Anesthetic Action on Kainate (Glutamate Receptor 6) Receptors
Kouichiro Minami, Marilee J. Wick, Yael Stern-Bach, Jo Ellen Dildy-Mayfield, Susan J. Brozowski, Elizabeth L. Gonzales, James R. Trudell and R. Adron Harris

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