Fast excitatory synaptic transmission in the mammalian CNS is mostly mediated by AMPA receptors (AMPARs), ligand-gated ion channels that are activated by glutamate released from the presynaptic terminals (1–4). On activation, AMPARs provide the transient excitatory postsynaptic current (EPSC) that depolarizes the membrane and initiates downstream processes, such as the generation of action potentials or synaptic plasticity (5, 6). The time course and amplitude of AMPAR-mediated EPSCs exhibit considerable variability among neurons and synapses and strongly depend on the properties of the postsynaptic AMPARs (7, 8).

AMPARs are tetrameric assemblies of \( \alpha \) subunits with distinct properties that are encoded by the glutamate receptor (Glur) genes Glur-A to Glur-D (9–11) or GluA1–4 according to the International Union of Basic and Clinical Pharmacology nomenclature (12) and their variations resulting from alternative splicing and RNA editing (13–15). In most central neurons, multiple variants of these Glur proteins are expressed and assembled into heteromultimeric channels that display a wide range of gating kinetics and Ca\(^{2+}\) permeabilities (16–19). In addition to the \( \alpha \) subunits, the properties of the AMPARs are modulated by a family of transmembrane AMPAR regulatory proteins (TARPs) (20, 21). The TARPs coassemble with the GluR proteins and through direct protein-protein interactions affect the gating, permeability, and pharmacology of the AMPARs (21–25). Furthermore, the TARPs influence the number and subcellular localization of AMPARs by promoting their trafficking to the plasma membrane and their targeting to the synapse (26, 27).

The profound impact of the TARPs led to the assumption that almost all AMPARs in the mammalian brain may be assembled with these auxiliary subunits (28, 29). However, only a minor portion of the AMPAR complexes in the rat brain (~30%) are associated with \( \gamma \) and \( \gamma \)-3, the TARPs with the most widespread expression pattern (30, 31) (Fig. 1A, arrowhead). It is, therefore, possible that native AMPARs contain further yet-unidentified protein constituents that may be identified by proteomics, as described below.

**References and Notes**

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Figs. S1 to S4

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Proteomic analysis of AMPAR complexes from rat brain. For proteomic analysis of native AMPARs, we used affinity purifications (APs) with three different antibodies specific either for GluR-A and GluR-B (anti–GluR-A, anti–GluR-B) or the TARPs γ-2 and γ-3 (anti–γ-2/3) (fig. S2, B and C) on solubilized membrane fractions prepared from total rat brain (36) (fig. S3). These protein preparations contained high-molecular-weight complexes of AMPARs (~0.7 megadalton) assembled from GluR and TARP subunits as visualized by two-dimensional gel separations using blue native polyacrylamide gel electrophoresis (BN-PAGE) and denaturing SDS-PAGE (36) (fig. 1A and fig. S1). Total eluates of APs with the AMPAR subunit–specific antibodies or several pools of preimmunization immunoglobulins G (IgGs) serving as negative controls were analyzed by high-resolution nanoflow liquid chromatography tandem mass spectrometry (nano-LC-MS/MS). The results of these MS-analyses showed that all four GluR proteins (GluR-A to GluR-D) were specifically and abundantly [i.e., IPQ score and \(q_{	ext{norm}}\) score, respectively] (36) retained by each of the three AMPAR subunit–specific antibodies (table S1); the peptides retrieved by mass spectrometry provided extensive coverage for the primary sequence of the individual GluR isoforms (61, 75, 56, and 52% for GluR-A to GluR-D, respectively). Moreover, MS/MS spectra obtained from both anti-GluR and anti–γ-2/3 eluates identified five members of the TARP family, with significant peptide yields for γ-2 and γ-3, and smaller yields for γ-4, γ-7, and γ-8 (table S1). A sixth TARP isoform, γ-5, was only observed in eluates of anti–GluR-A APs, albeit in small amounts (table S1).

In addition to the known AMPAR subunits, our MS analyses consistently identified cornichon homolog 2 (CNIH-2) (fig. 1) and cornichon homolog 3 (CNIH-3), closely related members of a conserved family of small transmembrane proteins that was first described in Drosophila (fig. 1, C and D) (37–39). Both cornichon proteins were specifically copurified at high yield with all three AMPAR subunit–specific antibodies (table S1), which suggests that CNIH-2 and CNIH-3 are robustly integrated into AMPAR complexes in rat brain.

Coassembly of native and heterologously expressed AMPARs and cornichon proteins. Coassembly of the two cornichon proteins with native AMPARs was confirmed by subsequent reverse-purification using an antibody specific for CNIH-2 and CNIH-3 (anti–CNIH-2/3) (fig. S2A) (36) on membrane fractions from rat brain. The respective eluates, besides the cornichon homologs, contained the AMPAR α subunits GluR-A to GluR-D, as well as the TARP isoforms γ-2, γ-3, and γ-4 (table S1). In addition, the cornichon-AMPAR assembly was corroborated by Western probing of the two-dimensional gel separation of the antibody-shift assay from Fig. 1A with the anti–CNIH-2/3 antibody; this assay separated the γ-2/3–associated AMPARs from those devoid of these TARPs by the additional mass introduced via target-specific binding of the anti–γ-2/3 IgG before the BN-PAGE. The major portion of the two cornichon proteins was not shifted by the γ-2/3 antibody, which indicated that CNIH-2 and CNIH-3 are predominantly assembled into γ-2/3-free AMPAR complexes (fig. 2A). These γ-2/3–free AMPAR complexes were effectively and completely shifted when anti–CNIH-2/3 IgG was used.

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**Fig. 1.** Proteomic analysis identifies cornichon proteins as subunits of native AMPARs. (A) Two-dimensional gel separation of AMPAR complexes from rat brain without (top) and with (bottom, antibody-shift assay) anti–γ-2/3 added to the solubilized membrane fraction; both gel separations were probed by Western blot with the indicated antibodies. Size (BN-PAGE) and molecular mass (SDS-PAGE) are indicated. Note that only a minor fraction of AMPARs (arrow; 32% by densitometric analysis) is shifted by the anti–γ-2/3 antibody. (B) (Top) High-performance liquid chromatography chromatogram of peptide fragments of an anti–GluR-B eluate. (Middle and bottom) MS- and MS/MS-spectra of a peptide unique for CNIH-2 (m/z value of 541.27246). The complete MS/MS fragment \(y^x\)-ion series is indicated together with the amino acid sequence derived from the mass differences (in carboxy-to-amino-terminal direction). (C) Hydrophathy plot (Kyte-Doolittle, window of 12 amino acids) of the rat CNIH-2 protein; horizontal bars denote sequence stretches long enough to span the membrane. (D) Dendrogram (Clustal method) of the cornichon family of proteins.
in the antibody-shift assay (fig. S4), which strongly suggested that all AMPARs not associated with TARPs are coassembled with CNIIH proteins.

The participation of cornichons and TARPs in native AMPARs was further investigated by relative quantification of CNIIH-2 and γ2 protein in the aforementioned APs. The mass traces (m/z peak volumes) of peptides unique for CNIIH-2 and γ2 were quantified on the basis of calibration curves determined for these peptides in a dilution series with heterologously expressed tagged versions of both proteins (36). In APs with anti–GluR-A and anti–GluR-B, the ratio of copurified CNIIH-2/γ2 was between 6/1 and 7/1 (Fig. 2B) or, equivalently, ~85% of the purified AMPARs were associated with CNIIH-2, whereas ~15% partnered with the TARP γ2. The excess of copurified CNIIH-2 over γ2 was independent of the GluR composition of the AMPARs (Fig. 2B) and suggested that cornichons, similar to TARPs proteins, may be directly assembled with the GluR subunits (25). This was tested in APs with anti–GluR-A and anti–CNIIH-2/3 on membrane fractions prepared from Xenopus oocytes and cultured cells (36) that heterologously expressed CNIIH-2 and heteromeric GluR-A/GluR-B AMPARs either alone or in combination. Robust and specific copurification of GluR-A and CNIIH-2 was observed with both antibodies when used on oocytes coexpressing AMPARs and CNIIH-2 (Fig. 2C).

Together, proteomic and biochemical analyses indicated that the cornichon proteins CNIIH-2 and CNIIH-3 are integral constituents of the majority of native AMPARs. They are intimately associated with the pore-forming GluR subunits.

Expression profile of cornichons in the CNS.

Next, the expression profile of CNIIH-2 and CNIIH-3 in the rat brain was investigated by immunohistochemistry using the anti–CNIIH-2/3 antibody. Anti–CNIIH-2/3 immunoreactivity was observed throughout most regions of the brain. Examples of expression of CNIIH-2/3 in the neocortex, hippocampal formation, and cerebellum are depicted in Fig. 3. In all these areas, CNIIH-2/3 immunoreactivity was found in various types of neurons, including neocortical and hippocampal pyramidal cells and cerebellar Purkinje cells, as well as in glial cells, such as Bergmann glia in the cerebellum or astrocytes in the hippocampus (Fig. 3, A and B). Neither of the two cornichon proteins was detected in cerebellar granule cells ([Fig. 3A], right), where AMPAR-mediated synaptic transmission crucially depends on the presence of the TARP γ2 (40, 41). In the hippocampal CA1 region, the anti–CNIIH-2/3 immunoreactivity was localized to the plasma membrane of both postsynapses and extrasynaptic sites (dendritic shafts, spines of pyramidal cells), as seen with post-embedding immunogold electron microscopy (EM) (Fig. 3C).

Enhanced surface expression of AMPARs by cornichons. Auxillary subunits affect both processing and biophysical characteristics of the pore-forming α subunits of various ion channels (42), which prompts respective analyses for the cornichon proteins. To examine CNIIH-mediated effects on AMPAR trafficking, we used heterologous expression of the flop splice variant of GluR-A (GluR-Ao) in cultured cells and Xenopus oocytes either alone or in combination with CNIIH-2 or CNIIH-3. Surface expression of the resulting AMPARs was monitored either by staining a hemagglutinin (HA) epitope in the extracellular N terminus of GluR-Ao (cultured cells) or by recording glutamate-activated currents in whole oocytes (36). Coexpression of either cornichon isoform markedly enhanced the HA-based surface immu
$n = 13$; $0.28 \pm 0.04$ ms, $n = 6$, for AMPARs with γ-2 and CNIH-2, respectively); however, they differed considerably in their deactivation kinetics, as reflected by the current decay when the agonist was removed (Fig. 5A, inset). Thus, either accessory subunit slowed the deactivation of GluR$_{A_1/I_2}$ receptors, but, whereas slowing by γ-2 was moderate as reported earlier (21, 22, 24), deceleration by CNIH-2 was substantial and

Fig. 3. Expression profile of CNIH-2 and CNIH-3 in CNS neurons and glial cells. (A) Immunostaining (red, top; white, bottom) of neocortex (Cx), hippocampal formation (Hipp) and cerebellum (Cb) with anti–CNIH-2/3. Immunoreactivity was observed in various cell types throughout the cortical layers (including both pyramidal and stellate cells in layer V), in CA1/CA3 pyramidal cells and sub-populations of hilar neurons in the hippocampus (including mossy cells), and in Purkinje neurons, as well as in the molecular layer (ml) of the cerebellum. Granule cells in the cerebellum (granule cell layer, gc) were not stained. DG, dentate gyrus. DAPI staining of nuclei is in blue. (B) CNIH-2/3 immunoreactivity in radial Bergmann glia in the cerebellum (upper left) and hippocampal astrocytes (lower left) identified by double-labeling with an antibody against the glial fibrillary acidic protein (GFAP, middle, and both signals merged, right). (C) Electron micrographs of CNIH-2/3 immunoreactivity in the CA1 region of the adult rat hippocampus detected by post-embedding immunogold-EM. Immunoparticles were found over (arrows) and at the edge (open arrowhead) of asymmetrical synapses between axon terminals (t) and dendritic spines (s) of pyramidal cells. Gold particles were also found at the extrasynaptic plasma membrane (filled arrowheads) of dendritic shafts (den) and spines of pyramidal cells. Scale bars, 0.2 μm.

Fig. 4. Cornichons enhance surface expression of AMPARs in cultured cells and Xenopus oocytes. (A) Density of extracellularly HA-tagged AMPARs is increased when CNIH-2 is coexpressed, as visualized by anti-HA immunoreactivity (without membrane permeabilization). (B) Summary of GluR-A surface expression in cells transfected with GluR$_{A_0}$ or co-transfected with GluR$_{A_0}$ and CNIH-2 and CNIH-3, respectively. Values of luminescence (means ± SEM) obtained with 27 (CNIH-2) and 8 (CNIH-3) culture dishes from seven and two independent transfections, respectively; luminescence data were normalized to the GluR$_{A_0}$-expressing control cells. (C) Representative current traces recorded from oocytes expressing GluR$_{A_0}$ or GluR$_{A_0}$/CNIH-2 AMPARs upon application of 1 mM glutamate ± 0.5 mM TCM; current and time scaling as indicated. (D) Summary of the CNIH-mediated increase in GluR$_{A_0}$ currents elicited by 1 mM glutamate ± 0.5 mM TCM. Data are means (± SD) of normalized current amplitudes from two batches of oocytes (6 to 10 oocytes per batch); current amplitudes were normalized to the mean current of the GluR$_{A_0}$-expressing control oocytes.
varied according to its expression level (Fig. 5A and fig. S5). Closer analysis (of the current decay) revealed that the slowing of deactivation by γ-2 and CNIH-2 resulted from a more complex process of channel closure. Although the deactivation time course of GluR-Ai/Bi receptors was adequately described by a single exponential function (time constant of 0.96 ± 0.24 ms, n = 15) (Fig. 5D), two exponential components were required for adequate fitting of the current decay in accessory subunit-containing GluR-Ai/Bi receptor complexes (Fig. 5, B and D) [see also (36)]. The distinct effects of γ-2 and CNIH-2 on deactivation kinetics predominantly resulted from the distinct amplitudes of the respective slow component, which was 8 ± 4% (n = 5) in γ-2-containing complexes, but 30 ± 7% (n = 13) in GluR-Ai/Bi-CNIH-2 receptors (P < 0.01, Mann-Whitney U test) (Fig. 5D).

At lower expression levels of CNIH-2, this slow component was largely reduced, and deactivation could be approximated with a monoexponential function albeit with a time constant for deactivation (τ_{deactivation}) slower than that of GluR-Ai/Bi (fig. S5). As CNIH-2 did, CNIH-3 also prolonged deactivation kinetics of heteromeric GluR-Ai/Bi receptors (Fig. 5D).

The impact of the cornichon proteins on channel gating was also examined in AMPARs assembled from the flop splice variants of GluR-B and GluR-D (GluR-B0/D0), heteromeric channels that display the fastest deactivation and desensitization kinetics of AMPARs (19). Both CNIH-2 and CNIH-3 slowed the deactivation kinetics ~3.6-fold (Fig. 5).

**Fig. 5.** Cornichons slow deactivation kinetics of AMPARs. (A) Representative current responses of AMPARs recorded upon 1-ms applications of 1 mM glutamate (indicated above the current trace) in giant oo-patches excised from Xenopus oocytes expressing GluR-Ai/Bi (black trace) or coexpressing GluR-Ai/Bi and either γ-2 (blue trace) or CNIH-2 (red trace). All complementary RNAs were injected at equal amounts; current and time scaling as indicated. (Inset) Current responses at expanded time scale; agonist application indicated by the horizontal bar. (B) Channel deactivation of GluR-Ai/Bi–CNIH-2 AMPAR complexes is a bi-exponential process. Continuous red line is fit of the sum of two exponentials (dashed lines, single components) with the time constants (τ_{fast}, τ_{slow}) and relative amplitude of the slow component (A_{slow}) as indicated. The current response of GluR-Ai/Bi–γ-2 complexes from (A) is shown for comparison. (C) Representative responses of GluR-B0/D0 and GluR-B0/D0–CNIH-2 AMPARs upon 1-ms applications of 1 mM glutamate in experiments as in (A). (D) Summary of the fit parameters of channel deactivation obtained with AMPARs of the indicated molecular composition. Data are means (± SD) of 6 to 15 patches. Time constants are shown as solid bars, open bars denote A_{slow} in GluR-Ai/Bi–containing AMPARs.

**Fig. 6.** Cornichons reduce desensitization of AMPARs without affecting recovery from desensitization. (A) Superimposed current responses of the indicated AMPARs to a 100-ms application of 1 mM glutamate (indicated above the current trace) in experiments as in Fig. 5. (B) Summary of the values for τ_{desensitization} (solid bars) and the relative amplitude of the nondesensitizing current component (relative I_{ss}, white bars) obtained from monoexponential fits to the current decay recorded from the indicated AMPARs in experiments as in (A). Data are means (± SD) of 6 to 15 patches. (C) Recovery of GluR-Ai/Bi–CNIH-2 AMPARs from steady-state desensitization recorded with a double-pulse protocol (pair of a 100-ms and a 50-ms glutamate pulse separated by increasing time intervals) in a giant oo-patch. Data points are peak currents recorded during the second pulse and normalized to the maximal current (recorded during the first glutamate application). Red line is the result of a monoexponential fit to the data points (τ_{recovery} = 69.2 ms). (Inset) Original current recordings; red trace is response with a recovery interval of 128 ms. (D) Values for τ_{recovery} obtained from fits as in (C) with the indicated AMPARs. Data are means (± SD) of three to eight patches.
2.61 for pure GluR-Bo/Do; 2.56 for CNIH-2 and CNIH-3, respectively (Fig. 6C). Unlike the gating transitions described for this family of proteins in Drosophila, chicken, and yeast, where cornichons were shown to operate as cargo receptors for the export of certain growth factors from the endoplasmic reticulum (37, 38, 47), it thus appears that the members of the cornichon family of transmembrane proteins may have multiple functions in cell physiology.

**Implication for AMPAR-mediated signaling in the CNS.** The influence of CNIH-2 and CNIH-3 on the gating of AMPARs may be viewed as stabilizing the open state impairing channel closure either upon agonist removal or upon conformational changes that trigger receptor desensitization (48). As a consequence, the time course of both deactivation and desensitization was slowed by up to several-fold in AMPARs of various subunit composition (Figs. 5 and 6).

Immuno-EM on the hippocampal CA1 region (Fig. 3C) suggests that CNIH proteins may be incorporated into both postsynaptic and extrasynaptic AMPARs. If coassembled into postsynaptic AMPARs, the cornichons will slow the decay time course of EPSCs, often determined by AMPAR deactivation kinetics. If present in extrasynaptic receptors, CNIH-2 and 3 may enhance the effects of glutamate spillover from the release sites to more distant locations, leading to the activation of receptors that would otherwise desensitize. Such spillover effects may be relevant in mossy fiber synapses on hippocampal CA3 pyramidal neurons and hilar mossy cells and in parallel and climbing fiber synapses on cerebellar Purkinje cells, in which the presynaptic elements form closely spaced release sites (49, 50) and the postsynaptic neurons abundantly express the two cornichon proteins (Fig. 3). Thus, effects on postsynaptic and extrasynaptic receptors will slow EPSCs, prolonging the time course of excitatory postsynaptic potentials and thus enhancing temporal summation of synaptic events (Fig. S6). Whether neurons specialized on synaptic integration (such as hippocampal pyramidal cells) and others specialized on coincidence detection (such as neurons in the auditory pathway or GABAergic interneurons in the cortex) differentially express the cornichon proteins remains to be determined. In conclusion, our results establish cornichons CNIH-2 and CNIH-3 as a new class of accessory AMPAR subunits and thus provide novel molecular determinants for the modulation of neurotransmission in the CNS.

**References and Notes**
Reports

Brightly Fluorescent Single-Walled Carbon Nanotubes via an Oxygen-Excluding Surfactant Organization

Sang-Yong Ju, William P. Kopcha, Fotios Papadimitrakopoulos

Attaining high photoluminescence quantum yields for single-walled carbon nanotubes (SWNTs) in order to broaden their optoelectronics and sensing applications has been a challenging task. Among various nonradiative pathways, sidewall chemisorption of oxygen provides a known defect for exciton quenching through nanotube hole doping. We found that an aliphatic (dodecyl) analog of flavin mononucleotide, FC12, leads to high dispersion of SWNTs, which tend to aggregate into bundles. Unlike other surfactants, the surface organization of FC12 is sufficiently tight to exclude oxygen from the SWNT surface, which led to quantum yields as high as 20%. Toluene-dispersed, FC12-wrapped nanotubes exhibited an absorption spectrum with ultrasharp peaks (widths of 12 to 25 milli–electron volts) devoid of the characteristic background absorption of most nanotube dispersions.

The ability to readily assign the (n,m) chirality of semiconducting single-walled carbon nanotubes (SWNTs) by means of photoluminescence excitation (PLE) mapping (1), together with their photostability (2), holds promise for applications in optoelectronics (3), biological imaging (2, 4), and sensing (4). Although the optical properties of SWNTs are excitonic in nature (5), these structures exhibit low-fluorescence quantum yields. Possible causes include low-lying, nonradiative states (dark excitons) (6) or various defects that, as a result of the large exciton diffusion length (~90 nm) in SWNTs, contribute to substantial photoluminescence quenching (7, 8). Oxygen in particular, in the presence of an acid or neutral environment (9), can quench photoluminescence through hole doping and subsequent nonradiative Auger recombination (8, 10).

To make matters worse, nanotube bundling (11, 12), along with chemical defects resulting from covalent functionalization (13) and nanotube inhomogeneities (14), can further decrease or completely quench nanotube luminescence. Individual SWNTs can have photoluminescence quantum yields as high as 8% (15), but solution-suspended SWNTs have shown much lower quantum yields [i.e., 1.5% for polyfluorene (PFO)–wrapped SWNTs (16), 1.1% for purified DNA-wrapped SWNTs (17), and less than 0.1% for surfactant-micellarized nanotubes (16, 17)]. Most SWNT surfactants allow oxygen to interact and dope these nanotubes, and are sufficiently labile that they allow the nanotubes to reform bundles (9). Here, we show that a low-molecular-weight, organic-soluble analog of flavin mononucleotide, FC12, imparts considerable individualization in toluene and other aromatic solvents (i.e., o-xylene and benzene). In addition, the tight self-organization of FC12 around SWNTs leads to an effective exclusion of oxygen that affords quantum yields as high as 20%.

Flavin mononucleotide (FMN), a common redox cofactor related to vitamin B2, was recently shown to self-organize around SWNTs through a helical conformation (18). Such helical wrapping (Fig. 1, A and B) originates from two sets of self-recognizing H-bonds that “stitch” the neighboring FMN moieties into a continuous helical ribbon (Fig. 1A), the concentric π–π interaction of the isoloxazine ring with the underlying graphene sidewalls (Fig. 1B), and a soluble d-ribitol phosphate side group that imparts effective solubilization in aqueous media. In an effort to broaden flavin-based dispersion in organic solvents, we synthesized an isoloxazine derivative with an aliphatic (dodecyl) side group, termed FC12. The synthetic route of FC12 involves two facile steps with an overall yield of ~35% (19). FC12 dispersions of CoMoCAT (Co-Mo bimetallic catalyst synthesized) SWNTs (20) were obtained by sonicating 1 mg of FC12, 1 mg of SWNTs, and 4 ml of various solvents for 4 hours at 300 W. The mixture was centrifuged for 20 min at 10,000g, which eliminated visible SWNT bundles in various solvents (i.e., benzene, toluene, o-xylene, ethylacetate, tetrahydrofuran (THF), pyridine, acetone, and N,N-dimethyl formamide (DMF)). Table 1 summarizes the physical properties of these solvents as a function of dielectric constant (ε). SWNT photoluminescence was observed for only some of these solvents: benzene, toluene, o-xylene, ethylacetate, THF, and acetone (see below).

PLE maps for benzene, toluene, ethylacetate, and acetone show that the photoluminescence intensity (~315,000 counts) of FC12–(6,5)-SWNTs in toluene dispersion is 15 to 20 times

36. Materials and methods are available as supporting material on Science Online.
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Materials and Methods
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