trophysiological results suggest that this effect may be mediated by way of a direct pathway from the raphe to the SPN's. Although other mechanisms such as disfacilitation or disynaptic inhibition involving a spinal interneuron cannot be definitively excluded, the weights of the combined evidence suggest that the spinal raphe inhibits the SPN's by a direct pathway.

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References and Notes


11. A cytoarchitectonic analysis of the mediulary nuclei for the pigeon; however, our preliminary evaluation of the complex suggests an organization similar to that of the cat [E. Taber, A. Brodal, F. Walberg, J. Comp. Neurol. 114, 161 (1960)].


15. Stimuli were 0.5-msec, cathodal pulses delivered by a constant-current stimulator through No. 30 stainless-steel insulated insect pins insulated to give tip exposures of ~100 μm. Electrode localization was verified histologically from 50-μm calibrations stained with cresyl echt violet. Thresholds were set at stimulus intensities of 30 to 60 μA with supramaximal responses at 50 to 100 μA. The SPN's were identified on the basis of collision [I. Darian-Smith, G. Phillips, R. D. Ryan, J. Physiol. (London) 168, 129 (1963)] of a spontaneous discharge with an antidromic discharge elicited by bipolar stimulation of the preganglionic and sympathetic ganglion 14 [J. B. Cabot and D. H. Cohen, Brain Res. 131, 73 (1977)]. Recording electrodes were 4M NaCl micropipettes with tip resistances of 6 to 10 meegohms.

18. The collision test was applied in all cases, and raphe recording sites were verified histologically.

19. Blood pressure and heart rate were recorded by conventional methods (14). Stimulating electrodes were located in the raphe while the animals were anesthetized with ether; the animals were allowed to recover for 2 to 3.5 hours before stimulation. Stimulus trains were delivered for 3 or 5 seconds at frequencies of 25, 50, or 100 Hz. Threshold was at intensities of 25 to 50 μA with supramaximal responses at 30 to 100 μA.


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Zea diploperennis (Gramineae): A New Teosinte from Mexico

Abstract. A perennial teosinte or “wild maize” endemic to the Cerro de San Miguel, Sierra de Manantlan, Jalisco, Mexico differs from Zea perennis by dimorphic rhizomes, robust habit, and a larger number of longer, laxer tassel branches. The fact that it is a diploid (2n = 20) has taxonomic and agronomic significance. The seeds are used locally for food.

Earlier this year, Guzmán (1, 2) reported his remarkable rediscovery of perennial teosinte, thought extinct in the wild since 1921 (3), at two sites in southern Jalisco, Mexico. Subsequently, both sites were visited by three of us (H.H.I., J.F.D., and R.G.M.), and specimens, seeds, and rhizomes were collected and initial analyses were made. This report confirms Guzmán’s conclusion regarding the Ciudad Guzmán population—that it is, indeed, conspecific with the tetraploid (2n = 40) Zea perennis (Hitchcock) Reeves and Mangelsdorf, originally discovered in this area by Hitchcock in 1910. However, the plants from the second location, Cerro de San Miguel, though similar in many ways, are a clearly distinct diploid taxon, here described for the first time:
inged, strongly green-nerved, the
nerves usually clustered marginally near
he apically ciliate prominent lateral
ings; outer glume strongly enclosing inner
lume.

Female spikes sessile or often borne
on long peduncles, frequently tipped by
short male racemes; fruit cases 5 to 10
per spike, trapezoidal-cylindrical, 6 to 8.2
mm on the long side, 2.5 to 4.5 mm on
the short side, 4 to 5 mm in diameter;
when mature light sepia to grayish brown
speckled with dark brown or nearly
black; weight of 100 mature fruit cases
7.12 g.

Chromosomes number: 2n = 20; mei-
osis regular with ten bivalents (4).

MEXICO: JALISCO: many, often
tense, colonies, mostly among tall
grasses and herbs (Dahlia coccinea,
thalictrum), in deep soft soil, often on
edge of (or in) small streams, and some-
times on edge of (but not in) maize fields
or in grazed pastures, on what was
formerly open Pinus-Quercus (ellig-
ica?)-Carpinus caroliniana forest: at
case of rocky north-northeast-facing up-
permot slopes of Cerro de San Miguel
(east end of Sierra de Manantlan,
just north of and below saddle (crest) at La
Ventana (104°13’W, 19°31’45”N), near an
Indian hut surrounded by five gigantic
Yucca (elephantipes?) trees, 20 km due
south of El Chante, 7 km east-northeast
of El Durazno (Municipio de Cuautitlán,
altitude 2250 to 2400 m, 22 September
1978, H. H. Itis, R. Guzmán M., J.
Doebley, and A. Lasseigne No. 450.

The holotype is in the Herbario de la
Universidad de Guadalajara (Zapopán);
isotypes (to be distributed) in B, BH,
BM, CHAPA, ENC, F, GH, ILL,
K, L, LJ, MEX, Mich, MO, NA,
PA, TAES, TEX, UC, US, WIS, XAL
(i).

Another collection from the same pop-
ulation was distributed as Zea perennis
(the location data given on this label (see
below) and by Guzmán (1) are not quite
correct):

Campos cultivos de maíz cerca del bosque
frio de pino, Cerro de la Ventana San Miguel,
15 km al E de la comunidad indígena de
Cuzalapa, Municipio de Cuautitlán, Jalisco,
1700 m alt., 15 December 1977, R. Guzmán
M. 777 [in ARIZ, Universidad Autonoma
Guadalajara, Universidad Guadalajara, Zapop-
án, Mich (5)].

This collection included mature seeds
which will be distributed with the type
material.

Common name: “Chapule,” “Maíz
Chapule,” or “Milpilla.”

It is of interest that the local people re-
port grinding up and mixing the kernels
with maize for use as food in hard times.

Similar to Zea perennis, Z. diploper-
ennis differs by its dimorphic rhizomes
with much shorter internodes (Fig. 1),
those of Z. perennis being usually 1 to 3
cm long; by its more open root system
which is not densely sod-forming, by the
larger number of, and longer and laxer
tassel branches (Fig. 2); by wider and
longer leaves; and by its considerably
more robust habit (Fig. 2).

The implications of this discovery are
considerable. (i) Being morphologically
primitive, this diploid wild maize could
give clues to the evolution of Zea, and
specifically to the origin of the suppos-
dedly autotetraploid Z. perennis (6), its
probable descendant. (ii) Since it is a
diploid perennial, and interfertile with
maize, as shown by F1 hybrids, grown
from field-collected seeds at the Univer-
sidad de Guadalajara, this new species
should provide geneticists and maize
breeders with a potentially valuable
source of germ plasm, and may lead to
the development of perennial maize.

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Cerebral Glucose Utilization: Local Changes During and After Recovery from Spreading Cortical Depression

Abstract. Cerebral glucose utilization is markedly increased in most areas of the cerebral cortex and reduced in many subcortical structures during spreading cortical depression. During recovery, cortical glucose utilization is still elevated, but the increased metabolic activity is distributed in columns running perpendicularly through the cortex.

Spreading cortical depression, a phenomenon first described by Leão in 1944 (1), remains a puzzling and still poorly understood response of the cerebral cortex to a variety of noxious stimuli. It can be elicited by mechanical, electrical, thermal, and chemical stimuli (2) and is characterized by a spread of transient intense neuronal activity followed by depression in all directions from the site of initiation at a rate of 2 to 5 mm/min (2). This rate of spread is similar to that seen in the Jacksonian march of convulsions or the development of the scotomata of migraine in man (2, 3). The electrophysiological changes consist of depolarization and decreased electrical activity of neuronal units, depression of amplitude of the electroencephalogram, increased electrical impedance, and a negative shift in the d-c potential of the affected cortex (2). There is also evidence of chemical changes in the depressed cortex, for instance, a release of K+ and an increase in extracellular K+ (4), decreased cortical PO2 (2), decreased concentrations of glycogen, glucose, and phosphocreatine (5), and increased concentrations of inorganic phosphate and lactic acid (5). Some of these chemical changes are suggestive of increased energy metabolism, but measurements of cerebral cortical energy metabolism in spreading cortical depression have not been reported. We have, therefore, employed the [14C]deoxyglucose method (6) to determine the regional rates of glucose utilization within the brain during and after the evocation of spreading cortical depression.

The experiments were performed on normal male Sprague-Dawley rats weighing between 370 and 410 g. The procedure for measuring local cerebral glucose utilization has been described (6). Briefly, polyethylene catheters were inserted into a femoral artery and vein under light halothane-nitrous oxide anesthesia, and the animal was then restrained by application of a loose-fitting abdominal-pelvic plaster cast. Holes, approximately 2 to 3 mm in diameter, were drilled through the skull over the occipitoparietal cortex on both sides of the head to expose the dura, which was then kept covered with mineral oil. At least 2 hours were then allowed for complete recovery of the animal from the effects of anesthesia.

In one group of animals spreading cortical depression was induced in one cerebral hemisphere by the application of a filter paper disk soaked in 3M or 5M KCl to the exposed dura on that side. Another disk soaked in 0.15M, 3M, or 5M NaCl was applied to the exposed dura on the opposite or control side. Both disks were replaced with freshly soaked disks at 15- to 20-minute intervals until the end of the experimental procedure. The animals so treated remained conscious, but spreading cortical depression appeared within 3 to 5 minutes after application of the KCl disks and was manifested by a marked hemiparesis and hemianesthesia on the side of the body contralateral to the side of KCl application. Measurement of local cerebral glucose utilization was initiated 15 to 20 minutes after the first application of the KCl and NaC disks by the administration of a pulse of 50 μCi of 2-deoxy-d-[1-14C]glucose (specific activity, 50 to 55 μCi/μ mole) into the femoral venous catheter. Arterial blood samples were rapidly drawn immediately after the pulse and at timed intervals for 45 minutes. The blood samples were immediately centrifuged to separate the red cells, and the plasma samples were stored on ice until usually sequently analyzed for glucose and [14C]deoxyglucose concentrations as described (6). At the end of the 45-minute period, the animal was decapitated, and the brain was removed as rapidly as possible, frozen in Freon XII chilled to −6°C to −7°C with liquid nitrogen, sectioned and subjected to quantitative autoradiography as described (6). Local cerebral glucose utilization was calculated from the time courses of the plasma [14C]deoxyglucose and glucose concentration and the tissue d-4 concentration by the operational equation of the [14C]deoxyglucose method (6).

In another group of animals spreading cortical depression was induced by the application of KCl directly on the surface of the parietal cortex. In these experiments the animal was reanesthetized with intravenous pentobarbital approximately 2 hours after recovery from the halothane-nitrous oxide anesthesia, the exposed dura was opened, and artificial cerebrospinal fluid containing 20 to 8 mM KCl was applied to one side of the parietal cortex and artificial CSF without added KCl was applied to the other side. The d-4 potential of the cortical surface was monitored continuously by means of the Marshall glass pen electrodes (outside diameter, 2 mm) (2). The recording electrode was applied to the surface of the cortex approximately 3 mm from the site of KCl application, and the reference electrode was placed in the subcutaneous tissues of the back of the neck. The outputs of the electrodes were amplified in a differential amplifier and displayed on the face of a Tektronix type RM56 oscilloscope or recorded by means of a Beckman model R611 polygraph. Local cerebral glucose utilization was measured under two sets of conditions in these experiments: (i) during sustained spreading cortical depression manifested by repeated waves of negative shifts of the d-4 potential caused by repeated applications of KCl, and (ii) immediately after return of the d-4 potential to the normal value after a single wave of depression.