branes, whereas the effects of (+)-[^{3}H]-SKF10,047 on isolated guinea-pig ileum preparation have been described by Su *et al.* (*Life Sci* **28:** 2519, 1981), and Kromer *et al.* (*Naunyn Schmiedebergs Arch Pharmacol* **321:** 218, 1982). The present work was thus carried out in order to characterize binding sites for (+)-[^{3}H]-SKF10,047 in guinea-pig myenteric plexus membrane preparations. A saturable specific binding was found when (+)-[^{3}H]-SKF10,047 concentrations were increased from 1 to 50 nM. The equilibrium was reached in about 30 min. Dissociation was complete in 20–30 min. Specific binding was linear with protein concentration up to 1.2 mg/ml. In the range of concentrations explored, a binding site with K_D of 42.2±2.2 nM and a Bmax of 3.19±0.34 pmole/g tissue was determined from Scatchard plot. Morphine and naloxone were inactive up to 10^{-4} M. Among the other compounds tested, haloperidol, imipramine, ketocyclazocine and propranolol were the most potent to displace (+)-[^{3}H]-SKF10,047 from guinea-pig myenteric plexus membranes. In contrast PCP was much less active (IC_{50}=2.5 μM) a result which is at variance with those reported with guinea-pig whole brain membranes (IC_{50}=65 nM). These experiments suggest that *sigma* receptors are present in the guinea-pig myenteric plexus and put forward a putative physiologic role of *sigma* receptors at the gastrointestinal tract level.

PUTATIVE ROLE OF PCP-TCP RECEPTORS IN THE EFFECTS OF KINDLING. *Rondouin, G., I. Chaudieu, J. M. Kamenka and R. Chicheportiche. CNRS LP 8402-INSERM U 249, Ecole Nationale Superieure de Chimie, 8 rue de l'Ecole Normale, 34075 Montpellier Cedex, France; *Laboratoire de Medecine Experimentale, Bd Henri IV, 34060 Montpellier Cedex, France.

Phencyclidine (PCP) has been reported to delay the rate of kindling in rats. This drug and other *sigma* compounds were also reported to block long term potentiation, which mechanisms are partially related to those of the kindling effect. This study investigated the effects of thienylcyclidine (TCP) and some TCP derivatives in kindled rats. TCP and GK103 neither significantly reduced the duration of afterdischarges in kindled rats nor had a clear-cut anticonvulsant effect. The effects on behavioral stages of kindling were rather related to the ataxic effect of these two drugs. Conversely, GK115 blocked kindled seizures without producing an ataxic side-effect. Kindling also induced modifications in the binding parameters of ^{3}H-TCP, both in the cortex and in the cerebellum. These results will be discussed according to the multiple binding sites of these compounds.

THE RAT BRAIN PHENCYCLIDINE (PCP) RECEPTOR: A PUTATIVE K CHANNEL. Sorensen, R. G. and M. P. Blaustein. Department of Physiology, University of Maryland School of Medicine, Baltimore, MD 21201.

^{86}Rb efflux studies on rat brain synaptosomes (Bartschat and Blaustein, *PNAS* **83:** 189, 1986) show that PCP selectively blocks a voltage-dependent, non-inactivating K channel. This suggests that the brain PCP receptor may be associated with this K channel. The present study provides PCP receptor binding data which support this proposal. Aminopyridines (AP) and tetraalkylammonium ions (TAA), compounds which block voltage-dependent K channels, compete with [^{3}H]PCP for binding to rat brain synaptic membanes. Their orders of potency are: for the APs, 4-AP=3,4-diAP > 2-AP >> 3-AP, and for the TAAs, tetrabutylammonium (TBA) > tetraethylammonium > tetramethylammonium. These sequences agree with those reported for the ability of the APs and TAAs to block K channels or to increase neurotransmitter release (an effect which results from the block of K channels). The nature of the interactions of the APs and TAAs at the PCP receptor were further studied. Reciprocal and Schild plot analyses indicate that TBA is a competitive inhibitor at the PCP receptor. 4-AP has a more complex interaction, probably an allosteric inhibition: the results suggest that 2 mol of 4-AP are required to displace 1 mol of PCP at the receptor. These data show that K channel blockers interact directly with the brain PCP receptor. Additional data also implicate the PCP receptor as a K channel. One, the order of potency of PCP and several analogs for block of the synaptosome K channel, [TCP (thienyl analog) > m-amino-PCP > PCP > m-nitro-PCP], is the same as the order of affinities of these compounds for binding to the PCP receptor and for producing behavioral deficits. Two, the stereospecific binding of the "sigma" opiate, N-allylnormetazocine (NANM), [(+NANM > (−)NANM] and the dioxolane, dioxadrol, [dexoxadrol >> levoxadrol], to the PCP receptor are similar to those determined for their block of the synaptosome K channel. Reciprocal and Schild plot analyses demonstrate that (+)NANM and dexoxadrol are competitive inhibitors at the PCP receptor. Taken together, these results provide strong evidence that the brain PCP receptor is part of a K channel. We suggest that the behavioral deficit produced by PCP intoxication results from the ability of PCP to block this K channel. (Supported by NINCDS.)

STUDIES OF NEWER SYNTHETIC OPIOIDS: THE CARDIAC AND KINETIC EFFECTS IN CHILDREN AND PIGLETS. Stiller, R. L., P. J. Davis, D. R. Cook, M. D. Ingram, J. M. Perel and C. M. Roeber. Departments of Psychiatry, Anesthesiology and Pharmacology, University of Pittsburgh School of Medicine, Pittsburgh, PA 15213.

Alfentanil (A), a structural analogue of fentanyl, is about