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18. The conditions in the sheet metal observation arena (25 by 25 cm) were 20°C; relative humidity 60 to 80 percent; light intensity, 700 lux; and laminar wind flow, 71 cm/sec. A sample (1 μl) of solution containing either 1 ng of synthetic or 1 ME of natural extract followed by 5 μl of Skellysolve B were placed onto a filter paper (5 by 7 mm; Whatman No. 1) skewered to a metal thumbtack. After the solvent evaporated in front of the exhaust tube at the downwind end, the thumbtack was placed with forceps so that the paper hung just above the surface 4 cm upwind of a female. Females were scored on four points: whether they (i) walked upwind and touched the paper; (ii) walked upwind but did not touch the paper; (iii) began walking upwind; or (iv) did not move at all during a 10-second exposure to the treatment.
19. Eleven percent of females walked upwind in response to 3 ME of fraction 4, compared to 3 percent for Skellysolve B blank (not significantly different at $P < .05$ according to a chi-square 2×2 test of independence with Yates' correction); 29 percent walked upwind in response to fraction 3 (significantly different at $P < .05$); $N = 35$ for all treatments.
20. Eighty percent of females walked upwind in response to 10 ng of **1** plus 3 ME of fractions 6 plus 7, compared to 45 percent in response to 10 ng of **1** alone (significantly different at $P < .05$ according to a chi-square 2×2 test of independence with Yates' correction); the response to Skellysolve B blank was 13 percent; $N = 40$ for all treatments.
21. The optical rotation of **2** gave a negative sign $[\alpha]_D^{21} = -133°$, at a concentration of 0.01 g/ml in chloroform. Since H. Arakawa, N. Torimoto, and Y. Masui [*Liebigs Ann. Chem.* **728**, 152 (1969)] determined the absolute configuration of (−)-mellein to be *R*, compound **2** also has the *R* configuration.
22. Compound **3** had GLC retention times on OV-101 identical to the authentic *Z* isomer and different from the authentic *E* isomer.
23. Compounds **3** and **4** produced similar mass spectra, yielded different hydrogenated products, and **4** was readily epimerized to **3** [E. Demole and M. Still, *Helv. Chim. Acta* **45**, 692 (1962); H. Tamka and S. Torii, *J. Org. Chem.* **40**, 462 (1975); H. Fukui, K. Koshimizu, Y. Yamazaki, S. Usudo, *Agric. Biol. Chem.* **41**, 189 (1977)].
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Sensory and Motor Functions of Spinal Cord Substance P

Abstract. *Low doses of D-Pro²-D-Phe⁷-D-Trp⁹-substance P, a specific substance P antagonist, depressed the scratching and biting behaviors elicited by intrathecal injections of substance P, and cutaneous application of algesic substances. Higher antagonist doses caused hindlimb paralysis. This suggests that substance P is a neurotransmitter for primary nociceptor afferents and may also have an important function in motor control.*

The principal neurotransmitters for efferent neurons passing through the ventral roots have been known for more than 30 years. However, no sensory neurotransmitter entering the spinal cord via the dorsal roots has been convincingly identified. In 1953, Lembeck (*1*) first suggested that substance P (SP) might be a sensory neurotransmitter. Since the elucidation of the SP structure as Arg-Pro-Lys-Pro-Gln-Gln-Phe-Phe-Gly-Leu-Met-NH_2 (*2, 3*) dorsal root SP has been shown to be present in small diameter primary afferent neurons making synaptic contact onto dendrites of neurons in the spinal cord dorsal horn (*4*). Electrical stimulation of these small diameter afferents leads to a release of SP into spinal cord perfusates (*5*). Furthermore, SP has been shown to excite dorsal horn neurons that can also be excited by intense cutaneous heat (*6*) or by more carefully defined noxious stimuli (*7*).

More recently it has been shown that SP, when injected into intrathecal spaces surrounding the spinal cords of mice, evokes an intense biting and scratching behavior (*8*). This type of behavior, similar to behaviors associated with chronic pain in rodents (*9*), is undoubtedly sensory in nature; the animals precisely direct their mouths and paws to their cutaneous surfaces in an apparently purposeful fashion. In order to further delineate the association of this behavior with pain sensation, we have now mimicked this behavior by coating the skin of the mice with algesic substances. In addition, we have antagonized the responses of these mice to algesic agents by the use of a specific SP receptor antagonist. Combined with the previously obtained data (*1, 4, 8*), this information provides evidence that SP is a neurotransmitter for primary nociceptor afferents. However, data are also presented suggesting that: (i) SP may not be the sole neurotransmitter for nociceptor afferents, and (ii) that spinal cord SP may also play an important role in motor control.

Capsaicin, the active ingredient of Hungarian red peppers, is known to produce intense burning inflammatory pain when applied to human skin (*10*), possi-

Table 1. Capsaicin-induced scratching and biting. The irritant was swabbed on with a Q-Tip. The observation time was the 5 minutes that immediately followed the irritant application. Results are expressed as the mean ± S.E.M. ($N = 8$).

Site	Chemical	Biting and scratching episodes	
		Number	Time spent (seconds)
Ear	Capsaicin	14.1 ± 2.7 (scratching)*	
	Ethanol	1.3 ± 0.5	
Foot	Capsaicin	42.5 ± 9.0 (biting)*	51.9 ± 10.2*
	Ethanol	0	0
Back	Capsaicin	23.4 ± 6.7 (scratching)*	12.6 ± 4.1*
	Ethanol	4.5 ± 1.3	2.1 ± 0.8

*Significantly different from control ($P < .05$, *t*-test).

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Table 2. Effects of D-Pro^2-D-Phe^7-D-Trp^9–SP on capsaicin-induced foot biting. The irritant was swabbed on the skin of the foot with a Q-Tip. The observation time was the 5 minutes immediately after the irritant was applied. Observers were blinded as to material injected. The results are given as means ± S.E.M. (N = 8). The vehicle was 2 μl of 0.1 percent crystal violet in 0.25 percent methylcellulose. Only animals with intraspinal dye marks at autopsy were accepted for data analysis to ensure that the drug was actually injected intraspinally.

Injection		Biting episodes	
Site	Material	Number	Time spent (seconds)
Spinal cord	Vehicle	31.8 ± 5.1	47.0 ± 5.3
Spinal cord	6.5 nmole of D-Pro^2-D-Phe^7-D-Trp^9–SP	14.7 ± 5.7*	25.4 ± 8.4*
Intravenous	Vehicle	41.3 ± 6.0	42.0 ± 7.4
Intravenous†	6.5 nmole of D-Pro^2-D-Phe^7-D-Trp^9–SP†	39.5 ± 4.8	39.8 ± 7.1
Uninjected		42.5 ± 9.0	51.9 ± 10.2

*Significantly different from all other groups ($P < .05$, t-test). †In separate experiments, doses as high as 130 nmole were ineffective. Higher doses produced "sedative-like" depressant effects.

bly by releasing SP (*11*). In order to determine whether the acute scratching response observed with intrathecal SP is a response appropriate for pain sensation, the cutaneous surfaces of male mice (18 to 22 g) were painted using Q-Tips soaked with capsaicin solutions. Animals were placed in individual plastic chambers and observed for a 5-minute period (*12*). Intense scratching and biting reactions were observed with capsaicin solutions applied to the feet, ears, and shaven backs of the mice (Table 1). In contrast, mice would only occasionally elicit a few weak wiping motions when their skins were coated with alcohol. Thus, the scratching and biting behavior evoked by intrathecal SP is a behavior that mice also utilize as a response to chemically induced inflammatory pain.

D-Pro^2-D-Phe^7-D-Trp^9–SP has been shown to specifically antagonize SP receptors of guinea pig ileum (*13*) and rat colon (*14*). We have now found that D-Pro^2-D-Phe^7-D-Trp^9–SP antagonizes SP's spinal cord action. The antagonist, given in doses (per animal) of 0.3, 1.0, 3.0, 10, and 30 μg (0.20, 0.65, 2.0, 6.5, and 20.0 nmole), was coadministered intrathecally with 18 pmole of SP. Each dose was tested in six animals. The threshold dose for antagonism was 0.65 nmole. The SP response was totally antagonized when the antagonist dose was raised to 2.0 and 6.5 nmole. Using Spearman-Karber analysis (*15*), we found that the dose required to antagonize the SP response in 50 percent of the animals was 1.0 nmole (0.7 to 1.3 nmole, 95 percent confidence interval). The effect was not due to any nonspecific anesthetic-like action because 6.5 nmole of the antagonist did not antagonize the scratching syndrome induced by somatostatin (*16*).

Intrathecal injections (but not intravenous injections) of 6.5 nmole of this antagonist dramatically depressed the responses of mice to cutaneous capsaicin application (Table 2). These data indicate that the capsaicin-evoked sensory experience is dependent on the endogenous SP system present in the spinal cord dorsal horn (*4*). Indeed, SP now appears to fulfill most of the anatomical, biochemical, and pharmacological criteria (*17*) required to identify it as a neurotransmitter for primary nociceptor afferents. Thus, although it has been suggested that SP may possess neuromodulator properties (*6, 18*), we believe that primary afferent SP functions more like a classical transmitter (*19, 20*).

In the tail-flick and hot-plate reflex tests traditionally used to detect narcotic analgesics 6.5 nmole of the antagonist was without effect. Raising the dose to 20.0 nmole was sometimes effective in prolonging tail-flick reflexes, but this dosage also elicited significant motor deficits not seen with lower doses. The motor syndrome varied considerably among different animals. Either the tail or one or both of the hindlimbs became flaccid. In the most dramatic cases, the animals would drag their hindlimbs as they walked. However, a flaccid tail did not mean the animals were incapable of tail movement because short latency tail flicks sometimes occurred in animals showing this syndrome. It is not yet clear as to what role SP antagonism plays in these motor deficits. Nevertheless, motor neurons are excitable by SP (*21*) and a significant SP neuronal system descends from the brainstem to the spinal cord ventral horn where motoneurons reside (*22*).

The marked depression of capsaicin-evoked behaviors by D-Pro^2-D-Phe^7-D-Trp^9–SP supports the previous suggestion that SP antagonists are likely to be novel analgesic agents (*7*). The reason for the antagonist's lack of activity on acute thermal pain tests is not yet clear. It is possible that the stimuli or responses (or both) in such tests are too rapid or intense for this antagonist to block. The depression of tail-flick reflexes sometimes observed with high antagonist doses could have been interpreted as analgesic activity if the motor deficits did not appear. However, it is also possible that the neural systems for acute thermal pain are anatomically or neurochemically distinct from those responsible for inflammatory pain. This hypothesis is supported by neurophysiological experiments associating specific nociceptor types with specific primary afferents (*23*).

Although some important details remain to be settled, sufficient evidence now exists to accept SP as a highly probable central neurotransmitter for primary nociceptor afferents. These results confirm the 1935 suggestion of Dale (*24*) that the sensory neurotransmitter for inflammatory pain should be the mediator of the "axon reflex" since SP has recently been demonstrated to possess such a mediator role (*25*).

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Chemical Self-Defense by Termite Workers: Prevention of Autotoxication in Two Rhinotermitids

Abstract. *Soldiers of the lower termites* Prorhinotermes simplex *and* Schedorhinotermes lamanianus *(Isoptera, Rhinotermitidae) have electrophilic contact poisons used in colony defense. Workers of these termites die when exposed to the defense secretion of the other species, but survive when exposed to chemicals from conspecific soldiers. Detoxication occurs by an initial substrate-specific reduction of the electron-deficient double bond of the nitroalkene (*Prorhinotermes simplex*) or vinyl ketone (*Schedorhinotermes lamanianus*) followed by complete catabolism to acetate.*

Chemical defense has evolved numerous times in the termite families Rhinotermitidae and Termitidae (*1*). Three defensive strategies are employed by chemically armed soldier termites (*2*): (i) biting, with the addition of an oily, toxic or irritating secretion from the frontal gland reservoir (Termitinae), (ii) brushing a copious amount of a hydrophobic contact poison onto the cuticle of an attacker (Rhinotermitidae), or (iii) ejection of an irritating glue-like secretion (Nasutitermitinae). Each of these modes of chemical defense requires a strategy for chemical self-defense (*3*), that is, a mechanism for conspecifics to avoid intoxication. Termite workers of the species *Prorhinotermes simplex* (Prorhinotermitinae) survive exposure to vapors and liquid forms of a volatile, toxic nitroalkene (*4*) secreted by conspecific soldiers during an attack on the colony. Similarly, *Schedorhinotermes lamanianus* (Rhinotermitinae) workers survive exposure to volatile vinyl ketones (*5*) produced by their soldiers in response to disturbance. We now report that the termite workers of these two species have substrate-specific alkene reductases which, in the presence of a reduced nucleotide cofactor, catalyze the reduction of the electron-deficient double bond of the unsaturated electrophilic group. The saturated compounds are subsequently recycled in vivo by way of catabolism to acetate. This represents a rare example of an electrophilic insecticidal compound that is detoxified by initial reduction rather than by oxidation or conjugation (or both) (*6*, *7*).

Radioactively labeled defense secretion compounds were synthesized (Fig. 1) (*8*). Condensation of [1-^{14}C]tetradecanal (*8*) with nitromethane in methanolic sodium methoxide (*4*) followed by stirring the nitroalcohol with acetic anhydride-pyridine gave (*E*)-1-nitro-1-[2-^{14}C]pentadecene (**1**) (0.05 mCi/mmole) in 37 percent chemical yield after chromatography and recrystallization from cold hexane. The vinyl ketone was prepared (*5*, *6*) by reaction of 11-[1-^{14}C]dodecenal (*8*) with vinylmagnesium bromide followed by oxidation of the allylic alcohol with manganese dioxide to give 1,13-[3-^{14}C]tetradecadien-3-one (**3**) (0.32 mCi/mmole) in 53 percent yield after chromatography. Unlabeled nitroalkene **1** and vinyl ketone **3** were also prepared for comparative toxicity studies and for monitoring detoxication by gas chromatography.

Twenty worker termites of *Schedorhinotermes lamanianus* (*9*) were treated with the labeled vinyl ketone [3-^{14}C]**3** by topical application of 100 μg per termite and were held in sealed dishes for 24 hours at 27°C. The termites were then homogenized in methanol, the extract was centrifuged, and the supernatant was subjected to reverse-phase high-performance liquid chromatography (*10*). Three peaks of radioactivity could be observed during gradient elution from water to methanol. Peak 1 (36 percent of soluble radioactivity) eluted at the solvent front and was shown to contain [^{14}C]acetate by purification of the *p*-bromophenacyl derivative (*8*). Peak 2 (21 percent) cochromatographed with the glutathione conjugate of **3** (*8*, *11*) under these high-performance liquid chromatography conditions; however, a higher resolution separation (*8*) revealed the absence of glutathione or cysteine conjugates, and the identity of this peak is uncertain. Peak 3 (43 percent) cochromatographed with starting vinyl ketone **3**, but lacked the ultraviolet absorption at

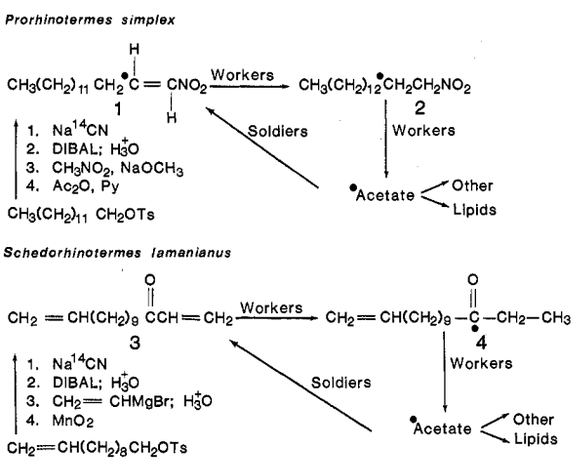

Fig. 1. Synthesis and detoxication of radioactively labeled soldier defense secretions. (Top) Nitroalkene **1** is converted first to nitroalkane **2** and then to acetate. (Bottom) Vinyl ketone **3** is converted first to ethyl ketone **4** and then to acetate. Biosynthesis of the nitroalkene **1** from acetate by soldiers has also been demonstrated (*8*).

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