in 1*M* NaCl extracts of calvaria in the experiments of Vuust and Piez (*8*).

Our studies demonstrate that a proteolytic activity, active at neutral *p*H, is capable of conversion of procollagen to collagen in bone. The existence of a preformed enzymatic activity is consistent with the observation that conversion of procollagen to collagen proceeds despite inhibition of collagen synthesis by cycloheximide. The enzymatic activity is not trypsin-like and is not a serine protease as indicated by lack of inhibition with soybean trypsin inhibitor or DFP. However, since relatively unpurified extracts were used as a source of enzymatic activity, it is possible that the physiologically important enzyme is susceptible to inhibition by these compounds and that, under these circumstances, conversion of procollagen is achieved in a less specific fashion by extraneous resistant enzymes in the extract.

Recently, cattle afflicted with a heritable disorder, dermatosparaxis, were found to contain a dermal collagen fraction which was defective in its fibrogenic properties and contained chains higher in molecular weight than α chains (*9*). This collagen fraction may represent procollagen, or a derivative thereof, which accumulates as a result of a defect in procollagen peptidase. Relatively large amounts of a collagen fraction having some of the characteristics of procollagen were also identified in the culture medium of normal human fibroblasts (*3*). Unlike procollagen, however, this medium fraction does not dissociate to pro-α chains under denaturing conditions. Procollagen might escape limited proteolysis in cell culture if procollagen peptidase were inhibited by a factor in the fetal calf serum used as a component of the culture medium or if the enzymatic activity were bound to the cell membrane and thence unable to efficiently cleave a substrate capable of diffusion into the medium.

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Epidemic Strain of Venezuelan Equine Encephalomyelitis Virus from a Vampire Bat Captured in Oaxaca, Mexico, 1970

Abstract. *A vampire bat,* Desmodus rotundus, *captured in Oaxaca, Mexico, in August 1970, was found to be infected with the epidemic strain of Venezuelan equine encephalomyelitis virus at the same time that an equine epizootic was occurring there.*

In 1968 an explosive and lethal outbreak of encephalitis in horses occurred in Guatemala (*1*). The strain of Venezuelan equine encephalomyelitis (VEE) virus that was isolated from horses there is antigenically related to the epidemic strain of VEE virus that has been isolated in Venezuela (*2*) and other Latin American countries (*3*). By 1969 this virus had spread to Costa Rica (*4*), and by 1970 into the southeastern Mexican states of Chiapas and Oaxaca (*5*). In early 1971 the virus appeared to have "jumped" into the Tampico, Tamaulipas (Mexico) area, with the occurrence of equine deaths there, also (*6*).

The existence of VEE virus in Mexico was first established in 1962 (*7*), but its geographic range appeared limited to the southeastern states of the republic (*8*). In 1966, an equine encephalitis epizootic in the Tampico area (*9*) killed approximately 300 of 1000 horses. Of 231 surviving horses 52 (22.5 percent) had hemagglutination-inhibiting antibodies to VEE virus 3 months after the epizootic (*10*).

During the 1970 outbreak in Mexico various specimens were collected from the affected area of eastern Oaxaca and tested. We now report on one isolation from that study.

Eighteen bats were examined for virus: these included six *Balantiopteryx plicata,* eight *Mollosus* sp., and four unsexed vampire bats (*Desmodus rotundus*). The vampires were collected at an abandoned well in San Francisco Ixhuatan, Oaxaca, on 10 August 1970. At the time of capture they were exsanguinated by cardiac puncture; the blood was allowed to clot, and serum was removed, frozen, and stored. When the animals were killed, the submaxillary salivary glands, brown fat, brain, and viscera (lung, heart, kidney, liver, and spleen) were removed; separate, sterile instruments were used for each organ or set of organs; the specimens were frozen at −70°C. The specimens were taken to the Instituto Nacional de Investigaciones Pecuarias, Palo Alto, D.F., Mexico, and later sent to the Center for Disease Control in Atlanta, Georgia, where they were processed for virus isolation.

Four of six suckling mice inoculated intracerebrally with a 10 percent clarified suspension of the viscera of one of the four vampire bats (No. 111) died 2 days after the inoculation. The virus was established in suckling mice by two subsequent intracerebral passages. The titer of the material of the isolate after the third passage was $10^{10.8}$ suckling mouse intracerebral LD_{50}/ml. The isolate was identified as VEE virus by neutralization tests in weanling mice with the use of hyperimmune mouse ascitic fluid prepared

Table 1. Identification of bat 111 isolate as VEE virus by neutralization test performed in suckling mice. The results are expressed as the log of the neutralization index; VEE, Venezuelan equine encephalomyelitis; EEE, Eastern equine encephalomyelitis; WEE, Western equine encephalomyelitis. Strains are shown in parentheses.

Virus	Neutralization by hyperimmune ascitic fluid to:		
	VEE (FE3-7C)	EEE (NJO)	WEE (Fleming)
Bat 111	2.7	0	0
VEE (GJ9/1BJ)	4.7	0	0
EEE (NJO)	< 1.0	2.9	< 1.0
WEE (Fleming)	< 1.0	< 1.0	3.0

Table 2. Identification of bat 111 isolate as VEE virus, type IB by kinetic hemagglutination-inhibition test (*3, 22*). The optimum *p*H for hemagglutination was 6.0. Results are expressed as units inhibited.

Serum dilution	Antigen					
	Bat 111	283424 IB	ICA IB	3880 ID	Mena II IE	Mex64A99 IE
Costa Rica, IB						
1 : 320	> 32	> 16	> 16	> 16	≧ 32	> 32
1 : 640	> 32	> 16	> 16	≧ 16	16	> 32
1 : 1280	≧ 32	≧ 16	≧ 16	4	2	≧ 32
1 : 2560	16	8	8	2	2	2
Mena II, IE						
1 : 80	16	8	≧ 16	4	≧ 32	> 32
1 : 160	< 2	2	8	2	16	≧ 32
1 : 320	< 2	< 2	1	< 2	4	4
1 : 640	< 2	< 2	< 1	< 2	2	1

against the endemic Florida (FE3-7C) strain (*11*) of VEE virus (Table 1). Kinetic hemagglutination-inhibition studies (Table 2) demonstrated the close relation between the isolate and an epidemic strain of VEE virus, type IB. The titer of serum from bat 111 was 1 : 20 by a 90 percent plaque-reduction neutralization test in duck embryo cells (*12*); thus, circulating antibody and virus in the viscera were present simultaneously. Neither antibody nor virus was detected in the other 17 bats, nor was virus recovered from other tissues of bat 111.

Vampires subsist on a strict blood diet and frequently feed on horses; in fact, rabid vampire bats cause more than 10,000 equine deaths every year in Mexico (*13*). In horses infected with the epidemic strain of VEE, viremia titers may approach $10^{7.2}$ suckling mouse intracerebral LD_{50}/ml (*14*). This may be enough to cause infection of a susceptible vampire bat by ingestion of infected blood, since this species commonly consumes 20 to 25 ml of blood a day (*15*). On the other hand, this bat may have been bitten by mosquitoes that had taken a blood meal from an infected horse. Cases of fatal equine encephalitis had occurred within 500 yards of the location of capture of bat 111.

Many arboviruses, including VEE virus (*16*), have been isolated from bats collected in areas where these viruses are endemic (*17*). Moreover, Sulkin and his co-workers have accumulated data which indicate that bats could serve as ideal reservoir hosts for arboviruses (*18*). Vampires cohabit with many other species of bats (*19*), including the Mexican freetail (*Tadarida brasiliensis mexicana*) (*19, 20*), which migrates in massive numbers to the southwestern United States every spring (*21*), and there appears to be ample opportunity for transfer of the virus to areas far from an infected focus.

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Δ^9-Tetrahydrocannabinol: Dose-Related Effects on Timing Behavior in Chimpanzee

Abstract. *Δ^9-Tetrahydrocannabinol, at doses within the effective range for humans, was administered orally to chimpanzees with stable, efficient timing performances maintained by multilink chained schedules of food reinforcement. Reinforcements decreased with increasing dose, because of decreased frequencies of total operant timing responses and decreased accuracy of the timing performances which did occur. Higher doses exerted an effect for up to 3 days.*

One difficulty encountered in interpreting results from animal studies of Δ^9-tetrahydrocannabinol (Δ^9-THC), the presumed principal psychoactive constituent of marihuana, has been the high doses generally needed to produce behavioral effects. This drug, in oral doses of about 0.200 mg/kg, has been reported to produce marihuana-like effects in humans (*1*). As part of an investigation of its behavioral and toxicological effects, oral doses ranging from 0.125 mg/kg to 4.0 mg/kg were administered to three chimpanzees (*2*)