UPDATE OF TRANSMISSION STUDIES 19.1.94

SE 1802 Transmissibility of BSE to pigs by injection with brain homogenate

The remaining four control pigs are clinically normal and due to be slaughtered in March 1994 (5 years post inoculation).

Mouse bioassay of tissues from challenged affected and control pigs were initiated in November 1993.

SE1803 Transmissibility of BSE to pigs by oral exposure to brain homogenate

The challenged pig killed 40 month p.i. due to intercurrent disease showed no significant pathology on histopathological examination. Remaining 4 challenged and 5 control pigs are healthy (44 months p.i.)

Mouse bioassay of tissues from 19-24 month kill of challenged and control pigs (84-93 to 97-93) are currently incomplete.

SE1804 Transmissibility of BSE to cattle by oronasal exposure to placentae of affected cattle.

The remaining 6 challenged and 4 control cattle are clinically normal (50 months p.i.).

Mouse bioassay of tissues from 24 month kill of challenged and control cattle complete but awaiting histopathological examination. No clinical signs suggestive of spongiform encephalopathy recorded.

SE1805 Transmissibility of BSE to domestic fowl by injection with brain homogenate.

1 challenged bird died overnight (42 months p.i.) following a period of ataxia - histopathological examination pending. 2 further challenged birds are also showing neurological signs of ataxia and tremor (43 months p.i.).

All affected birds are cock birds. The remaining 4 challenged hens are clinically normal (43 months p.i.).

2 control birds were culled due to intercurrent disease (40 and 43 months p.i.). No significant lesions were observed in one and histopathological examination is pending on the other.

The remaining 6 control birds are clinically normal.

SE1806 Transmissibility of BSE to domestic fowl by oral exposure to brain homogenate.

1 challenged cock bird was necropsied (41 months p.i.) following a period of ataxia, tremor, limb abduction and other neurological signs. Histopathological examination failed to reveal any significant lesions of the central or peripheral nervous systems.

1 other challenged cock bird is also showing ataxia (43 months p.i.). The remaining 2 challenged cocks and 5 hens are clinically normal (43 months p.i.).

Further examinations are in progress to determine the cause of morbidity in these studies (SE1805 and SE1806).

For controls see SE1805.

SE1809 Comparative efficiencies of the bioassay of BSE infectivity in cattle and mice.

All challenged and control cattle remain healthy (12 months p.i.).

Mouse inoculations were carried out in January 1993.

SE1812 Transmissibility of BSE to cattle by injection with brain homogenate.

The remaining 6 control cattle were necropsied 5½ - 6 years post inoculation. All were clinically normal. Histopathological examination is pending.

SE1813 Transmissibility of scrapie to pigs by oral exposure to brain homogenate.

A pool of homogenised brains from confirmed sheep scrapie cases was fed to a challenge group of twelve piglets in November/December 1993. A group of 12 piglets receiving only the normal ration act as controls.

All challenged and control pigs are healthy.

An aliquot of the pool of homogenised sheep scrapie cases was prepared for a standard end point titration of infectivity by mouse inoculation in October 1993.

SE1901 Pathogenesis of experimental BSE in cattle

Kill 6 (22 months p.i.) was completed in October 1993. Control and challenged cattle were clinically normal and no significant lesions were observed on histopathological examination of spinal cord and medulla.

Remaining challenged and control cattle are currently clinically normal (25 months p.i).

Mouse bioassay for infectivity of tissues

	Date of inoculation	Status
Kill 1	113-92 to 268-92	Incomplete
Kill 2	275-92 to 49-93	Incomplete
Kill 3	56-93 to 124-93	Incomplete
Kill 4	126-93 to 196-93	Incomplete
Kill 5	203-93 to 13-94	Incomplete
Kill 6	327-93 to date	Incomplete

SE1902 Effect of oral inoculum dose on attack rate and incubation period of BSE

A few mild clinical signs have been observed in some of the attack rate animals (24 months p.i.) but these, as yet, are not sufficiently manifest to be confident of a positive clinical diagnosis.

S A C HAWKINS G A H WELLS