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ERGOT OF CEREALS: A LITERATURE REVIEW AND SURVEY OF INCIDENCE IN TRADED GRAIN

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by

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Final report of an eight month project at ADAS – Cambridge. The work commenced in February 1992 and was funded by a grant of £11,300 from the Home-Grown Cereals Authority (Project No. 0001/1/92).

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1. SUMMARY

Although the pathogen Claviceps purpurea seldom causes serious losses of yield, the alkaloids which its sclerotia ('ergots') contain are so toxic both to humans and to their animals that ergot contamination of grain lots greatly reduced their value. Postal surveys of grain merchants revealed that contamination of grain could not only reduce the price paid to the farmer but might lead to the complete rejection of consignments. The surveys showed the incidence of contamination to be particularly high in spring wheat and rye in the East Midlands and East Anglia.

Mature ergots may fall to the ground or be harvested with the grain. In the latter case, they may serve to introduce the pathogen into other fields if the contaminated grain is used for seed. In either case, they lie dormant over winter, germinating (after a period of chilling) in the spring. The fruiting bodies which develop from the germinating ergots produce 'ascospores' which are able to infect the cereals or grasses at the time of anthesis. Spores of another kind ('conidia') are produced on the first infected inflorescences and these, transmitted by insects or rainsplash, serve further to spread the disease within the crop. Ultimately, infected florets produce more sclerotia in place of healthy grain. Sclerotial germination, spore production and infection are all very dependent on weather conditions. Levels of the disease are likely to be highest in cool, wet seasons.

Cross pollinated cereal species, in which the glumes gape for a long period during anthesis, are most susceptible to the disease. Thus, rye, triticale and durum wheat are more susceptible to infection than are bread and feed wheats and barley. In closed flowering species, any factor which reduces pollination, and/or causes the glumes to gape longer, increases the risk of infection.

Early flowering grasses can act as alternative hosts for the fungus allowing it to build up within or around a field before the cereal crop flowers.

Strains of the pathogen especially well adapted to particular species or groups of species are known to exist, but the strains appear not to be very distinctly differentiated and to vary from area to area. In England, a strain which attacks blackgrass will spread very readily to wheat, and ergot is most frequently found on wheat in fields infected with this weed.

Animals and poultry fed on a contaminated grain (or grazing contaminated pastures) can suffer severely from the toxic effects of the ergot alkaloids. Symptoms vary with dose and with the species affected but include gangrene (leading to loss of extremities), abortion and, in extreme cases, death.

Ergotism in humans is now rare but in past centuries has sometimes reached epidemic proportions in rye growing areas. Gangrenous and convulsive forms of the disease have been recorded, the latter apparently having been more prevalent where the diet of sufferers was deficient in vitamin A.

Despite their toxicity, ergot alkaloids have found a number of uses in medicine, more particularly in obstetrics and in the treatment of migraine. Cultivation of ergot for pharmaceutical use has been practised in many countries since the Second World War.

Strategies advocated for the control of ergot include the use of uncontaminated seed, rotation of crops, deep ploughing to bury the sclerotia and the control of weed grasses. The use of fungicides sprayed onto the ground to prevent infection, has been tried but (at present) finds no place in commercial practice. The use of biological control agents has also been investigated. Certain fungicides applied as seed treatments will suppress the germination of sclerotia occurring as contaminants in the seed lot.

There is a need for further research on the role of weed grasses in ergot epidemiology, and on the effects of the new generation of fungicides on the infection process. Spray timing trials using these materials on carefully monitored plots artificially contaminated with sclerotia could yield valuable information on both epidemiology and control.

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2. GLOSSARY

AETIOLOGY - the science of the causes of disease.

ANTHER - the plant organ in which pollen is produced. In grasses and cereals, the male organs of the plant (the stamens) are comprised of anthers borne on fine stalks (filaments) which, at the time of anthesis (q.v.), elongate to push the anthers out through the gaping glumes.

ANTHERIDIA - the male organs of the lower plants and fungi. In the Ascomycetes, antheridia are cylindrical cells at the tips of hyphae which grow alongside the ascogonia (q.v.). At fertilisation, the contents of the antheridium pass into the ascogonium within which nuclear fusion takes place.

ALKALOIDS - nitrogenous organic compounds produced in certain plants and fungi, apparently as the end products of metabolic processes, which have no known function in the plants themselves but which are often physiologically active in higher animals. The alkaloids of higher plants include drugs such as morphine and quinine and stimulants such as nicotine and caffeine.

ANTHESIS - that stage in the flowering of grasses and cereals when the anthers are exposed outside the glumes. In closed-flowering, self-fertile species, pollination has already occurred within the floret before the glumes gape to allow the anthers to emerge. In open-flowering, cross pollinated species, the glumes gape for a much longer period to allow pollen from other plants to reach the stigmata.

ASCOGONIA - the female organs of Ascomycetes from which, after fertilisation by the antheridia (q.v.), the asci (q.v.) develop.

'ASCOMYCETES' - fungi belonging to the Subdivision ASCOMYCOTINA, the members of which are characterised by the production of 'asci' (q.v.).

ASCUS (pl. ASCI) - a sac-like cell, produced after a sexual process involving the fusion of two nuclei, in which 'ascospores' (q.v.) are formed.

ASCOSPORES - spores formed within an ascus after the nucleus has undergone 'reduction division' - i.e. after it has divided in such a way that the pairs of chromosomes brought together by earlier nuclear fusion are again separated

(one of each pair passing into each of the daughter nuclei). Usually two further divisions follow the 'reduction division' to produce eight nuclei in each ascus and around each nucleus a single ascospore is formed.

CAPITULUM (pl. CAPITULA) the terminal rounded head of a clava within which perithecia (q.v.) develop.

CLAVA (pl. CLAVAE) - a club-like stroma (q.v.)

CONIDIUM (pl. CONIDIA) - vegetative spore, the formation of which is not preceeded by nuclear fusion and reduction division (cf. 'ascospore').

GLUMES - the chaffy scales enclosing the florets of grasses and cereals.

HYPHA (pl. HYPHAE) - one of the fine threads or filaments of which all fungal structures are composed. Hyphae may be vegetative or spore-bearing.

OVARY - the organ which encloses the ovules from which, after fertilisation, the seeds of flowering plants develop.

PERITHECIUM (pl. PERITHECIA) - a flask-shaped fungal organ within which asci develop.

PHLOEM - the vessels within the vascular systems of the higher plants which are primarily responsible for translocating the sugars produced in the leaves to other parts of the plant.

SCLEROTIUM (pl. SCLEROTIA) - a firm to very hard mass of fungal hyphae, sometimes incorporating tissues from the host plant, which enables the fungus to survive in the absence of its host and which eventually germinates to produce vegetative hyphae or, as in the case of ergots, spore-bearing stromata (q.v).

STIGMA (pl. STIGMATA) - the receptive organ (in cereals and grasses a feathery structure) to which pollen grains adhere and on which they germinate, the pollen tubes then growing down the stigma to the ovary.

STIPE - the stalk of a fungal organ; the lower part of a clava which bears the capitulum.

STROMA (pl. STROMATA) - a body, formed of mass of vegetative hyphae, on or within which fungal spores are produced.

VASO-CONSTRICTION - constriction of the blood-vessels.

XYLEM - the vessels within the vascular systems of the higher plants which translocate water and mineral nutrients from the roots to other parts of the plant.

3. INTRODUCTION

Although it seldom causes serious losses in yield, ergot (Claviceps purpurea) ranks as one of the more important fungal diseases of cereals and grasses as the highly toxic alkaloids it produces render contaminated grain or feed poisonous to both animals and man.

Because of their toxic nature, and because their presence in contaminated seed can serve to introduce the disease into a field, very low tolerance levels are set for the presence of ergot sclerotia in grain lots, whether the grain is destined for milling, feed or seed. The presence of the disease can thus greatly reduce the price the farmer receives and can greatly increase the cost of cleaning the grain for sale to the end user.

Historically, the disease has been of even greater significance as the cause of the 'ignis sacer', the 'holy fire,' which often decimated whole populations in medieval Europe. As late as 1772, the fungus is known to have changed the course of history when a severe outbreak of ergotism amongst his cavalry obliged Peter the Great of Russia to abandon a campaign against Turkey (Ramsbottom, 1954).

On a more positive note, the toxins produced by the fungus have medicinal properties (they were being used in obstetrics as long ago as the 16th century) and the cultivation of ergot for pharmaceutical purposes has been practised in many countries (Sastry, 1986).

This review examines the economic significance of the disease with a special reference to the United Kingdom (UK) and presents the results of a recent survey on how merchants perceive its importance, describes the causal organism of the disease, the symptoms it produces, its life history and the effect of weather on its aetiology. It discusses the factors influencing the susceptibility to infection of our major cereals and looks at the role of grasses as alternative hosts of the pathogen. The importance of the ergot alkaloids is discussed both with reference to their mammalian and avian toxicity and to their use in medicine. Cultural, chemical and biological control measures are outlined and the need for further R&D work on the pathogen is discussed.

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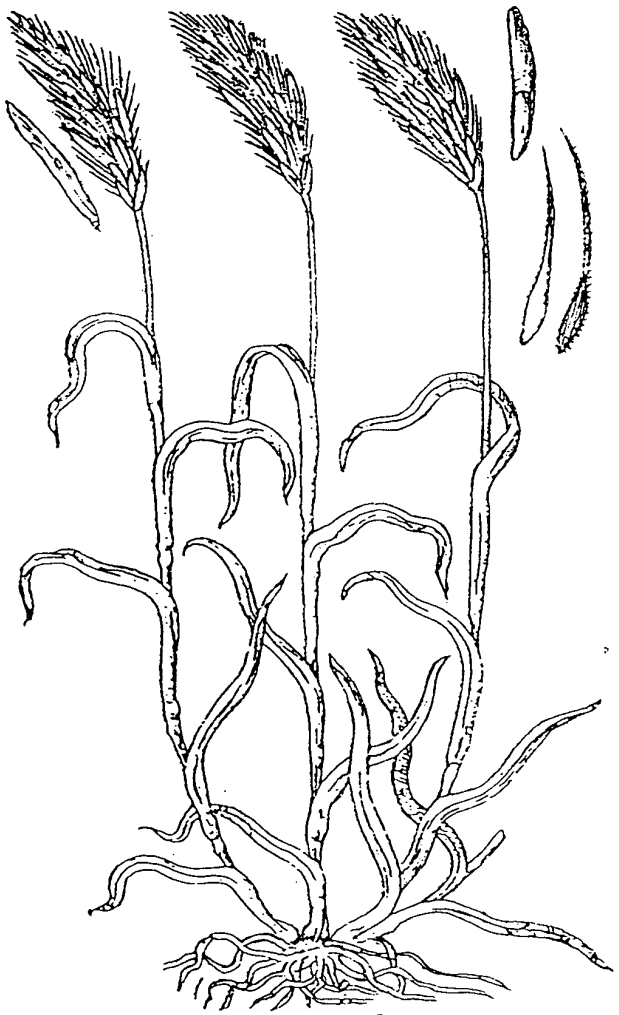

Fig. 1. The first reference to ergot in Adam Lonicer's 'Kreuterbuch', 1582. The first illustration of ergot from Casper Bauhin's "Theatrum Botanicum", ed 1658 (both produced from Barger, 1931)

4. THE ECONOMIC SIGNIFICANCE OF ERGOT

Yield losses caused by ergot are generally very low except in the very occasional seasons when severe epidemics occur on susceptible species of cereals. Melhus & Kent (1948), for example, noted that in the years 1932 to 1934 inclusive, the disease reduced the yield of rye (the most susceptible of cereals) over the whole of the USA by only 0.5%. However, the same authors noted that in 1921, when a severe epidemic affected durum crops in the northwest of the United States, it was not uncommon to find fields with 50% of the ears attacked, and wheat offered for sale showed 10% ergots by weight.

More important than the loss of yield, however, is the effect of ergot on the value of the grain. In 1921, buyers of the Crosby Milling Company at Minneapolis, directed to purchase 20 railcars of durum wheat, could find only eight carloads which they considered suitable for the manufacture of macoroni. Of these eight, seven were rejected at the mill because of the presence of ergot (Melhus & Kent, 1948).

Compared with losses such as those described above, ergot causes only insignificant yield loss in cereals in the UK. Nevertheless, the effect of contamination on the saleability of the grain can still cause serious problems to the farmer. In the 1978 epidemic in the North of Scotland (the worst experienced in the UK in recent years), levels in grain lots seldom reached 0.5% by weight. However, 0.5% was well above the legal limit of 0.1% by weight in grain destined for compound feed (Anon., 1976) and the even lower limit (0.05%) for wheat sold into intervention.

In recent years, farmers' problems in disposing of ergotised grain have been exacerbated by a continued tightening of the regulations. EC directives laid down that basic seed should contain no more than 1 piece of ergot, and certified seed not more than 3 pieces of ergot, per 500g. The UK higher voluntary standard demanded that for basic seed no ergots, and for certified seed no more than one ergot, should be present in the 1 kg sample examined. Given a normal seed rate of 157 kg/ha (1.5 cwt/acre), one piece of ergot in 1 kg seed could provide 3460 sources of inoculum in a 20 hectare field. By the early 1980s, there was already a growing feeling in the industry that this threshold was too high. In 1989, the United Kingdom Agricultural Seed Trade Association (UKASTA) fixed yet tighter standards for ergot contamination for

feed grain setting a limit of 0.001%, for all other grain the standard was set at zero (Anon., 1989).

4.1 Merchants' perceptions of the incidence of ergot in cereals

4.1.1 Survey of ergot contamination in grain yield stocks in 1991 and 1992

To ascertain the seriousness with which ergot is currently regarded by seed merchants in different areas of the UK, a questionnaire was prepared by the ADAS Ergonomics Unit (see Appendix I) and The United Kingdom Agricultural Supply Trade Association Ltd (UKASTA) kindly agreed to send this out (with pre-paid reply envelopes) with their July newsletter. About 700 UKASTA members received the questionnaire, but of these only some 300 would have been involved in the grain trade. Forty three were returned but 4 of these could not be used in the analysis (little or no grain bought in past 2 years, or information given too sparse to be useful). One additional questionnaire was filled in by a merchant who was not a member of UKASTA. The returns came from both large and small businesses. Not everyone indicated the tonnages they handled, so it was often impossible to relate the incidence of ergot to the total number of grain lots.

The results obtained are summarised in Table 1.

TABLE 1. Responses from grain merchants to the ergot questionnaire

Areas from which grain was bought	Number of merchants replying	Number seeing ergot in past two years
1. Scotland, Cumbria	10	2 (barley in Scotland, wheat in Northumberland)
2. Yorkshire, Lancashire, Cheshire, Lincolnshire, Humberside	5	3 (wheat)
3. West Midlands and Wales	5	4 (wheat)
4. Norfolk, Suffolk, Essex, Herts Cambs, Beds, and Bucks.	12	9 (wheat, barley, oats, rye)
5. Wiltshire, Gloucestershire Dorset, Oxon, Berkshire, Hants, I.o.W.	5	2 (wheat and barley in Oxon, & Gloucestershire)
6. Cornwall, Devon, Avon & Somerset	3	0
TOTAL	40	20

It will be seen that half the merchants returning the questionnaires had encountered ergot during 1990 and 1991. The incidence of grain samples contaminated with ergots was highest in Eastern and Central areas and lowest in the far north and the south west.

Area 1 (Scotland & Borders). The one Scottish merchant who reported finding the ergot dealt only in barley. He had rejected 2 loads in 1990 and 3 in 1991 because of ergot contamination. While he could not be certain of the actual location, he thought that the source of the contaminated grain was in the Grampian Region. His experience as an adjudicator in other cases led him to believe that the disease was increasing in his area (Grampian, Banff, Buchan & Moray). Only one case was reported from the Border counties of England.

Area 2 (North Central). One merchant, who in the past 2 years had handled over 2000 consignments of winter wheat from North and East Yorkshire and Lincolnshire, had rejected over 200 consignments because of ergot contamination. The disease appeared to be most common in the Whitby area and on the wolds of north Lincolnshire. A second merchant operating in the Masham area of Yorkshire had, over the past 2 years, rejected 2 out of 200 wheat

samples and 2 out of 260 barley samples because of ergot. A third respondent whose trading area extended from South Yorkshire to Northumberland had encountered 2 contaminated consignments in 1990 and 4 in 1991, all from South Yorkshire.

Area 3 (the West Midlands & Wales). The 4 merchants reporting ergot from this area had provided detailed information which is summarised in Table 2.

TABLE 2. Ergot incidence in the West Midlands and Wales in 1990 and 1991

	Winter wheat	Spring wheat	Barley	Oats	Triticale
No. of consignments handled in 1990 and 1991	4,145	127	2,050	175	3
No. for which price was reduced due to ergot	14	10	0	0	0
No. rejected due to ergot	2	0	0	0	0
Total samples with ergot	16	10	0	0	0
Samples contaminated as % total samples	0.4	7.9	0	0	0

The merchants in this area operated in the counties of Shropshire, Herefordshire, Staffordshire, west Worcestershire, Powys, Clwyd and north Monmouthshire. Within this area, ergot was reputed to be most common in Shropshire and Herefordshire.

Area 4. East Anglia & North Home Counties. Returns received from this area did not allow for such ready analysis as did those from the West Midlands. However, the information from three large operators may serve to illustrate the relative incidence of the disease on the various cereal species (Tables 3, 4 & 5). Note that in Tables 3 & 4, the information on quantity of grain bought is presented in terms of tonnes rather than of consignments.

TABLE 3. Data from a merchant operating in Norfolk, Suffolk and Cambridgeshire

		Winter wheat	Spring wheat	Barley
Tonnage handled	1990	135,000	9,000	10,000
	1991	164,000	12,000	12,000
% for which price was reduced due to ergot	1990	nil	15%	nil
	1991	nil	20%	nil
Price reduction/tonne			£14	
No. of loads rejected due to ergot	1990/1	nil	10	nil

Contaminated grain was not recleaned but was downgraded to feed.

TABLE 4. Data from a merchant operating in Essex, Suffolk, Cambridgeshire and Hertfordshire

		Winter wheat	Spring wheat	Durum	Barley	Oats	Rye
Tonnage handled	1990	107,000	2,000	2,000	12,500	2,000	150
	1991	110,000	2,000	2,000	15,000	2,500	200
Tonnage for which price was reduced	1990	500	100	100	200	nil	nil
	1991	250	50	50	nil	50	nil
Price reduction/ per tonne		£10	£10	£10	£10	£10	-
No. of loads rejected in 1990 and 1991		7	5	4	2	2	1

TABLE 5. Data from a merchant operating in Norfolk, Suffolk and Essex

	Winter wheat	Spring wheat	Durum	Barley	Oats	Rye	Triticale
No. of consignments handled in 1990 and 1991	5,200	120	20	3,100	120	450	22
No. for which price was reduced	5	6	0	0	0	0	0
Price reduction	£5	£5	-	-	-	-	-
No. of loads rejected	4	4	0	0	0	0	0

Spring wheat and (at least in one merchant's experience) durum were particularly prone to contamination with ergot. One correspondent observed that the spring wheat cv. Tonic appeared to be more susceptible than cv. Axona to infection by Claviceps purpurea.

In view of the well attested historical association between ergot and rye (Barger, 1931), surprisingly little ergot was found in the East Anglian rye crops. Apart from the crop rejected by the merchant cited in Table 4, only one other case of ergot in rye was recorded on any of the questionnaires received.

That ergot caused one merchant to reject 50 tonnes of oats is noteworthy as the disease is very rarely found on this crop.

Area 5. South. Returns from this area suggested that ergot was particularly severe in the Cotswold area of north Oxon, and Gloucestershire. One merchant operating over a wide area of southern England commented that the ergots were seldom encountered in grain grown south of the M4. The higher susceptibility of spring than of winter wheat was again noted. Price reductions due to the presence of the ergot ranged from £3/t to £15/t and the disease caused occasional samples to be downgraded from milling to feed, or from feed to export status.

In summary, the survey suggested that ergots were rarely found as contaminants in barley or oat grain. Winter wheat was more frequently affected but it was spring wheat and (in the few areas where it was grown) durum which were most frequently contaminated with the disease. The information on rye and triticale was too limited to draw firm conclusions on the incidence of the ergots in grain from these two species but, given that rye is generally regarded as the most susceptible of the cereals, it was surprising that so few cases of ergot contaminated rye grain were reported.

Price reductions for contaminated crops ranged from £2/t to £15/t but many respondents said that they simply would not buy contaminated grain. A typical comment from one large company was that, "All grain ... will be rejected if one piece of ergot is found. It is then the responsibility of the supplier to clean or dispose of the grain. All grain is thoroughly tested before purchase and will not be purchased if ergot is present unless it has been thoroughly cleaned using a gravity separator".

Most of those responding to the question "was there evidence that ergot was associated with grass weed infestation?" answered in the affirmative. One, however, implied that while this was true for ergot on winter wheat it was not true for spring wheat or barley.

A number of respondents made full and helpful use of the "Comments" box to tell of their experiences of past "ergot years" even if they had not encountered the problems in 1990 or 1991. Comments included the following:-

"We find ergot is very seasonal and we have not had a bad season for ergot for some years. There is no doubt that there is a connection between grass weeds and infected fields .... We have never had a problem in marketing the very small % which we occasionally observe" (Scottish Borders).

"Ergot seen very little in last 2 years although earlier seasons have occasionally been bad. I guess one year in 6 or 7 years is bad" (Essex).

"We consider ... ergot to be prevalent in some seasons and not others. There have been only 2 very bad ergot years in the last 30 in Bedfordshire and Cambridgeshire and probably another 3 or 4 years when it caused moderate problems."

"Always suspicious of spring sown wheat, [ergot] was prevalent in spring wheat of 1990 harvest and worse in 1988 harvest. ... Associate ergot with low fertility conditions" (Oxfordshire/Gloucestershire/Warwickshire).

"Ergot, although present most years to a greater or lesser degree is only a problem about one year in ten. 95% of the mills will take slight traces on an allowance basis" (Herefordshire/Worcestershire/Shropshire).

"From over 25 years of experience of grain buying ... it is clear that the incidence of ergot contamination is directly attributable to the prevailing weather conditions at flowering and the degree of grass weed infestation within the crop" (Hertfordshire/Bedfordshire/Buckinghamshire).

"We have seen several bulks of wheat on farms contaminated with ergot but only two incidents involving actual deliveries of milling wheat being affected. On these occasions the consignments of wheat were rejected and returned to the farm. This incurred a double cost to the grower including additional haulage and a reduced feed wheat price. The most recent case of this type produced a reduced price of approx £20 per tonne. All incidents of ergot that we have seen have been associated with grass seed infestation" (Shropshire/Staffordshire/Herefordshire).

"The majority of wheat that we are offered direct by farmers, contaminated with bunt or ergot, we attempt to market for pig feed and always advise the buyer before negotiating the sale. As a service to our customer, we always advise on how they can preclude a re-occurrence of the problem, whether it be

bunt or ergot ... We would never accept any wheat contaminated with either ergot or bunt into our flour mill, no matter how small the level. Similarly we would not look to sell any 'contaminated' grain to any end user, other than a pig farmer who was made aware of the problem first. We find that certain of these 'home mixers' take advantage of the discount price available but only include into rations at very low percentages"
(Yorkshire/Lincolnshire/Nottinghamshire).

And finally a plea from a store-keeper:-

" I feel that ergot contamination is on the increase. It is difficult to detect unless above the 0.5% level. The futures contract and feed barley contract should not have a 'nil' clause since it means that the store-keeper has to be perfect and that is impossible even if the intake system is extremely strict" (Scotland).

Merchants who returned our questionnaire in 1992 were invited to furnish additional information on grain bought after 1992 harvest. 16 replies were received and of these 5 reported that ergot contamination had been encountered. The replies suggested, that ergot had not been a serious problem after the 1992 harvest but, even so, in parts of East Anglia up to 8% of spring wheat samples were contaminated and this resulted in price reductions averaging £24/tonne.

4.1.2 The ten-year survey

While the results obtained from the 1990/91 survey, and from the more limited survey carried out after the 1992 harvest, were of considerable interest, it was accepted that these years may not have been typical in terms of the incidence of ergot. It was decided, therefore, seek additional information on merchants perceptions of how serious a problem ergot contamination had been over a ten year period. A brief report on the previous findings was prepared for a newsletter which was sent out to all UKASTA members on 31 March 1993. An accompanying questionnaire sought information (for winter, spring and durum wheat, winter and spring barley, oats, rye and triticale) on:-

i) the number of years during the past decade in which ergot had been perceived as a major problem.

ii) the proportion of consignments which, in the worst years, had been so badly contaminated that they were:-

a) rejected

b) bought at a reduced price.

Seventy one merchants returned the questionnaires but, though all supplied valuable information, some presented it as subjective comments which it has not been possible to include in the following table (Table 6). Information on durum wheat and oats is not included in the table as very few merchants traded in the fomer commodoty, and only one reported problems in the latter.

Because of differences in the trading areas of the merchants responding to the two surveys the regional break-down of information in table 6 is slightly different from that given in Table 1.

TABLE 6. Responses of grain merchants to the second UKASTA Questionnaire

Areas from which grain was bought	Crop	Number of respondents regarding ergot as a problem in the number of years incicated.							
		0	1	2	3	4	5	6	7
1. Scotland and Northumberland	W. wheat	6	2	-	1				
	S. wheat	2							
	S. barley	0	1						
2. Lincolnshire, Yorkshire Co Durham	W. wheat	8	1	1	1	1			
	S. wheat	7	1	1	-	2			
	W. barley	3							
	S. barley	3							
	Rye	3							
	Triticale	3	-	1					
3. West Midlands and Wales	W. wheat	4	1	1					
	S. wheat	1	1	2					
	W. barley	0	2	1					
	Rye	0	0	0	1				
4. East Midlands	W. wheat	-	-	4	1				
	S. wheat	-	-	1	4	-	-	1	
	W. barley	2	1	-	-				
	S. barley	2	-	-	-				
	Rye	-	-	-	-	5	-	-	1
5. East Anglia and north Home Counties	W. wheat	2	7	2	1	1			
	S. wheat	1	3	2	3	1	1		
	W. barley	6	1						
	S. barley	6	2	1					
	Rye	1	1	-	1				
	Triticale	2							
6. Southern England	W. wheat	4	2	1					
	S. wheat	4	-	2					
	W. barley	4	1	-					
	S. barley	4	-	-					
	Rye	1	-	-					
7. South West	W. wheat	2	1						
	S. wheat	3							
	W. barley	2							
	S. barley	2							

The survey data show ergot contamination to have presented most frequent problems on wheat (especially spring wheat) amd rye in the East Midlands and East Anglia.

The years considered to have been particularly bad for ergot contamination are indicated in table 7.

TABLE 7. Years in which ergot contamination was particularly high

Areas from which grain was bought	Crop	Number of respondents regarding ergot as a problem in the harvest years indicated.									
	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992
1. Scotland and Northumberland	1	-	-	1	-	1***	-	-	1	-	-
2. Lincolnshire, Yorkshire C Durham	-	1	1	1	-	2	1	-	1	1	-
3. West Midlands and Wales	-	-	-	-	-	-	-	-	-	2**	2**
4. East Midlands	-	-	-	-	-	1	2	1	1	-	-
5. East Anglia and North Home Counties	-	1	-	1	1*	5	2*	-	1	-	2
6. Southern England	-	1	-	-	-	-	1*	-	1	2	-
7. South West	-	-	-	-	-	-	-	-	-	-	1
TOTAL	1	3	3	3	1	9	6	1	4	4	5

* especially on spring wheat

** rye and winter barley especially badly affected

*** especially on spring barley

The figures in table 7 should be treated with a degree of caution as tighter restrictions on the sale of contaminated seed has heightened awareness of the disease in the past few years and may have altered merchants' perceptions of what constitutes a problem year.

A few examples may help to illustrate the cost of contamination to the industry in high ergot years in areas where the disease is severe:-

i) in the 1991 and 1992 seasons one merchant in the West Midlands rejected 5% and 20% of rye samples because of ergot contamination. (in the same years only 0.1% of winter barley and 0.001% of winter and spring wheat samples were rejected).

ii) a merchant in the Eastern Counties indicated that in years of high contamination he could reject up to 5% of rye and spring wheat samples, and reduce the price on 25% of rye and 20% of spring wheat samples. In the same years rejections and price reductions in winter wheat samples would be less than 1%.

iii) a Suffolk merchant said that in the two bad ergot years he had recently encountered he had reduced the price on 25-30% of spring wheat, and 5-10% of winter wheat and spring barley samples.

It should be stressed, however, that these figures were extreme examples and very few merchants had encountered such high levels of contamination.

Other comments on the questionnaires highlighted the problems of ergot contamination in imported Canadian wheat and imported hybrid rye seed. The high levels of grass ergots in some UK wheat samples was also a cause for some concern. It was suggested the most of the contamination seen in barley and oats was due to the presence of grass ergots.

One respondent expressed the opinion that ergot levels had been higher in the 1970s when direct drilling of cereals was popular, another voiced concern lest incidence might provide higher after the ploughing out of set-aside if weed grasses were allowed to flower in the set-aside year.

ERGOT

(CLAVICEPS PURPUREA)

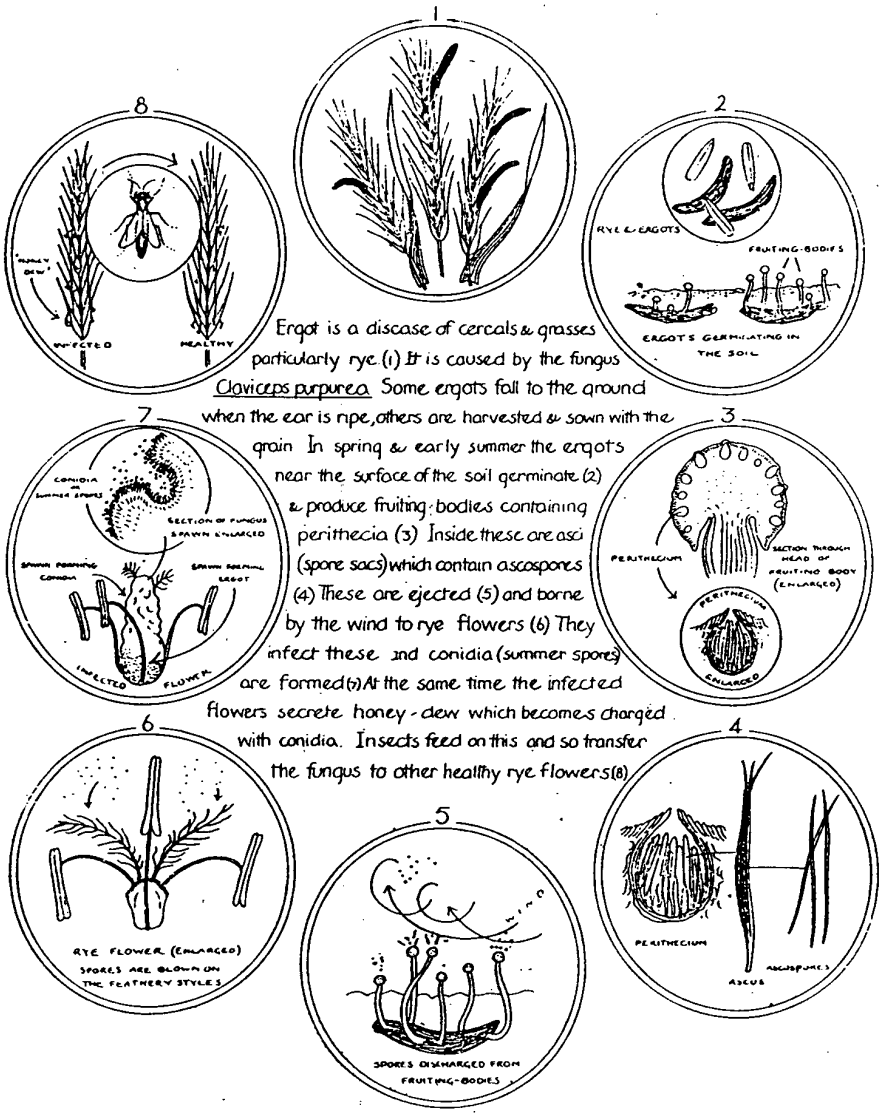

Fig.2. The life cycle of ergot, from Ann Murray's original drawing produced for inclusion in "Diseases of Cereals" by W A R Dillon Weston (1948).

5. THE CAUSAL ORGANISM

Claviceps purpurea, the organism which causes ergot, is a member of the 'Ascomycetes', a major group of fungi which contains many important plant pathogens. The group is characterised by the fact that sexual reproduction results in the production of spores in sacs or 'asci' from which, in most cases, they can be forcibly ejected and thus become air-borne. In most Ascomycetes, vegetative spores, known as 'conidia', are also produced; in this case, spore production is not preceded by sexual fusion. The genus Claviceps is a member of that group of Ascomycetes known as "Pyrenomycetes" in which the asci are produced in flower shaped "perithecia".

Over 30 species of Claviceps have been described. Three of these are found on sedges (Cyperaceae), the remainder on grasses and cereals (Gramineae). In general, each species of Claviceps is confined either to a single genus of grasses or sedges or, less frequently, to a group of related genera within a single tribe. C. purpurea is the exception to this rule in that it has a much wider host range and has been recorded on 200 species in 50 genera representing 17 tribes within the sub-family Pooidea (Loveless, 1967). A fuller account of the taxonomic position of C. purpurea is given in Appendix II.

6. SYMPTOMS AND LIFE HISTORY

The life cycle of the fungus is (first described by Tulasne, 1853) illustrated at Figure 2.

The word 'ergot' is derived from the Old French 'argot' meaning 'a cock's spur' (Fowler & Fowler, 1964). The name refers to the hard, purple to black, spur-like bodies (sclerotia) which protrude from the florets of infected cereals and grasses. These sclerotia differ in size and shape according to the host. In rye, they can be up to 30 mm long whilst in Agrostis spp, they may be no more than 2mm (Bradley-Jones, 1965). In rye, and many grasses, they tend to be thin and curved; in wheat they are thicker, shorter and straighter. Often they are little bigger than, and roughly the same shape as, the grains which they replace.

As the plant dies and its seeds ripen, so too the ergots in the ears reach maturity. In the case of cereal ergots, they may fall to the ground or be harvested with the grain. The use of contaminated grain for seed may serve to introduce the pathogen into other fields.

6.1 Germination of the sclerotia

Whether they fall naturally to the ground or are sown with the seed, the sclerotia lie dormant in the soil until, usually after a period of chilling, they germinate in the spring or early summer.

Sclerotia from any one source usually germinate over a period of about six weeks. In any one location, the time at which germination begins varies with the host species from which the sclerotia are derived. In a study by Wood & Coley Smith (1982) ergots from cocksfoot began to germinate in early April, those from barley, meadow foxtail and couch grass in late April/early May and those from wheat, triticale, ryegrass and several other grasses in mid- June.

The germinated sclerotia produce white stalks, known as 'clavae', the tips of which grow towards the light eventually changing colour through yellow and red to purple. As it grows, each clava becomes differentiated into a stalk, or 'stipe', and a rounded head or 'capitulum'. Stipe and capitulum together constitute a 'stroma' (pl. 'stromata'). Germinating sclerotia, clavae and capitulum are illustrated in Figure 3.

The rate of elongation of the clavae is more rapid in the darkness than in the light and the final cessation of elongation appears to be linked with a fall in relative humidity (Hadley, 1968).

The larger the sclerotium the greater the number of stromata it will produce (Cooke & Mitchell, 1966) and though a single cereal ergot usually produces about six such fruiting-bodies, many more (up to 60 per sclerotium) may sometimes be found (Butler & Jones, 1949). If stromata are destroyed before sporulation, others will develop to take their places (Cooke & Mitchell, 1967).

If a small sclerotium lies on the soil surface, the clavae it produces may be no more than 1mm long, but larger sclerotia produce longer clavae (Cooke & Mitchell, 1966) and those from buried sclerotia grow even longer to bring the

capitula above the surface of the soil. Bretag & Merriman (1981) found that more clavae were produced from sclerotia buried at 10 to 30 mm than from either those left on the surface or from those buried at 40 mm depth. Sclerotia buried at 50cm germinated but their clavae never reached the soil surface, so no capitula were formed.

6.2 Production and release of ascospores

Within the capitulum, a sexual process involving the fusion of nuclei from specialised 'male' ('antheridial') and 'female' ('ascogonial') cells takes place. This is followed by the development of flask-shaped cavities known as 'perithecia' which open through narrow channels in warty protuberances on the capitulum. The perithecia are lined with elongated sacs (asci) within each of which eight thread-like (1-2 μm x 70-120 μm) ascospores are produced.

Ascospore discharge normally begins about 8 days after the appearance of the stromata (Mantle & Shaw, 1976). The ascospores may exude from the perithecia to collect in a sticky liquid on the surface of the stroma to be dispersed by rainsplash or by visiting insects (Leach, 1940). More frequently, they are forcibly ejected (to heights of 20-80 mm) through the open necks of the perithecia to be caught up and disseminated by air currents. The clavae are negatively geotropic and positively phototropic. During ascospore release the stipes twist so as successively to turn different groups of perithecia towards the light (Barger, 1931). This ensures that the ascospores are ejected upwards and have the maximum chance of being caught up in air currents.

Ascospore release normally continues over a period of about five weeks (Wood & Coley Smith, 1982). It has been estimated that a single ergot may produce a quarter of a million ascospores (Hewett, 1982) and, even though most of these will fall harmlessly to the ground or land on plants of non-susceptible species, some are likely to reach the flowers of grasses or cereals.

6.3 The "sphacelia" stage

When deposited on the stigmata of susceptible grass or cereal flowers, the spores germinate to produce fungal threads ('hyphae') which grow round the ovaries to cause lesions at their bases. Through this wound, a mixture of xylem and phloem sap (equivalent to a 2.33 molar sugar solution) flows into the floral cavity thus providing the fungus with a source of nutrients and

aiding its colonisation of the ovary. Within a few days of infection, masses of vegetative spores ('conidia') are produced and are carried in the exuding sap, as a droplet of 'honeydew', to the outside of the floret. This conidial stage of the fungus was first described by Léveillé 1827 (cited by Barger, 1931) who named it Sphacelia segetum. It is still often referred to as the *sphacelia* stage of the life history. (*Sphacelia* is derived from the Greek *sphakelos* = gangrene).

It has been estimated that a millilitre (ml) of honeydew may contain as many as 10^8 conidia (Shaw, 1986). The sweet liquid is very attractive to insects which spread the pathogen to surrounding healthy plants where the process of infection and sporulation is repeated. Wind and driving rain may also serve to spread the conidia, as may contact between diseased and healthy ears as they move in wind, but insect transmission is of particular importance at this stage in the life history of the pathogen. Atanasoff (1920, cited by Leach, 1940) listed more than 40 different insects that had been observed feeding on ergot honeydew. Mercier (1911, cited by Leach, 1940) showed that the fungus gnat Sciara thomae could disseminate the spores both as external contaminants on its body or by their passing unharmed through its digestive tract.

Potentially, the spread of conidia by insects could lead to the widespread dissemination of the fungus within a field but there appears to have little research investigating this aspect of epidemiology. In one study in Finland, counts of ergots in susceptible rye fell by 95% within 30 yards of a severe, artificially infected source (cited by Hewett, 1982).

6.4 Formation, dispersal and overwintering of sclerotia

In many, but not all (Neill, 1941a), of the infected florets the 'sphacelia stage' is succeeded, after about 12 to 16 days, by the formation of sclerotia ('ergots') in place of the seeds. An infected ovary hardens as it is penetrated by the fungus and eventually it is impossible to distinguish its remains from the hard mass of fungal tissue which has replaced it. If an ergot is not formed in a floret, honeydew production may continue for a much longer period. When collecting ergots from lyme grass (Elymus arenaria), Wood & Coley-Smith (1982) found honeydew still being produced in November, and in grasses such as Poa annua which flower the year round, the fungus may occasionally over-winter in the sphacelial stage (Vizoso et al., 1984).

The ergots found on some grass species have evolved effective methods of dissemination. Those on Brachypodium sylvaticum, for example, remain attached on the host florets, the glumes of which carry hooked awns which become attached to the coats of passing animals. Those on Calamagrostis epigeios adhere to glumes equipped with parachutes of hairs and are thus dispersed by wind, and those on certain aquatic grasses (e.g. Glyceria fluitans) contain air which enables them to float and be dispersed by water (Stager, cited by Barger, 1931). Cereal ergots have had the most effective of all agents of dispersal - man himself. Harvested with the grain, they can be carried in seed corn all over the world.

However they are dispersed, the new crop of ergots ultimately fall to the ground where they lie dormant during the autumn and winter. During the dormant period, the sclerotia are vulnerable to attack by micro-organisms and mycophagous animals so, although they remain viable for up to 9 or 10 months (Neill, 1941a), many will be degraded before this time.

Survival of the sclerotia is influenced by soil type and by the depth to which they may be buried during subsequent husbandry operations. Bretag & Merriman (1981), working in Australia, compared survival of sclerotia over a period of 32 weeks in a clay loam (pH 5.9) and a self mulching grey clay (pH 8.2) in Victoria. More viable sclerotia were recovered from the latter soil than from the former. In the clay-loam soil more degradation occurred when the sclerotia were buried at 50 mm or 150 mm than when they were left on the soil surface. On the clay soil, increased degradation occurred only when the sclerotia were buried to a depth of 150 mm.

Eventually, with the arrival of spring, the sclerotia germinate and the cycle begins again with the production of a new crop of apothecia.

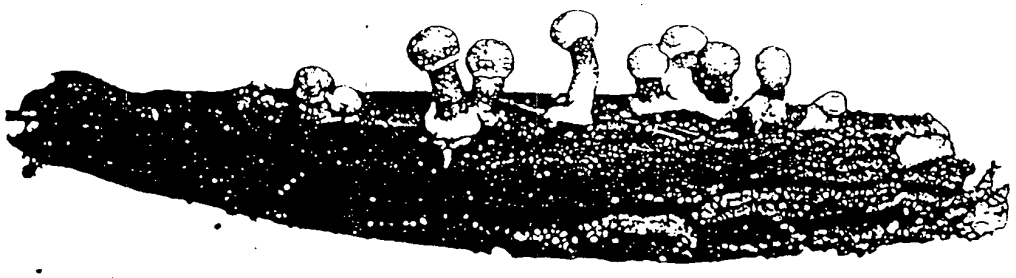

Fig.3. Germinating ergot from rye (x16) (reproduced from Barger, 1931)

Fig.4. Later stage of above (x8) (reproduced from Barger, 1931)

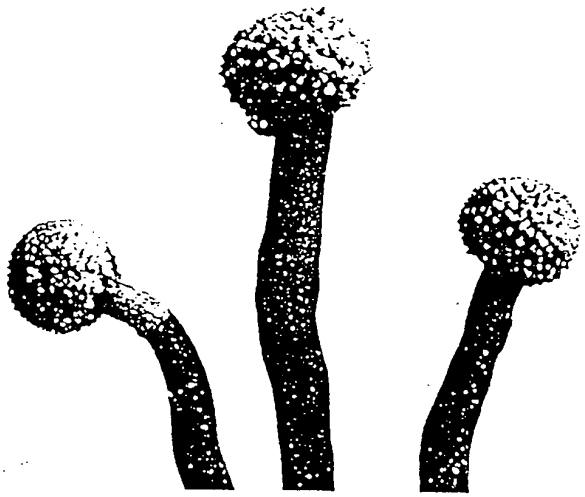

Fig.5. Fully developed capitula (x14) (reproduced from Barger, 1931)

7. EFFECT OF WEATHER ON DISEASE DEVELOPMENT

The effect of weather on ergot development was noted as long ago as 1575 when Joannes Thalius observed the disease (in the Harz Mountains) in a hot summer after a wet spring, conditions which Barger (1931) observed were ideal for its optimal production. Smith (1884) cites a report that ergot was particularly common in marshland wheats in East Anglia after the abnormally wet and sunless summer of 1879. Subsequent research has provided a clearer idea of the way in which the prevailing weather can affect the various stages in the development of the disease.

7.1 Effect of temperature on sclerotial germination

The findings of various earlier workers on the germination of ergot sclerotia were summarised by Mitchell & Cooke (1968a). After they are fully formed, the sclerotia appear to need a 'rest period' (said to be at least 8 weeks in the case of rye ergots) before further development can take place. Thereafter, a period of chilling is normally required to activate germination, though there seem to be occasional exceptions to this rule (Petch, 1937). Kirchhoff (1929) showed that rye ergots required 5-60 days at a temperature of 0-10°C to activate germination and Brentzel (1947, cited by Mitchell & Cooke, 1968a) suggested that alternate periods of freezing and thawing would stimulate germination of sclerotia from wheat, barley, rye and couch grass.

Mitchell & Cooke (1968a) investigated the chilling periods required to initiate germination in ergots from a range of hosts. Table 8 illustrates their findings with meaned data from sclerotia obtained from 3 sources: marram grass (Ammophila arenaria) from Yorkshire and Jersey and sea couch grass (Agropyron pungens) from Lincolnshire.

TABLE 8. Effect of chilling period on subsequent germination of sclerotia (ergots) of Claviceps purpurea

Duration of cold period (weeks)	Final % germination after chilling periods at the temperatures indicated		
	0°C	5°C	10°C
2	2	2	0
4	26	18	1
6	50	49	18
8	70	66	28
10	68	80	48

Germination was not induced by temperature lower than -5° or above 10° (Mitchell & Cooke, 1968a)

The main storage materials in ergot sclerotia are lipids. Sclerotia that have been chilled are able to transform and utilise these reserves more rapidly than are non-chilled sclerotia. Associated with this increase in lipid metabolism is an increase in both respiration rate and the ability to take up water from the environment (Mitchell & Cooke, 1968b).

Although the germination process is activated by chilling, actual germination with the production of stromata requires a return to higher temperatures. Under laboratory conditions, Cooke & Mitchell (1967) induced sclerotia from reed grass (Phalaris arundinacea) to germinate by allowing them to imbibe water for 2-4 hours and then keeping them at 5°C for 8 weeks. Stromatal development began 15 days after the sclerotia were transferred to room temperatures (18-21°C), reaching a maximum 16 days after that.

Authorities are divided as to the optimal temperature range for stromatal production. Thus, while Krebs (1936) suggested that 9-15°C provided optimal temperatures for the germination of sclerotia from rye, Kirchhoff (1929, cited by Mitchell & Cooke, 1968a or b) stated that germination of sclerotia from the same host ceased below 10°C and was most prolific at 18-20°C. Differences in the populations studied by these workers (the one in Germany, the other in Russia) may explain the differences in their results. Mitchell & Cooke

(1968a), working with sclerotia from Phalaris, found that: 95% would germinate between 10 and 20°C; at 25°C only 50% germinated; and above this temperature germination percentage fell off still further with the sclerotia becoming colonised by moulds.

Given that germination is influenced by temperature, it is not surprising that the sclerotia from the same host species will germinate at different times in different years and in different locations. For example:-

- cereal ergots in a field in Lincolnshire germinated over the period 6 June to mid July in 1972, and over the period 14 June to 5 August in 1974 (Wood & Coley-Smith, 1982);

- in 1973, germination of wheat ergots in the south of England began in mid-May - earlier in a sheltered situation in London than in rural locations (Mantle & Shaw, 1976)

- in the Versailles region of France, wheat ergots began to germinate in mid-April (Rapilly, 1969, cited by Mantle & Shaw, 1976);

- ryegrass ergots germinated from early June to mid July in Devon in 1955-7 (Jenkinson, 1958), and from mid June to mid August in Hull in 1973 (Wood & Coley-Smith, 1982),

In the above examples, germination tended to be earlier in warmer situations. This suggests that it is the time of onset of warm weather, rather than the time at which the chilling period occurs, which is of over-riding importance in determining the onset of germination.

7.2 Effect of temperature and humidity on ascospore release and production of conidia

Ascospore liberation begins about 7 days after germination (Cooke & Mitchell, 1967). Vladimirsky (1939, cited by Butler and Jones, 1949) observed that ascospore production was favoured by relative humidities (r.h.) of 76 to 78%. However, Hadley (1968) noted that while ascospore discharge was very heavy at 77% to 86% r.h., and remained quite heavy at r.h. values up to 94%, very few spores were discharged at 100% r.h.. He postulated that this would tend to

Marshall compared her own observations with those of Vladimirsky (1939) who noted that maximum development of ergot on rye in the Soviet Union occurred in regions where there was a high relative humidity (not less than 74%), a temperature of not more than 13-15°C at the beginning of flowering, and a prolonged flowering period of 14 days.

The last severe ergot epidemic in Eastern England occurred in 1981. A moderate level of infection in 1980 had provided ample inoculum for disease development in 1981 when cool wet weather in late May favoured infection of blackgrass and a return to similar conditions when the wheat was in flower favoured spread from the grass to the grain crop (Long, 1981).

High rainfall during the flowering period of wheat crops was also implicated in the severe epidemic experienced in the north of Scotland in 1978. An edited version of a previously unpublished paper by Watson and Osborne describing this epidemic is appended to this review (Appendix III).

discourage ascospore discharge in very humid conditions of the early morning before the stigmata of the host plants were exposed and receptive.

Optimal conditions for the production of the spacelial conidia are provided by atmospheric humidities in excess of 74% and temperatures not exceeding 13 to 15°C (Vladimirsky, 1939, cited by Butler & Jones, 1949).

7.3 Effect of weather on infection

Cool, wet weather at the time of anthesis not only favours the production and dispersal of the fungal spores, it also reduces the release and dispersal of pollen and extends the period of host susceptibility (see also Section 5). Hot, dry weather, on the other hand desiccates the stromata on the soil surface, dries up the honeydew and reduces the period of host susceptibility by favouring efficient pollination (Shaw, 1986).

Marshall (1960) reviewed the degree of ergot contamination found in wheat, barley and rye seed samples tested at the Official Seed Testing Station, Cambridge in the 40-year period from 1918 to 1957. Contamination was always highest in rye and lowest in barley, but in all three species definite fluctuations in severity were recorded:-

- in rye, ergots were most frequently recorded in 1918-29, 1927-8, 1936-9, 1945-8 and 1954-6. Four of these five periods were marked by high average relative humidities (74% or more) and low maximum temperatures (19°C) in June.

- a run of 'high ergot' years tended to end with a summer when in June humidity was less than 70% and temperature more than 21°C (e.g. 1940, 1949 and 1957).

However, high levels of infection occasionally occurred in years of unfavourable weather (e.g. 1936-39) and incidence was sometimes low when weather appeared to be favourable (e.g. 1929 and 1953). Marshall suggested that these anomalies might have been due to the presence or absence of large amounts of inoculum left from the previous year, and/or to the fact that weather conditions in the rye-growing areas differed from those at Cambridge where her own records were taken.

8. SUSCEPTIBILITY OF THE HOST TO ERGOT INFECTION

8.1 The "infection window"

Inoculation experiments carried out by Wood & Coley-Smith (1982) on male sterile barley showed that the flowers were susceptible to infection from the time the ears emerged from the boot. Susceptibility decreases after pollination as the growth of the pollen tubes towards the ovary is followed by the collapse of the tissues of stigmata and styles (Shaw, 1986), and the ovaries become resistant to infection about 5 days after fertilisation (Puranik & Mathre, 1971).

Wood & Coley-Smith (1982) showed that individual ovaries of male sterile barley remained susceptible for some 3 weeks, the period of maximum susceptibility lasting only about 10 days. The crop as a whole remained susceptible for a much longer period than this as flowers on late formed tillers were vulnerable to infection by conidia produced on the primary tillers.

Working on the highly susceptible wheat cultivar 'Waldron', Watkins & Littlefield (1976) showed that the flowers were more susceptible to infection in the 5 days before anthesis than in 5 days after (their inoculations being most successful on the day before anthesis). Inoculation 7 days after anthesis failed to produce infection.

Under field conditions, the period during which infection is likely to occur is (at least so far as self-fertile species are concerned) much shorter than these inoculation experiments would suggest. To infect a floret, the fungus must gain access to the ovary. In self-fertile species and cultivars, pollination normally takes place within the closed flower before anthesis. There is thus no need for the glumes to stay open to receive pollen from other flowers and the glumes gape for only a very short time. It is only during this brief period that infection can occur.

8.2 Susceptibility of cereal species

In open flowering species such as rye, triticale and durum wheat (Triticum durum), the glumes gape for a much longer period and the risk of infection is consequently much higher. It is for this reason that such species are much

more susceptible to ergot than are the commonly grown cultivars of bread and field wheats (T. vulgare) and barley (Shaw, 1986).

The relative susceptibilities of commonly grown cereal species are illustrated in Table 9 from data collected at the Official Seed Testing Station at Cambridge (Hewett, cited by Wood & Coley-Smith, 1980a).

TABLE 9. Ergot in cereal seed in the UK 1967 - 1972

	% samples contaminated				
	1967-8	1968-9	1969-70	1970-71	1971-72
Rye	9.1	4.5	5.3	0.9	3.1
Wheat	1.9	2.2	2.1	0.8	1.9
Barley	1.2	1.0	0.9	0.8	0.7
Oats	0.8	1.1	0.7	0.4	1.5

Not surprisingly, the male sterile lines used in plant breeding and in the production of hybrid cereals are particularly susceptible to infection since they are entirely dependent on cross pollination and their glumes therefore gape longer than those of self-fertile plants.

Spring wheat is frequently observed to suffer more severely from ergot than winter wheat. Marshall (1960) attributed this to its later flowering period rather than to any inherent susceptibility in spring wheat cultivars. Spring wheat flowers when the pathogen has had a chance to build up on nearby winter sown crops which can thus act as a strong source of inoculum.

8.3 Effect of pollen density

Watkins & Littlefield (1976) postulated that the high susceptibility of wheat cv. Waldron was due to the high degree of cross pollination which took place in this cultivar and which could result in the glumes staying open longer than in other cultivars. Particularly severe infections recorded in Waldron in 1971 were associated with low temperatures (down to -7°C) in the 2-3 weeks before heading. Temperatures of -3 to 2.5°C in the period 1-5 weeks before

heading were known to cause non-functional anthers in wheat so it was likely that pollen production was particularly poor in cv. Waldron in 1971. Flowers would remain unfertilised and glumes would gape for longer than usual, allowing ample time for infection to take place.

A similar situation occurred in England in 1987 when the bread wheat cv. Moulin was particularly badly ergotised. Moulin always tended to be a poor producer of pollen and conditions in 1987 were such as to exacerbate this trait. Self fertilisation frequently failed to take place and the glumes of the unfertilised flowers remained open for a much longer period than usual. The pathogen was thus presented with an ideal opportunity for infection.

That ergot is frequently more common along the field edge is due, at least in part, to the fact that the density of pollen is lower towards the edge of the crop and the competition between ergot spores and pollen grains on the stigmata of the plants is thus reduced (Hewett, 1982). This effect is often augmented by infected grasses in the hedge bank increasing the level of inoculum in this part of the field.

8.4 Effect of unevenness of crop

That the ears on late tillers are particularly susceptible to ergot infection is frequently observed. Such late ears become susceptible when the pathogen has had a chance to build up in its sphacelia stage on the first formed tillers, and when the main pollen shower is over. Incidence of ergot is thus likely to be particularly high in uneven crops, in wheelings or where careless herbicide application has delayed flowering in parts of the field (Hewett, 1982).

8.5 Effect of crop nutrition

8.5.1 Nitrogen

Naylor & Munro (1992) found that the numbers and the percentage by weight of ergot in grain from triticale plots in a 'rate of nitrogen x fungicide' trial increased with the amount of nitrogen applied. The weight of the individual sclerotia was unaffected by nitrogen rate.

8.5.2 Copper

Evans et al. (1992) noted that soil-applied copper significantly reduced the incidence of ergot in wheat grown on copper deficient soil. Copper deficiency causes malformation of the ears with the upper spikelets frequently failing to produce grain. Such malformation is likely both to reduce pollen production and to result in the glumes gaping longer.

8.6 Effect of fungicide programme

While certain fungicides applied at the time of anthesis can reduce the incidence of ergot (see section 13.2), Naylor & Munro (1992) found that a fungicide programme applied for the control of foliar disease and based on prochloraz and fenpropimorph applied at early flag leaf (GS37) and ear emergence (GS59) increased the incidence of the disease in triticale. While the effect of the fungicides would have been due to their having reduced competition from other micro-organisms, Naylor & Munro considered it more likely that it was due to their having had a growth regulatory effect on the plants.

9. THE ROLE OF GRASSES IN ERGOT AETIOLOGY

9.1 Host range on grasses in the UK

Wood & Coley-Smith (1980a) list 25 species of wild and cultivated grasses on which C. purpurea was found in the UK between 1969 and 1974. A few additional species were mentioned by Loveless (1971) and Mantle et al. (1977) as sources of sclerotia used in their experiments. Between them, these papers mention 33 grasses on which the fungus has been found in the UK in recent years:-

Agrostis canina	- brown bent
Agropyron pungens	- sea couch grass
Alopecurus geniculatus	- marsh foxtail
Alopecurus myosuroides	- blackgrass
Alopecurus pratensis	- meadow foxtail
Anthoxanthum oderatum	- sweet vernal grass
Ammophilia arenaria	- marram grass
Arrhenatherum elatius	- oat grass
Brachypodium sylvaticum	- slender false brome
Bromus racemosus	- smooth brome
Dactylis glomerata	- cocksfoot
Deschampsia caespitosa	- tufted-hair grass
Deschampsia flexuosa	- wavy hair-grass
Elymus arenarius	- lyme grass
Elymus repens	- couch grass
Festulolium loliaceum	- meadow fescue x perennial ryegrass
Festuca arundinacea	- tall fescue
Glyceria fluitans	- flote-grass
Holcus lanatus	- Yorkshire fog
Holcus mollis	- creeping soft-grass
Hordeum murinum	- wall barley
Lolium multiflorum	- Italian ryegrass
Lolium perenne	- perennial ryegrass
Melica uniflora	- wood melick
Monilia caerulea	- purple moor grass
Nardus stricta	- mat-grass
Phalaris arundinacea	- reed-grass
Phleum pratense	- timothy
Phragmites communis	- reed

Poa annua	- annual meadowgrass
Puccinellia distans	- reflexed poa
Spartina anglica (sic)	- (? S maritima)
Spartina x townsendii	- cord grass

The most commonly infected grass species listed by Mantle et al. (1977) and by Wood & Coley-Smith (1980a) are given in Table 10.

TABLE 10. Grass species commonly infected with ergot

Grass species	Number of collections made from different sites 1969-74 (Wood & Coley-Smith, 1980a)	Frequency of infection (Mantle et al., 1977)
Alopecurus myosuroides		Very common
Alopecurus pratensis		Common, but only after a wet spring
Anthoxanthum oderatum	6	
Arrhenatherum elatius	12	
Dactylis glomerata	17	Common
Elymus repens	11	Very common
Festuca arundinacea	5	
Holcus lanatus	11	Very common
Lolium perenne	15	Very common in late summer
Glyceria spp.		Common
Monilia caerulea	5	
Phleum pratense	5	

That Alopercurus myosuroides is absent from Wood & Coley-Smith's list is probably a reflection of their collections having been made on British Mycological Society forays which would tend to ignore the arable fields where this grass is a common weed. Mantle et al. (1977) noted that although ergots were found only occasionally on Poa annua, the disease was difficult to observe on this host and they thought that infection of this very common arable weed was probably far more common than was often supposed.

9.2 Strain specialisation within the pathogen population

That C. purpurea might be divided into a number of 'biologic forms' each restricted in the grass species which it would infect was first suggested by the Swiss mycologist Stager (1903, 1905, 1908, 1922, 1923 cited by Barger 1931). Stager claimed to have identified 5 special forms of the fungus:-

i) a race that would attack rye, wheat, barley, Festuca pratensis, Bromus sterilis and a few species of Poa.

ii) a race that would attack Lolium perenne and Bromus sterilis but not rye (Stager (1922) later queried the validity of this race).

iii) a race that would attack rye, Anthoxanthum oderatum and certain other grasses but not barley.

iv) a race confined to Glyceria fluitans.

v) a race confined to Brachypodium sylvaticum.

vi) a race confined to Phalaris arundinacea.

Noting that most ergot species are very restricted in their host range, Loveless (1964) expressed doubts as to whether Claviceps purpurea, with its very wide host range, should really be considered to be a single species. He suggested that, while the ergot fungi collected from all the grasses within this range might appear morphologically similar, close investigation of the sphacelial spores might enable C. purpurea to be broken down into a number of species each of which might be found to have a much more restricted host range. In a later study of 101 samples of honeydew from 31 species of grasses and cereals, Loveless (1971) was able to group isolates from the various host species according to their conidial characteristics. He found that these groups bore a close similarity to the 'biologic forms' which Stager had characterised by their host range.

While Loveless's work appeared to confirm Stager's findings, those findings had earlier been challenged by Campbell (1957) who managed successfully to inoculate rye, wheat and barley with 420 isolates of the pathogen from 38

species of grasses; and to inoculated 47 species of grasses with honeydew from rye.

The apparent contradiction between the observations of Stager and Loveless on the one hand and of Campbell on the other may be due, at least in part, to the fact that some of the 'biologic forms' of the pathogen are characterised not by their inability to infect species outside their normal host range but by their ability to infect such species only with difficulty.

This was indicated by the work of Mantle et al. (1977) who inoculated male sterile wheat with 40 isolates from 19 species of grass and cereals. Thirty seven of the 40 isolates infected the wheat, but only 15 could be considered 'highly infective' (>63% inoculated florets produced sclerotia); the remainder were only 'weakly infective' (<32% successful inoculations):-

> 'Highly infective' isolates included those from other wheats, triticale, rye, barley and Alopercurus myosuroides.
>
> 'Weakly infective' isolates included those from Lolium spp., Dactylis glomerata, Phleum pratense, Holcus spp. and Anthoxanthum oderatum.
>
> Both 'highly infective' and 'weakly infective' isolates were obtained from Alopecurus pratensis and Poa annua.

A strain of the pathogen from wheat was highly infective on Dactylis glomerata, Alopecurus spp. and Festuca pratensis but would not infect Lolium spp. or Anthoxanthum oderatum.

There was no evidence that the strains described by Mantle et al. (1977) could be distinguished by any morphological differences but they did show chemical differences with respect to the proportions of the major alkaloids which they contained (in this study, alkaloid content was used simply as a taxonomic characteristic and the authors did not discuss the toxiological significance of their findings). In particular, from whatever hosts they were obtained, strains which were highly infective to wheat and had a high ergotoxine content.

Using thin layer chromatography, Mantle et al. (1977) analysed 241 samples of grass and cereal ergots from all over the British Isles. They found that:-

i) the alkaloid spectra in sclerotia from wheat and blackgrass were always very similar with ergotoxine level being never less than, and almost always greater than, the level of ergotamine;

ii) sclerotia from meadow foxtail and annual meadow grass usually gave alkaloid spectra in which ergotoxine was low in comparison with ergotamine, but there were examples from both grasses in which the alkaloid spectra closely resembled those obtained from wheat ergots (isolates from these grasses had been sometimes highly infective and sometimes only weakly infective to wheat);

iii) amongst ryegrass ergots, only one was found in which the ratio of ergotoxine to ergotamine was greater than unity; this sample had been obtained from a single infected ryegrass plant found in an ergotised wheat crop infested with ergotised blackgrass;

iv) most sclerotia from timothy, oat-grass and the Holcus species resembled those commonly found on ryegrass rather than those found on wheat;

v) although sclerotia from cocksfoot often resembled those from wheat in their ergotoxine/ergotamine ratios, they could usually be distinguished by their high content of ergosine or of another unidentified alkaloid.

Data collected by Mantle & Shaw (1977) clearly illustrate the differential spectra of alkaloids in cereals and weed grasses growing in close proximity on a single farm (Table 11).

TABLE 11. Alkaloid spectra in grass and cereal ergots from a farm at Warborough, Oxfordshire 1972

(+++ major component, ++ minor component, and trace component of alkaloid spectrum)

	Ergotamine	Ergosine	Ergotoxine
Holcus lanatus	+++	0	+
Lolium perenne	+++	+	++
Arrhenatherum elatius	+++	++	++
Dactylis glomerata	+	+++	+++
Barley	++	++	+++
Wheat	++	+++	+++
Alopecurus myosuroides	++	+++	+++
A. pratensis	++	+++	+++

NB. 'Ergotoxine' is a mixture of ergocornine, ergocristine and ergocryptine.

Small amounts of lysergic acidamide and ergometrine were commonly found in sclerotial extracts Holcus lamatus also contained an unidentified alkaloid (+++).

The similarity between the alkaloid spectra of wheat, blackgrass and, in this case, meadow foxtail ergots will be noted. Inoculation experiments showed that, on this farm, the meadow foxtail was carrying a strain infective to wheat. The flowering sequence of meadow foxtail (which was found to be producing honeydew by 30 May), blackgrass, and wheat provides opportunity for the pathogen to build up on the two weed grasses before spreading onto the wheat. The ergots from Yorkshire fog, ryegrass, oat-grass and, to a lesser extent, cocksfoot all differed from the wheat ergot in their alkaloid spectra suggesting marked stain differences, but even had the strains from these grasses been able to infect wheat, their hosts flower too late for honeydew from them to be of any great importance in infecting the wheat plants.

The work of Mantle et al (1977) and Mantle & Shaw (1977) supports the views of earlier workers that C. purpurea exists as several distinct strains. However, the strains which they categorised by infectivity and biochemical properties do not correspond well with the groups based on conidial morphology delineated by Loveless (1971). Nor do the strains which they describe appear to be so closely confined to particular hosts as are the 'forma speciales' of many other pathogens. A highly complex and rather fluid situation seems to exist.

The one firm conclusion which can be drawn is that the strain commonly found on blackgrass is highly infective to wheat.

The link between wheat ergot and blackgrass has long been recognised (Batts, 1956) and is well supported by field observations such as those of Mantle et al. (1977) whose survey of 25 wheat crops in 1973 showed a close association between the incidence of the weed in the fields and the incidence of ergots in the ears. However, with the possible exception of meadow foxtail (and perhaps annual meadow grass), other grass weeds appear to be less important in the epidemiology of the disease in England than was once supposed.

In Scotland, where blackgrass is uncommon, the spectrum of strains may be different to that found in England and other grasses may have a more important role to play. In the 1978 epidemic in northern Scotland, infection of couch grass and rye grass was very common (Watson, 1979b). The spectrum of alkaloids in the Scottish wheat ergots differed from that of English wheat and blackgrass ergots but was very similar to that found in ryegrass ergots from Scotland (see Appendix III).

The existence of strains which can infect both ryegrass and wheat has also been demonstrated in Australia by Bretag & Merriman (1981) who showed ryegrass to be the primary source of wheat ergot in Victoria Province.

Analysis of ergots from Scottish barley in 1986 showed them to be very rich in ergosine but with no ergotamine - in marked contrast to the Scottish wheat and ryegrass ergots which were rich in ergotamine but contained relatively low levels of ergosine (B.G. Osborne in a letter to S Wale, pers comm.).

10. THE TOXICOLOGY AND PHARMACOLOGY OF ERGOT

10.1 The ergot alkaloids

Seven isometric pairs of indole alkaloids have been isolated from the sclerotia of Claviceps purpurea (Table 12). One of each pair is derived from lysergic acid and is physiologically active. The second is derived from isolysergic acid and has little physiological activity (Osborne & Watson, 1980). An eighth alkaloid, ergomonamine, differs from the others in that it is not an indole derivative. Yet other alkaloids have been isolated from ergot sclerotia from various of the grass hosts of the fungus (Sastry, 1986).

TABLE 12. The more important alkaloids found in ergots

Lysergic acid derivatives	Isolysergic acid derivatives
ergocornine	ergocornine
ergocristine	ergocristinine
ergocryptine	ergocryptinine
ergometrine	ergometrinine
ergosine	ergosinine
ergostine	ergostinine
ergotamine	ergotaminine

The way in which, in a given location, the ratios of certain of these alkaloids vary according to the host species has already been discussed. The situation is further complicated, however, by the fact that these ratios can also vary between populations of C. purpurea in different parts of the world. Total alkaloid content also varies considerably between different populations. Osborne & Watson (1980), for example, reported that the total alkaloid content of ergots collected in Scotland in 1978 fell between the contents of the two main types of commercial ergot, i.e. Spanish and Portuguese (0.2-0.25%) and Eastern European (0.4-0.5%). The Scottish ergots had an ergometrine content similar to those from Eastern Europe and were rich in ergotamine which the Spanish ergots do not contain.

Physiologically, the ergot alkaloids are highly active. Ergocornine, ergocristine and ergocryptine (the 'ergotoxine group') have an inhibitory

effect on the central nervous system; ergotamine, ergosine and ergometrine act on the smooth muscles of the uterus, blood vessels, stomach and intestines (Ramsbottom, 1954).

The highly toxic nature of these ergot alkaloids renders the sclerotia highly poisonous to both man and his animals.

10.2 Ergot poisoning in animals

Kobert (cited by von Tubeuf, 1897), working primarily with cattle and fowls, described the following symptoms associated with ergot poisoning:-

i) gastro-enteritic symptoms, excessive salivation accompanied by redness, blistering, inflammation, wasting and gangrenisation of the mouth epithelium, with similar changes to the epithelium of the gut producing vomiting, colic and diarrhoea,

ii) gangrenisation and mummification of extremities, consisting of a drying up, a drying-off and a detachment of extremities such as nails, ears, tail, wings, claws, toes and point of tongue,

iii) spasmodic contraction of the uterus and consequent abortion,

iv) nervous phenomena such as insensibility, blindness, paralysis etc,

Most outbreaks of ergotisim in the UK occur amongst cattle. The first symptom to be noticed is usually lameness. Affected limbs are generally swollen and raw flesh is often exposed at the back of the foot (Dickens, 1974).

10.2.1 Cattle

The effects on cattle of ingesting ergots from a heavily infected crop of ryegrass was described by Woods et al. (1966). The vaso-constricting effects of the toxins caused sloughing of hooves and the development of granulomatous lesions which in some instances became infected with the bacteria Corynebacterium pyogenes (which can cause supperative lesions in cattle) and Fusiformis necrophorous (known as a cause of gangrenous dematitis in horses). 0.1% ergot in high protein rations made cattle more vulnerable to heat stress (Seaman, 1971).

10.2.2 Sheep

Sheep are known to be sensitive to ergot poisoning, especially in late pregnancy. In the late 1980s, 7 lambs died on a Scottish farm after being fed barley screenings heavily contaminated with grass ergots (S. Wale, North of Scotland College of Agriculture, pers. comm). The concentration of ergot in the bottom of the feed trough was measured as 2% by weight. Outbreaks of ergotism amongst sheep, are however, rare. Low levels of concentrate intake reduce the risks of their eating enough contaminated cereals to harm them, and their habit of selective grazing leads them to avoid the mature grass heads which contain the sclerotia. The present author has known a flock of pregnant ewes to graze a heavily ergotised crop of ryegrass without ill effect.

10.2.3 Pigs

Ergot sclerotia are less toxic to pigs than they are to ruminants as the pigs eliminate the alkaloids more efficiently (Whittmore et al 1977). Thus, while pigs fed on a contaminated grain show reduced growth rates, and occasionally internal lesions, they are less prone to develop the symptoms of 'classic ergotism' as described by Kobert. In the experiments of Whittmore et al (1977), pigs would tolerate a diet including up to 0.7% ergot, but their intake declined when the ergot content was increased to 1.4%. The sclerotia appeared to be less unpalatable when they were fresh than when they had been stored for a year, perhaps because changes during the storage period had increased the characteristic aroma of the sclerotia.

10.2.4 Horses

Ergot poisoning in horses leads to general depression of the vital functions; pulse and breathing are weak, and the sensory organs less sensitive than usual. The disease generally progresses rapidly leading to muscular paralysis and eventually death (Rojdestvensky, 1927). Buffon (cited by Rojdestvensky, 1927) found that horses which had grazed a field of infected Agropyron began to lose their hooves, and in some cases their manes and tails, on the following day. Wilcox (cited by Rojdestvensky, 1927) mentioned a case in which horses died within 6 to 12 hours after eating hay with a high content of

ergot. When lesser amounts of ergot have been ingested over a long periods there may be diarrhoea, colic, vomiting and signs of abdominal pain (West, 1985).

10.2.5 Other domestic mammals

In addition to the species already mentioned, ergot poisoning has been recorded in goats, mules, dogs and cats and has been induced experimentally in guinea pigs (Rojdestvensky, 1927; Fuller, 1968; Sastry, 1986).

10.2.6 Poultry

Rojdestvensky (1927) fed 2 young cockerels with a daily ration of 8 g ergot mixed with steamed oats or in a millet mash; the birds usually ate about half of what was given them. On the fifteenth day their combs and wattles developed cyanosis; after 30 to 32 days they could no longer stand and they had lost much weight despite the fact that their appetites were normal, on the 35th and 36th days they died.

Rotter et al - (1985) found that as the concentration of wheat ergot (0.31% total alkaloids) increased from 1 to 8% in the diet of Leghorn and broiler chickens, performance progressively decreased. Broiler chickens were slightly more sensitive than Leghorns to the effects of ergot. Generally, after 3 and 4 weeks, chickens given 1 or 8% dietary ergot had about a 10% or 80% lower relative weight gain than control birds. Mortality was low on diets containing up to 3% ergot but above this concentration increased dramatically and progressively.

Poultry will probably avoid eating ergots contaminating whole-grain feeds (Watson, 1978).

10.2.7 Bees

Ergot honeydew, being rich in sugars, proteins and minerals, is attractive to bees but the honey they make from it less fragrant than that made from nectar. It is also injurious and bees are said to have difficulty in surviving the winter if they have stored honey of this kind (Kabloukoff, cited by Rojdestvensky, 1927).

10.2.8 The need for caution in interpreting data from animal experiments

The variation in alkaloid content between ergot populations demands a degree of caution when interpreting data on the dose rates necessary to cause symptoms. This was clearly demonstrated by Whitmore et al. (1977) who used both East Anglian and American ergots in their experiments. In the former, ergotoxine comprised 80%, and in the latter 30%, of the total alkaloids. Pigs fed on diets containing the English ergots showed lesions in their stomachs, small intestines and livers. Those fed on the American ergots showed none of these symptoms.

The age of the ergots can also influence their toxicity. Moller (cited by Rojdestvensky, 1927) stated that 4g of fresh ergot are sufficient to cause visible symptoms of poisoning in hens; a little later 10g were necessary to cause the same symptoms, and 5 month old ergot caused hardly noticeable symptoms of poisoning.

10.2.9 'Safe' levels of ergots in feed

It has been generally recommended that animals should not be given feeds containing more than 0.1% ergot by weight (Young & Marquardt, 1982). This accords with the provisions of the Fertiliser and Feedstuffs Regulations Amendment No. 840 (Anon.,1976) which required that ergot should represent no more than 0.1% by weight of grain sold for animal feed. The work of various authors cited by Young & Marquardt (1982) suggests that, at least so far as poultry are concerned, this should allow a sufficient margin for safety:-

i) growth of leghorn chicks was not affected when their rations contained 0.3% wheat ergot (O'Neill & Rae, 1965)

ii) growth of broiler chicks was not affected when their rations contained 0.8% triticale ergot (Bragg et al., 1970)

iii) laying hens tolerated 0.4 - 0.6% dietary ergot without egg production being affected (O'Neill & Rae, 1965)

At higher levels of ergot intake, chicks showed poor growth, increased feed gain ratios and mortality.

While a 0.1% level of ergot in a feed sample should not cause problems, care needs to be taken if grain with this level of contamination is used for feed. As a bulk of grain is handled, sclerotia fragments tend to move towards the bottom of the bulk. The grain which is fed last may therefore have a higher concentration of ergot in it than the concentration in the bulk as a whole.

10.3 Ergot poisoning in humans

The effect of ergot toxins on humans has been described by many authors over the past four hundred years. The description of symptoms given by Ramsbottom in (1954) is worth quoting in full:-

> "Ergotism shows two distinct types of poisoning, gangrenous and convulsive.
>
> In gangrenous ergotism the preliminary symptoms are followed, after a few weeks, by the limb affected becoming swollen and inflamed, violent burning pains alternating with very cold. Gradually the affected part becomes numbed, then shrunken and mummified. The extent of the gangrene varies from the mere shedding of nails and the loss of fingers and toes, to that of whole limbs. Sometimes the course of the disease is very rapid, the prémonitory signs of gangrene appearing after 24 hours.
>
> Convulsive ergotism shows pronounced nervous symptoms, with twitchings and tonic spasms of the limbs, and strong permanent contractions, particularly of the hands and feet. In severe cases the whole body is subject to sudden, violent, general convulsions. One of the early symptoms, which often continues, is the numbing of the hands and feet, and a tingling sensation "as if ants were running about under the skin" (formication)".

The gangrenous type of ergotism is said formerly to have been common in parts of France and the convulsive form in Eastern Europe but some overlapping of symptoms is likely to have occurred.

There is evidence that the convulsive form occurred in subjects deficient in vitamin A (this form of the disease was most common in areas where dairy products were in short supply), but variation in the ratio of alkaloids in the ergots may also have played a part.

From the description of past plagues, it is possible to trace the history of ergotism back long before its connection with C. purpurea was properly understood. The first record of the disease dates from AD 857 when the area around Kanten in Lower Rhineland was smitten by 'a plague of swollen blisters' which 'consumed the people by a loathsome rot, so that their limbs were loosened and fell off before death' (Ramsbottom, 1954).

From AD 945 onwards, a number of outbreaks were recorded in France where the burning sensations experienced by the victims earned the disease the name of *'ignis sacer'* or *'ignis Beatae Virginis'*. Later it became known as St Anthony's fire - perhaps because of the hallucinations suffered by victims were reminiscent of the visions of devils which appear to have afflicted the saint. For whatever reason, it was to St Anthony that sufferers turned for help when all human ministrations failed and the Order of St Anthony at one time had 390 houses, in various countries, in which they cared for those suffering from the disease. As late as the 18th century, the hospital of the Order of St Anthony in Vienna maintained a collection of withered and blackened limbs, relics of sufferers from the disease who had sought succour there (Haggard, 1929).

The hallucinations produced by ergot poisoning were not, however, invariably diabolic in nature. They could be ecstatic, visionary and sometimes quasi-mystical. Matossian (1982) has educed evidence to show that the more dramatic manifestations of religious fervour which marked the revivalist 'camp-meetings' of 18th and 19th century America most frequently occurred when those meetings were held in rye growing areas and at times (immediately after harvest) when ergot contaminated flour would be at its most toxic. One sceptical Methodist preacher described people at the meetings falling into trances 'and when they came to they professed to have seen heaven and hell, to have seen God, angels, the devil and the damned; they would prophesy, and, under the pretence of Divine inspiration, predict the time of the end of the world, and the ushering in of the great millennium'.

Serious outbreaks of convulsive ergotism occurred in Germany during the 17th and 18th centuries and continued into the 1880s though their incidence declined with improving agricultural practices and with the increase in potato growing which followed the famine of 1770/71.

Russia has suffered numerous epidemics, usually of the convulsive form of the disease, the most recent being in 1926 when over 11,000 people were affected (some fatally) by the disease. In this latter epidemic, the proportion of ergot in the rye ranged from 1 to 26.7% by weight in the various districts affected. The disease occurred when 1% contamination was present; 7% contamination led to death. As a result of the epidemic, the USSR fixed the permissible level or ergot in flower at 0.15%, the corresponding allowable level in Germany being 0.1% (Dixon, 1932).

In the UK, presumably because we have never been so dependent on rye as our continental neighbours, ergotism has been rare. Apart from some rather obscure references to 12th century epidemics (Barger, 1931), the one recorded case of gangrenous ergotism occured at Wattisham, Suffolk where a plaque in the now de-consecrated church states that:-

> "This Inscription Serves to Authenticate the Truth of a Singular Calamity which Suddenly Happened to a Poor Family in this Parish, of which Six Persons lost their Feet by a Mortification not to be Accounted for. A full Narration of their Case is Recorded in the Parish Register & Philos: Transactions for 1762"

The six unfortunates were the wife and five children of a poor labourer who himself escaped with numbness of the hands and lost finger nails. For a fuller account of this outbreak, as recorded by Smith (1884), see Appendix IV.

A more recent outbreak of mild convulsive ergotism occurred in Manchester in 1927 amongst Jewish immigrants from Central Europe who ate bread made from a mixture of wheat and rye flour. The grain from which the rye flour had been prepared yielded 0.9% ergot by hand picking but colorimetric analysis suggested an ergot content of 1.5%, equivalent to between 0.18 and 0.3% in the flour (Dixon, 1932). Given a daily consumption of 0.5 kg, flour containing 0.3% ergot would provide a dose of 1.5 g of ergot per day.

Modern methods of grain cleaning enable millers to achieve very high standards in removing ergots from contaminated grain and, if any sclerotia should survive the cleaning process, only 20% of the initial ergot content of the wheat remains in flour at 75% extraction rate (Osborne & Watson, 1980). During bread baking, a further 50% reduction in alkaloid content occurs, the pharmacologically active alkaloids being reduced to a greater extent than the

less active ones (Wolf et al., 1988). Not surprisingly, therefore, ergotism has been almost unknown since the Second World War. Almost - but not quite.

In 1951, in the French village of Pont-Saint-Espirit, all those who ate bread from a particular bakery (including the baker and his family) were stricken with symptoms which bore a so close a resemblance to those of classic ergotism that there seems little doubt that ergot poisoning was responsible. This conclusion appears to have been supported by medical, analytical and circumstantial evidence though, at the time, the official enquiry into the incident failed to endorse this verdict (Bouchet, 1980). One point at issue was the fact that some of the victims had eaten so little of the contaminated bread that it seemed impossible that they should have developed such severe symptoms from the very small amounts of ergot which they would have consumed. A possible explanation put forward by some was that the poisoning was due not so much to alkaloids derived from lysergic acid but from lysergic acid itself which would be toxic at much lower concentrations than its derivatives. It was postulated that under certain circumstances the fungus could produce lysergic acid at unusually high concentrations and that this was responsible for the Pont-Saint-Esprit epidemic (Fuller, 1968).

Amongst the remarkable symptoms associated with the Pont-Saint-Espirit outbreak were chronic insomnia, alternating euphoria and deep depression, hallucinations of (generally malign) animals and a compulsion to throw oneself from upper-storey windows. The latter condition is said to be common amongst those taking LSD as a psychadelic drug. To keep his thoughts from this irrational desire, one sufferer wrote endlessly. Poems he produced at this time give a disturbing insight into a mind brought to the end of its tether by the effects of ergotism (Appendix IV).

Even if the lysergic acid theory remains unproven, the 1951 outbreak certainly suggests that ergot can occasionally be even more toxic than had previously been supposed, and it underlines the need for continued vigilance in our efforts to avoid contamination of grain destined for human consumption. Such vigilance becomes increasingly important at a time when there is increasing interest in the consumption of unprocessed cereals (Schoch & Schlatter, 1985).

Fig.6. A sufferer from ergotism appeals for help to St Anthony. Woodcut from a book on military surgery by Gessdorff, 1535. (Reproduced from Barger, 1931).

10.4 The medicinal uses of ergot

For at least 400 years, and probably for much longer, ergot has been used in folk medicines for easing childbirth. The earliest published reference to ergot (Lonicer, 1582, cited by Barger, 1931) mentioned its use as a means of inducing labour, a repeated dose of three sclerotia (about 0.5 g) being recommended. It was widely used by 16th and 17th century midwives in France, Italy and Germany, and by the late 18th century powdered ergot (known as *'pulvis ad partum'*) was being administered by some contintental physicians. That such early usages were not without risk is suggested by the fact that the use of ergot by midwives was forbidden in Hannover from 1778 and in the Palatinate from about 1790 (Barger, 1931).

It was from an immigrant German woman that the American doctor John Stearns first learned of its value in the early 19th century. After studying its effects for some years, Stearns introduced ergot to the medical establishment in a letter to the periodical 'Medical Repository of New York' (Stearns, 1808: cited by Barger, 1931). The drug was admitted to the first edition of the United States Pharmacopoeia in 1820 and to the Pharmacopoeia of the London College of Physicians in 1836.

During the latter half of the 19th century, other medicinal uses of ergot were discovered, in particular it was used in the treatment of migraine and its vaso-constrictive properties were exploited in the control of bleeding in delicate organs such as the stomach, oesophagous, intestine and urinary tubes. In more recent times, the use of ergot derivatives has been investigated in connection with the treatment of such diverse conditions as breast cancer, Parkinson's disease, acromegaly and prostatic hypertrophy (Sastry, 1986). Ergot has also found a place in homeopathy for the treatment of gangrene, Raynauds disease and circulatory difficulties of various kinds (Coulter, 1981).

Direct preparations of ergot, such as *'ergota praeparata'* (powdered and defatted ergot), are seldom if ever used in modern medicine but alkaloids extracted from the fungus still find a place in the pharmacopoeia. The properties of the most important, ergometrine and ergotamine, may be summarised as follows (Parish, 1987):-

ergometrine (used as ergometrine maleate, sometimes referred to ergonovine maleate) causes contractions of the uterus and is used to prevent bleeding from the uterus after childbirth. If the drug is properly used, adverse effects are rare but it can occasionally cause nausea and vomiting and, if used intravenously, heightened blood pressure.

ergotamine (used as ergomatine tartrate) stimulates and, in large doses, paralyses the endings of the sympathetic nerves; it constricts small blood vessels and also the uterus. Its main use has been to control migraine but care is needed in its dose as it can cause unpleasant side-effects. In large repeated doses it can produce all the symptoms of ergot poisoning:- coldness of the skin, severe muscle pain, gangrene, thrombosis, angina, alteration of the pulse rate and blood pressure, confusion, paralysis and convulsions. (It has recently been claimed (Murray & Pizzaro, 1990) that, while the intramuscular injection of ergotamine tartrate is definitely effective against migraine, taken orally it has only a placebo effect).

For further information on the pharmacology of ergot alkaloids and their analogues, see the account given by Reynolds (1989).

11. THE CULTIVATION OF ERGOT

So valuable are the drugs derived from its sclerotia that ergot has long been considered a valuable commodity. It was actively traded from the 1820s onwards, Russia and Spain being the main sources of supply. By the 1920s, improvements in agriculture in these two countries had led to a fall in natural contamination, and the possibility of cultivating the fungus was already being investigated. This research was given added impetus during the Second World War when European sources of the drug were denied to Commonwealth countries. In New Zealand, Neill (1941a) successfully inoculated rye with conidia produced in culture medium from a Hungarian strain of ergot. Unfortunately, the alkaloid content of the original strain was low and, in the second generation grown in Auckland, no alkaloids at all were produced so the ergots were worthless. As an alternative to cultivated ergot, schoolchildren in New Zealand were encouraged to collect ergots from grasses (those from tall fescue being favoured) to supply Britain's needs (Neill, 1941b). Instructions on the harvesting and cleaning of such wild ergots were given by Bell (1941).

After the war, the Ministry of Health was concerned to maintain UK supplies of ergot for medicinal purposes. Departmental records show that, in 1952, NAAS Plant Pathologists at Cambridge were asked to comment on the plant pathological implications of a trial (carried out by the Wellcome Research Laboratories) which involved the artificial inoculation of 10 acres of rye at Little Cressingham in the Brecklands of Norfolk. In the event, commercial production of ergot never developed in the UK, but it provided an important source of the drug in a number of other European countries.

After the severe epidemic in England in 1964, contaminated wheat was actively sought by at least one seed merchant for sale to the drug companies. A letter from this time in the files of the ADAS Plant Pathology Department (now Plant Diagnostic Laboratory) at Cambridge describes the methods used for the cultivation of ergot in Switzerland:-

> "Rye is grown for this purpose in many parts of the country ... A variety with very stiff straw is used and the seed is supplied by the drug people who are contracting the crop with the growers. Inoculation is carried out by a fleet of machines owned by the company. This is done when the ... ears have just emerged. The rye is drilled in strips to enable the

machines to travel in the alley-ways without damaging the crop. The first and best ergots are brushed off by hand. Later the field is combine harvested and the remaining ergots separated from the rye." (Keyworth, in a letter to Brenchley, 1964).

Although some alkaloids are now produced synthetically by fermentation processes, cultivated ergot remains an important source of these drugs. Sastry (1986) estimated the worldwide annual production to be between 282 and 395 tonnes (Table 13).

TABLE 13. Production of ergot by country (Sastry, 1986)

Country	Area (ha)	Yield (tonnes)
Hungary	2000 - 2500	100.0 - 125.0
Czechoslovakia	1000 - 1500	50.0 - 75.0
Switzerland	800 - 1200	40.0 - 60.0
USSR	500 - 1000	25.0 - 50.0
Spain	300 - 350	15.0 - 17.5
Netherlands	250 - 300	12.5 - 15.0
Poland	200 - 250	10.0 - 12.0
India	150 - 200	7.5 - 10.0
East Germany	150 - 200	5.0 - 7.5
Austria	100 - 150	5.0 - 7.5
Yugoslavia	100 - 150	5.0 - 7.5
France	80 - 100	4.0 - 5.0
Bulgaria	50 - 60	2.5 - 3.0
TOTAL	5680 - 7060	282.5 - 395.0

With the recent introduction of new and safer drugs for the control of migraine, the use of ergotamine is likely to decline. This could significantly affect the demand for cultivated ergot.

12. DECONTAMINATION OF GRAIN

Washing of grain before milling so that 'the black grains would float uppermost' was recommended as long ago as 1699 (Hellwig, cited by Barger, 1931). Flotation in a solution of common salt (sodium chloride) has been advocated by a number of authors since the technique was developed in Russia in the early 1900s (Jaczewski, 1904, cited by Weniger, 1924). Gussow (1929) recommended that contaminated grain should be immersed in a solution prepared by dissolving 40 lb of salt in 25 gallons of water. On stirring, the sclerotia came to the surface and could be skimmed off. The grain was then washed with water and dried quickly to prevent injury to germination. Dickens (1974) endorsed this treatment for use with small quantities of barley seed but specified that the seed should be pre-soaked in water for about 3 hours before treatment. Nobbe (1904, cited by Barger, 1931) preferred a saturated solution of potassium chloride to sodium chloride, claiming that it did not damage the seed and suggesting that it could be used afterwards as a fertiliser.

The steeping and subsequent drying of grain is a time-consuming and costly process and it was advocated only because normal methods of grain cleaning had proved ineffective. Weniger (1924) commented that "cleaning of grain with ordinary cleaning and fanning mills will remove a large percentage of the unbroken, elongated ergot sclerotia from rye seed, but still leaves many broken ones and also those which do not develop beyond the size of the normal grain. A still larger proportion of ergot sclerotia remain in wheat seed when cleaned by the fanning mill since a larger proportion of them are about the same size as the normal grain". Methods of grain cleaning have obviously improved immensely since Weniger's day, though seed-sized sclerotia still make grain cleaning difficult - and the decontamination of grass seed even more so (Bradley-Jones, 1965).

The most recent data (Table 14) on the effectiveness of seed cleaning in reducing ergot levels appear to be those produced at the North of Scotland College of Agriculture in 1978 (Watson, 1979a).

TABLE 14. Effect of dressing on ergot content of grain

Original ergot content	After first dressing				After second dressing			
	Dressed grain	Dressings*			Dressed grain	Dressings*		
		coarse	fine	bulked		coarse	fine	bulked
	% ergot, by weight, of dry matter							
0.08	0.02	3.2	0.7	1.0	-	-	-	-
0.11	0.08	2.0	1.0	1.3	0.03	5.0	2.0	1.4
0.30	0.18	14.6	0.5	1.4	0.04	16.0	0.4	1.9

* coarse dressings: over 5.16 mm; fine dressings: through 2.2 mm

Dressing effectively reduced ergot levels in the grain. The high ergot levels in the dressings serve as a reminder that dressings from ergotised crops should not be used in feed mixtures.

13. CONTROL MEASURES

13.1. Control by management of husbandry

Summarising ADAS advice on the control of ergot, Dickens (1974) advocated the use of clean seed, ploughing and rotation of crops, the control of blackgrass in cereal crops, and the topping of headland grasses to prevent their flowering and acting as a source of honeydew. He recommended intensive grazing followed by periodic topping during the late summer to prevent the disease building up in pasture land.

Deep ploughing of fields which have suffered ergot attacks provides a very effective way of eliminating ergot inoculum as sclerotia buried below 250 mm do not germinate (Barger, 1931) and the clavae of those buried at 50 mm will seldom reach the surface (Bretag & Merriman, 1981).

Rotation of crops is also very effective as only graminaceous crops are infected and sclerotia normally germinate within one year or not at all (Barger, 1931). There is, of course, a need for good grass weed control in the break crops to prevent the carry over of inoculum. Gilmour (1982) has suggested that if there is no alternative but to take another cereal crop immediately after an infected crop of wheat, then spring barley, rather than a second wheat, should be sown.

The topping of hedgerow grasses is recommended, in particular, if blackgrass or meadow foxtail are present but one fenland farmer who has had problems with the disease has expressed grave concern at the possible ecological effects (particularly on nesting birds) of mowing his dyke sides just before grass anthesis.

Juxtaposition of early and late flowering cultivars should, where possible, be avoided, and husbandry should be such as to prevent an irregular crop in which some plants come into flower a long time before others (Barger, 1931).

Bretag (1985) found that burning ergot-contaminated wheat stubbles considerably reduced the number of viable sclerotia (an observation which, since the ban on straw burning, will remain of merely academic interest in the UK).

Hewett (1982) has suggested that when infection is seen, the edges of the crop (where infestation is likely to be highest) should be harvested separately to prevent the contamination of the whole bulk.

If, for some reason, ergot contaminated seed has to be used, Gussow (1929) recommended that seed should be drilled rather than broadcast and Bretag & Merriman (1981) advocated deep drilling to bury the sclerotia to such a depth that the clavae are unable to reach the surface. However, while deep drilling has been shown to be partially effective under experimental conditions, it has not been proven to be effective under commercial conditions where sowing depth is seldom precise (Shaw, 1986).

13.2 Fungicidal control

Benzimidazole (MBC) fungicides applied at the time of anthesis have been shown to have some prophylactic effect against the pathogen (Wood & Coley-Smith, 1980b) and such sprays have sometimes been employed by growers on the highly susceptible durum wheats (Peake, Harlow Agricultural Merchants, pers. comm). However, to be effective, the chemical must have access to the stigmata and ovaries and this can take place only when the florets are open. With florets of different tillers in the crop, and different florets on individual ears, opening at different times, spray timing presents obvious difficulties. Wood & Coley-Smith (1980b) suggested that for male sterile lines used to produce hybrid seed, sprays should be applied every 3 days between the time the first flowers open until harvest.

In glasshouse experiments, Hardison (1977) applied sodium azide or one of the azole fungicides (fenarimol, nuarimol or triadimefon) to the surface of soil contaminated with sclerotia from ryegrass. All the treatments suppressed ascocarp formation, the azoles being particularly effective. Triadimefon 'Bayleton' prevented the germination of the sclerotia at rates as low as 1.2 mg/92 cm^2 of soil surface. In field trials, however, spray and granular formations of the same fungicides gave incomplete control of sclerotial germination. Sodium azide was more effective but timing of application proved critical: 1.1.21 kg/ha suppressed stromatal formation almost completely when applied on 29 April or 11 May, but was ineffective when applied on 14 April.

Hampton (1984, cited by Shaw, 1986) found that triadimefon would prevent ascocarp formation in laboratory studies of Claviceps paspali but was relatively ineffective when applied to ergot infested plots in the field.

Shaw (1986) has pointed out the difficulties in applying fungicides to overwintered sclerotia which will often be covered by a litter of leaf debris.

Seed treatment Fungicide treatment of contaminated seed can reduce the risks of attack by reducing the viability of the sclerotia in the seed-lot. The use of such seed treatment was first investigated by Brentzel (1947) who found that treatment with formaldehyde or ceresan (2-methoxyethyl mercury chloride) completely inhibited the germination of ergots from barley.

Shaw (1986) treated sclerotia from wheat and triticale with phenylmercury acetate (as 'Agrosan D'), bitertanol + fuberidazole and triadimenol + fuberidazole ('Baytan'), all the chemicals being applied at the rates recommended by the manufacturers for seed-treatment. (It had previously been shown (Shaw, 1984) that, of the constituents of 'Baytan', triadimenol, but not fuberidazole, had activity against the fungus). The sclerotia were buried in the autumn in field soils at sites in Surrey and Essex and at the Welsh Plant Breeding Station, Aberystwyth. Contrary to Brentzel's findings, the organomercury had little or no effect on sclerotial germination but both of the other fungicide treatments reduced germination and few ascocarps emerged above the soil surface in the following spring. Pots of blackgrass placed in plots containing the treated sclerotia developed less ergot than those placed in plots containing untreated sclerotia.

In further trials, Shaw (1988) tested triadimenol plus fuberidazole on ergots from 10 different sources in trials at Great Dunmow, Essex and at the Welsh Plant Breeding Station. At both sites, both dry and flowable formulations of the fungicide reduced sclerotial germination, but the level of reduction was greater by 5-12% at Dunmow than at Aberystwyth. The Welsh site was on more freely drained soil and was subject to much higher rainfall than the Essex site. Under these conditions, there would have been more leaching of the fungicide during the winter months and a consequent diminution in its effectiveness. At a third site (in Wiltshire), Shaw (1988) found that, while untreated sclerotia decayed during the summer, many of those treated with fungicide were preserved and germinated in the following year, though still with reduced ascocarp production.

13.3 Biological control

Shaw's observations (1988) on the longevity of fungicide treated sclerotia suggested that hyper-parasitic fungi were responsible for hastening their decomposition. As long ago as 1884, Smith found "Fusicladium" (his figure reveals it to have been a Fusarium species) growing, apparently as a hyper-parasite, on ergot sclerotia on ryegrass. Kirulis (1942) considered the Fusarium avenaceum which he found growing on ergots in Latvia to be a hyper-parasite. More recently, Mower et al. (1975) have shown that a clone of Fusarium roseum 'sambucinum' acts as a highly effective biological control agent (BCA) for ergot. It will break down sclerotia and, when applied to the sphacelia stage, prevent the sclerotia developing. Infected sclerotia, if not broken down, show greatly reduced germination. The Fusarium also had the ability to break down ergotamine into substances which were found to be harmless when administered to rats and rabbits. However, even if a BCA active against ergot was commercially available, its application on a field scale to sclerotia protected by plant debris, or buried in the surface layers of the soil, would present even more difficulties than were encountered in the application of fungicides. The application of a BCA to contaminated seed might prove more effective, but, to be economically viable, it would need also to control other seed-borne disease (as does 'Baytan').

13.4 Genetic control

Platford & Bernier (1970, cited by Shaw, 1986) claimed resistance for one durum and one spring wheat cultivar in Canada, but in neither case did the resistance prove stable when exposed to a wide range of strains of the fungus. Willis (1953) found differences in the numbers of ergots in the various cultivars in variety x fertiliser trials in Hertfordshire, in particular cv. Cappelle Desprez contained almost twice as many ergots as any other cultivar. It may be, however, that the differences were linked with chance differences in the level of contamination of the different seed stocks from which the cultivars were grown; certainly Marshall (1960) found no differences between Cappelle Desprez and other cultivars in her studies of data from the Official Seed Testing Station. In the Scottish epidemic of 1978, ergots were confined mainly to cv. Maris Huntsman and were negligible or absent in other cultivars, including Cappelle Desprez (Watson, 1979b - see also Appendix III). Maris Huntsman was not more prone to open flowering than other cultivars though, as Watson observed, it did have shorter glumes and it may be that this made it

easier for the ascospore to enter the florets. These apparently conflicting observations support the conclusion of Shaw (1986) that "there have been no substantiated reports of physiological resistance to ergot in temperate cereals".

While there may be no genetic factors conferring physiological resistance to ergot, other genetically determined characteristics may reduce the plant's susceptibility to infection. Breeding against the tendency to open flowering should, for example, help to reduce susceptibility to the disease. The increased incidence of ergot in barley in the early 1950s may have been due to the introduction of the more open-flowered Scandinavian cultivars such as cv. Carlsberg and Rika (though this was not substantiated by OSTS data in 1956/57 (Marshall, 1960) when these cultivars were no more heavily infected than were the closed-flowering English cultivars). Selection against the tendency to produce later tillers could also help to reduce susceptibility to the disease (Shaw, 1986).

14. SUGGESTIONS FOR FUTURE WORK

14.1 Strains of ergot and the role of grass hosts

Despite excellent work carried out over many years, our understanding of the physiologic strains of C. purpurea remains fragmentary. The significance of blackgrass in the epidemiology of the disease is clear, but the role played by other grasses, and the way in which this role may vary from location to location, still requires further elucidation.

14.2 The effect of weather on epidemiology

As has been shown, there is a large body of information on the effects of weather parameters such as temperature and humidity on the various stages in the life history of the ergot fungus. Despite this, however, while the retrospective interpretation of epidemics has been relatively easy, it has proved difficult accurately to predict the severity of infection from the weather in spring and summer. In closed flowering cereals, the final level of infection is determined by conditions pertaining during a very brief period during anthesis. A knowledge of these conditions needs to be linked to information on the early build-up of inoculum, and on the time of flowering of the various cultivars so that disease development can be modelled and the need for fungicides more accurately assessed.

14.3 Spray timing and choice of fungicide

Epidemiological modelling would facilitate the accurate timing of sprays for the control of the disease. Careful timing is of particular importance while we have to rely on the MBC fungicides which have no curative activity against the disease. Spray timing trials would enable the accuracy of the models to be predicted. Fungicide comparison trials should investigate both the prophylactic and curative effects of new materials.

It is suggested that the following trials should be carried out:-

A. The efficacy of an MBC fungicide and one or two of the newer triazole materials should be compared on the highly susceptible durum wheat in small plot trials inoculated in the autumn with sclerotia. Single sprays of the materials under test should be applied at weekly intervals from

ear emergence to the milky ripe stage, and should be compared with unsprayed controls and with full spray programmes.

B. The fungicide showing most promise in the first series of trials should be further evaluated, using closer (3-4 day) spray intervals, on bread/feed wheat in the folllowing situations:-

i) with and without blackgrass contamination,
ii) autumn-sown winter cultivar vs. spring sown spring cultivar.

Close observations should be made on disease development (e.g. presence of honeydew on both crop and weed grasses) and these should be linked with weather records collected from as near as possible to the trial.

The trials should be conducted at 5 or 6 sites in climatologically differing parts of the country and over a period of at least three years to enable spray timing to be compared in different epidemiological situations.

14.4 <u>The need for an awareness leaflet for farmers and merchants</u>

i) to alert farmers and merchants to the symptoms of the disease (both honeydew and sclerotial stages in cereals and weed grasses),

ii) to summarise the current regulations governing the degree of contamination allowed in grain destined for seed, feed, milling etc,

iii) to spell out the risks of feeding contaminated grain to livestock,

iv) to outline the risks of infection in the various cereal species and the way in which these risks are influenced by weather conditions,

v) to present advice on the best methods of controlling the disease and of cleaning contaminated grain.

15. ACKNOWLEDGEMENTS

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Grain Contamination Survey

ADAS

1. Business Details

Business Name

Address

Postcode

Tel No.

Contact Name

2. Grain Details

Please complete as much of the table as possible for grain that you have purchased in the last two years

Grain Type	Quantity bought in 1990-1992 (Tons)	County/Parish	Price paid per ton £	Was grain price reduced?		Approx. %age reduction	Was price reduction due to ergot contamination?		Was any grain rejected due to ergot?		Approx %age rejected	Was there evidence of grass infestation in the rejected or reduced price grain?		Approx %age of grass infestation
				Yes	No		Yes	No	Yes	No		Yes	No	
Rye														
Triticale														
Durum wheat														
Winter wheat														
Spring wheat														
Barley														
Oats														

3. Grain Details

Please complete as much of the table as possible for grain that you have purchased in the last two harvest years. (*Approximate figures only are required*)

Grain Type	Quantity of grain bought (*in tonnes*)		If price was reduced due to **ergot**, please give the approx. *tonnage* of reduced price grain		If price was reduced due to **bunt** * please give the approx. *tonnage* of reduced price grain		What was the average **%age** reduction in price over the two years as a result of contamination?		Areas where contaminated samples were bought. (*District, parish or nearest town*)		Total number of consignments rejected over the two years due to contamination		Was there evidence that ergot was associated with grass weed infestation? (*Please tick*)		Was there evidence that bunt was associated with home saved seed? (*Please tick*)	
	1990	1991	1990	1991	1990	1991	Ergot	Bunt	Ergot	Bunt	Ergot	Bunt	Yes	No	Yes	No
Winter wheat																
Spring wheat																
Durum wheat																
Barley																
Oats																
Rye																
Triticale																

* or covered smut (barley)

4. Grain Re-cleaning

Over the last two years approximately what percentage of bought grain have you re-cleaned due to ergot contamination?

Grain Type	Approx. %age re-cleaned
Winter wheat	
Spring wheat	
Durum wheat	
Barley	
Oats	
Rye	
Triticale	

5. Effect of Grain Contamination on Marketing of Wheat

Please enter the approximate number of contaminated wheat grain lots sold in 1990/1991 and the type of market

Intended Market	Ergot contaminated		Bunt contaminated	
	No. of wheat grain lots sold	Actual market	No. of wheat grain lots sold	Actual mark
Seed				
Milling				
Feed				
Export				
Intervention				
Other (please specify).....				

ADAS/HGCA - ERGOT SURVEY

	Number of years over the past ten in which ergot has been perceived as a major problem	In the worst years what proportion of the consignments you handled were so affected by the disease that: a) they were rejected	b) price was reduced
Winter wheat			
Spring wheat			
Durum wheat			
Winter barley			
Spring barley			
Oats			
Rye			
Triticale			

Comments: (dates of the bad years would be helpful)

Name of Company:

Address:

Area from which grain is purchased:

16.2 APPENDIX II

Taxonomy of the ergot fungus

First considered to be abnormalities of the rye grains themselves, ergots were finally regognised as being fungal structures and were given the name Sclerotium clavus (De Candolle, cited by Barger, 1931). The clavae developing from the germinating sclerotia were at first thought to be growths of a separate fungus which the mycologist Fries (cited by Barger, 1931) names Cordyceps purpureum. Similarly, the honeydew stage was thought to be a distinct organism and was named Sphacelia segetum by Lévéillé (1827, cited by Barger, 1931). The full life history of the fungus and the part played in it by these three seemingly separate organisms, was finally established by Tulsane (1853, cited by Barger, 1931) who retained the specific epithet given by Fries but changed the genetic name to Claviceps, hence Claviceps purpurea (Fr.) Tul.

Claviceps purpurea can be classifed as follows (Webster, 1970):-

Division: MYCOTA - the true fungi (as opposed to the 'slime moulds' and their allies).

Subdivision: ASCOMYCOTINA - fungi in which sexual reproduction results in the production of spores in sacs or 'asci' from which, in most cases, they can be forcibly ejected and thus become air-borne. In most ascomycetes, vegetative spores, known as 'conidia', are also produced. In this case, spore production is not preceded by sexual fusion.

Class: PYRENOMYCETES - Ascomycetes in which the asci develop as a layer lining a flask shaped 'perithecium'.

Order: HYPOCREALES - Pyrenomycetes in which the perithecia are fleshy and brightly coloured, or are embedded in pale or brightly coloured, fleshy fruiting bodies known as 'stromata'.

Family: CLAVICIPITACEAE - a family of the Hypocreales, distinguished mainly by microscopic features, most of whose members as parasitic on grasses but some of which parasitize insects.

Genus: CLAVICEPS - the 'ergot' fungi

36 species of Claviceps have been described. Of these, 3 occur on sedges and the remainder on grasses. Most species are confined either to a single genus of grasses or sedges or, less frequently, to a group of related genera within a single tribe.

Species: CLAVICEPS PURPUREA (Fr.) Tul. C. purpurea has a much wider host range than other Claviceps species and has been recorded on 200 species in 50 genera representing 17 tribes within the sub-family Pooidea (Loveless, 1967).

16.3 APPENDIX III

SOME FACTORS INFLUENCING AN OUTBREAK OF ERGOT (CLAVICEPS PURPUREA) INFECTION OF WINTER WHEAT IN NORTHERN SCOTLAND

The following is an edited version of an unpublished paper by R D Watson (North of Scotland College of Agriculture) and B G Osborne (Flour Milling and Baking Research Association, Chorleywood). Details of the data presented in the paper can be obtained from S Wale, School of Agriculture, Aberdeen or D J Yarham, ADAS, Cambridge.

ABSTRACT

The location of 32 crops of wheat infected with ergot during an epidemic in 1978, and the variety of 19 of these crops were determined. One variety (Maris Huntsman) appeared to be more susceptible than others to infection. The source of infection was concluded to be grasses and not cereals. From the analysis of the alkaloid content of ergots, it was concluded than perennial ryegrass was the likely source of infection for the winter wheat. The distribution of infected crops showed no correlation with below average temperatures during the flowering period, but was closely correlated with abnormally high rainfall patterns during that part of the period in early July. It was concluded that the abnormally wet weather conditions during the flowering period of winter wheat in 1979 were a major cause of the epidemic with varietal susceptibility playing a lesser role.

INTRODUCTION

Sporadic outbreaks of ergot infection of cereals remain troublesome in the United Kingdom, but are rare in northern Scotland though the disease occurs commonly in grasses there. In 1978, ergot infection appeared widely in winter wheat crops in this area. The opportunity was taken to elucidate the causes of the epidemic.

MATERIALS AND METHODS

Thirty-two infected crops were located and in 19 of these the variety was ascertained. Alkaloid analysis was undertaken of ergots from 22 crops and a

similar analysis made of 14 samples of grass ergots from the 1980, 83 and 84 seasons, to provide a guide to the origin of the wheat infection.

No retrospective data on the flowering period of winter wheat crops in 1978 was available, but the period of head emergence in local winter wheat crop trials was determined as a guide to the flowering period. Temperature and rainfall for the deduced flowering period, and segments of that period, were then examined for relationships between weather patterns and the location of infected crops. Weekly and monthly averages of rainfall and temperatures for 9 stations in northern Scotland were analysed for their deviation from the average and compared with the distribution of infected crops. Isopleths of rainfall, expressed as a percentage of the 15 year monthly average were prepared for June, July and the periods 20 to 25 June, 26 to 30 June and 1 to 6 July, and plotted against the locations of infected crops. The deviations of rainfall and temperatures in 1978 from the 15 year averages were calculated.

RESULTS

Location of infected crops

The infected crops were grouped within the north eastern corner of the Scottish Lowlands, covering only a fraction of the total Scottish wheat growing area. Two meteorological stations lie within the area (Fyvie and Craibstone) and one on its border (Banff) and provided useful data on local weather conditions.

Varietal susceptibility

Ergots were found in 16 crops of cv. Maris Huntsman, 2 of cv Atou and 1 of Flanders. In 2 fields split between Maris Huntsman and Maris Nimrod, Huntsman showed significant infection when the other varieties had none or only trace levels of infection. In 3 cases where different fields on the same farm were of Maris Huntsman and other cultivars, again Huntsman showed significant infection when the other varieties showed trace levels or none.

Period flowering of winter wheat

Data from 4 winter trials in the North College area showed 50% head emergence occurred between the 16 to 18 June.

The weather throughout that period was about 1.2°C cooler than average, but there was no correlation between the degree of deviation from the norm and location outwith or within the area of the epidemic.

Considering the 2 stations sited within the area of the epidemic (Fyvie Castle and Craibstone) and one at its margin (Banf), the shortfall in seasonal temperature was not even particularly unusual.

Isopleths for June showed no correlation of rainfall with geographic distribution of the outbreaks. However, a dry period in the first half of the month and a wet period in the second combined to produce near normal average monthly rainfall. This could thus have concealed the influence of the wetter second two weeks when the wheat crops were in flower. Isopleths for July showed a strong correlation between the distribution of rainfall and the location of infected crops. Examining the crucial 4 weeks of flowering of 14 June to 7 July individually, the two periods up to 30 June showed no fit. Indeed the period 26 to 30 June had a very low rainfall and relative humidity. However, rainfall and the distribution of infected crops showed a close correlation for the period of 1-7 July.

DISCUSSION AND CONCLUSIONS

Location of infected crops

It is certain that the crops located with ergot represent only a small fraction of the total number of infected crops within that area. However, the combined observations of advisory pathologists and general agricultural advisers in the field, farmers and grain merchants, all of whom had been thoroughly alerted by publicity in the media, failed to produce the discovery or reporting of a single infected crop outwith that area. Considering that all farmers inspect their crops regularly as they approach harvest, and that ergot infection of cereals is a striking disease quickly noticed by growers,

it can reasonably be deduced that the geographic area approximately defined by the 32 infected crops represents the extent of the outbreak.

Varietal susceptibility

86% of the infected crops were of the cultivar Maris Huntsman, a cultivar which occupied only 75% of the total average of wheat grown in the area. This and the observations from fields split between Maris Huntsman and Maris Nimrod, and of comparisons of closely situated fields under Huntsman and other varieties, where Huntsman showed significant levels of infection and other varieties did not, suggests that Huntsman is more susceptible to ergot infection. Observations by ADAS on infected crops in 1981 (unpublished) also indicated that Huntsman was a more susceptible variety. Varietal susceptibility thus probably played a part in the epidemic.

Source of infection of winter wheat

Whether wheat was infected by ascospores arising directly from germinating ergots, or by conidia derived from the honeydew stage in an infected cereal host, the infection must have arisen originally from ergots carried over from 1977. The two possible sources of these ergots are grasses or cereals. Ergot infection of cereals is very rare in Northern Scotland. In the 5 or more years pervious to 1978, for example, not a single observation of ergot infection of cereals was reported to the advisory services or observed by their staff, despite the many crop and trial inspection carried out by pathologists and other advisory staff of the North of Scotland College of Agroculture. The experience of grain merchants was similar. Grasses, and not cereals were therefore the source of infection.

Ergot occurs widely on various grass species in northern Scotland in most years. The intensity of infection varies with the year, but no observations had been made on the level of infection in 1977. Ergot infection in grasses was observed to be unusually widespread in 1978 with species such as Elymus repens Agrostis tenuis, Lolium perenne, Dactylis glomerata and Phleum pratense carrying heavy infestations.

In a series of papers, (e.g. Mantle and Shaw, 1977; Mantle et al; 1977), Mantle and his co-workers showed that C. purpurea exists in a number of physiological races, each with a specialised host range of grass species and

usually one or more cereal. They also demonstrated that the spectrum and proportions of alkaloids found in ergots were characteristic for each race of the pathogen. Ergots from wheat and blackgrass (Alopecurus myosuroides) in Southern England were similar in being rich in ergotoxine, with smaller amounts of argosine and ergotamine, suggesting the two hosts shared the same race. Both were dissimilar in this respect to ergots from perennial ryegrass and other grasses. Based on this evidence, and evidence from cross infection experiments, field observations on infected crops, and infection rates on wheat plots seeded with wheat ergots and mingled with various grass hosts, they concluded that ryegrasses were not a significant source of wheat infection, the chief source of which, they concluded, was blackgrass.

This conclusion cannot apply in Scotland, where blackgrass does not occur. Further Osborne & Watson (1980) showed Scottish wheat ergots were rich in ergotamine with lesser amounts of other alkaloids, making them distinct from Spanish and European cereal ergots. Their results also demonstrated that they were distinct from both English wheat ergots, and English blackgrass ergots in their pattern of alkaloid spectrum and content, as analysed by Mantle et al. (1977) but, significantly, were similar to perennial ryegrass ergots examined by these workers.

The chromatograms obtained in the present study showed that the alkaloid pattern for wheat and perennial ryegrass are quite similar to each other and distinct from creeping softgrass, cocksfoot and fescue. More specifically, both wheat and perennial ryegrass are rich in ergotamine and have relatively low levels of ergosine. All the samples examined had relatively low levels of the ergotoxine group (ergocornine, ergocryptine and ergocristine) but while the wheat and perennial ryegrass contained all three constituents of ergotoxine, ergocorine was absent in the other grass ergots. On the basis of the semiquantative criteria employed by Mantle & Shaw (1977), it seems evident therefore that, of the species examined, perennial ryegrass is the likely source of ergot infection for wheat in Northern Scotland.

It can be shown that the ergocryptine/ergotamine and ergocryptine/total alkaloid ratios for wheat and perennial ryegrass ergots are not significantly different at the 5% level. The ergotamine/total alkaloids ratios are, however, significantly different at the 1% level. Therefore, while the ranges of the data in this study overlap with those previously reported for wheat

(Osborne & Watson, 1980), it cannot be unreservedly concluded that the wheat and perennial ryegrass ergots share the same alkaloid spectrum.

Weather pattern during the winter wheat flowering period

From examination of the date of 50% head emergence in winter wheat trials over a number of years, it is evident that 50% head emergence at 16 to 18 June, as occurred in 1978, was normal. Since the 4 trial sites were deliberately widely spaced in climatically diverse areas of the winter wheat land, and since these events are regulated largely by day length, it seems very likely that these observations were a good indication of the stage of growth of the general run of crops.

Flowering in wheat commences 4 days after head emergence and is completed in 6 to 8 days in dull, wet weather as prevailed over much of the flowering period in 1978. Flowering, therefore, took place in 1978 at least over the period 20 to 30 June. The experience of specialist crop trial officers and crop husbandry advisers has shown that variation between crops, even within one variety, would extend this period by a week on either side. The full flowering period of winter wheat in northern Scotland would therefore have been 14 June to 7 July.

There is no evidence that the slightly below average temperatures during the flowering period of winter wheat played a significant role in initiating the epidemic. However, there was a close fit between above average rainfall and the distribution of infected crops for the period from the 1 to 7 July. Parallel data for rainfall over exactly the same periods, expressed in absolute terms and as a % of average, for individual stations was not available, but the very high rainfall for the period 28 June - 4 July is also striking. It may be significant, in this respect, that at the two meteorological stations within the area of the epidemic, and at the one on its borders, there was rain on 6 out of these 7 days. It therefore seems highly likely that the high rainfall during part of the flowering period of winter wheat crops played a crucial role in initiating the epidemic. This hypothesis is stengthened by the absence of any epidemic in 1979, when the weather pattern did not repeat itself. Only one infected crop was reported despite the high levels of potential inoculum that must have been available from grass ergots and locally shed cereals ergots from the previous year, and the development of very high levels of ergot infection in various grass species in

1979, which could have acted as a source of infection through conidia arising at the honeydew stage.

16.4 APPENDIX IV

A CASE OF GANGRENOUS ERGOTISM IN ENGLAND

The following extract from Smith's "Diseases of Field and Garden Crops" describes what appears to have been the only recorded case of gangrenous ergotism in the UK. Commenting on the case, Barger (1931) noted that the diet of the family included "dried peas, pickled pork, cheese and milk". They would not, therefore, have been suffering from the vitamin A deficiency which can predispose sufferers to the convulsive, rather than gangrenous, form of the disease.

"In the Philosophical Transactions (1763) vol. lii., part ii., for the year 1762, p. 526 is a printed extract from a letter from the Rev. James Bones, M.A. of Wattisham, near Stowmarket, Suffolk, to George Baker, M.D., F.R.S., relating to a case of mortification of limbs in a family there.

The letter says that on Sunday, 10th January, Mary, daughter of John and Mary Downing, sixteen years old, felt a violent pain in her left leg, which, in a hour or two, also affected her foot, and particularly her toes. On the next day her toes were much swollen, and black spots appeared on them. By degrees the whole foot became swollen and black. The pain, which was now chiefly in her toes, was, as she said, as if dogs were gnawing her. The blackness and swelling advanced upwards by slow degrees, till they reached the knee, where the flesh broke, and a great discharge followed. In a little time the flesh of her leg putrified and came off at the ankle together with the whole foot, leaving the leg bones bare. Her other foot and leg were affected in a few days, and decayed, nearly by the same degrees and the same manner. She had then an abscess formed in one of her thighs. In a subsequent note it is stated that this girl, who had sat for fourteen days in a chair, and for seven days without any feet, or flesh on her leg bones, had at length consented to have the bones taken off.

Mary, the mother, was seized within a few hours of her daughter's first seizure with the same violent pain under her left foot, or, as she sometimes said, in her left leg. Her toes, foot, and leg were affected in the same manner as her daughter's; and in a few days her other leg and foot suffered in like manner. The flesh of one leg had separated, and come off at the knee, leaving the bones bare, which she would not suffer to be taken off. The other

foot had rotted at the ankle. Her hands and part of her arms were, from the first attack, without sensation, and her fingers contracted. In a subsequent letter it is stated that the mother "still remains in bed with her leg bones bare, which she will not suffer to be taken off".

In four or five days after the eldest daughter and the mother were first affected, Elizabeth, aged fourteen years, Sarah, aged ten, Robert, aged six, and Edward, aged four, were all taken on the same day with violent pains in the feet and legs, chiefly in the left.

Elizabeth was seized only in one leg and foot, which, during three weeks, she could not set on the ground, but stood all the time on the other foot, leaning against the chimney; after which, being taken in the same manner in the other foot, she was obliged to lie down. One foot mortified and came off at the ankle, and the other leg near the knee.

Sarah was taken in one foot, which mortified and came off at the ankle. The other leg suffered in the same manner, and also separated at the knee.

Robert was taken in both feet. His legs separated at the knees ... Edward was taken in both feet, which separated at the ankle ... etc.

The report then describes the death of an infant, whose hands and feet turned black after death, and the illness of the father, whose fingers were benumbed, contracted, and black, the nails coming off, and two of the fingers becomming ulcerated. Then follows a description of the persons afflicted and their food, but no hint is given as to the presence of ergot.

In the subsequent letter, before referred to, it states at page 530 that the family had no rye, but had been "used to buy two bushels of clog-wheat, or revets, or bearded wheat ... every fortnight. Of this they made their household bread". The wheat, it appears, had been laid, and was thrashed separately lest it should spoil the samples. It was not mildewed or grown, but discoloured and smaller than the other. It made bad bread and worse puddings. A labouring man who used the bread was affected with a numbness of both his hands for about four weeks. His hands were continually cold, and his finger ends peeled. One thumb was at the time of the report still without any sensation."

16.5 APPENDIX V

The following poem, translated from the original French, was written by M. Marcel Delacquis of Pont-Saint-Espirit while suffering from convulsive ergotism in the summer of 1951 (Fuller, 1968).

Dark hours - ashes
That fall and cover up
All the things, and the soul.

Dark hours - opaque vapour
That comes up from the bottom of everything
And fatally like an ebb
Invades the voids of life.

Dark hours - monotonous song
In a minor key, sad and full of torture;
Rhythmic sting of the daggar
Into the mass of brain.

Dark hours - acid taste
That makes the mouth grin
The whole tragedy of life,
The dark hours.

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(The list includes some of the more important references referred to as having been cited in the works of other authors).

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