

ADIASPIROMYCOSIS OF WILD SMALL MAMMALS IN AUSTRIA

Z. HUBÁLEK, W. SIXL*, Z. ŠEBEK**, D. STÜNZNER*, H. TROGER* and M. VALOVÁ**

Institute of Parasitology, Czechoslovak Academy of Sciences, Prague, *Institute of Hygiene, University of Graz, Graz, and ** District Hygiene Station, Jihlava

Abstract. Adiaspores of the fungus *Emmonsia crescens* Emmons et Jellison were found in the lungs of 10 rodents (*Clethrionomys glareolus*, *Microtus agrestis*, *M. oeconomus*, *Apodemus flavicollis*) out of 195 small mammals examined (Soricidae, Microtidae, Muridae) belonging to 15 species, trapped in May and June 1977 in various localities and habitats of Austria. This is the first evidence of adiaspiromycosis in Austria, and apparently also the first European record of adiaspores in *M. oeconomus*.

The information (Dvořák 1969, Dvořák et al. 1973) on the geographic distribution and frequency of adiaspiromycosis, a pulmonary disease of mammals caused by the fungi of the genus *Emmonsia* (predominantly by *E. crescens* Emmons et Jellison), is relatively scarce. This infection has been observed in some European countries (Bulgaria, Czechoslovakia, Finland, France, German Democratic Republik, Great Britain, Italy, Norway, Portugal, Spain, Sweden, Switzerland, USSR, Yugoslavia), but no search for adiaspiromycosis has been carried out in Austria. During an investigation of natural foci of various diseases in Austria, organized by the Institute of Hygiene, University Graz, a number of small mammals was examined for the presence of adiaspores of *Emmonsia*.

MATERIAL AND METHODS

Table 1 shows a survey of 195 mammals examined. The animals were trapped in various localities and habitats from May 24 to June 3, 1977 (Table 2). The lungs of the mammals were placed in plastic ampoules (2-ml capacity; see Bárdóš and Hubálek 1976) containing cca 1 ml of a 5 % formaldehyde solution, sealed, and transported to a stationary laboratory. Potassium hydroxide (2 %) solution was then substituted for the formalin, and the lung tissue was examined microscopically after a period of 2 days at 20 °C.

RESULTS

Adiaspores of *E. crescens* were found in 10 rodents (Tables 1, 3) of the species *Clethrionomys glareolus* (8.8 % infected), *Microtus agrestis* (3.6 %), *M. oeconomus*, and *Apodemus flavicollis* (6.2 %), the insectivores did not show the disease.

All infected animals were adults. However, only 15 juvenile of subadult mammals were examined and therefore, no conclusion should be drawn from this result. The distribution of adiaspiromycosis was not proved to be significantly (chi-square test, $P > 0.30$) sex-correlated; the infection rates of males and females were, respectively: *C. glareolus* 11.1 % and 6.7 %; *A. flavicollis* 7.4 % and 4.8 %, the average in all mammals 7.0 % and 3.7 %.

Table 1. Survey of the mammals examined, and the frequency of adiaspiromycesis

	The number of animals		Infection rate
	examined	infected	
Insectivora			
Soricidae, Shrews	18	0	0 %
<i>Sorex araneus</i> Linnaeus, Common Shrew	18	0	0 %
<i>S. alpinus</i> Schinz, Alpine Shrew	5	0	
<i>Neomys fodiens</i> Pennant, Water Shrew	4	0	
<i>N. anomalus</i> Cabrera, Miller's Water Shrew	8	0	
Rodentia	1	0	
Microtidae, Voles	177	10	5.6 %
<i>Clethrionomys glareolus</i> (Schreber), Bank Vole	94	7	7.4 %
<i>Pitymys subterraneus</i> (de Sélys-Longchamps), Pine Vole	57	5	8.8 %
<i>Microtus arvalis</i> (Pallas), Common Vole	6	0	
<i>M. agrestis</i> (Linnaeus), Short-tailed Vole	2	0	
<i>M. oeconomus</i> (Pallas), Root Vole	28	1	3.6 %
Muridae, Mice and Rats	1	1	1/1
<i>Micromys minutus</i> (Pallas), Harvest Mouse	83	3	3.6 %
<i>Apodemus flavicollis</i> (Melchior), Yellow-necked Field Mouse	1	0	
<i>A. sylvaticus</i> (Linnaeus), Wood Mouse	48	3	6.2 %
<i>Rattus norvegicus</i> (Berkenhout), Brown Rat	11	0	0 %
<i>R. rattus</i> (Linnaeus), Black Rat	4	0	
<i>Mus musculus</i> Linnaeus, House Mouse	1	0	
Total	18	0	0 %
	195	10	5.1 %

DISCUSSION

The mean frequency of the small mammals infected by *E. crescens* in Austria (5.1 %) is comparable to other surveys of Rodentia and Insectivora in Europe and elsewhere: e. g., Norway 1.0 % (Jellison et al. 1960 b), Sweden 3.3 % (Paldrok and Zetterberg 1962), USSR 6.1 % (Sharapov 1969), Czechoslovakia 1.9 % (Prokopič 1971), Portugal 0.8 % and Spain 3.9 % (Doby et al. 1971 a), France 14.1 % (Doby et al. 1971 b), Bulgaria 2.5 % (Zlatanov and Genov 1975).

Microtus oeconomus was proved for the first time as the host of *E. crescens* in Europe; the sole previous record in this species is apparently from Siberia, where 5.9 % of 563 animals were infected (Sharapov 1969). In *M. agrestis*, adiaspores of *E. crescens* were observed in Norway (Jellison et al. 1960 b), Finland (Jellison et al. 1960 a), Sweden (Jellison et al. 1961 b), Switzerland (Hörning and Hörning-Pezenburg 1962), USSR (Sharapov 1969, 5.5 % infected), France (Doby et al. 1970, 4.7 %) and Czechoslovakia (Prokopič 1971, 3.4 %). *Clethrionomys glareolus* was found to be the host of *E. crescens* in Great Britain (Tevis 1956, 31 %), Norway (Jellison et al. 1960 b), France (Jellison et al. 1961 a, Doby et al. 1971 b, 17.8 %), Switzerland (Hörning and Hörning-Pezenburg 1962, 18.2 %), Sweden (Paldrok and Zetterberg 1962, 11.1 %), Czechoslovakia (Prokopič et al. 1965, Prokopič 1971, 2.5 %), USSR (Sharapov 1969, 7.4 %), Bulgaria (Pavlov et al. 1971) and Spain (Doby et al. 1971 a, 5.3 %). Adiaspores in *Apodemus flavicollis* were noted in Sweden (Jellison 1956, 5.7 %), Great Britain (Tevis 1956, cca 3.3 %), Czechoslovakia (Prokopič et al. 1965, Prokopič 1971, 2.9 %), USSR (Sharapov 1969) France (Doby et al. 1971 b, 20.3 %) and Bulgaria (Pavlov et al. 1971, 23.5 %).

Among the more frequent species in this study, *C. glareolus* seems to be the most often infected host. *A. flavigollis* was also infected relatively frequently, but no spherules of *E. crescens* were observed in a closely related species *A. sylvaticus*. The infection rates found in Czechoslovakia (Prokopič 1971) were 2.9 % and 0.3 % for *A. flavigollis* and *A. sylvaticus*, respectively, in Bulgaria (Pavlov et al. 1971) 23.5 % and 6.4 %, in France (Dobý et al. 1971b) 20.3 % and 12.7 %. The differences between both species have been significant for Czechoslovakia ($P < 0.02$) and Bulgaria ($P < 0.01$), and it appears that the incidence of adiaspiromycosis in the wood mouse is, in general, markedly lower than in the Yellow-necked field mouse. The reason might be the variability between the habitats of both species: *A. flavigollis* prefers more humid and cool places in woods, whereas *A. sylvaticus* usually occurs in more dry habitats in open country: edges of woods, bushes, etc. (Kratochvíl and Rosický 1953). According to Sharapov (1972),

Table 2. List of localities and habitats, and the infection rates of adiaspiromycosis

Locality, approx. elevation (a.s.l.) Date	Habitats	The number of animals		Infection rate
		examined	infected	
Steiermark Grambach bei Graz, 380 m May 24	A deciduous and mixed forest, brook banks	61	6	9.8 %
Pöls a.d.W., 320 m May 25	A deciduous wood (Querceto-Carpinetum)	29	1	3.4 %
Zwaring- - Wundschuh 310 m May 25	A humid lowland forest, dominated by alder	21	0	0 %
Wundschuh bei Graz, 330 m May 26	Buildings and gardens in the village	19	0	0 %
Tobelbad bei Graz, 350 m "Vogelfarm" May 26	A mixed wood (edge), brook banks	25	1	4.0 %
Mühlen bei Neumarkt "Hochmoor" 1100 m May 28	Peat-moor with small groves of birches and willows	21	1	4.8 %
St. Georgen bei Neumarkt 1000 m May 29	Shrubs along mountain brooks	11	0	0 %
Burgenland — Neusiedler See Mörbisch, 130 m June 1	A tip ("Müllplatz") near a lowland forest	4	0	0/4
Oggau; Weiden 120 m June 2; 3	The lake littoral with reed, cattail, sedges and willows	4	1	1/4

Table 3. List of the mammals infected by *Emmonsia crescens*

No.	Species	Sex Age	Loca- lity	No. of adias- pores per lung	Adiaspore diameters (wall thickness in parentheses), μm — examples			
A 32	<i>C. glareolus</i>	♂ ad.	Grambach	2	375	(24),	351	(25)
A 51	<i>C. glareolus</i>	♀ ad.	Grambach	1	222	(12)		
A 59	<i>C. glareolus</i>	♂ ad.	Grambach	19	457	(44),	429	(24), 423 (32), 398 (29), 391 (46), 387 (38), 366 (37), 362 (41), 331 (37)
A 62	<i>C. glareolus</i>	♀ ad.	Grambach	1	262	(34)		
A 87	<i>C. glareolus</i>	♂ ad.	Pöls	2	404	(46),	370	(28)
A 236	<i>M. agrestis</i>	♀ ad.	Mühlen	102	485	(48),	441	(35), 413 (46), 403 (33), 400 (46), 388 (37), 366 (41), 345 (41)
A 437	<i>M. oeconomus</i>	♂ ad.	Oggau	2	335	(32),	320	(30)
A 40	<i>A. flavicollis</i>	♂ ad.	Grambach	1	193	(22)		
A 42	<i>A. flavicollis</i>	♂ ad.	Grambach	63	178	(18),	153	(19), 150 (18), 141 (21), 141 (18), 137 (23), 135 (10), 123 (15), 109 (9), 75 (7), 69 (12), 69 (8), 49 (4)
A 208	<i>A. flavicollis</i>	♀ ad.	Tolbebad	3	337	(26),	200	(28), 156 (9)

Remark: The females A 51, A 208 and A 236 were pregnant.

the development of the mycelial phase of *E. crescens* in nature should be generally better at humid and sheltered sites than at more isolated places with a low humidity of the soil.

We found no adiaspores in the lungs of Soricidae and synanthropic species *Mus musculus*, *Rattus norvegicus* and *R. rattus*. This observation is in agreement with a relatively very low incidence of adiaspiromycosis in shrews and synanthropic rodents as described in other surveys (Sharapov 1969, Doby et al. 1971b, Dvořák et al. 1973).

A seemingly higher over-all infection rate of males (7.0 %) against females (3.7 %) proved to be insignificant, perhaps because the number of infected animals was not high enough for a more reliable statistical evaluation. Prokopič (1971) analyzed a more extensive material (162 infected mammals), but he also found only insignificant differences between males and females (and adults vs. subadults, too) in the frequency of adiaspiromycosis. On the other hand, the male white mice were observed to be more susceptible to experimental intraperitoneal infection by *E. crescens* than the females (Hamáček et al. 1972).

A rough estimation is possible of the probable time of the infection onset, evaluating the size of the adiaspores produced in the host (Dvořák et al. 1973). There are, of course, some limitations of this method (cf. Boisseau—Lebreuil 1970, 1972, Dvo-

řák et al. 1973), e. g., differences of the adiaspore mean size among strains of *E. crescens*; different dimensions of adiaspores produced from a strain in various hosts; a simplifying assumption of the linear growth of the adiaspore diameter in time. Nevertheless, it seems likely that most mammals were infected in the winter 1976/77 or early spring 1977, while the specimen A 42 in late April or early May 1977; the specimens A 59, A 87 and A 236 seemed to contract the disease in the autumn or summer 1976.

АДИАСПИРОМИКОЗ ДИКИХ МЕЛКИХ МЛЕКОПИТАЮЩИХ В АВСТРИИ

З. Губалек, В. Сиксл, З. Шебек, Д. Штюнзнер, Г. Трогер и М. Валова

Резюме. Обнаружены адиаспоры грибка *Emmonsia crescens* Emmons et Jellison в легких 10 грызунов (*Clethrionomys glareolus*, *Microtus agrestis*, *M. oeconomus*, *Apodemus flavicollis*) из общего числа 195 исследованных мелких млекопитающих (Soricidae, Microtidae, Muridae), относящихся к 15 видам и добытых в мае и июне 1977 г в разных местах их обитания в Австрии. Эта находка адиаспиромикоза является первой в Австрии и по-видимому первым в Европе свидетельством обнаружения адиаспор у *M. oeconomus*.

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A. Graw: Viren — Parasiten unseres Lebensraumes. *Heidelberger Taschenbücher, Band 192, Springer-Verlag, Berlin—Heidelberg—New York 1977, 179 pp. Price 19.80 DM.*

Small in scope, but extensive in content this book gives an outline of virology in all basic trends concerning animal and plant viruses. It opens with a historical introduction which is followed by five comprehensive chapters.

The first chapter, entitled "Virion", deals with the structure, biochemical properties and classification of viruses and with some basic methods used in relevant studies.

The second chapter, entitled "Virus in laboratory", summarizes knowledge on the mechanism of virus multiplication. Virological experimental systems, all most important diagnostic methods are also discussed. The chapter ends with a survey of data on virus interference.

The third chapter entitled "Virus as parasite" is concerned with virus reservoirs and provides the most important information on virus pathogenesis. It also treats viruses as mutagens, their role in human embryopathy and foetopathy, and their importance as oncogenic factors.

The fourth chapter entitled "Our control of viruses" reviews virus disinfection, antiviral

chemotherapy, antiviral protection by means of interferon, and covers immunotherapy and immunoprophylaxis.

The last chapter indicated as "Supplement" presents a table for the calculation of density gradient used in centrifugation, and a nomogram for the determination of the nucleic acid content.

All chapters are profusely and well documented. At the end of the book there is a list of references divided according to relevant chapters, and a subject index.

This is an excellent handbook not only for virologists, but also for other research workers concerned with microbiology. Besides presenting a good orientational outline of most important up-to-date trends and knowledge achieved in virology, the book is a valuable guide, mainly for those who are not directly involved in virology and unfamiliar with the abundant virological terminology. It will also provide useful information for parasitologists. Although the book had to be very brief, sometimes using only definitions, its style makes it intelligible and appealing to a wide professional audience.

MUDr. D. Málková, D.Sc.