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CAN EMMONSIA CRESCENS EMMONS ET JELLISON 1960 — THE CAUSATIVE AGENT OF ADIASPIROMYCOSIS — BE DISTRIBUTED BY THE EXCREMENTS OF MICE?

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Abstract. It has been confirmed in experiments that the conidial stage of the causative agent of adiaspiromycosis can be distributed by mouse excrements. Conidia, and possibly also mycelial fragments, of *Emmonsia crescens* survive the passage through the digestive tract of mice. They are capable of growth, reproduction and of causing animal infection. The importance of this finding for the circulation of *E. crescens* in nature, and for the origin of a natural focus of adiaspiromycosis, has been pointed out by the author.

Most importance, as regards the distribution of adiaspiromycosis, has been ascribed to the saprotrophic, conidial stage of its aetiological agent, the fungus *Emmonsia crescens*. It has been suggested that infection is acquired mainly by inhalation of the conidia (aleuries) of this fungus (Otčenášek et al. 1972). This mode of infection occurred, apparently, also in the case of adiaspiromycosis in the lung of man (casus Viničné-Šumperk) (Kodousek et al. 1971). In the host tissue, the conidia convert into large chlamydosporic formations (spherules, adiaspores) occupying the inside of a granuloma of typical morphology, e.g., Hejtmánek and Kodousek (1972). No confirmation is available on the fact that elements of *E. crescens* are released by either animals or man into the external environment.

Dvořák et al. (1970) suggested that *E. crescens* lives as a saprophyte in the soil, mainly in the burrows of small mammals. The conidia of this fungus can be easily distributed by air. Apparently, they are carried to the body surface of the inhabitants of the burrows and contaminate also the food of these animals. By licking the pelt and consuming food contaminated with conidia, these may easily penetrate the digestive tract of the animals and be released with the excrements to the external environment. In this way, the environment may be constantly contaminated with conidia of *E. crescens* and, hence, represents a potential source of infection. This mode of distribution of adiaspiromycosis presupposes that *E. crescens* survives the passage through the digestive system of the animals. We have tried to confirm this possibility in experiments.

MATERIAL AND METHODS

In our experiments, we used cultures of *Emmonsia crescens* Emmons and Jellison 1960, strain EC-1 (= 1815 from Dr. J. Dvořák, Dr.Sc., Pardubice), which we left to grow for one month on Sabouraud's glucose agar plates at 26 °C.

The experiments were performed in two variants and were three times repeated.

1. Conidia with the mycelium were taken from the surface of the *E. crescens* culture and suspended in water. Conidial density was not assessed because we did not intend to make a quantitative evaluation. The water suspension of conidia and hyphal fragments was the only liquid available to the experimental mouse (strain H), which was kept on a Larsen diet in the metabolic cage (a system of barriers constructed by Kavalier Glassworks Czechoslovakia) with forced air-circulation.

2. The agar plate with the *E. crescens* culture was cut into prisms (length of the sides approximately 3 mm); these were mixed with crushed, sterile pellets of the Larsen mixture at a ratio of approximately 1 : 5. This mixture, and pure water, was the only food of the mouse (strain H) kept under these conditions. In both variants, the mice was starved 24 hrs previous to the application of the pathogenic material.

After three days the mice were killed, disinfected by submergence into a 1 % solution of Ajatin, and dissected. Samples taken from the content of the stomach, the small and large intestine above the anal pore (sample volume 0.25—0.5 cm^3) were suspended in 30 ml of sterile physiological saline. The suspension was adequately diluted in sterile saline and spread in amounts of 0.1 ml on plates with Sabouraud's glucose medium. The plates were incubated at 26 °C, the remnants of the suspension injected intraperitoneally to 15 mice of the conventional strain H. After 22 days, the mice were killed and inspected in post-mortem for the presence of adiaspiromycotic granulomas.

Three mice of the same strain were used as controls; they were not infected with pathogenic material and examined in the same way. Technical assistence in this work was given by Mrs. A. Hanelová, Mrs. J. Valová and Mr. J. Jančèek to whom our thanks are due.

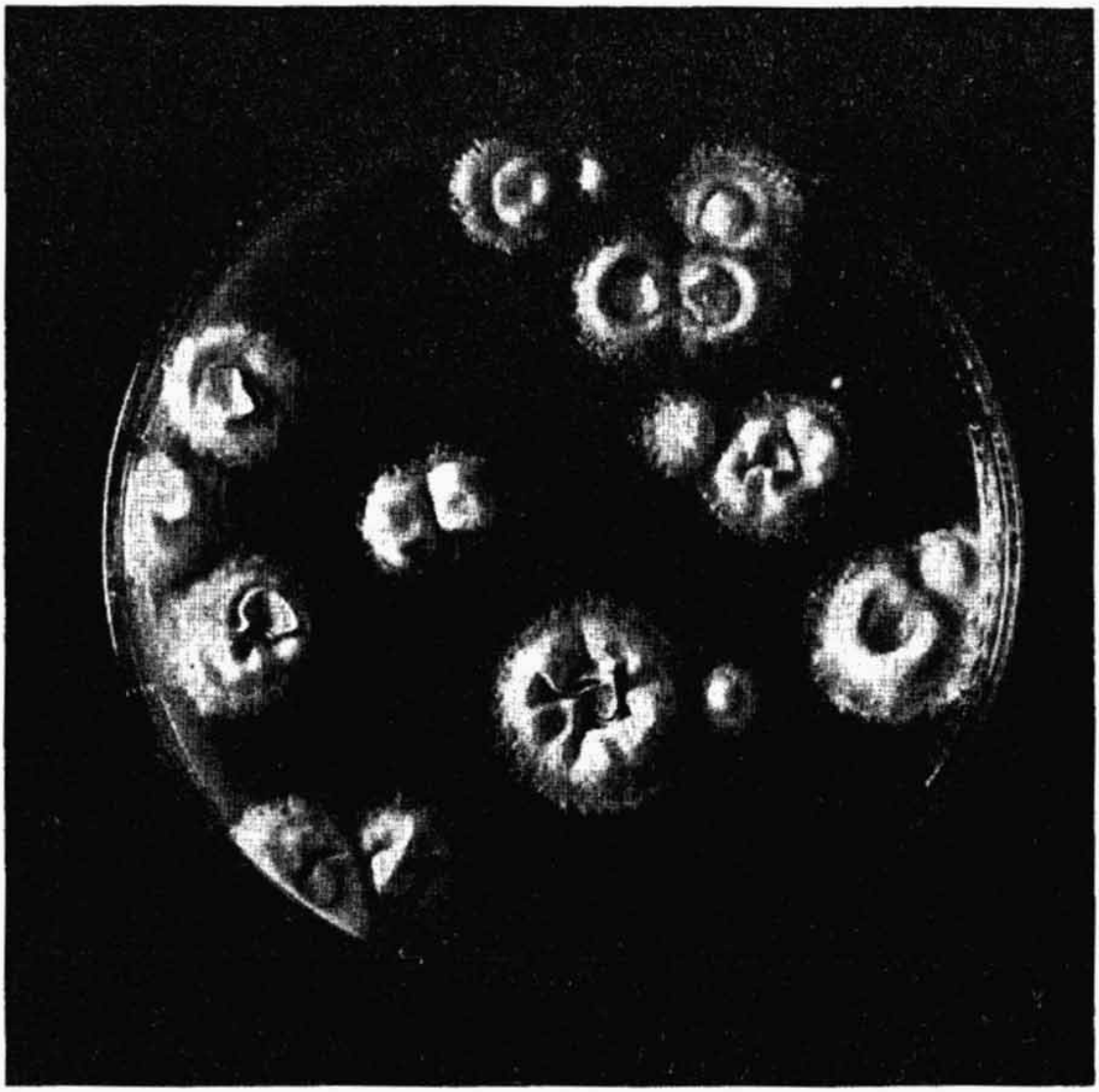

Fig. 1. Recultivation of *Emmonsia crescens* from the intestinal content of mice. (Sabouraud's glucose agar, 26 °C, 14 days).

RESULTS

From the content of the stomach, the small intestine, and from faeces formed in the large intestine of per os inoculated mice, typical colonies of *Emmonsia crescens* (Fig. 1) started to grow on the agar plates within 6—14 days. Sometimes, we found as many

as 0.5—2 million viable *E. crescens* elements in approximately 0.25 cm³ of samples from the intestinal content or from that of stomach. Although the number of viable elements appeared to be extremely high, it did not indicate whether all elements had survived the passage through the intestinal tract; neither could we derive from it how many of the viable elements were conidia, how many hyphal fragments. The reason for this is the fact that the actual dose of pathogenic material received by the mouse had not been characterized quantitatively.

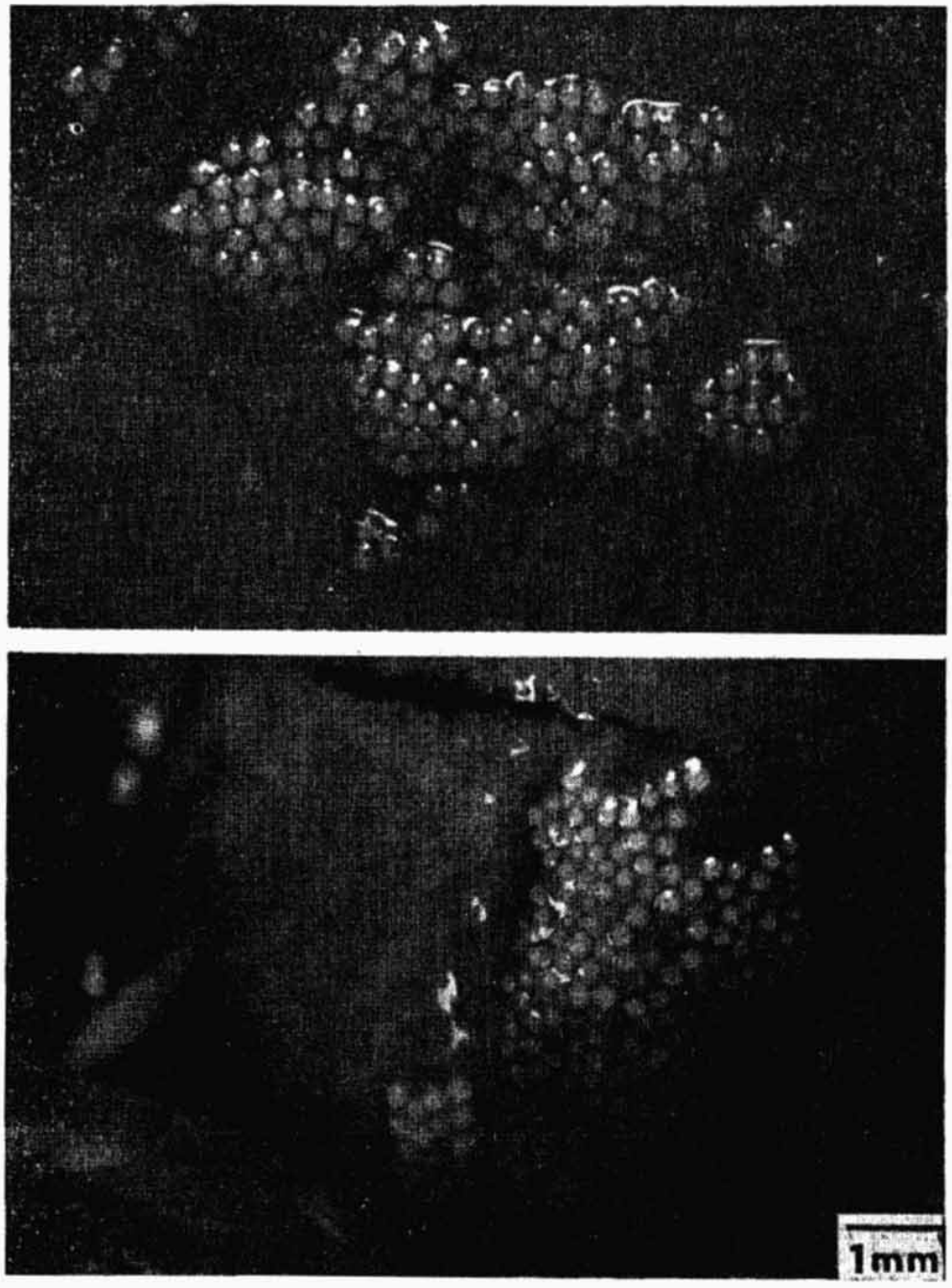

Fig. 2. Adiaspiromycotic granulomas on the peritoneum (top) and on the kidney (bottom) in mice on day 22 of i.p. inoculation (10 : 1).

In mice, inoculated intraperitoneally with the same material as that used on the agar plates, we found numerous adiaspiromycotic granulomas (Fig. 2) with typical adiaspores. Their description has been given in an earlier paper (Hejtmánek and Kodousek 1972).

The presence of *E. crescens* was not confirmed in the digestive tract of mice from the control group. The metabolic cage proved to be very suitable for these purposes and may be recommended also for inoculation by inhalation.

DISCUSSION

Our experimental work confirmed that conidia of *Emmonsia crescens* (and, possibly, also hyphal fragments) pass the digestive tract of mice in a viable state. The faeces of mice and, possibly, those of other mammals ingesting food containing conidia of *E. crescens*, are a potential source of adiaspiromycosis, because they contain viable elements of the causative fungus, which are capable of growing, reproducing and causing infection.

The knowledge of the distribution of *E. crescens* by the excrements of mice completes the present concept of the circulation of *E. crescens* in the field (Dvořák et al. 1966). It may influence the origin of natural foci of adiaspiromycosis, the existence of which has first been pointed out by Rosický et al. (1967). Hejtmánek and Herodek (1958) confirmed in experiments that this mode of distribution of infection was possible with the causative agents of dermatophytoses (genera *Microsporum* and *Trichophyton*), candidiasis (*Candida albicans*), hyphomycoses (genus *Aspergillus*) and phycomycoses (genus *Rhizopus*). Its importance in the epidemiology of several mycotic infections may be greater than anticipated.

МОЖЕТ *EMMONSIA CRESCENS* EMMONS ET JELLISON 1960 — ВОЗБУДИТЕЛЬ АДИАСПИРОМИКОЗА, РАСПРОСТРАНЯТЬСЯ ЧЕРЕЗ ЭКСКРЕМЕНТЫ МЫШЕЙ?

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Резюме. Неоднократные опыты показали, что конидиальная стадия возбудителя адиаспиромикоза может распространяться через экскременты мышей. Конидии и возможно также фрагменты мицелия грибка *Emmonsia crescens* остаются жизнеспособными во время прохода через пищеварительный тракт мышей. Они способны расти, размножаться и вызывать заражение животных. Автором указано значение такого обнаружения для циркуляции *E. crescens* в природе и для возникновения природного очага адиаспиромикоза.

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R. S. Shults, E. V. Gvozdev: Osnovy obshchey gelmintologii (Fundaments of general helminthology), volume II, The Biology of Helminths. *Publishing House NAUKA, Moscow 1972, 515 pp., 170 figs.*

The first, successful, volume of the series "Fundaments of general helminthology", published in 1970, is followed by volume two "The Biology of Helminths". In this new book, the authors present a complete survey of concepts and studies on life cycles, with characteristics of the processes involved, and of the physiology of helminths.

The introductory chapters deal with the system of the supertypo Scolecida enabling the definition of helminths as scolecida living as parasites during a certain phase of their life. The twofold external environment of helminths, i.e., the system: parasite-host-environment, is discussed on p. 8—22. This phenomenon involves problems of the endo- and exoecology of helminths. The chapter dedicated to host categories and to studies on the ontogenesis of helminth is of utmost importance. It is logically followed by an evolution of epidemiological aspects. In the chapter on host categories arranged in accord with the degree of natural susceptibility of the hosts to helminths, the authors have demonstrated, on a number of examples host-parasite relationships and possibilities of their evolution in connection with the factors determining the organism to be utilized as the host of the parasite, and the phenomenon of the exchange of hosts. The general lay-out of these introductory chapters offers a good orientation throughout the problems solved in this book.

The next part deals with problems of general biology (p. 23—182). The individual chapters discuss the various forms of helminths in relation to the external environment. An evaluation is given of the degree of bonds between the helminth and its environment (ecto- and endohelminths), the possibilities of the release of the helminth or its developmental stages into the external environment, their movements (migration), the routes and mechanisms of entering their hosts (from a general point of view). Attention has been given to the migration of endohelminths in the organism of vertebrates (cavity- and tissue migration), and to the various forms of tissue migration. In addition, brief characteristics are given of the migration of monogeneans and that of helminths in the organism of invertebrates. In conclusion, a comprehensive survey is given of the phenomenon of migration of helminths in the organism of their hosts. The following chapter contains data on the location of the helminths in vertebrates and invertebrates, on the duration of development and longevity of the helminths, on fertilization and fertility. A separate chapter is dedicated to variability and to morphological anomalies in helminths, which are important for a better understanding of several taxonomic problems. Cases of hyperparasitism are illustrated in a number of examples, and brief reference is made to palaeohelminthic findings.