

Mutations blocking development of perithecium

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Recommended Citation

Ho, C. C. (1972) "Mutations blocking development of perithecium," *Fungal Genetics Reports*: Vol. 19, Article 3. <https://doi.org/10.4148/1941-4765.1861>

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Abstract

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Ho C.C. **Mutations** blocking development of the **protoperithecium** in **Neurospora**.

The development of the protoperithecium or the female **sexual organ** in **Neurospora**, though essential for sexual development, is nevertheless **dispensable** for completion of the life cycle, due to alternative **vegetative** reproduction by **conidia and** vegetative hyphae. **Mutants defective** in the

formation of protoperithecio are therefore **valuable non-lethal developmental mutants** in efforts to discover those genes that are responsible for the initiation of a **developmental pathway**. For genetical studies, these female-sterile mutants can be used or male parents to fertilize the **protoperithecia** from **strains** of opposite **mating** type.

Three **classes** of **mutations** blocking the different **stages** of the development of protoperithecio have **now** been obtained. All of them are **spontaneous mutations**. In most cases (except **ty-1** and **ty-2**) only mutants with **normal vegetative** morphology and good growth rate were chosen, so that it can be certain that the mutation specifically **affects** the development of **protoperithecia**. It is **already** known that several **morphological** mutants, such as the modifiers of the **colonial** temperature-sensitive mutant (Terenzi and Reissig 1967 Genetics 56:321), have defective protoperithecio and are **female** sterile.

The mutations of the first group (**ff-1**, **ff-2** and others) specifically prevent the formation of **protoperithecia** and have **no** effect on vegetative morphology or **nutritional requirements**. The **ff-1** mutation was mapped on the right **arm** of **linkage group II** between **org-5** and **try-3** (Tan and Ho 1970 Molec. Gen. Genet. 107:158). Another protoperithecio-less mutant also maps on linkage group **II**, but its precise location is not known. The location of **ff-2** is **uncertain**.

The second **class** of **mutants** (**ty-1**, **ty-2**) was first discovered by Westergaard, and the regulation of their **tyrosinase** synthesis was studied intensively (Horowitz et al. 1960 J. Mol. Biol. 2: 96). They form a few small **protoperithecia**, which are **generally** defective in function. **Rarely**, a few of their protoperithecio can be **mated** to form perithecia. The mutant **ty-1** has an **abnormal vegetative** morphology called "velvet", in that the **aerial** hyphae are short and **bear** few conidia. Velvet is **inseparable** from female sterility. Most **ascospores** of **ty-1** are **also probably lethal**, as indicated by a large deficiency of **ty-1** in the progeny of **all** crosses as determined by **random ascospore analysis**. The aerial hyphae of **ty-2** are **also** shorter than those of the wild type. The mutant **ty-1** was **tentatively** mapped by Walker (1963 Neurospora News 1.3: 15) near **tyrosine-1** on the far right end of **linkage group III**. The present work confirms his result. The gene is located to the right of **albino-2** on the right **arm** of **linkage group 1**. It is not **allelic** to the **T** locus (Horowitz and Fling 1956 Proc. Natl Acad. Sci. U. S. 42:498), the structural gene of **tyrosinase**, which is proximal to **al-2**.

The **last class of mutants (ff-6) produces** many **large** and black **protoperithecia** which **cannot** be mated **to form perithecia**. They **also** excrete large **amounts of black** pigments, **presumably** melanin, into the medium. This excretion of pigments may **not** be the cause of **the** functional defect, for there **are** similar **excretor** mutants which **are** female fertile. The gene **ff-6** is located close to **ty-1**.

The **protoperithecia-less mutants (ff-1, ff-2)** are strong candidates for the regulatory gene or genes that switch on the development of **protoperithecia**. If this is **true**, it is **expected** that these mutants may **show a** deficiency of the **various** enzymes and proteins involved in the development of this **organelle**. The **nature of the ff-6** mutation is unknown. ■ ■ ■ Division of Genetics, School of Biological Sciences, University of Malaya, Kuala Lumpur, Malaysia.