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A DOUBLE TELO TRISOMIC FOR THE SEVENTH CHROMOSOME IN PEARLMILLET

IN the progeny of a self pollinated Semidwarf trisomic of pearlmillet[1], which is trisomic for the 3rd chromosome, one out of 29 plants had a chromosome constitution of $2n = 13$ normal *plus* two small chromosomes. The small chromosomes were clearly unequal in length and at pachytene were identified as telocentrics. The short and the long telos corresponded to the short and long arms of the 7th chromosome of pearlmillet, which is the shortest and possesses a satellite on the short arm[2]. Only one such normal nucleolus organising chromosome was observed in the present material. This chromosome and the short telo were almost always directly associated with the nucleolus in PMC's at prophase I, but not the long telo. A plant of such a chromosome constitution is called a double telo trisomic according to the nomenclature of Kimber and Sears[3].

In pearlmillet, the normal 7th chromosome pair occurs usually as a rod bivalent at meiosis but also rarely as a ring bivalent or two univalents. In the present plant in PMC's at prophase I and metaphase I no pairing was observed between the telos. However, in 68% of the 25 cells examined at metaphase I, the long telo paired with the normal chromosome to form a heteromorphic bivalent. In 4% of the cells, a short telo paired with the normal chromosome to form a heteromorphic bivalent. In 16% of the cells the two telos were paired one on either end of the normal chromosomes to form a heteromorphic chain trivalent and in 12% of the cells the three chromosomes remained as univalents.

In an earlier report on double telo trisomics for a different chromosome of pearlmillet[4] centromere misdivision of one submetacentric chromosome was considered to have given rise to two telocentrics. A similar mechanism is possible in the present case involving a 7th chromosome univalent in the trisomic parent.

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TOP NECROSIS VIRUS DISEASE OF PEA FROM INDIA—A NEW RECORD

THE survey of local fields of Agra during 1975–77 revealed an important necrosis virus disease of pea (*Pisum sativum* L.).

The infected plants exhibited withering of the top (top necrosis) and greyish-brown discolouration of leaves, petioles and stems. Internally the host tissues were highly necrosed. Severely infected pea plants die premature death, whereas plants with mild infection develop malformed pods with wrinkled or even abortive seeds. Under glass house conditions ($25° \pm 3°$ C), typical systemic symptoms of the disease were reproduced on the healthy pea plants but only with occasional top necrosis.

The traditional methods of graft (wedge and cleft grafts) and sap inoculations (using phosphate buffer of pH 7·0 and carborundum powder) gave 100% transmission. The virus was also found to be transmitted through seeds (upto 13·6%) and by nematode vector (*Trychodorus* sp.).

The physical properties were studied at room temperature ($25° \pm 3°$ C) using *Chenopodium ameranticolor* Coste and Regn as local lesion host. Its dilution end point lies near 1:10,000, the thermal death point at 64° C and longevity *in vitro* and *in vivo* for 5 and 15 days respectively.

The present virus disease is a new record from India as it has been found to differ from all other virus diseases reported on peas[1–3].

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