Defective I elements introduced into Drosophila as transgenes can regulate reactivity and prevent I-R hybrid dysgenesis

Received: 24 October 1994 / Accepted: 23 March 1995

Abstract The I-R hybrid dysgenesis syndrome is characterized by a high level of sterility and I element transposition, occurring in the female offspring of crosses between males of inducer (I) strains, which contain full-length transposable I elements, and females of reactive (R) strains, devoid of functional I elements. The intensity of the syndrome in the dysgenic cross is essentially dependent on the reactivity level of the R females, which is ultimately controlled by still unresolved polygenic chromosomal determinants. In the work reported here, we have introduced a transposition-defective I element with a 2.6 kb deletion within its second open reading frame into a highly reactive R strain, by P-mediated transgenesis. We demonstrate that this defective I element gradually alters the level of reactivity in the three independent transgenic lines that were obtained, over several generations. After > 15 generations, the transgenic Drosophila show strongly reduced reactivity, and finally become refractory to hybrid dysgenesis, without, however, acquiring the inducer phenotype. Induction of a low reactivity level is reversible – reactivity again increases upon transgene removal – and is maternally inherited, as observed for the control of reactivity in natural R strains. These results demonstrate that defective I elements introduced as single-copy transgenes can act as regulators of reactivity, and suggest that some of the ancestral defective pericentromeric I elements that can be found in all reactive strains could be the molecular determinants of reactivity.

Key words Hybrid dysgenesis • Reactivity • I element • LINE • Drosophila

Introduction

I-R hybrid dysgenesis in Drosophila melanogaster is observed when inducer males that contain functional transposable I elements are crossed with reactive females devoid of such elements (Picard and L'Héritier 1971; reviewed in Bregliano et al. 1980; Bregliano and Kidwell 1983). The resulting female offspring – called SF females (for stérilité femelle) – show reduced fertility, and their germline manifests high frequency I element transposition, high mutation rate, and chromosome non-disjunction (Picard et al. 1978; Picard 1978c; reviewed in Finnegan 1989; Bucheton 1990). It has been demonstrated that the intensity of the hybrid dysgenesis syndrome is essentially dependent on the reactive strain used in the dysgenic crosses; actually, reactive Drosophila strains fall into categories from strongly-reactive to weak-reactive, with respect to the level of SF sterility they produce in a dysgenic cross, ranging from complete sterility to nearly normal fertility, respectively (Bucheton et al. 1976; Picard 1978c). Back-crossing of females from strong-reactive strains with males from a weak-reactive strain showed that the level of reactivity is a peculiar cellular state transmitted mainly maternally from one generation to the next, which is ultimately determined by the nuclear genome (Bucheton and Picard 1978; Bucheton and Bregliano 1982); stabilization of the reactivity level takes about ten generations, and detailed analysis showed that each of the three major chromosomes is involved in the determination of reactivity levels (Bucheton and Picard 1978; reviewed in Finnegan 1989; Bucheton 1990). Reactivity is also influenced by physiological and environmental factors such as ageing and temperature (Bucheton 1978; 1979a, b). The level of reactivity of females decreases with age or when they are raised at high temperature. As the reactivity level is maternally transmitted, it is possible to shift a strong-reactive stock to a low reactivity level by producing each...
generation from old or heat-treated females (see also Fig. 1). However, this process is always reversible: reactivity returns to its initial level, if for about five generations, only young females raised at low temperature are used.

An important question, then, concerns the molecular basis of the control of reactivity in Drosophila strains. One clue is provided by the observation that contamination of a highly reactive strain by complete I factors, which takes place either after a dysgenic cross or by I transgenesis (Picard 1976; Pritchard et al. 1988), results in the generation of an inducer strain (Picard 1978a; Pelisson and Bregliano 1987); such a strain can be considered, formally, as having very low reactivity, since crosses between inducer males and females of an inducer strain have been demonstrated to be non-dysgenic (the female offspring is fertile). To determine whether these observations are actually relevant to the biology of reactivity control, we introduced into a highly reactive strain an I element rendered defective for transposition, thus creating a stable single-copy transgene. Under these conditions we could generate transgenic lines that were not inducer strains, but which manifested a very low reactivity level. Induction of low reactivity takes several generations, is maternally transmitted and is reversible upon transgene removal by chromosome substitution. These results demonstrate that a defective I element introduced as a single copy transgene into Drosophila regulates reactivity, with characteristic features – maternal inheritance, latency, reversibility – as in natural R strains; this might suggest that ancestral defective I elements, which can be found in the pericentromeric domains of all chromosomes of reactive and inducer strains (Bucheton et al. 1984; Crozatier et al. 1988; Vaury et al. 1990), could be the natural determinants of reactivity (see Crozatier et al. 1988; Chaboissier et al. 1990; Discussion).