



Fig. 4. A scheme for chromosomal alterations in *Myrmecia (pilosula)* $n=1$. Solid circles at the primary constrictions represent active NOR sites. Arrowheads indicate loci where chromosomal mutations were assumed. The A_1^M chromosome is a hypothetical acrocentric chromosome with an extraordinarily elongated heterochromatic short arm (=pseudo-acrocentric). For details see text

next alteration ($A_2' \rightarrow M_2$) seems to be somewhat unusual. In A_2' and M_2 the size of the heterochromatin and the location of the chromosomal gap are exactly the same (Figs. 1 and 2). The only difference between them is the location of the centromere and NOR (Fig. 3). The topological pattern of the chromosomal gap precludes the chromosomal alteration from A_2' to M_2 or the reverse by a simple pericentric inversion. The only possible solution seems to be a centromere shift from the terminal of A_2' to the euchromatin-heterochromatin junction of M_2 . If we accept this assumption, we have to assume a shift of the active NOR site also, because there is no active NOR site in the subterminal region of the heterochromatic short arm of M_2 which corresponds to the primary constriction of A_2 (compare Fig. 3h and i).

The third event is concerned with the origin of $M_{(1+2)}$. Based on the C-banding patterns of $2K_4$ ($=1M_{(1+2)} + 1ST_1 + 1A_2'$) (Fig. 2i), we concluded that $M_{(1+2)}$ was induced by the so-called telomere fusion which occurred at the short arm terminals of ST_1 and A_2' (see Fig. 4, arrowheads). The centromere and NOR of $M_{(1+2)}$ obviously originated from those of ST_1 (compare Fig. 3e and f). If this interpretation is correct, we have to assume inactivation of the centromere and NOR in the short arm of $M_{(1+2)}$, which is homologous with A_2 or A_2' (Figs. 2i, 3e, h and 4).

Many cases of centromeric inactivation following telomere fusion have been reported recently in mammals (for details see Imai 1988; Imai et al. 1988b). We have found the same phenomenon in the ant *Ponera scabra*, and also centromeric 'reactivation' in *Myrmecia (piriventris)* H185-

302 (Imai et al. 1988a). These observations suggest that the inactivation of the centromere and NOR found in $M_{(1+2)}$ of *M. (pilosula)* $n=1$ is not an unacceptable assumption. The M_2 induction by the centromere and NOR shift mentioned above may be another example of inactivation and reactivation of the centromere and NOR, although we need further supporting evidence at the molecular level.

So far as our observations are concerned, the most simple but reasonable interpretation for the karyotypic alterations in *M. (pilosula)* $n=1$ may be that its ancestral karyotype was $2K_0 = 2A_1^M + 2A_2$ (i.e., $2n=4$), and the lowest karyotype, $2K_1 = 2M_{(1+2)}$ having $2n=2$, was derived secondarily from a hypothetical karyotype $2K' = 2ST_1 + 2A_2'$ by telomere fusion between ST_1 and A_2 , as summarized in Figure 4.

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