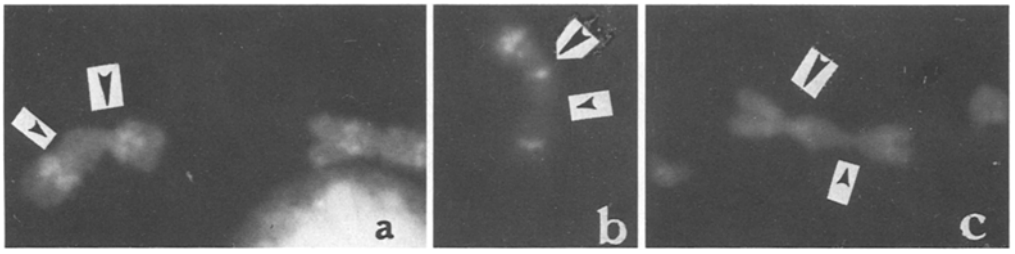

Figure 2. A panel of three dicentrics showing differential replication of pericentric heterochromatin associated with the two centromeres. The active centromeres are marked by arrows. (*a*) shows a dicentric in which one centromere is separated while the other is tightly holding the two sister chromatids. The prematurely separated centromere had completed replication of heterochromatin before BRdU was added to the culture. The segment showing BRdU incorporation below this centromere is late replicating euchromatin. The centromere that is not divided shows replication in the centromeric region as well as pericentric heterochromatin. (*b*) represents a dicentric showing replication of the region around the active centromere. The inactive centromere shows only a sign of slackening and there is no evidence of replication of either the centromeric region or the surrounding pericentric heterochromatin. Notice, however, a euchromatic block in the middle of the bottom third of this chromosome which replicates after the heterochromatin associated with the prematurely separating centromere has replicated. This pattern of replication was used to identify the position of the prematurely separating centromere in situations where neither of the two centromeres had separated (*c*).

tightness with which the two centromeres hold the chromatids together. Another 12 chromosomes showed localization of the label in only the central region of the late separating centromere. However, the mere dot-like location and weak fluorescence of the label did not appear on the photographs.

These results suggest that in mouse L-cells (i) all heterochromatin does not replicate late, (ii) there are regions of euchromatin which replicate later than certain regions of pericentric heterochromatin or regions of repetitive DNA (it is not known if the late replicating, apparently euchromatic, regions carry repetitive DNA in this cell line. It is unlikely though, that large blocks of euchromatin-like regions as seen in figure 2a would be entirely composed of repetitive DNA and yet show no C-bands), (iii) the timing of replication of heterochromatin blocks of more or less similar composition, as found in mouse, may possibly depend upon the location of the heterochromatin, and (iv) the prematurely separating centromere in the dicentric under discussion replicates its associated pericentric heterochromatin consistently earlier than that associated with the active centromere. For the present study it is immaterial whether or not the two blocks of heterochromatin have similar composition. It is entirely possible that the timing of centromere separation and the sequential separation observed in various genomes[6-8] is a reflection of the timing of replication of the pericentric heterochromatin, i.e., the sooner the pericentric heterochromatin completes its replication, the sooner a particular chromosome separates at its centromere. Thus, the timing of centromere separation may reflect the replication of this region and associated DNA sequences followed by its 'maturation' and resulting separation into daughter units. The heterochromatin of prematurely separating centromere apparently causes maturation of its centromere ahead of the maturation of equivalent region of the active centromere; hence, the former separates before the latter. A consistently sequential pattern of centromere separation in multicentric chromosomes may even suggest, a previously postulated but unpopular belief[10], that there exists some sort of interaction (or communication) between various centromeres in a given chromosome. This may result in the hierarchy leading to premature separation in dicentrics.

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Genotypic differences between the mouse adenovirus strains FL and K87[1]

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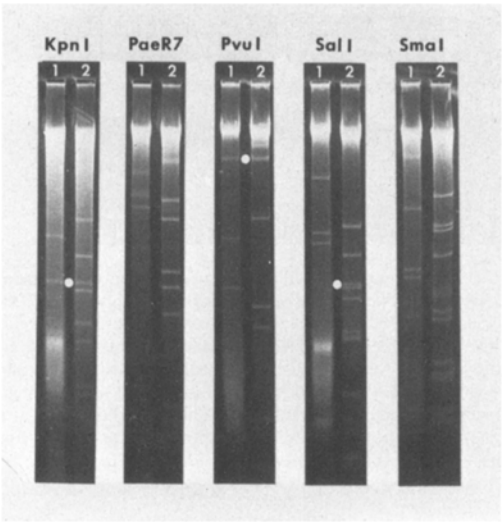
Summary. Restriction endonuclease analysis was used to compare the genome of mouse adenovirus (MAd) strains FL and K87. Large differences were found between the *Kpn* I, *Pae*R7, *Pvu* I, *Sal* I and *Sma* I restriction profiles of the prototype strains. MAd-FL and MAd-K87 thus represent two distinct species of mouse adenovirus.
Key words. Adenovirus; DNA; mouse; restriction endonuclease; virus disease.



Electrophoretic patterns of (1) MAd-FL and (2) MAd-K87 DNAs after cleavage with five restriction endonucleases. Electrophoresis of DNA fragments was carried out in a 1 % agarose slab gel at 50 V for 16 h. DNA in the gel was stained with ethidium bromide and photographed under ultraviolet light illumination. Comigrating MAd DNA fragments are indicated by white dots.

Only one species of murine adenovirus (MAd) is until now recognized in the literature[2, 3]. MAd prototype strains FL and K87 differ, however, in pathogenicity and tissue tropism[4–7]. Serological tests also indicate that the two viruses are antigenically distinct[8–10]. Under these circumstances, it seemed interesting to use restriction endonuclease analysis of viral DNA as a means to further differentiate the MAd strains.

The FL and K87 viruses were propagated in primary mouse kidney cells and embryonic mouse cells, respectively, as described earlier[9]. Viral DNA was extracted from MAd-infected cells by the SDS-pronase-phenol method developed earlier for canine adenoviruses[11]. Aliquots containing about 0.5 µg of MAd-FL or MAd-K87 DNA were digested for 2 h at 37 °C under appropriate buffer conditions with restriction enzymes (5–10 units) either prepared in our laboratory (*Pae*R7) or purchased from Bethesda Research Laboratories (*Kpn* I, *Pvu* I, *Sal* I and *Sma* I). Digestion products were resolved by electrophoresis for 16 h at 50 V in a 1 % agarose gel as described previously[12]. DNA in the gel was stained with ethidium bromide (1 µg/ml) and photographed under UV light illumination.

According to the restriction profiles shown in the figure, the FL and K87 strains represent two distinct species of MAd. The number of *Kpn* I, *Pae* R7, *Pvu* I, *Sal* I and *Sma* I restriction fragments is, effectively, different for the two viruses. MAd-FL and MAd-K87 DNA fragments rarely comigrate in the gel (white dots). Hexanucleotide sequences recognized by the five restriction endonucleases used in this work are thus located at very distinct positions on each type of MAd DNA molecules.

Together with biological and immunological differences already found[4–10], our results confirm the existence of at least two distinct adenovirus species in the mouse. The size of each restriction fragment in the gel has now been evaluated using the 1 kilobase ladder from Bethesda Research Laboratories as a molecular weight marker. Results indicate that the MAd-FL and MAd-K87 genomes comprise 32 and 31 kilobase pairs, respectively. Similar values were published earlier by Larsen et al.[13, 14] for MAd-FL DNA.

The usefulness of restriction endonuclease analysis in the identification and classification of DNA viruses is also confirmed. This sensitive and specific technique was recently used in our laboratory to distinguish between canine adenovirus types 1 and 2[15, 16]. With the present results, we can only support the concept of genome typing for species distinction. As suggested earlier by Whetstone[17], the definition of an adenovirus species should include both serotype and genotype. In this perspective, the discovery of new adenovirus species in rodents is more than probable.

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Karyotypic differentiation in an agamid lizard, *Japalura swinhonis swinhonis*[1]

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Summary. Three populations of a Taiwanese agamid lizard *Japalura swinhonis swinhonis* exhibit karyotypes distinct from each other. Lizards from the northern area have $2n = 46$ all acrocentric chromosomes, whereas animals from two localities in the central region possess $2n = 36$ and $2n = 40$ chromosomes, respectively, including several biarmed elements.
Key words. Japalura s. swinhonis; Reptilia; Agamidae; karyotype.