weak histocompatibility difference between the two strains.

It was apparent that the tumors were due to something common to these inoculums, presumably mammary tumor virus which is usually present in the erythrocytes of mice of both sexes that harbor the agent (*9*). Many erythrocytes are present in any spleen or liver preparation, and it appeared likely that mammary tumor virus was carried by the DBA/2J male cell donors. Male DBA/2 mice may harbor mammary tumor virus (*10*), as indicated by studies at the Jackson Laboratory.

In conclusion, graft-versus-host disease that results from the inoculation of parental spleen cells differing at more than one histocompatibility locus failed to induce a significant number of malignant lymphomas in the recipient mice. Neither were tumors observed in the mice inoculated with Rous sarcoma virus, possibly because partially purified preparations were used rather than crude extracts (*11*). This suggests that factors other than GVHD itself (such as mammary tumor virus in the present study) may have been involved in those experiments in which malignant lymphomas occurred (*3, 4*). The relation of immunologic phenomena to neoplastic proliferation remains to be clarified.

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Mosaic Ruler

Mills's idea (*1*) for a "more economical" version of my (*2*) hypothetical mosaic unit ruler has come to my attention. He qualifies his suggestion by either omitting unit 21.6 cm, or incorporating it differently from the other units. However, I find so many (Fig. 1) classical floor-mosaic patterns this size compared with the other mosaic unit sizes that I regard it probable that, at least from the mosaicists' point of view (*3*), unit 21.6 cm was as basic as the others, and I would expect it to appear like the others on their rulers.

However, while Mill's ruler is simpler in the sense of having fewer calibrations, having made one, I find it much trickier to use than mine which is simply marked with each unit in turn from a zero at one end. The latter arrangement happens to coincide with that usually found on other ancient rulers.

This problem may come to be resolved, for, following Ledin's comment (*4*), a picture (*5*) has come to light leading to the possibility (*6*) that an original mosaicist's ruler may be contained in a burial in the Catacombs of Priscilla at Rome.

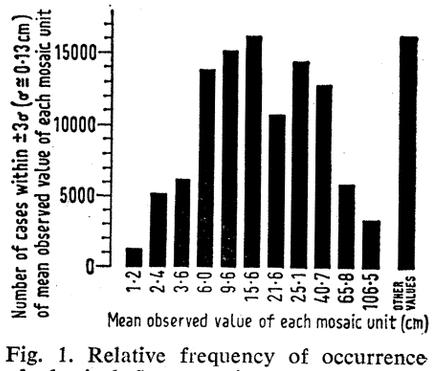

Fig. 1. Relative frequency of occurrence of classical floor-mosaic pattern sizes in sample of 121,265 observations.

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Dorsal Root Potentials Produced by Stimulation of Fine Afferents

Concerning the reports (*1–3*) that volleys in afferent unmyelinated fibers produce a negative dorsal root potential (DRP) in contrast to an earlier finding (*4*) such that impulses in fine afferents were said to produce a positive DRP, Zimmerman (*1*) says that his finding abolishes "one of the basic postulates of a recent pain theory" (*5*) and Vyklicky *et al.* (*3*) state that their results deny "a basic tenet" of the theory. The paper to which they refer proposed no more than that the input-output relations of hypothetical dorsal horn cells were modulated by what was termed a "gate control mechanism." Impulses arriving in certain fine afferent fibers tended to open the gate by facilitation, while certain large fibers closed it by inhibition. A possible presynaptic mechanism was discussed, but there was doubt as to whether the mechanism of the modulation was presynaptic, postsynaptic, or both. To emphasize this uncertainty, the diagram of the gate control mechanism showed a box around both pre- and postsynaptic structures. The location of the facilitating mechanism was never a "basic postulate," let alone a "tenet." The theory does require that some modulating mechanism should exist but does not specify its location. Evidence continues to accumulate that a modulating mechanism does exist. For example, lamina 5 cells and flexor motoneurons are facilitated by some fine afferents and inhibited by some large afferents (*2, 6*). Irrespective of the sign of DRP's, we have still to face the exist-