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.

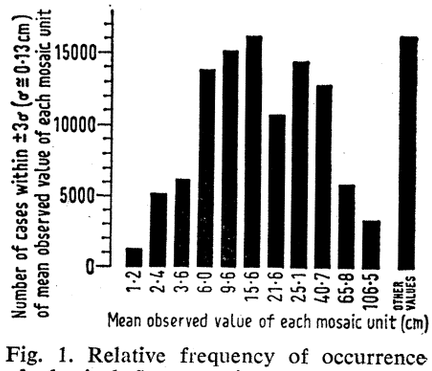

Fig. 1. Relative frequency of occurrence of classical floor-mosaic pattern sizes in sample of 121,265 observations.

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1 December 1969

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-

ence of these modulations and to explain their mechanisms.

To our surprise Zimmerman (*1*) has intentionally arranged the blocking electrodes with the cathode closest to the cord. In the study by Mendell and Wall (*4*), the anode was proximal to the cathode since it was known that impulses would be generated at the cathode and would bombard the cord unless they were blocked by a more proximal anode. In order to test the effects of Zimmerman's technique, we duplicated his experiment, using the same type of blocking equipment and electrode arrangement (Fig. 1). As the blocking current increased to the level used by Zimmerman, an afferent barrage was generated and continued throughout the plateau phase of the block, during which he had delivered this test afferent volley. Examination of his published result shows that the same effect was occurring in his experiment. The base line activity is increased in line *i* of his figure 1, although the effect is not so obvious as in our Fig. 1 because his amplification was less than ours and because the shape of his records shows that high-frequency responses were partially abolished by filtering. In our experiments, the height of the individual spikes in the afferent barrage generated by the blocking electrodes was small and therefore one must assume that certain fine afferents were active. Zimmerman's test volley evidently arrived at a spinal cord already under this steady bombardment. His failure to record positive DRP's may have been a consequence of an inhibition or saturation of central mechanisms produced by the ongoing barrage generated by his blocking electrodes.

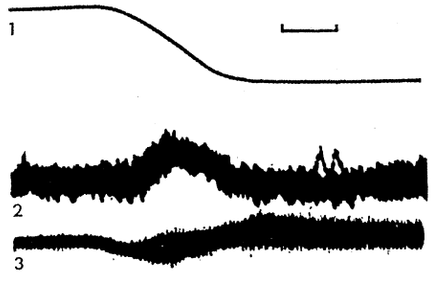

Fig. 1. Impulses generated by Zimmerman's blocking method (*1*). The cat was spinalized at Cl, and was unanesthetized. The sural nerve was mounted on a pair of blocking electrodes with the cathode closest to the spinal cord. Line 1 shows the time course of the onset of the blocking current which rose over 1.2 seconds from zero to a plateau level of 25 μa. Time mark, 1 second. Line 2 shows complex dorsal root potential evoked by the onset of the blocking current. DRP was recorded from a rootlet of L7 dorsal root. Line 3 recorded from a pair of electrodes on the sural nerve proximal to the blocking electrodes. Filters were set to record from 6 hz to 60 khz. The record shows that impulses were traveling in the nerve toward the spinal cord when so-called blocking currents were applied.

When we repeated the original experiments (*4*), using Zimmerman's technique but with the anode proximal, it was evident that impulses in fine fibers did generate positive DRP's (Fig. 2). The recordings were made from cats with the cord sectioned at C1 as described (*4*). The blocking current was produced by a device which generated a linearly rising current followed by a constant current plateau. The rising phase lasted 1.2 seconds, and the transitions were smoothed over 200 msec (Fig. 1, line 1). Stimuli to the sural nerve were delivered at 5-second intervals and the resulting DRP's were averaged by a Linc 8 computer. The results show that a positive DRP begins after 40 to 50 msec when a blocking current prevents the arrival of most of the high-velocity impulses in A beta fibers. This relatively short latency means that fine myelinated afferents (A delta) must play a role in generating the positive DRP, since no impulses in unmyelinated afferents could have arrived at the cord in so short a time. This was recognized even in the title of our paper under attack (*4*).

We now know from Casey and Blick (*7*) that the technique of anodal polarization fails to block all the A delta afferents even when the A delta component of the compound action potential has disappeared. Therefore, we can no longer conclude that Mendell and Wall (*4*) ever generated volleys limited to unmyelinated fibers. We can conclude however that predominantly positive DRP's are recorded after the arrival of a mixed volley of impulses in predominantly fine fibers, that is, A delta and C fibers.

It has been known for some time that the tendency to generate positive dorsal root potentials depends on the excitatory state of the spinal cord. Lloyd (*8*) showed that anesthesia prolonged the negative component of the DRP and abolished the positive. Mendell and Wall (*4*) showed that repetitive stimulation of afferents of large diameter reduced negative and increased positive DRP's. The same effect is shown in Fig. 2 where the small initial negative DRP is eliminated by repetitive stimulation of a nearby dorsal rootlet with a stimulus strength adequate for only the largest fibers. The duration of the negative DRP produced by maximal peripheral stimulation is an index of the tendency to generate positive DRP's. Lloyd's negative DRP's lasted 75 msec (*8*). We have never seen

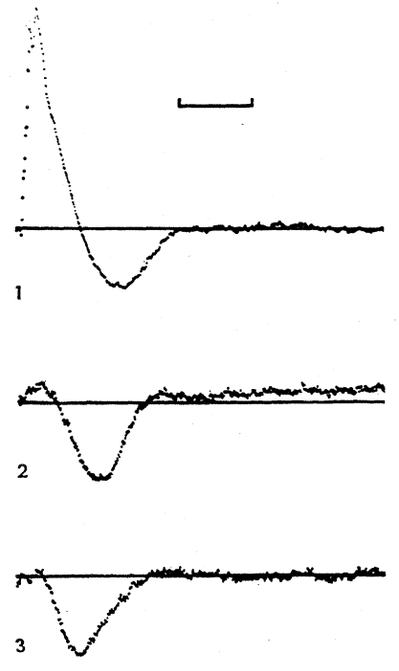

Fig. 2. Dorsal root potentials generated by sural nerve stimulation under three conditions. The cat was spinalized at C1; it was unanesthetized, and all peripheral nerves were intact. Stimulus to sural nerve every 5 seconds. Recording of DRP on L7 dorsal rootlet. The average of eight responses are displayed. Time line, 100 msec. (Line 1) DRP produced by maximal stimulation of sural nerve. The afferent volley monitored on the sural nerve showed a compound action potential containing A beta, A delta, and C waves. (Line 2) DRP produced by the same stimulus to sural nerve as in line 1 but with potential blocking of the afferent volley. The blocking electrodes were arranged with the anode closest to the cord and both blocking electrodes were proximal to the two stimulating electrodes. The test stimulus was delivered at 200 msec after the blocking current had reached its plateau level of 25 μa. The A beta component of the compound action potential in the monitored afferent volley was no longer visible, but since there is a small early negative DRP, some impulses in A beta fibers must have escaped the block (*4*). Large volleys traveling at A delta and C velocities were still recorded. (Line 3) Sural test stimulation and block were the same as in line 2. A rootlet from rostral L7 dorsal root was continuously stimulated at 100/sec, 0.01 msec, 0.25 volt. This conditioning stimulus generated a small steady negative DRP and eliminated the phasic negative DRP which followed the sural test shock.

sural nerve stimulation produce a negative phase lasting longer than 80 msec in an adequately circulated unanesthetized spinal cord. In Zimmerman's experiment (*1*), the cord must have been severely depressed since the negative DRP lasts 135 msec. In the experiments of Franz and Iggo (*2*), all peripheral nerves had been sectioned, a procedure which will reduce central activity by abolishing the ongoing afferent barrage, and the negative DRP lasted 130 msec. In the experiment of Vyklicky *et al.* (*3*), the cord was sectioned at L1, and all but one afferent nerve from the leg had been cut; both these operations reduce the amount of central activity. We conclude that the results described (*1–3*) were from relatively inactive cords under conditions known to exaggerate the negative and eliminate the positive DRP's.

However, under the conditions of the experiments (*1–3*), certain unmyelinated fibers were shown to produce negative DRP's. Their stimuli or blocking did not allow a test of whether other fine fibers would have produced positive DRP's under those conditions. In our experiments where mixed volleys in fine fibers produced predominantly positive DRP's, we observed intermittent long latency components in the positive wave which, had they been observed in isolation, would have appeared similar to the C DRP of Franz and Iggo (*2*). An example of this negative trend during a positive DRP was illustrated by Mendell and Wall (*4*) in Fig. 4. It therefore seems likely that both negative and positive DRP's can be generated by volleys in fine afferents, depending for their relative sizes on the state of the preparation and on which specific group of afferents are stimulated.

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27 October 1969

Calcium and Salt Tolerance of Plants

LaHaye and Epstein (*1*) have reported that calcium increases the salt (sodium) tolerance of bean plants and imply that this effect has not been appreciated. However, a distinction needs to be made between the tolerance of plants to soil salinity and exchangeable sodium.

Since sodium salts affect soils in special ways, saline and sodic soils have been carefully distinguished (*2*). By definition, saline soils contain enough calcium to meet the ordinary nutritional requirements of plants. In studying the salt tolerance of plants, therefore, calcium is always present at a concentration of at least a few milliequivalents per liter in the root medium (*3*), and the reaction of plants to salinity does not involve calcium deficiency effects except as they may be induced by high concentrations of other salts (*4*).

In sodic soils, in which the concentration of exchangeable sodium is more than 15 percent, calcium and magnesium concentrations decrease as sodium increases. In nonsaline, sodic soils, therefore, calcium and magnesium are often deficient for plant growth. This phenomenon is well known, and the tolerance of plants to high concentrations of sodium and low concentrations of calcium and magnesium has been studied (*5*). As has been reported by LaHaye and Epstein (*1*), sodic soil conditions cause an accumulation of sodium in the tops of bean plants (*5*). Furthermore, the absolute concentration of calcium is critical since, with the same proportions of exchangeable cations, the effect of high concentrations of exchangeable sodium is apparent only when the soluble salt content is low and the absolute calcium concentration is therefore low (usually about 1 meq/liter or less) (*4*, *6*).

Thus salt damage does not depend primarily upon a low or deficient calcium level and sodium-calcium relations of sodic soils and plants grown on them have been amply studied.

LaHaye and Epstein's statement that 50 meq of NaCl per liter in the presence of adequate calcium has no effect on the growth of beans is contrary to numerous reported studies in which appreciable reductions in growth and yield have been observed at such salt concentrations (*3*). The discrepancy is probably due to the short period of observation (7 days) by LaHaye and Epstein, to a probably mild set of growing conditions (not specified), and to considering the gross aspects of calcium deficiency rather than the more subtle effects on growth and yield.

The report by LaHaye and Epstein is related to tolerance of plants to sodic soil conditions, not to salinity. The results of LaHaye and Epstein can be attributed to their having followed the erstwhile frequent practice of studying salt uptake by plants from single-salt solutions in short-term experiments and concluding inappropriately that growth responses to salinity also involved single-salt and calcium-deficient solutions.

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3 November 1969; revised 23 December 1969

The report of LaHaye and Epstein struck a responsive note in my memory, and I pulled from my files *Cornell Agricultural Experiment Station Memoir No. 2*, "The action of certain nutrient and non-nutrient bases on plant growth," by M. M. McCool. I quote from this 1913 publication.

"Kearney and Cameron (1902), employing alfalfa and lupine, found that the greatest endurable concentration of sodium chlorid is .02 mol., while in the presence of calcium chlorid the amount of sodium may be raised to .2 mol.

"When *N*/50 NaCl is employed, slight development of tops occurs but there is no root extension. When seedlings are placed in *N*/100 NaCl, the development