ever, the primary disadvantage is that there is no proof that seeing cytogenetic abnormalities is an absolute indication of mutation. One can visualize a spectrum of damage from such severe damage that the cells die without ever getting into mitosis, to other cells that are made incapable of cell division but survive in a post-mitotic state, with or without a change in functional proteins; and finally, gene mutations, in which cells can still go on to divide but have alterations in the functional proteins produced.

Evidence for the first two types of change seems well established, and this is of course important in our consideration of damage to genetic material, and is a very important consideration in teratogenesis and perhaps in aging. The mutations are less easily confirmed, however. If chromosome breakage is important in mutation, it would express the view that the breaks are an indicator system, since most of the cells with visible unstable chromosome abnormalities would probably go on to cell death. Work correlating mutation and chromosome breakage after chemical treatments is in an early stage when compared to the data available for X-rays. However, Kihlman has pointed out that there is good correlation between substances that are capable of producing mutations in various systems and those that can produce chromosome abnormalities (*3*). There is almost 100 percent correlation between chromosome aberrations produced in mammalian cells in tissue culture and mutagenic effects whenever there was data available on both effects (see table 5) (*3*). The correlation between mutagenesis and chromosome aberrations in plant root tips was good, but not as good as for mammalian cells in tissue culture.

The absolute answer to this question of chromosome breaks serving as an indicator for gene mutation will probably come from the studies that are presently starting in somatic cell genetics when these are correlated with, and studied in conjunction with, cytogenetics. The type of work referred to here is the ability of an agent to induce drug resistance in somatic cells which reflects the loss of functional enzyme, as for example resistance to BUdR, because it is no longer incorporated into the cell due to the loss or modification of the thymidine kinase enzyme. This approach is well exemplified in recent studies in which Chinese hamster cells were treated with BUdR and nutritionally deficient mutants were selected by growing cells in restrictive media in which the nutritionally deficient mutants cannot divide, and then adding an agent that will kill dividing cells (*5*). Similarly, selective culture techniques have been used to isolate L glutamine auxotrophs or 8-azaguanine resistant Chinese hamster cells and to compare the incidence of these mutants in cultures treated with chemical mutagens and control cultures (*1*). Human male cells and genes on the X-chromo-