consists of the following: (i) In contrast to PHA, PHA-IM must be added back to cultures after virus inoculation and wash in order to enhance virus replication. This finding makes unlikely, but does not totally exclude, the possibility that small amounts of PHA were present in the PHA-IM preparation and caused virus enhancement when added back. (ii) In order to exclude the possibility that PHA-IM contained PHA, experiments were conducted which indicated that PHA-IM enhanced virus replication but did not agglutinate leukocytes; in a series of dilution experiments we were unable to separate the leukoagglutinating and enhancing properties of PHA. (iii) Experiments in collaboration with T. C. Merigan indicate that PHA-IM at high concentrations reversed the PHA-interferon-induced plaque reduction of VSV in a primary human foreskin fibroblast culture assay system; the mechanism of reversal remains to be determined. PHA alone had no effect on VSV plaque formation in this system.

In the search for the mechanism of PHA-induced enhancement of VSV in human leukocyte cultures, we have focused our attention on the lymphocyte transformed by PHA into blast cells (*11*). This approach is due in part to the repeated observation of a close correlation between blast cell formation and virus enhancement, which to our knowledge is a relationship not satisfactorily demonstrated before. Recent experiments indicate that PHA-IM, as well as PHA, induces blast cell formation in uninfected leukocyte cultures. Morphologic studies of PHA- or PHA-IM–treated cultures have revealed that VSV infection produces a tenfold reduction in the number of blast cells and marked damage to many of the remaining blast cells as compared with uninfected treated cultures. The observation that blast cells undergo cytopathic effects in infected cultures suggests that VSV replicates in this cell type. This conclusion is further supported by our recent finding that cultures of lymphocytes (99 percent homogenous) prepared in siliconized glass bead columns (*12*) are incapable of supporting significant VSV replication, but the addition of PHA induces both blast cells and VSV replication. The cell type supporting VSV replication in buffy coat cultures in the absence of PHA has not yet been determined. Thus it appears that VSV enhancement is intimately tied to blast cell formation and to those biochemical alterations implicit in this cellular transformation.

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Protein Synthesis during First Cleavage of Sea Urchin Embryos

I was not surprised to find contradictory results, published a month apart, concerning the rate of protein synthesis during the first cleavage in the sea urchin embryo. Gross and Fry (*1*) found no decline in the rate of protein synthesis in fertilized eggs of *Strongylocentrotus purpuratus* through the first cycle of cell division. Sofer, George, and Iverson (*2*), working with the fertilized eggs of *Lytechinus variegatus*, found a decline in the rate of protein synthesis as the cells began the first cell division.

For the past 2 years I have been working on this same problem with the eggs of *Lytechinus pictus* and *Strongylocentrotus purpuratus*, and, much to my bewilderment, I have obtained both results (that is, the continuation and the decline of protein synthesis as the eggs entered the first mitotic division). As far as I have been able to determine, the disparity in my own results is not due to (i) species used; (ii) the amino acid used (both Gross and Fry, and Sofer, George, and Iverson used C^{14}-leucine; I have used C^{14}-valine and C^{14}-phenylalanine); or (iii) the time of year or the region in which the eggs were collected.

After 2 years of repeating experiments and wondering how many more times they should be repeated, and refraining from publishing either of these conflicting results, I find that I have been scooped on both counts.

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