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Einar Lundsgaard

Word has come from Copenhagen of the death, during the last days of 1968, of Einar Lundsgaard. In these times of rush and short recall, I am particularly keen to refresh the memory of the scientific community about his truly great discovery which is now slipping into the background. Personally and scientifically, I encountered Lundsgaard during my period of maturation and I owe him much. I was in Meyerhof’s laboratory in Heidelberg when he came in 1930: tall, blond, and very Danish, with his handsome wife, Helle. There we first met and became friends, and later when I moved to Copenhagen in 1932 and stayed until 1939, we saw each other a great deal. In the fall of 1967, his friends and colleagues went to Copenhagen to celebrate the 40th anniversary of the discovery of what
he called the α-lactacid contraction of iodoacetate-poisoned muscle. When this startling news reached us in Meyerhof’s laboratory, it was very upsetting to our group which looked upon glycolytic lactic acid as the link between metabolic energy generation and muscle contraction.

The interest in iodinated organic compounds which might have metabolic actions similar to thyroxin induced Lundsgaard to study iodinated acetic acid. Its injection into animals, however, yielded a rather unexpected effect: for a few minutes the animal behaved quite normally, but suddenly it turned over and its muscles became rigid. Such rigor was dependent on prior muscle activity since denervated or curarized muscles did not respond. But when rigor developed, the expected burst of lactic acid was missing. Lundsgaard elegantly solved the puzzle. He showed that: (i) iodoacetate inhibited glycolysis; and (ii) that poisoned muscle performed a limited number of normal contractions at the expense of dephosphorylation of creatine phosphate, then newly found in muscle and in need of a function. The rigor mortis-like condition developed when the limited supply of creatine phosphate was exhausted.

Lundsgaard had discovered that the muscle machine can be driven by phosphate bond energy, and he shrewdly realized that this type of energy was “nearer,” as he expressed it, to the conversion of metabolic energy into mechanical energy than lactate acid. He was right, because it soon developed that the glycolytic reaction is a feeder of phosphate bond energy and not of acid. On the way, Lundsgaard also provided an enormously useful tool for studying enzyme mechanisms. iodoacetate has become one of the standard reagents for SH-blocking in enzymes. Thus, iodoacetate inhibits glycolysis because it blocks the functional SH in phosphoglyceraldehyde dehydrogenase.

In the middle 30’s, Lundsgaard became professor of physiology at Copenhagen University and trained many biochemists and physicians. Herman Kalekar was one of his graduate students. And, even though I did not formally work with Lundsgaard, I consider myself his pupil. My subsequent work was profoundly influenced by his discoveries which changed our concepts of metabolic energy transformation.

Fritz Lipmann

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18 APRIL 1969

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