in perspective a possible physiological role of somatomedin, it is therefore of interest to consider possible points of similarity and difference between somatomedin and other known growth-promoting factors.

A. Insulin

It has been well established that insulin, both *in vivo* and *in vitro*, has a stimulatory effect on protein synthesis. Insulin added *in vitro* to isolated tissues stimulates the incorporation of labeled amino acids into proteins and increases the proportion of ribosomes in polysomes (Manchester, 1972). As pointed out earlier, insulin in high dosages can stimulate the incorporation of sulfate and thymidine into cartilage (Salmon and DuVall, 1970a; Van Wyk *et al.*, 1972b). Furthermore, high dosages of insulin can partially replace serum in stimulating DNA synthesis in cultures of chicken embryo fibroblasts (Temin, 1967). On the other hand, insulin present in normal serum cannot account for the effect of serum on cartilage or fibroblasts.

The plasma levels of insulin, measured by radioimmunoassay, are high in patients with acromegaly and low in patients with hypopituitarism. It would therefore appear that growth hormone participates in the regulation of insulin secretion (Luft and Cerasi, 1964; Luft *et al.*, 1967, 1969). The mechanism of this action of growth hormone is still unknown. Furthermore, growth hormone *in vivo* stimulates protein and RNA syntheses even in the absence of insulin as shown on isolated liver preparations (Jefferson and Korner, 1967). Thus, insulin itself does not seem to be the hormone mediating the anabolic action of growth hormone.

B. Nonsuppressible Insulinlike Activity (NILA)

Serum contains far more insulinlike activity, measured by bioassay on the epididymal fat pad, than can be accounted for by the level of insulin measured by radioimmunoassay. When assayed by its ability to stimulate glucose oxidation in the fat pad immunoreactive insulin accounts for only 7% of the total insulinlike activity (ILA) in serum (Froesch *et al.*, 1963). The remainder of this biological activity, NILA, has been studied by many workers (Froesch *et al.*, 1963, 1967; Bürgi *et al.*, 1965, 1966; Jacob *et al.*, 1968; Poffenbarger *et al.*, 1968; Oelz *et al.*, 1970a,b; Humbel *et al.*, 1971). Few studies have been directed toward the influence of HGH on ILA and NILA. In patients with acromegaly,