17a and 17b methylene signals with respect to those of 13a and 13b. Such a conjugation reaction is reminiscent of the detoxication processes[10] which most foreign compounds and some natural metabolites, usually unwanted by the organism, undergo in the body to produce more polar and relatively less active products.

Indeed, the occurrence of neobonellin in the body skin more than in the proboscis of female *Bonellia viridis* would suggest a role of this pigment in the storage or scavenger of bonellin, responsible, as reported[3-5], for the inhibition of growth and the development of masculinity of the worm larvae.

While the physiological role of these pigments in *B. viridis* awaits further investigation, we have found that both bonellin and neobonellin have a strong blocking effect on the development of sea urchin embryos.

Tests on fertilized eggs of *Sphaerichinus granularis*, with or without fertilization membrane, provided evidence that aqueous solution of these pigments in their natural forms (R=H)[11] readily inhibit cleavage at any stage of development, and cause a complete cellular lysis within a short time. These effects are qualitatively similar to those described by previous authors[4,5] for crude or partially purified pigment extracts of female *Bonellia viridis*. Interestingly enough, while in dilute solution up to 10^{-6} M bonellin and neobonellin are toxic for the embrionic cells, at lower concentration they produce a different effect resulting in the separation of blastomeres in the first stages of cellular division. Subsequently, the apparently undamaged blastomeres lose the ability for cleavage, although they continue to survive for several hours (figure).

The observed inability of separated embryonic cells to enter in mitosis is of interest in view of the possibility that bonellins can also be active on proliferating cells, such as tumoral tissues. Experiments in this direction are now in progress in our laboratory.

1 A preliminary account of this work was presented at the IX Congress of Italian Society of Marine Biology, Ischia (Italy), May 19-22, 1977.
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11 Notably, both bonellin and neobonellin in the form of the methyl esters show no activity on the development of sea urchin embryos.

Activators of serum lipoprotein lipase in alloxan diabetic rats

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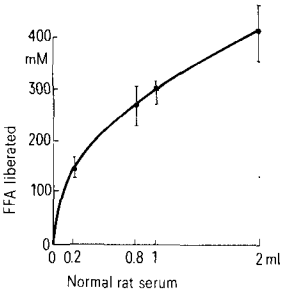
Summary. The amount of free fatty acid (FFA) liberated with intralipid in post-heparin guinea-pig serum, when serum from alloxan diabetic rat is incubated, is higher than that liberated when serum from healthy rats is used. The above effect is probably due to larger quantities of lipoprotein lipase present in the diabetic serum.

Endogenously produced lipids and lipids available in plasma from diet, when transported, provide a balanced caloric supply of energy to various organs and tissues. The normal metabolism of the lipoproteins responsible for this transport is dependent on the proper functioning of a group of lipolytic enzymes. Lipoprotein lipase (LPL) is primarily responsible for hydrolysis of the triglycerides present in chylomicra and very low density lipoproteins (VLDL) before their uptake as fatty acids by extrahepatic tissues. Heparin-released LPL represent the only enzyme system involved in the catabolism of triglyceride fatty acid (TGFA) in peripheral tissues[1].

Schnatz and Williams[2] studied the effect of acute insulin withdrawal on the LPL activity of epididymal fat from alloxan diabetic rats. They observed a rapid decline in LPL activity causing an inverse correlation with blood glucose, plasma triglycerides and fatty acids. They attributed the decline in LPL activity to the elevated circulating triglycerides of the uncontrolled diabetes.

It has been shown that heparin released LPL activity is dependent on the presence of a protein cofactor, which is a component of natural lipoprotein substrates[3]. Subsequently it has been observed that this cofactor protein increased the catalytic rate of LPL but was not a prerequisite for its activity. Its optimum pH is similar to the pH optimum of lipase (8.0-8.5) rather than the pH which is obtained in physiological states. Its reaction depends upon lipid-water interface. Its ability to induce an increase in the LPL activity is due to the approximation of a positively charged amino-acid residue to the lipase active site[4]. It is shown that preparations of human or rat post-heparin LPL were



The assay system consisted of 10% intralipid, tris-buffer (1.35 M; pH 8.4) 15% bovine albumin, ammonia solution (0.025 M), post-heparin guinea-pig plasma and varying amounts of rat serum.

Showing the amount of FFA liberated after incubation of post-heparin guinea-pig plasma with intralipid, control and diabetic rat serum

	Concentrations of rat serum 0.2 ml		1.0 ml	
	Control	Experimental	Control	Experimental
Mean values ± SEM (mM MFFA liberated)	151.1 ± 19.5	251.9 ± 13.0	298.9 ± 25.6	431.7 ± 26.7
t-values		5.0		3.5
Number of observations	5	5	6	8

Values shown were corrected from the circulating FFA before incubation at 37 °C.

activated only by apolipoprotein C-2 whose origin is extrahepatic[1]. It was therefore thought worthwhile to study the activators of LPL in the serum of alloxan diabetic rats.

The assay was conducted, based on the observation of Whayne and Felts[5]. They showed that guinea-pig post-heparin plasma did not hydrolyze intralipid unless it was activated by the addition of human or rat serum. The methods used in the experiment were conducted according to Chu et al.[6]. Serum of control and alloxan diabetic rats were used to activate the post-heparin plasma of guinea-pigs for the assay.

The response of the system is shown in the figure, which shows that the amount of FFA liberated during the digestion occurs actively till 2.0 ml of rat serum. It was therefore decided to use 2 concentrations to observe the difference between the normal and alloxan diabetic serum.

It can be seen from the table that at 0.2 ml and 1.0 ml of serum concentrations of control and diabetic rats that there has been a significant increase of FFA liberated when diabetic serum was used. This would probably mean that the presence of larger quantities of the activators of LPL in the diabetic serum activated the post-heparin treated guinea-pig plasma to a greater extent than the controls.

The mean values of blood sugar and FFA of the control and diabetic rats were respectively 120 and 444 mg %; 70.4 and 254.6 mM/100 ml of serum. The corrected elevation of plasma activators of LPL in the alloxan diabetic rat serum when compared to controls were 251.9 and 431.7 in 2 concentrations of 0.2 and 1.0 ml of serum respectively.

From these observations one would feel that lipolysis of the circulating triglycerides were occurring very rapidly in the insulin deficient diabetes. On the contrary, inspite of the elevated plasma activators the LPL activity is absent owing to lack of insulin[2]. It seems that insulin could influence LPL activity but could not influence the proteins which help to activate the process. These apoproteins belong to CII which usually predominate in the VLDL[6].

The bond between atherosclerosis and diabetes is still not very clear, since VLDLs play an important role in the atherosclerotic process[7], and apolipoprotein C has been demonstrated in the atherosclerotic lesion of both hyperlipidaemic and normolipidaemic individuals[8]. The presence of significantly high level of plasma activators in insulin deficient diabetes could possibly be the clue to the link between the 2 diseases.

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Viral adenosine triphosphatase

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Summary. The catalytic and immunological properties of an adenosine triphosphatase from different types of virus have been studied. The avian myeloblastosis virus has been found to be specialized in holding this enzyme in a highly active state as compared to other virus with respect to their host cell enzyme. Catalytically myeloblastosis virus and Rous virus ATPase behave alike, while that of the Reo virus is significantly different.

An adenosine triphosphatase (ATPase) activity, associated with avian myeloblastosis virus (AMV)[3], has been purified and characterized[4]. This enzyme is incorporated into the virus during cytoplasmic budding from its host cell myeloblast[5]. The partially purified myeloblastic ATPase did exactly match the properties of the virus enzyme[6]. ATPase activity has also been detected in the Rous virus and Reo virus. The Reo virus enzyme has been shown to differ strikingly from that of the AMV or Rous virus. The results have been incorporated in the present communication.

Materials and methods. AMV and AMV-infected myeloblast were kindly provided by Dr J.W. Beard and G.E. Houts, authorized by the office of Resources and Logistics of the Virus Oncology Program. Rous sarcoma virus was cultured on chicken embryonic cells in our laboratory by standard procedure[7]. Reo virus was generously supplied by Dr A. Shatkin of Roche Institute of Molecular Biology, Nutley, New Jersey. The ATPase activity of the Rous virus, AMV-infected myeloblast and chicken embryonic fibroblast was assayed by colorimetric estimation of Pi[8] as