tion of iodoacetate and cystein, the stability of the hormone is not maintained.

Discussion. The loss of the insulin's antigenic power (coinciding with the disappearance of its biological function) in the presence of cystein, implies the possibility that the disulphide groups of the molecule are also somehow involved in the control over its antigenic power. It is an established fact that the addition of OH ions to an isoelectric solution of cystein liberates protrons of the NH_3 group, with the consequent ionization of the 'thiol' group. In 1956 DE DEKEN et al.[7], studying the displacement of the UV-spectrum of absorption from the ionized cystein, suggested this mechanism. A consequence of this reaction are certain modifications directly connected with the structure of the hormone. At pH 7 these modifications are less profound but increase as the pH is increased.

The fact that iodoacetate is unable to protect the insulin from the inactivating effect of cystein, confirms the possibility that the amino acid action exceeds its reducing power to a much larger degree than the amount of reduced insulin present would seem to justify. It is necessary to account for the fact that reduction of only 10% of the total number of the disulphide bridges present in the molecule is capable of reducing the biological activity of the insulin by 87%[8]. On the other hand, iodoacetate behaves as an inhibitor of the antigenic power of the insulin[4]. The observations compiled in this work tend to confirm the reasonable supposition that the structural formation of insulin governs its antigenic potential.

Resumen. La capacitad de reacción de la insulina de bovino con un anticuerpo especifico desaparece completamente cuando, previamente, se incuba la muestra en presencia de Cisteina. Concentraciones de iodoacetato hasta $1 \cdot 10^{-2} M$ no son capaces de proteger a la hormona contra la acción inactivadora del aminoacido.

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The Effect of a Sudden Rise in Temperature on Strobilae of *Aurelia aurita*

A certain low critical temperature has been listed as one of the criteria for strobilation to occur in scyphistoma larvae of various species from temperate regions[1,2]. During the winters of 1962/63 and 1963/64, scyphistoma larvae of *Aurelia aurita* which had been induced to strobilate at a temperature below 8 °C continued to do so even when the temperature rose as high as 12 °C. As this temperature is higher than that recorded when strobilation occurs naturally, a series of experiments was conducted to see how far strobilation was temperature sensitive and whether scyphistoma larvae could adapt themselves to a rise in temperature once the strobilation mechanism had been initiated.

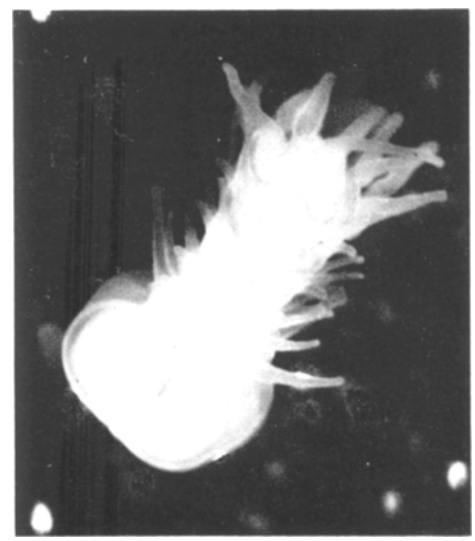
The collection and rearing of scyphistoma larvae has been described in an earlier report[3]. Strobilating individuals were taken either from the stock aquaria or from a low temperature experimental apparatus and were placed in darkened vessels in a water bath of the required temperature. In all other respects the conditions were identical to those the animals had previously been experiencing. It was found that while these animals were generally able to adapt themselves to a gradual rise in temperature up to 12–14 °C, a sudden rise produced the formation of tentacles characteristic of the scyphistoma in place of the ephyral lappets. Such a condition is shown in the Figure, where a 5 ringed strobila has produced 8 tentacles in place of the lappets in each ring.

The results of experiments where there is a sudden change in temperature are shown in the Table.

There are 3 main observations which can be made from these results: (1) Tentacles are formed most rapidly when the temperature is raised to 15 °C or above. If the temperature is not raised so high the effect is either absent or takes much longer to appear. (2) This effect is much more marked in individuals in which strobilation has not proceeded far, i.e. up to about 5 rings. In later stages the effect may take longer to appear, or may be completely absent. (3) There may also be a resorption of some of the proximal rings.

It is interesting to note that in all cases these changes tend to start at the proximal end of the strobilating portion of the scyphistoma and spread distally.

This apparent reversal of the strobilation mechanism has been observed to occur naturally by HAECKEL[4], who lists this tentaculate form as one of 8 variations of strobila. THIEL[5] in his observations on scyphistoma larvae in



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Experiment	Sudden temperature change (°C) From	To	No. of rings on strobila Initial	Maximum	Average time[b] (days)	No. of rings on strobila which were tentaculate on conclusion of observations
1964 1 and 2	11	15	1	..	5 ± 1	0[a]
			1	–		0[a]
			1	2		1[a]
			3	4		3[a]
			7	9		5
			8	10		8[a]
			11	12		8
1965/1	8	21	13	13	5	2
1966 1, 2, and 3	5	15	1	2	4 ± 2	2
			2	2		2
			3	4		4
			3	3		3
			3	4		4
			3	4		4
			4	7		0
			4	7		2
			6	8		7[a]
			6	8		2
			6	7		2
			9	14		7
			12	22	14.5 ± 3.5	1
			12	12		0
			13	16		0
			16	18		0
			17	22		1
			18	24		2
1966/4	5	7	2	19		0
			3	20		0
1966 5 and 6	5	10	3	5	16 ± 2	4[a]
			3	7		7
			5	5		3[a]
1966 7 and 8	5	10	5	(14)		0
			7	(22)		0
			8	(18)		0
			8	24		0

[a] These specimens exhibited resorption of the proximal rings to some extent; in other specimens the number of rings remained unchanged.
[b] These figures show the range in time between the start of the experiment and the first appearance of tentacles in place of lappets on the ephyral rings. Numbers in brackets indicate values at termination of the experiment.

the Kiel Fiord describes this form which he refers to as a 'polyp-strobila', and records that their appearance was subsequent to a drop in temperature to below 1 °C for over 3 months. More recently SPANGANBERG[6] has shown that this effect can be induced by a transference of strobilae from natural to artificial sea water (though it should be noted that her samples were from a population of *Aurelia* that strobilate at a much higher temperature than the European examples). These results emphasize the plasticity of form found in the tissues of the *Cnidaria*, the phylogenetic significance of which has been discussed by THIEL[5] and WERNER[7]. The apparent dependence on environmental conditions also raises the problem of how strobilation is regulated within the scyphistoma. It can be noted that there appear to be 2 distinct stages, the formation of ephyral rings as a result of the buckling of the body wall of the scyphistoma, and the morphogenesis of these into ephyra larvae. The reversion to polyp form begins in the most proximal rings; this could be interpreted on the basis of a gradient of activity controlling the metamorphosis of the scyphistoma into the strobila operating along the proximal-distal axis, and which has its centre at the distal end of the polyp. Recent work on the morphogenesis of *Hydra* by BURNETT[8] and LENTZ[9] has shown that neurosecretory material in the hypostome may be involved in the organization of body form. In *Aurelia* maintenance of strobilation could be controlled by a similar mechanism, dependent in this case on environmental changes[10].

Zusammenfassung. Scyphistomalarven von *Aurelia aurita* strobilieren normalerweise bei Temperaturen unter 10 °C. Die Strobilen können sich bei stetigem Ansteigen der Temperatur bis zu 14 °C normal entwickeln, ein plötzlicher Anstieg über 5 °C führt jedoch zur Entwicklung polypoider Tentakeln anstelle der Ephyra-Arme. Diese Formplastizität veranlasst die Frage nach der Regulation der Metamorphose des Scyphistoma zur Ephyra.

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Deposition of Fat in the Liver Following Administration of Caffeine

During the last years there have been many reports on the increase in non-esterified fatty acids in the blood with subsequent deposition of fat in the liver under the influence of numerous pharmacologically active substances, the effect of which depends partly on a direct attack on the lipolytic enzyme system of the adipose tissue, and partly on being supposed to come into action through the mediation of hormonal and neural carrier mechanisms (PAOLETTI[1]). Purine derivatives likewise belong to the fat-mobilizing drugs. However, as far as we know, no investigations have been made up to the present as to whether caffeine will also produce an accumulation of fat in the liver.

For this reason, white mice (NMRI breed) were given caffeine-sodium-benzoate in doses corresponding to 25 and 50 mg/kg of caffeine s.c. Following this, the content of esterified fatty acids in the liver and in the epididymal

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