accounts for the very low incorporation of 8-^{14}C-adenine (about 1%) into PNA compared with the incorporation of 8-^{14}C-guanine (about 50%) under the same conditions. Obviously with micromolar quantities, as used in these isotope experiments, small amounts of adenine, or hypoxanthine, do become incorporated; these would be undetectable with the concentrations used in the enzyme experiments.

Incorporation of 8-^{14}C-adenine and 8-^{14}C-guanine into *Rhodospirillum* PNA. (8-^{14}C-adenine added as adenine sulphate hemihydrate (S.A. 0.025 mC/mg) and 8-^{14}C-guanine added as the free base (S.A. 0.016 mC/mg). Each result is the mean of 4 determinations).

Counts added (cpm)	Counts in PNA (cpm)	Specific Activity (cpm/μM)		Ratio A/G
		Adenine	Guanine	
1. 195×10^3 (8-^{14}C-adenine)	2.09×10^3	4720	2440	1.94:1
2. 521×10^3 (8-C^{14}-guanine)	256×10^3	720	10400	0.07:1

Zusammenfassung. Das molare Verhältnis von Guanin zu Adenin in PNA von *R. rubrum* ist 1,23. Ganze Zellen von *R. rubrum* verbinden sich mit ^{14}C-Adenin in gleichem Mengenverhältnis zu PNA-Adenin und -Guanin, mit ^{14}C-Guanin aber fast ausschliesslich zu PNA-Guanin.

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Effect of some Hyper- and Hypo-Cholesteremic Drugs on the Cholesterol Biosynthesis by Liver Homogenates

There are some compounds that are capable of affecting the biosynthesis of cholesterol both *in vivo* and in liver slices, but whose mechanism of action has still not been identified.

Intravenous injection of Triton WR 1339 (arylalkylpolyether of phenol, Rohm & Haas, Inc., Philadelphia) has been shown to produce a rise in serum cholesterol[1,2] and FRANTZ and HINKELMAN[3] and BUCHER[4] found that hepatic cholesterol synthesis is accelerated 24 h after intravenous administration of Triton in rat.

Phenylethylacetic acid and its derivatives are known to produce a reduction in serum cholesterol of the hypercholesteremic animals and in the cholesterol biosynthesis of liver slices from acetate[5].

In order to ascertain whether any of these drugs may directly affect the metabolic pathways of cholesterol *in vitro* or whether they need the presence of the living cell, their effect on liver homogenates has been tested.

Materials and methods. A 30% rat liver homogenate containing the following in a volume of 2.5 ml, according to FRANTZ and BUCHER[6], is used: 0.08 *M* potassium phosphate buffer, pH 7.4, 0.03 *M* nicotinamide, 0.0048 *M* $MgCl_2$, 0.0008 *M* DPN, and 0.016 *M* sodium acetate. Three ml of the homogenate are distributed in two sets of 6 glass-stoppered 25 ml tubes. The first set (A) is added with 1 ml assay solution, the latter (B) with 1 ml water. All the tubes are sealed with 100% O_2 and maintained at 37°C for 2 h with shaking.

3 ml of the same homogenate are mixed in a third set of 6 tubes as control (C) with 1 ml water and 1 ml of assay solution.

Total cholesterol prior to incubation is determined according to Bloor.

At the end of incubation 1 ml of water to A and 1 ml of assay solution to B are added and immediately total cholesterol is determined from both the sets of tubes. pH values of all the tubes with and without assay solution at the end of experiment are controlled to be constant within $\pm$ 0.04.

Results. The mean cholesterol contents of liver homogenates in a typical experiment with Triton as assay solution are:

A) After incubation with Triton (final concentration in each tube 50 mg/ml): 249 mg%.

B) After incubation without Triton: 251 mg%.

C) Prior to incubation (control): 227 mg%.

Statistical analysis of the difference between the means B and C shows that incubation at 37°C stimulates a significant cholesterol biosynthesis from acetate in the liver homogenates at the rate of 10.6% (Student's t for 10 degrees of freedom = 3.69; $P < 0.01$). Triton is not capable to produce a rise in the rate of cholesterol biosynthesis at concentrations in the medium between 1 mg and 50 mg/ml.

Of the hypocholesteremic drugs phenylethylacetic acid (PEA), diphenylylethylacetic acid, 3-methyl-4-phenyl-3-butenoic acid (methylphenylvinylacetic acid[7]) and 2-methyl-4-phenylbutanoic acid (methylphenylbutyric acid[7]) have been tested.

The mean results of a typical experiment with sodium PEA as assay solution are: A) After incubation at 37°C with sodium PEA (final concentration in each tube 6 mg/ml): 237 mg%. B) After incubation at 37°C without sodium PEA: 240 mg%. C) Prior to incubation (control): 212 mg%.

Statistical analysis of the difference between the means B and C shows a significant Student's t (t for 10 degrees of freedom = 3.18; $P < 0.01$). In these experiments no inhibitory effect of PEA on the cholesterol biosynthesis by liver homogenates was evident at concentrations between 1 and 100 μM. Similar results were obtained in testing the other hypocholesteremic drugs mentioned.

Riassunto. Nè il Triton W R 1339 nè l'acido feniletilacetico sono capaci di modificare significativamente la biosintesi di colesterolo da parte di omogenati di fegato di ratto incubati a 37°C.

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