decrease meansa decrease in the aerobic metabolism with consequent conservation of food stores. However, this conservation could not be continued for longer periods of starvation. A prolongation of starvation period resulted in a rapid decline in the major nutrient stores of the body[3]. The gradual elevation of succinic dehydrogenase activity from its lowest level at 28 days might therefore indicate a rapid breakdown of the metabolites and their utilization for sustenence during starvation period. The data also suggest that metabolism of hepatopancreas is comparatively highly regulated. Thus while there is nearly a 50% decline in the enzyme activity in the hepatopancreas after 10 days of starvation, it is maintained at more or less normal level in the foot; after 28 days there is more than 85% reduction in the enzyme activity while there is only a 50% decline in the foot. This clearly suggests that there is preferential breakdown and utilization of food reserves from foot. The enzyme activity in the nervering is reduced to half its original level after 28 days of starvation but later there is gradual increase which touches almost normal level of activity. This suggests that the activity in the nervous system does not show any marked decline during starvation[13].

Zusammenfassung. Nachweis, dass die Dehydrogenase-Aktivität im Hepatopankreas, Fuss und Schlundring bei der Lugenschnecke *Ariophanta* sp. eine ausgesprochene Enzymanpassung an die veränderten Stoffwechselbedingungen während langfristiger Hungerperioden zeigt und damit auf einen Überlebensmechanismus hinweist.

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Cholestanol and Chenodeoxycholic Acid: Metabolites of Injected Cholesterol-4-^{14}C in Pigeon Bile

Previous studies from this laboratory have shown that cholestanol (5α-cholestan-3β-ol) is present in high concentrations in tissues and feces of the White Carneau pigeon[1,2]. This stimulated us to determine whether this stanol is of endogenous origin in this species. Except for an in vitro study by SHEFER et al.[3] using rat and guinea-pig livers, others[4,5] could not completely rule out the contribution of intestinal bacterial flora in the conversion of mevalonate and cholesterol into cholestanol. We examined the possibility of the conversion of injected cholesterol into biliary cholestanol in pigeons with bile fistulas, which would eliminate the role of intestinal bacteria in this conversion. Attention was also given to the formation of biliary chenodeoxycholic and cholic acids, which are the major bile acids in pigeon bile[6]. Although this conversion of cholesterol to bile acids has been demonstrated in a number of animals[7], it has not been shown in the pigeon.

Material and methods. An albumin emulsion containing 1–2 μC cholesterol-4-^{14}C was injected i.v. into adult White Carneau pigeons after a cannula was inserted in the bile duct. The bile was collected for 6 to 8 h. Biliary steroids were extracted and the neutral products were separated from bile acids after saponification and extraction[8]. Cholestanol was separated from cholesterol by chromatography on $AgNO_3$-impregnated silica gel G[2]. Bile acids were resolved by thin-layer chromatography on silica gel G using the solvent system consisting of iso-octane : isopropyl ether : acetic acid, 50 : 25 : 40 (v/v/v) and eluted from the gel with methanol and assayed for radioactivity[8]. Identification of the bile acids as based on the behavior of bile acids on thin-layer and gas-liquid chromatography[8] and crystallization with authentic standards.

Results and discussion. The bile was collected for 6 to 8 h after the i.v. injection of cholesterol-4-^{14}C. Early samples of bile were used to determine the formation of labeled cholestanol to minimize the recirculation of cholestanol, if formed, in the intestinal tract by the labeled sterol excreted from the intestinal cell. The neutral biliary fraction (5.2% of total radioactivity) obtained after saponification was subjected to chromatography on $AgNO_3$-impregnated silica gel G to separate cholestanol from cholesterol. The cholestanol fraction was then assayed for radioactivity. An average of 2.13% of the label present in the neutral sterol fraction was contributed by cholestanol. Cholestanol fractions from a number of bile samples were pooled and the identity of this compound was established by chromatography on 2 solvent systems

Radiochemical purity of biliary cholestanol-4-^{14}C

Thin-layer chromatography (TLC)	Rf values	
	Standard	Sample
Silica gel G [a]	0.23	0.23
$AgNO_3$-impregnated silica gel G [c]	0.53	0.53
Specific activity determination [b]	dpm/mg	
First $AgNO_3$ (TLC)	168.5	
Second $AgNO_3$ (TLC)	176.3	
Third $AgNO_3$ (TLC)	170.8	

[a] Solvent system, heptane : isopropyl ether : acetic acid, 65 : 40 : 4 (v/v/v). [c] Solvent system, chloroform : methanol : acetic acid, 100 : 1 : 0.2 (v/v/v). [b] Carrier cholestanol added, 10 mg.

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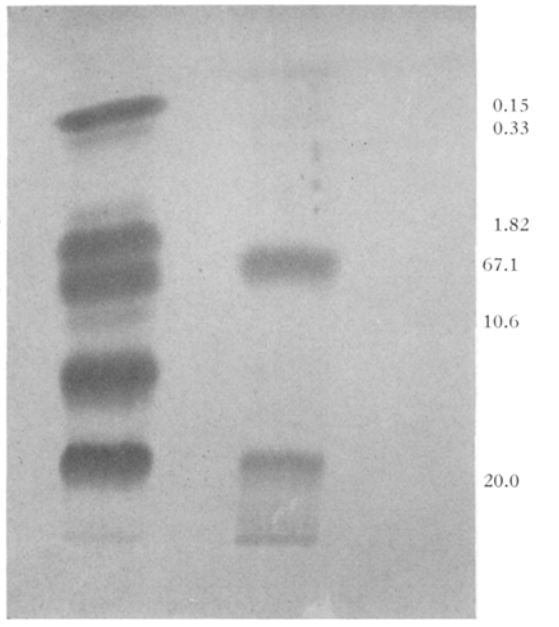

Distribution of radioactivity in biliary bile acids after injecting cholesterol-4-^{14}C (radioactivity expressed as percent of the total radioactivity in bile acids). Identification of bile acids: 1, cholic; 2, hyodeoxycholic; 3, ursodeoxycholic; 4, chenodeoxycholic; 5, deoxycholic; 6, 3β, 12α-dihydroxy cholanoic; and 7, lithocholic. Adsorbent = silica gel G. Solvent system, isoctane:isopropyl ether:acetic acid, 50:40:4 (v/v/v). The plates were stained with phosphomolybdic spray in methanol.

and crystallized to a constant specific activity with authentic cholestanol (Table). The contention that biliary cholestanol is of endogenous origin is supported by the observation that in vitro fecal cultures of the pigeon could not convert cholesterol-4-^{14}C into cholestanol[9].

The acidic fraction was subjected to thin-layer chromatography and the radioactivity present in various bile acid fractions was assessed (Figure). About 67.1% of the total radioactivity was present in the area corresponding to the authentic chenodeoxycholic acid. About 20% of the label was present in the cholic acid area. The rest of it was distributed between another dihydroxy bile acid (10.6%) and unidentified monohydroxy acids (2.3%). The formation of cholic and chenodeoxycholic acids has been demonstrated in many animal species[7]. However, it has not been shown in the pigeon to date. The identity of the label in chenodeoxy and cholic acids was confirmed by thin-layer chromatography of 2 different solvent systems[8] and crystallized to a constant specific activity. The identity of the other dihydroxy and monohydroxy bile acids could not be confirmed in this study.

Zusammenfassung. Es gelang, in Tauben die Umwandlung von injiziertem Cholesterol-C^{14} in Gallen-Cholestanol nachzuweisen, wobei Chenodeoxycholinsäure als Hauptmetabolit des Cholesterols-C^{14} in der Galle ermittelt wurde.

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Perinatal Changes of the Activity of Acyl-CoA: Monoglyceride Acyltransferase in the Rat Intestinal Mucosa

Dietary triglycerides are hydrolyzed in the lumen of the gastrointestinal tract into diglyceride, monoglyceride, free fatty acids and free glycerol[1]. In the small intestinal mucosa re-esterification of the absorbed lipolytic products occurs by two pathways, initiated either from α-glycerophosphate or monoglycerides. The monoglyceride pathway has been found to be more important in mammals, including man[2]. In this pathway acyl-CoA: monoglyceride acyltransferase catalyzes transacylation of activated fatty acids with absorbed monoglyceride forming diglycerides. It was found recently that a high fat diet evoked an increase[3] of this enzyme activity. The suckling period is naturally associated with a high fat intake, e.g., suckling rats' fat intake is 2–3 times higher than later in life[4]. In contrast, activity of pancreatic small intestinal lipase is approximately 10 times lower during the suckling period than in adult rats[5,6]. Thus, the question arose about the extent of the esterification processes in the small intestinal mucosa of suckling rats as compared to adult rats.

Methods. Studies were performed on rats of Charles River strain fed Purina Chow diet. Fetal rats (1 day before delivery) were obtained by caesarian section. 3 month-old males were used as adults. Microsomes from the mucosa (in fetuses and 3-day-old rats from the whole intestinal wall) from the entire jejunum and entire ileum were prepared from nonfasting rats according to Rodgers et al.[7]; materials from 8–9 fetal and suckling rats were always pooled and treated as 1 sample, adult rats were used individually. Activity of the acyl-CoA:monoglyceride acyltransferase was determined according to Rodgers[8],

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