The results with R. rubrum, and probably the other Rhodospirillum species (except R. photometricum), point to the possibility that in these bacteria an APS sulfotransferase like that found in algae and higher plants[13,17,18] is active. We know that our results do not present final proof for this possibility, and also that we cannot exclude the presence of APS sulfotransferase besides APS reductase in the phototrophic sulfur bacteria. Further studies with purified enzymes are under way.

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Effect of trypsin, S-adenosylmethionine and ethionine on L-serine sulfhydrase activity[1]

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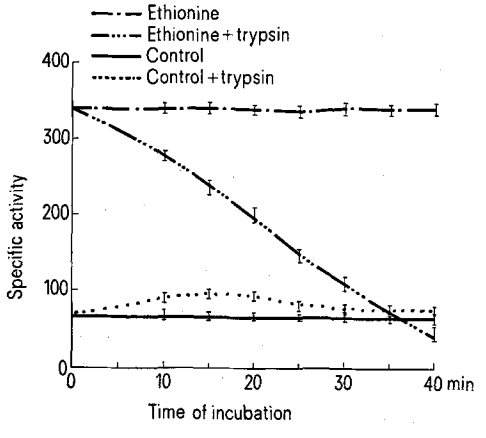
Summary. Trypsin causes an activation of serine sulfhydrase in the liver extracts from intact animals, but inhibits enzyme activity in the liver of ethionine treated rats. Trypsin also decreases an elevation of serine sulfhydrase activity caused by S-adenosylmethionine.

During the last few years, several studies have indicated that both serine sulfhydrase (EC 4.2.1.22) and cystathionine β-synthetase activities are due to a single enzyme[2–4]. Recently, it has been demonstrated that ethionine administration produces a significant activation of cystathionine β-synthetase and serine sulfhydrase in the rat tissues[4–6]. It is also found that S-adenosylmethionine affects as the activator of cystathionine β-synthetase[4,6]. Mudd and his coworkers[7] have shown that trypsin increases the activity of cystathionine-synthetase from an extract of human liver.

As the control mechanism of serine sulfhydrase action has not been known of sufficiently, we examined the interaction of trypsin, ethionine and S-adenosylmethionine in relation to the activity of this enzyme.

Effects of S-adenosylmethionine (SAM) and trypsin on L-serine sulfhydrase activity in vitro (nmoles cysteine/mg protein h)

No. of experiments	Medium for incubation (20 min at 37 °C)	Enzyme activity
9	Enzyme + SAM	99.6 ± 1.8
9	Enzyme + SAM + trypsin	81.5 ± 1.4
9	Enzyme + trypsin	97.3 ± 2.1
9	Enzyme	76.1 ± 0.9



In vitro effect of trypsin on L-serine sulfhydrase activity in the liver of rats treated with ethionine. Vertical bars indicate standard errors of the mean.

Material and methods. Albino rats weighing 160–220 g were used. DL-ethionine was dissolved in 0.9% NaCl and injected i.p. in a dose of 200 mg/kg. The control group received the corresponding volume of 0.9% NaCl. The animals were killed 3 h after the injection. Serine sulfhydrase was extracted and separated from serine dehydratase according to the Kashiwamata and Greenberg method[8]. Trypsin was dissolved in 0.1 M tris-HCl buffer pH 8.3 and added to the aliquots of enzyme extract in the amount of 0.5 y/mg protein. S-adenosylmethionine was dissolved in H_2O and added to the enzyme of control rat liver in the amount of 0.1 micromol per mg protein. Enzyme had been preincubated with S-adenosylmethionine 5 min at 37 °C before adding trypsin. After incubation at 37 °C, the activity of serine sulfhydrase was determined by the procedure of Stepien and Pieniazek[9]. Enzyme activity was expressed in nmoles cysteine per mg protein per h. Protein was estimated by the method of Lowry et al.[10]. DL-ethionine and S-adenosylmethionine were obtained from Calbiochem while trypsin was purchased from Merck.

Results and discussion. The effect of trypsin treatment on L-serine sulfhydrase activity is shown in the figure. Trypsin causes an increase in serine sulfhydrase activity in the liver extracts from intact animals. On the contrary, when trypsin was incubated with enzyme extracts prepared from the liver of the rats which had received DL-ethionine, a successive diminution in serine sulfhydrase activity was observed. The action of S-adenosylmethionine and trypsin upon serine sulfhydrase levels is presented in the table. It is evident from these data that S-adenosylmethionine as well as trypsin produce a remarkable rise in serine sulfhydrase. However, addition

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of trypsin to the enzyme preincubated with S-adenosylmethionine results in a marked decrease in serine sulfhydrase activity. As shown in the table, trypsin and S-adenosylmethionine act not synergistically, whereas the combined treatment with both trypsin and S-adenosylmethionine produced a smaller increase in serine sulfhydrase activity than did treatment with trypsin or S-adenosylmethionine alone. It is undoupted that trypsin has different effects upon control enzyme and enzyme altered with S-adenosylmethionine or ethionine. However, the nature of the interrelationships among various actions of trypsin, ethionine and S-adenosylmethionine is unknown. It is now well established that administration of ethionine to rats leads to a rapid synthesis of S-adenosylethionine in the liver[11-14]. Both S-adenosylmethionine (SAM) and S-adenosylethionine (SAE) were found to increase hepatic cystathionine β-synthetase[4,6]. The present findings, coupled with the reports cited above, suggest that action of ethionine on serine sulfhydrase is mediated by S-adenosylethionine. It is obvious from the results of this study that trypsin and S-adenosylmethionine (as well as S-adenosylethionine) act in a different manner to activate rat liver serine sulfhydrase. The exact mechanism of interaction of trypsin and S-adenosylmethionine or S-adenosylethionine is not clear as yet. Based on the information derived from these experiments, it is postulated that serine sulfhydrase activated by SAM or SAE is more liable for proteolytic action of trypsin. These data support the conclusion that activation of serine sulfhydrase by S-adenosylmethionine or S-adenosylethionine is due to some modification of the enzyme structure.

Since trypsin has been shown to cause an increase in cystathionine β-synthetase activity[7], the findings presented in this study confirm the opinion that cystathionine β-synthetase and serine sulfhydrase activities are associated with the same protein.

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Changes of the prostaglandin F_{2a} metabolism in early human placenta[1]

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Summary. The quantitative metabolism of PgF_{2a} was studied in different ages of early human placentae in vitro. The 15-OH-Pg-dehydrogenase became minimal at about the 9th week.

In the last 10 years, it was proved that prostaglandins take an important part in regulating pregnancy. It was demonstrated by Karim and Devlin[3], Karim and Hillier[4], Jouvenaz et al.[5] and Keirse et al.[6] that the endogenous prostaglandins (E_2 and F_{2a}) significantly increase in the amniotic fluid from early pregnancy till term. According to Willmann et al.[7], the main site of synthesis is the decidua and myometrium. Metabolism mostly takes place in the placenta which is very active as regards 15-OH-prostaglandin-dehydrogenase, as well as prostaglandin Δ^{13} reductase enzymes[8,9]. Carminati et al.[10,11] studied the quantitative metabolism of the PgF_{2a} and E_1 at various stages of pregnancy in the rat. Results of these investigations showed that metabolism became maximal between days 9 and 12 of gestation.

In our own work we have studied 15-OH-prostaglandin-dehydrogenase (15-OH-PGDH) activity in early human placentae during the course of gestation.

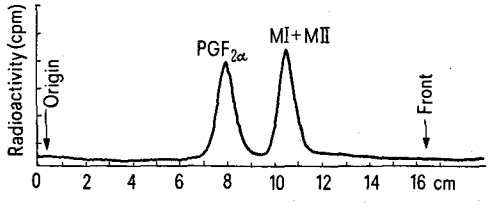

Fig. 1. Radiochromatogram demonstrating prostaglandin metabolism by homogenat of early human placenta. Solvent system, benzene:dioxan:acetic acid = 10:10:1; MI, 15-keto-PgF_{2a}; MII, 15-keto-13, 14-dihydro-PgF_{2a}.

Material and methods. 45 placenta tissues were obtained from interruption of pregnancies in healthy individuals. The length of pregnancy was established from the time which elapsed since the first day of final menstruation until the interruption. The placenta tissues were quickly separated and homogenized with 4 volumes of ice-cold Bücher medium[12] in a Potter-Elvehjem tissue grinder. The whole homogenates were centrifuged at 10,000 × g and 2 ml of supernatant incubated with 1.25 μCi H^3-PgF_{2a} (9.3 Ci/mMol, New England, Nuclear Corp.).

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