nen uns Untersuchungen von SLATER et al.[33,34] bemerkenswert, nach denen die während der Entwicklung des Seeigeleis transkribierte langlebige mRNA über wenig Poly(A)-Reste verfügt; erst nach der Aktivierung und Demaskierung der stabilen mRNA, die mit der Befruchtung des Eis einsetzen, werden die Oligo(A)-Sequenzen im Cytoplasma (nicht im Kern[30]) verlängert.

In diesem Zusammenhang erhebt sich die Frage, ob im quellenden *Agrostemma*-Embryo die Poly(A)-Sequenzen an neu transkribierte Hn-mRNA oder an gespeicherte Poly(A)$^-$-mRNA angefügt werden. Um diese aktuelle Problematik einer Klärung näherzubringen, haben wir die Hybridisierung ^{3}H-Uracil-markierter und ^{14}C-Adenin-markierter RNA miteinander verglichen. Da auf die Poly(A)-Sequenzen etwa 30 bis 40% der ^{14}C-Adenin-Markierung entfallen (Tabelle II), ist von vornherein ein erheblich höherer Hybridisierungsgrad der Adenin-markierten RNA zu erwarten. Die erhaltenen Befunde (9% bei ^{3}H-Uracil-markierter RNA, 19% bei ^{14}C-Adenin-markierter RNA, Tabelle I) sprechen unseres Erachtens dafür, dass die Mehrzahl der Poly(A)-Reste an neu synthetisierte Hn-mRNA angefügt wird; andererseits schliessen unsere vorläufigen Resultate die Möglichkeit einer Polyadenylation stabiler mRNA nicht völlig aus.

Zu klären bliebe weiterhin, ob mRNA aus trockenen *Agrostemma*-Samen tatsächlich Poly(A)-Sequenzen enthält oder ob die Bindung an Oligo-dT-Cellulose unspezifischer Natur ist. Wird RNA aus trockenen Embryonen vor der Hybridisierung mit Poly(U) geschüttelt, erfolgt keine Hybridisierung. Dieses Experiment (siehe Figur A) kann man als Hinweis auf die Existenz langlebiger Poly(A)-mRNA gelten lassen, der exakte Nachweis wäre beispielsweise durch Hybridisierung mit ^{3}H-Poly(U) zu erbringen[35].

Der geringe Anteil von Poly(A)-RNA an der Gesamt-RNA trockener Samen muss nicht zwangsläufig bedeuten, dass nur wenig langlebige mRNA in *Agrostemma*-Samen gespeichert wird. Durch die Affinitätschromatographie wird lediglich Poly(A)haltige RNA erfasst. Ob daneben noch Poly(A)$^-$-mRNA enthalten ist, wie das von WALBOT[23] bei Baumwollsamen vermutet wird, muss offen bleiben.

Die Synthese Poly(A)-haltiger RNA, die im Gegensatz zu Befunden anderer Autoren[11,13,23,36] bei *Agrostemma* unmittelbar mit der Stoffwechselaktivierung quellender Samen einsetzt, lässt eine wesentliche physiologische Bedeutung vermuten. An anderer Stelle soll gezeigt werden, dass die frühe Proteinbiosynthese quellender *Agrostemma*-Embryonen von einer vorangegangenen RNA-Synthese unmittelbar abhängig ist[37]. Inwieweit an der frühen Proteinsynthese neben neu synthetisierter mRNA auch stabile, in ruhenden Samen gespeicherte beteiligt ist, ist Gegenstand der zur Zeit laufenden Untersuchungen.

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Levels of β-Trace Protein and Lysozyme in Human Amniotic Fluids

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Summary. The levels of β-trace protein and lysozyme were estimated in amniotic fluids from normal fetuses and from fetuses with neuraltube defects. The values of these proteins in normal amniotic fluids were found to be similar to those detected in fetuses with anencephaly and spina bifida. The levels of lysozyme were shown to be correlated with gestational age.

Studies from BROCK et al.[2,3] have shown that high levels of α-fetoprotein (AFP) are present in the amniotic fluid of fetuses with anencephaly and/or spina bifida. Consequently, the estimation of this fetal protein in amniotic fluid is widely used as a diagnostic test for the antenatal detection of neural-tube defects[4].

Since the levels of AFP are elevated in the cerebrospinal fluid (CSF) from human fetuses less than 25 weeks old[5] it has been suggested that the abnormal, high values of this protein in the amniotic fluids of fetuses with 'open' neural-tube defects are caused by the transfer of AFP from CSF into the amniotic cavity.

Recently high counts of macrophages have been detected in amniotic fluids of fetuses with CNS defects[6,7] and it appears that this test may be useful for the diagnosis of such abnormalities. These observations have prompted us to estimate the amniotic levels of β-trace protein, a major component of CSF[8,9] and compare them with those of AFP, lysozyme (LZM) and albumin.

Materials and methods. 46 samples of amniotic fluids were collected by amniocentesis in the course of an investigation on the incidence of fetal chromosome abnormalities. Only samples from fetuses which were found free from chromosome abnormalities were tested. Soon after being collected, the amniotic fluids were centrifuged and the precipitates discarded.

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Amniotic fluids from 7 fetuses with 'open' types of neural-tube defects were kindly provided by Dr. MARY SELLER, (Guy's Hospital Medical School, London). The amniotic fluids were obtained between 9 and 24 weeks of gestation.

The levels of β-trace protein were estimated by the the single radial diffusion technique using a monospecific horse immune serum. The amount of β-trace protein was expressed as a percentage of a purified preparation of this protein (10 mg/l) isolated from the urine of a patient with plasma cell dyscrasia.

The estimation of AFP was carried out using the rocket (Laurell) method, as previously described[5] and the levels of albumin were measured by the single radial diffusion technique[10]. The concentrations of LZM in amniotic fluid were estimated by the lyso-plate method[11] using purified human LZM as a standard (100 mg/l). The levels of total protein were measured by the LOWRY method[12].

Results and discussion. As shown in Figure 1, the levels of β-trace protein in the amniotic fluids obtained from fetuses with anencephaly and/or spina bifida were similar to those detected in the samples from 9 fetuses without neural-tube defects. No relationship was observed between the amount of β-trace protein and those of AFP, albumin and total protein in the two groups of amniotic fluids (Figure 2).

These results are at variance with two short reports by Macri et al.[13,14] who were able to detect β-trace protein, by double diffusion in agar gel, in the amniotic fluid of a single human anencephalic fetus and in the amniotic fluids from rat fetuses with neural-tube defects, whereas the protein was not detected in samples obtained from normal fetuses. However, in both investigations the concentrations of β-trace protein and AFP were not estimated and the specificity of the immune serum against β-trace was not evaluated using a sample of the purified protein.

It has been shown that β-trace protein, originally described in CSF, is also present in the serum, urine, pleural and ascitic fluids[8,9]. These findings suggest that the small amount of this protein detected in all the normal human amniotic fluids tested is possibly derived from fetal urine. The lack of relationship between the levels of β-trace protein and AFP in amniotic fluid does not exclude leakage of both proteins from CSF in fetuses with neural-tube defects. Investigations in experimental animals suggest a different catabolism and rate of transfer across the amniotic membranes of the two proteins (HADDAD and ADINOLFI, unpublished observations).

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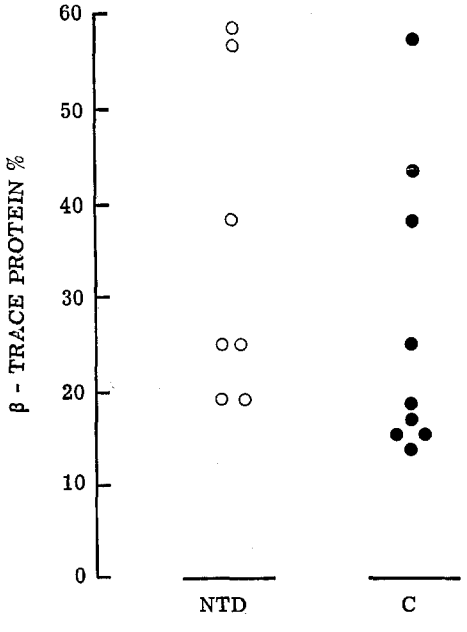

Fig. 1. Levels of β-trace protein in amniotic fluids from fetuses with neural-tube defects (NTD) (○) and controls (●) expressed as percentage of a standard preparation (see methods).

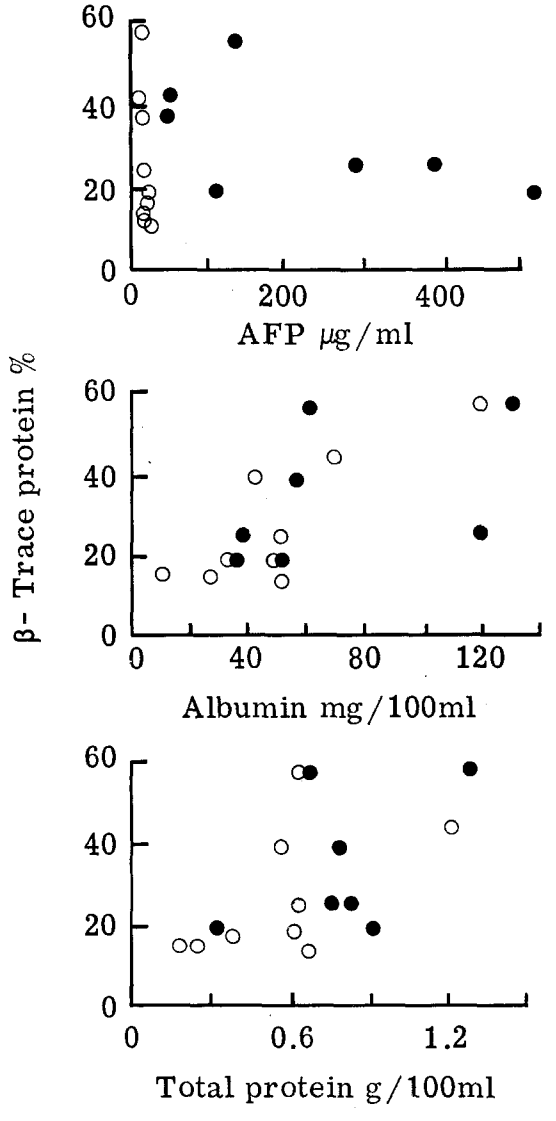

Fig. 2. Relationship between the levels of β-trace protein, AFP, albumin and total protein in amniotic fluids from fetuses with neural-tube defects (○) and controls (●).

Gestational age and levels of lysozyme in human amniotic fluids

Groups	Gestational age (weeks)	Number tested	Levels of lysozyme in individual samples (mg/l)	Mean values
A	9	1	4.8	
	10	4	1; 4.2; 6; 7.9	
	11	3	1.2; 2; 5	4.6
	12	5	1.2; 4.8; 5.9; 7; 8.8	
B	13	3	5; 6.8; 8	
	14	8	3.1; 4; 6; 6; 6; 7.2; 11.2; 13.2	
	15	1	4.4	7.4
	16	4	5.2; 8; 9.2; 15	
C	17	3	4.8; 7.2; 12.1	
	18	4	5.6; 8.8; 8.8; 15	
	19	2	9; 15.2	9.7
	20	3	5.8; 11; 13.9	
D	21	–		
	22	3	5.5; 8.5; 18	
	23	–		11.8
	24	2	8; 11.8	

The levels of LZM varied in 46 amniotic fluids from 1 to 18 mg/l, and considerable variations were observed in samples collected at about the same stage of gestation. LZM activity was detected in the amniotic fluid of the youngest fetus tested, 9 weeks old. When the samples were divided into 4 groups according to the age of the fetuses (Table) the mean levels of LZM in each group were found to increase as pregnancy progressed. The detection of LZM in amniotic fluids at an early stage of gestation is in agreement with the observed early synthesis of the enzyme during fetal development[15,16].

Normal levels of LZM were observed in the amniotic fluids from 7 fetuses with neural-tube defects; the individual values were found to range between 3 and 16 mg/l.

The high counts of macrophages observed in amniotic fluids of fetuses with CNS defects[8,9] do not seem to affect the levels of LZM. The clinical significance of estimating the levels of LZM in amniotic fluids to monitor bacterial infections of the fetus remains to be established.

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Deep Temporal Lobe Projections to the Nucleus of the Diagonal Band of Broca

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Summary. Electrophysiological studies with acutely prepared cats found that stimulation of deep temporal lobe structures (e.g., amygdala, prepyriform cortex) evoked responses in the nucleus of the diagonal band of Broca. An analysis of field, extra- and intracellular unitary responses points to the existence of a monosynaptic excitatory connection.

Early electrophysiological studies by Stoll et al.[2] and Gloor[3] suggested the presence of a pathway to the septal area from deep regions of the anterior temporal pole. Specifically, a pathway from the amygdala to the septal component of the diagonal band of Broca (nDBB)[4] has been suggested by anatomical techniques[5–8]. The cortex subjacent to the amygdala (e.g., the prepyriform cortex) might also project to the nDBB[9]. The present report investigates some of the physiological properties of these deep temporal lobe pathways to the nDBB.

Cats were anesthetized with thiamylal sodium (25 mg/kg) and chloralose (40 mg/kg). The septum and hippocampus were exposed by removing the overlying cerebral cortex and corpus callosum. Bipolar stimulating electrodes with an inter-pole distance of 0.5 mm were stereotaxically placed in the amygdala and prepyriform cortex. In most of the experiments, the amygdala stimulating electrodes were arranged so that the basolateral group or the anterior amygdaloid area from AP levels +12 to +15 was stimu-

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