these experiments were similar to those presented above for mouse explants: the addition of prolactin caused an increase in fatty acid synthesis, whereas the addition of neurohypophyseal hormones had no effect (data not shown).

The above experiments indicate that vasopressin contamination is not solely responsible for the stimulation of mammary function in vitro by prolactin preparations, since the substitution of vasopressin for prolactin does not stimulate fatty acid synthesis. In addition, prolactin preparations (rat B1 and ovine oPRL-14) with a 100-fold difference in vasopressin contamination show the same effects on MCFA synthesis at similar doses. However, these observations do not eliminate the possibility of synergy between prolactin and vasopressin.

Such interactions were examined in two ways. First, vasopressin was added to different doses of prolactin and the effect on MCFA synthesis by explants determined. Explants incubated with IF-medium and prolactin (oPRL-10; 10 an 100 ng/ml) had approximately 200% and 670% more incorporation into MCFA than explants incubated in IF-medium alone. Explants incubated with similar doses of prolactin plus vasopressin (100 pg/ml) had 220% and 610% more incorporation into MCFA than explants incubated in IF-medium alone. At each concentration of prolactin, the responses of explants incubated with or without vasopressin were not statistically different ($p > 0.1$). These results do not rule out possible synergistic interactions between vasopressin and prolactin, since oPRL-10 is contaminated with vasopressin. They suggest, however, that the effect of vasopressin must be maximal at the lowest dose of prolactin tested (10 ng oPRL-10 contains approximately 0.1 pg vasopressin).

Synergy between vasopressin and prolactin was investigated further by treating prolactin with anti-vasopressin antiserum. Excess anti-vasopressin antiserum precipitated over 85% of [^{125}I]vasopressin; treatment with additional antiserum did not precipitate more material. Treatment of ovine prolactin (oPRL-14) with excess antiserum removed 100% of the precipitable [^{125}I]vasopressin. As seen in the figure, incubation of explants with either treated or untreated prolactin caused a similar rise in MCFA synthesis.

The above experiments show that vasopressin does not directly stimulate the synthesis of milk-specific or total fatty acids in explants of mammary tissue from pregnant animals. In addition, this neurohypophyseal hormone does not synergistically interact with other hormones to affect this process. Thus, vasopressin contamination of prolactin preparations is not responsible for this lactogenic effect. A similar conclusion has been reported regarding the regulation of casein synthesis in mammary explants from pregnant rats[20]. The reason for the stimulation of fatty acid synthesis by vasopressin in the WRK-1 cell line is still unclear; however, it does not appear to reflect a differentiative process found in normal mammary tissue. The role(s), if any, that vasopressin may have on mammary gland function in vivo during lactogenesis is not addressed by this study. In addition, possible short term effects of this peptide on mammary fatty acid synthesis in vitro were not investigated.

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Normal adenylate-cyclase activity in platelets of patients with Huntington's disease

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Summary. Adenylate-cyclase activity and cyclic-AMP concentration in platelets of 7 Huntington's disease patients were found to be similar to control values.

Impaired platelet aggregation has been found in Huntington's disease patients[1]. The 2nd phase of aggregation[2] was decreased when ADP was used as an inducer, and 50% of the patients also showed disaggregation[1]. That the effect was consistent with an alteration of the release reaction was supported by reduced ^{14}C 5-HT release. However, the platelet serotonin uptake was normal[1].

We have found that the platelet response to ADP, in 4 patients, was normalized when 0,5 mM (final concentration) of sodium arachidonate was added to the platelet-rich plasma (unpublished results). This suggests that the cyclooxygenase activity is not affected in Huntington's disease.

Normally the magnitude of aggregation depends on the amount of ionized calcium and ATP available. Cyclic-AMP

removes ionized calcium from the cytosol, thus inhibiting the contractile processes[3]. Increased levels of c-AMP also inhibit platelet reactions[4]. The fact that the 1st aggregation wave was normal in our patients argues against the possibility of increased c-AMP[1]. However, as suggested by Holmsen[5], the degree of response depends on the intensity of the stimulus. A weak one will only produce a 1st aggregation phase. Similarly, it might also be possible that the inhibition produced by c-AMP was not strong enough to alter the 1st phase. Recent work by Johnson et al.[6] in dogs demonstrated that c-AMP plays an important role in regulating the response of platelets to the aggregating products of prostaglandin metabolism. For this reason we have studied the activity of adenylate-cyclase and the steady-state concentration of c-AMP in platelets of patients with Huntington's disease.

Venous blood from 7 Huntington's disease patients and 11 normal controls was collected in a fasting state, between 08.00 h and 09.00 h. The control group was matched for age and sex. None of the patients were under medication. Informed consent was obtained in all cases.

Adenylate-cyclase activity was determined in a platelet rich fraction by homogenizing the final pellet with 2 ml of 2 mM Trismaleate buffer pH 7.4, containing 2 mM EGTA[7]. The standard assay system (final volume 0.5 ml) was as described[8]. Cyclic-AMP was determined by radio-immunoassay (New England Nuclear). Statistical analysis was by Student's t-test.

As shown in the table, the adenylate-cyclase activity and cyclic-AMP content in platelets of Huntington's disease patients and controls were similar. It seems, therefore, that the platelet defect observed in Huntington's disease is not due to an increase in the levels of cyclic-AMP.

Adenylate-cyclase activity and cyclic-AMP content in platelets of Huntington's disease patients

Groups	Adenylate-cyclase activity (pmole/mg protein/2.5 min)	Cyclic-AMP (pmole/mg protein)
Huntington's disease	67.2± 10.1*	2.8±0.8
Controls	69.1±4.2	2.8±0.6
p	NS	NS

* Values expressed as mean± SE.

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Characterization of cellulase from *Humicola lanuginosa*

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Summary. In a carboxymethylcellulose-yeast extract medium, *Humicola lanuginosa* synthesized cellulase. The purified enzyme possessed a molecular weight of 71,000 daltons and exhibited optimum enzymatic activity at pH 5.0 and 45 °C.

Some species of *Humicola,* like a number of other thermophilic fungi, are able to hydrolyze cellulose substrates[1,2]. However, some workers have shown that *H. lanuginosa* is unable to synthesize cellulases[3-5]. Recently a group of workers reported an isolate of *H. lanuginosa* which was cellulolytic[6]. During our work on decomposition of organic matter by thermophilic fungi, we isolated a strain of *H. lanuginosa* which produced cellulase. The present work examines the production and purification of the enzyme.

Materials and methods. The isolate (UNIFE 147) of *Humicola lanuginosa* (Griff. and Maubl) Bunce employed was taken from decomposing compost and was identified by Professor M.H. Zoberi. The growth medium and the inoculation techniques were as previously described[7]. Experimental flasks were incubated at 45 °C for 6 days. Ability of the organism to degrade soluble and insoluble celluloses, measurement of cellulolytic activity and other experimental procedures were as previously described[7]. Purification procedures involving a combination of ammonium sulphate precipitation, batch adsorption on DEAE-Sephadex A-25, ultrafiltration (UM-10), gel filtration and ion-exchange chromatography were performed as described elsewhere[7-10]. One unit of cellulase activity was defined as the amount of enzyme in 1 ml of the reaction mixture that released reducing sugars equivalent to 1 µmole glucose. Specific activity was defined as units per mg protein.

Results and discussion. Humicola lanuginosa failed to grow in defined liquid media containing soluble (carboxymethyl-

Table 1. Growth and synthesis of cellulase by *H. lanuginosa* grown in liquid synthetic medium containing 1% carboxymethycellulose and different concentrations of yeast extract

Concentration (%, w/v) of yeast extract added to 1% (w/v) of carboxy-methylcellulose	Mycelial dry wt (mg)	Cellulase activity (units/mg protein)
0.00	0.0±0.0*	0.0 ± 0.0
0.05	75.7±2.8	0.21±0.02
0.10	99.5±1.5	0.36±0.01
0.15	152.8±2.9	0.65±0.03
0.20	186.8±1.1	0.91±0.05
0.25	219.6±1.8	1.10±0.02
0.30	221.5±2.1	1.13±0.01
0.35	229.1±3.7	1.13±0.03
0.40	230.5±2.8	1.12±0.05

* Each value represents the mean of 6 replicates with SE.