We investigated further whether ICSA of non-diabetic subjects with an antecedent mumps infection exhibit complement-dependent cytotoxicity against rat islets of Langerhans in vitro [8]. Out of a total of 24 subjects, the sera of 11 exhibited cytotoxicity. Ten out of the 11 subjects with cytotoxic sera had HLA DR3 and/or DR4, whereas DR3 was present in 3 out of 13 subjects with non-cytotoxic sera. There was no relationship between B-cell function in vivo and the capability of patient sera to damage islet cells in vitro. The results support the hypothesis that complement-dependent cytotoxicity of ICSA may be involved in the B-cell damage in some subjects with an antecedent mumps infection and diabetic risk. In the study of Helmke et al. [2], HLA-typing was carried out in 45 mumps-infected children. The typical HLA-associations as established for Type 1 diabetes were not seen in ICA-positive mumps patients. In agreement with the authors, we failed to establish any relationship between the most common diabetes-associated HLA markers and the occurrence of both ICA and ICSA in 125 randomly selected subjects with antecedent mumps infection [7]. In addition, there was no relationship between ICA or ICSA and B-cell function (fasting C-peptide and insulin response to OGTT) and haptoglobin phenotype in the study group. We have concluded from our study that the results do not support a close temporal relationship between mumps infection and onset of Type 1 diabetes mellitus. The view is in agreement with our findings of a screening programme for diabetes in 1080 unselected children from rural and urban communities who had had mumps 10–19 months previously [6]. 1069 (99%) of the 1080 subjects had no diabetes mellitus, diabetic symptoms or glucosuria. A positive urine screen was obtained in 11 subjects (1%) studied; all these cases finally had normal glucose tolerance tests. The prevalence of Type 1 diabetes mellitus in children (<16 years of age) in the year of the study was 42.9 per 100 000 subjects of the general national population. On the other hand, the prevalence of mumps infection in the general population at the ages studied was 5012.7 per 100 000 [6]. Thus, purely on a statistical basis, one would have expected a higher number of Type 1 diabetes cases if mumps were closely related to diabetes.

In conclusion, the pathogenetic role of mumps virus and islet cell antibodies as their possible relation to slow progressive B-cell destruction is still to be ascertained. We agree with Helmke et al. [2] that a virus infection could be the final insult to the immune system or islets which are undergoing destructive autoimmune processes leading to the onset of clinical diabetes. It seems likely that, if mumps infection has a diabetogenic role, it is only relevant to a few cases.

Yours sincerely,
K. P. Ratzmann

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Nontraumatic rhabdomyolysis during diabetic ketoacidosis

Dear Sir,

In a recent article, Moller-Petersen et al. [1] evaluated the frequency of nontraumatic rhabdomyolysis in diabetic ketoacidosis. They concluded that “the pathogenic mechanism leading to rhabdomyolysis in diabetic ketoacidosis remains unsettled”. In a young patient recently admitted for ketoacidosis and presenting rhabdomyolysis, we also looked for unusual causes of rhabdomyolysis (Table 1).

This 20-year-old female was previously in good health, and never presented cramp nor muscle pain. During two months she experienced fatigue and progressive weight loss; four days before entry into hospital, she complained of dyspnea, polyuria and vomiting. On admission, the diagnosis of ketoacidosis, clinically evident, was confirmed by glycosuria, ketonuria, hyperglycaemia (52 mmol/l), low plasma standard bicarbonate concentration (4.8 mmol/l) and arterial pH (7.03). Serum urea and creatinine were elevated (16.8 and 0.43 mmol/l respectively) and creatine kinase activity in serum was moderately increased to 120 U/l (N: 5–130). Serum phosphorus was normal (1.29 mmol/l). Treatment was immediately started with classical rehydration by intravenous saline containing sodium bicarbonate and potassium chloride combined with low-dose insulin given intravenously by a syringe pump. Glucose infusion was progressively introduced when glycaemia decreased. Clinical and biological improvement was rapidly obtained (Table 1). Surprisingly, serum creatine kinase activity increased dramatically to 7690 U/l on the next day after admission, then progressively decreased to normal values within 15 days. Usual causes of rhabdomyolysis [2] were rapidly excluded (no muscular trauma or compression, no intramuscular injection, normal electrocardiograms . . .).

We suggest the hypothesis of a metabolic disease such as McArdle disease, phosphofructokinase deficiency or other glycolytic defect, possibly revealed by ketoacidosis, and responsible for rhabdomyolysis in this case. To screen for disorders of anaerobic glycolysis, we used the procedure proposed by Layzer [3]. In fact, two weeks after ketoacidosis, all the tests proved normal: ischaemic exercise with sphygmomanometer cuff placed around the upper arm was possible during more than one minute, ischaemic exercise with ergometric bicycle produced a normal rise in blood lactate levels (from 1.30 to 7.10 mmol/l). Finally, muscle biopsy only demonstrated myolysis sequelae but did not find histoenzymologic abnormalities (particularly for phosphorylase activity).

In conclusion, we wish to emphasize that, in our opinion, enzymatic defects of glycolysis are probably not the cause of rhabdomyolysis during diabetic ketoacidosis. Moreover, in our observation, as in Moller-Petersen's case, it is noteworthy that maximal rhabdomyolysis occurred a few hours after beginning treatment. Rhabdomyolysis might therefore be related to rehydration and/or correction of hyperglycaemia and ketoacidosis. In this setting, the fall in serum phos-

phate due to the transfer of phosphorus in the first hours of therapy might play an important role. However, this seems unlikely if one considers the prospective study of Møller-Petersen et al. [1], which did not find a relationship between phosphate depletion and rhabdomyolysis.

Yours sincerely,

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Table 1. Evolution of biological parameters in a patient presenting diabetic ketoacidosis with rhabdomyolysis

	On admission	Hour 6	Hour 11	Day 2	Day 3	Day 4	Day 15
Glycaemia (mmol/l)	52	42.6	33	21.4	16.8	4.9	–
S-Urea (mmol/l)	16.8	17.1	18.6	14.6	16.2	9.8	2.2
S-Creatinine (mmol/l)	0.432	–	–	0.221	0.162	0.118	0.060
S-Sodium (mmol/l)	134	127	132	136	135	144	142
S-Potassium (mmol/l)	4	4.3	3.5	3.7	2.8	3.1	3.8
P-Standard bicarbonate (mmol/l)	5.1	9.1	13	14.5	17.5	16.1	26
Arterial pH	7.03	7.28	–	7.31	7.45	–	–
S-Calcium (mmol/l)	2.16	–	–	2.12	1.97	2.02	–
S-Phosphorus (mmol/l)	1.29	–	–	0.13	0.41	0.47	–
S-Glutamate oxaloacetic transaminase (U/l)	13	–	–	250	–	–	–
S-Creatine kinase activity (U/l)	120	–	–	7691	–	3371	38

Moderne klinische Diabetologie

Zweiter Internationaler Weiterbildungskurs für Fortgeschrittene in Deutscher Sprache

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