deionized water and eluted into a collector of 0.35 mm i.d. PTFE tubing. 75 µl of the collected eluate was then pumped into the graphite tube. The reproducibility of FI peak gradients was exploited through precise timing of the collection and dispensing of the eluate fraction so that the end sections of the eluate bolus, which contained only low analyte element concentrations were discarded. The complete cycle of operation took 150 s which conveniently fitted into the time interval between successive graphite furnace firings.

Results

A 26-fold enhancement in the peak area compared to the direct introduction of 50 µl was obtained with 60 s preconcentration. The precision for 0.1 µg/l Pb (n = 11) was 1.9% r.s.d., and a detection limit of 0.003 µg/l (3 σ) was achieved. Determination of Pb in sea water samples showed that the interfering matrix was almost completely removed during preconcentration, and determinations were made without using chemical modifiers. Results obtained for sea water standard reference materials using matrix-matched standards, and for river water using simple aqueous standards agree well with certified values (Table 1).

Table 1. Determination of lead in standard reference materials for sea and river water using FI on-line sorbent extraction preconcentration GF-AAS

Standard reference material	Sample loading period (min)	Concentration (ng/l Pb)	
		certified	found[a]
NASS-2 sea water	2	39 ± 6	37 ± 2
CASS-1 sea water	1	251 ± 27	267 ± 6
CASS-2 sea water	2	19 ± 6	22 ± 1
SLRS-1 river water	1	106 ± 11	117 ± 2

[a] Average and standard deviation of 6 preconcentrations/determinations

Conclusion

The results show that the principles and techniques of FI could be exploited to improve the performance and extend the capabilities of GF-AAS. It is quite exciting to anticipate the vast number of possibilities for further improvement of the GF-AAS technique when the wealth of chemical literature on ion-exchange, chelate formation, and solvent extraction is re-explored in the context of a new solution handling concept. The combination of GF-AAS with FI automated on-line sample manipulation and with the appropriate chemistry could create new analytical systems with unrivaled sensitivity and selectivity.

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Serum contents of immunoreactive pancreatic secretory trypsin inhibitor and seminal plasma acrosin-trypsin inhibitor in polytraumatized patients

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Human pancreatic secretory trypsin inhibitor (PSTI), a Kazal-type serine protease inhibitor, was isolated and extensively characterized by Bartelt et al. [1]. PSTI has been thought to exist only in the pancreas having the function to prevent autoactivation of trypsinogen in the pancreas and the pancreatic juice. Ogawa and coworkers ([4], review), however, found that immunoreactive PSTI is present also in other tissues and reported that raised serum levels of PSTI occur in various malignant diseases and after severe injuries.

Only very recently the elucidation of the amino acid sequence (manuscript in preparation) of HUSI-II, the acrosin-trypsin inhibitor of human semen [2], demonstrated unequivocally that this inhibitor also belongs to the Kazal family. The findings of Ogawa and coworkers [4] prompted us to investigate whether the plasma concentration of HUSI-II shows a similar response as that of PSTI in patients with polytrauma.

Methods

The clinical significance and pathophysiological relevance of a series of biochemical parameters was investigated in a comprehensive study including 69 patients with polytrauma. The average Injury Severity Score (ISS) was 36. The patients were subdivided in three groups: 11 patients died due to multiorgan failure, 29 patients overcame multiorgan failure and 29 survived without manifestation of organ dysfunction.

Radioimmunoassays for human PSTI and HUSI-II have been developed in our institute. They allow the determination of these two polypeptides in plasma in concentrations as low as 0.2 µg/l. A radioimmunoassay for neopterin is commercially

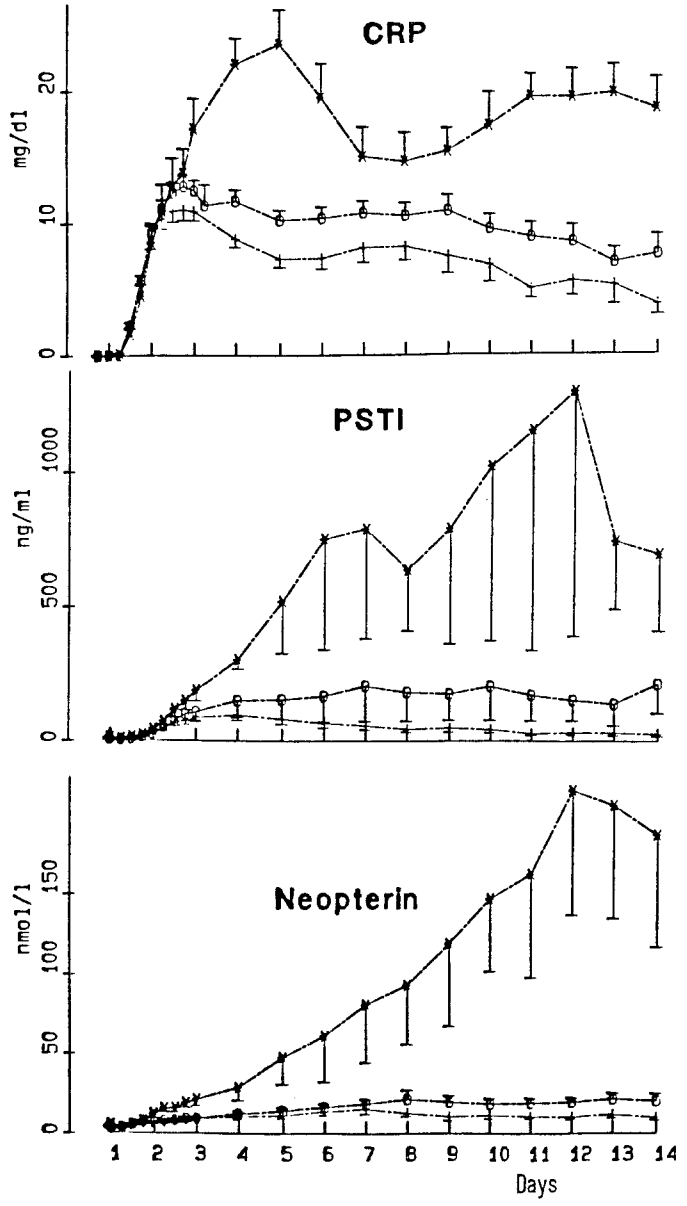

Fig. 1. Time course of C-reactive protein (CRP), pancreatic secretory trypsin inhibitor (PSTI), and neopterin (mean ± SEM) in polytraumatized patients. The patients were subdivided in three groups: letal multiorgan failure (n = 11) ×----×; reversible multiorgan failure (n = 29) ○----○; no organ failure (n = 29), +----+

available (Henning, Berlin). C-Reactive protein (CRP) was determined by an immunodiffusion assay (Behring-Werke AG, Marburg).

Results and discussion

Several of the specific and unspecific inflammation parameters of humoral and cellular systems investigated in this study allowed a discrimination of patients with lethal outcome and those who survived with and without multiorgan failure, respectively. The plasma concentrations of neopterin (endproduct of the macrophage GTP metabolism [3], CRP and PSTI permitted already on the third posttraumatic day a discrimination of surviving and not surviving patients (mean survival time: 16 d, range: 6–28 d).

The plasma concentration of PSTI in healthy persons is about 6 µg/l and that of HUSI-II about 0.3 µg/l which is close to the lower limit of detection in the routine radioimmunoassay. In plasma of polytrauma patients PSTI levels rise almost steadily from day 2 to day 12 post trauma. This delayed increase is similar to that of neopterin and somewhat different from the steeper increase of the acute phase reactant CRP (Fig. 1). The highest PSTI concentration observed in our study was 1.5 mg/l.

In contrast to PSTI no change in the concentration of HUSI-II in the plasma of patients with polytrauma could be detected.

Concluding from its comparatively high plasma concentration and the significant increase after severe injury, PSTI seems to have other, not yet understood functions in addition to its role to prevent autoactivation of trypsinogen. In contrast, the low plasma concentration of HUSI-II and the fact that no concentration increase is observed in polytrauma patients indicate that HUSI-II has not specific function under acute phase conditions. It may be deduced further that the physiological role of HUSI-II is restricted to the reproductive system where it has to prevent deleterious proteolysis by acrosin.

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