



### Effect of Cernilton on Platelet Aggregation In Vivo

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The finding that platelets contain a mitogen for arterial smooth muscle cells [7] has provided a major mechanism for the concept that platelets may be causal agents in atherogenesis. The platelets of arterial blood have been depicted as significant factors in early atherogenesis as well as in late thrombotic complications of advanced atherosclerosis.

Antiplatelet agents are able to suppress the increased platelet aggregation and endothelial cell loss as well as the intimal lesions caused by induced homocystinemia [6].

Our earlier preliminary studies indicated to the possibility of decreased platelet aggregation, especially in vitro, under the influence of Cernitin [8]. The purpose of this investigation is to check the antiplatelet activity of Cernilton in vivo.

#### Materials and Methods

Twenty healthy subjects, ten women and ten men, 39 to 56 [mean 45] years old were studied. Subjects had normal medical histories, physical examinations, screening blood chemistries, blood counts and urinalyses. Each subject was fully informed of the nature of the studies.

Cernilton [AB Cernelle, Vegeholm] was given orally 2 tablets three times daily before meals over 2 weeks.

Platelet aggregation as well as blood serum lipids level was measured in all the examined subjects.

The platelet aggregation was tested using an Elvi 840 apparatus with the method of Born [2].

55  $\mu\text{mol}$  solution of ADP was added to the platelet rich plasma.

Total lipids were determined according to Zöllner and Kirsch [10], triglycerides by the method of Eggstein and Kreutz [4] and total cholesterol after Blaszczyzyn [1]. The data reported were based on a comparison between the results obtained on entry into the trial, after 1 week and after 2 weeks of treatment with Cernilton.

Student's t-test was used for comparing differences between means.

#### Results

Platelet aggregation [Table 1] expressed by means of threshold of aggregation as well as by means of speed of aggregation was diminished in subjects receiving Cernilton. Threshold after one week was practically unchanged, however after two weeks of treatment with Cernilton it was increased by 82% as compared with initial value, the difference being statistically significant [ $p < 0.02$ ]. Speed aggregation was significantly diminished both in the first phase and in the second phase of aggregation.

The effect of Cernilton on serum lipid fractions is summarized in Table 2.

Total lipids level was lowered insignificantly – after 1 week by 11% and after 2 weeks by 18%. Triglycerides concentration in subjects receiving Cernilton was decreased by 18%. Triglycerides concentration in subjects receiving Cernilton was decreased by 18% after 1 week and by 35% after 2 weeks of management. Both differences were statistically significant. Diminution of the total cholesterol level was also observed. It was decreased by 25% after 2 weeks of Cernilton administration in comparison with the initial value.

## Discussion

Platelet is reported as playing important roles in cardiovascular diseases. Yamazaki et al. [9] demonstrated hyperaggregable platelets in patients with coronary artery disease, and Frishman et al. [5] proved that platelet aggregation threshold in response to ADP and epinephrine was increased in patients with angina pectoris.

Platelets initiate thrombosis by aggregating at the site of previous vascular injury and it is speculated that altered platelet aggregability may play a significant role in the development and progression of atherosclerotic lesions [3].

The results of this trial show that Cernilton in clinically acceptable doses decreases the platelet aggregation significantly, illustrating the importance of investigating the effect of a drug in vivo.

The present study also clearly indicates that Cernilton is able to affect lipid concentration in the blood serum, even in the cases revealing normal values.

Both factors – platelet aggregation as well as lipid metabolism disturbances are of fundamental importance for development of atherosclerosis and ischemic heart disease. Therefore, therapeutic implications of the obtained results under the influence of Cernilton should be considered and discussed.

## Conclusion

Preventive and therapeutic significance of Cernilton for atherosclerosis and ischemic heart disease should be taken into account.

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Table 1. Platelet aggregation [mean ± SE].

Period of examination	Threshold of aggregation [umol]	Aggregation speed [deprees]	
		In I phase	In II phase
Initial value [I]	1.13 ± 0.13	69.5 ± 1.95	41.5 ± 3.16
After 1 week [II]	1.15 ± 0.14	59.3 ± 2.44	33.2 ± 1.60
After 2 weeks [III]	2.06 ± 0.34	53.4 ± 3.25	28.8 ± 1.70
P	I/II	>0,5	<0,005
	I/III	<0,02	<0,001
	II/III	<0,02	>0,1

Table 2. Effect of Cernilton on serum liquid fractions [mean ± SE].

Period of examination	Total lipids [g/l]	Triglycerides [mmol/l]	Total cholesterol [mmol]
Initial value [I]	8.40 ± 0.88	3.39 ± 0.25	7.26 ± 0.23
After 1 week [II]	7.50 ± 0.58	2.80 ± 0.50	6.32 ± 0.42
After 2 weeks [III]	6.86 ± 0.68	2.20 ± 0.21	5.49 ± 0.11
P	I/II	>0,2	>0,2
	I/III	>0,1	<0,05
	II/III	>0,2	>0,5