observation further emphasizes the functional[1] and structural[2] similarities between motile and muscular cells. Because of these analogies, cells with motile activity could be a suitable system to investigate abnormalities affecting the muscular fibre, such as Duchenne muscular dystrophy.

In our investigations, however, no differences between creatine phosphokinase from leukocytes of normal and dystrophic subjects has been found. The specific activity of the enzyme, the *Km* at different pH, the pH optimum, and the resistance to heat denaturation were similar in both cases, thus indicating that dystrophic leukocytes contain normal amounts of normal creatine phosphokinase molecules.

This last point seems to exclude the leaking of creatine phosphokinase from the leukocytes, at variance with the dystrophic muscle[11–13]. Thus the postulated alteration of membrane permeability to creatine phosphokinase seems to be restricted to the dystrophic muscular fibre and not extended to the leukocyte. Similarly, the recently reported structural alteration of the creatine molecule in human dystrophy[14] seems to be restricted to the muscle fibre. Muscular and leukocyte phosphokinases must therefore be under independent genetic control[15].

Riassunto. Nei leucociti polimorfonucleati umani, cellule capaci di movimenti durante la fagocitosi, è stata osservata la presenza dell'enzima creatina fosfocinasi. Questa è un'altra analogia tra cellule fornite di movimenti e fibre muscolari. Il confronto di alcune caratteristiche dell'enzima isolato da leucociti provenienti da soggetti sani e distrofici non rivela alcuna differenza significativa.

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Copper and Zinc Levels in the Blood Serum and Urine of Bilharzial Hepatic Fibrosis

In our previous work on bilharziasis, the urinary porphyrins, uro and coproporphyrins[1], as well as the urinary amino acids[2], were found to be excreted in large amount, expecially in advanced cases of hepato splenomegaly patients. Moreover, it appears from the work of WATSON and SCHWARTZ[3] that the urinary as well as feacal porphyrins uro- and coproporphyrins are excreted as zinc complexes. While working on rats, VAN CAMPEN et al.[4] found that there is an interrelationship between zinc concentration and copper absorption. Therefore, it appears of interest to study the serum and urinary level of both zinc and copper in bilharzial hepatic fibrosis, and to compare with the results obtained from normal healthy individuals.

Materials and methods. 25 patients, 17 adult males and 8 adult females, in different stages of bilharzial hepatic fibrosis were selected for this study. These cases were subjected to thorough clinical examination, liver function tests, total protein as well as differential blood serum proteins were estimated by electrophoresis. Serum and urinary zinc and copper levels were determined in the patients studied and compared with the results obtained from 10 normal individuals.

Determination of serum and urinary zinc and copper was performed according to the method of WILLIS[5], using the atomic absorption spectrophotometer SP 90 A.

Results. The Table indicates the range and average values of serum and urinary zinc and copper of the studied bilharzial patients, compared with the results obtained from 10 normal healthy individuals. Normal serum zinc ranges from 105 to 208 and the average found was 185 µg/100 ml. In urine the range lies between 0.185 and 0.610 mg/24 h, while the mean urinary zinc recorded was 0.400 mg/24 h. Normal Serum copper, however, ranges from 65–190 µg/100 ml and the mean value 125 µg/100 ml. Urinary copper, however, varied from traces to 0.027 with a mean value of 0.016 mg per day.

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Range and average values of urinary and blood serum zinc and copper of normal controls and patients studied

Cases	Zinc		Copper	
	Serum (µg/100 ml)	Urine (mg/24 h)	Serum (µg/100 ml)	Urine (mg/24 h)
Normal control				
Range	105–208	0.185–0.610	65–190	0.000–0.027
Mean ± S.E.	185 ± 9	0.400 ± 0.05	125 ± 15	0.016 ± 0.002
Bilharzia hepatosplenomegaly				
Range	112–890	0.200–1.800	90–800	0.000–0.070
Mean ± S.E.	341 ± 38	1.113 ± 0.2	338 ± 31	0.021 ± 0.003

In bilharzia patients, the range of serum zinc lies between 112 and 890 with a mean value of 341 µg/100 ml, that is, there is an increase of about 84% more than the normal mean. The zinc level in urine of bilharzia patients (1.113 mg/24 h) is markedly increased. The increase is about 3 times the normal mean (0.4 mg/24 h).

As shown in the Table, serum copper of bilharzia patients ranges between 90 and 800 µg/100 ml with a mean value of 338 µg/100 ml which is about 3 times the average normal serum copper, (125 µg/100 ml). However, for copper level in urine of the patients, only 4 cases showed an increased amount, the remaining cases did not show any significant change from the normal maximum level of urinary copper.

Discussion. The main pathway of zinc excretion appears to be by the faeces, little is normally excreted by way of the urine. Zinc excretion in urine has been studied recently by some investigators. VALLEE et al.[6] reported that 457 µg of zinc per day was lost in urine in normal adult, while PRASAD et al.[7] found in normal adult urine an amount of 447 µg of zinc per g of creatinine. In this investigation, the average amount of urinary zinc obtained from 10 normal subjects (7 adult males and 3 females) was found equal to 0.4 mg per 24 h (Table).

According to MCCANE and WIDDOWSON[8], the amount of urinary zinc does not vary appreciably with the dietary levels of zinc. However, a significant zincuria may occur in certain pathologic conditions such as nephrosis, post alcoholic hepatic cirrhosis and certain types of porphyria patients[3].

In this investigation, the average amount of zinc excreted in the urine of hepato-splenomegaly bilharziasis was 1.113 mg zinc per day which is about 3 times the normal mean. This increase was found to be statistically significant, $p < 0.005$. This is in agreement with the finding of VALLEE et al.[9], who reported a mean value of 1.03 mg of zinc per day excreted in the urine of patients with liver cirrhosis. In the normal plasma, however, zinc was found to be at a concentration of 185 µg/100 ml which is near the value found by VIKBLADH[10] and KOCH[11], who found a normal mean value of 124 and 120 µg/100 ml respectively, while PRASAD[7] and BERFANSTAN[12] reported it as 102 and 110 µg/100 ml. The average serum zinc found in bilharziasis was 341 µg/100 ml. Compared with our normal average serum zinc, 185 µg/100 ml, there is an increase of about 84%, which is statistically significant, $p < 0.001$.

The universal distribution of copper suggested that this element participates in life processes as catalyst[13], and it was found to be a component of a variety of oxidative enzymes. Moreover, copper is stored in the liver[14] particularly in the parenchymal cells. This fact also encouraged us to estimate its level in bilharzial hepatic fibrosis.

As shown in the Table, the mean value of serum copper of the patients studied was found to be 338 µg/100 ml: a concentration which is about 3 times the normal mean of 125%. The difference is statistically significant, $p < 0.001$. However, no significant changes occured in the urinary copper of most of the patients. Serum copper found in our normal control, 125 µg/100 ml, agreed with the finding of WILLIAMS[15] and of LAHEY et al.[16] who found also an increase in the plasma copper during subacute chronic infections. The normal average urinary copper 0.016 mg/day is near that found by BUTLER and NEWMAN[17], who estimated it equal to 0.018 mg/day.

As regards the protein fractions, the α_2-globulin is also increased in most of the patients studied. This increase was found to correlate with serum copper content. In human beings 5–7% of the plasma copper is diffusible and present as free ion which is in equilibrium with and loosely attached to albumin[18]. The remaining 93–95% of plasma copper is carried with α_2-globulin. The rise of α_2-globulin is associated with chronic liver damage, as occurs in the bilharzial hepatic cirrhosis. Also the serum copper is increased with all conditions in which there is chronic tissue damage as tuberculosis and liver cirrhosis[16].

Conclusion. 25 cases of bilharzia hepatic-splenomegaly were clinically examined and biochemically studied for their blood and urinary zinc and copper contents. The following conclusions may be drawn: 1. A marked increase in urinary zinc excretion appeared, but the increase is less in serum zinc. 2. The increase of serum copper correlate with the increase of α_2-globulin and parallel the severity of liver damage. 3. No significant change in urinary copper excretion was observed.

Résumé. On a étudié le contenu en cuivre et en zinc du sérum et de l'urine des malades infectés par la bilharzie, la plupart avec cirrhose du foie. On a trouvé une augmentation notable du zinc et du cuivre dans le sérum et une augmentation élevée du zinc dans l'urine.

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Einfluss von Silymarin auf Keimung und embryonales Wachstum von Gartenkresse (*Lepidium sativum* L.)

Influence of Silymarin on Germination and Embryonic Growth of Garden Cress (*Lepidium sativum* L.)

Mit dem Sammelnamen «Silymarin» bezeichnet man nach WAGNER[1] die Gesamtheit der wirksamen Inhaltsstoffe der Droge Fructus Cardui Mariae[2], der Früchte der Mariendistel (*Silybum marianum* Gaertn.). Dieser Wirkstoffkomplex zeichnet sich nach den Befunden von VOGEL et al.[3,4] durch eine ausgeprägte antihepatotoxische Wirkung aus.

Um die Isolierung und Strukturaufklärung der wirk-