Table II. RNA concentration in the liver ($n = 5$)

Age (days)	RNA concentration (μg/100 mg of tissue)		Significancy of intergroup differences (age of the quail in days)									
	Mean ± S.E.	C.V. (%)	0	7	14	21	28	35	42	49	56	63
0	12.84 ± 0.69	10.74										
7	16.10 ± 0.82	10.18	+									
14	19.66 ± 0.96	9.76	+++	+								
21	21.22 ± 0.36	3.34	+++	++	−							
28	33.63 ± 1.15	6.80	+++	+++	+++	+++						
35	50.86 ± 4.11	16.15	+++	+++	+++	+++	++					
42	67.82 ± 1.70	4.99	+++	+++	+++	+++	+++	++				
49	57.22 ± 5.95	20.78	+++	+++	+++	+++	++	−	−			
56	54.76 ± 7.80	28.45	+++	+++	++	++	+	−	−	−		
63	55.74 ± 2.66	9.62	+++	+++	+++	+++	+++	−	++	−	−	
70	56.88 ± 1.60	5.62	+++	+++	+++	+++	+++	−	++	−	−	−
G-test			significant									

n = number of animals in a group; +, $0.05 > P < 0.01$; ++, $0.01 > P < 0.001$; +++, $P < 0.001$; −, $P > 0.05$.

tion (Table II) increases progressively from the time of hatching up to the age of 42 days. This is then followed by a marked decrease lasting until the 56th day of age and subsequently a period of stabilization. The increase in RNA concentration is probably correlated with animal growth and the growth of the liver or, respectively, the increase is related to the intensity of protein synthesis by which this growth is mediated. The relatively low decrease of RNA concentration after the 42nd day may be caused by a lower level of protein synthesis in the retarded phase of growth. On the other hand, the stabilization of RNA concentration, which is on a relatively high level after the 56th day may be related to egg-laying which was present in the quail. It is also to be pointed out that RNA variability is considerably higher than DNA variability and that large and significant differences are also observed among the individual age groups. It can be presumed that a high variability of RNA concentration is closely related to the variability in growth intensity, as well as to the degree of egg laying.

In this study we have confirmed the observation that DNA concentration under normal conditions does not change and is relatively stable, even after initiation of egg-laying[2]. This contradicts the results of DAWSON[6], who reported that between the first and the third month of age there occurs a rapid decrease in DNA concentration in the liver of the rats. Regarding RNA concentration and its variability, our resulte confirm those previously reported[1–5].

Zusammenfassung. Während des Wachstums der japanischen Wachtel, *Coturnix coturnix japonica*, ändert die DNA-Konzentration der Leber nicht; die RNA-Konzentration ist hingegen erhöht. Die letztere ist mit der Eiweissynthese bei Wachstum und Eiproduktion korreliert.

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Decrease of Arylsulfatase A Activity in Plasma in Hemorrhagic Shock

Elevation of plasma lysosomal enzyme activity in shock is known since JANOFF's studies[1]. In hemorrhagic shock, labilization of lysosomes was first observed by BITENSKY[2]. In the past decade, changes in the level of lysosomal enzymes in shock have been studied by several authors[3–8].

In the course of our hemorrhagic shock experiments, together with other lysosomal enzymes we have measured the activity of arylsulfatase A (ASA) and have found that its activity decreases rather than increases during hypotension. To our knowledge, no decrease of any other lysosomal enzyme activity in shock has yet been reported.

The experiments were performed on 52 dogs of both sexes (mean body weight: 11 kg). In sodium pentobarbital anesthesia, polyethylene cannulae weer introduced into both femoral arteries and a femoral vein for the purpose of bleeding, blood pressure measurement, sampling and reinfusion. The animals were given 500 U of heparin per kg body weight. After a 30 min control period, bleeding was begun from the femoral artery into a glass reservoir and mean arterial blood pressure was lowered to 40 mm Hg. Blood pressure was maintained at this level for 90 min

[1] A. JANOFF, G. WEISSMANN, B. W. ZWEIFACH and L. THOMAS, J. exp. Med. *116*, 451 (1962).
[2] L. BITENSKY, J. CHAYEN, G. J. CUNNINGHAM and J. FINE, Nature Lond. *199*, 493 (1963).
[3] A. E. DUMONT and G. WEISSMANN, Nature, Lond. *201*, 1231 (1964).
[4] N. G. SUTHERLAND, G. BOUNOUS and F. N. GURD, J. Trauma *8*, 350 (1968).
[5] T. BARANKAY, G. HORPÁCSY, S. NAGY and G. PETRI, Medicina exp. *19*, 267 (1969).
[6] G. HORPÁCSY, S. NAGY, T. BARANKAY, T. TÁRNOKY and G. PETRI, Enzymologia biol. clin. *11*, 324 (1970).
[7] T. M. GLENN and A. M. LEFER, Circulation Res. *27*, 783 (1970).
[8] P. E. FREDLUND, P. A. ÖCKERMAN and J. O. VANG, Am. J. Surg. *124*, 300 (1972).

ASA activity (mU/ml) in the plasma of dogs in hemorrhagic shock ($n = 52$)

Period	Control	Hypotension		Post-reinfusion		
Sample No.	1	2	3	4	5	6
Mean	11.04	7.59	7.23	8.49	9.98	10.74
Median	9.58	6.24	6.24	7.29	8.33	8.74
20th percentile	7.20	5.04	4.99	5.12	6.08	6.58
80th percentile	13.49	9.33	8.91	9.96	12.58	14.62
P [a]		$P < 0.05$	$P < 0.01$			

[a] Probability of random occurrence of difference from the control value.

when blood from the reservoir was reinfused intravenously Following reinfusion the animals were observed for an additional 90 min. Blood samples (5 ml) were taken in the control period, in the 10th and 80th min of the hypotensive period and 30, 60 and 90 min after the end of reinfusion. ASA activity was determined in plasma by the method of BAUM, DODGSON and SPENCER[9] using *p*-nitrochatechol sulphate in acetate buffer, pH 5, as substrate and an incubation period of 180 min. Statistical analysis was done by the method of WILCOXON[10]. Percentiles[11] were used to estimate variation.

Results are summarized in the Table. It can be seen that ASA activity in plasma shows a decreasing tendency already at the beginning of the hypotensive period, and is significantly lower than control values by the end of the period. The low ASA activity begins to rise somewhat after reinfusion and reaches approximately control values at the end of the experiment.

We can offer the following two hypotheses for the interpretation of these results: 1. Release of ASA occurs from organs the perfusion of which is severely diminished during shock, and thus lesser amounts of enzyme can reach the general circulation. 2. During shock, inhibitors are released which decrease the activity of ASA in plasma. We have devised and commenced experiments in an attempt to study these possibilities.

Zusammenfassung. Bei 52 Hunden wurde in einem 90 Minuten dauernden Blutungs-Schock mit einem Blutdruck von 40 mm Hg eine 30%ige Verminderung der Aktivität der Arylsulfatase A im Plasma beobachtet.

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10 July 1973.

[9] H. BAUM, R. S. DODGSON and B. SPENCER, Clin. chim. Acta *4*, 453 (1959).
[10] F. WILCOXON, S. KATTI and R. A. WILCOX, *Critical Values and Probability Levels for the Wilcoxon Rank Sum Test and the Wilcoxon Signed Rank Test* (American Cyanamid Company and Florida State University, New York, Tallahassee 1963).
[11] L. HERRERA, J. lab. clin. Med. *52*, 34 (1958).

Optical Diffraction Studies on Stimulated Single Fibres of Frog Muscle (*Hyla caerulea*)

When striated muscle is stimulated, the actin and myosin filaments slide together producing a contraction of several percent in each of the sarcomeres, the repeating structural units about 2 μm long which contain the filamentary mechanism. Because of the regularity of these sarcomeres the muscle acts upon an incident beam of light as a diffraction grating with a spacing equal to the length of 1 sarcomere. Observations of changes in the diffraction pattern thus enable changes in sarcomere length to be followed during muscular action[1-4].

By direct ciné-photography of the diffraction pattern during tetanus CLEWORTH and EDMAN[2,3] investigated the presence of the fluctuations in the sarcomere length which had been reported by LARSON et al.[4] and by GOLDSPINK et al.[5]. We have employed the same technique to determine the rate of filament sliding during an isometric contraction and to elucidate the nature of sarcomere length changes during a twitch.

Methods. Single fibres were dissected from the dorsal part of the semitendinosus muscle of a tree climbing frog (*Hyla caerulea*) and mounted horizontally (between a tension-transducer and a rigid arm) in frog Ringer's solution (NaCl, 115 mM; KCl, 2.5 mM; $CaCl_2$, 1.8 mM; Na_2HPO_4, 2.15 mM; NaH_2PO_4, 0.85 mM; pH 6.9) at 7 °C. A laser beam was directed upon the fibre at right angles to its axis and the zero and 1st order diffraction lines were displayed on a screen about 40 cm away. A moving-film camera focussed on the screen provided a continuous record of the spacing between the diffraction orders during activation. The fibre was stimulated transversely by a pair of platinum wire electrodes (0.007″ diameter) located immediately adjacent to the laser beam, so that the illuminated sarcomeres were the first to contract. A single 200 μ sec supramaximal pulse or a train of such pulses at an appropriate frequency was used to produce a twitch or a tetanus as required. The stimulating pulses were recorded on the film by including a synchronized light-emitting diode in the field of view (as seen in the Figure).

Results. Trace A in the Figure illustrates the typical sarcomere response in tetanus obtained from a region located midway along the length of a muscle. The resting sarcomere length was 2.6 μm and the temperature 7 °C.

[1] A. SANDOW, J. cell. comp. Physiol. *9*, 37 (1936).
[2] D. CLEWORTH and K. A. P. EDMAN, Science *163*, 296 (1969).
[3] D. CLEWORTH and K. A. P. EDMAN, J. Physiol., Lond. *227*, 1 (1972).
[4] R. E. LARSON, M. J. KUSHMERICK, D. H. HAYNES and R. E. DAVIES, Biophys. J. *8*, MA4 (1968).
[5] G. GOLDSPINK, R. E. LARSON, R. E. DAVIES, Experientia *26*, 16 (1970).