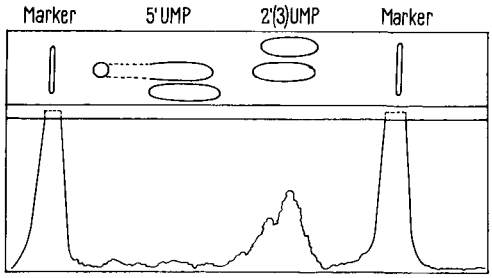
acid-ammonia[11] and isopropanol-water-ammonia[12] as developing systems. For each solvent the running time was 3 h. The spots localized by UV-illuminations were scraped off, suspended in scintillation fluid and counted. The scintillation fluid had the following components: toluene 500 ml, dioxane 500 ml, methanol 300 ml, PPO 6.0 g, POPOP 0.150 g, naphthalene 100 g, plus 4% Cab-o-sil.

Nucleotide composition

Substance	By UV	By ^{32}P distribution			
	Column	Column		Thin layer chromatography	
	%	Counts per min	% of total	Counts per min	% of total
2′(3′)-CMP	33.3	159	7.0	15.5	12
2′(3′)-GMP	30.8	200	8.8	11.5	9
2′(3′)-UMP	17.2	1735	75.7	92.0	73
2′(3′)-AMP	18.7	194	8.5	7.5	6
Recovery		2288	92.5	126.5	91

CMP = cytidine-monophosphoric acid; GMP = guanosine-monophosphoric acid; UMP = uridine-monophosphoric acid; AMP = adenosine-monophosphoric acid.



Radiochromatogram of the UMP fraction. Chromatography on Whatman No. 3 mm paper. Developing solvent, 6:3:1 absolute ethanol-2% (w/v) H_3BO_3-NH_4OH (density 0.9)[10]. Bottom: reproduction of a radiochromatogram of 2′(3′)-UMP; full-scale deflection = 1000 counts per min. Top: reproduction of the separation of two authentic samples of 5′-UMP and 2′(3′)-UMP.

In addition, the uridylate fractions were chromatographed on paper with 0.5 μmoles authentic uridine-5′-monophosphoric acid (5′-UMP) using the borate system[13], which separates 2′(3′)-nucleotides from 5′-nucleotides.

Results and conclusions. The ^{32}P distribution in the alkaline hydrolysate of the labelled RNA extracted is shown in the Table. 75% of the ^{32}P counts were found in 2′(3′)-UMP. This is confirmed by the results of the paper chromatography shown in the Figure. The incorporation is mostly not terminal. In fact, when UTP labelled in the pyrimidine ring was used as precursor, only 29% of the radioactivity was found associated with the nucleoside. It is concluded that a subcellular fraction which apparently contains no DNA and probably derives from the microsomes is able to incorporate uridylate into RNA when UTP is the precursor. The incorporation is largely not terminal and not random. In some aspects, the system described here is similar to the pigeon liver microsome system of STRAUS and GOLDWASSER[13]. It would be interesting to determine whether the peculiarity of the product is due to a specific mechanism at work in vivo or to an artefact due to the extraction of the enzymatic activity from the cell.

Riassunto. Una frazione subcellulare preparata dalla corteccia cerebrale del vitello è capace di catalizzare l'incorporazione di uridilato nell'acido ribonucleico (RNA) quando UTP è usato come precursore. L'acido ribonucleico sintetizzato risulta costituito per il 75% di residui uridilici di cui soltanto il 29% risulta terminale.

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Attack Elicited by Forebrain and Hypothalamic Stimulation in the Chicken

Defensive reactions, fear and rage have been provoked in mammalian species[1], including man[2], by stimulating brain structures extending from the amygdala to the mesencephalon. Directed attack has been elicited in cats by stimulation of the amygdala[3], the hypothalamus[4–6], the medial thalamus[7] and the mesencephalon[5]. In birds, attack has been elicited by stimulating the hypothalamus and the preoptic area of the pigeon[8] and the brainstem of the chicken[9]. The location of the electrodes is not specified in the aforementioned papers. In ducks attack against

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nearby birds has been caused by stimulation of the nucleus mesencephalicus dorsalis lateralis[10].

The present experiments were performed on 29 adult White Leghorns: 14 hens, 15 cocks. In all 159 sites in various parts of the basal forebrain and the diencephalon were stimulated. Only points giving a directed attack response are dealt with in this report.

Steel electrodes with non-insulated tips of 0.2–0.4 mm were implanted stereotaxically with the aid of an atlas[11]. The stimulation was begun 1–2 days after the operation. The birds were connected to the stimulator by a flexible cable which allowed them freedom of movement in the experimental cage. Unstimulated chicken were used to study the evoked social reactions. Monopolar stimulation was used. The indifferent electrode was a steel screw in the supraorbital skull. The stimulus consisted of negative monophasic square waves at a frequency of 50 pulses/sec, and a pulse duration of 2 msec. The stimulus train varied from about 5 sec to some minutes. The milliamperage of the stimulating (peak) current was monitored on an oscilloscope. The attack thresholds ranged from 0.05–0.35 mA at different sites. The electrode positions were histologically verified by studying serial frontal sections stained alternately for nerve cells and myelinated fibres.

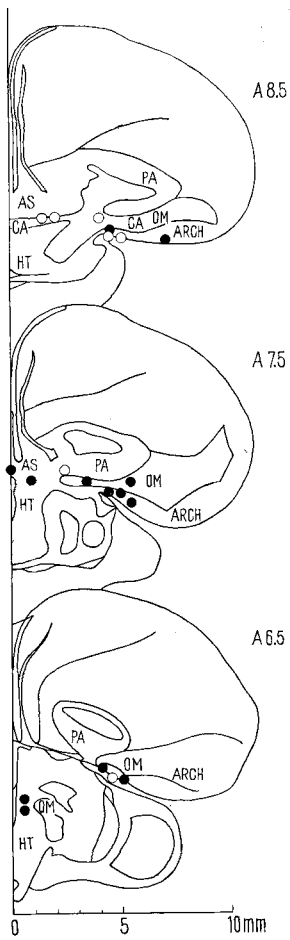

Fig. 1. Brain sites giving rise to attack regularly (●) or occasionally (o) on repeated stimulation. They are plotted (combining the points from the left and right sides) on frontal sections beginning at the anterior commissure (A 8.5) and continuing posteriorly at 1 mm intervals. Abbreviations: ARCH, archistriatum; AS, area septalis; CA, commissura anterior; HT, hypothalamus; OM, tractus occipitomesencephalicus; PA, paleostriatum augmentatum.

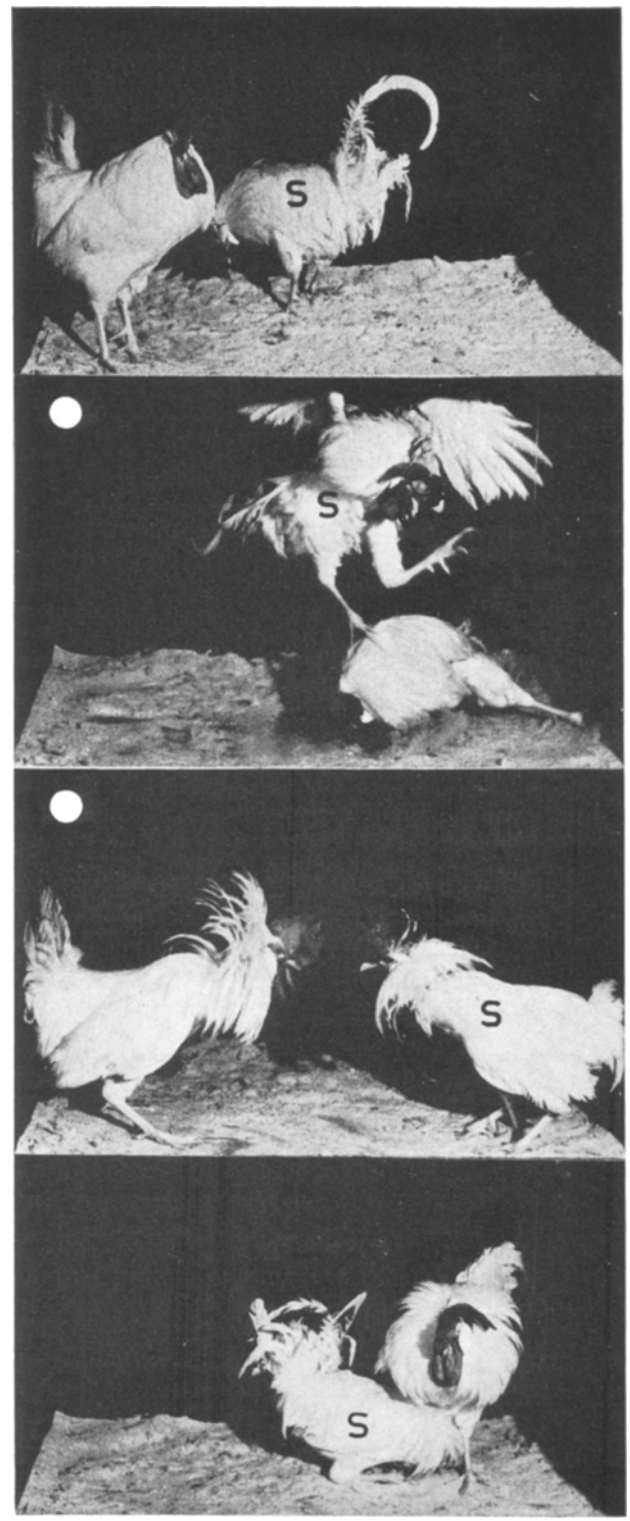

Fig. 2. A photographic series of a fight elicited by stimulating (at 0.1 mA) the medial archistriatum of the smaller cock (s). When stimulated (white dots) the cock attacks a stronger companion and a fight begins. Note the threat postures with ruffled collars. When stimulation is terminated, the smaller cock hides its head submissively under the body of its recent opponent.

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Directed attack and intra-species fights were induced by stimulating 20 brain sites in 14 chicken. This response was obtained from the medial archistriatal region, including the nucleus taeniae, the tractus occipito-mesencephalicus, the area septalis and the hypothalamus. In Figure 1 the active points are plotted on frontal sections of the stereotaxic atlas. In 13 of these the attack was obtained regularly, and in 7 cases actual attacking occurred only occasionally but was observed more than once. A characteristic threat display preceded or accompanied the attacks in most cases (Figure 2). Aggression infrequently overlasted the stimulation for some minutes. Truly spontaneous fights were not seen. In the majority of the cases, circling to the non-stimulated side occurred during the threat display. Vocalizations of varying intensity were noted in about half of the responses. At three electrode sites the aggressive response pattern was intermingled with behaviour suggesting fear and escape tendencies.

Of the 20 attack responses, only 3 were obtained from hens. In them all the active points were within 1.5 mm of the midline, and no attacks were elicited from the archistriatal region. This difference between the sexes might depend on hormonal factors, since cerebral implants of androgen into capons have been reported to augment aggressive behaviour, when implanted 'in and ventral to the paleostriatum'[12]. This definition of the hormone-sensitive area seems to overlap partly with the lateral points of my material, though electrical stimulation of the paleostriatum did not cause attack, except at the borderline of the occipito-mesencephalic tract.

Lesions in the ventral medial archistriatum have been reported to have a taming effect on wild ducks and electrical stimulation of the same region to cause mainly escape behaviour[10].

The avian archistriatum and the mammalian amygdala are regarded as being anatomically homologous. The occipito-mesencephalic tract, which connects the archistriatum with preoptic, hypothalamic and mesencephalic centres, forms, together with the anterior commissure, the avian stria terminalis system[13]. In the light of the presented data, the general arrangement of brain structures related to affective reactions in birds appears to correspond to that delineated for cats[14]. In view of the great phylogenetic distance and the differences in many brain structures between birds and mammals, this would suggest that the general plan for the central regulation of aggressive behaviour was laid out early during the phylogenesis of vertebrates.

Zusammenfassung. Elektrische Reizung begrenzter Stellen des medialen Archistriatums (Amygdala), Tractus occipito-mesencephalicus, Area septalis und des Hypothalamus vom Haushuhn, löst gezielten Angriff auf einen Artgenossen aus.

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Dämpfung des Kältezitterns durch Pentobarbital und Pethidin bei Ratten

Die Beeinflussung der Temperaturschwelle für das Kältezittern durch Pethidin und Pentobarbital wurde an intakten Ratten geprüft, die sich in einem Wasserbad befanden. Die Temperatur des Wasserbades konnte durch Zufuhr von kaltem und warmem Wasser kontinuierlich verändert werden. In Äthernarkose wurde eine Kanüle zur Durchführung von Injektionen in die rechte V. jugularis eingebunden; eine bipolare, chlorierte Silberdrahtelektrode wurde zur Ableitung der elektromyographischen Aktivität im linken M. gastrocnemius fixiert. Registriert wurde das Elektromyogramm und das Mechanomyogramm, das mit Hilfe eines Piezokristalles über dem Muskel abgeleitet wurde. Die Körpertemperatur wurde thermoelektrisch im Rektum gemessen.

Die Bestimmung der Temperaturschwelle, bei der ein Kältezittern durch Abkühlung ausgelöst wurde, erfolgte bei zwei Gruppen von Tieren. Die Tiere der einen Gruppe (39 Ratten) befanden sich während der Durchführung des Versuches in einer flachen Pentobarbitalnarkose (5 mg/kg i.v. halbstündlich); unter dieser Bedingung setzte das Zittern bei einem Absinken der Rektaltemperatur unter 36,0 °C ein (Tabelle). Die Tiere der anderen Gruppe (21 Ratten), die sich nach beendeter Operation (Äthernarkose) in wachem Zustand befanden, zeigten ein Kältezittern bei einer Rektaltemperatur von 36,6 °C. Pentobarbital führte demnach zu einer geringgradigen Erhöhung der Schwelle des Kältezitterns. Der Unterschied der Schwellenwerte beider Gruppen ist zwar sehr klein, liess sich jedoch mit einem $p < 0{,}05$ statistisch sichern.

Pethidin senkte in einer Dosierung von 2 mg/kg die Schwellentemperatur für das Zittern. Ein Kältezittern trat bei den wachen Tieren erst bei einer Rektaltemperatur von 35,3 °C auf, während bei den Tieren, die ausserdem

	Schwellentemperatur (°C $\pm \sigma$) des Kältezitterns		Signifikanz
	Kontrolle	Pethidin 2 mg/kg	Kontrolle-Pethidin
a) Tiere in Pentobarbitalnarkose	*36,0* $\pm$ 0,54 $n = 39$	*33,0* $\pm$ 0,95 $n = 10$	$p < 0{,}001$
b) Wache Tiere	*36,6* $\pm$ 1,73 $n = 37$	*35,3* $\pm$ 1,16 $n = 12$	$p < 0{,}01$
Signifikanz a) gegen b)	$p < 0{,}05$	$p < 0{,}001$	

n = Anzahl der Versuche