activity affected little in contrast with mammalian guanase[7]. In addition, sepiapterin could not be attacked by guanase from rat liver[8].

Both sepiapterin and isosepiapterin were deaminated by the enzyme, whereas the following pteridines were inactive as a substrate: 2-amino-4-hydroxypteridine[9], 2-amino-4-hydroxy-6-methylpteridine[9], 2-amino-4-hydroxy-6,7-dimethylpteridine[10], xanthopterin, and 2-amino-4-hydroxypteridine-6-carboxylic acid[11]. It was reported that bacterial pterin deaminase[12] has rather low substrate specificity and attacks some pteridines, including 2-amino-4-hydroxypteridine, 2-amino-4-hydroxy-6-methylpteridine and 2-amino-4-hydroxypteridine-6-carboxylic acid. From the substrate specificity, it seems likely that sepiapterin deaminase is different from the bacterial enzyme.

The enzyme exhibits its activity in the range of pH 6 to 10. The enzyme deaminates sepiapterin both under aerobic and anaerobic conditions. From the structure of sepiapterin and xanthopterin-$B_2$ together with the above fact, the deamination reaction may be formulated as follows:

$$\text{sepiapterin} + H_2O \rightleftharpoons \text{xanthopterin-}B_2 + NH_3$$

Purification and further characterization of the enzyme are being undertaken.

*Zusammenfassung.* Im Seidenspinner (*Bombyx mori*) wird ein Enzym nachgewiesen und angereichert, welches das gelbe Pigment Sepiapterin zu Xanthopterin-$B_2$ desaminiert. Ferner wird die Substratspezifität des Enzympräparates abgeklärt.

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## Primary Afferent Depolarization Produced by Vagal Visceral Afferents

Previous work[1] showed that stimulation of vagal and aortic afferents depressed laryngeal reflexes with a time course similar to that of primary afferent depolarization (PAD) in the spinal cord[2-4]. This suggested that the depression in reflex activity could be due, at least in part, to PAD produced by the visceral afferents which course through the vagus nerve. This seemed of interest because, in addition to its possible significance for the understanding of the mechanisms involved in the inhibitions produced by vagal and aortic stimulations[5-7], previous studies on PAD produced by afferent stimulation have been concerned only with the actions of somatic, i.e. muscle and cutaneous, afferents[2-4].

*Methods.* The observations were performed in 20 adult cats anaesthetized with sodium pentobarbital (35–40 mg/kg i.p.), paralysed with gallamine triethiodide and maintained by artificial respiration. The floor of the fourth ventricle was exposed by cerebellectomy, and a stimulating microelectrode was placed in the solitary tract nucleus (STN) region, 4–6 mm rostral to the obex and 1.5–2.5 mm lateral to the midline, at depths ranging from 0.7–1.2 mm from the bulb surface. Antidromic responses were monophasically recorded from the central end of the ipsilateral superior laryngeal nerve (SL), which consists of afferent fibres only[8]. Stimulating and recording electrode pairs were placed on the central end of the ipsilateral vagus and aortic nerves. Arterial blood pressure was recorded from the femoral artery.

*Results.* The antidromic SL responses produced by STN stimulation were usually increased when preceded by vagal and aortic nerve stimulation. As shown in Figure 1A and D, hyperexcitability of the SL nerve terminals resulted mainly from the activation of vagal afferents whose threshold ranged from 1.6–5.0 times that of the most excitable fibres in the nerve (T). In 5 experiments stimulus strengths of 5–10 T added a small excitability increase, and this was correlated with the appearance of an intermediate-threshold group of fibres in the vagal electrogram (Figure 1B). No changes were observed for stimulus strengths of 10 up to 50 T.

The vagal afferents producing excitability increases of the SL terminals also induced a blood-pressure fall when stimulated at 50 c/sec (Figure 1C). Both effects had about the same threshold and grew similarly with increasing stimulus strengths. It is therefore concluded that the afferent fibres producing the blood-pressure fall are the same ones producing hyperexcitability of the SL terminals.

The lowest-threshold afferent fibres in the vagus nerve seem to originate from pulmonary stretch receptors, while those with intermediate threshold are attributed to arterial pressoceptors and rapidly adapting tracheal receptors[9]. In order to test possible actions from pul-

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monary stretch receptors, the amplitude of the SL antidromic responses was observed during lung inflation. By this procedure, pulmonary stretch receptors coursing through the vagus nerve are strongly stimulated[9]. As shown in Figure 2A, the amplitude of the SL antidromic responses was increased by lung inflation, and the time course of this effect closely paralleled that of the simultaneously recorded intratracheal pressure changes. Section of both vagus nerves abolished the effects produced by inflating the lungs, leaving only a delayed excitability increase which was particularly noticeable during the return of intratracheal pressure to control values (Figure 2B). Since this delayed effect was abolished after picrotoxin injections (Figure 2C), it is suggested that activation of extravagal afferents, presumably thoracic and diaphragmatic ones[10,11], may also contribute to the observed excitability changes produced by lung inflation.

Experiments similar to that illustrated in Figure 1 showed that the low threshold aortic afferents (1.1–5 T), which are known to originate from circulatory pressoceptors, and to produce reflex hypotension[7,9], also increased the excitability of the SL afferent terminals. Intermediate and high threshold afferents, which respectively originate from aortic chemoceptors and slow conducting pressoceptors[7], do not seem to produce any excitability change.

Hyperexcitability of the SL nerve terminals was already detectable 10–12 msec after the first of the conditioning stimuli and reached its maximum between 20 and 50 msec. After this the effect gradually decayed but was still measurable at 150–200 msec intervals. The time course of the effects produced by vagal and aortic nerve stimulations was about the same and resembles that of the hyperexcitability of Ia, Ib muscle and cutaneous afferents produced by bulbar and cerebellar stimulations[4].

If, as in the spinal cord, PAD is paralleled by presynaptic inhibition[2] the results presented in this report would indicate a visceral presynaptic control of the reflex activities induced by the mucosal receptors of the larynx. Since there is no reason to believe that PAD produced by vagal and aortic afferents is restricted to the SL nerve terminals only, it is also suggested that presynaptic inhibition would participate in the reflex reduction of vasomotor and respiratory activity[12] as well as in other

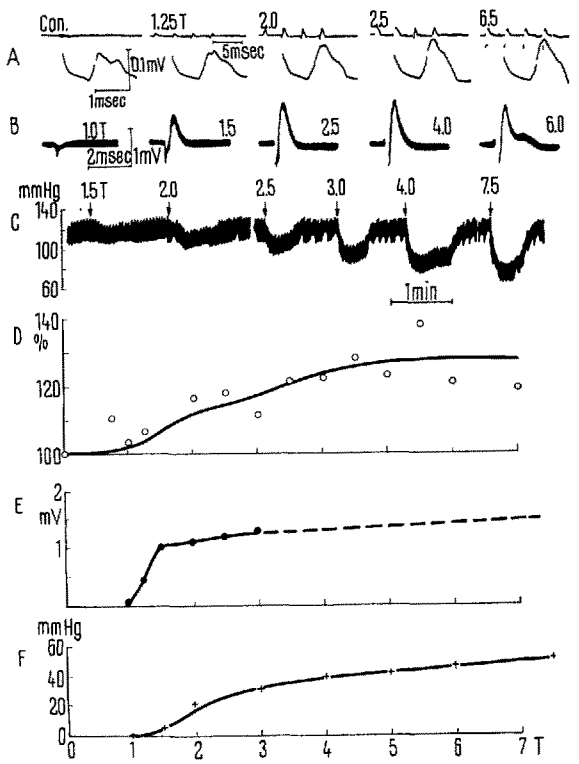


Fig. 1. Effects of vagal stimulation on antidromic SL responses and on arterial blood pressure. The lower traces in A show the antidromic SL test response produced by constant stimulation (45 V, 0.1 msec) 25 msec after the onset of a conditioning train of 4 stimuli (330/sec, 0.01 msec duration) applied to the central end of the ipsilateral vagus nerve at increasing strengths. Vagus nerve electrograms are shown by the upper traces in A and in B (at higher magnification and sweep speed, 10–20 superimposed records at 50/sec). Hypotension produced by 50/sec vagal stimulation is shown in C. In all records and graphs, strengths of vagal stimulation are expressed as times threshold (T) of the most excitable fibres in the nerve. The graphs were constructed with data partly shown in A, B, and C. D shows % change of the antidromic SL response amplitude, 100% being control values. E, amplitude of early vagal component (broken line shows estimated height where actual values could not be measured due to stimulus artifact), and F, magnitude of the blood-pressure fall. Body temperature 37 °C.

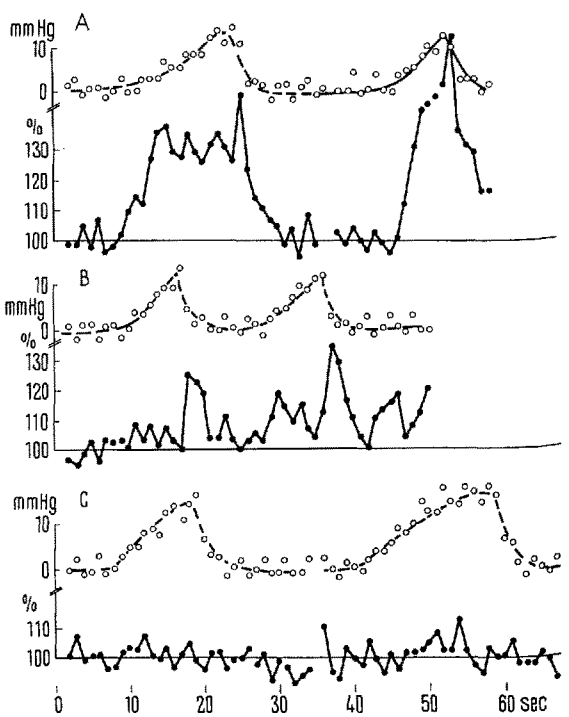


Fig. 2. Effects of lung inflation on antidromic SL nerve responses. Dots show % changes of the antidromic SL test response amplitude, 100% being control values before inflation periods. Circles show intratracheal pressure at the moment of stimulus application. A, intact animal; B, after section of both vagus nerves; and C, 20 min after 1 mg/kg i.v. picrotoxin. The SL nerve terminals were stimulated once per second with constant pulses (48 V, 0.1 msec). Body temperature 36.2 °C.

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inhibitory actions induced by stimulation of vagal and aortic afferents[5–7].

*Résumé.* La stimulation électrique des fibres afférentes vagales et aortiques les plus excitables issues des presso-récepteurs pulmonaires et artériels, augmente l'excitabilité des terminaisons du nerf laryngé supérieur dans le noyau du tractus solitaire. Des changements d'excitabilité des terminaisons afférentes furent également obtenus pendant l'activation physiologique des récepteurs vagaux sensibles aux variations de pression intrapulmonaire.

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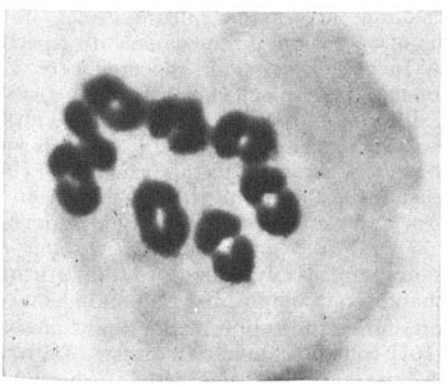
## A Nullisomic Plant in Diploid *Chrysanthemum*

*Chrysanthemum carinatum* Schousb., distinguished from other species of this group by its carinated involucral bracts, is a common garden annual and has 18 as its diploid chromosome number. Following observations of spontaneous chromosomal interchanges in many of its geographically isolated populations, interchange heterozygotes from several populations were intercrossed with a view to finding whether the interchanges present in these samples involved common chromosome pairs[1]. A monosomic plant having 17 chromosomes, and a chimeral plant having pollen mother cells with 18 and 16 chromosomes, were discovered when $F_1$ hybrids from one of these crosses were analysed cytologically for meiotic chromosomal associations[2,3]. Among the progeny of a cross involving these 2 abnormal plants, 1 plant was marked out from the others by its dwarf stature and stunted growth. This plant was found to have 16 chromosomes, forming regularly 8 bivalents in pollen mother cells (Figure), instead of a normal complement of 18 chromosomes. Meiotic process and microsporogenesis in the nullisomic were normal and the pollen grains appeared fertile as judged by the carmine-stainability test. Since this species is self-incompatible, the nullisomic plant was crossed to disomics in reciprocal crosses. None of these crosses yielded viable seeds, indicating that the deficient gametes did not function.

In addition to the genera *Prunus* and *Morus*, *Chrysanthemum* shows the highest known level of naturally occurring polyploidy with a wide range of somatic chromosome numbers extending from 18–198 (i.e. 22 *X*)[4]. Spectrum of aneuploid chromosome variation exhibited by the vegetatively propagated, more common garden forms of this genus is indeed striking[5,6]. In the species *indicum*, *rubellum* and *maximum*, for example, most of the phenotypic variations that exist are through whole chromosomes being lost and giving cytologically unbalanced progeny which survive. Equally interesting are the genetic mechanisms to promote fitness and adaptability discovered in sexually propagated diploid forms of this genus and the inherent capacity of the diploid genome to withstand extensive interchromosomal rearrangements[7,8]. The present report of a nullisomic plant in a basically diploid species is also significant, since such gross chromosome deficiencies are expected to be viable only in organisms evolved through polyploidy and having consequent duplication of whole chromosomes or parts thereof. Similar situations may possibly lead to a decrease in the basic number of chromosomes in this genus, as appears to have occurred in the genus *Crepis*. Undoubtedly, evolutionary development in the genus *Chrysanthemum* is of considerable cytogenetic interest, since such a method of evolution as that of the ornamental chrysanthemums is not yet known in any other plant[9].



Photomicrograph of a pollen mother cell from the nullisomic plant showing 8 bivalents. × 2040.

*Zusammenfassung.* Bei *Chrysanthemum carinatum* wurde eine nullosome Form während Kreuzungsversuchen entdeckt.

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