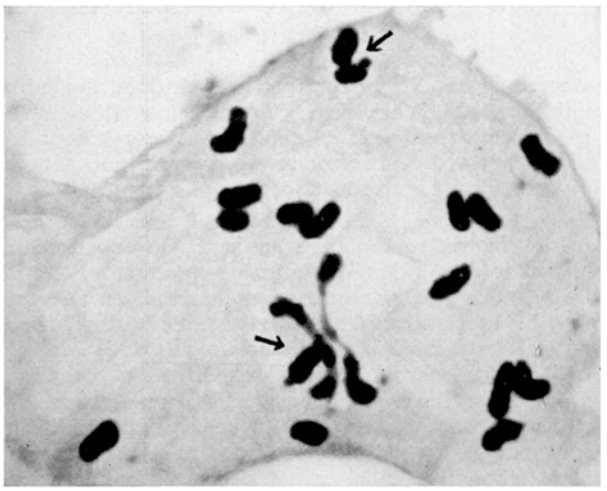
percentage of germinable pollen contained nonfunctional male gametes. The stimulus of pollination provided by such pollen might have induced the parthenogenetic development of the egg cell. The highest frequency of haploids (12 haploids among 9600 plants) occurred when P^{32} was applied to pots at the rate of 50 μc/8 lb of soil just prior to the initiation of microsporogenesis in the main tiller. PERSON[5] also found haploid plants in the progeny of the wheat variety Thatcher treated with P^{32}. The application of internal sources of radiations such as radioisotopes at a suitable stage in the reproductive development of plants may thus prove to be a promising method of inducing haploidy. The optimum dosage and stage of treatment may vary from crop to crop and would have to be worked out separately for each plant species. When X-rays or Cobalt 60 γ rays are used for irradiation, the best results may be obtained when inflorescences are irradiated at the end of microsporogenesis and prior to the completion of the mitotic divisions in the pollen grain.



PMC of a haploid N. P. 809 plant showing 17 univalents and 2 bivalents. Several univalents show s-s associations. Sat. I univalent is attached at the distal end to a bivalent. Sat. II chromosome is also marked by an arrow (magnification × 1400).

An interesting feature of meiosis in haploid plants of *T. aestivum* (technically these are 'poly-haploids') is the occurrence of side to side (s–s), end to end (e–e), and end to side (e–s) associations among univalents or univalents and bivalents besides the formation of regular bivalents. PERSON[5] concluded from a quantitative statistical analysis of the relationship between the number of bivalents and the frequency of occurrence of s–s and e–e associations, that e–e associations arise from accidents in positioning while s–s pairs are caused by chromosome homology. RILEY and CHAPMAN[6] have, on the other hand, expressed the view that s–s, e–e, and e–s associations may all have a similar origin, heterochromatic attraction being the probable causal factor. During a critical study of the types of chromosome associations found in the haploid plants obtained by us, we observed that only localized and specific regions of chromosomes are involved in these associations. Using the satellited chromosomes as markers, it was found that Sat. I chromosome (the longer of the two satellited chromosomes present in the haploid complement of bread wheat) was associated with the other univalents only at the distal region. The associations in which this chromosome was involved were thus always of the e–e or e–s type (Fig.). Sat. II chromosome was not involved in any e–e association. This type of specificity of the associated chromosome segments would support the view of RILEY and CHAPMAN[6] that heterochromatic affinity or some similar cause may be the factor governing the different types of secondary associations observed in poly-haploids of bread wheat.

We are indebted to Dr. B. P. PAL and Dr. S. M. SIKKA for their interest in this study.

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Zusammenfassung

Bestrahlung von Weizenähren (*Triticum aestivum* L. Varietät N.P. 809) mit X-Strahlen (5200 r) kurz vor dem Blühen sowie geeignete Behandlung von Topfpflanzen oder Karyopsen der gleichen Pflanze mit P^{32} oder S^{35} erzeugen haploide Formen.

Reversibility of a Facilitatory Action of Reserpine on the Central Nervous System, by Methylamphetamine

Reserpine has been shown to possess a facilitatory action on the maximal tonic-extensor seizure response evoked by electrical- or Metrazol stimulation[1]. This action can be antagonized by various anticonvulsants which augment the depressive action of Reserpine[2]. As Methylamphetamine is an effective antagonist of the sedative action of Reserpine[3], it seemed of interest to determine whether it

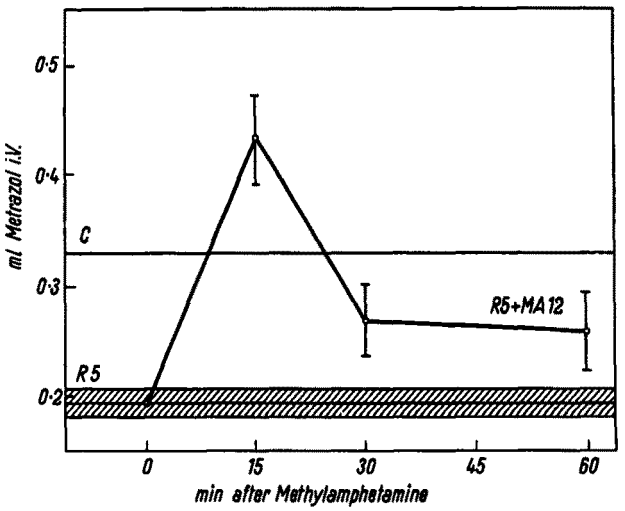

Fig. 1.—*Mean threshold (± s.e.m.) for tonic-extensor seizures on mice induced by Metrazol.* Lines parallel with abscissa indicate average of controls (*C*; $n = 39$), and threshold, 150 min after injection of Reserpin 5 mg/kg (*R5*; $n = 28$); *R5 + MA12* = Reserpin 5 mg/kg, 150 min later Methylamphetamin 12 mg/kg; at various intervals after the latter the Metrazol test followed (for each point $n = 10$). Control values indicated a double peaked frequency distribution.

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[6] R. RILEY and V. CHAPMAN, Heredity *11*, 195 (1957).

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counteracts or enhances the facilitatory action of Reserpine on Metrazol induced seizures.

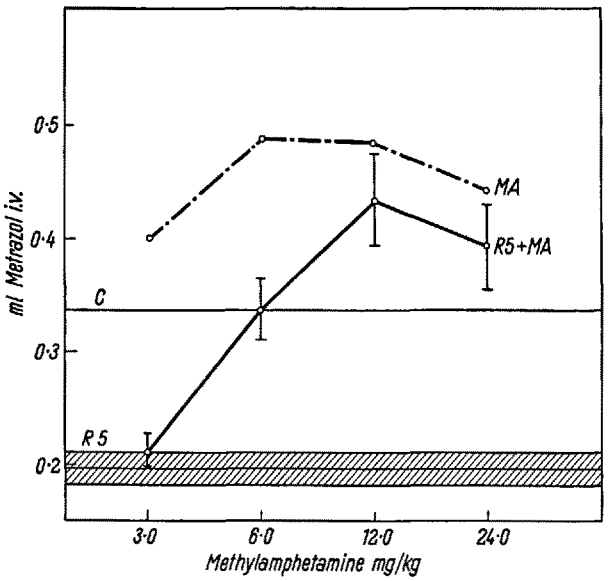

Fig. 2.—*Mean threshold (± s.e.m.) for tonic-extensor seizures on mice induced by Metrazol.* Metrazol was injected 15 min after various doses of Methylamphetamine(abscissa). *MA* = Methylamphetamine (each point, $n = 15$). *R5 + MA* = Reserpin 5 mg/kg injected 150 min before Methylamphetamine (each point, $n = 15$). *C* and *R5* same as in Figure 1. No normal frequency distribution was obtained for *C* and *MA*.

Metrazol-convulsions were induced in white mice (20–21 g) by infusion of a 0·5% solution into a tail vein at the rate of 0·05 ml every 10 sec until the occurrence of maximal tonic-extensor seizures (ORLOFF, WILLIAMS, and PFEIFFER[4]). (+)-Methylamphetamine hydrochloride was injected intraperitoneally $2^1/_2$ h after a constant dose of Reserpine (5 mg/kg intraperitoneally).

Reserpine considerably lowered the threshold for tonic extensor seizures (Fig. 1, 2). When Methylamphetamine 12 mg/kg was injected after Reserpine, the animals woke up a few minutes later from their somnolent, motionless state, and at the same time the low extensor seizure threshold was raised (Fig. 1). This action reached its maximum 15 min after the injection of Methylamphetamine. Thereafter various doses of the latter were injected after Reserpine and the Metrazol test was carried out 15 min later. Figure 2 demonstrates that Methylamphetamine in doses between 3 and 24 mg/kg raised the extensor seizure threshold of Reserpine pre-treated mice, the logarithm of the dose and the action showing approximately a linear relation within the dose range of 3 and 12 mg/kg. Methylamphetamine alone was followed by a marked elevation of seizure threshold; a complete abolition of tonic extensor seizures, as described by CHEN and BOHNER[5], could not be observed after the Methylamphetamine doses used in these experiments.

From the fact that substances like Iproniazid and Methylamphetamine both antagonize the sedative action of Reserpine[6] and abolish its CNS facilitatory action (Iproniazid[7]), it can be concluded that the state of alertness and spread of seizure discharge are closely connected.

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Zusammenfassung

Die durch Reserpin herabgesetzte Schwelle für tonische Streckkrämpfe, ausgelöst durch Metrazol (Cardiazol), konnte durch Methylamphetamin erhöht werden. Methylamphetamin ist ein wirksamer Antagonist der sedativen Wirkung des Reserpins.

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Paradoxical Effect of Quinidine upon Coronary Flow of the Isolated Heart at Different Temperatures

According to the few experimental data available, quinidine does not affect the coronary flow either in the dog heart-lung-preparation (BODO[1]), in the perfused dog heart (ELEK and KATZ[2]), or in the revived human heart perfused by the Langendorff method (KOUNTZ[3]).

In our experiments, isolated rabbit hearts perfused by the Langendorff method and subjected to experimental ventricular fibrillation (rectangular impulses of 10 ms duration and 30 Hz frequency) were used. In an other series, the hearts were allowed to beat spontaneously. The isolated hearts were perfused with warm (38°C) and cold (26°C) Locke solution alternately. In all experiments, quinidine has been administered in form of single injections of 2 mg drug in 1 ml Locke solution. Injections were given to the region of the orifice of the coronary vessels through a thin catheter placed in the aortic cannula.

At 38°C temperature, quinidine, after a possible slight increase of 30–60 s duration, consistently depressed the coronary flow in fibrillating hearts, whereas at 26°C the same doses increased it markedly. The same was found to hold for hearts beating spontaneously.

Table Ia summarises the maximal changes in the coronary flow in fibrillating hearts at different temperatures. 'Maximal flow' means the highest value of coronary flow observed during the drugs action, expressed in ml/min.

Table Ib gives the changes in the total flow during drug action. 'Total flow' indicates the amount of Locke solution in ml, flowing through the coronaries from the onset of drug action until the evanescence of the latter. 'Changes in the total flow' means the difference between the above value and the flow observed in the absence of the drug during a corresponding control period.

Table IIa and IIb represent the corresponding changes in the spontaneously beating heart.

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