Fig. 1. METAPHASE I IN THE THRLE SPECIES HYBRID, *G. barbadense* × *G. arboreum* × *G. sturtii* A C D) SHOWING 1 V, 2 IIIs, 14 IIs AND 13 Is. (× 1200).

Fig. 2. UNIVALENTS FROM TWO OTHER METAPHASE I PLATES. PROBABLE C UNIVALENTS ARE BRACKETED. (× 1,800).

World, and *sturtii* genomes, and hence *G. sturtii* may be dismissed as a possible parent in the origin of New World cottons.

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Development of Gametocytes from Extra-erythrocytic Forms in *Plasmodium gallinaceum*

It was shown in a previous paper¹ that if fowls inoculated with emulsions of organs containing extra-erythrocytic forms of *Plasmodium gallinaceum* are subjected to intense and continuous quinine treatment (150 gm./kgm. body weight quinine hydrochloride daily), they eventually become heavily infected with extra-erythrocytic forms in the complete absence of erythrocytic schizogony. During quinine treatment the red cells become infected with small non-pigmented parasites derived directly from extra-erythrocytic forms. These small non-pigmented forms do not develop further unless the quinine is stopped or the dose diminished, in which case they undergo the normal cycle of development in red cells. The same results are obtained in birds subjected to

the bites of infected mosquitoes, *Aedes aegypti*, and to continued intensive quinine treatment. With the interruption of quinine treatment the parasites in the red cells, all derived from extra-erythrocytic forms, undergo normal development which can be readily followed by examining the blood at various intervals.

Discussing these findings with us, Brigadier J. A. Sinton asked whether or not gametocytes are derived from extra-erythrocytic forms.

Experiments on birds infected by the bites of *A. aegypti*, and subjected to intense quinine treatment which was stopped only after red cells were infested with small non-pigmented parasites, showed beyond all possible doubt that among the merozoites produced by extra-erythrocytic forms some invade red cells and develop directly into gametocytes. Young gametocytes can be recognized 27½ hr. after the cessation of quinine, and they approach their maximum size before the first cycle of erythrocytic schizogony is completed.

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Effect of *p*-Amino-Benzoic Acid on the Toxicity of *p*-Amino-Benzene-Sulphonamide to Higher Plants and Fungi

MOST of the published observations on the antagonism between *p*-amino-benzoic acid and *p*-amino-benzene-sulphonamide relate to the growth of bacteria, but work carried out in these laboratories and elsewhere shows that this antagonism also obtains with higher plants and fungi.

Working with isolated tomato roots growing in tissue culture, Bonner¹ has described a growth inhibition caused by *p*-amino-benzene-sulphonamide which is reversed in the presence of *p*-amino-benzoic acid. More recently I have observed a similar response from complete plants growing under more natural conditions. In a series of experiments connected with the control of seed-borne fungoid diseases of wheat, the following observation was made. *p*-Amino-benzene-sulphonamide in fine powder form was applied to wheat at the rate of 0.01 per cent, and the grains were sown in soil. The emergence was markedly reduced, and those plantlets which did emerge were stunted. If the grains are soaked in aqueous solutions of *p*-amino-benzene-sulphonamide and are subsequently sown in moist sand at 22° C., percentage germination after five days is not affected, but the coleoptile and roots are stunted. Addition of *p*-amino-benzoic acid to the aqueous solution of *p*-amino-benzene-sulphonamide cancels this stunting effect, as is shown by the series of experiments recorded in Tables 1 and 2.

TABLE 1. MEAN PERCENTAGE GERMINATION OF WHEAT SEEDS: FIVE DAYS AT 22° C.

Concentration of <i>p</i> -amino-benzene-sulphonamide solution	Concentration of <i>p</i> -amino-benzoic acid			
	Nil	0.1 p.c.	0.25 p.c.	0.5 p.c.
Nil	100.0	100.0	100.0	100.0
0.5 per cent	100.0	97.3	98.6	100.0
1.0 per cent	100.0	98.6	100.0	96.0