resistance. This was postulated by ElNaghy and Linko[2] and from this point of view the aglucone appears to meet several of the criteria which would be required for its classification as a phytoalexin[7]. It is, however, difficult to imagine that variations in the rate and degree of hydrolysis of the glucoside combined with the possible differential sensitivity of different races of the rust fungus to the aglucone do, in fact, account for the pattern of resistance of a single variety of wheat to a number of different races of rust. There are, undoubtedly, many other chemical factors involved. Nevertheless, so far as we are aware, this is the only instance in which it has been possible to demonstrate a correlation between the general level of resistance to rust and the concentration of a single metabolite. The problem therefore deserves a more detailed investigation.

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Effect of Human Growth Hormone on 'Insulin Basic Protein Complex'

Young and George[1] report that, in their immunoassay system utilizing resin separation, the addition of human growth hormone (HGH) to fasting human serum causes a decrease in 'percentage antibody-bound' of iodine-131 labelled insulin, and they infer that HGH releases free insulin from a serum protein-bound form. We have been unable to confirm this phenomenon in a double-antibody immunoassay system[2,3].

(1) The addition of HGH to fasting human sera incubated with anti-insulin serum and ^{131}I-insulin causes no alteration in the percentage of antibody-bound ^{131}I-insulin (Table 1).

Table 1. Addition of HGH to Fasting Human Sera causes no Change in the Percentage of ^{131}I-insulin bound by Antibody
(In double antibody assay system; corresponding standard curve is shown for reference)

	Bound ^{131}I-insulin (%) (final volume 1·0 ml.)						
Amount HGH added	Fasting human sera		Buffer solution ± human insulin				
	Serum *A* 0·1 ml.	Serum *B* 0·5 ml.	0 μU	0·6 μU	1·25 μU	2·5 μU	5·0 μU
0	61·0	50·5	66·0	61·4	60·2	55·8	48·1
10 mμg	61·7	—	—	—	—	—	—
33 mμg	—	50·7	66·7	—	—	—	—
100 mμg	61·4	—	—	—	—	—	—
5 μg	61·2	—	—	—	—	—	—

Table 2. Addition of Insulin to Fasting Human Serum causes no Change in the Percentage of ^{131}I-HGH bound by Antibody
(Corresponding standard curve shown for reference)

	Bound ^{131}I-HGH (%) (final volume 0·7 ml.)				
Amount insulin added	Fasting human serum 0·1 ml.	Buffer solution ± HGH			
		0 mμg	0·1 mμg	0·2 mμg	0·4 mμg
0	—*	56	54	51	39
100 μU (human)	56	58	—	—	—
1,000 μU (bovine)	56	59	—	—	—

* No measurable growth hormone in this serum sample in previous assay (percentage bound ^{131}I-HGH in 0 mμg buffer tube = 48 per cent; and in 0·1 ml. serum tube 47 per cent).

(2) The addition of human insulin to human serum, incubated with anti-HGH serum and ^{131}I-HGH, similarly does not impair percentage antibody-bound ^{131}I-HGH (Table 2).

Hence the hypothesis that HGH and insulin can interact at a basic protein binding site cannot be supported. We suggest that Young and George's findings represent an artefact of their anion-exchange resin method. It seems likely that factors in human serum plus the HGH solution alter the binding reaction of the resin, causing a false decrease in 'antibody-bound ^{131}I-insulin'. Such methods require a monitoring device to ensure that complete separation of the antibody-globulin is attained, such as the use of ^{131}I-γ-globulin in control samples[4].

While both hormones are probably bound in serum–insulin to α-1-globulin[5] and HGH to α-2-macroglobulin[6], not only are these different binding proteins, but our data indicate that it is unlikely that either displaces the other from a binding site.

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Gibberellin-like Activity of Helminthosporol and Helminthosporic Acid

Helminthosporol (I) was isolated from a culture of *Helminthosporium sativum* by Tamura *et al.*[1], and was shown in this and subsequent studies[2] to stimulate the growth of the second leaf sheath of rice seedlings. However, (I) did not promote the growth of dwarf maize, nor did it give positive results in some other biological tests. Subsequently, Tamura and Sakurai[3] prepared a variety of related compounds and showed that (I) and helminthosporic acid, (II), promoted the elongation of rice seedlings and of lettuce seedling hypocotyls grown in light[4]. Thus, at high dose levels, these substances sometimes showed gibberellin-like activity. Various substances related to (−)-kaurene are probably on the biosynthetic pathway to gibberellic acid, (III), in *Gibberella fujikuroi*[5,6], and probably in higher plants also[7]. Some of these substances give positive results in a number of biological tests for gibberellins[8,9], as does steviol[10]. *Gibberella* converts steviol to an unidentified substance having activity in a wider range of test systems[11].

In view of the importance of gibberellic acid to the malting industry[12], and the interest attached to its mode of action, it seemed worthwhile to investigate the ability of (I) and (II) to trigger the release of sugars from de-embryonated barley *in vitro*, a release that is catalysed by hydrolytic enzymes synthesized *de novo* in the aleurone layer[13], since this response has always seemed specific for gibberellins. The evident similarity of (I) and (II) to the fused rings *C* and *D* of the kaurene derivatives, the gibberellins and steviol, and the fact that, unlike the other substances mentioned, (I) and (II) have only two rings rather than four, suggest that they may approximate to the minimal chemical structure needed to elicit a gibberellin-like response. In addition, (I) and (II) were tested for their ability to counteract the dwarfing effect of 2-chloroethyl-trimethylammonium chloride (CCC) on the hypocotyls of lettuce seedlings[14]. This compound prevents the synthesis of gibberellic acid by *Gibberella*[15], and so may stop gibberellin synthesis in higher plants. The results, Table 1, show that (I) and (II) are indeed able to reverse dwarfing due to CCC but less well than gibberellic acid itself, a result very similar to that obtained by Tamura and Sakurai[3], using light as the dwarfing agency.

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