slightly different in the folded (intramolecularly hydrogen bonded) than in the extended (intermolecularly hydrogen bonded) configurations of the polypeptide chain has been utilized to study the types of folding in polypeptide preparations.[5] Samples of polyglycine (average molecular weight 540), polyleucine (average molecular weight 960) and 1,1-co-glycine-L-leucine polypeptide (average molecular weight 1250) were dried at 55° and 0.1 mm., and their infrared absorption spectra determined in Nujol mull in the Baird Associates, Inc., I. R. Spectrophotometer, using a calcium fluoride prism.[11] Figures 1 to 3 give the spectra for these polypeptides. The carbonyl stretching mode for polyglycine occurs at about 1630 cm.$^{-1}$ (Fig. 1) in agreement with the published data of Ambrose and Elliott.[2] In poly-L-leucine, this peak occurs at about 1640 cm.$^{-1}$, with a shoulder at about 1660 cm.$^{-1}$ (Fig. 2). This shoulder is more pronounced in the curve for the copolymer (Fig. 3), and by comparison with the published work of Ambrose and Elliott, would indicate the presence of more of the folded structure.[2] Since it has been shown that the extended form predominates in short polypeptides,[7] which were used here, more striking shifts in the location of the peaks would be unexpected.

Discussion

Ambrose and Elliott[2] have pointed out that polypeptides of glycine are predominantly intermolecularly hydrogen bonded, and are soluble only in solvents which are capable of breaking these bonds.

It is at first surprising that the introduction of the large hydrophobic isobutyl side chain of leucine into the glycine polypeptide should increase its water solubility. However, this can be explained in part on the basis that the bulky side chain has reduced the amount of intermolecular hydrogen bonding. Consequently, fewer hydrogen bonds have to be broken in order to allow the molecules to pass into solution.

Bamford and associates,[7] on the basis of polarized infrared spectral data found that large side chains favored the folded form in polypeptides containing a single amino acid. It is of interest that similar results are obtained with polypeptides containing two amino acids and prepared in aqueous solutions. These findings, together with the observation that the polypeptides resulting from the copolymerizations are not only more soluble but also of higher average molecular weight than the polypeptides containing a single amino acid, gives support to the hypothesis[6] that the relative amounts of the folded and extended forms may be an important factor in determining the solubility of polypeptides. It would seem reasonable then to expect that the particular distribution of amino acids in the peptide chains of proteins would likewise affect the arrangement of the chains and the extent of intermolecular hydrogen bonding, and so influence the solubility behavior of the proteins.

MADISON 6, WISCONSIN

(11) We wish to thank Mr. Donald R. Johnson for making the infrared absorption measurements.

[CONTRIBUTION FROM THE DEPARTMENTS OF NEUROLOGY AND MICROBIOLOGY, COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, AND THE NEUROLOGICAL INSTITUTE, PRESBYTERIAN HOSPITAL]

Some Configurational Requirements and Dimensions of the Combining Site on an Antibody to a Naturally Occurring Antigen[1]

BY ELVIN A. KABAT

RECEIVED FEBRUARY 18, 1954

Oligosaccharides with 1 → 6 linkages inhibit the precipitation of human antidextran of 1 → 6 specificity and oligosaccharides with 1 → 4 specificity inhibit precipitation of antidextran with non 1 → 6 specificity by a dextran containing mainly 1 → 6 and 1 → 4 linkages. In each instance the homologous trisaccharide is much more effective than the homologous disaccharide. From the data on the inhibiting capacities of a variety of oligosaccharides of different structures the dimensions of the combining site of each type of antidextran are inferred to be complementary to an open chain of at least three α-D-glucopyranose units of homologous structure and probably to part or all of a fourth unit.

The classical studies of Landsteiner and co-workers[2] have established unequivocally the structural complementariness of the antibody combining group to the haptenic or determinant group of the antigen employed for immunization. These investigations as well as the more quantitative studies of subsequent workers[3,4] were carried out exclusively with artificial antigens prepared by introducing various determinant groups of known structure into the protein molecule. While the low molecular weight hapten introduced was capable of combining with the anti-hapten to inhibit precipitation of the the anti-hapten by a precipitating hapten or antigen, it was never possible to infer that any hapten used optimally satisfied the configuration requirements of the combining site on the antibody, since these may have been directed toward some unit larger than the group introduced into the antigen and may have involved not only the hapten but an indeterminate number of amino acids of the protein to which the hapten group had been attached. Indeed, Hooker and Boyd[5] showed that antibodies to a *p*-azophenylarsonate protein were inhibited

(1) This investigation was carried out under the William J. Matheson Commission and the Office of Naval Research (Contract #Nonr-266 (13)), Navy Department, Washington, D. C.

(2) K. Landsteiner, "The Specificity of Serological Reactions," Second Edition, Harvard University Press, Cambridge, Mass., 1945.

(3) L. Pauling, D. H. Campbell and D. Pressman, *Physiol. Rev.*, **23**, 203 (1943).

(4) D. H. Campbell and N. Bulman, "Progress in the Chemistry of Organic Natural Products," Vol. 9, L. Zechmeister, Editor, Vienna, 1952, p. 443.

(5) S. B. Hooker and W. C. Boyd, *J. Immunol.*, **25**, 61 (1933).

more strongly by tyrosine azophenylarsonate than by *p*-aminophenylarsonate alone.

The recent finding in this Laboratory[6] and confirmed by Maurer[7] that dextran was antigenic in man made available a tool *par excellance* for the elucidation of the structural requirements of the combining site of the antidextran molecule. From a study with dextrans[8] containing varying proportions of 1 → 6, 1 → 4, and 1 → 3 linkages,[8-11] of the quantitative precipitin curves of the antidextrans produced in various individuals by the injection of 1 mg. of dextran, it was concluded that most individuals produced antibody to some multiple of the 1 → 6 glucosidic unit.[6] The serum of one individual contained a mixture of antibodies, a small amount being antibody of 1 → 6 specificity, but most of the antibody having a specificity for the non 1 → 6 linkages.[6]

In the present communication, it is shown that certain oligosaccharides can inhibit precipitation of antidextran by dextran and that the inhibition is specific in that oligosaccharides of the isomaltose series (1 → 6) inhibit the antibody with 1 → 6 specificity while those of the maltose series do not. Furthermore a substantial portion of the antibody with the non 1 → 6 specificity is shown to be specific for the maltose (1 → 4) series of oligosaccharides. With both the 1 → 6 and the 1 → 4 type antibodies the homologous trisaccharide was found to be a much stronger inhibitor on a molar basis than the homologous disaccharide. The results of inhibition studies with these and a variety of other oligosaccharides are best interpreted as indicating that the combining site on each of the two varieties of antidextran has dimensions complementary to a chain at least three α-D-glucosidopyranose units, and probably to part or all of a fourth unit.

Experimental

Materials and Methods.—Two samples of human antidextran were employed, from individuals 30 and 9. The quantitative precipitin curves on the sera of an earlier bleeding from these two individuals have been described in detail.[6] The former had produced antibody showing 1 → 6 specificity while the latter had produced but a small amount of antibody of 1 → 6 specificity with the bulk of the antibody having specificity for the non 1 → 6 linkages[6]; the serum samples were designated $30D_{-4}$ and $9D_{-4}$ and contained about 72 and 24 μ g. antibody N per ml. precipitable by native dextrans 236 and 1299, respectively. Dextrans N150N and 236 prepared from the NRRL B512 strain were kindly supplied by Drs. F. Schulz and M. B. Bachmann of Commercial Solvents Corporation; dextrans 1299 S-3 and 1355 S-4 by Dr. Allene Jeanes of the Northern Regional Research Laboratory; the analytical data were also obtained from these sources.[6,8-10]

THE DEXTRAN SAMPLES EMPLOYED IN THIS STUDY

	Types of linkage from periodate oxidation[8-11] 1 → 6 %	1 → 4 %	1 → 3 %
Clinical dextran N150N	97–98		3–2
Native dextran 236	96		4
Native dextran NRRL 1299 S-3	50	50	0[a]
Native dextran NRRL 1355 S-4	57	9	35

[a] The periodate oxidation value of this dextran is anomalous and the sample is actually known[11,12] from infrared and optical rotation studies to contain about 9% of 1 → 3 like linkages.

Oligosaccharides included isomaltotriose and isomaltose supplied by Dr. Allene Jeanes[13a]; isomaltose, cellobiose and isomaltitol[13b] from Dr. M. L. Wolfrom of The Ohio State University, maltotriose[14]; maltotetraose, maltopentaose[15] and xylotetraose[16] from Dr. Roy L. Whistler of Purdue University; maltose hydrate from Dr. D. P. Langlois of A. E. Staley and Company; maltoheptaose, panose (4α-isomaltosyl-D-glucose), cycloheptaamylose and cycloöctaamylose from Dr. Dexter French of Iowa State College; and 4α-isomaltotriosyl-D-glucose[17] from Dr. John H. Pazur of the University of Nebraska. Most of the samples had been purified chromatographically and separated from higher and lower oligosaccharides. Without the generous coöperation of these individuals, this work would not have been possible. A sample of gentiobiose from Concord Laboratories also was used.

Inhibition Assay.—The procedure employed consisted in the addition of known quantities of oligosaccharide to a measured volume of antiserum; 0.5 ml. of antiserum $30D_{-4}$ and 1.0 ml. of antiserum $9D_{-4}$ were used. After 30 minutes at 37° a suitable quantity of dextran was added to each tube and to tubes containing the same amount of antiserum with no oligosaccharide. Controls with antiserum alone were also included. The total volume was 2 ml. After mixing, the tubes were incubated at 37° for one hour and placed in the refrigerator for one week with mixing twice daily. The tubes were then centrifuged in a refrigerated centrifuge, the precipitates washed twice with chilled saline, dissolved in a drop of *M*/2 NaOH, diluted to 2.5 ml. and a 2-ml. aliquot analyzed by the Folin–Ciocalteu tyrosine reagent as described by Heidelberger and MacPherson[18] for the micro-quantitative precipitin method.[19] The color developed was read in a Beckman spectrophotometer at 7500 Å. and converted to antibody nitrogen by means of factors determined for human γ-globulin and antibody.[20] The percentage of inhibition produced by each concentration of oligosaccharide was calculated. The conditions employed were shown to result in equilibrium since the same degree of inhibition was produced within experimental error by addition of the dextran to the antibody and, when definite opalescence developed, addition of the inhibitor.

In carrying out inhibition studies it was observed that cycloheptaamylose, when added to the antiserum at a concentration of 2.3 μmole, gave a fine precipitate quite unlike an antigen antibody precipitate. Normal human serum also was found to precipitate with cycloheptaamylose at this concentration. Oddly enough no precipitation was obtained with cycloöctaamylose under these conditions so that it could be used satisfactorily as an inhibitor. Precipitation did not occur with 0.45 μmole of cycloheptaamylose, however, and it was tested for inhibiting power at this concentration.

Evaluation of Amylase Activity.—In studies on the maltose series of oligosaccharides, the hydrolytic action of serum amylase on these oligosaccharides producing maltotriose and maltose[21] would create uncertainty as to the concentration of inhibitor present. The amount of maltotetraose

(6) E. A. Kabat and D. Berg, (a) *Ann. N. Y. Acad. Sci.*, **55**, 471 (1952); (b) *J. Immunol.*, **70**, 514 (1953).

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(8) A. Jeanes, W. C. Haynes, C. A. Wilham, J. C. Rankin, H. M. Tsuchiya and C. E. Rist, Abstracts Papers, Am. Chem. Soc., **122**, 14A (1952).

(9) A. Jeanes and C. A. Wilham, THIS JOURNAL, **72**, 2655 (1950); **74**, 5339 (1952).

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(11) J. C. Rankin and A. Jeanes, *ibid.*, **125**, abstracts (1954).

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(13) (a) A. Jeanes, C. A. Wilham, W. Jones, H. M. Tsuchiya and C. E. Rist, THIS JOURNAL, **75**, 5911 (1953); (b) M. L. Wolfrom, A. Thompson, A. N. O'Neill and T. T. Galkowski, *ibid.*, **74**, 1062 (1952).

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(15) W. J. Whelan, J. M. Bailey and P. J. P. Roberts, *J. Chem. Soc.*, 1293 (1953).

(16) R. L. Whistler and C. C. Tu, THIS JOURNAL, **74**, 3609 (1952).

(17) J. H. Pazur and D. French, *J. Biol. Chem.*, **196**, 265 (1952).

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(21) W. J. Whelan and P. J. P. Roberts, *J. Chem. Soc.*, 1298 (1953).

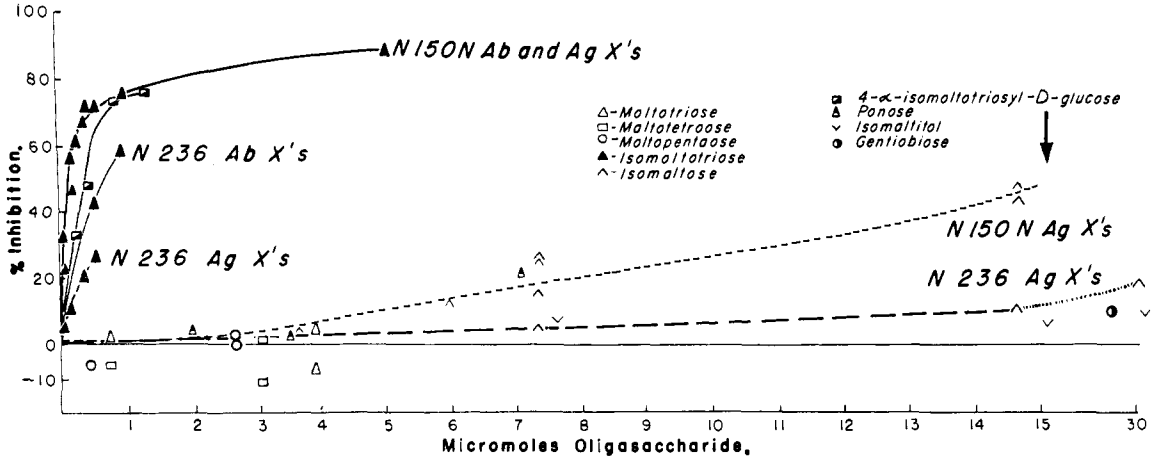

Fig. 1.—Inhibition of precipitation of dextran–antidextran $30D_4$ of $1 \rightarrow 6$ specificity by various oligosaccharides; 0.5 ml of antiserum, 4 μg. of N150N for antibody excess, 10 μg. of N150N for antigen excess, 5 μg. of N236 for antibody excess, 25 μg. of N236 for antigen excess points; total volume 2.0 ml.

Inhibitor used	Amt. present, μ mole	Dextran used	Inhib., %	Additional points not on graph Inhibitor used	Amt. present, μ mole	Dextran used	Inhib., %
Glucose	56	N150N Ag x's	6	Glucose	55	N236 Ag x's	5
Cycloöctaamylose	2.1	N150N Ag x's	3	Glucose	110	N236 Ag x's	7
Gentiobiose	51	N150N Ag x's	30	Gentiobiose	51	N236 Ag x's	8
Xylotetraose	9	N150N Ab x's	0	Maltose	59	N236 Ag x's	9
Glucose	110	N150N Ab x's	25	Xylotetraose	9	N236 Ag x's	4
Gentiobiose	51	N150N Ab x's	30	Cellobiose	7.5	N236 Ab x's	4
Cellobiose	7.5	N150N Ab x's	7	Isomaltotriose	0.2	N236 Ab x's	31

and maltopentaose split by the amylase in serum $9D_{-4}$ was determined under the conditions used in the inhibition assay. It was found that 35 and 60% of the maltotetraose and maltopentaose were split, respectively.

Results

The findings in antiserum $30D_{-4}$ are summarized in Fig. 1. The specificity of the inhibition for the α $1 \rightarrow 6$ glycosidic linkage is evident, in that the oligosaccharides isomaltotriose, isomaltose, 4α-isomaltotriosyl-D-glucose and panose containing one or more $1 \rightarrow 6$ linkages inhibit precipitation, while oligosaccharides (maltopentaose, maltotetraose, maltotriose, maltose, cellobiose) with $1 \rightarrow 4$ glycosidic linkages do not inhibit. The requirement for an open chain structure is suggested since some inhibition can be obtained by very high concentrations of gentiobiose and glucose (data with Fig. 1). Among the $1 \rightarrow 6$ oligosaccharides isomaltotriose is a much better inhibitor than isomaltose, about 0.15 μmole of the former being required for 50% inhibition as compared with over 15 μmole of the latter using N150N as the precipitating dextran; with N236 about 0.40 μmole of isomaltotriose and about 30 μmole of isomaltose were required for about 20% inhibition. 4α-Isomaltotriosyl-D-glucose was somewhat less effective as an inhibitor than isomaltotriose, 0.5 μmole being required for 50% inhibition as compared with 0.15 μmole (Fig. 1), but was many times more effective than isomaltose or panose. Isomaltitol, however, was much poorer as an inhibitor than isomaltose and failed to inhibit significantly even in a concentration of 30 μmole. The points for isomaltitol and gentiobiose in Fig. 1 were obtained using dextran N150N and should be compared with the other oligosaccharides using this dextran.

Figure 1 also shows that the degree of inhibition obtained with a given oligosaccharide is less with a native than with a clinical dextran. With native dextran a higher concentration of inhibitor is required in the antigen excess region than in the antibody excess region.

The data for antiserum $9D_{-4}$ are given in Fig. 2. This antiserum had previously been shown to contain a small amount of antibody of $1 \rightarrow 6$ specificity with most of the antibody being of non $1 \rightarrow 6$ specificity. The inhibition data in Fig. 2 were obtained with native dextran B1299 S-3 which contained a very large proportion of $1 \rightarrow 4$ linkages and only 9% of $1 \rightarrow 3$ like linkages.[11,12] Under these conditions the specificity of this system for oligosaccharides with $1 \rightarrow 4$ glycosidic linkages is seen. Only slight inhibition with isomaltotriose and isomaltose was found; this probably was due to inhibition of the small amount of $1 \rightarrow 6$ antibody present. The maltose series of oligosaccharides, however, inhibited at much lower concentrations. The requirement for a terminal glycosidic residue is evident from the failure of cycloöcta- and cycloheptamylose to inhibit in concentrations comparable to that of the non-cyclic oligosaccharides. The greater degree of inhibition with panose is probably due to its simultaneously inhibiting the small amount of $1 \rightarrow 6$ antibody as well as the $1 \rightarrow 4$ antibody. The shape of the curve suggests more rapid inhibition with small amounts while both antibodies are being inhibited. After inhibition of the $1 \rightarrow 6$ antibody is complete further inhibition parallels that of maltose.

The most striking finding in Fig. 2 is that maltotriose, maltotetraose and maltopentaose are much better inhibitors than maltose or panose and are

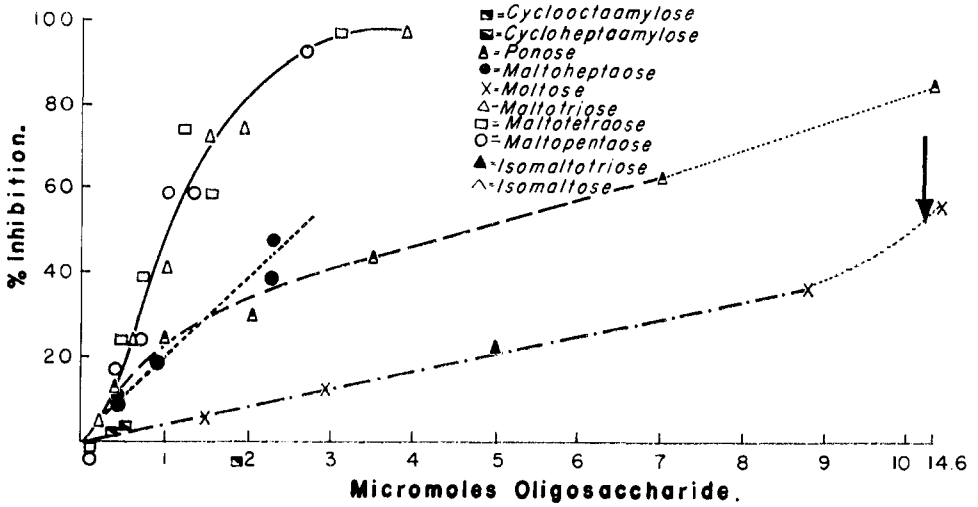

Fig. 2.—Inhibition of precipitation of dextran–antidextran $9D_{-4}$ of 1 → 4 specificity by various oligosaccharides: 1.0 ml. antiserum, 100 μg. of dextran B1299 containing 50% each of 1 → 6 and 1 → 4 linkages; total volume 2.0 ml.

Additional points not on graph

Inhibitor used	Amt. present, μ mole	Inhib., %	Inhibitor used	Amt. present, μ mole	Inhib., %
α-Methyl galactoside	57	−2	Gentiobiose	25.5	17
Xylotetraose	4.5	3	Glucose	26	23

about equally effective on a molar basis in inhibiting precipitation of antidextran by dextran. Taken alone, these findings would establish the requirements of the antibody-combining group as involving two α-glycosidic linkages and a minimum of two and a maximum of three pyranose rings. However, these data are rendered somewhat uncertain to the extent that any amylase activity might have split maltotetraose and maltopentaose.[21a] Since maltopentaose is split into maltotriose and maltose, and the latter is a very weak inhibitor, the potency of maltopentaose would approach that of maltotriose as enzymatic digestion became more complete and, unless maltopentaose were a much better inhibitor than maltotriose, the two would appear equally effective if any appreciable amylase action occurred. As previously indicated, about 60% of the maltopentaose was split under the conditions of the experiment.[22] In the case of maltotetraose, however, since amylase action produces two molecules of maltose which is a much poorer inhibitor, the effectiveness of maltotetraose as an inhibitor would be reduced proportionately to the amount split by amylase. In this instance about 35% of the maltotetraose was hydrolyzed by amylase. It is possible, therefore, that had there been no amylase present, maltotetraose would have been about one-third more effective as an inhibitor on a molar basis than maltotriose. Were this the case, then the dimensions of the antibody combining site would fit more closely a unit of three 1 → 4 α-glycosidic units with a minimum of three pyranose rings plus carbon 4 of the fourth ring up to a maximum of the entire tetrasaccharide.

Figure 2 also shows that maltoheptaose on a molar basis is substantially less effective as an inhibitor than are either maltotriose, maltotetraose or maltopentaose. If amylase action were complete in this instance, one molecule of maltotriose would be formed for each molecule of maltoheptaose split and the maltoheptaose should have been as effective an inhibitor as the maltotriose. The findings, therefore, indicate that the maltoheptaose is not completely split by amylase and suggest that its inefficiency as an inhibitor is probably due to steric hindrance from its helical structure.[23]

Using antiserum $9D_{-2}$ and dextran NRRL B1355 S-4 containing 35% 1 → 3 and only 9% 1 → 4 glycosidic units, it was found that 1.0, 0.74, and 0.70 μmole of maltotriose, maltotetraose and maltopentaose produced no inhibition of precipitation, as compared with 37, 35 and 19% inhibition when B1299 S-3 with 50% 1 → 4 glycosidic units was used. These findings strongly suggest that antibody with specificity involving the 1 → 3 linkage is present in this antiserum and that 1 → 4 oligosaccharides are ineffective in preventing formation of an antigen antibody aggregate involving other than 1 → 4 linkages.

The finding that maltotriose and maltotetraose are more effective than maltose in inhibiting precipitation by dextran B1299 S-3 indicates that this dextran must contain short chains with two or three 1 → 4 linkages in sequence and therefore that all of the 1→ 4 linkages do not occur as branch points on a primary chain of 1 → 6 linked units.[24]

Discussion

The data presented make it possible to define within much closer limits than hitherto possible the dimensions of the grouping on the antibody complementary to a natural antigen molecule. The un-

(21) (a) This argument would not be seriously influenced by the recent finding that maltotriose is split into glucose and maltose at a very slow rate, *cf.* J. H. Pazur, *J. Biol. Chem.*, **205**, 75 (1953).

(22) The author is indebted to Drs. E. J. Hehre and Dexter French for calling to his attention the possibility of amylase action and its consequences.

(23) E. G. V. Percival, "Structural Carbohydrate Chemistry," Prentice-Hall, Inc., New York, N. Y., 1950.

(24) This interpretation of the data was suggested by Dr. Fred Senti of the Northern Regional Research Laboratory.

certainties in the use of proteins containing artificially introduced groups already have been indicated. In studies of the cross reaction of artificial carbohydrate protein antigens containing glucuronic, cellobiuronic and gentiobiuronic acids with certain antipneumococcal sera and their inhibition by glycosides of these sugars,[25,26] Goebel was able to establish certain specificity relationships, but no data on the relative effectiveness of oligosaccharides of various sizes were obtained.

It is evident for two kinds of antidextran, one having 1 → 6 and the other 1 → 4 specificity, that the homologous trisaccharide is enormously more effective as an inhibitor than the corresponding disaccharide and that each system is specific for the 1 → 6 or 1 → 4 repeating structure, respectively. These findings are uncomplicated by the possibility of albumin binding since Dr. M. E. Carsten in this Laboratory has shown that such binding does not occur with these oligosaccharides. That panose in each instance is no more effective than the homologous disaccharide (allowing in antiserum $9D_{-2}$ for the presence of some 1 → 6 antibody) suggests that the place of attachment of the third pyranose ring is also of crucial importance. These observations immediately place a lower limit on the size of the antibody combining site as being complementary to a unit of two α-glycosidic linkages and three pyranose rings.

In defining further the dimensions of the antibody combining site, the data at this time do not permit a definitive answer, but suggest that a homologous tetrasaccharide might fit more closely than the homologous trisaccharide. In the case of the 1 → 6 antibody, 4α-isomaltotriosyl-D-glucose is only about one-third as effective as an inhibitor as isomaltotriose. This would suggest that the attachment of the fourth pyranose ring through a maltose linkage at carbon 4 interferes with the approach of this oligosaccharide to the antibody combining site. Since this antibody has formed to a long string of 1 → 6 linked glucopyranosides, one would expect that, if the antibody combining site were complementary only to a trisaccharide, the fourth unit would not interfere The finding of interference might therefore suggest that a unit larger than a trisaccharide, perhaps a tetrasaccharide, would be most complementary to the antibody grouping. Further suggestive but not conclusive evidence is obtained from the results with antibody from which, correcting for the 35% splitting of maltotetraose by the amylase in serum, the tetrasaccharide would be more effective as an inhibitor than the trisaccharide.

A more precise answer to this question must await the availability of isomaltotetraose for inhibition studies in the 1 → 6 system or the preparation of antidextran of 1 → 4 specificity free from amylase. Further studies along these lines are in progress. A more precise expression of these data in actual dimensions will require analysis from three dimensional models. In view of the known heterogeneity of antibodies even to a single antigen, it may be that these data should be considered representing a statistical average of the dimensions of the combining sites of all of the antibody groupings present.

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(25) (a) W. F. Goebel, *J. Exp. Med.*, **64**, 29 (1936); (b) W. F. Goebel and R. D. Hotchkiss, *ibid.*, **66**, 191 (1937).

(26) W. F. Goebel, *ibid.*, **72**, 33 (1940).

[CONTRIBUTION FROM GENERAL ELECTRIC RESEARCH LABORATORY, SCHENECTADY]

The Preparation and Properties of Some Organosilicon Sulfides and Sulfones

BY GLENN D. COOPER

RECEIVED FEBRUARY 18, 1954

Organosilicon sulfides of the type $\equiv SiCH_2SR$ were obtained by the reaction of the corresponding chlorosubstituted silane or siloxane with sodium mercaptides. The corresponding sulfones may be prepared by oxidizing the sulfides with hydrogen peroxide in acetic acid, if siloxane bonds are not present; the sulfone derivatives of the siloxanes were obtained when monoperphthalic acid in ether is used as the oxidizing agent. The sulfones underwent silicon–carbon cleavage very readily when treated with bases, but were much more resistant than the corresponding ketones, esters, and nitriles to cleavage by acids. The siloxanes were much more susceptible to silicon–carbon cleavage than the silanes.

As part of a program of study of the properties of organosilicon compounds having functional groups attached to carbon, a number of types of compounds having sulfur attached to a carbon atom α to silicon have been prepared, in most cases by the displacement of chlorine from a chloromethylsilane or -siloxane by a sulfur-containing compound or ion, such as thiourea, sulfide ion, mercaptide ion, thiocyanate ion or hydrosulfide ion. Nucleophilic reagents may attack organosilicon compounds having halogen on a carbon atom α to silicon either at the silicon atom, resulting in silicon–carbon cleavage, or at the carbon atom, resulting in the normal displacement reaction.[1] In addition, if siloxane bonds are present, extensive siloxane rearrangement may occur.[2] The sulfur compounds, presumably because of their high reactivity as nucleophilic reagents and relatively low basicity, gave in each case good yields of the normal products, without excessive silicon–carbon cleavage or siloxane rearrangement. The thiocyanates[3] and mercaptans[4] have been previously described; in this paper the preparation and properties of some organosilicon sulfides, sulfones and sulfonium iodides having sulfur attached to the α-carbon atom are reported. Since this work was completed,

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(3) G. D. Cooper, *ibid.*, **76**, 2500 (1954).

(4) G. D. Cooper, *ibid.*, **76**, 2499 (1954).