Table 2

	1	2	4	8	16	32	64	128	256
x/12156	(+)	gw	+	+	+	+	+	++	++
x/12157	w	(+)	+	+	++	++	++	++	++
x/12158	w	(+)	+	+	+	++	++	++	++
x/12159	–	w	(+)	(+)	+	+	+	++	++
x/12160	–	w	(+)	(+)	+	+	+	++	++
x/12506	(+)	+	+	+	++	++	++	++	++
x/12507	(+)	(+)	+	+	+	++	++	++	++
x/12508	(+)	+	+	+	+	+	++	++	++
x/12509	–	–	–	–	w	(+)	+	++	++
Mr. K.	–	(+)	(+)	+	+	++	++	++	++

Saline control—anti-B 1/10 + an equal volume 2 per cent B red cells in saline: ++.

(9) Mr. K's serum gave no reactions at 4°, 18° or 37° C. with known A_4 cells[7], suggesting that the serum was not from a group O person.

By the time the investigations reported here were complete, Wiener and Gordon's report[8] was just published of a blood, the red cells of which grouped as O—β, yet the saliva of this person contained A substance. They claim this as a further sub-group of A and propose the name A_m because of serological similarities between the blood they describe and the bloods of certain monkeys[9]. No claim is made that the blood reported here is a new sub-group of B, although indeed it may be. This part of the investigation awaits specimens (blood and saliva) from the relatives of the propositus.

Other possibilities that have to be considered are that this blood is a further example of the type described by Levine *et al.*[10] which was phenotypically *O*, but was proved to be of genotype *BO*, but the gene *B* was suppressed. However, while further investigation is necessary, one major difference between the blood described by Levine *et al.* and the blood here reported is that the former contained anti-H, whereas the blood we have examined did not possess such an antibody. Unfortunately, we did not test Mr. K's cells with anti-H.

An alternative possibility is that this blood is a defective B type in which the alcohol-soluble form of the antigen is lacking while the water-soluble form has developed normally. The defect may conceivably have resulted from the absence of normal complementary genes for the development of the alcohol-soluble antigen system, or from the influence of a modifying gene upon the alcohol-soluble system. This was suggested by Gammelgaard[11] to describe a blood A_2, which had little or no evidence of the A antigen in the red cells and yet the saliva was rich in A substance. Indeed, Gammelgaard's A_x and Wiener's A_m may be identical, and the latter may not be a new form of blood group A as claimed by Wiener.

The blood reported here is not an exception to Landsteiner's Law, providing the presence of the antigens is not confined to the red cells. In further work on the human chimera which will be reported later[6], we have observed that the chimera, although phenotypically *O—A*, possesses no anti-A agglutinins, but possesses A substances in her serum when not pregnant. However, when pregnant her serum possesses no A substances—even though she has A cells in her circulation—and at this time possesses an anti-A agglutinin. It would appear from the observations made on the present blood and on that of the human chimera that the presence or absence of agglutinins in the serum is more dependent upon group-specific substances in the serum than on the group-specific substances in the red cells.

It is hoped to carry out further studies on this blood and on that of members of this person's family, and to give a fuller report elsewhere.

We wish to thank Dr. A. E. Mourant, of the Medical Research Council Blood Group Reference Laboratory, London, for his suggestions.

Note added in proof. Since submitting this report for publication, members of Mr. K's family have been tested and his father and one sister have the identical variety of blood group B.

[1] Landsteiner, K., *Wien. Klin. Woch.*, **14**, 1132 (1901).
[2] Dunsford, I., *Vox Sanguinis*, **3**, 3 (1953).
[3] Dunsford, I., and Bowley, C. C., "Techniques in Blood Grouping" (Oliver and Boyd, 1955).
[4] Wiener, A. S., *Amer. J. Clin. Path.*, **25**, 495 (1955).
[5] Dunsford, I., *et al.*, *Brit. Med. J.*, ii, 81 (1953).
[6] Dunsford, I., and Stacey, S. M., to be presented at VIth Congress International Society of Blood Transfusion, Boston, U.S.A., 1956.
[7] Dunsford, I., and Aspinall, P., *Ann. Eugenics*, **17**, 30 (1952).
[8] Wiener, A. S., and Gordon, E. B., *Brit. J. Haemat.*, **11**, 305 (1956).
[9] Wiener, A. S., *et al.*, *J. Immunol.*, **45**, 229 (1942).
[10] Levine, P., *et al.*, *Blood*, **10**, 1100 (1955).
[11] Om. Sjieldne Svage A–Receptorer Hos. Mennsket–A. Gammelgaard–Nyt Nordisk Forlag. Arnold Busk, 100–105 (1942).

'LIVING' POLYMERS

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POLYMERIC molecules are born in an initiation process, they grow by a propagation process, and finally they 'die' in a termination process. This death is regulated by the conditions prevailing in the polymerization process, and thus, the rate of death, the average molecular weight of the polymer formed, and its molecular weight distribution are all well-determined functions of experimental conditions.

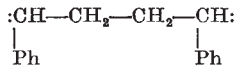
An interesting situation arises when a polymerization process does not involve a termination step. The polymeric molecules then 'live' for an indefinite period of time, and such a system should be profitable in many investigations. A 'living' polymer does not grow indefinitely, nor does its molecular weight increase beyond certain limits. Any growth requires food, and the food for a growing polymer is the monomer. Consequently, if the supply of monomer is exhausted the growth is interrupted, although the living ends are potentially able to grow further if an additional amount of monomer is available.

The exclusion of natural death does not mean immortality either. Reagents may be visualized which convert a living end into an unreactive dead end. Treatment of living polymers with such reagents represents, therefore, a killing process. The difference between the normal termination process—described here as a natural death—and the killing process lies in the unavoidable character of the former reaction, whereas the latter takes place at the time chosen freely by the experimenter. The advantage of such a termination is obvious.

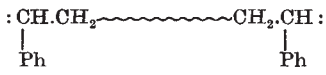
Recently we have succeeded in preparing in our laboratories living polymers by carrying out an anionic polymerization in a non-acidic solvent, such as, for example, tetrahydrofurane. The growing end of an anionic polymer is a carbanion. If the polymerization is proceeding in a solvent which is a proton donor, then the transfer of a proton from the acidic solvent to the carbanionic end of a growing polymer terminates the polymerization. Such a mechanism operates, for example, in polymerization initiated by sodium and carried out in liquid ammonia. However, this termination is impossible in tetrahydrofurane solution.

In solvents which solvate poorly the ions formed, the termination may occur by returning an electron from a carbanion to the cation (say Na^+). Such a reaction probably terminates the polymerization initiated by sodium in hydrocarbon solution. However, tetrahydrofurane solvates the ions well, and therefore in this solvent the re-formation of neutral species is energetically unprofitable. Hence, this second termination mechanism does not operate in tetrahydrofurane either.

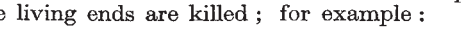
One assumes finally that the anionic polymerization carried out in tetrahydrofurane solution does not involve any termination step, and in order to examine this assumption the following experiments were carried out with styrene as the monomer. The polymerization was initiated by the intensely green complex naphthalene anion, sodium cation. This initiating catalyst was discovered by Scott[1] and the mechanism of this initiation was elucidated recently in our laboratories[2]; we showed that it involves an electron transfer process from the naphthalene anion to the monomer, for example, styrene. Consequently, negative monomer ions are formed which eventually dimerize into species such as:



and the latter grow by a carbanionic mechanism until the monomer supply is exhausted. Thus, living polymers of the following structure are formed:



The —Ph.CH: ends of the living polymers are bright red—this is essentially the colour of the negative benzyl ions. Hence, the green colour of the catalyst solution (naphthalene anion) turns red instantaneously when introduced to the styrene solution. In absence of air or moisture the red colour persists for days, without any apparent change of intensity. However, when air or moisture comes in contact with the solution, the red colour disappears —the living ends are killed; for example:



Hence, the observation of colour is the simplest manifestation of living polymers.

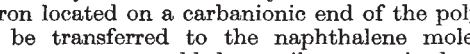
At this juncture one may wonder whether an electron located on a carbanionic end of the polymer may be transferred to the naphthalene molecule. Such a process would be easily recognized. The anionic ends deprived of an electron are transformed into radical ends. The latter undergo an irreversible dimerization process. Consequently, the final result would be the change of the colour from red to green, and an increase in the viscosity of the solution resulting from polymerization of polymers. Neither of these effects was observed, showing that the proposed electron transfer is too slow to be seen. This result reflects, of course, the much higher electron affinity of benzyl radical as compared with, say, naphthalene.

Now, if additional amounts of styrene and of tetrahydrofurane are added to the red solution of a living polymer, further polymerization ensues. If the ratio of styrene to solvent is chosen to be identical to that in the first batch, then the *concentration* of polystyrene formed remains unaltered by the second polymerization. Nevertheless, the viscosity of the solution increases enormously. This increase in viscosity, coupled with the fact that both portions of styrene are converted quantitatively into a polymer, proves that the second batch of monomer polymerized on the living ends of the previously prepared polymer.

This experiment suggests an interesting and novel method for the preparation of block polymers. After completion of the first polymerization process, a second monomer is added to the still living polymers formed from the first monomer. Thus, block polymers of the type *AA...A.B.B...B.A.A...A* are produced. For example, polymerization was initiated in styrene solution. When the reaction was ended, isoprene was added and further polymerization ensued. After killing with water, a polymer was obtained which was shown to contain neither polystyrene nor polyisoprene. Thus, the nature of the block polymer is established.

It is interesting to compare the effect of two killing agents, namely, water and oxygen. If the viscosity of the red solution of living polymers is determined and then a drop of water is introduced, the colour will disappear but the viscosity of the solution remains unaltered. This killing process leads to a transfer of a proton from a water molecule to the carbanionic end of the living polymer, and such a process is not expected to change the molecular weight of the polymer. On the other hand, if oxygen is introduced into the red solution, the colour disappears again, but simultaneously with this effect the viscosity of the solution increases considerably.

It is believed that oxygen represents the class of killing reagents which removes one electron from the carbanionic ends. The high electron affinity of the oxygen molecule favours this reaction:



Consequently, the living ends are transformed into radicals, and the latter undergo dimerization. (It is possible that the dimerization process involves oxygen bridges due to the action of other oxygen molecules. This, however, does not change the essential characteristic features of the phenomena described here.) Since our living polymers possess two living ends, the dimerization process leads to polymerization of polymers, and consequently, to a considerable increase in the viscosity of the solution.

I hope that these brief comments will show how versatile is the chemistry of living polymers. A detailed description of this work will be published later.

[1] Scott, N. D., Walker, J. F., and Hansley, V. L., *J. Amer. Chem. Soc.*, **58**, 2442 (1936). Scott, N. D., U.S. Patent 2,181,771 (1939).

[2] Szwarc, M., Levy, M., and Milkovich, R., *J. Amer. Chem. Soc.*, **78**, 2656 (1956).