The reaction of hog gastrin to rabbit anti-hog gastrin serum was studied in agar gel diffusion plates. One major antigenic component and at least three minor antigenic components were demonstrated, emphasizing the impurity of gastrin prepared by this method. Dog and human gastrin reacted with homologous rabbit antiserum similarly showed multiple antigenic components. An attempt was made to demonstrate a cross reaction between hog, dog, and human gastrins when they were reacted with rabbit anti-hog gastrin serum. No such cross reaction could be shown in agar gel diffusion plates in spite of the cross reaction achieved in the inhibition experiments described above. These results may be interpreted to mean that the active secretory principle in gastrin prepared by the Gregory method is in too low a concentration to form a crossreacting precipitin band demonstrable by the relatively insensitive agar gel diffusion method (7). Another interpretation might be that not all antigenantibody complexes precipitate, particularly if the antigen molecule is small or has only one antigenic site. Thus, gastrin may be a small, univalent, antigenic molecule.

The following conclusions are suggested. Gastrin produced by the Gregory method is a relatively impure substance with several demonstrable antigenic components. The active acid secretory principle contained in this preparation may be in low concentration or may be a small, univalent antigen which does not stimulate the production of precipitating antibody. Antiserum reacted with crude hog gastrin is capable of neutralizing the physiologic effect of hog, dog, and human gastrin, illustrating a cross reaction among the species mentioned. Gregory's method of preparing gastrin almost certainly excludes contamination of the final product with histamine which stimulates gastric secretion. However, the possibility that trace amounts of histamine might be present has been considered despite the fact that biologic assay does not reveal any histamine (4). The present experiments add further evidence that, in gastrin prepared by the Gregory method, histamine is not the substance that induces acid secretion (8).

> WILLIAM R. WADDELL* J. PHILIP LYTHGOE[†] ANTHONY P. MONACO

Department of Surgery, Harvard Medical School and Massachusetts General Hospital, Boston

References and Notes

- 1. J. S. Edkins, J. Physiol. (London) 34, 133
- J. S. Edkins, J. Physiol. (London) 34, 133 (1906).
 S. A. Komarov, Proc. Soc. Exptl. Biol. Med. 38, 514 (1938); Rev. can. biol. 1, 191 (1942).
 B. Uvnäs, Acta Physiol. Scand. 9, 296 (1945); J. E. Jorpes, O. Jalling, V. Mutt, Biochem. J. 52, 327 (1952).
 R. A. Gregory and H. J. Tracy, J. Physiol. (London) 149, 70P (1959).
 M. Heidelberger and F. E. Kendall, J. Exptl. Med. 62, 697 (1935).
 M. W. Wilson and B. H. Pringle, J. Immunol. 73, 232 (1954).
 E. Kabat and M. M. Mayer, Experimental

- 7. E. H. Kabat and M. M. Mayer, Experimental Immunochemistry (Thomas, Springfield, Ill., ed. 2, 1961), pp. 88-89.
- This work was supported in part by grant No. A-1836 (C2) from the National Institutes of Health. 8 supported in part by grant
- Present address: University of Colorado Medical Center, Denver.
 Recipient of the Dickinson medical traveling scholarship and a Wellcome travel grant.

14 July 1961

Erythrocyte Automosaicism in Some Persons of Known Genotype

Abstract. We have estimated the proportions of non-A and non-B erythrocytes in some members of four families in which both parents were blood group AB, including two B homozygotes. The exceptional cell frequencies were not discernibly correlated with age. The B homozygotes had fewer non-B exceptional cells than their AB parents or sibs, but too many to represent independent losses of the B alleles. These results are not in agreement with the simplest expectations based on the mutational hypothesis of automosaicism.

Red cell populations in human A or AB individuals and in Phaseolus lectinpositive pigeons are heterogeneous, containing a proportion of non-A or lectinnegative cells that is rather stable, but subject to individual variation. This proportion is increased by P³² treatment in man or by whole-body x- or gamma irradiation in pigeons (1). Part of the induced increase is stable for an indefinite time, which suggests that the phenotypic heterogeneity among the red cells reflects a corresponding genetic diversity among the reproducing erythropoietic cells. The presence of such exceptional cell populations generated within an individual has been called automosaicism to distinguish it from chimerism involving admixture of cells from different zygotes.

Two criteria bearing on the spontaneous origin of automosaicism are its relations to age and to zygosity. If it is mainly caused by accumulation of genetically altered stem cells during adult life, a correlation with age is expected. If it is caused by independent events affecting the two ABO alleles in the diploid cell, then the proportion

of exceptional cells in a homozygote should approximate the square of the proportion in a comparable heterozygote.

Through the cooperation of the Chicago Blood Donors Association, we were able to study four families in which both parents in each family are group AB. These included two B homozygotes and related persons of widely disparate age. The genotypes and ages of the persons studied are shown in the figure captions. The experiments indicate, first, that non-A and non-B cell proportions in AB children are rather similar to those in adults; second, that the proportions of non-B cells in the B homozygotes, although from 1/5 to 1/20 of those in related heterozygotes, were still from 50 to 200 times higher than the squares of the proportions in the heterozygotes.

The proportions of non-A and non-B cells were estimated by the isotope dilution method previously described (2). Saline suspensions of the cells were labeled with Cr⁵¹ and passed through repeated agglutinations and separations (stages) in the presence of unlabeled "carrier" cells. Within five or six stages the rate of removal of labeled cells per stage usually becomes negligible and the remaining proportion of labeled cells approximates the true inagglutinable fraction. In the present experiments, unlike those previously reported, human anti-A and anti-B sera were used, rather than lima bean anti-A lectin. The sera were obtained from donors selected for the production of commercial typing sera, unselected sera having proved unsatisfactory. In all experiments with anti-B, B cell carrier was used, whereas A_1 carrier was used in all those with anti-A.

Isotope dilution curves with anti-B were obtained for the AB and B persons in three of the families (Fig. 1). The AB persons show no consistent age effect. The results in family II suggest the possibility of a non-B frequency characteristic of the B allele, with Br.O. having received his mother's B allele and Wa.O. his father's. This interpretation does not find further support in family III, nor in the non-A proportion in family II; we regard the result as fortuitous. The B homozygotes are distinct; De.O. has a non-B frequency 10 to 20 times lower than in sibs and parents, and Ka.F. has 5 to 10 times lower.

The results with anti-A are shown in Fig. 2. Evidently the proportions of non-A and non-B cells can be quite different in the same AB individual. Again, no age effect is apparent. Families I and IV involved AB bloods inagglutinable by anti-A₁, but partially agglutinable by anti-A, a high proportion of free cells being evident on slide tests. These individuals are phenotypically A_3B , but the genotypes are questionable because the A_2B genotype has been reported, in certain pedigrees, to yield a phenotype resembling A_3B (3). For our present purposes the subgroup



29 DECEMBER 1961

allele will be provisionally called A_3 . In family I the result with Ch.N. was technically unsatisfactory, but the subgroup individuals appear to show different isotope dilution curves. In family IV the A₈B phenotypes are remarkably constant. The inagglutinable frequencies in the A1A3 persons (in contrast to those in B homozygotes) are quite consistent with independent behavior of the alleles; that is, they approximate the product of the non-A proportions in the A₁B and A₃B persons. The similarity of phenotypic expression of the A subgroup allele in family IV, whether in the presence of the B allele or in the minor cell population that has lost A₁, suggests that in this instance it is appropriate to designate the subgroup allele as A₃.

These results do not exclude an age effect, but they show that an age-independent component is preponderant. Two age-independent sources of mutant cells can be imagined: first, mutant stem lines established early, perhaps during the prenatal period, and second, mutations occurring in the clones that intervene between the stem lines and the erythrocytes. The mutational hypothesis now requires the assumption that mutant cells arising from either or both of these sources greatly outnumber those that arise from the accumulation of mutant stem lines during postnatal life.

The results with the B homozygotes present further difficulties for the mutational hypothesis, since statistically independent events in the two B alleles can account for only a minor fraction of the cells that have apparently lost the B phenotype. It would be necessary to assume that the mutations in homozygous cells are highly correlated, or that genotypic selection has altered the stem cell populations in the required manner, or that a large proportion of exceptional cells in homozygotes are cell phenocopies. The presence of exceptional cells in homozygotes and the inequality of non-A and non-B proportions in AB persons is evidence against somatic segregation (4) as a major cause of automosaicism. Finally, the magnitude of the difference in exceptional cell proportions between homozygote and heterozygote suggests that the isotope dilution method may permit determination of homozygote frequencies in population surveys.

> K. C. Atwood* F. Jay Pepper

Division of Biological Sciences, University of Chicago, Chicago, Illinois

2101

References and Notes

- 1. K. C. Atwood and S. L. Scheinberg, Science K. C. Atwood and S. L. Scheinberg, Science 127, 1058 (1958), abstr.; J. Cellular Comp. Physiol. 52, suppl. 1, 97–123 (1958); S. L. Scheinberg and R. P. Reckel, Genetics 44, 533 (1959), abstr.; K. C. Atwood and D. Megill, Science 130, 1411 (1959), abstr.; S. L. Scheinberg and R. P. Reckel, Genetics 45, 621 (1960); —, Science 131, 1887 (1960).
 K. C. Atwood and S. L. Scheinberg, Science 129, 963 (1958)
- **129**, 963 (1959). 3. L. E. Young and E. Witebsky, J. Immunol.
- **51**, 111 (1945). H1 (1943).
 R. B. Goudie, Lancet 272, 133 (1957); K. C. Atwood, Proc. Natl. Acad. Sci. U.S. 44, 1054
- (1958). Present address: Department of Microbiology,
- University of Illinois, Urbana.

18 September 1961

New Source of the j₂ Gene Governing Jointless Pedicel in Tomato

Abstract. A multiple-flowered, jointless pedicel, single plant appeared in the tomato variety 146 in 1958. The gene governing the jointless pedicel character was found to be identical with the j_2 gene found in a tomato strain from the Galapagos Islands. The character is considered to be a mutant and of potential value in the development of mechanically harvestable tomatoes.

An off-type, single plant of the tomato variety 146 (1), with multiple flower clusters, was found in a commercial field in 1958. Plants grown in 1959 from seed of this plant produced flower clusters with jointless pedicels. The clusters were bifurcate, differing from those of plants with the j_1 gene (2). They were similar in appearance to the jointless pedicel clusters of strain LA 315 received from C. M. Rick. Strain LA 315 was derived from LA 166, a collection of Lycopersicon pimpinellifolium from Indefatigable Island of the Galapagos Island group (3). The jointless pedicel character of strain

LA 315 was reported by Rick to be different from that determined by the gene j_1 and was assigned the symbol j_2 (3, 4). As far as we know this has been the only report of the j_2 gene.

A series of crosses was made between the 146 selection and normal jointed, jointless j_1 type, and jointless j_2 type, using LA 315 for the last cross, with the results shown in Table 1.

Crosses 1, 2, and 3 indicated that 146 selection pedicel type was recessive to normal jointed, was conditioned by a single factor, and was like j_2 pedicels in appearance. Crosses 4, 5, and 6 indicated that 146 selection was not identical with jointless j_1 , because the F1 was jointed, both jointless types appeared in the F₂, and the two backcross populations were distinct, each approaching 1:1 ratios for jointed and jointless, with the backcross parent pedicel type only represented.

Crosses 7, 8, and 9 established the identity of the j_2 gene for jointless pedicel in 146 selection and LA 315, with all populations having j_2 pedicels.

In contrast with clusters of 1 to 6 flowers in most j_1 tomato strains or varieties, the j_2 clusters, as grown at Riverton, New Jersey, have from 7 to 30 or more flowers per cluster, although actual fruit set is usually 1 to 4 per cluster. The 146 selection differs from LA 315 in lacking the large and leaflike calyces and the ovoid fruit described for the latter (3, 4). The character behaves normally in crosses and has been combined with both determinate, sp and dwarf, d characters and with the double recessive, d, sp. Germination of seed is normal.

The jointless pedicel character results in a minimum of stems remaining on picked ripe fruit. This characteristic may be useful in the development

Table 1. Classification of tomato plants for pedicel type.

Cross	Parent plants	Gener- ation	No. of plants of phenotype		
			Jointed	Jointless j ₁ type	Jointless j ₂ type
1	146 sel. × Jointed	F 1	11	0	0
1	146 sel. × Jointed	F_2	72	0	16
2	146 sel. $F_1 \times Jointed$	BC1	41	0	0
3	146 sel. $F_1 \times 146$ sel.	BC1	12	0	10
4	146 sel. \times Jointless i_1	\mathbf{F}_1	6	0	0
4	146 sel. \times Jointless i_1	\mathbf{F}_2	18	3	1
5	146 sel. $F_1 \times Jointless i_1$	BC1	13	7	0
6	146 sel. $F_1 \times 146$ sel.	BC1	12	0	10
7	146 sel. \times LA 315 i_2	\mathbf{F}_1	0	0	4
7	146 sel. \times LA 315 i_2	\mathbf{F}_2	0	0	46
8	146 sel. $F_1 \times 146$ sel.	BC1	0	0	21
9	146 sel. $F_1 \times LA$ 315 j_2	BC1	0	0	. 22

of mechanically harvestable tomato varieties (5).

The origin of the j_2 character in 146 is unknown. The LA 315 j₂ was not grown in local experimental plantings until 1960, whereas the 146 selection appeared in 1958. This eliminates the possibility that a chance field pollination from LA 315 introduced the gene to the 146 variety.

GEORGE B. REYNARD Campbell Soup Company, Agricultural Research Department, Riverton, New Jersey

References

G. B. Reynard, Proc. Plant Sci. Seminar. 93 (1960), Campbell Soup Co., Camden, N.J.
 L. Butler, J. Heredity 27, 25 (1936).
 C. M. Rick, Am. J. Botany 43, 687 (1956).
 ..., Tomato Genetics Cooperative Rept. No. 6, 23 (1956).
 S. K. Ries and B. A. Stout, Proc. Am. Soc. Hort Sci 75 632 (1960).

Hort. Sci. 75, 632 (1960).

7 August 1961

Sources and Isotopic Composition of Atmospheric Sulfur

Abstract. In nonindustrial areas the prime source of SO_4^{--} in rain and snow is atmospherically oxidized H₂S that is produced predominately along coastal belts by anaerobic bacteria. The δ S³⁴ analyses of atmospheric SO_4^{--} vary from +3.2 to +15.6 per mil in contrast to +20.7 per mil for sea water SO₄⁻⁻. Contrary to previous studies based on CI^{-}/SO_{4}^{-} ratios, sea spray SO_{4}^{-} is a minor Cl⁻/SO₄ ratios, sea spray SO_4^{--} source.

Studies on the relationship between variations in isotopic composition of sulfur and the genesis of sulfide minerals (1)have led us to encounters with the complexities of the biogeochemical cycle of sulfur. One significant portion of this cycle is the role of sulfur in the atmosphere.

Sulfur occurs in the atmosphere predominately, if not exclusively, as SO_4^{--} in aerosols and in SO2 and H2S gas. The second form, and in all probability the third, is oxidized to SO₃, which in the presence of moisture subsequently forms hygroscopic SO_4^{--} nuclei. The SO_4^{--} in aerosols is returned to the earth in precipitation of rain and snow-in variable concentrations between essentially 1 to 10 mg of SO_4^{--} per liter of precipitation.

Three sources for SO_4^- in the atmosphere have been suggested. The first and most obvious source, especially to urbanites, is industrial SO₂, which, as shown by Junge (2), is certainly quantitatively insufficient to account for the total amount of SO_4^{--} in the atmosphere. The second source is H₂S produced by anaerobic

SCIENCE, VOL. 134