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SP' antigen
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Red Cell Sp₁ Antigen Change Associated with *in vivo* Poly-Agglutinability¹

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Sp₁ is the name given by MARSH and JENKINS [2] to the high-frequency red cell antigen recognized by some cold auto-agglutinins. One of the serological features distinguishing anti-Sp₁ from anti-I is the difference in reactivity after treatment of the red cells with the enzymes, ficin and papain. Anti-I is enhanced by using enzyme-treated red cells but the Sp₁ red cell receptor is inactivated. MARSH and NICHOLS [3] subsequently reported *in vitro* experiments showing that some bacterial filtrates containing proteolytic enzymes caused activation of the red cell T-antigen but at the same time caused denaturation of Sp₁. All of these antigen changes are believed to result from bacterial enzyme activity.

This report presents a case of *in vivo* poly-agglutinability which was accompanied by loss of the red-cell Sp₁ antigen.

The patient, a woman aged 47 years, had a pelvic abscess. *Clostridium perfringens* and *Clostridium butyricum* were isolated from the wound. Her red cells were poly-agglutinable (T-active) and were strongly agglutinated by group AB serum and by a saline extract of *Arachis hypogea* [1]. We were unable to establish the Sp₁ status of the patient's red cells before they became poly-agglutinable.

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MARSH and JENKINS [2] in tests on the red cells of more than 20,000 persons did not find any to be Sp₁-negative, and it is reasonable to assume, therefore, that this patient was Sp₁-positive.

To avoid difficulties caused by normal serum anti-T, a heat eluate into saline from a serum containing high-titer anti-Sp₁ was prepared by absorption and elution with group O red cells. The eluate gave clear reactions but with a prozone effect, a phenomenon we have encountered previously in sera of this specificity. Titrations against various red cells showed the characteristic reactions of anti-Sp₁, but gave no reactions with the patient's poly-agglutinable red cells (table I).

Table I. The results of titrating an eluate containing anti-Sp₁ against various red cells

Dilutions of eluate in buffered saline pH 6.0								
Cells	1/1	1/2	1/4	1/8	1/16	1/32	1/64	Score
Group A ₁ untreated	+	++	++	++	+	±	W	39
Group O untreated	+	++	++	++	+	+	±	42
Group O papainized	W	—	—	—	—	—	—	2
Poly-agglutinable	—	—	—	—	—	—	—	0

Tests at 4°C

The patient's red cells showed slightly enhanced reactions when used in comparative titrations against normal red cells, with high-titer anti-I serum, and the results closely paralleled those obtained in the *in vitro* studies of MARSH and NICHOLS [3].

Disease processes may modify the activity of a number of red-cell antigen determinants. These results show that loss of cell Sp₁ antigen, probably caused by bacterial enzyme activity, is another disease-associated cell antigen change.

References

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