The Lewis Blood Group System and Secretor Status

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**THE LEWIS BLOOD GROUP SYSTEM AND SECRETOR STATUS**

Le was first described in 1946 by Mourant, when it was named L. Le was first described in 1948 by Andresen.

In 1945, Sneath and Sneath observed that red cells lacking Le and Le will take up these antigens from plasma containing them. Equally, red cells expressing either Le or Le will give these up to plasma lacking them. In other words, the antigens were found to be soluble.

Between 1948 and 1951, Grubb and Brendemoen independently observed that the saliva of Le(−) individuals strongly inhibited anti-Le, and that the saliva of the majority of Le(+) individuals also inhibited anti-Le, but did so less strongly.

In 1956, Möllison et al demonstrated that this phenomenon also occurred in vivo. From this it can be seen that:

- Lewis antigens are not intrinsic to red cells.
- They are located on type 1 glycosphingolipids that are adsorbed onto the red cells from the plasma.
- Lewis, therefore, is not strictly speaking, a red cell blood group!

**Genotypes and phenotypes and their relationship with the Secretor gene**

At a basic level, if you do not inherit a Lewis gene (LE, or, as it is now named, FUT3), whether you inherit a Secretor gene (SE, or, as it is now named, FUT2) of not, you will be Le(a−b−); if you do inherit a Lewis gene, but you do not inherit a Secretor gene, you will be Le(a−b−). If you inherit a Lewis gene, and you inherit a Secretor gene, you will be Le(a+b+).

Table 1. The interaction between the LE/FUT3 gene and the SE/FUT2 gene, and their influence on the Lewis phenotype.
based molecules, and so, like the A, B and H antigens, they are not direct gene products. The gene products are α-1-4-fucosyltransferase (LE/FUT3) and α-1-2-fucosyltransferase (SE/FUT2). The LE/FUT3 direct gene product cannot function, unless the SE/FUT2 direct gene product is present (rather in the same way that the A and B gene products cannot function, unless the H gene product is present and functioning). A schematic of the two carrier molecules can be seen in Figure 1.

There are six antigens recognised by the International Society of Blood Transfusion (ISBT) within the Lewis Blood Group System. These can be seen in Table 2.

Lewis phenotype frequencies

The normal figures for Lewis types can be seen in Figure 2; however, for various reasons, such as infancy and pregnancy (see below for explanations), such figures should only be taken as true for individuals from the age of approximately two and upwards, and who are not pregnant.

Lewis antigens in newborns and infants

Most newborn babies type as Le(a-b-), for the first month of their life, as the production of Lewis fucosyltransferase is at very low levels. If they are going on to produce Lewis antigens in pregnancy, it has been known for many years that the increased incidence of the Le(a-b-) phenotype is due to a weak or mutated Secretor status. This is due to a weak or mutated Secretor status.

Lewis antigens in pregnancy

It has been argued that the Le(a-b-) phenotype in adults is the result of a ‘passive’ process, that is, the Le(a-b-) phenotype is due to the absence of Lewis fucosyltransferase activity, resulting in the absence of the Le(a-b-) phenotype. However, it is now thought that this is not so, and that the Le(a-b-) phenotype is due to the absence of Lewis fucosyltransferase activity in the bone marrow.