

KIR / HLA-C

Genotyping

KIR / HLA-C genotyping allows determining the risks of the embryo being rejected by the maternal immune system



The human immune system (IS) is prepared to eliminate anything that it recognizes as foreign in order to offer appropriate protection against pathogens. During pregnancy, the mother's IS faces an element –the embryo– which presents “non-self” antigens from the father in cases of pregnancies with their own eggs; or from the father and the donor in oocyte donation cases. The ability of the mother's IS to “tolerate” the embryo, despite recognizing it as foreign, is essential to achieve a successful pregnancy.

Recent studies have revealed that alterations in the immunoregulatory mechanisms of the maternal response to the fetus may be responsible for some cases of female infertility, implantation failure or miscarriage¹⁻⁴.

KIR / HLA-C genotyping is a genetic test that allows to assess the risks of the embryo being rejected by the maternal immune system, and thus to direct medical interventions in order to achieve a successful pregnancy.

The process



Collection of blood from the patient and the partner/donor



Shipment at ambient temperature



Genotyping KIR and HLA-C



Result in 15 working days



KIR / HLA-C genotyping

A quality embryo, a uterus prepared to receive it and an interaction between the two that allows the embryo to nest and develop correctly are essential for gestation to develop correctly. For this to take place, the maternal immune system must be able to tolerate the embryo. This is achieved by the interaction of the embryo with the immune cells present in the uterus (called uterine NK cells). These cells recognise the embryo through receptors on the surface of the embryo called KIR, which bind to identification fragments on the surface of the embryo called HLA-C, one inherited from the mother and one inherited from the father.

Studies have shown that certain combinations of KIR and HLA-C are more likely to lead to problems in pregnancy¹⁻⁵.

KIR / HLA-C genotyping can determine whether there is a good match between the uterine KIR receptors and the HLA-C presented by the embryo. If this is the case, the maternal-fetal tolerance process will develop properly and the pregnancy will proceed without complications. If not, the embryo acceptance process will be compromised and with it the evolution of the pregnancy.

Benefits & value-added

- KIR / HLA-C genotyping will allow us to:
- Identify the cause of repeated miscarriages and implantation failure.
- Prevent possible complications during pregnancy, such as pre-eclampsia.
- Decide on the optimal number of embryos to be transferred.
- Choose a donor compatible with the future pregnant woman, both in the case of sperm and oocyte donation.

Process in details

KIR / HLA-C genotyping is performed on the couple's DNAs. For this, a blood sample (collected in a purple lid, EDTA-tube) from each member of the couple is required.

In cases of oocyte or sperm donation, donor HLA-C genotyping is required.

Post-testing indications

In the field of fertility immunological incompatibilities may affect essential reproductive processes which, ultimately, may result in implantation failure, recurrent miscarriages and preeclampsia. Several studies have shown that severe pathologies associated with pregnancy increase significantly on pregnant women with an incompatible maternal-fetal combination^{1,3,5}.

When the combination of maternal KIR and HLA-C of the future embryo is incompatible, it is advisable to:

- Transfer a single embryo to avoid exposing the mother to a double load of incompatible HLA-C.
- Immunomodulatory treatment can also be prescribed to the expectant mother to increase maternofetal tolerance.
- In cases of oocyte or sperm donation, donor HLA-C genotype and maternal KIR matching is recommended to ensure maternal-fetal compatibility.

1. Moffet A, Colucci F. J Clin Inves. 2014; 124 (5): 1872-1879.

2. Alecsandru D y col. Hum Reprod. 2014 Dec; 29(12): 2637-43.

3. Alecsandru D, García-Velasco JA. Fertil Steril. 2017 Jun; 107(6): 1273-1278.

4. Morin SJ y col. Fertil Steril. 2017 Mar; 107(3): 677-683.e2.

5. Moffet A, Hiby SE. Placenta. 2007; 28 (Supp A): S51-6.

