

# **Implantation is irrelevant for the moral status of the embryo**

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# Abstract

We argue that although implantation marks biologically an important step in the embryonic development, it is irrelevant for the moral status of the embryo, just like it is for the sex or for Down syndrome.

# A main objection against the moral status of the pre-implantation embryo

“In the fertilized egg the genetic program is certainly present. However, for the program’s processing the embryo requires the symbiosis with the maternal organism. This is indispensable. The implantation (by which the embryo comes directly in cellular contact with another individual) is biologically one of the most discontinuous things one can imagine. [...] The human embryo acquires the full developmental potentiality, and therefore the moral status, only after implantation and interaction with the mother’s organism.”

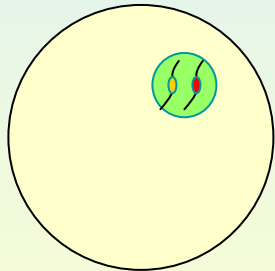
See: Ch. Nüsslein-Volhard, *Das Werden des Lebens*, Munich: Verlag C.H. Beck, 2004, p. 189-191. 1995 Nobel Laureate in Medicine.

In this presentation we prove the inconsistency of the claim that “the human embryo acquires the moral status only after implantation”.

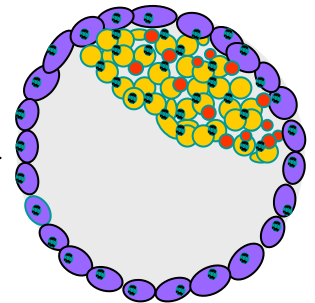
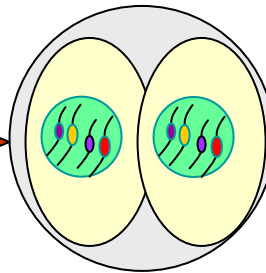
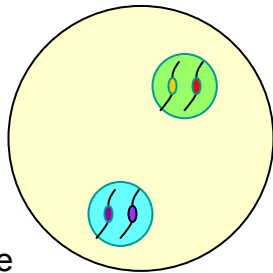
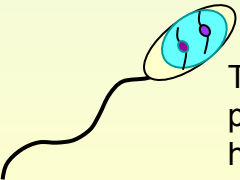
# Genetic information

In sexual reproduction, each individual inherits one set of chromosomes from each parent: for each maternal chromosome, there is a corresponding paternal one. The chromosomes contain the **genetic information**. Normally there are two copies of each gene: one copy in a maternal chromosome and another copy in the corresponding paternal one.

The egg cell or oocyte provides the maternal genome consisting in half of the chromosomes



The sperm cell provides the paternal genome consisting in half of the chromosomes



For the sake of simplicity we do not represent the so called *second polar body*, a cell containing the other half of the chromosomes of the oocyte, which are discarded only after fertilization.

# Human chromosomes

The nuclei of human cells usually contain 23 pairs of chromosomes (in total 46 chromosomes), one pair is formed by the **2 sex chromosomes**.

In **females** the sex chromosomes consist in two **X chromosomes**: one X of maternal origin, and one X of paternal origin.

In **males** the sex chromosomes consist in one **X chromosome** of maternal origin, and one **Y chromosome** of paternal origin.

Each maternal chromosome carries the same genes as the corresponding paternal chromosome, however in males some genes of the **X chromosome** do not have a corresponding copy in the **Y chromosome**.

# Implantation is irrelevant for determining the baby's sex

Individuals with the abnormality called *Turner's syndrom* have only **one X chromosome**, and therefore 45 (instead of 46) chromosomes. These individuals are **females**.

Individuals with the abnormality called *Klinefelter's syndrom* have **two X chromosomes** and **one Y chromosome**, and therefore 47 (instead of 46) chromosomes. These individuals are **males**.

This means that the presence of the **Y chromosome** is decisive for unleashing the developmental program that leads to a baby boy.

The fact **that an embryo becomes a girl and not a boy** depends on the embryonic information, but **does not depend at all on implantation**.

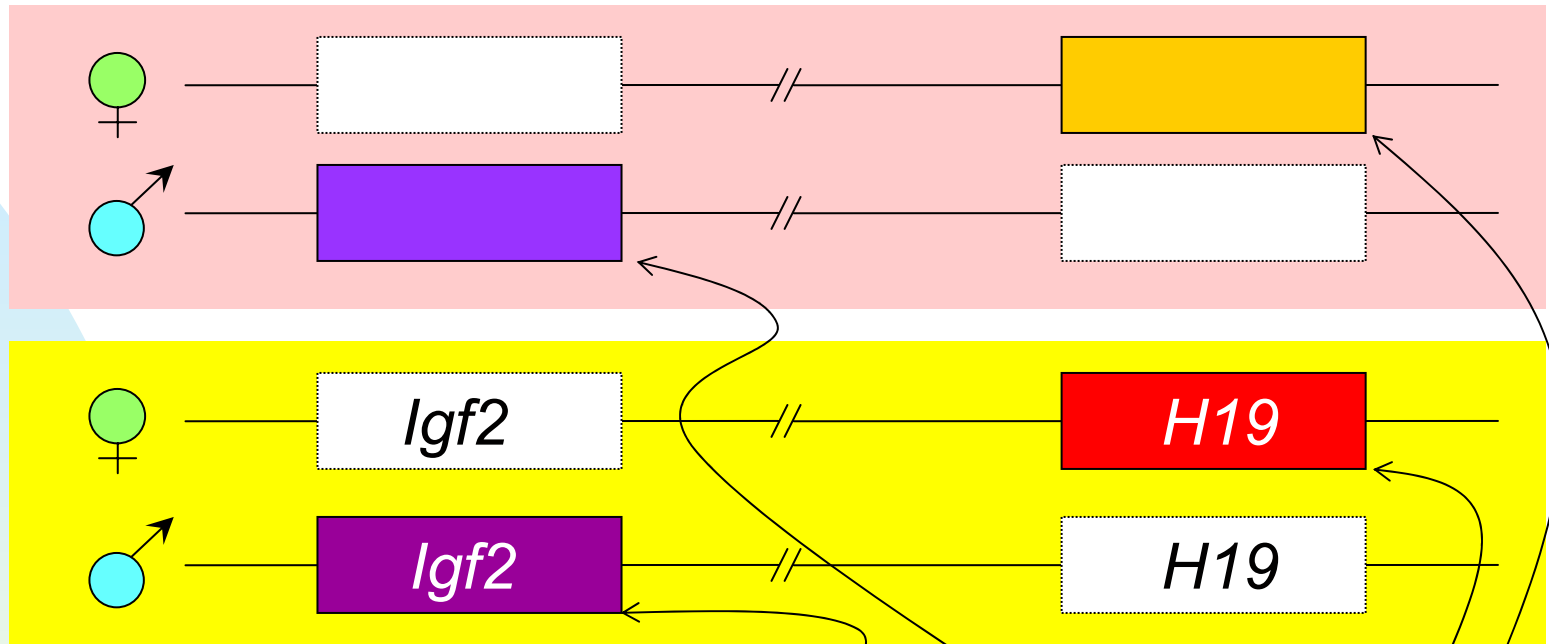
# Epigenetic information

The maternal and paternal genomes are not exactly equivalent, but are endowed with different *imprints* (chemical changes) **determining whether the copy of a gene becomes expressed or not** during embryonic development.

*Imprints* refine the genetic information through an epigenetic one. According to the **epigenetic information**, certain genes become expressed only from the maternal chromosomes, and other genes become expressed only from the paternal chromosomes.

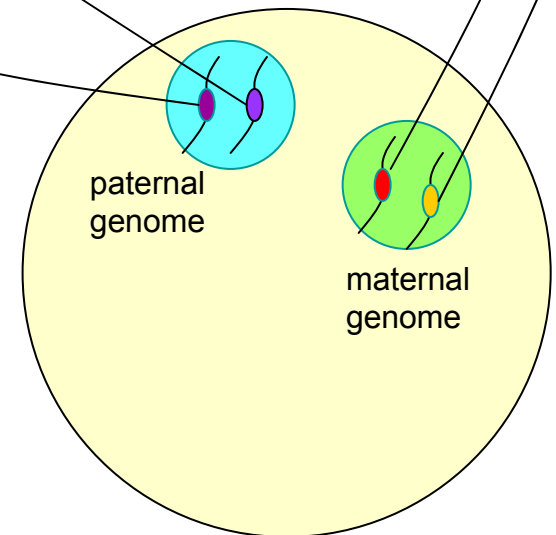
(Loebel and Tam 2004)

# The epigenetic state of the fertilized egg



In a normal mouse embryo, resulting through the fusion of a **sperm** cell and an **egg** cell, *H19* and several other genes become expressed only from the maternal chromosomes, and *Igf2* and several other genes become expressed only from the paternal chromosomes. Silent genes are represented in white.

(Kono et al. 1996)



# The epigenetic information determines how the embryo develops

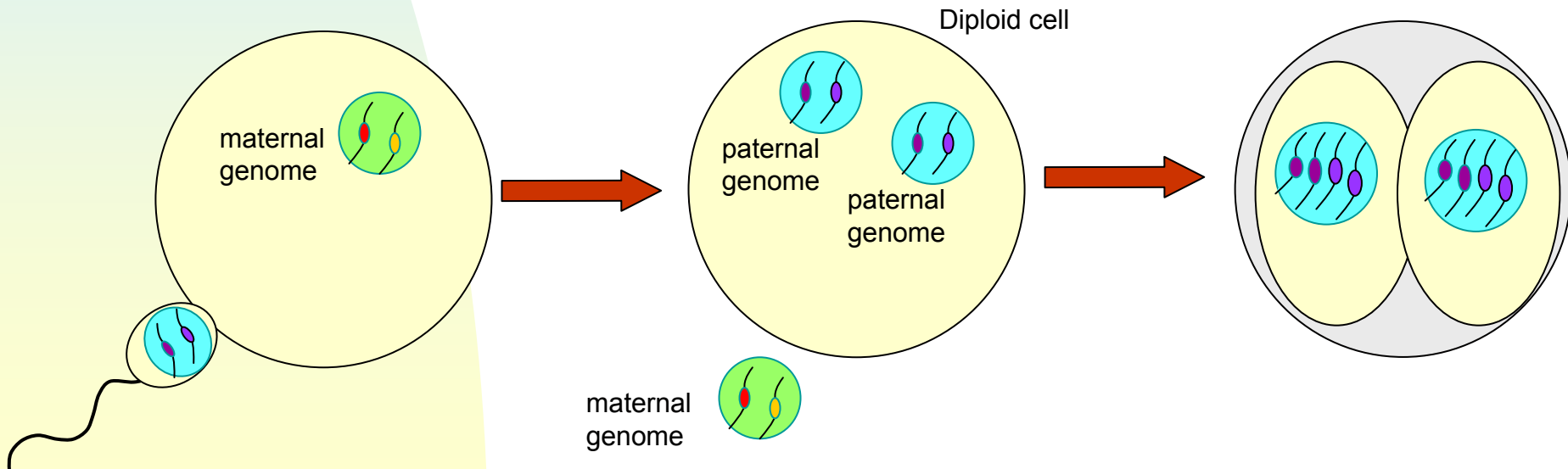
In order for normal development to take place, it seems necessary for the absence of activity of one gene in a gamete to be counterbalanced by the presence of the corresponding active gene from the other gamete.

In certain cases, development fails if there are two active copies of the same gene (one copy in the maternal and another copy in the paternal chromosome).

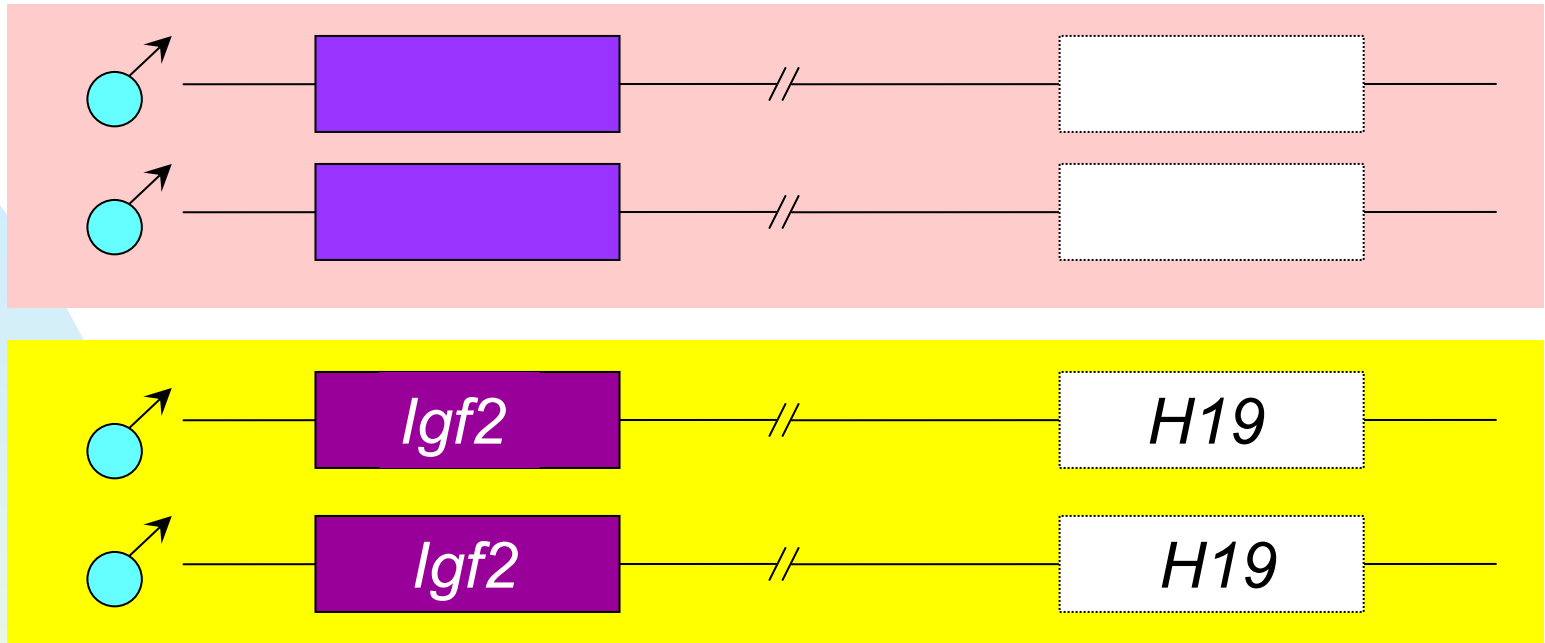
# Androgenotes

Abnormalities during the gametes fusion can produce an *androgenote*, i.e. a diploid egg cell containing only paternal genome.

*Androgenotes* arise through elimination of the maternal genome together with replication of the sperm cell chromosomes. (Sometimes they may also result through fusion with two sperm cells).



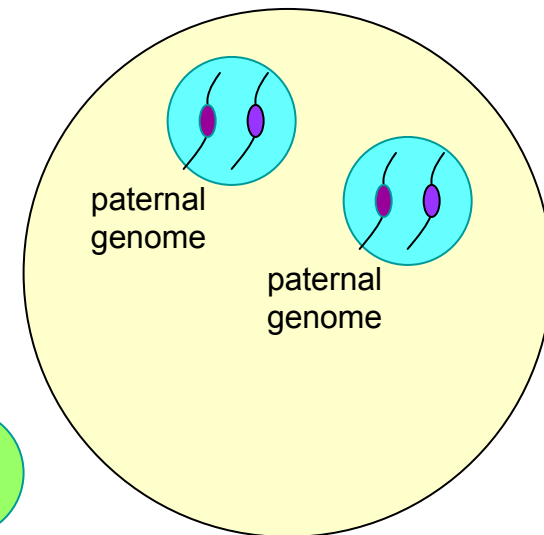
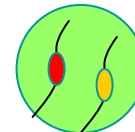
# The epigenetic state of *androgenotes*



In androgenotes, *Igf2* and several other genes become twofold expressed, whereas *H19* and several other genes remain inactive.

Such an *androgenote* cleaves and develops like an early normal embryo, but after implantation produces a *hydatiform mole*, without a proper embryo.

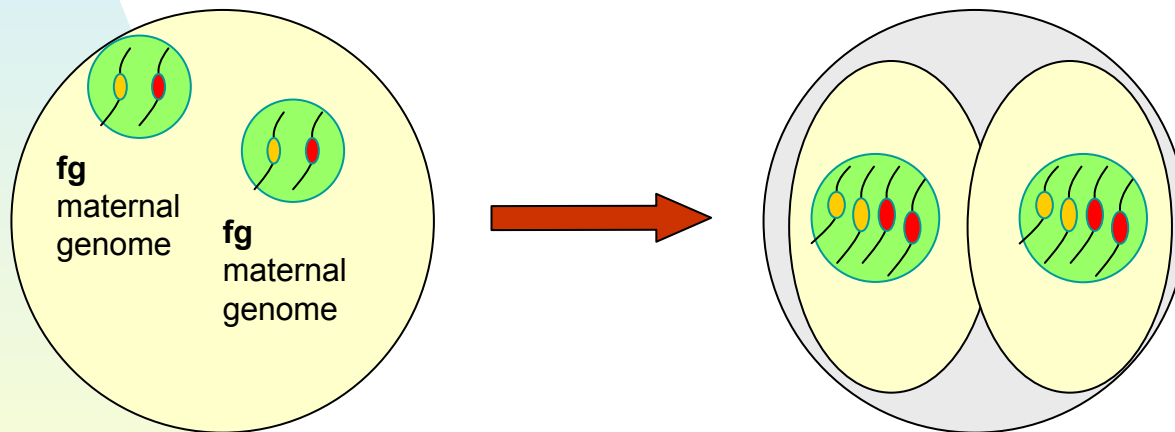
maternal genome



# *Standard parthenotes*

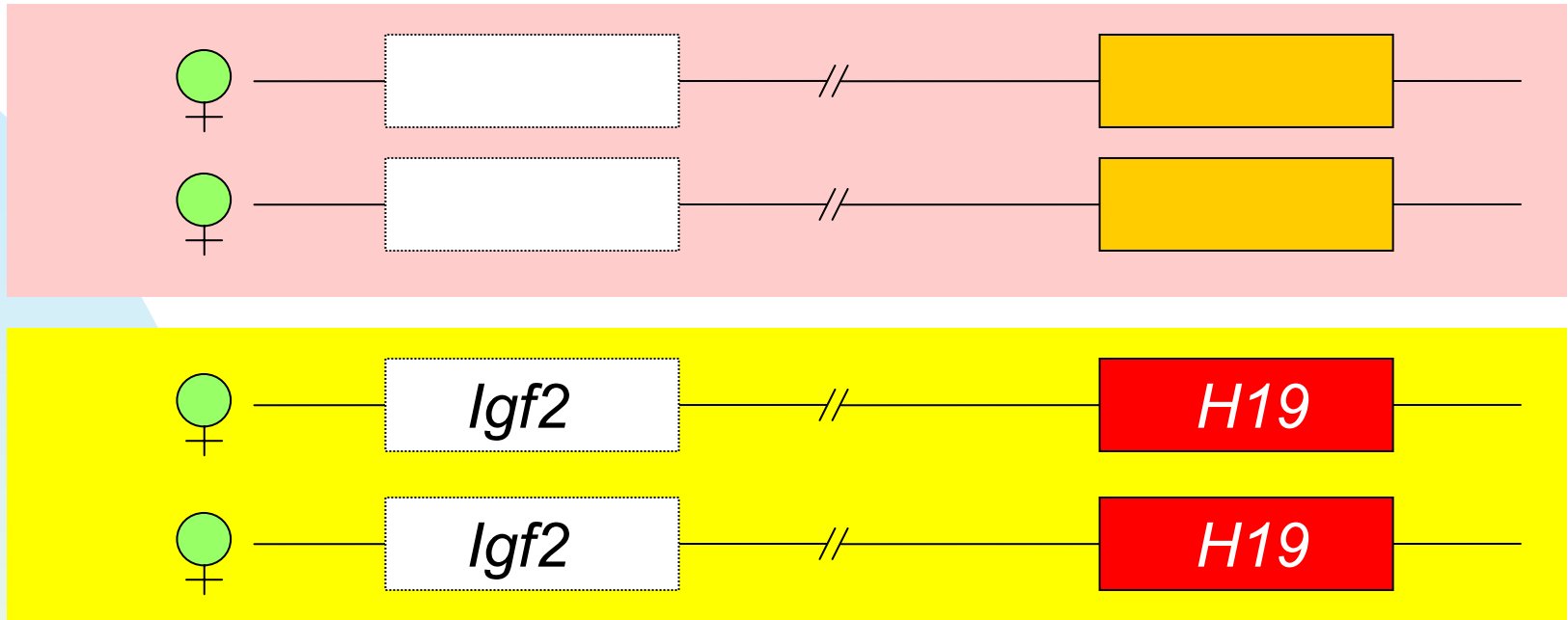
Fully-grown (fg) human secondary oocytes contain two sets of chromosomes, one of which is normally discarded within two hours of fertilization. Using a chemical treatment to prevent this, one produces diploid *parthenotes* with both sets of chromosomes coming from the mother.

(Rogers et al. 2004)



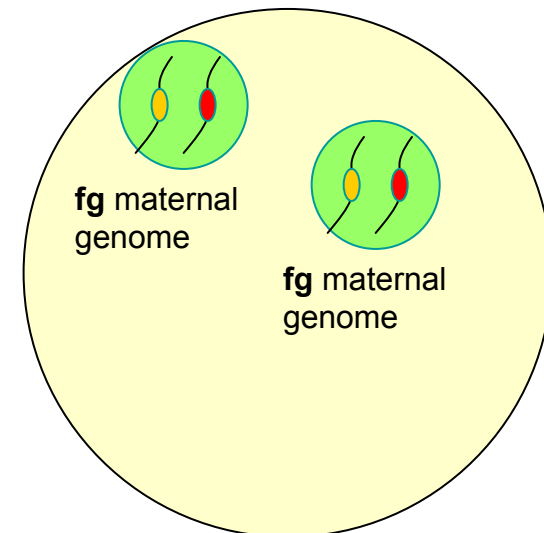
These parthenotes appear to undergo the same changes as naturally fertilized eggs: They undergo cleavage divisions for four or five days, and some form blastocysts. Observations in mouse models show that parthenotes are capable of undergoing early post-implantation development.

# The epigenetic state of *standard parthenotes*



In a egg containing only chromosomes from fully-grown (fg) oocytes, *H19* and several other genes become twofold expressed, whereas *Igf2* and several other genes remain inactive.

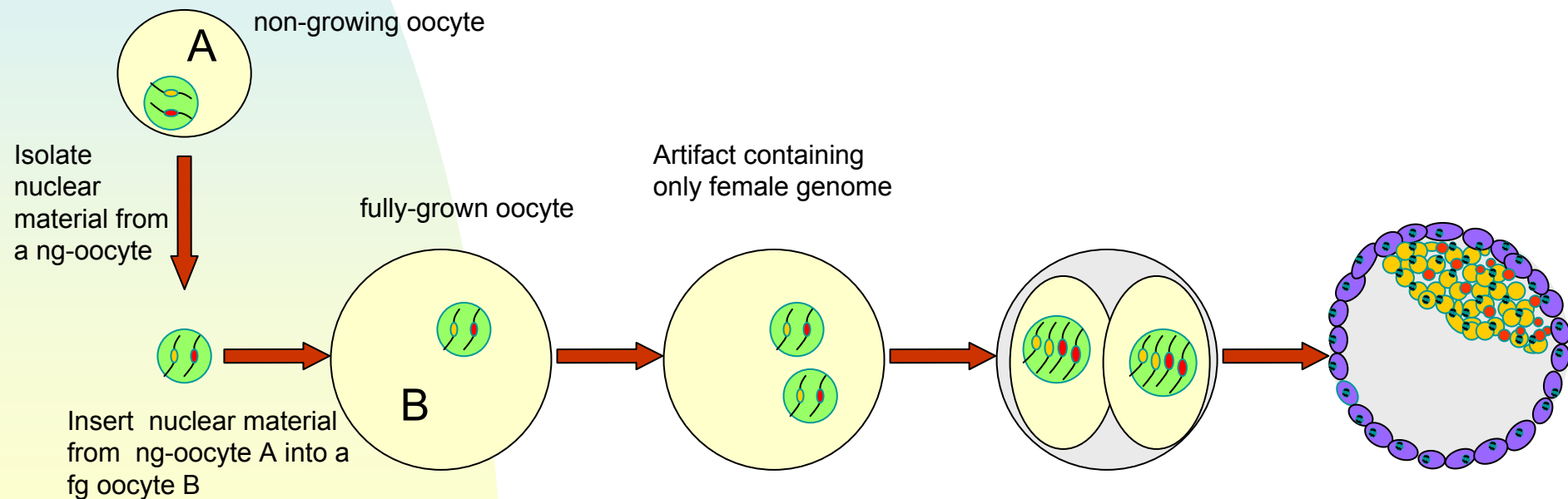
Such a mouse *parthenote* can develop only till **12 days**, reaching heart beating but not the neural activity responsible for spontaneous fetal motility.



(Gardner et al. 1990)

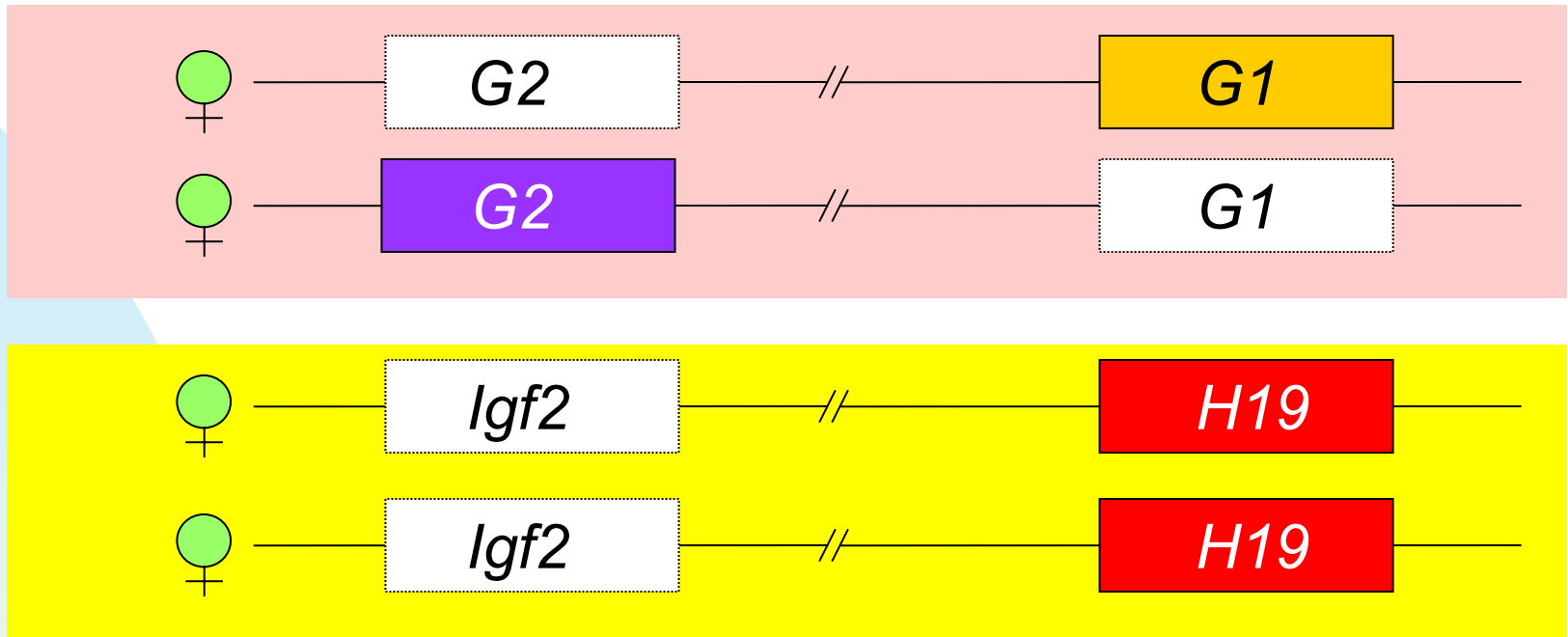
# "ng parthenotes" (mice)

Transferring the nucleus of a non-growing (ng) oocyte (egg cell obtained from a new born mouse) into a fully-grown oocyte one constructs a diploid cell, which contains two sets of chromosomes, all of them from maternal origin. Such artifacts are often called *ng-parthenotes*. We refer them as *H19/Igf2 mutants* too.



# The epigenetic state of “ng parthenotes”

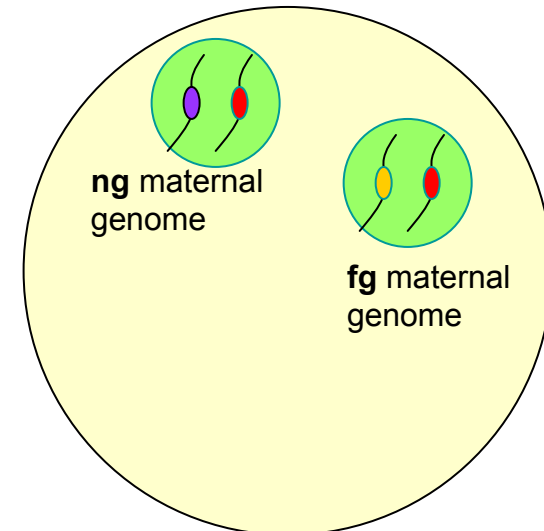
*H19/Igf2* mutants



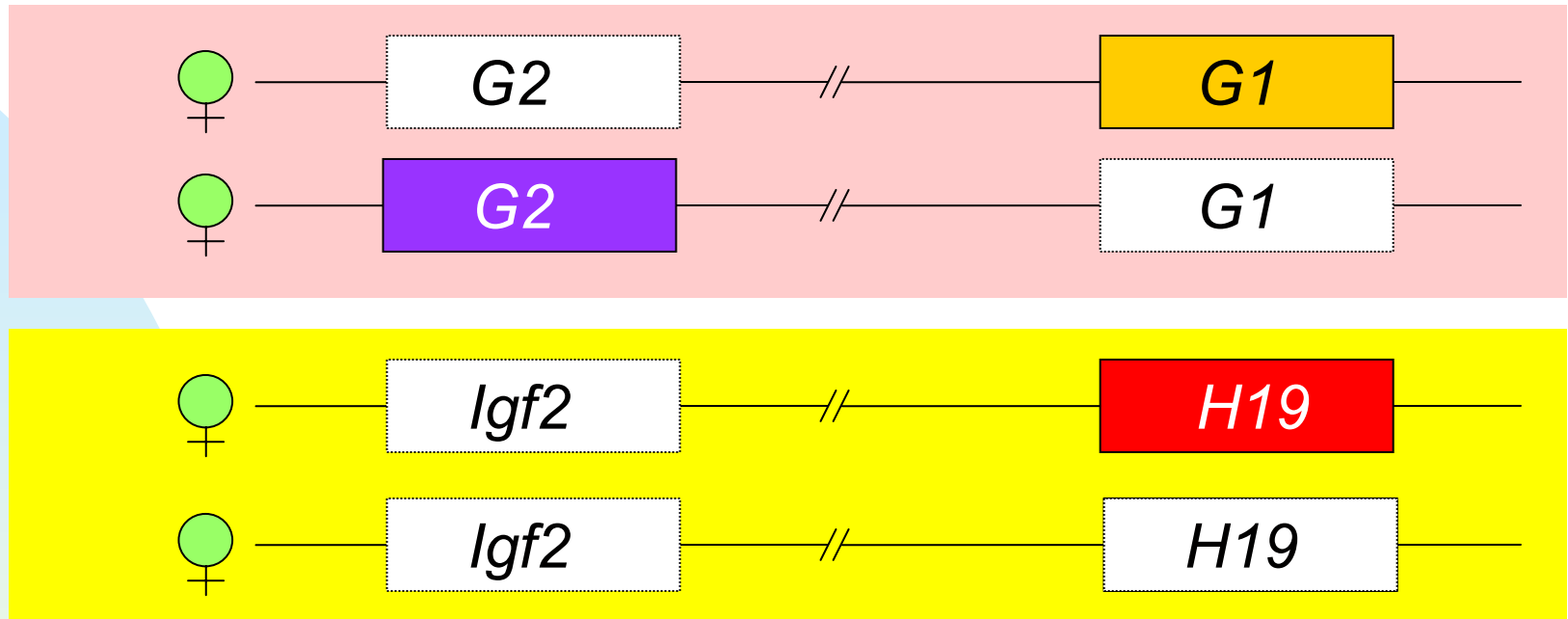
In a ng-parthenote several genes as *H19* are twofold expressed and both copies of several other genes as *Igf2* remain inactive, whereas some genes (*G1* and *G2*) are normally expressed (only one copy expressed).

Such a *H19/Igf2* mutant mouse or „ng parthenote“ develops till **13.5 days**. At **day 12.5** it shows breathing, legs and hands movements.

(Kono et al. 1996)



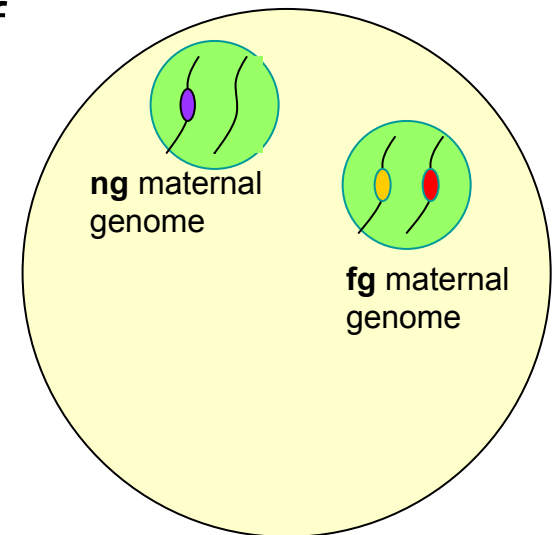
# The epigenetic state *Igf2* mutants



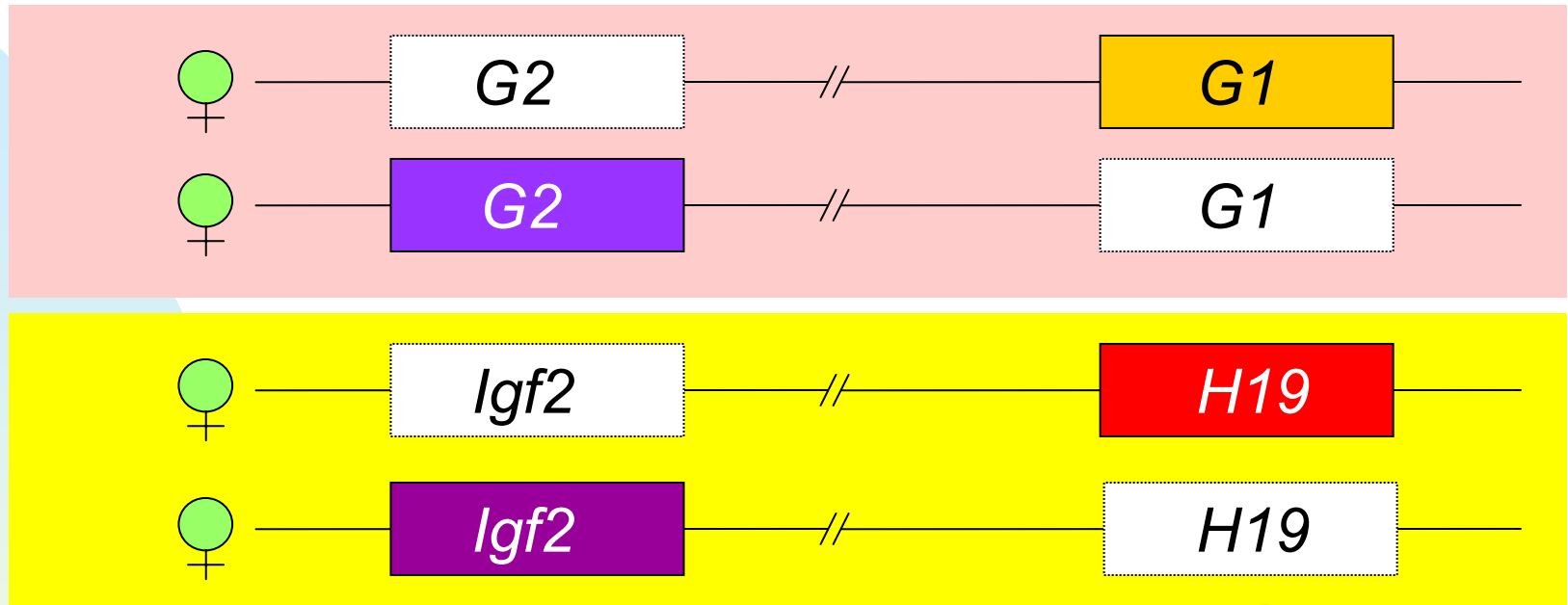
By knocking out the gene *H19* in the nucleus of the non-growing oocyte before transfer, one produces an artifact in which *H19* is monoallelically (single) expressed, whereas both *Igf2* copies remain inactive.

Such a mouse artifact develops till **17.5 days**.  
(Birth in mice happens at 19.5 days)

(Kono et al. 2002)

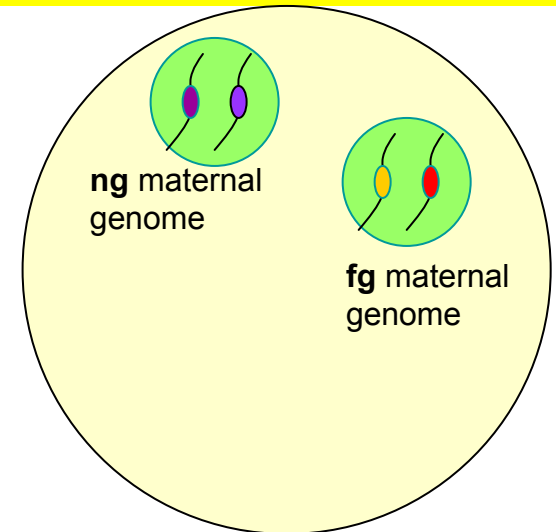


# Producing an epigenetic state equivalent to the fertilized egg's one



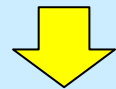
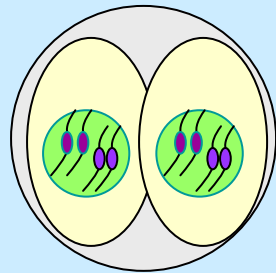
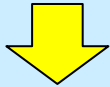
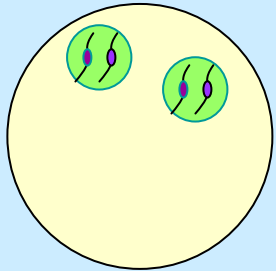
By knocking out the gene *H19* and activating the gene *Igf2* in the nucleus of the non-growing oocyte before transfer one produces an epigenetic state equivalent to the fertilized egg's one, i.e. such a mouse artifact develops to **birth** at **19.5 day**.

(Kono et al. 2004)



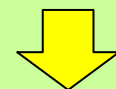
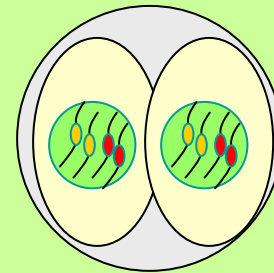
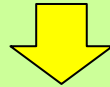
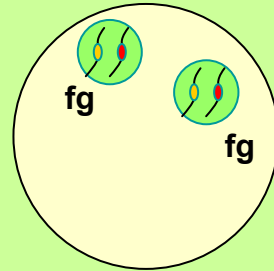
# Neural activity is specifically determined by embryonic information

## Androgenote (only paternal genome)



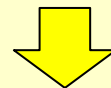
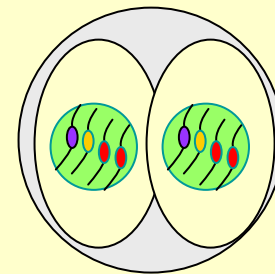
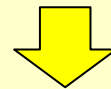
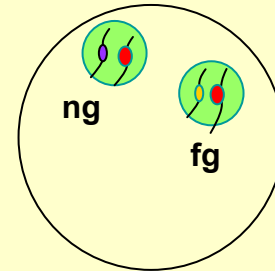
can implant,  
but produces a  
hydatiform mole  
without  
proper embryo

## Parthenote (only maternal genome)



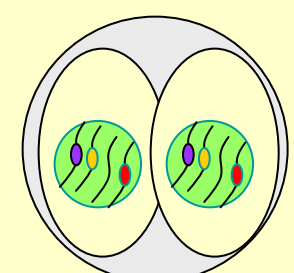
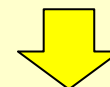
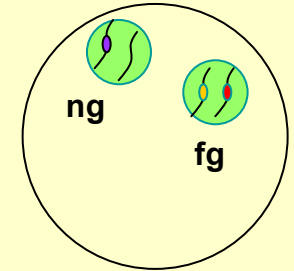
(in mice) can develop  
only till day 12  
exhibiting  
**heart beating** and blood  
circulation

## *H19/Igf2* mutant (“ng parthenote“: 2 active *H19*, 2 silent *Igf2*)



(in mice) can develop  
only till day 13.5.  
At day 12.5 shows  
**fetal motility**  
(neural activity)

## *Igf2* mutant (two silent copies of *Igf2*)



(in mice) can develop  
only till day 17.5,  
reaching advanced  
**fetal motility**  
(neural activity)

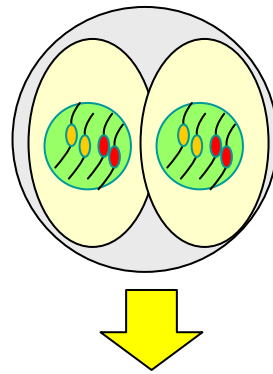
# Implantation is irrelevant for determining whether an embryo develops neural activity or only heart beating

The fact that an embryo exhibits neural activity **and not** only heart beating is determined by embryonic information, and not by the interaction with the mother's organism.

# What would be the fate of *standard human parthenotes* in a well functioning uterus?

Since human embryos exhibit well established heart beating and blood circulation after the 4th pregnancy week...

*Standard parthenote*



... one can assume that *standard human parthenotes* would probably develop till about 4-5 weeks, hence reaching heart beating and blood circulation stage, but would not go beyond

# Spontaneous fetal motility is considered the sign of neural activity

In humans, spontaneous movements, and in particular breathing movements, appear between the 7th and 15th week of pregnancy.

See: [De Vries, Visser and Prechtl 1982](#)

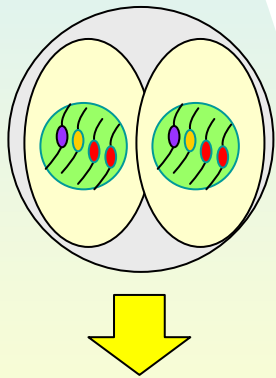
and the footages:

[www.createhealth.org](http://www.createhealth.org)

[www.mamma.ch/de/hintergrund\\_embryo.htm](http://www.mamma.ch/de/hintergrund_embryo.htm)

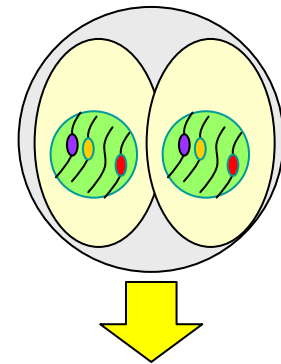
# What would be the fate of human equivalents to mouse mutants in a well functioning uterus?

Since in humans spontaneous movements, and in particular breathing movements, appear between the 7th and 15th week of pregnancy...



ng parthenote  
(*H19/Igf2* mutant)

... one can assume that a human ng parthenote would most likely develop further than 7 weeks, and hence reach spontaneous fetal motility



*Igf2* mutant

... one can assume that a human *Igf2* mutant would most likely develop further than 10 weeks, and hence reach advanced spontaneous fetal motility

# Spontaneous movements

By *spontaneous movements* we mean movements of the human body like arm, leg, and lip movements, head flexion and rotation, eye and breathing movements. Even if they are often unconscious and even intentionless, they are potentially will-directed movements, i.e., they can always be directed by the will when chosen. In contrast to these movements of the lips, tongue, eyes, fingers, etc., it is not possible to use “heart beating” to communicate messages.

Movements which are not caused by some stimulus external to the body, but which are strongly programmed by biology and cannot be brought under the immediate control of the will, we call *autonomous* movements.

Another kind of movement that escapes the control of the will is a nerve *reflex* reaction that has an external cause. Neither autonomous movements nor reflexive ones are meant to be connoted by our term “spontaneous”.

(Huarte and Suarez 2004)

# Assumption about the human soul

(derived from the standard definition of death)

In accordance with the nowadays accepted clinical criteria for stating death, we assume that:

- The human soul cannot be separated from the biological processes inducing the neural activity responsible for spontaneous movements and in particular, spontaneous breathing.
- A human organism exhibiting heart beating and blood circulation but without potentiality for performing spontaneous movements is not a human person.

# Conclusion

Neural activity is determined by **embryonic information** (epigenetic state), just like baby's sex and Down syndrome are determined by **embryonic information** (Y chromosome and trisomy 21, respectively).

Accordingly, although the interaction with the mother's organism (after implantation into the uterus) marks biologically an important step in the embryonic development, this interaction is irrelevant for the embryo's moral status, just as it is for the sex or for Down syndrome.

# May the moral status of an embryo be affected by epigenetic alterations?

We assume that a genomic anomaly that directly inhibits the emergence of the neural activity responsible for spontaneous motility (a DIANA anomaly\*) excludes the moral status of a person.

Consequently:

- ***Androgenotes and Standard Parthenotes are not persons***
- ***“ng parthenotes“ (H19/Igf2 mutants) and “Igf2 mutants“ are persons***

\* See *Presentation: “Diana genomic anomalies. Distinguishing between a sick embryo and a pseudo-embryo”*, at [www.embryoperson.org](http://www.embryoperson.org)

# Does an epigenetic alteration preventing implantation exclude the moral status of a person?

The fact that a human egg results in an abnormal and lethal growth at a very early developmental stage is not a sufficient condition to exclude the moral status of a person. The resulting biological entity may very well be a sick-embryo i.e., a sick person.

See *Presentation*: “Diana genomic anomalies. Distinguishing between a sick embryo and a pseudo-embryo”, at [www.embryoperson.org](http://www.embryoperson.org)



**End**