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Review article

# Maternal control of oocyte quality in cattle "a review"

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ABSTRACT

The oocyte is a central regulator of multiple aspects of female fertility, including ovarian follicular development and early embryogenesis. During its prolonged diplotene arrest, the oocyte is subjected to endogenous (i.e., reactive oxygen species from metabolism) and exogenous (i.e., heat stress, malnutrition) sources of damage-inducing factors, which may lead to a progressive deterioration of oocyte quality. A deficit in oocyte competence can lead not only to a failure of fertilization but also to a lower developmental rate after fertilization. Thus, an appropriate environment for growth and maturation of the oocyte, *in vivo* and *in vitro*, is critical to ensure optimal oocyte quality. The objectives of the current review are to give an overview of some maternal key factors that influence oocyte quality in cattle and describe some of the findings to date in the hope of obtaining competent oocytes that could be used for clinical and applied purposes.



**Contents**



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## 1. Introduction

The intrinsic ability of oocytes to resume meiosis, accept spermatozoa for fertilization, cleave after fertilization, and facilitate proper embryonic development that leads to the production of healthy offspring is defined as oocyte competency (Sirard et al., 2006). The acquisition of oocyte developmental competence is the major challenge for assisted reproductive technologies (ARTs) and still an issue that has always captured the attention of reproductive biologists. The origin of the oocyte and more specifically, the environment in which oocyte growth and maturation occur (Lequarre et al., 2005) has been implicated as an important determinant of the subsequent developmental competence of the oocyte. This area of reproductive biology is particularly important in cattle due to relatively high levels of early embryonic failure which are often related to poor oocyte quality because of the predominant role of maternal factors (Sirard et al., 2006). In cattle, estimates indicate that fertilization rate is 90% with an average calving rate of about 55%, suggesting an embryo/fetal mortality of about 35%; further, 70–80% of total embryonic loss occurs between days 8 and 16 after insemination (Diskin et al., 2006).

The oocyte undergoes a remarkably long and complex journey within the follicle prior to reaching MII in a state that renders it fully competent for fertilization which is a tightly regulated and rapid process that relies on maternal control mechanisms established during oocyte maturation. During its development, the oocyte becomes equipped with complete maternal molecular machinery that proves adequate for successful fertilization and early embryonic development. Any exogenous factors with negative influences or intrinsic oocyte abnormality will compromise the fertilizing potential of oocytes. As a prerequisite to obtain a healthy embryo is first obtaining a healthy oocyte. Thus, improved access to maternally regulated factors will allow a better understanding of the mechanisms and pathways regulating gametogenesis and embryogenesis that can possibly alleviate the reduced cattle reproductive performance observed worldwide, and may facilitate discovery of novel contributors to early embryonic development with the ultimate goal of improving oocyte quality.

## 2. Maternally inherited components

### 2.1. Maternal transcripts

In mammals, during prenatal development, oogonia undergo mitosis from primordial germ cells to form diploid oocytes (Wassarman and Albertini, 1994). These diploid oocytes enter into meiosis I, which ultimately generate haploid oocytes that can be fertilized with haploid sperm to form viable zygotes. In cattle, shortly before birth, oocytes are arrested at prophase of the first meiotic division (Wassarman and Albertini, 1994). The oocyte then should resume and complete meiosis as well as cytoplasmic maturation before successful fertilization can take place. The ability of oocytes to resume and complete meiosis, is acquired progressively during follicular and oocyte growth and is associated with nuclear and cytoplasmic changes (Mermillod et al., 1998).

The nuclear changes include chromatin condensation, nuclear envelope assembly, and cytoskeletal organization (Jones, 2004), whereas cytoplasmic changes include relocation and modification of organelles, acquiring functional $Ca^{2+}$ release mechanisms, capacity to decondense the chromatin of the fertilizing sperm (for review, Eppig, 1996), and the production and accumulation of maternal mRNAs in oocytes that are likely to be critical for oocyte maturation and successful pre-implantation development (Albertini et al., 2003). These transcripts are necessary to guide the early stages of embryonic development prior to the activation of embryonic transcription which is not robustly activated until the 8- to -16 cell stage (Camous et al., 1986). Thus, the maternal to embryonic transition depends heavily on post transcriptional control of maternal transcripts accumulated during oocyte maturation (Bettegowda and Smith, 2007). Since factors from the oocyte cytoplasm and products of its gene expression control many processes central to the early development of the whole organism, it is not surprising, therefore, that a decline in oocyte quality has a profound impact on the developmental competence of the embryo.

The maternal mRNA population is highly diverse, and supports a range of different functions during oocyte maturation and after fertilization, such as pronuclear formation and fusion (Philipps et al., 2008), the first cell division (Tang et al., 2007), embryonic gene transcription (Bultman et al., 2006) and cleavage-stage embryogenesis (Ma et al., 2006). The expression of embryonic messages gradually increases during the progression of embryonic development from the zygotic stage to the blastocyst stage (Memili and First, 2000) and the major onset of embryonic genome activation (EGA) occurs at a species-specific cell stage. In mice, it occurs at the 2-cell stage (ZGA, zygotic genome activation) (Kidder and McLachlin, 1985), at the 4-cell stage in the pig (Whitworth et al., 2004), at the 6- to 8-cell stage in monkeys (Schramm and Bavister, 1999), and at the 8- to 16- cell stage in humans (Tesarik et al., 1987) and bovines (Camous et al., 1986), suggesting potential species differences in mechanisms and mediators of the maternal-to-embryonic transition.

The maternal mRNA population also changes extensively during these periods (Zeng et al., 2004), and the vast change in maternal mRNA recruitment is not chaotic, but rather seems to follow a very carefully orchestrated pattern wherein large groups of maternal mRNAs may be