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Characteristics of morphological changes in developmentally retarded bovine embryos

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要旨

It has been shown that embryos with delayed blastocyst formation have a higher frequency of spindle structural abnormalities and lower implantation ability compared to embryos that quickly form a blastocoel in human IVF zygotes (Hashimoto et al., 2013). It has also been revealed that reduced mitochondrial function of embryos at morula stage results in delayed subsequent development (Morimoto et al., 2021). To understand the characteristics of morphological changes in developmentally delayed embryos, we measured the time required for development to each stage in bovine IVF zygotes with delayed blastocyst formation (Day 8 blastocysts) and embryos that formed the blastocoel at normal timing (Day 7 blastocysts).

The number of cells at the beginning of culture was 5.1 in Day 7 blastocysts and 4.9 in Day 8 blastocysts, respectively, with no statistical difference (p = 0.25). The time required to initiate compaction was 92 h and 99 h for Day7 and Day8, respectively, with no statistical difference. However, the compaction completion time in Day 8 blastocyst (122 h) was delayed (P<0.01) compared with Day 7 blastocyst (113 h). The time required for blastocyst development (183 h) was prolonged in the growth retarded embryos (P<0.01) compared with Day 7 blastocyst (161 h). The time required from 8 cell to compaction start (Day 7: 24 h and Day 8: 24 h, P=0.90) did not differ. On the other hand, a trend toward an increase in the time required from the start of compaction to the completion of compaction (Day 7: 21 h vs. Day 8: 24 h, P=0.06) was observed, and the time required from the completion of compaction to the start of blastocoel cavity formation (Day 7: 41 h vs. Day 8: 49 h, P<0.01), and the time required from the start of blastocyst cavity formation to blastocyst (Day 7: 8 h vs. Day 8: 13 h, P<0.01) were significantly increased.

In developmentally retarded embryos (Day 8 blastocysts), the delay was observed from the start of compaction, and the delay time tended to expand increasingly thereafter. In

bovine embryos, the activation of the embryonic genome is thought to begin at the 8-cell stage. We believe that insufficient gene expression related to the activation of the embryonic genome, especially mitochondrial function, is the cause of the developmental delay, and we plan to further our analysis focusing on mitochondrial function in the future.

Hashimoto et al., Hum Reprod. 2013;28:1528-35.
Morimoto et al., J Assist Reprod Genet. 2020;37(8):1815-1821.