Optimal levels of DHEA for pregnancy may be reduced by antibiotics

I suggest that the basis of the findings of Muanda and colleagues in their *CMAJ* article[1] is reduced dehydroepiandrosterone (DHEA) (i.e., antibiotics reduce arylsulfatase, which reduces availability of DHEA).

Dehydroepiandrosterone sulfate (DHEAS) is the precursor of the active molecule, DHEA. If sulfatase activity is reduced, DHEA is not optimally produced.[2,3]

It is my hypothesis that evolution selected DHEA because it optimizes replication and transcription of DNA (i.e., genes). Therefore, DHEA levels affect all tissues, and all tissues compete for available DHEA, especially the brain. (I think evolutionary selection of DHEA produced mammalia.[4])

Dehydroepiandrosterone naturally begins to decline around the ages of 20 to 25 years and reaches very low levels in old age.[5] When DHEA levels are low or decreasing, all tissues are adversely affected; when DHEA levels are too low, death occurs.

Dehydroepiandrosterone is known to be involved in conception and fertilization and, when it is at low levels, treatment with DHEA can increase the probability of conception.[6] Treatment with DHEA has been shown to reduce miscarriages.[7]

A mother produces DHEA for herself and her fetus. I suggest that treatment with these antibiotics may reduce production of DHEA and, therefore, increase the probability of miscarriage, in this case, an evolutionary mechanism that saves the life of the mother. The need for use of antibiotics in these women may indicate that they already have low levels of DHEA.

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