## "Maternal Stress Responses and Anxiety During Pregnancy: Effects on Fetal Heart Rate" (2000), by Catherine Monk, William Fifer, Michael Myers, Richard Sloan, Leslie Trien, and Alicia Hurtado

In 2000, Catherine Monk, William Fifer, Michael Myers, Richard Sloan, Leslie Trien, and Alicia Hurtado published "Maternal stress responses and anxiety during pregnancy: Effects on fetal heart rate," hereafter "Maternal stress: Effects on fetal heart rate," in which the authors conducted a study on how pregnant women's stress and anxiety affects the health of their fetuses. Previous studies had shown that stress and anxiety during pregnancy could cause fetal abnormalities. In their article, Monk and colleagues reported that the fetuses of anxious pregnant women were more likely to have elevated heart rates and increased stress when exposed to stressors than fetuses of non-anxious women. The authors' findings indicated that fetuses of anxious women display more biological markers of stress than fetuses of non-anxious women.

In "Maternal stress: Effects on fetal heart rate," Monk and her colleagues report the findings of their research on how maternal stress during pregnancy affects fetal health and development. Monk worked in the Department of Psychiatry and the Behavioral Medicine Program at Columbia University in New York City, New York. Co-authors William Fifer, Michael Myers, and Richard Sloan were also affiliated with Columbia University, while Leslie Trien and Alicia Hurtado were associated with the New York State Psychiatric Institute in New York City, New York. All of the authors focused on how fetuses were affected by the pregnant woman's pre-birth stress.

Prior to the work of Monk and her co-authors, other researchers had associated the stress and anxiety experienced by pregnant women with fetal risks and abnormalities. For example, stress experienced by pregnant women had been linked with low fetal birth weights and premature birth. Researchers had also linked exposure to prenatal stress with low Apgar scores, which is a measure of the health of an infant immediately after birth. During the 1990s, scientists conducted animal experiments that suggested a relationship between maternal stress during pregnancy and offspring's ability to regulate stress. For example, researchers found that rats exposed to prenatal stress expressed more anxiety and fear once born. Other researchers found that monkeys prenatally exposed to stress exhibited poorer motor skills. Scientists also conducted human studies during the 1990s that pointed to a relationship between maternal emotions during pregnancy and altered fetal physiology and behavior. For example, a 1995 study reported that infants born to depressed women had altered central nervous system development, exhibited less activity and endurance, and exhibited more irritability. The authors of "Maternal stress: Effects on fetal heart rate" looked further into how prenatal stress impacts fetal physiology and behavior.

Monk and colleagues aimed to directly measure the effects of maternal biological responses to stress on fetal behavior. In "Maternal stress: Effects on fetal heart rate," the authors describe how they measured a number of pregnant women's biological responses to stress, how fetal heart rate changed as a result of exposure to maternal stress, and how fetal heart rate responded to maternal anxiety, which the authors used to create two different stress-level groups. After identifying those effects, Monk and her co-authors concluded that fetuses of anxious women were more likely to have elevated heart rates when exposed to stressors than fetuses of non-anxious women, which showed that prenatal exposure to stress may have negative effects on a fetus.

In the methods section, the authors outline how they selected the women they worked with and how

they measured those women's stress levels. The authors experimented on twenty pregnant women, all of whom were nonsmokers and thirty-five to thirty-eight weeks into pregnancy. The mean age of the pregnant women was twenty-six years old. To collect data, the authors instructed the women to complete a self-report measure of state anxiety, which is a questionnaire that measures anxiety. The authors describe that they then attached electrodes to the pregnant women to measure the electrical activity generated by the heartbeat of the woman and the fetus. The electrodes also monitored the women's breathing. The authors measured the women's blood pressure throughout the experiment and used an ultrasound to measure fetal heart rate.

Continuing in the methods section, the authors describe one of tests they used to induce stress in their test subjects. To monitor the stress, the authors measured the heart rate and blood pressure of the women because the experience of stress can increase blood pressure due to increased heart rate. As part of the experiment, the authors had the women lie down and complete one of two cognitive tasks, which the women completed using a keypad to log their answers while remaining silent. One of the cognitive tasks, which the researchers assigned to the women randomly, involved mental arithmetic. In the task, Monk and her colleagues presented the women with a four-digit number on a computer and asked them to subtract from that number serially by seven, starting with the number originally presented on the screen. Once an answer was entered, the current number on the screen disappeared. The researchers instructed the women to subtract as quickly and as accurately as they could. The authors administered that first test to induce stress in the pregnant women so that the authors could measure the biological markers of stress in the women and the fetuses.

Concluding their methods section, the authors describe the second test they used to induce stress in the women, the Stroop Color and Word Test. In the test, Monk and her colleagues presented the women with the name of a color written in a color that may or may not be the same color as the written color name. The women had to select the key on the keypad that indicated the color of the letters presented. If a woman answered incorrectly, the screen immediately indicated that the answer was incorrect, creating a more stressful situation for the woman being tested. The authors explain that the women were allowed to practice their given cognitive task for one minute before data collection began. At the start of data collection, the women sat quietly for five minutes to establish a baseline for all the measurements mentioned previously. The women then completed their given task for five minutes and concluded with a three-minute recovery period. During each cognitive task, the authors told the women to work a little faster at one-minute intervals, to create a more stress-inducing situation than previously experienced. At the end of the recovery period, women completed the same questionnaire that they had completed before.

In the results section, the authors report on their findings that fetuses of anxious pregnant women exhibit more biological markers of stress as a result of exposure to prenatal stress than fetuses of non-anxious women. The authors relate the results of the stress-inducing cognitive tests on maternal blood pressure, breathing rate, heart rate, and fetal heart rate. At baseline, the pregnant women had a mean level of 2.8 self-reported stress on a ten-point scale and a mean level of 7.2 self-reported stress directly following the cognitive tasks, which showed that the tests successfully created the intended stressful situation. The cognitive stress that the tests created caused a significant increase in maternal blood pressure and respiratory rate, while maternal heart rate was less effected. In analyzing maternal anxiety and stress scores, the authors explain that the women could be broken up into two major groups, a higher than average anxious group, which the authors referred to as [ANX(+)], and a lower than average anxious group, which the authors referred to as [ANX(-)]. Women in [ANX(+)] generally had higher anxiety levels than women in the [ANX(-)] group at baseline. Women in the differing groups also had very different blood pressure responses to the stressful cognitive tasks. [ANX(-)] women had much greater increased blood pressure in response to the tasks than [ANX(+)] women, who had no major blood pressure changes when exposed to stress. In other words, women with generally lower anxiety had more noticeable biological responses to the stress-inducing tasks than women with generally high anxiety levels. However, neither group displayed significant heart rate changes during the cognitive tasks, while both groups showed significantly increased respiratory rates, indicating increased stress in both groups. However, fetuses of [ANX(+)] women experienced much greater increases in heart rate during the stressed period than fetuses of [ANX(-)] women, meaning that fetuses of women with high anxiety experience more

biological markers of stress throughout pregnancy. From this, the authors conclude that maternal anxiety and stress impacts fetal heart rate, a measure indicative of the experience of stress.

"Maternal stress: Effects on fetal heart rate" has been cited 256 times as of early 2018 and helped set the precedent for other studies about maternal anxiety and stress and their effects on fetuses and developing children. The article also set a precedent for the corresponding authors' research on perinatal stress. Following the publication of "Maternal stress: Effects on fetal heart rate," Monk continued her study linking fetal heart rate reactivity to long-term developmental risks in fetuses. By linking increased fetal heart rate to prenatal exposure to stress, the authors identified a specific example of the significance of maternal stress on the health of a fetus. In so doing, they identified the important role of maternal psychology in the health and development of a fetus.

## Sources

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