

## A Mathematical Model for the Fetal Movement

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### Abstract

Depression is prevalent in pregnant women, affecting 10-25% of women. A mathematical model is used to study the prenatal depression which effects on the fetus and newborn. The results are obtained by mathematical formulas.

**Keywords:** Depression, Fetal movement, Mean time between failures (MTBF), Pregnancy, Projection Model.

### I. Introduction

The most widely used traditional reliability growth tracking model and reliability growth Projection model address reliability growth based on failure modes surfaced during the test. With the Projection model all corrective actions are delayed until the end of test [2, 3].

Supposed a development testing program begins at time 0 and is conducted until time T and stopped. Let N be the total number of failures recorded and let  $0 < X_1 < X_2 < \dots < X_N < T$  denote the N successive failure times on a cumulative time scale. We assume that the Non-Homogeneous Poisson Process (NHPP) assumption applies to this set of data. Under the basic model the maximum likelihood estimates (MLEs) for  $\lambda$  and  $\beta$  (numerator of MLE for  $\beta$  adjusted from N to N-1 to obtain unbiased estimate) are

$$\hat{\lambda} = \frac{N}{T^{\hat{\beta}}}, \quad \hat{\beta} = \frac{N-1}{\sum_{i=1}^N \frac{T}{X_i}} \quad \dots (1)$$

The achieved or demonstrated failure intensity and Mean time between failures are estimated by

$$\hat{\lambda}_{CA} = \hat{\lambda} \hat{\beta} T^{\hat{\beta}-1}, \quad \dots (2)$$

$$\hat{M}_{CA} = [\hat{\lambda}_{CA}]^{-1} \quad \dots (3)$$

It is important to note that this model does not assume that all failures in the data set receive a corrective action. Based on the strategy some failures may receive a corrective action and some may not.

#### 1.1. Notation

- t - Test time
- T - Total test time
- $X_i$  - The i-th successive failure time
- N - Total number of failures
- $\lambda_A$  - Type A modes failure intensity

- $\lambda_B$  - Type B modes failure intensity
- $\lambda_{CA}$  - Achieved failure intensity
- $\lambda_A$  - Achieved failure intensity of MTBF

### II. Projection model

Suppose a system is tested for time T. During the testing problem failure modes are identified, but all corrected actions are delayed and incorporated at the end of the test phase. These delayed corrective actions are usually incorporated as a group and the result is generally a distinct jump in the system reliability. The projection model [4], estimates this jump in reliability due to the delayed fixes. This is called a "Projection."

The projection model places all failure into two groups, A and B. Type A failure modes are all modes such that if seen during test no corrective action will be taken. This accounts for all modes for which management determines that it is not cost-effective to increase the reliability by a design change. Type B failure modes are all modes such that if seen during test a corrective action will be taken. This type A and Type B determination helps define the reliability growth management strategy. The basic projection model assumes that the Type A failure modes has constant failure intensity  $\lambda_A$ , the i-th Type B failure mode follows the exponential distribution with failure rate  $\lambda_i$ , and the initial failure intensity for Type B failure modes is  $\lambda_B$ .

An effectiveness factor (EF)  $d_j$  is the fraction decrease in  $\lambda_j$  after a corrective action has been made for the j-th Type B mode. The failure rate for the i-th Type B failure mode after a corrective action is  $(1-d_j)\lambda_j$ . In Practice, for application of the Projection model, the EFs are assigned based on engineering assessments, test results, etc.

The system failure intensity is constant, say,  $\lambda_S$ , during the testing and then jumps to a lower value due to the incorporation of corrective actions. The intensity at the end of the test T, before delayed corrective actions are introduced into the system, is the achieved intensity. the reciprocal of the intensity is the achieved mean time between failure (MTBF)  $M_S$ .

We estimate the achieved failure intensity  $\lambda_S$  by  $\hat{\lambda}_S = \hat{\lambda}_A + \hat{\lambda}_B$ ,  $\hat{\lambda}_A = N_A / T$ ,  $\hat{\lambda}_B = N_B / T$  ... (4)

The estimated achieved mean time between failure  $M_S$  at time T, before the jump is  $\hat{M}_S$ . We estimate the jump next.

The estimated projected failure intensity is

$$\hat{\lambda}_P = \hat{\lambda}_A + \sum_{j=1}^M (1 - d_j) \frac{N_j}{T} + \bar{d} \hat{h}(T) \quad \dots (5)$$

Where  $\bar{d} = \frac{\sum_{j=1}^M d_j}{M}$  is the average EF, and

$$\hat{h}(T) = \hat{\lambda} \hat{\beta} T^{\hat{\beta}-1}, \quad \dots (6)$$

The projection model  $\hat{\lambda}$  and  $\hat{\beta}$  for use only the M first occurrence failure times of the seen and unique Type B failure modes.

### III. Application

Prenatal depression increases in severity from the first to the second trimester [7], Negatively affecting fetal development and neonatal outcome. Prenatal and perinatal complications include higher rates of placental abnormalities, preeclampsia and spontaneous abortion [10]. Depressed women are also more likely to deliver prematurely, and they often have neonates who require intensive care for postnatal complications including bronchopulmonary dysplasia and interventricular hemorrhage.

Neonates of depressed mothers are also at greater risk for being low birthweight (<2500 g) and small for gestational age (<10<sup>th</sup> percentile) [6], with low birth weight being one of the leading causes of fetal growth retardation, the second leading cause of perinatal mortality. These infants continue to experience growth retardation across the first year of life [11].

Behaviourally, biochemically and physiologically fetuses and neonates of depressed mothers also differ. Fetuses of depressed women show elevated heart rates, greater activity levels and increased physiological reactivity. Newborns of depressed mothers perform less optimally on the

Brazelton neonatal behaviour assessment scale, and they show less positive affect. Their negative affect continues into later infancy, and their cortisol responses to mild stressors are predictive of negative affect even at the toddler stage. Infants of depressed mothers also show inferior mental, motor and emotional development, and later social and emotional problems during childhood including less emotional well-being as well as internalizing and externalizing problems [9].

Although postpartum depression was the focus of these studies, at least one study had suggested that the depressed mothers showed depressed affect as early as their third trimester of pregnancy, suggesting prenatal depression effects as well or a combination of prenatal and postnatal environmental conditions. According to a multivariate cumulative risk model, infants of depressed mothers may have a genetic predisposition and/or may be exposed to a chemically and physiologically imbalanced prenatal environment. This can result in the development of neurobehavioural dysregulation that then interacts postnatally with their mothers' poor arousal modulation and negative interaction style, in turn, potentially leading to later psychopathology.

Because prenatal depression had been noted in many of these mothers, infants of depressed mothers were then studied even earlier, shortly after birth. In these studies, the Brazelton neonatal behaviour assessment scale was administered with 24 h after birth. In one study, newborns of depressed (N = 47) versus newborns of non-depressed mothers (N = 36) received inferior orientation and motor scores, and they showed more irritability and less activity, robustness and endurance during the assessment [1]. The facial expressions of newborns born to depressed (N = 20) and non-depressed mothers (n = 20) during the Brazelton assessment and during the modeling of happy, sad and surprised faces. Newborns of mothers with depressive symptoms had lower scores on the orientation cluster of the Brazelton scale. During the facial expression modeling, the newborns of the depressed mothers also showed less attentiveness and fewer facial expressions in response to the modeled happy and surprise facial expressions.

### IV. Methodology

Prenatal depression also affects fetal activity [5]. In this study, pregnant women with (N = 45) and without (N = 45) symptoms of depression (CES-D scores greater than 16) were given ultrasound examinations across the second and third trimesters. Fetal movements (single limb, multiple limb, gross body) were recorded for 5 min prior to a standard ultrasound examination. The analyses revealed that the fetuses of depressed women were more active at 5, 6 and 7 months gestation (see Fig.1).

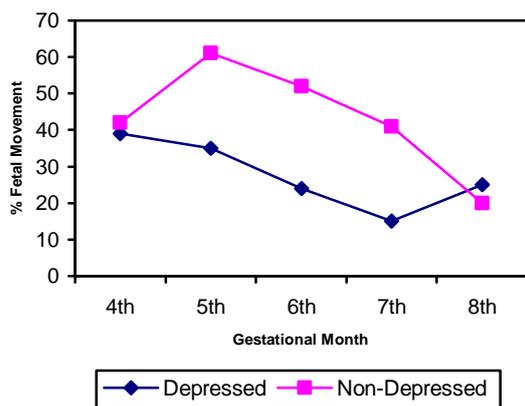


Fig. 1. Activity in fetuses of depressed and non-depressed mothers

### V. Mathematical Result

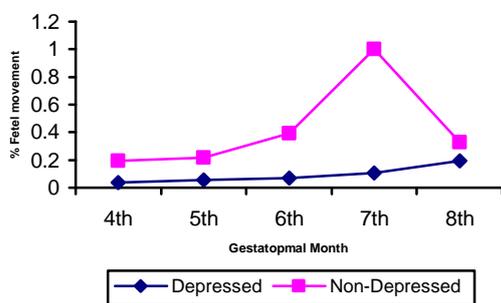


Fig. 2. Activity in fetuses of depressed and non-depressed mothers

### VI. Conclusion

Neonates of depressed mothers mimic their mothers' prenatal biochemical / physiological profile. From the mathematical model this study found that fetal activity was affected by prenatal depression which is explained in section 5. This result is useful for medical professional for the future references.

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