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PRETERM LABOR

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Abstract
Preterm birth is traditionally defined as the presence of uterine contractions of sufficient frequency and intensity to effect progressive effacement and dilation of the cervix prior to term gestation (between 20 and 37 wk).
Keywords: Preterm; labor, birth

Introduction:

The sub-grouping of preterm birth is based on gestational age depending on gestational age at birth **(Keelan and Newnham, 2017)**. Infants born at gestational age 32-36 weeks are classified as moderately preterm, infants born at gestational age 28-31 weeks are classified as very preterm, and infants born at gestational age less than 28 weeks are classified as extremely preterm. The term "near-term" has been used for infants born closest to term at 34-36 gestational age (Table, 1). Prenatal corticosteroids and tocolytic treatment are no longer administered in gestational age more than 34 weeks **(Romero et al., 2014)**.

Preterm labor precedes almost half of preterm births and preterm birth neonatal mortality in the United States. Approximately 15 million neonates are born premature worldwide yearly, implying a rate of one out of ten neonates **(Lawn et al., 2010)**. The rate of preterm birth is still rising. Preterm birth is the leading cause of mortality in new-borns. Long-term morbidity in survivors comprises of learning disabilities, hearing loss, behavioural problems and visual disturbances. Inequalities of prevalence and survival rates vary considerably. The prevalence of preterm birth is the highest in Africa and South Asia **(Keelan and Newnham, 2017)**.

Table (1): Gestational age categories.

Categories	*Week and days*
Late preterm birth	34 to 36+6
Moderately preterm birth	32 to 33+6
Very preterm birth	28 to 31+6
Extremely preterm birth	Less than 28

Mechanism of preterm labor:

The exact mechanism(s) of preterm labor is largely unknown but is believed to include decidual hemorrhage, (eg, abruption, mechanical factors such as uterine overdistension from multiple gestation or polyhydramnios), cervical incompetence (eg, trauma, cone biopsy), uterine distortion (eg, müllerian duct abnormalities, fibroid uterus), cervical inflammation (eg, resulting from bacterial vaginosis [BV], trichomonas), maternal inflammation/fever (eg, urinary tract infection), hormonal changes (eg, mediated by maternal or fetal stress), and uteroplacental insufficiency (eg, hypertension, insulin-dependent diabetes, drug abuse, smoking, alcohol consumption) **(Howson et al., 2013)**.

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Perinatal mortality rates:

In developed countries the preterm birth rate is about 8% of all pregnancies1. In the Netherlands, perinatal mortality was 9.7 per 1000 total births. Preterm births (> 22.0 weeks and < 37.0 weeks) accounted for 75.3 % of all perinatal mortality with a mortality risk of 82.6 per 1000 births (Schaaf et al., 2011). Short-term and long-term effects of preterm birth on society and health systems are profound5. Reducing the burden of child mortality due to preterm birth, is therefore one of the eight Millennium Development Goals. Hence, prevention of preterm birth is essential to reduce the high mortality and the considerable risk of lifelong impairment **(Murray et al., 2010)** (Fig, 1).

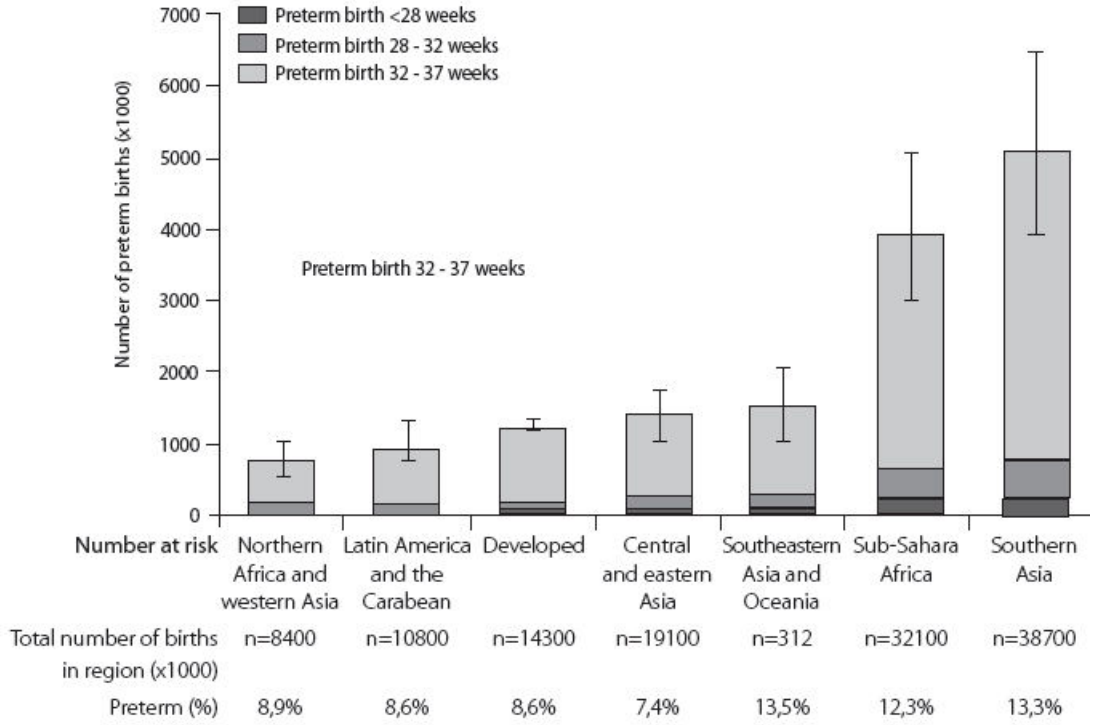

Figure (1): National, regional and worldwide estimates of preterm birth rates in the year 2010 for selected countries: a systematic analysis and implications.

Risks of preterm birth:

Preterm birth can occur spontaneously or for medical reasons (iatrogenic preterm birth). In this thesis we focus on spontaneous preterm birth. The two pillars in the strategy to reduce preterm birth are prevention and care for women who present with threatened preterm labour. The underlying pathophysiology of spontaneous preterm birth is not completely understood and has probably multiple causes, which makes prevention strategies challenging **(Smith et al., 2009)**. Care for women with (threatened) preterm birth include interventions such as tocolysis, bed rest or placement of a cervical cerclage. These measures have limited effectiveness **(Smith et al., 2009)**.

Various risk factors and methods of screening have been identified. The most important risk factor which predicts preterm birth, is a history of premature birth15. Nulliparous women do, however, not benefit from this knowledge. Therefore, most preterm births occur in women initially classified as low risk. A second important risk for preterm birth is a multiple pregnancy. These pregnancies, however, are not the scope of this thesis. In this thesis we focus on singleton pregnancies. Nulliparous women do, however, not benefit from this knowledge. Therefore, most preterm births occur in women initially classified as low risk. A second important risk for preterm birth is a multiple pregnancy. These pregnancies, however, are not the scope of this thesis. In this thesis we focus on singleton pregnancies. Methods of screening for preterm birth include testing for fetal fibronectin and ultrasonographic cervical length measurement **(Smith et al., 2009)**.

Cervical length measurement between 20-24 weeks gestation is thought to be the best method of screening for premature birth. A strong association between a short cervix in the second trimester and a subsequent

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premature birth has been shown by several studies **(Plunkett et al., 2008)**. Thus, in a low-risk population of women (defined as nulliparous women and women without a history of preterm birth), women with a short cervical length between 20-24 weeks of gestation are at risk **(Chang et al., 2013)**.

The argument for a relationship between physiological immaturity and risk of poor neonatal outcomes among infants born late preterm and early term is made on the basis of the observed dose-response relationship between gestational age and neonatal risk, for example, found that the rate of severe respiratory morbidity declined steadily with increasing gestational age from 19.8% at 34 weeks to 0.28% at 39 to 41 weeks **(Keelan and Newnham, 2017)**.

There is also evidence of functional immaturity of specific body systems at 34 to 36 weeks gestation, and neonatal morbidity associated with late preterm birth can be explained as follows:

- Respiratory morbidity. Infants born late preterm have immature lung volume and structure. This results in delayed fluid absorption, insufficient surfactant, and inefficient gas exchange.
- Hyperbilirubinemia. Infants born late preterm have increased bilirubin production and decreased bilirubin elimination. This is exacerbated by their poor suckswallow mechanism, which results in inadequate breast milk intake, dehydration, and increased bilirubin circulation.
- Sepsis. The immune systems of infants born late preterm are immature, and this is exacerbated by feeding difficulties which may prevent them from being breastfed.
- Hypoglycemia. Infants born late preterm have an immature system of glucose regulation; they may therefore not adapt adequately to the drop in glucose supply experienced immediately after birth with the removal of the placenta.
- Temperature instability. Infants born late preterm have an immature epidermal barrier due to incomplete development of adipose tissue as well as a higher ratio of surface area to birth weight. They also have greater difficulty than term infants regulating their body temperature.
- Neurological morbidity. The brains and central nervous systems of infants born late preterm are under-developed and are more vulnerable to extrauterine insults, such as handling and ventilation, which may disrupt blood pressure and lead to bleeding in the brain.

Fetal maturation is a continuous process with no threshold. Therefore, infants born early term would be expected to be physiologically mature compared to their late preterm counterparts and immature compared to those born full term **(Chang et al., 2014)**.

Reasons for Early Birth:

Although the physiological immaturity of infants born late preterm and early term seems clear, it is possible that poor outcomes among these infants are associated not only with being born early but also with the reasons for being born early. Exposure to pathological conditions in utero may act through early birth to cause poor outcomes and may even exacerbate the risk of poor outcomes among those born early **(Beck et al., 2010)**.

Studies conducted by Basso and Wilcox demonstrate the impact of the complex relationship between preterm birth and the reasons for preterm birth on neonatalutcomes. In the first study, Basso and Wilcox **(Chang et al., 2014)** estimated the overall expected proportion of neonatal mortality due to immaturity alone by summing gestational agespecific mortality rates among singletons with “optimal birth weight” for gestational age. They reasoned that mortality among these supposedly healthy infants must be due to immaturity and not the reasons for preterm birth (which would likely result in smaller birth weight for gestational age). They then compared this expected neonatal mortality rate with the actual neonatal mortality rate among U.S. singleton births (1995-2002). They concluded that 49% of neonatal mortality was due to immaturity alone and 51% of neonatal mortality was, in fact, due to underlying pathologies experienced in utero **(Katz et al., 2013)**.

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