GBS has been the principal pathogen in the neonate for many years with mortality rates ranging from 4-50% prior to the initiation of new guidelines. Infants that survived suffered from mental retardation, hearing loss, or vision loss. Mothers were also at risk developing over 50,000 clinical infections per year.

Early onset disease is acquired by horizontal transmission rather than vertical transmission of early onset and hence unaffected by IAP (3). Late-onset disease is probably related more to persistent maternal anogenital carriage of GBS than to infected breast milk, except when mastitis is present (3). Term and preterm infants are equally susceptible to late-onset disease, and maternal obstetric complications are uncommon (1).

Late onset GBS infection (3).

Though treatment for early vs late onset disease is very similar, the presentation of illness has distinct characteristics in relation to onset of symptoms. Early onset disease, defined as signs and symptoms within the first 7 days of life, consists primarily of generalized septicemia, pneumonia, and meningitis with more than 80% of neonates presenting initially with signs of respiratory distress. This is opposed to late onset GBS, classified as onset between 7-99 days of life with typical onset at 3-4 weeks, which presents with nonspecific findings that can progress to septicaemia and meningitis if not caught and treatment started early on in presentation. Skin manifestations are common in late-onset GBS infection with the most common being cellulitis or cellulitis/adenitis generally of the submandibular area (4). Disseminated skin infections such as the one in our patient are very rare in both the setting of late onset GBS as well as presentation of late onset GBS in pre-term infant.

Early fetal loss, premature rupture of membranes, and preterm delivery of low-birthweight infants are also associated with GBS colonization (1) With the advent of standard antenatal screening and the utilization of Intrapartum Antiseptic Prophylaxis (IAP) in at risk women in the 1990’s, as well as the institution of National Guidelines in the late-90’s and early-2000’s we have seen a dramatic decrease in the rates of early onset GBS-related sepsis, however late-onset GBS sepsis continues to persist at the same rate of approximately 0.3-0.4 case per 1,000 live births (50% of all cases) despite these advancements (2) (See Fig. 1), with preterm infants being affected nearly 6 times more frequently and with higher severity than term infants.

We present a case of an extremely preterm neonate with severe skin necrosis and cellulitis at 8 weeks of life. This unusual condition is later revealed to be a unique presentation of late GBS infection.

Case Presentation
A 23 1/7 week GA neonate is born by emergent c-section to a mother with foul-smelling vaginal discharge and PPROM (72 hours). Maternal labs are negative except for HSV 1/2 (no active lesions), including GBS (treated with IAP).

At delivery, APGARs are 1/1/6; the patient requires ventilation. Empirical ampicillin and gentamicin are begun. Initial blood cultures are negative; maternal placental studies display funisitis and acute chorioamnionitis. The patient has a critical clinical course and is treated with a prolonged course of broad spectrum antibiotics until DOL 95. The hospital course is complicated by two PICC line infections.

At 8 weeks of age, the patient presents with a blistering lesion on the left lateral thigh, which rapidly progresses to further skin lesions. Lab work reveals leukopenia, thrombocytopenia, and bandemia. Despite escalation of antibiotics, the condition advances and cellulitis versus necrotizing fasciitis is suspected.

On DOL 61 the patient is transferred to a higher level of care with extensive skin erythema, edema, and breakdown of the buttocks, bilateral lower extremities, and left upper extremity. With concern for tissue necrosis, plastic surgery takes the neonate to the OR for a bilateral lower extremity fasciotomy with bilateral upper extremity fasciotomy. There is no recurrence of GBS on any further cultures collected since the initial negative culture.

The patient continues with a complex clinical course but there is no recurrence of GBS on any further cultures collected since the initial negative culture.

Discussion
Late onset GBS disease rate has remained unchanged and remains a risk despite the initiation of IAP. This is understandable given that it is thought that late-onset disease is acquired by horizontal transmission rather than vertical transmission of early onset and hence unaffected by IAP (3). Late-onset disease is probably related more to persistent maternal anogenital carriage of GBS than to infected breast milk, except when mastitis is present (3). Term and preterm infants are equally susceptible to late-onset disease, and maternal obstetric complications are uncommon (1). However, IAP has recently been shown to result in both delayed and milder presentation of late onset GBS infection (3).

Conclusion
Late-onset GBS continues to be a serious risk especially in preterm infants, and many times presents without suspected respiratory symptoms. One must consider this when evaluating an infant presenting with nonspecific infectious complaints.

References
** List of references provided upon request**