Female urinary incontinence, from pregnancy to menopause: a review of epidemiological and pathophysiological findings.

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Abstract

Hypotheses that might explain urinary incontinence during pregnancy and after childbirth have been examined. Urinary incontinence prevalence reaches a maximum during pregnancy and decreases after childbirth. Cesarean delivery is associated with lower rates of stress incontinence than vaginal delivery. Women delivered by cesarean section differ from women who had a vaginal delivery through pre-existing characteristics associated with the incontinence risk, producing a selection bias. The only randomized trial (intention of vaginal delivery versus elective caesarean section) was not conclusive. The suspected etiological factors consist of congenital elements, obesity, aging, pregnancy and vaginal delivery. They are likely to have an effect at different times and portions of the urethral sphincter complex. Unfortunately there exist no cohorts facilitating description of the natural history of female urinary incontinence. Given the small amount of usable data available, it is not possible to draw any conclusions concerning the possible long-term protective effects of cesarean section.

Abbreviations: BMI, body mass index; LE, level of evidence; MUI, mixed urinary incontinence; SUI, stress urinary incontinence; UI, urinary incontinence; UUI, urge urinary incontinence.

Key-words: Urinary incontinence, pregnancy, delivery, cesarean, epidemiology.

Conflict of Interest:

The authors have no conflict of interest to declare.

Introduction

Urinary incontinence (UI) is a symptom frequently encountered in women, and it can evolve into a disabling pathology necessitating costly treatment (1-3). Despite its frequent occurrence, many aspects of its pathophysiology remain uncertain. Urinary continence is due to the fact that bladder pressure remains lower than urethral closure pressure, and incontinence may result from bladder or urethral impairment; when closure pressure is lower than bladder pressure, leakage occurs. Three types of incontinence are generally distinguished: stress urinary incontinence (SUI); urge urinary incontinence (UUI), and mixed urinary incontinence (MUI), which associates the first two.

The pathophysiology of UUI has yet to be clearly elucidated. It appears to be connected with poor transmission or processing of information between the bladder and the nervous system (4). The main identified disorders with regard to SUI are impaired urethral support and sphincter deficiency (5). Nobody really knows why, how, and at what precise moment these disorders arise (6).

In a majority of cases urinary incontinence in women is stress-related before the age of 50 and mixed after the age of 50 (7). The time lapse between average age at first childbirth and average age at the time of SUI surgery ranges from 20 to 30 years; this wide range underscores the difficulty of establishing a causal relation between childbirth and SUI. And yet, the obstetrical trauma hypothesis according to which vaginal childbirth results in irreversible traumatic lesions to the urinary continence system is generally accepted. Some practitioners even favor caesarean section as a way of preventing urinary incontinence (8). Our objective in this review of the literature is to examine the arguments supporting the traumatic obstetrical theory and, if need be, to put forward alternative hypotheses that might more adequately explain the occurrence of urinary incontinence in women.

Methods

The studies included in our manuscript originate from two sources. The first is a review of available epidemiologic or clinical studies in which urinary incontinence was the main outcome of interest and obstetrical factors (related to pregnancy or delivery) were analyzed as explanatory factors. We have included a review of all cohorts as regards urinary incontinence during first pregnancy and postpartum (22 studies). The second part consists of a panel of selected studies on the pathophysiology of urinary incontinence associated with anatomical, hormonal, or physiological changes during pregnancy and after childbirth. Selection of these studies was not based on a systematic search; they are rather cited to illustrate some underlying pathophysiological mechanisms that may explain the epidemiological evidence.

The level of evidence [LE] is indicated for each study through the standard model developed by the Oxford Centre for Evidence-Based Medicine (www.cebm.net): LE1, randomized comparative studies; LE2, cohort studies; LE3, case-control studies; LE4, case-series.

We wish to emphasize that most available information on the connection between childbirth and middle-aged UI came from cross-sectional studies that could not determine the onset of urinary symptoms. Only three teams have published results on longitudinal follow-up of women studied for more than two years subsequent to childbirth (9-11).

Results

Urge urinary incontinence, pregnancy and delivery

UUI prevalence in young women is quite low (7% at 30 years of age in EPINCONT, see below); it is higher in older women (13% at 50 years of age and 19% at 70) [LE3b] (7). During pregnancy, its prevalence increases as the term approaches (12% at end of pregnancy) [LE2b] (12). In the first stages of postpartum, UUI prevalence is lower in cases of cesarean section [LE2a] (13). However, in the long term after childbirth, cross-sectional and longitudinal observational studies have not shown any association between parity and UUI, or mode of delivery and UUI [LE2a] (13-17). Given the lack of connection between UUI and pregnancy or delivery, the remaining review will be essentially devoted to SUI.

Association between obstetrical trauma due to vaginal delivery and stress urinary incontinence

SUI prevalence is higher in older women, and reaches a maximum level at approximately the age of 50 (27% in EPINCONT) [LE3b] (7,18). In a majority of studies on the subject less SUI prevalence following cesarean section has been found in comparison with vaginal childbirth [LE2a] (11,13,15,19). EPINCONT is a large-scale cross-sectional survey that compared SUI prevalence in 669 women aged from 20 to 64 years having delivered only by cesarean section and 11 299 women who had only vaginal deliveries (15). SUI prevalence was higher in the vaginal childbirth group (OR 2.4; IC95% 1.7-3.2) [LE3b]. Leijonhufvud cross-checked the Swedish birth registry from 1973 to 1982 with the records of surgical interventions from 1973 to 2005 (20), and his study shows a heightened likelihood of SUI surgery in cases of vaginal childbirth (compared with cases of cesarean childbirth) [LE3b]. The randomized trial by Hannah, who compared planned cesarean births with planned vaginal delivery for breech

presentation at term (21), showed lower SUI prevalence in the planned cesarean group three months later (4.5 vs. 7.3%) [LE1b]. The risk of postnatal UI was aggravated when the second stage of labor was prolonged, and lessened in cesarean childbirth, especially when the section was carried out prior to labor or at the onset of labor [LE2b] (22). SUI prevalence 12 years after childbirth was 55% in the event of only vaginal deliveries as opposed to 40% in the event of only cesarean deliveries [LE2b] (10). These observations suggest a causal link between the trauma of vaginal childbirth and SUI.

Obstetrical trauma

A childbirth-related pelvi-perineal trauma may be immediately observed by simple clinical examination or using complementary postnatal tests (23). Perineal tears frequently occur, particularly on the occasion of a first childbirth, in which a 1.8% to 16% prevalence of third-degree perineal tear has been reported [LE2b] (24,25). After vaginal childbirth, investigations have shown increased pudendal nerve terminal latency, widening of the levator hiatus, perineal descent, and increased urethral mobility at stress [LE2b] (26,27). Dietz found a reduction of reflex pelvic floor activation on coughing that is more marked after vaginal delivery (28). Twenty per cent of primiparas show levator ani lesions at MRI after vaginal childbirth, and the lesions are associated with an increased risk of SUI (72% with SUI in cases of MRI-evidenced lesion vs. 45%) in this cross-sectional study of 160 primiparas examined 9-12 months subsequent to vaginal delivery [LE3b] (29). Dietz and Lanzarone objectified similar levator ani lesions through ultrasonography in 36% of primiparas having undergone vaginal childbirth and examined 2-6 months subsequent to delivery (30). In their work, no link between levator ani lesions and SUI was reported [LE3b].

Vaginal delivery may consequently entail the creation of irreversible lesions that could be aggravated by aging and subsequent childbirth, finally leading to urinary incontinence. Such is the theory put forward by DeLancey, who strongly suggested that an obstetrical trauma paved the way to urinary incontinence, which can occur immediately if the trauma is severe or later in cases where the trauma is partially repaired (31).

Shortcomings of the trauma paradigm

Several observations call the predictions given by the trauma model into question: SUI prevalence is maximal not postpartum, but rather in the prenatal stage (Figure 1), during which 30% to 50% of pregnant women have reported SUI [LE2b] (22,32-35). Such findings mean that the pathology (SUI) may actually precede exposure (delivery) and suggest that mechanisms other than obstetrical traumatism are involved prior to delivery in the genesis of SUI.

A follow-up of 305 primiparas carried out by Viktrup (32) shows a remission of incontinence over the first 12 months of postpartum, with SUI prevalence being divided by 6 as it decreased from 19% in the immediate postpartum period to 6% three months later and to 3% after one year [LE2b]. A comparable drop in SUI prevalence following delivery and over the initial months postpartum has been observed by others in the past (33-36) or by contemporary authors identified in our bibliographic review (Figure 1), which recapitulates all published longitudinal studies pertaining to primiparous women (22,37-57). These observations tend to suggest a deleterious, yet reversible, effect of pregnancy on urinary continence.

Partially reversible obstetrical trauma

In a majority of cases, perineal exploration undertaken at some time after childbirth show that perineal measurements are quite similar to those reported before childbirth or improved in comparison with measurements undertaken just after delivery. In the study published by Sultan (26), the terminal latency of the pudendal nerve had reverted to normal at six months postpartum in eight of the 12 women for whom it had increased at six months postpartum [LE2b]. Snooks showed that the terminal latency of the pudendal nerve had returned to normal at eight weeks postpartum, as had perineal descent after five years [LE2b] (58,59). In the work carried out by Toozs-Hobson (27) no difference was observed as regards bladder neck mobility or levator hiatus between prenatal dimensions and the dimensions reported six months after vaginal childbirth [LE2b]. Later postpartum, between 3-6 months and 2-3 years, Shek and Dietz did not observe any improvement in levator hiatus dimension (60) [LE2b]. However, in this study, two women (of 12) recovered from levator avulsion, and bladder neck mobility showed improvement for 53 women delivered vaginally with no further births (60) [LE2b]. These observations tend to suggest that several obstetrical lesions of the pelvic floor may spontaneously heal during the months following delivery. In that case, vaginal childbirth would account for largely reversible lesions of the urinary continence system.

Such findings may help to elucidate observations showing that the apparent protective effect of a cesarean is attenuated in time. In the randomized trial performed by Hannah (21,61), the protective effect with regard to SUI observed at three months postpartum in the planned cesarean group (RR = 0.6; IC95%: 0.4-0.9) diminished and was no longer significant 24 months subsequent to childbirth (RR = 0.8; 0.6-1.1) [LE1b]. Over the same time interval, the fraction of SUI risk attributable to vaginal delivery was halved, decreasing from 38% at three months to 18% at 24 months [LE1b]. Over the long term, after the age of 50, according to the EPINCONT (15) as well as GAZEL studies (62), there exists no significant difference in SUI

prevalence between women having undergone vaginal childbirth and women having undergone cesarean childbirth, even though association with parity remains [LE3b]. In the cohort followed by MacArthur and his colleagues for up to 12 years following childbirth (10), 40% of the women having delivered only by cesarean section reported urinary incontinence [LE2b]. These observations suggest that parity, or pregnancy itself, may contribute to the occurrence of UI independently of the mode of delivery (19,32,63).

Possible selection bias

Moreover, the apparent protective effect of cesarean reported in observational studies, including studies based on registers, may be explained in part by a selection bias. Prospective studies showed that prior to childbirth, women who are going to have a vaginal delivery present higher urethral mobility (27,64) and levator hiatus (27), than those who are going to have cesarean delivery [LE2b]. It may be the case that some particular tissue or muscle characteristics entail both a higher risk of cesarean birth and a lower risk of SUI. In other words, the mode of delivery could be a consequence of pelvic tissue quality rather than a cause, and it is likewise possible that cesarean delivery selects women at little risk of developing SUI. This type of preliminary difference between women delivering by cesarean as opposed to vaginal childbirth does not substantiate observational studies attributing a protective effect to caesarean section. The existence of this kind of selection bias in the EPINCONT cross-sectional study (15) is suggested by the fact that on average, the women delivering by cesarean were four years younger than those delivering vaginally (36 vs. 40 years), had higher BMI (26.2 versus 25.4 kg/m²) as well as lower parity (1.7 versus 2.2), and had more recently given birth (8 vs. 12 years) to children of lower weight (3396 vs. 3542g). Since higher age is a cesarean section risk factor in low-risk nulliparas [LE3b] (65), the opposite difference would have been expected.

Alternative hypotheses are required

As a result, the obstetrical trauma theory does not suffice as a stand-alone explanation of stress urinary incontinence occurring either at the end of pregnancy or with aging. The 241-woman cohort followed by Viktrup over 12 years (11,12,38) underscores the highly diversified histories of urinary incontinence in women and consequently suggests comparably diversified etiologies. In the women presenting with SUI 12 years after initial childbirth, SUI appeared for the first time during pregnancy in 39% of cases, just after delivery in 15%, and after the postpartum period in 46% of cases [LE2b]. Subsequent to an initial remission during the first year of postpartum, (3% one year after delivery), SUI prevalence increased over the years following the initial childbirth (30% at 5 years and 42% at 12 years) [LE2b].

It is equally necessary to explain why SUI occurring transitorily during pregnancy constitutes a risk factor for later appearance of SUI [LE2b] (9,11,19).

Individual susceptibility

The hypothesis of a congenital factor may help to elucidate SUI observed in young or nulliparous women and the similarity frequently observed among members of the same family (66-69). Buchsbaum (69) indeed showed striking SUI concordance among sisters, regardless of parity [LE3b]. In EPINCONT (70), SUI prevalence is higher in the daughters of mothers with declared UI (OR 1.3; IC95% 1.2-1.4) [LE3b]. A case-control survey revealed three times more cases of SUI in a first-degree relative when the woman in question suffered from SUI [LE3b] (71). Dietz et al found a correlation for bladder neck mobility (r= 0.61) in nulliparous identical female human twins [LE3b] (72) The authors estimate that 59% of the variance in bladder neck mobility in nulliparas may be explained by recessive genes, while the rest of the variance would be due to individual environmental factors. Pregnancy may act as a presenting

symptom of underlying individual SUI-related fragility of partially congenital origin. King and Freeman (73) have shown that prior to delivery, primiparas who later suffer from postnatal SUI already present higher urethral mobility than those who will remain continent (7.1 vs. 4.8 mm) [LE2b]. Postpartum, resolution of the physiological, hormonal and anatomical modifications specific to pregnancy may explain the SUI remission observed over the months following delivery. At a later time, the effects of aging could provoke an additional impairment of continence mechanisms, that is to say, a new alteration leading to a SUI relapse in women with individual susceptibility.

Aging

In women as in men, aging is accompanied by a gradual rarefaction of the striated muscle cells in the urethral sphincter [LE3b] (74). Pelvic floor strength is reduced and levator hiatus is increased in older women [LE3b] (75). Urethral closure pressure likewise declines in older women [LE3b] (6,76). Pelvic floor denervation is greater in older women [LE3b] whether or not they once bore children (77). SUI prevalence regularly increases with age [LE3b] in parous and nulliparous women (15). With time, the aging effect is likely to dilute the risk attributable to obstetrical factors; little by little, any causal factor connected with the obstetrical trauma is prone to dwindle. DeLancey (29) showed the existence of an association between SUI and MRI levator ani lesion in women of an average age of 30 who were examined 9–12 months postpartum [LE3b]; no comparable link was found in a case-control study conducted by the same team in women of an average age of 48 [LE3b] (5). In a population of women consulting an urogynecological unit, mean age 55, levator ani lesion was negatively associated with stress UI [LE3b] (78).

A role specific to pregnancy

Individual susceptibility and aging do not suffice to explain why the SUI risk is greater in women who have given birth, even by cesarean section, than in nulliparas. In EPINCONT (15), the age-adjusted risk with regard to all types of UI, of SUI, and of mixed UI was in fact greater in women having delivered by cesarean section than in childless women: OR = 1.5 (1.2–1.9), 1.4 (1.0-2.0) and 1.7 (1.2-2.5), respectively [LE3b]. A heightened risk for women having given birth by cesarean section in comparison with nulliparas has also been found in other studies [LE3b] (62,79,80). Regardless of the mode of delivery, it is consequently highly likely that pregnancy has a specific impact on continence mechanisms, with long-term clinical effects that may be connected with mechanical or metabolic phenomena. Between the prenatal and postpartum stages, the observed volume of the urethral sphincter decreased by 8.2% in the women delivering vaginally and by 10.6% in those giving birth by cesarean section [LE2b] (27). A 10 cm H_20 drop in urethral closure pressure is likewise observed following childbirth, and it remains similar, whether delivery be vaginal or by cesarean [LE2b] (81). Tetzschner (82) highlighted the lengthening of pudendal nerve terminal latency 12 weeks after childbirth, and the figure is similar in women having benefited from spontaneous vaginal delivery and in those having undergone a cesarean section [LE3b]. Weidner (83) discovered an alteration of urethral sphincter electromyography (EMG) in pregnant nulliparas as compared with non-pregnant nulliparas of the same age, and this alteration was still present six months after delivery [LE3b]. Dietz (84) observed increased urethral mobility in pregnant as opposed to non-pregnant nulliparas [LE3b].

Weight gain may partially explain the increased SUI prevalence during pregnancy, postpartum remission and SUI relapse with age. In EPINCONT (85), a high BMI was associated with each type of UI [LE3b]. A high BMI is also associated with the UI specific to pregnancy [LE3b] (37). Postpartum weight loss is associated with remission of UI [LE2b]

(39). Apart from this kind of mechanical effect, a metabolic effect of pregnancy cannot be ruled out. Indeed, pregnancy entails numerous hormonal or metabolic modifications that may be involved in UI occurrence. Estrogens are known to modify collagen synthesis in the tendons [LE2c] (86), and collagen metabolism is modified in the event of SUI [LE3b] (87). Estrogen administration after menopause favours the occurrence of SUI [LE1b] (88). Several metabolic disorders observed in the metabolic syndrome (enlarged waist, high triglyceride level, low HDL cholesterol, arterial hypertension, hyperglycemia) are encountered during pregnancy and are likewise associated with urinary disorders in the mature adult. In a population of 984 women aged over 40 years, Kim et al (89) discovered an association between the presence of at least three elements of the metabolic syndrome and SUI [LE3b]. Brown et al (90) underlined an association between SUI and diabetes or raised fasting glycemia levels in 1461 women [LE3b].

The metabolic and hormonal modifications connected with pregnancy may both explain some of the urinary symptoms observed during pregnancy and entail long-term consequences. Indeed, the long-term metabolic impact of pregnancy is known in the context of other chronic pathologies, such as diabetes: the number of previous pregnancies increases the risk of abnormal glycemia in non-pregnant women [LE3b] (91). In a manner analogous to UI, the prevalence of diabetes is raised during pregnancy, and there is an association between the diabetes of pregnancy and the diabetes of maturity.

Discussion

SUI prevalence is maximal at the end of pregnancy, it diminishes postpartum and is lower in cases of cesarean delivery. SUI prevalence increases with age and reaches a maximal level around the age of 50. After 50, SUI prevalence is similar in women delivered only by cesarean and women delivered vaginally. Several pathophysiological mechanisms are likely to be involved to explain these epidemiological observations.

Urethral mobility increases during pregnancy (84). It would be interesting to learn if a higher mobility is associated with a higher risk of SUI during pregnancy. There exists a link between heightened prenatal urethral mobility and postnatal SUI (73), and pregnant women with a high prenatal urethral mobility are more likely to deliver vaginally (27,64). Higher urethral mobility may be provoked by a number of physiological, metabolic or hormonal modifications induced by pregnancy. Hormones such as estrogens or relaxin are involved in collagen metabolism (86). In postmenopausal women, estrogen therapy increases the risk of SUI (88), and a higher level of serum relaxin has been associated with SUI during pregnancy (92). Pregnancy also modifies carbohydrate metabolism and diabetes is associated with a heightened UI risk (90). Unfortunately, studies on this subject are lacking. Van Geelen did not find a correlation between closure pressure changes and female hormones changes during pregnancy (81).

Urethral closure pressure and sphincter volume diminish after childbirth (27,81). Urethral closure pressure is lower in primiparous stress incontinent women than in primiparous continent women (6). Urethral electromyography is altered in stress incontinent women (93). The pathophysiological mechanism of this alteration of the urethral sphincter is not well-known, and it may be related to pregnancy itself. The Childbirth and Pelvic Symptoms study has shown an alteration of urethral electromyographic parameters during pregnancy that persists six months after delivery (83). The diminution of closure pressure and sphincter

volume observed following childbirth is similar in women delivered by cesarean section and in women delivered vaginally, and it persist six months after delivery (27,81).

The pelvic floor function may be altered during vaginal delivery by muscle injuries as avulsion, widening levator hiatus, diminution of reflex pelvic floor activation on coughing, or increased urethral mobility at stress (27-30). The relation between these lesions and *de novo* postnatal SUI remains uncertain. DeLancey observed an association between postnatal SUI and urethral mobility (6). On the other hand, the same team did not observe any association between major levator ani injury and postnatal UI or SUI (94). Similarly, Shek did not find any association between de novo postnatal SUI and levator avulsion occurrence, or increased urethral mobility (95). Some obstetrical alterations of the pelvic floor may be reversible, while others are not. As regards women having vaginally delivered, measurement of the levator hiatus and bladder neck mobility carried out 6 months postpartum yields figures similar to those of prenatal measurements (27). For postpartum levator ani avulsions, Shek and Dietz observed a recovery in two cases (out of 12) (60).

Postpartum remission of SUI (Figure 1) may be explained by partial healing (31), or as a result of compensation that might restore continence despite persistence of the lesions. The compensation may be due to the pelvic floor muscles, which assume a urethral support role, and it may be favored by pelvic floor training. Since muscle force diminishes as one grows older, SUI may recur as the years pass (Figure 2). If the obstetrical trauma hypothesis were the only explanation, then the protective effect of a planned cesarean delivery would in all likelihood be observed at any age, the incidence of SUI (appearing after childbirth) would be closely associated with the mode of delivery, and SUI prevalence would necessarily be similar in nulliparas and in women having given birth by cesarean section.

According to the reversible trauma hypothesis with a proper effect of pregnancy, postnatal remission of SUI (Figure 1) would find its explanation in the resolution of hormonal and

metabolic changes associated with pregnancy and the spontaneous healing of the traumatic lesions due to vaginal childbirth. Concerning the relapse associated with aging (Figure 2), this would be provoked by tissue aging and be aggravated in case of multiple exposures to pregnancy. If the reversibility hypothesis were altogether accurate, then the cesarean section protective effect would disappear as time passes, and SUI at a mature age would be more closely connected with the number of pregnancies than with the mode of delivery.

Conclusion

At different stages of women's lives, at least five etiological factors can be considered to help explain SUI occurrence: a congenital factor, obesity, aging, pregnancy and vaginal delivery. At different moments, these factors are likely to affect different parts of the urethral sphincter complex. Some, such as aging, are continuous; others, like pregnancy, are occasional; still others, such as weight gain, are reversible. This is why risk factors are significant during certain phases of women's lives, and not during others. At present we do not have a sufficient number of cohorts to allow us to describe the complete natural history of SUI in women or to distinguish age-associated risk from the risk linked with pregnancy and the risk associated with delivery. Childbirth is followed by remission of SUI, and the main indicators measuring pelvic floor quality show improvement postpartum. Cesarean section is associated with a lower rate of postpartum SUI and less frequent recourse to remote SUI surgery. However, it is difficult to postulate this association as causal. In fact, women undergoing cesarean section differ from women undergoing vaginal childbirth with regard to several prenatal characteristics connected with the UI risk. Moreover the one available randomized trial (planned vaginal delivery vs. planned cesarean for breech delivery at full term) is not conclusive.

Further studies still need to be elaborated so as to better understand the respective impacts of pregnancy and obstetrical trauma on urinary continence.

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Legends of figures

Figure 1

Urinary incontinence (continuous line), and stress urinary incontinence (broken line) prevalence (with 95% confidence interval) during first pregnancy and postpartum in longitudinal studies (22,37-57).

Figure 2

Theoretical prevalence of stress urinary incontinence (SUI) in women in accordance with the number of pregnancies, types of deliveries, and age.

The large black line represents SUI prevalence of stress urinary incontinence in nulliparas. The double black line its course during the first pregnancy and afterwards, and the double gray line its course during the second pregnancy and afterwards in women delivered vaginally. The continuous line depicts SUI in accordance with the hypothesis of a reversible obstetrical trauma, and the broken line in accordance with the hypothesis of an irreversible traumatism. Women with cesarean deliveries, thin black line, are located somewhere between nulliparas (large black line) and primiparas (continuous double line) according to the theoretical model of pregnancy-specific effect.

Figure 1

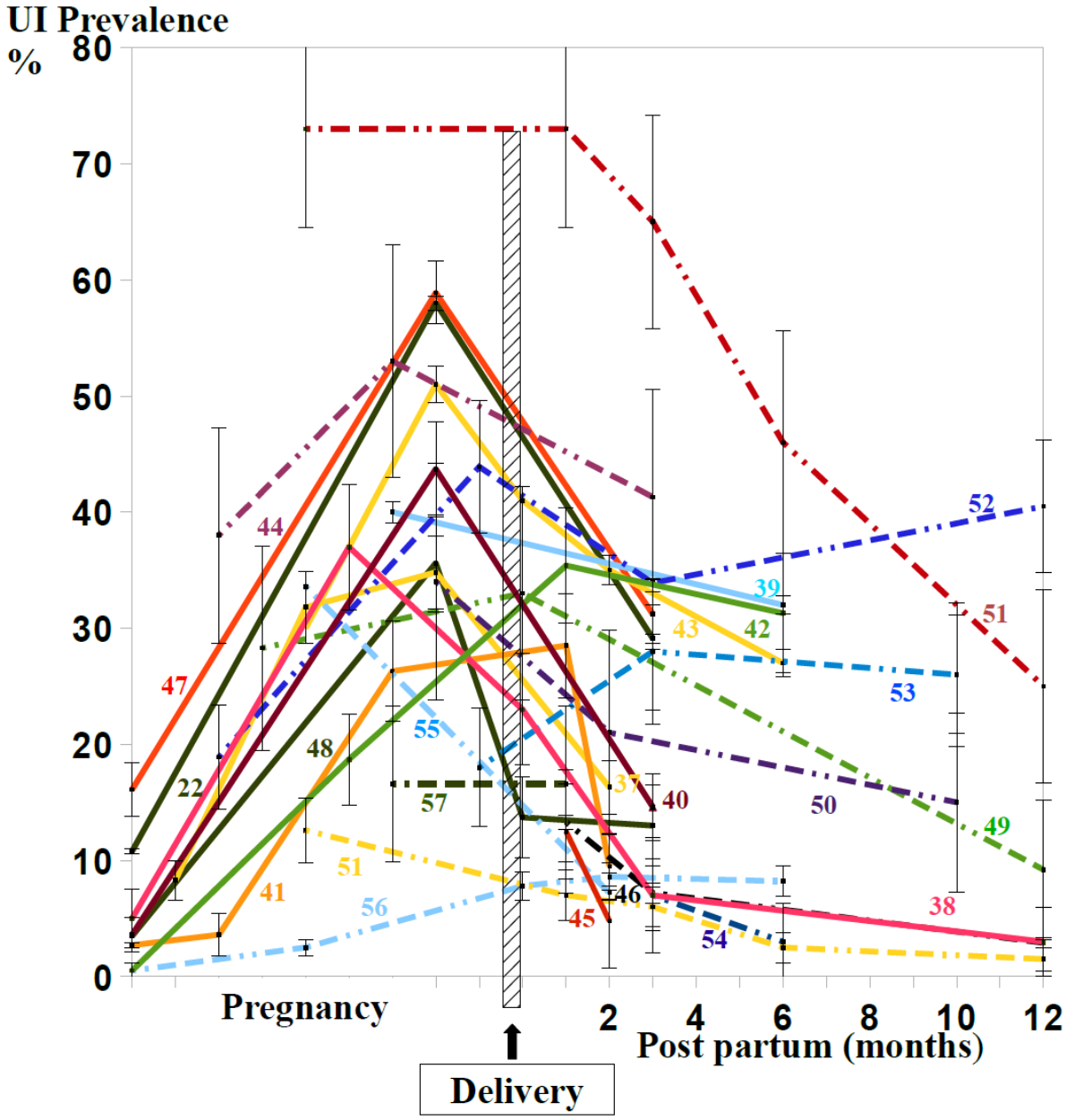
UI Prevalence
%
80
70
60
50
40
30
20
10
0
Pregnancy
Delivery
2
4
6
8
10
12
Post partum (months)
47
44
22
48
41
55
57
51
56
37
40
45
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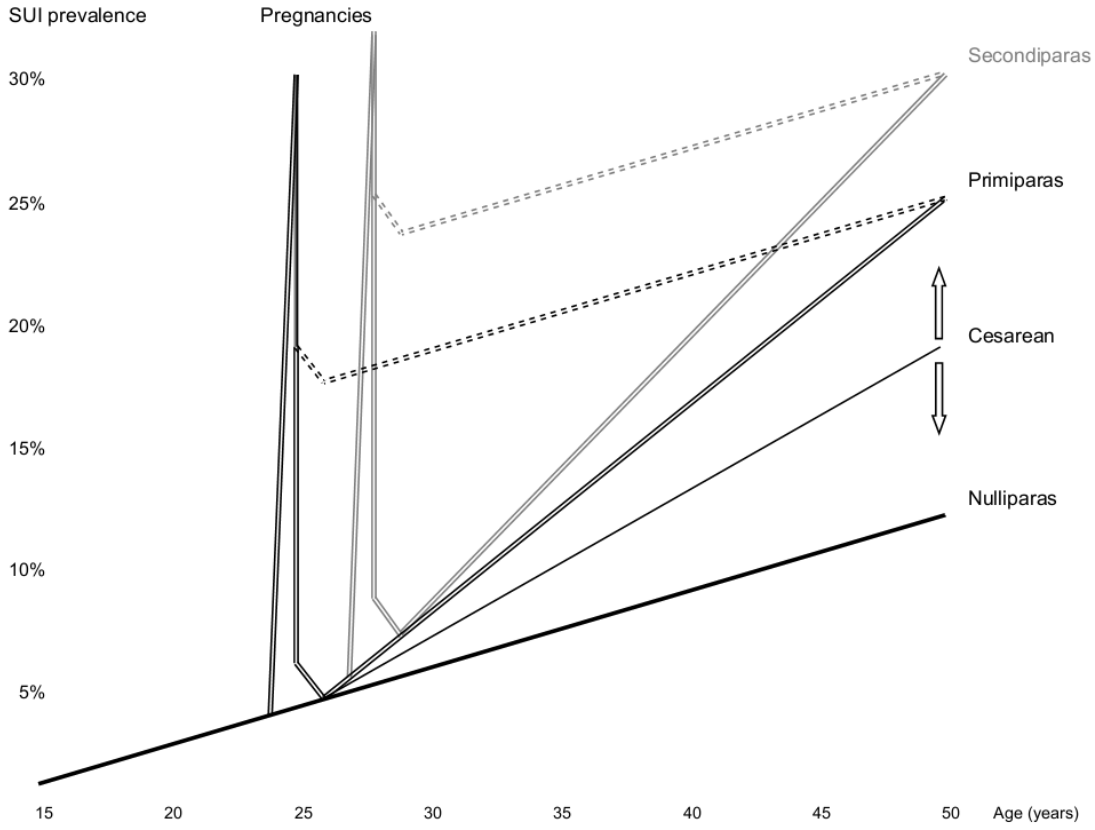
Figure 2


SUI prevalence
Pregnancies
30%
25%
20%
15%
10%
5%
Secondiparas
Primiparas
Cesarean
Nulliparas
15
20
25
30
35
40
45
50
Age (years)