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Stimulating an immune response? Oral sex is associated with less endometritis

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Abstract

Introduction—Adaptive immunity requires antigenic priming of the lymphatic system. As lymphatic tissue is abundant in the oropharynx, oral sex could lead to effective immune stimulation and prevent pelvic inflammatory disease (PID).

Objective—To determine whether oral sex could be a protective factor for PID.

Method—The relationship between self-reported oral sex and endometritis was analysed among 619 women with clinically suspected PID who participated in the PID Evaluation and Clinical Health (PEACH) study.

Results—Nearly one quarter of participants reported oral sex in the past 4 weeks. These women also reported a higher number of sexual partners, a new partner within the past 4 weeks, and a higher frequency of sexual intercourse (all $p < 0.03$). They were more likely to smoke ($p<0.0001$) and use alcohol ($p<0.004$) and recreational drugs ($p<0.02$). Participants reporting oral sex were significantly less likely to be black or to have a positive test for *Neisseria gonorrhoeae* (7.8% vs 21.6%, $p= 0.001$).

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Women who disclosed oral sex were significantly less likely to have endometritis after adjusting for race, number of partners, recent new partner, smoking, alcohol use, and drug use (adjusted OR 0.5 (0.3 – 0.8)).

Conclusion—This is the first paper showing a negative association between oral sex and endometritis. This may be mediated by a protective immune response in the genital tract following priming in the pharynx. This hypothesis needs to be tested in further studies.

Introduction

Upper genital tract infection and inflammation (pelvic inflammatory disease or PID) is one of the most important complications of sexually transmitted infections (STIs) in women. Even adequately treated PID can lead to serious long-term sequelae including tubal factor infertility, ectopic pregnancy, and chronic pelvic pain[1–5].

PID is an expensive illness. The estimated medical cost per case of PID in the United States is $1995 USD[6] for direct care and $1,592 USD[7] for care for sequelae.

PID usually occurs following ascension of infection from the lower to the upper genital tract. An appropriate immune response is important to prevent ascending infection and PID[8]. Adaptive immunity depends on effective presentation of an antigen to the lymphatic system. Koelman et al[9] have suggested that pharyngeal exposure to paternal antigens is more effective in stimulating the adaptive immune system than vaginal exposure. Research by Johansen et al[10] demonstrates that lymphocytes stimulated in the pharynx can be later detected in the endocervix. Thus, we hypothesized that effective priming of the immune system through exposure of STI antigens in the pharynx could reduce the risk of ascending STIs and the development of PID through lymphocyte migration to the genital tract.

Objective

To test the hypothesis that oral sex could lead to more effective immune stimulation than vaginal sex only, resulting in a reduced frequency of ascending infection, we undertook a secondary data analysis from the PEACH study, a randomized controlled trial investigating inpatient versus outpatient treatment of PID.

Method

The PEACH study was undertaken between March 1996 and February 1999 in 13 clinical sites in the United States. It enrolled women aged 14 to 37 presenting to emergency departments, ambulatory clinics, and STI units with symptoms suggestive of PID. Women presenting with a history of pelvic discomfort for a period of 30 days or less, findings of pelvic organ tenderness on bimanual examination, and leukorrhea and/or mucopurulent cervicitis and/or untreated but documented gonococcal or chlamydial cervicitis were offered participation in the study. Women with a current or recent pregnancy or pelvic surgery, antibiotic use within the preceding 7 days, previous hysterectomy or bilateral salpingooophorectomy, tuboovarian abscess, allergy or intolerance to study medications, or homelessness were excluded. Human subject use approval was obtained at each participating institution, and all participants provided informed consent. The details of the methodology are described elsewhere[11].

Participants

Eight hundred thirty-one women meeting all study criteria were enrolled. Our analyses are restricted to 619 participants with complete data on self-reported oral sex and histologically categorized endometritis.

Exposure and Outcome Measures

All measures were obtained within one hour and before treatment was initiated. A 20 minute standardized interview administered by trained study personnel collected demographic data and the social, medical, reproductive, contraceptive and sexual history of participants, as well as information about current or recent reproductive tract symptoms. The interviewer used standardized, scripted questions and interviewer training ensured that all participants were asked exactly the same questions. Questions about general sexual activity and oral sex were introduced with the following statement: “Now I'd like to ask you some questions about your sex life. These questions are somewhat personal. They help us to determine your risk of having sexually transmitted infection. When I say "sex", I mean whenever a man puts his penis into your vagina.” Participants were then asked to answer the following questions with either yes or no: “1) Have you had sex in the last 4 weeks?; 2) Have you had oral sex in the last 4 weeks?; 3) Have you had anal sex in the last 4 weeks?; and 4) Have you used a condom in the last 4 weeks?;” We did not provide any definition of “oral sex” and the question did not differentiate between fellatio and cunnilingus. Further, whether or not ejaculation occurred was not recorded.

All participants then underwent a comprehensive gynaecologic examination with particular attention on physical findings suggestive of pelvic infection or other gynaecologic disorders. Swabs of vaginal fluid were obtained for microscopy and pH measurement, along with Gram staining for diagnosis of bacterial vaginosis[12] (BV) using Nugent’s criteria. Endocervical swabs were collected for culture for *Neisseria gonorrhoeae* (NG), polymerase chain reaction (PCR) testing for *Chlamydia trachomatis* (CT) (Roche Diagnostics, Branchburg, NJ), and *Mycoplasma genitalium* (MG) PCR (in house assay described elsewhere.)[13]

An endometrial specimen was obtained using a sterile endometrial sampler (Unimar Pipelle de Cornier; CooperSurgical, Shelton, CT) after antiseptic cleansing of the exocervix and endocervix to reduce specimen contamination. The presence of *Neisseria gonorrhoeae, Chlamydia trachomatis* and *Mycoplasma genitalium* in the endometrium was detected by culture and PCR, respectively.

Formalin-fixed, paraffin-embedded endometrial tissue was sectioned and stained with both hematoxylin– eosin and methyl-green pyronine (the latter to highlight plasma cells). A modification[14] of the criteria proposed by Kiviat et al[15] was used to define endometritis. A classification of endometritis was given upon finding at least five neutrophils per ×400 field in the endometrial surface epithelium in the absence of menstrual endometrium and/or at least two plasma cells per ×120 field in the endometrial stroma.

Statistical Analyses

Demographic, social, behavioral and microbiological and histological variables traditionally associated with STIs were selected a priori and classified into dichotomous and polychotomous categories and compared between women reporting and not reporting oral sex by the chi-square test of proportions. Multiple logistic regression was used to determine if oral sex was associated with a decreased risk of having endometritis at baseline, after adjustment for potential confounders. Covariates were selected if they were significantly associated with oral sex in bivariate analyses. As variables measuring sexual history were co-linear, the model included a select set of these variables, including number of partners and self-report of a new sexual partner within the past four weeks. All analyses were carried out using SPSS version 18.0.

Results

Among the 619 women in our sample, nearly two-thirds were under 25 years old, almost three-quarters self-identified as black, approximately one quarter were unemployed or looking for work, and just under one quarter reported education beyond high school (Table 1). Ethnicity was the only demographic variable significantly associated with reporting recent oral sex ($p<0.001$), with black women being the least likely to report this activity (Table 1).

Approximately one quarter of participants reported that they had engaged in oral sex in the past 4 weeks. Compared to women who did not report oral sex, these women reported a significantly higher number of sexual partners and a higher coital frequency. They were also significantly more likely to have had anal sex ($p< 0.0001$) or a new partner in the past 4 weeks ($p< 0.03$). In contrast to women who did not report oral sex in the past 4 weeks, those who did report oral sex were also significantly more likely to report smoking, alcohol and recreational drug use ($p<0.0001$, $p<0.004$, $p<0.02$ respectively). There were no significant differences between the two groups of women in reported condom use, oral contraceptive use or vaginal douching.

Participants reporting oral sex were significantly less likely to have a positive test for *Neisseria gonorrhoea* (7.8% vs 21.6%, $p= 0.001$). Although not statistically significant when stratified by race, the association between oral sex and gonococcal infection was similar among blacks and whites (black women: 13.6% vs. 26.1%, $p=0.074$; white women: 7.8% vs. 21.6%, $p=0.3$). There were no statistically significant differences in the rates of *Mycoplasma genitalium*, *Chlamydia trachomatis* infection, mucopurulent cervicitis, leukorrhea or BV between women reporting and not reporting oral sex.

Women who disclosed oral sex in the past 4 weeks were also significantly less likely to have endometritis (OR 0.6 (0.4 – 0.9), Table 2), a finding that remained robust after adjusting for race, number of partners, new partner, smoking, alcohol use, and drug use. Restricting the analysis to those with STI pathogens or BV did not show any significant associations between oral sex and endometritis, although all relationships were in the hypothesized direction and demonstrated a lower likelihood of endometritis among women reporting oral sex. Among women with *Neisseria gonorrhoeae*, there was a borderline significant 80% lower likelihood of having endometritis.

Discussion

Our study showed that women presenting to emergency departments, ambulatory clinics, and STI units with signs and symptoms suggestive of PID were significantly less likely to have endometritis (OR 0.6 (0.4 – 0.9)) if they reported oral sex in the last 4 weeks.

The major strengths of our study include the large sample size, histological categorization of endometritis, and the ability to generalize to populations at risk for STIs and PID. However, there are a few limitations to note. First, while the association between oral sex and a reduced risk of endometritis is strong and biologically plausible, analyses were cross-sectional and thus we are unable to demonstrate a causal relationship. Second, the original data had been collected for a different purpose and only basic information about oral sex was collected. It is not clear how women understood the question and if it was interpreted as fellatio, cunnilingus or both. The hypothesized immunological mechanism for the observed association between oral sex is only hypothesized for women engaging in fellatio. We believe that the ambiguity in the definition of oral sex may have mainly introduced non-differential misclassification, serving to weaken the association between oral sex and the absence of endometritis and has not biased our conclusion. We did not ask why women had

oral sex. It is thus possible that some women with pelvic pain could have used oral sex to avoid dyspareunia. This could lead to differential misclassification if the dyspareunia as it could have reduced the risk of genital STI exposure. However, we did not find any evidence of reduced risk behavior among women reporting oral sex in our study population.

Women reporting oral sex were more likely to report other risk factors for STIs such as alcohol[16, 17] and recreational drug use, new sexual partnerships, anal sex[18], higher coital frequency, and a higher number of life-time sexual partners[19], as well as smoking, which has been identified as an independent risk factor for upper genital tract infection[20]. Despite the associations between oral sex and factors traditionally associated with increased STI risk, controlling for these behavioral variables did not change the relationship between oral sex and lower rates of endometritis. What is more, women in our sample who reported oral sex, compared to those who did not, were significantly (prior to adjusting for race) less likely to have gonorrhoeal and no more likely to have chlamydial infection. While it is possible that some participants practiced oral and anal sex to reduce the risks associated with unprotected vaginal sex (pregnancy and loss of technical virginity) we do not believe that this explanation is responsible for the magnitude of reduction in gonorrhoea prevalence (7.8% vs 21.6% p=0.001, (prior to adjusting for race). Participants who reported oral sex were also significantly more likely to report more frequent vaginal sex, a new partner or anal sex in the last 4 weeks. This suggests that in our study, oral sex was not a substituted behavior for vaginal-penile sex. Alternatively, dyspareunia could be the result of prevalent genital tract infection which should lead positive rather than a negative association between oral sex and genital tract infection (i.e. the opposite of our finding).

It is possible that social desirability bias may have affected disclosure of oral sex, and the clustering of risk factors in women who disclosed oral sex could suggest that women who report oral sex might be less affected by such bias. However, such misclassification would be expected to weaken rather than distort the association. We believe that the relatively low rate of oral sex is better explained by the infrequent practice of oral sex reported in the late 1990s among predominately black populations[21].

Confounding by race may explain the association between oral sex and a lower gonorrhoea prevalence as black women report less oral sex[21] and may have a two to four times higher gonorrhoea prevalence than white women[22]. Following stratification by race the negative association between oral sex and gonorrhoea prevalence did not reach statistical significance (black women p=0.074; white women: p=0.3). Confounding by race could however not explain the negative association between oral sex and endometritis.

Secondary analysis always carries a risk of spurious associations. We believe that the risk for this is small as our analysis was driven by independently generated hypothesis. Lastly, endometritis is a strong indicator of salpingitis but it is not the same and our paper only assesses endometritis as defined above[15]. The absence of endometritis does not exclude PID and endometritis might not be associated with the same risk of infertility[23]. However compared with laparoscopically diagnosed PID, endometritis has a sensitivity of 70%–89% and a specificity of 67%–92%[24–26] and is probably the best surrogate measurement for PID where a gold standard diagnosis with laparoscopy and fimbrial biopsy is not possible. We accept that the use of a surrogate measurement may have introduced a bias.

Interpretation

Our findings are consistent with our hypothesis that pharyngeal exposure to antigens can stimulate an adaptive immunological response in the genital tract. Experimental research by Johansen et al[10] showed that lymphocytes primed in the nasopharynx can later be found in the endocervix. Johansson et al[27] showed that antigenic exposure of the pharynx has been

found to result in a stronger immune response (IgA) in women than antigenic exposure of the vagina. Cuburu et al[28] were able to induce a genital B cell and T cell response following sublingual immunization.

Our findings are consistent with our hypothesis that pharyngeal exposure to antigens is more effective in the induction of an adaptive immunological response than vaginal exposure. Lymphatic tissue is abundant and exposed in the pharynx while it is sparse and not exposed in the vagina. It is thus possible that pharyngeal exposure could be more effective than genital exposure in priming or boosting the immune system. If this were the case, one could expect that STIs could be cleared faster from the oropharynx than the genital tract. This hypothesis is supported by the findings of Winkström et al[29] who showed that of 128 women with confirmed genital chlamydial infection who also practiced unprotected receptive oral sex, only 9 had a positive pharyngeal strand displacement amplification (SDA) test for chlamydia. An alternative explanation is that genital priming of the immune system could reduce pharyngeal carriage.

Oral sex reduced the cervical prevalence of gonorrhoea (prior to adjusting for race) but did not of chlamydia or *Mycoplasma genitalium*. We believe that this could be explained by differences in pharyngeal exposure to STI antigens. Urethral discharge is more likely in gonorrhoeal than in chlamydial or mycoplasmal infection. Unless ejaculation occurs, pharyngeal STI antigen exposure relies on the presence of antigens on the glans penis. Moncada et al[30] showed that self collected swabs from the glans penis had a good sensitivity of the nucleic acid amplification tests for *Neisseria gonorrhoeae* (>92%) but not for *Chlamydia trachomatis* (56 to 68%).

The immune response primed in the pharynx could also have a protective effect in the genital tract. It could either lead to a faster elimination of STI pathogens or by prevention of ascending infection. The former is supported by our finding of lower gonorrhoea prevalence in women who reported engaging in recent oral sex (prior to adjusting for race) and by research in animal models, showing that oral vaccination in the mouse model reduced the bacterial load of genital chlamydial infection[31]. Faster elimination of pathogens from the lower genital tract may in turns reduce the risk of ascending genital tract infection.

We are not the first to formulate the hypothesis that oral sex could stimulate the immune system. Pre-eclampsia, a hypertensive disorder of pregnancy, is associated with maternal exposure to paternally derived antigens of the fetus. Pre-eclampsia is more frequent in women who have not had a prolonged period of unprotected sex with father of the foetus prior to conception[32–34]. Koelman et al[9] found that women who practiced oral sex with their partner had lower odds of developing pre-eclampsia and attributed this to the improved recognition HLA derived peptides in the seminal fluid through oral sex.

While our findings are plausible it is important not to overstate our case. Our findings need to be confirmed in other studies specifically designed to test our hypothesis. However if the association could be confirmed it could have important implications for health care and public health. National surveys of sexual behaviour in the U.K. and U.S. show that oral sex is part of the normal sexual repertoire[35,36]. Oral sex has traditionally been viewed as high risk behaviour. However, our data suggests that instead it might have a protective effect. As condom promotion, hormonal contraception[37] and chlamydia screening have been inconsistently linked to reductions in STIs or PID, the discovery of new factors protective against lower and upper genital tract infection is of particular importance.

Our findings of a potentially beneficial effect should be interpreted with caution. There is strong evidence that unprotected oral sex in high risk situations contributes to STI transmission in men who have sex with men[38] and male clients of commercial sex

workers[39]. Transmission of oncogenic HPV through oral sex has been assumed to be the driver in the increase of oral cancers[40] and the transmission of other bacterial or viral STIs including HIV has been reported. On the other hand if oral sex reduces the bacterial load of STIs in the lower genital tract, then unprotected heterosexual, non transactional oral sex may not carry many health risks and may have advantages both for the individual and the population.

Conclusion

This is the first study showing that oral sex was associated with a reduced likelihood of histologically confirmed endometritis among patients with clinically suspected PID, even after adjustment for traditional STI risk factors. We believe that this association is best explained through effective priming of the immune system through oral sex, a hypothesis supported by studies in reproductive immunology. We hope that our findings will stimulate the research community to test our hypothesis further. In particular it would be interesting to measure the immune response in women with pharyngeal and genital tract STIs.

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Dr Haggerty had had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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Summary

Secondary analysis of the PEACH data suggests that among women presenting with signs and symptoms of PID, those who reported oral sex were less likely to have endometritis (adjusted OR 0.5 (0.3 – 0.8) than those who did not report oral sex.

Table 1

Characteristics of Women Reporting and Not Reporting Oral Sex at Baseline

	Oral Sex + N=145 n (%)	Oral Sex− N=474 n (%)	p-value
Demographic Characteristics			
Age			
<=19	37 (25.5)	120 (25.3)	0.70
20–24	62 (42.8)	187 (39.5)	
>=25	46 (31.7)	167 (35.2)	
Race			
Black	69 (47.6)	370 (78.1)	<.0001
White	51 (35.2)	62 (13.1)	
Other	25 (17.2)	42 (8.9)	
Education			
< HS grad	51 (35.2)	186 (39.2)	0.65
HS grad	58 (40.0)	173 (36.5)	
> HS grad	36 (24.8)	115 (24.3)	
Activity past 4 weeks:			
Working or homemaker	96 (66.2)	296 (62.4)	0.64
Student	10 (6.9)	48 (10.1)	
Unemployed/Looking for work	36 (24.8)	117 (24.7)	
Disabled/other	3 (2.1)	13 (2.7)	
Sexual History (past 4 weeks) and Contraception History			
Had sex	145 (100.0)	374 (78.9)	<.0001
Number of partners (mean, sd)	1.8 (5.0)	0.87 (.62)	0.02
New partner	21 (14.5)	40 (8.4)	0.03
Frequency of sex/week (mean, sd)	4.8 (6.7)	2.2 (3.1)	<.0001
Anal sex	10 (6.9)	7 (1.5)	<.0001
Contraception History			
Oral Contraception			
No	127 (87.6)	329 (88.0)	0.91
Yes	18 (12.4)	45 (12.0)	
Condom Use			
Consistent condom use	16 (11.0)	46 (12.3)	0.69
Inconsistent condom/no contraception	129 (89.0)	328 (87.7)	
Behavioral Characteristics			
Smoking Status			

	Oral Sex + N=145 n (%)	Oral Sex− N=474 n (%)	p-value
Never smoked	44 (30.3)	259 (54.9)	<.0001
Past smoker	10 (6.9)	28 (5.9)	
Current smoker	91 (62.8)	185 (39.2)	
Alcohol Use past 4 weeks *			
None	48 (33.1)	230 (48.7)	0.004
<= 15 drinks/week	82 (56.6)	209 (44.3)	
> 15 drinks/week	15 (10.3)	33 (7.0)	
Other recreational drugs			
None	90 (65.7)	358 (77.5)	0.02
Marijuana	44 (32.1)	100 (21.6)	
Other drugs	3 (2.2)	4 (0.9)	
Douching History Past 4 Weeks			
None	88 (60.7)	271 (57.4)	0.48
Any	57 (39.3)	201 (42.6)	
Clinical Characteristics at Baseline			
Baseline STI (cervical and/or endometrial)			
CT	29 (22.5)	102 (23.6)	0.80
GC	8 (7.8)	79 (21.6)	0.001
GC and/or CT	37 (27.4)	179 (40.5)	0.006
MG	13 (12.0)	48 (13.3)	0.73
Mucopurulent Cervicitis	70 (53.4)	269 (62.0)	0.08
Leukorrhea	115 (80.4)	406 (86.9)	0.05
BV	66 (51.6)	262 (60.2)	0.21

*Alcohol units: wine, 1 glass = 4 ounces; beer, 1 can = 12 ounces; hard liquor, 1 shot = 1 ounce

Table 2

Association between Oral Sex and Endometritis

	Oral Sex+ n (%)	Oral Sex n (%)	OR (95% CI)	OR_{Adj} (95%CI)*
Entire Cohort (N=619)				
Endometritis+	56 (38.6)	246 (51.9)	0.6 (0.4 – 0.9)	0.5 (0.3 – 0.8)
Endometritis−	89 (61.4)	228 (48.1)	p=0.005	p=0.007
Subset of CT+ (N=131)				
Endometritis+	19 (65.5)	71 (69.6)	0.8 (0.3 – 2.0)	0.6 (0.2 – 1.9)
Endometritis−	10 (34.5)	31 (30.4)	p=0.675	p=0.356
Subset of GC+ (N=87)				
Endometritis+	4 (50.0)	65 (82.3)	0.2 (0.05 – 1.0)	0.2 (0.01 – 2.2)
Endometritis−	4 (50.0)	14 (17.7)	p=0.032	p=0.176
Subset of MG+ (N=61)				
Endometritis+	7 (53.8)	35 (72.9)	0.4 (0.1 – 1.5)	0.2 (0.03 – 1.2)
Endometritis−	6 (46.2)	13 (27.1)	p=0.188	p=0.082
Subset of BV+ (N=328)				
Endometritis+	31 (47.0)	146 (55.7)	0.7 (0.4 – 1.2)	0.5 (0.3 – 1.0)
Endometritis−	35 (53.0)	116 (44.3)	p=0.202	p=0.062

*Adjusted for race, number of partners, new partner, smoking, alcohol use, drug use