

Review

What is the evidence for non-sexual transmission of gonorrhoea in children after the neonatal period? A systematic review

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Abstract

International consensus guidelines state that *Neisseria gonorrhoeae* infection in pre-pubertal children is always, or nearly always, sexually transmitted. A systematic literature review does not concur with this. *N gonorrhoea* was believed to solely sexually transmitted when first identified in the 1880s. However it became recognised that when the infection was introduced into children's institutions, it rapidly spread among pre-pubertal girls. The medical literature records over 40 epidemics involving about 2000 children in Europe and the United States. Communal baths, towels or fabric, rectal thermometers and caregivers hands were identified as means of transmission. Although sensitive to heat and drying, gonorrhoea may remain viable in pus on cloth for several days. Several unusual accidental transmissions are reported, often due to contamination from laboratory samples. Indirect transmission occurs in epidemics of conjunctivitis in third world rural populations. Spread of infection can occur via contaminated hands of infected caregivers. While all paediatric cases of gonorrhoea must be taken seriously, including contact tracking and testing, forensic medical examiners should keep an open mind about possible means of transmission. Doctors and lawyers need to be cognisant of the large body of literature demonstrating both sexual and non-sexual means of transmission of gonorrhoea in children.

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1. Introduction

International guidelines and other authorities state that *Neisseria gonorrhoeae* infection in pre-pubertal children is always, or nearly always, a sexually transmitted disease.^{1–16} All these documents conclude that transmission after the neonatal period is likely to be sexual and are strongly supportive of sexual abuse.

In New Zealand, the finding of gonorrhoeal infection in a pre-pubertal child beyond the neonatal period is considered diagnostic of sexual abuse.¹⁷ The presence of this infection designates sexual abuse a medical certainty with urgent action required by the social agencies,

police and the courts. It was assumed that all 14 reported cases of gonorrhoea in pre-pubertal children in Auckland between 1991 and 2002 must have been due to sexual abuse with the child requiring urgent placement 'in a safe household'.¹⁸ In many cases there was no disclosure and the perpetrator was deduced 'based on who was in contact with the child during the incubation period'. Several families fled the country; some children were taken into custody and some prosecutions occurred. One mother was considered to be the perpetrator and lost custody of her child.

Clearly sexual transmission should be immediately considered whenever gonorrhoea is found in a pre-pubertal child. Missing a case of sexual molestation has serious social and legal consequences. However what is the possibility that this organism can be spread non-sexually on occasion? Removing a child from its parents on the basis

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of a wrongful assumption of sexual abuse, or charging an adult with sexual assault, may have taken an equally damaging toll. What is the evidence on which the guidelines and other authorities base their statements?

Of concern, the guidelines either do not refer to primary studies to back their claims, or they rely on a 1984 review article which claims to have summarised ‘*all studies of gonococcal infections in children since 1965*’.¹⁹ However this is not a systematic review; it fails to refer to much of the published literature during that period and omits reference to a number of pertinent studies before their review time, as well as those published since.

Therefore the aim of this review is to systematically examine the literature on the possible non-sexual modes of transmission of *Neisseria gonorrhoeae* in children after the neonatal period.

2. Method

A comprehensive literature review was conducted on modes of non-sexual transmission of gonorrhoea. The Cochrane library, Medline (1966–2006), Embase (1980–2006) and CINAHL (1982–2006) databases were searched using MeSH terms *Neisseria gonorrhoeae*; child; child, pre-school; disease transmission; sexually transmitted diseases, bacterial and the non-MeSH terms non-sexual and non-venereal infection. Grey literature sources included policy statements and guidelines on websites of appropriate professional bodies. A large number of additional studies were identified by checking reference lists of retrieved papers. Key authors were contacted. Selection criteria were all experimental or observational studies regarding non-sexual transmission. Papers not published in English were excluded but reviews reporting details of case series not published in English were included in cases of institutional epidemics. Both in vitro and in vivo transmission were included.

Because this is a review of aetiology rather than intervention, the level of evidence available is from observational studies (cohort studies, case control studies, case series and case reports) rather than randomised controlled trials.

3. Results

Sixty one primary sources of case reports and series of both in vitro and in vivo studies were accessed and reviewed. A further three reviews^{20–22} were secondary sources of 11 case series of institutional epidemics. These have been included because the primary sources were unable to be accessed or were not in English. An evidence table was produced (Table 1) and the papers were graded according to whether all or some of the contained cases were due to definite, probable or possible non-sexual transmission. As well as sources of primary data, a number of guidelines containing un-evidenced opinion data plus background information were reviewed. A number of references

were excluded because they were opinion-based or fell beyond the scope of this review (such as case series of inoculating male volunteers with gonococci or treatment outcomes).^{23–34}

The large and diverse body of literature recovered from this search strategy is reviewed under a number of sub-headings.

3.1. Identification of the *N gonorrhoeae* organism

Neisseria gonorrhoeae is a gram negative diplococcal bacterium. It can be diagnosed microscopically by seeing the bacteria inside cells from gram-stained smears of secretions, or by culture of the bacteria on selective media wiped with infected swabs. However there are many other species of *Neisseria* (for example, *N lactamica*, *N cinerea*, *N meningitidis*). Non-gonococcal *Neisseria* species are part of the normal flora in the mouths and throats of adults and children.³⁵

In cases with no prior suspicion of sexual abuse, the *Neisseria* should be confirmed as a gonococcus before a sexual abuse investigation is instigated. In a study where 40 bacterial isolates identified as *N gonorrhoeae* from children under age 15 were submitted to the Sexually Transmitted Disease Laboratory Program at the Centers for Disease Control to confirm the identity of the bacteria, 14 proved to be species other than *N gonorrhoeae*. Unnecessary investigations for child sexual abuse were conducted in eight cases.³⁵ Given the limitation of any single test, it is important that identification of *N gonorrhoeae* is made by testing isolated organisms rather than by direct identification of organisms in patient samples (which might wrongly identify a commensal *Neisseria* as *N gonorrhoeae*). The isolate should be able to be stored and the identity reconfirmed if necessary.

3.2. Sites of infection of *N gonorrhoeae* in the pre-pubertal child

Neisseria gonorrhoeae infects mucus membranes, for example, inside the mouth, the conjunctivae of the eyes, the urethra, the vagina and cervix and the anal canal.

Lack of oestrogen renders the mucous membrane of a pre-pubertal girl's vagina more delicate than that of an adolescent or adult and particularly susceptible to infections such as gonorrhoea.³⁶ The vaginal pH in pre-pubertal and pubertal girls is 6.5–7.5 and 3.5–4.5, respectively. The relatively alkaline environment of the pre-pubertal vagina is much more easily colonised and infected on exposure to *N gonorrhoea* than that of post-pubertal girls because the susceptible epithelium (membranous tissue) is considerably more superficial. Whereas in the adolescent and the adult it is the cervix and uterus that becomes infected, in the child the infection is usually vaginal, with purulent discharge, redness and swelling of the vaginal lips.³⁷ The pre-pubertal child is susceptible to gonococcal vulvovaginitis for several reasons:

Table 1
Summary of studies with definite or possible cases on non-sexually transmitted gonorrhoea

Study	Study type	Location	Population	Gender/age	Gonococcal infection	Mode of transmission	Non-sexual transmission (some or all)
Matters et al. ⁵⁹	Case series	Outback Australia	447 Reported cases, 242 confirmed	77% 0–9 years 5% >14 years 113 male; 129 female	Epidemic of conjunctivitis	Probable hands, cloths or bush flies	Definite
Anon ⁶²	Case series	Outback Australia	13 Cases	Age and gender not specified	Epidemic of conjunctivitis	Probable hands, cloths and/or bush flies	Definite
Mikru ⁵⁵	Case series; case control series	North Omo, Ethiopia	9075 Cases; 216 cases/146 controls	55% <5 years No significant gender difference	Epidemic of conjunctivitis	Probable hands, cloths and/or bush flies	Definite
Merianos ⁶⁰	Case series prospective study	Central Australia	432 Cases Aboriginal	68% Age 0–9 years aged 0–4 27 × more likely than adults No gender difference	Epidemic of conjunctivitis	Probable hands, cloths and/or bush flies	Definite
Van Buynder ⁵⁸	Case series	Western Australia	42 Reported cases, 19 confirmed Aboriginal	Cases peaked in 5–9 years group No gender difference	Epidemic of conjunctivitis	Probable hands, cloths and/or bush flies	Definite
Monger ¹⁰¹	Case series	Central Australia	20 Cases Aboriginal	90% Aged 6 months–9 years >50% 0–4 years No gender difference	Epidemic of conjunctivitis	Probable hands, cloths and/or bush flies	Definite
Brennan ⁵⁷	Case series	Central Australia	140 Cases	Age range 6 week–63 years; only eight cases >15 years 33% 2–5 year old	Epidemic of conjunctivitis	Probable hands, cloths and/or bush flies	Definite
Matters ⁵⁶	Case series	Outback Australia	23 Cases Aboriginal	3–18 years Aboriginal	Epidemic of conjunctivitis	Probable hands, cloths and/or bush flies	Definite
Lipsiatt ⁵⁰	Case report	Texas, USA	1 Case	3 years old boy	Accidental transmission, throat	Ingestion of laboratory specimen (chocolate agar)	Definite
Diena ⁵¹	Case report	Ottawa, Canada	1 Case	Male adult	Accidental transmission, conjunctivitis	Inoculation by laboratory specimen (strap of mask)	Definite
Bruins ⁵²	Case report	Indianapolis, USA	1 Case	37 years old male	Accidental transmission, conjunctivitis	Inoculation by laboratory specimen (spray from syringe)	Definite
Valenton ⁵³	Case series	Manilla, Phillopinnes	13 Cases	7 Males, 6 females Age 17–33 years	Accidental transmission, conjunctivitis	Inoculation of eyes by infected urine	Definite
Dobszay ¹⁰² German	Experimental inoculation	Germany		Various items contaminated either artificially or by letting infected children play with them	Organism grown from rabbit skin after 54 h, wet linen after 24 h, dry linen after 1/2 h, rubber, water and wood after 2 h, metal after 10 min	Inoculation various fomites	Probable in vitro Secondary source described by Benson ⁴³
Scholtz	Experimental				Organism recover from bath water after 24 h	Contaminated bathwater	Possible Secondary source described by Leishman ⁴¹

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Table 1 (continued)

Study	Study type	Location	Population	Gender/age	Gonococcal infection	Mode of transmission	Non-sexual transmission (some or all)
Elmros ⁴⁰	Experimental inoculation	Sweden	10 Tests	N/a	Urethral pus on glass slide or towel Organism recovered after 17 h on glass; 24 h on towel	Inoculation glass slide or towel	Definite in vitro
Alausa ⁴²	Experimental inoculation	Ibadan, Nigeria	Three experiments	N/a	Pus suspension on cloth Organism recovered after 2–3 h if cloth damp	Inoculation cloth	Definite in vitro
Gilbaugh ⁴⁵	Experimental inoculation infected suspensions or pus	Portland	15 Sections toilet seat; 15 strips toilet paper	N/a	Pus: organism recover from seat and paper after 2–3 h	Inoculation toilet seat and toilet paper	Definite in vitro
Srivastava ⁴⁴	Experimental inoculation	Cardiff	At various intervals, exudate cultured	N/a	Pus on range of sterile test materials Organisms recovered from most materials after 24–48 h, from few materials (cotton swab, white cardboard and wooden spatula) ≥ 72 h	Hard substances (glass, plastic, cellophane, wood, cardboard and paper and soft substances (cotton swab, cotton gauze, linen handkerchief, cotton towel, tissue paper and lubricated condom)	Definite in vitro
Czeri ¹⁰³ German	Case series	Buda-Pesth	26 Cases	25 Girls, 1 nurse	Institutional epidemic of vulvovaginitis, 1 case ophthalmia (nurse)	Admission to hospital of case of vulvovaginitis Possible bedding, instruments, bandages, mutual contact	Probable Secondary source (described by Baer ²¹ and Hamilton ¹⁰⁴)
Lennander ¹⁰⁵ Swedish	Case series	Stockholm, Sweden	18	Girls in children's hospital	Institutional epidemic of vulvovaginitis	Admission to hospital of case of vulvovaginitis	Probable Secondary source (described by Baer ²¹)
Leszynski ¹⁰⁶	Case series	New York City	35	Girls in Hebrew Orphan's Home	Institutional epidemic of vulvovaginitis and ophthalmia (18)	Admission of four cases of vulvovaginitis started epidemic	Probable Secondary source (described by Baer ²¹)
Dusch ¹⁰⁷ German	Case series	Heidelberg	9	Girls in children's hospital	Institutional epidemic of vulvovaginitis	Admission to hospital of case of vulvovaginitis	Possible Secondary source (described by Baer ²¹)
Succhar ¹⁰⁸ French	Case series	Lavey	11 and 12 (2 series)	Girls in hospital	Two institutional epidemics of vulvovaginitis	Hydrotherapy common bath	Possible secondary source (described by Baer ²¹)
Skutch ¹⁰⁹ German	Case series	Posen, Poland	236 Cases within 8–14 days	Girls 6–14 years using public bath	Epidemic of vulvo-vaginitis	Common use of public bath, towels and soap	Probable Secondary source (described by Abt, ²⁰ Baer ²¹ and Hamilton ¹⁰⁴)
Fischer ¹¹⁰ German	Case series	Altona	40 Cases	Girls in children's hospital	Institutional epidemic of vulvo-vaginitis	Developed infection following admission of case	Probable Secondary source (described by Abt ²⁰ and Baer ²¹)

Weill ¹¹¹	French	Case series	France	20 Cases	Children in hospital	Institutional epidemic of anogenital infection	Developed infection following admission of case of vulvovaginitis. Probable rectal thermometer. Cases ceased once instrument disinfected	Possible Secondary source (described by Baer ²¹)
Sheffield ⁶⁹		Case series	New York City	66 Cases	65 Girls in Hebrew Orphan's Home 4–14 years 1 boy age 10 (infected eye)	Institutional epidemic of vulvovaginitis ± ophthalmia (6), peritonitis (4), synovitis (1)	Developed infection following admission of case. Common bathing 20–30 per bath Boy used towel of infected child	Highly probable
Cnopf ¹¹²	German	Case series	Nuremberg	10 Cases	Infants in children's hospital	Institutional epidemic	Developed infection following admission of case of ophthalmia	Possible Secondary source (described by Baer ²¹)
Skiba-Zaborowska ¹¹³	German	Case series	Zurich	42 Cases	Girls in children's hospital	Institutional epidemic of vulvovaginitis	Developed infection following admission. Epidemic ceased when children with vaginal discharge isolated on admission	Probable source (described by Baer ²¹)
Koplik ⁷³		Case series	New York	10 Cases	Girls in children's ward	Institutional epidemic	Developed infection following admission of two cases of boys with rectal infection. Further cases prevented by strict isolation and testing of children at admission	Probable Thorough isolation techniques stopped epidemics
Kimball ¹¹⁴		Case series	New York, USA	63 Cases	Girls in Babies' Hospital	Institutional epidemic of vulvovaginitis	Developed infection in hospital following admission of infected girl	Probable Secondary source (described by Baer ²¹)
Bauer ²¹		Case series	Chicago, USA	19 Cases	18 Girls, 1 boy in hospital 6 months–13 years	Institutional epidemic of vulvovaginitis and urethritis	Probable contamination from rectal thermometer and transportation in hospital cart with infected children. Epidemic controlled by strict isolation procedures	Probable
Welt-Kakels ¹¹⁵		Case series	New York	190 Cases	Girls at Mount Sinai Hospital 0–13 years 59% 2–6 years 96% <10 years	Institutional cases of vulvovaginitis	Mostly developed in hospital	Possible–insufficient detail
Cotton ⁷¹		Case series	Chicago	19 Cases	18 Girls, 1 boy in hospital	Institutional epidemic vulvovaginitis, ophthalmia	All but two developed in hospital. Probable transmission by fomite, especially rectal thermometer or nurses. Epidemic controlled by strict isolation techniques	Probable
Holt ⁷²		Case reports	New York	28 Cases	26 Cases arthritis (19 male) 2 vulvovaginitis	Institutional epidemic	Vulvovaginitis transmitted by nurse	Highly probable
Hamilton ²²		Case series	Chicago	82 Cases	Vulvovaginitis Cook County Hospital 1–13 years 75% <7 years	Institutional epidemic vulvovaginitis	Cared for in the same wards, shared bath tub and nurses Epidemic controlled by identifying cases and strict isolation regimes	Highly probable

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Table 1 (continued)

Study	Study type	Location	Population	Gender/age	Gonococcal infection	Mode of transmission	Non-sexual transmission (some or all)
Seippel ⁷⁴	Case series	Chicago	535 Cases over 2 years	Mostly girls, about 60 boys at Cook County Hospital 73% <6 years	Institutional epidemic vulvovaginitis, urethritis, conjunctivitis	Transmitted by bedding, towels, bath tubs, fingers of nurse; Possible sexual on occasion	Possible Insufficient detail regarding timing on infection with respect to admission Highly probable
Cooperman ⁷⁵	Case series	Philadelphia	67 Cases	Rectal and vulvovaginal infections. Fifty three cases arthritis. All infants in same ward, Mt. Sinai Hospital	Institutional epidemic rectal infection, vulvovaginitis and arthritis	Probable transmission through rectal thermometer	Highly probable
Burry ⁸⁷	Case series	Kansas	36 Cases	31 Girls, 5 boys. <10 years	Vulvovaginitis/urethritis	Possible non-sexual contact with infected cases 6 cases sexual or suspected sexual	Possible
Shore ⁷⁶	Case series	Alaska	15 Cases	8 Girls, 6 boys 71% <6 years	Vulvovaginitis, urethritis, conjunctivitis	Bed-sharing with infected relatives; contaminated hands of mothers Reported non-sexual but possible in some cases	Probable
Doyle ⁸¹	Case report	Birkenhead, UK	2 Cases	Girl age 3 and her brother age 2 years	Conjunctivitis	Shared towels with infected parents	Probable
Allue ⁸⁵	Case series	Oklahoma, USA	8 Cases	Aged 2–13 years, 2 boys	Conjunctivitis, urethritis, vulvovaginitis, arthritis	Two probable non-sexual for infected neonatal sibling (1 conjunctivitis, 1 vulvovaginitis 6 probable sexual	Probable (2 cases)
Tunnessen ⁸⁸	Case report	New York, USA	5 Cases	5 years old girl, 2 years old sister, 7 years old cousin, 12 years old uncle and mother	Vulvovaginitis/urethritis	Probable non-sexual contact, possible sexual	Possible
Doyle ⁹²	Case report	Southampton, UK	1 Case	8 months old boy	Conjunctivitis	Probable shared towel of 21 years old lodger with salpingitis	Probable
Osaba ⁸⁴	Case series	Nigeria	17 Cases	Age 2–9 years		Three sharing towels or underpants with infected relatives 1 housemaid positive contact. Sexual probable in 2 cases	Possible
Auman ⁸²	Case report	Raleigh, USA	1 Case	Girl 5 years	Vulvovaginitis	Probable sharing bed with 16 years old infected female cousin Sexual improbable	Probable
Folland ⁸⁶	Case series	Nashville, USA	67 Cases	<10 years	Vulvovaginitis (48), urethritis (11), conjunctivitis (8)	Possible non-sexual transmission by family members in some case; probable sexual transmission in 18	Possible

Low ⁹³	Case series	Kansas, USA	43 Cases	38 Girls, 5 boys 26% <10 years Crowded households	Vulvovaginitis, nasal, rectal, throat	Adolescents probably sexual five cases probably non-sexual	Probable
Felman ⁷⁸	Case report	New York, USA	1 Case	Girl 3 years	Vulvovaginitis	Transmission from infected mother	Possible
Reynolds ⁷⁹	Case report	Cambridge, UK	1 Case	Girl 3 years	Vulvovaginitis	Infected Filipino nursemaid Sexual improbable	Probable
Frewen ⁸⁴	Case series	Toronto, Canada	18 Cases	Girls 2–10 years	Vulvovaginitis	Eight shared bed with infected family member; five from infected households; three probable sexual contact	Possible
Meek ⁸⁴	Case series	Maryland, USA	45 Cases	35 Girls, 10 boys age 1–9 years	Vulvovaginitis/urethritis	Either non-sexual or sexual household contact. Sexual contact uncommon or unsuspected children <3 years	Possible
Alausa ¹¹⁶	Prospective study	Ibadan, Nigeria	37 Cases	Girls 1–10 years 71% 1–6 years	Family contacts mostly female. Vulvovaginitis	Possible fomite transmission (towel, bedding, underclothes) Possible sexual	Possible (probable in some cases)
Ingram ⁸⁹	Case series	Raleigh	17 Cases	Age 0–4 years	Vulvovaginitis/urethritis	Four lived with older infected female members; Six had sexual contact	Possible
Alexander ⁹⁸	Case series	Alabama, USA	36 Cases	Aged 1–10 years 32 girls, 4 boys	Household contacts vulvovaginitis/urethritis	Possible non-sexual contact with infected cases Some sexual	Possible
Alexander ⁹⁸	Case series	Alabama	36 Cases	32 Girls, 4 boys age 1–11 years	Vulvovaginitis/urethritis	Infected household or non-household associates 18; sexual contact 18	Possible
Ismail ⁸³	Case series	Malaysia	16 Cases	Girls age 1–9 years 75% <6 years From low socio-economic over-crowded households	Vulvovaginitis	Possible non-sexual household contacts or fomite transmission. Sexual contact suspected in 3	Possible
Nair ⁹⁹	Case reports	Baltimore, USA	14 Index cases	12 Girls, 2 boys Age 2–12 years From low socio-economic over-crowded households	Vulvovaginitis/throat	Nine siblings or other children contacts tested positive. Either non-sexual or sexual household contact	Possible
Lewis ⁸⁴	Case reports	Philadelphia, USA	4 Cases	Three boys aged 11, 15 and 26 months; 1 girl age 8 years	Conjunctivitis	Shared bed ± wash cloth with infected relatives	Probable
Low ⁷⁷	Case report	Brazil	3 Cases	2 Sisters 2 and 3 years; 1 girl 2 years	Vulvovaginitis	Transmitted by infected mother's hands	Probable
Ingram ⁹⁷	Case series	Raleigh, USA	84 Cases	Girls 1–12 years	Vulvovaginitis	Either non-sexual or sexual contact	Possible
Dada-Adegbola ⁹⁰	Case series	Ibadan, Nigeria	84 Cases	82 Girls, 2 boys Age 1–10 years	Vulvovaginitis/urethritis/ ±conjunctivitis	30% Fathers and 22% Mothers tested positive. Nine had history of sexual contact	Possible
Kelly ¹⁸	Case series	Auckland, New Zealand	14 Cases	12 Girls, 1 boy age 3–8 years	Vulvovaginitis/urethritis	Five positive relatives including baby's eye, mother, cousin, uncle, father. No prior disclosure or concern about sexual abuse All assumed sexual abuse because positive GC	Possible
Dayan ¹⁰⁰	Case report	Plane Russia to Sydney	1 Case	Girl age 8 years	Vulvovaginitis	Probable self-inoculation from toilet paper wiped on infected seat	Probable

- the vulva lacks the protection of adult labial fat pads and pubic hair and the labia minora tend to open when the child squats;
- the vulval skin is thin, delicate and sensitive;
- the vulva are susceptible to irritation, infection, drying, chapping and blistering;
- the vagina is an excellent bacterial culture medium being warm, moist and of neutral to slightly alkaline pH;
- the vagina has a thin, atrophic, mucosa lacking in oestrogen;
- antibodies that may be present in adolescents and adults are lacking;
- children may have poor local hygiene.^{38,39}

3.3. Recovery of *N gonorrhoeae* from inanimate objects (in vitro studies)

Gonococcus flourishes at 25°–39 °C. It is killed by heat at 55 °C for 5 min, dies quickly under natural environmental conditions and is vulnerable to drying. However gonococci have been recovered from pus on linen kept moist with sterile saline after 5 h and in one case after 22 h, although could not be recovered by culture after 2 h if the cloth was kept dry.³² Gonococcal pus placed on glass slides and on towel kept at room temperature has been shown to survive for up to 24 h on the towel and 17 h on the slide.⁴⁰ The medicolegal importance of the fact that ‘towels and linen soiled with gonorrhoeal pus may convey contagion so long as the pus has not dried up’ was noted by Marshall in 1914.²⁹ Leishman recorded in 1916 that Scholtz ‘has cultivated it from infected bathwater after 24 h’.⁴¹ The primary source of this experiment (Scholtz) was not accessed.

A series of in vitro experiments culturing gonococcus from contaminated pieces of cloth found that when conditions were kept warm and humid, the cloth would remain damp and the organism could be recovered after 2–3 h.⁴² These experiments indicate that in humid conditions, contaminated towels, bedding and underclothes may remain sufficiently damp to allow transmission of the infection.

Benson⁴³ describes a series of experiments conducted by Dobszay (Archiv fur Kinderheilkunde 1992;99:102). Dobszay took cultures at frequent intervals from various items contaminated either artificially or by letting infected children play with them. Gonococcus was able to be grown from rabbit skin after 54 h, wet linen after 24 h, dry linen after 1/2 h, rubber, water and wood after 2 h and metal after 10 min. Positive cultures were not able to be obtained in soapy water at 10 min, which suggests that washing with soap is likely to prevent spread of the infection.

Experiments were conducted to determine the survival periods of gonococci on a variety of materials that might be contaminated by an infected person.⁴⁴ A droplet of fresh

gonococcal pus was placed on a range of sterile test materials including hard substances (glass, plastic, cellophane, wood, cardboard and paper) and soft substances (cotton swab, cotton gauze, linen handkerchief, cotton towel, tissue paper and lubricated condom). At various intervals, the exudate deposited was scraped with a sterile disposable scalpel onto plates of culture medium which were incubated for 4 days and then tested for growth of *N gonorrhoeae*. Gonococci were recovered from most of the materials after 24–48 h, and from a few materials (cotton swab, white cardboard and wooden spatula) up to 72 h or more. The authors conclude ‘It is possible that gonorrhoea is transmitted non-venereally more often than is usually acknowledged, and these results may have medicolegal and social significance’.

An experiment involving 72 random swabs from public toilets did not grow any *N gonorrhoeae*.⁴⁵ Suspensions of *N gonorrhoeae* inoculated on toilet seats and toilet paper did not survive after the inoculants had dried (within 10 min). However survival of gonococci was markedly enhanced in purulent suspensions, from which organisms could be recovered from both toilet seats and toilet paper 2–3 h later. The authors conclude that ‘mere sitting on a contaminated seat is not sufficient’ to acquire the infection, however ‘contaminated toilet paper has greater potential as a direct source than do toilet seats’.

3.4. Intrauterine and perinatal (vertical) transmission of *N gonorrhoeae*

Gonococcal infection in the newborn can be acquired either in the uterus (following membrane rupture during labour) but more usually through contamination from the birth canal during delivery.^{46,47} Although the infection usually effects the eyes (gonococcal ophthalmia neonatorum),³⁴ vertical transmission can also result in genital, respiratory, skin or joint infection.^{48,49}

3.5. Accidental transmission of *N gonorrhoeae*

Several cases of accidental transmission of *N gonorrhoeae* are reported in the literature. While these are case reports, they are strong evidence because there is laboratory confirmation of the causative agent. Transmission of pharyngeal gonorrhoea in a child resulted from a laboratory technician leaving her 3 year old son in her car where he accessed and ingested some chocolate agar from a culture plate.⁵⁰ The agar remnants grew *N gonorrhoeae* and the gonococcal organism was cultured from a throat swab of the boy taken 8 days after the event (swabs taken at 1, 2, 4 and 6 days were negative).

A further accidental infection occurred when a laboratory technician, who was working with mice infected with a *N gonorrhoeae*, was struck on the right eye with the strap of his face mask.⁵¹ He developed severe gonococcal conjunctivitis in that eye within 24 h. In this case the

organism was transmitted to his eye by the strap of his face mask.

Another laboratory-acquired gonococcal conjunctivitis resulted from a laboratory technician accidentally spraying her face and left eye with fluid containing *N gonorrhoeae*.⁵² She immediately rinsed her eyes with tap water but developed a gonococcal infection in her left eyes 13 days later.

Gonococcal conjunctivitis is also reported in 13 Filipino patients who had the unusual cultural practice of using their own urine as an eyewash.⁵³ Seven underwent urethral smears and cultures, all of which were positive, and the cause of the eye infection was determined to be the infected urine.

3.6. Epidemics of non-sexually transmitted conjunctival gonorrhoea

In much of the sub-continent of Africa, there is lack of adequate facilities for proper diagnosis, treatment and epidemiological surveillance of gonorrhoea.⁵⁴ This allows for epidemics of non-sexually transmitted gonorrhoea to occur no longer seen in the western world. For example, in Ethiopia over 9000 cases of gonococcal conjunctivitis were reported during 8 months in 1987–88 in a single district.⁵⁵ This outbreak affected both sexes and all age groups, but particularly children under 5 years of age. The epidemic curve suggested horizontal transmission. Routine surveillance data showed that there was no concurrent genital gonorrhoea outbreak and genital transmission could not explain a community-wide outbreak.

Similar outbreaks have occurred in Aboriginal communities in rural Australia. An outbreak of 30 cases of gonococcal conjunctivitis in Aboriginal settlements in the Northern Territory in 1981 affected mostly children under the age of five.⁵⁶ A similar outbreak occurred at the isolated community of Warburton in Western Australia. In 1987, 140 cases of gonococcal conjunctivitis were seen in a relatively short time period in another Aboriginal community.⁵⁷ Cases ranged between 6 weeks and 63 years of age, but only eight cases were older than 15 years. Two to five year olds were most likely to be infected, with 35% of the children in this community affected. A smaller eruption occurred in two isolated Aboriginal communities in Western Australia in 1991.⁵⁸ Again the number of cases peaked in 5–9 age group, with no differences in gender of the children.

Another outbreak of non-sexually transmitted gonococcal conjunctivitis occurred in 1997 with 447 cases identified in Aboriginal communities in Central Australia and the Kimberley region.⁵⁹ This outbreak had similar associated environmental factors to previous ones and 77% of cases were under 10 years old.

A prospective study of an outbreak of 432 identified cases of gonococcal conjunctivitis in 1991 used a community survey to explore risk factors.⁶⁰ Children aged 0–4 years had the highest attack rate, and were 27 times

more likely to develop conjunctivitis than adults. There was no difference in attack rate by gender. Cases were more likely to have unwashed faces and/or hands than those without the disease. Surveillance data indicated no increase in anogenital gonorrhoea preceding or concurrent with this epidemic. The epidemic involved a community living in sub-standard housing and overcrowded conditions with insufficient water supply, poor sanitation and sewerage disposal and inadequate food hygiene. The epidemic was preceded by unseasonably heavy summer rains and humidity with a subsequent explosive increase in fly density. Similar conditions preceded the 1987 epidemic reported above.⁵⁷ This epidemic shows comparable clinical and epidemiological features to the Ethiopian one in 1991.⁵⁵ The role of the fly as a possible vector remains speculative but is a distinct possibility.⁶¹ In 1998, 13 confirmed cases of non-sexually transmitted gonococcal conjunctivitis were notified in the Northern Territory and Western Australia.⁶² The gonococcal conjunctivitis management protocol was instigated urgently in both locations and no further cases were identified.

These epidemics were detected by laboratory testing and then controlled using antibiotics. The epidemiological evidence presented includes case series, case controlled study and prospective study.

3.7. Epidemics of non-sexually transmitted gonorrhoea in children's institutions

Due to the development and availability of laboratory testing and antibiotics for treatment, gonorrhoea is now a relatively rare disease in the developed world. In the past, gonorrhoea was commonly endemic and gonococcal infection in children acquired both sexually and non-sexually from contaminated articles was a frequently reported occurrence in textbooks and peer-reviewed papers.^{27,43,44,63–65}

The gonococcus bacterium was discovered by Neisser in 1879 but finally confirmed as the cause of the venereal disease gonorrhoea after it was successfully cultured and humans infected experimentally by Bumm in 1884.²² Originally it was believed 'that sexual contact alone could be responsible for such infection' but the frequent 'occurrence of cases in institutions for little girls, where there was no possibility of venereal infection' slowly convinced the medical fraternity that *N gonorrhoea* was extremely contagious and could be both sexually and non-sexually transmitted.

Epidemics of vaginal gonococcal infection were a serious problem in children's wards and orphanages, usually through some failure in nursing care.⁶⁶ Thermometers, enema nozzles, examination gloves, nurses' aprons, towels and bedding have been implicated as agents of transmission.⁶⁷ A typical scenario was that an infected child would be admitted to the hospital or children's home. The original source of infection for this index case might have been sexual transmission. However within the institution the infection would then spread rapidly without sexual transmission to other children. Because there was no antibiotic

treatment this could lead to serious and sometimes fatal disease. The epidemics would often continue until strategies to isolate the infected children and identify the source of transmission (nurses' hands or fomites) were implemented. The pattern of spread was clearly due to contamination and not sexual abuse of the children in the institutions.

By 1917 *'the extreme contagiousness of gonococcus infection among girls under puberty was universally admitted'*.⁶⁸ Once an infected girl was admitted to an institution, *'only the strictest hygiene will prevent its rapid spread'*. This review identified approximately 2000 cases in gonococcal epidemics in over 40 hospital and children's homes reported in the medical literature. With the advent of antibiotics, gonorrhoea is no longer endemic and epidemics in institutions an event of the past. The knowledge of these non-sexual epidemics has largely been lost to modern literature.

These cases are listed in Table 1 and examples follow. In August 1890, 236 cases were reported in little girls in the city of Posen, traced to the sharing a public bath.^{20,22} In 1896, a number of epidemics of gonorrhoea in children's institutions are described.⁶⁹ The infection typically spread very rapidly and the younger the child, the greater the likelihood of contracting it. In some children's asylums it appeared that the infection was spread through bathing 20–30 girls together in a large bathtub. The epidemic stopped once a shower bath was constructed. Physicians were aware that the infection could be spread through baths, beds, clothing and towels. Sheffield warns institutions not to admit girls with vaginal discharge *'unless they can convince themselves that this is not of gonorrhoeal origin'*, because of the significant likelihood of spread through contamination. *'If a child with vaginitis associated with gonorrhoea is admitted unrecognized to an institution where personal hygiene is not perfect, in about 3 or 4 days almost all the little girls have the disease'*.³⁶

Five cases of vulvo-vaginitis were reported in the West End Nursery, Boston City Hospital, in 1893, possibly transmitted by sharing of infected towels and beds.⁷⁰ Nineteen cases of gonorrhoea (18 girls and 1 boy) were reported in the children's ward of Chicago hospital in 1 year between August 1902 and September 1903.⁷¹ Investigation indicated that the infection was introduced by an infected 2 year old boy and another infant with typhoid fever in August 1902. These two patients *'appear to have furnished the infection for this entire epidemic'*. The most likely vector was rectal thermometers in many cases.

In 1904 Baer reviewed 19 epidemics involving 696 cases of gonorrhoeal vulvovaginitis in young girls reported by 17 institutions.²¹ The first record dated from 1878. In many of these children's hospitals and orphanages the infection followed admission of an infected child. The disease spread rapidly but once the source of infection was identified and the affected children isolated, the outbreaks ceased and no new cases would occur. Epidemics would also abruptly stop once the mode of transmission was identified

and addressed. A number of institutions traced the mode of transmission to common baths. Other modes of transmission were attributed to towels, wash rags, diapers, bandages, bed linens, instruments and children's hands. Transmission by nurses' hands was also strongly implicated. Transmission was traced to the use of inadequately cleaned rectal thermometers in two institutions. *'Following appropriate methods of disinfection of this instrument, the epidemic promptly ceased'*.

In 1903 evidence considered conclusive proof that gonorrhoea could be transmitted from one child patient to another by nurses was reported in the Babies Hospital in New York.⁷² Despite thorough disinfecting of napkins or using cotton pads that were burnt once soiled; providing each child with a separate thermometer; keeping bottles and nipples separate; abolishing sponges, wash cloths and bathing in tubs, *'case after case developed as long as children remained in the ward with those who were infected'*. It was eventually identified that the infection was carried by nurses in the process of feeding, bathing and changing the napkins of the infants. The spread was brought under control by the use of rubber gloves by the nurse when required to care for *'gonococcus cases and others at the same time'*.

Also in 1903 Koplik detailed methods to prevent the spread of gonococcal vulvovaginitis in hospital services.⁷³ Such outbreaks were seen as *'the most annoying scourges'* of children's hospitals and prevention *'of greatest value and importance'* because once a child became infected she was *'exposed to the dangerous consequences of the disease and a menace to others'*. Using stringent isolation procedures, Koplik was able to eliminate the spread of this disease within his New York Hospital children's wards. Upon admission all children underwent examination for the presence of urethral, vaginal or rectal discharge and cultures taken of vaginal secretions. No child was bathed in the common tub until pronounced free of discharge. All patients with a suspicious discharge were placed under strict isolation. This involved issuing of individual combs, basins, bedpans and thermometers kept at the child's bedside. Bar soap was replaced by liquid soap. Diapers were made of cheesecloth and thrown away when soiled. The child's bed was surrounded by a red bandage to warn nurses and all linen marked VD (vaginal discharge). Soiled linen was sterilised in the hospital steriliser before being sent to the general laundry. Bedpans, catheters, nozzles and douche apparatus were marked with the patient's name and kept aside from similar utensils in the ward. The nurse attending the patient was not allowed to take the temperature or touch the genitals of any other patient in the ward. This system in safeguards resulted in the regular epidemics of gonococcal vulvovaginitis in the children's wards being completely eliminated.

In 1917, it was also reported of the frequent spread of gonorrhoea through contact with *'contaminated bedding, towels, sponges, toilet seats, bath tubs, the fingers of the nurse, rectal thermometers, underwear, etc'*.⁷⁴ Over 500

cases of gonorrhoeal vulvovaginitis were identified in 12 children's institutions in the 12 months of 1910. While contamination was identified as the most frequent means of communicating the disease, the author also recognised that girls could also become infected through sexual assault. Children infected in hospitals were much more likely to be infected if they were very young – for example of the 330 cases seen at the Children's Annex of the Cook County hospital, 73% were aged under 6 years.

In 1924, 67 babies in the same hospital ward contracted gonococcal infections within a single month.⁷⁵ There were 182 babies born in the hospital during this period and none cared for in a different ward developed the disease. Fifty-three of the infected babies developed gonococcal arthritis. Laboratory diagnoses were made by microscopic evidence of gonococci confirmed by bacteriologic cultures. The source of the infection was believed to be either a laundress or an infected woman giving birth, with gonococcal organisms introduced to the nursery by a baby with infected discharges on its body. It was then transmitted to other infants by the nurses via contaminated rectal thermometers, wash basins or laundry. There was no possibility in this context of the infection being transmitted by sexual abuse of the babies. Other cases have occurred where children have developed anogenital gonorrhoea having been in hospital since birth, or admitted to medical and surgical wards with negative vaginal swabs then subsequently developing positive cultures.³²

3.8. Non-sexual person-to-person and fomite transmission of *N gonorrhoeae*

While most cases of person to person transmission of *N gonorrhoeae* are likely to be sexually transmitted, the literature also reports non-sexual or likely non-sexual cases of person to person transmission, particularly related to over-crowded living conditions as well as fomite transmission by towels, bedding and other cloths. In some cases the transmission might either be by contaminated hands or fomites. In other cases it is not possible to establish whether the transmission was sexual or non-sexual.

Spread of gonococcal infection to young children has been thought to be transmitted by infected mothers or other caregivers with contaminated hands in a number of cases,^{76–79} and to older children through contaminated bedding, sharing of infected towels or underclothes,^{54,80–84} or non-sexual contact with infected family members^{32,85–92} or friends (child-to-child transmission).^{93–99}

A recent case is reported of an 8 year old pre-pubescent girl contracting gonorrhoea during 72 h in transit flying from Rome to Sydney via Moscow.¹⁰⁰ The toilets were dirty and the child always used a piece of toilet paper to wipe the seat before using it. The child is believed to have contracted the disease via auto-inoculation (that is, transmitting the infection via her finger) while using mixed toilets in a crowded plane. Another unusual case of indirect transmission is the report of a hospitalised soldier contract-

ing gonorrhoea from sharing a bed urinal bottle with an infected patient.¹⁹

4. Discussion

When *Neisseria gonorrhoea* was first identified in the 1880s it was believed to be strictly a sexually transmitted disease. However throughout the world it became recognised that once the infection was introduced into a children's hospital or other institution, it would rapidly spread among pre-pubertal girls. The medical literature records over 40 epidemics involving about 2000 children in Europe and the United States. Communal baths, towels and other cloth, rectal thermometers and caregivers hands were all identified as means of transmitting the infection, for which there was no adequate treatment prior to the discovery of penicillin. While it generally caused vulvovaginitis, to which young girls are particularly susceptible, it would also cause conjunctivitis and more serious complications including arthritis, and could be fatal on occasions.

Strong evidence of non-sexual transmission includes epidemics of conjunctivitis, accidental inoculations and evidence from in vitro studies of the ability to culture from fomites if *N gonorrhoea* solutions or pus are kept damp. In the cases of conjunctival epidemics in Africa and Australia in the 1980s and 1990s, the epidemics were detected by laboratory testing and then controlled using antibiotics. The epidemiological evidence presented includes case series, case controlled study and prospective study. Evidence from the conjunctival epidemics is important because it demonstrates that this infection can be readily transmitted in a manner that is not mucous membrane to mucous membrane.

The literature regarding possible non-sexual transmission through contact with infected household members and from towels, bedding and clothing, particularly likely to occur under conditions of over-crowding and poor hygiene, is weaker evidence. It may not be possible to determine whether the infection was spread through sexual abuse or through non-sexual means in household situations. However the bulk of this evidence suggests that this does occur on occasions, particularly when there is conjunctivitis because this is unlikely to be sexually transmitted and more likely to be through infected hands or fomites.

Put together, there is overwhelming evidence that *N gonorrhoeae* can be both sexually and non-sexually transmitted in pre-pubertal children, particularly girls. While international authorities and guidelines acknowledge the rare possibility of non-sexual transmission, their opinions clearly lack the evidence base revealed by this literature review. Fomite transmission may not be acknowledged.¹⁰ Sgroi suggests that the institutional epidemic described by Cooperman⁷⁵ of 67 babies acquiring *N gonorrhoeae* rectally and in their joints while staying in a crowded hospital ward were actually all cases of unrecognised sexual abuse.¹³

The validity of the childhood institutional epidemics may be questioned because of the age of the evidence or because it is believed that sexual abuse of children went unidentified in these days. However when *N gonorrhoeae* was first discovered in the 1880s it was considered to be exclusively sexually transmitted and early papers do recognise and discuss the importance of sexual transmission.

Control of an epidemic requires both effective surveillance and response. First the epidemic must be detected by some recorded increase in numbers of cases. Then an intervention needs to be implemented to prevent or to treat the cases, and ongoing monitoring must demonstrate that either no more cases are occurring, or in situations where a condition is endemic that the number of cases has dropped back down to the usual level. This is not new science. After John Snow was able to demonstrate that a London epidemic of cholera resulted from contaminated water from a particular public water pump in 1845, it was then understood that cholera is a water-borne disease and in general, education and sanitation are the limiting factors to prevent an epidemic.

Even studies that are over 100 years old often demonstrate considerable scientific rigour. There was microscopic and microbiological diagnosis of the organism. In the epidemics of gonococcal infection in children in the 19th and early 20th century, once the sudden rise in cases was detected, physicians looked for ways the infection might be being transmitted, took action and then recorded whether new cases still occurred. For example, there were cases traced to sharing of a common bath, and the epidemic stopped when this practice was discontinued. In the Babies Hospital in New York, the transmission was finally traced to nurses' hands, and cases ceased once the nurses wore rubber gloves.⁷²

Differentiation between sexual and non-sexual transmission of gonorrhoea in children is difficult. In the past the possibility of sexual abuse may have been overlooked in children infected with *N gonorrhoeae*. It is likely that some cases identified as transmission via fomite were actually sexually transmitted.

Conversely, current thinking is that gonorrhoea in children is definitive, or nearly always definitive, evidence of sexual abuse or contact. However there is conclusive evidence that gonorrhoea may be transmitted by non-sexual means. While all paediatric cases of gonorrhoea need to be taken seriously, including conducting relevant contact tracking and testing, forensic medical examiners should keep an open mind about possible means of transmission. They should consider the likelihood of sexual abuse on a case by case basis. Missing a case of sexual abuse has serious social and legal consequences. However removing a child from its parents on the basis of a wrongful assumption may have equally damaging sequelae. Doctors and lawyers need to be cognisant of the large body of literature demonstrating both sexual and non-sexual means of transmission of gonorrhoea in children.

References

1. Adams J. Approach to the interpretation of medical and laboratory findings in suspected child sexual abuse: a 2005 revision. *The APSAC Advisor*, 2005. p. 7–13.
2. Adams JA, Girardin B, Faugno D. Adolescent sexual assault: documentation of acute injuries using photo-colposcopy. *J Pediatr Adolesc Gynecol* 2001;**14**(4):175–80.
3. Adams JA, Harper K, Knudson S. A proposed system for the classification of anogenital findings in children with suspected sexual abuse. *Adoles Ped Gyn* 1992;**5**(2):73–5.
4. American Academy of Pediatrics. Gonorrhoea in prepubertal children. *Pediatrics* 101(1 Pt 1):134–5, 1983;**71**(4):553.
5. American Academy of Pediatrics Committee on Child Abuse and Neglect. Gonorrhoea in prepubertal children. *Pediatrics* 1998;**101**(1 Pt 1):134–5.
6. Anonymous. American Academy of Pediatrics Committee on Child Abuse and Neglect: Guidelines for the evaluation of sexual abuse of children. *Pediatrics* 1991;**87**(2):254–60.
7. Anonymous. Guidelines for the evaluation of sexual abuse of children: subject review. American Academy of Pediatrics Committee on Child Abuse and Neglect. *Pediatrics* 1999; **103**(1):186–91.
8. APSAC. Glossary of terms and the interpretations of findings for child sexual abuse evidentiary examinations Written by Interpretation of Physical Findings in Sexual Abuse subcommittee headed by John McCann. Chicago: American Professional Society on the Abuse of Children; 1998.
9. Centers for Disease Control and Prevention. Sexually transmitted diseases treatment guidelines 2006. 2006;**55**(RR-11):139.
10. Hammerschlag MR. Sexually transmitted diseases in sexually abused children: medical and legal implications. *Sex Transm Infect* 1998;**74**(3):167–74.
11. Kellogg N. Committee on Child Abuse and Neglect. Clinical Report: the evaluation of sexual abuse in children. *Pediatrics* 2005;**116**(2):506–12.
12. Royal College of Physicians. Physical Signs of sexual abuse in children. Report of a working party of the Royal College of Physicians of London, London, 1997.
13. Sgroi SM. Pediatric gonorrhoea and child sexual abuse: the venereal disease connection. *Sex Trans Dis* 1982;**9**(3):154–6.
14. Sgroi SM. Childhood gonorrhoea. *Med Aspects Hum Sexual* 1982;**16**:118–41.
15. Thomas A, Forster G, Robinson A, Rogstad K. Clinical Effectiveness Group. National guideline for the management of suspected sexually transmitted infections in children and young people. *Sex Transm Infect* 2002;**78**(5):324–31.
16. Thomas A, Forster G, Robinson A, Rogstad K. Clinical Effectiveness Group Association of Genitourinary M, Medical Society for the Study of Venereal D. National guideline for the management of suspected sexually transmitted infections in children and young people. *Arch Dis Child* 2003;**88**(4):303–11.
17. Shand C, Broadmore J, MacDonald J, Gellatly R, Hurst C. *The medical management of sexual abuse*. 5th ed. DSAC; 2002.
18. Kelly P. Childhood gonorrhoea in Auckland. *N Z Med J* 115 (1163):U201, 2002 Oct 11.
19. Neinstein LS, Goldenring J, Carpenter S. Nonsexual transmission of sexually transmitted diseases: an infrequent occurrence. *Pediatrics* 1984;**74**(1):67–76.
20. Abt I. Gonorrhoea in children. *JAMA* 1898;**31**(17):289–94.
21. Baer J. Epidemic gonorrhoeal vulvo-vaginitis in young girls. *J Infect Dis* 1904;**1**(19):313.
22. Hamilton A. Gonorrhoeal vulvovaginitis in children with special reference to an epidemic occurring in scarlet fever wards. *J Infect Dis* 1908;**5**:133–57.
23. MRC Working Party. Resistance of gonococci to penicillin : interim report by a Medical Research Council working party appointed to

- examine the resistance of gonococci to penicillin. *The Lancet* 1961;**278**(29):226–30.
24. Cruickshank R, Dugid J, Maramion B, Swain R. *Chapter 27: Neisseria. Medical Microbiology: the practice of medical microbiology*. 12th ed. Edinburgh: Churchill Livingstone; 1975. p. 399–402.
 25. Cruickshank R. *Mackie & McCartney's handbook of bacteriology*. 10th ed. Edinburgh: Livingstone; 1960.
 26. Rein MF. Nonsexual acquisition of genital gonococcal infection. *N Engl J Med* 1979;**301**(24):1347.
 27. Wilcox R. *Textbook of venereal diseases and treponematoses*. 2nd ed. London: William Heinemann Medical Books; 1964.
 28. Bigger J. *Handbook of bacteriology*. 6th ed. London: Bailliere Tindall & Cox; 1949.
 29. Marshall C. *Syphilology and venereal diseases*. 3rd ed. London: Bailliere Tindall & Cox; 1914.
 30. Cohen MS, Cannon JG. Human experimentation with *Neisseria gonorrhoeae*: progress and goals. *J Infect Dis* 1999;**179**(Suppl 2): S375–9.
 31. Cohen MS, Cannon JG, Jerse AE, Charniga LM, Isbey SF, Whicker LG. Human experimentation with *Neisseria gonorrhoeae*: rationale, methods, and implications for the biology of infection and vaccine development. *J Infect Dis* 1994;**169**(3):532–7.
 32. Cohn A, Steer A, Adler E. Gonococcal vaginitis: a preliminary report on one year's work. *Venereal Dis Inform* 1940;**21**:208–20.
 33. Felman YM, Nikitas JA. Gonococcal infections in infants and children. Medical and epidemiologic considerations. *New York State J Med* 1979;**79**(7):1063–5.
 34. Israel KS, Rissing KB, Brooks GF. Neonatal and childhood gonococcal infections. *Clin Obstet Gynecol* 1975;**18**(1):143–51.
 35. Whittington WL, Rice RJ, Biddle JW, Knapp JS. Incorrect identification of *Neisseria gonorrhoeae* from infants and children. *Pediatr Infect Dis J* 1988;**7**(1):3–10.
 36. Altchek A. Pediatric vulvovaginitis. *Pediatr Clin North Am* 1972;**19**(3):559–80.
 37. Altchek MA. Gonococcal vaginitis in children. *Med Aspects Hum Sexuality* 1982;**16**(1):46–51.
 38. Altchek A. Pediatric vulvovaginitis. *J Reprod Med* 1984;**29**(6): 359–75.
 39. Woods CR. Gonococcal infections in neonates and young children. *Semin Pediatr Infect Dis* 2005;**16**(4):258–70.
 40. Elmros T, Larsson PA. Survival of gonococci outside the body. *Br Med J* 1972;**2**(810):403–4.
 41. Leishman W, Keogh A, Melville C. *A manual of venereal diseases*. 2nd ed. London: Oxford University Press; 1916.
 42. Alausa K, Sogbetun A, Montefiore D. Effect of drying on *Neisseria gonorrhoeae* in relation to nonvenereal infection in children. *Nigerian J Pediatr* 1977;**4**(1):14–8.
 43. Benson R, Steer A. Vaginitis in children. *Am J Dis Child* 1937;**53**: 806–24.
 44. Srivastava AC. Survival of gonococci in urethral secretions with reference to the nonsexual transmission of gonococcal infection. *J Med Microbiol* 1980;**13**(4):593–6.
 45. Gilbaugh JH, Jr., Fuchs PC. The gonococcus and the toilet seat. *N Engl J Med* 1979;**301**(2):91–3.
 46. Treadwell P. Sexually transmitted diseases in neonates and infants. *Semin Dermatol* 1994;**13**(4):256–61.
 47. Fletcher JL Jr, Gordon RC. Perinatal transmission of bacterial sexually transmitted diseases. Part I: Syphilis and gonorrhea. *J Fam Pract* 1990;**30**(4):448–56.
 48. Babl FE, Ram S, Barnett ED, Rhein L, Carr E, Cooper ER. Neonatal gonococcal arthritis after negative prenatal screening and despite conjunctival prophylaxis. *Pediatr Infect Dis J* 2000;**19**(4):346–9.
 49. Desenclos JC, Garrity D, Scaggs M, Wroten JE. Gonococcal infection of the newborn in Florida, 1984–1989. *Sex Transm Dis* 1992;**19**(2):105–10.
 50. Lipsitt HJ, Parmet AJ. Nonsexual transmission of gonorrhea to a child. *N Engl J Med* 1984;**311**(7):470.
 51. Diena BB, Wallace R, Ashton FE, Johnson W, Platenaude B. Gonococcal conjunctivitis: accidental infection. *Can Med Assoc J* 1976;**115**(7):609.
 52. Bruins SC, Tight RR. Laboratory-acquired gonococcal conjunctivitis. *Jama* 1979;**241**(3):274.
 53. Valenton MJ, Abendano R. Gonorrhoeal conjunctivitis. Complication after ocular contamination with urine. *Can J Ophthalmol* 1973;**8**(3):421–7.
 54. Osoba A, Alausa K. Vulvovaginitis in Nigerian children. *Nigerian J Pediatr* 1974;**1**:26–32.
 55. Mikru FS, Molla T, Ersumo M, Henriksen TH, Klungseyr P, Hudson PJ, et al. Community-wide outbreak of *Neisseria gonorrhoeae* conjunctivitis in Konso district, North Omo administrative region. *Ethiop Med J* 1991;**29**(1):27–35.
 56. Matters R. Non-sexually transmitted gonococcal conjunctivitis in Central Australia. *Commun Dis Intell* 1981;**13**:3.
 57. Brennan R, Patel M, Hope A. Gonococcal conjunctivitis in Central Australia. *Med J Aust* 1989;**150**(1):48–9.
 58. van Buynder P, Bailey S, Adams J, Talbot J, Sullivan H, Waddingham A, et al. A cluster of non-sexually transmitted gonococcal conjunctivitis in the Pilbara, Western Australia. *Western Aust Notifiable Dis Bull* 1992;**2**(6):534–6.
 59. Matters R, Wong I, Mak D. An outbreak of non-sexually transmitted gonococcal conjunctivitis in Central Australia and the Kimberley region. *Commun Dis Intell* 1998;**22**(4):52–6. discussion 57–8.
 60. Merianos A, Condon RJ, Tapsall JW, Jayathissa S, Mulvey G, Lane JM, et al. Epidemic gonococcal conjunctivitis in central Australia. *Med J Aust* 1995;**162**(4):178–81.
 61. Weinstein P. The Australian bushfly (*Musca vetustissima* Walker) as a vector of *Neisseria gonorrhoeae* conjunctivitis. *Med J Aust* 1991;**155**(10):717.
 62. Anonymous. Gonococcal conjunctivitis outbreak. *Commun Dis Intell* 1998;**22**(3):39.
 63. Catterall R. *A short textbook of venereology*. 2nd ed. London: English Universities Press; 1975.
 64. Mackie T, Cruickshank R. *Mackie & McCartney's handbook of bacteriology*. 10th ed. Edinburgh: Livingstone; 1976.
 65. Schofield C. *Sexually transmitted diseases*. 2nd ed. Edinburgh: Churchill Livingstone; 1975.
 66. King A, Nicol C. *Veneral diseases*. 3rd ed. London: Bailliere; 1975.
 67. Grimble A. *McLachlan's handbook of diagnosis and treatment of venereal diseases*. 5th ed. Edinburgh: Livingstone; 1969.
 68. Gittings C, Mitchell G. Review of the literature of the past five years on gonococcus vulvovaginitis in childhood. *Am J Dis Children* 1917;**13**:438–56.
 69. Sheffield H. Contribution to the study of infectious vulvo-vaginitis in children, with remarks upon purulent ophthalmia, and a report of sixty-five cases. *Am Medico-Surgical Bull* 1896;**9**(30):726–31.
 70. Morse J. Five cases of gonorrhoeae in little girls. *Arch Pediatr* 1894;**11**:596–8.
 71. Cotton A. An epidemic of vulvovaginitis among children. *Arch Pediatr Adolesc Med* 1905;**22**:352–5.
 72. Holt L. Gonococcus infections in children, with especial reference to their prevalence in institutions and means of prevention. *New York Med J Philadelphia Med J* 1905;**81**:589–93.
 73. Koplik H. Prophylactic measures to prevent the spread of vulvovaginitis in hospital services. *Arch Pediatr* 1903;**10**:735–41.
 74. Seippel C. Venereal diseases in children. *Illinois Med J* 1912;**22**:50–6.
 75. Cooperman M. Gonococcus arthritis in infancy. *Am J Dis Child* 1927;**33**:932.
 76. Shore WB, Winkelstein JA. Nonvenereal transmission of gonococcal infections to children. *J Pediatr* 1971;**79**(4):661–3.
 77. Lowy G. Sexually transmitted diseases in children. *Pediatr Dermatol* 1992;**9**(4):329–34.
 78. Felman YM, William DC, Corsaro MC. Gonococcal infections in children 14 years and younger. Epidemiologic and other lessons drawn from a survey of 30 instances. *Clin Pediatr* 1978;**17**(3):252–4.

79. Reynolds V, Oates JK, Newsom SW. Prepubertal gonococcal vulvovaginitis: a penicillin-resistant infection treated with cefotaxime. *The Lancet* 1979; 28;2(8135):206–7.
80. Lewis LS, Glauser TA, Joffe MD. Gonococcal conjunctivitis in prepubertal children. *Am J Dis Child* 1990;144(5):546–8.
81. Doyle JO. Accidental gonococcal infection of the eyes in children. *Br Med J* 1972;1(792):88.
82. Auman GL, Waldenberg LM. Gonococcal periappendicitis and salpingitis in a prepubertal girl. *Pediatrics* 1976;58(2):287–8.
83. Ismail R, Toh CK, Ngeow YF. Gonococcal vulvovaginitis among female children in Malaysia. *Sex Transm Dis* 1985;12(3):114–6.
84. Frewen TC, Bannatyne RM. Gonococcal vulvovaginitis in prepubertal girls. *Clin Pediatr* 1979;18(8):491–3.
85. Allue X, Rubio T, Riley HD Jr. Gonococcal infections in infants and children. Lessons from fifteen cases. *Clin Pediatr (Phila)* 1973;12(10):584–8.
86. Folland DS, Burke RE, Hinman AR, Schaffner W. Gonorrhoea in preadolescent children: an inquiry into source of infection and mode of transmission. *Pediatrics* 1977;60(2):153–6.
87. Burry VF, Thurn AN. Gonococcal infections in prepubertal children. *Mol Med* 1971;68(9):691–2.
88. Tunnessen WW Jr, Jastremski M. Prepubescent gonococcal vulvovaginitis. *Clin Pediatr (Phila)* 1974;13(8):675–6.
89. Ingram DL, White ST, Durfee MF, Pearson AW. Sexual contact in children with gonorrhoea. *Am J Dis Children* 1982;136(11):994–6.
90. Dada-Adegbola HO, Oni AA. Review of cases of children with gonorrhoea—source of infection. *Afr J Med Med Sci* 2001;30(4):347–51.
91. Meek JM, Askari A, Belman AB. Prepubertal gonorrhoea. *J Urol* 1979;122(4):532–4.
92. Doyle JO. Accidental gonococcal infection of a child's eye. Unusual source of infection. *Br J Vener Dis* 1974;50(4):315–6.
93. Low RC, Cho CT, Dudding BA. Gonococcal infections in young children. Studies on the social, familial, and clinical aspects of 11 instances. *Clin Pediatr* 1977;16(7):623–6.
94. Emele FE, Anyiwo CE. Prevalence and horizontal propagation of gonococcal infections among Nigerian children. *Acta Paediatr* 1998;87(12):1295–6.
95. Potterat JJ, Markewich GS, King RD, Merceyck LR. Child-to-child transmission of gonorrhoea: report of asymptomatic genital infection in a boy. *Pediatrics* 1986;78(4):711–2.
96. Odugbemi T, Onile BA. Pediatric gonorrhoea: is it receiving adequate attention? *Am J Reprod Immunol Microbiol* 1988;18(1):32–4.
97. Ingram DL, Everett VD, Flick LA, Russell TA, White-Sims ST. Vaginal gonococcal cultures in sexual abuse evaluations: evaluation of selective criteria for preteenaged girls. *Pediatrics* 1997;99(6):E8.
98. Alexander WJ, Griffith H, Housch JG, Holmes JR. Infections in sexual contacts and associates of children with gonorrhoea. *Sex Transm Dis* 1984;11(3):156–8.
99. Nair P, Glazer-Semmel E, Gould C, Ruff E. Neisseria gonorrhoeae in asymptomatic prepubertal household contacts of children with gonococcal infection. *Clin Pediatr (Phila)* 1986;25(3):160–3.
100. Dayan L. Transmission of Neisseria gonorrhoeae from a toilet seat. *Sex Transm Infect* 2004;80(4):327.
101. Monger K, Brennan R. Gonococcal conjunctivitis outbreak in a Northern Territory Aboriginal community. *Northern Territory Commun Dis Bull* 1992;1(5):5.
102. Dobszay L. Klinische Beiträge zur Kenntnis der kindlichen gonorrhoe. *Archiv Kinderheilkunde* 1933;99(102):37–46.
103. Czeri. *Wien Med Wochenschr* 1885;35:352.
104. Yordan EE, Yordan RA. The hymen and tanner staging of the breast. *Adolescent Pediatr Gynecol* 1992;5(2):76–9.
105. Lennander. *Hygiea* 1885;47:505.
106. Leszynski. Leukorrhoea as the cause of a recent epidemic of purulent ophthalmia in one of our city charitable institutions. *New York Med J* 1886;43:352.
107. Dusch. *Deutsche Med Wehnschr* 1888;14:831.
108. Succhard. *Rev mens des mal de l'enfant*. 1888;6:265.
109. Skutsch. Vulvo-vaginitis gonorrhoeica bei kleinen Mädchen. *Jena*. 1891.
110. Fischer. *Deutsche Med Wehnschr* 1895;21:861.
111. Weill, Barjon. *Archive Med Exp D'anat Path* 1895;7:418.
112. Cnopf. *Munch Med Wehnschr* 1898;45:1141.
113. Skiba-Zaborowska M. Eine Epidemie von vulvo vaginitis kleiner Mädchen. *Zurich*; 1898.
114. Kimball. Gonorrhoea in infants, with a report of eight cases of pyremia. *New York Med Record* 1903;64:761.
115. Welt-Kakels S. Vulvovaginitis in little girls: a clinical study of 190 cases, part 1. 1904;80(8 Oct):693–8.
116. Alausa KO, Osoba AO. Epidemiology of gonococcal vulvovaginitis among children in the tropics. *Br J Vener Dis* 1980;56(4):239–42.