



THEME

Pelvic pain



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Pelvic inflammatory disease

BACKGROUND

Pelvic inflammatory disease (PID) is a treatable condition with serious long term sequelae. The recognition and diagnosis of PID can be challenging due to the wide spectrum of disease and clinical presentation.

OBJECTIVE

This article discusses the presentation, aetiology, diagnosis, management, and prevention of PID.

DISCUSSION

A high index of suspicion and a low threshold for treatment for women at risk of PID may help decrease the burden of serious associated morbidity. Screening for chlamydia in young sexually active women may reduce the incidence of PID.

Pelvic inflammatory disease (PID) is defined as inflammation of the upper genital tract including the endometrium, fallopian tubes and/or contiguous structures that follows infection from micro-organisms that ascend from the cervix and/or vagina.¹

Clinical presentation

The clinical presentation of PID may vary from severe, debilitating abdominal pain associated with tubo-ovarian abscess requiring hospitalisation to subclinical, asymptomatic infection for which the patient may not seek health care. It has been estimated that a clinical diagnosis of PID is incorrect in 33% of cases and of all true cases, up to 60% will be subclinical, 36% mild to moderate, and 4% severe.¹

No individual sign or symptom is pathognomonic of PID and correct diagnosis may rest upon clinician experience. One Australian study found the most common symptoms were abnormal vaginal discharge, lower abdominal pain and dyspareunia with adnexal tenderness, cervical motion tenderness and cervicitis most frequently seen on examination.² For those with more severe disease, systemic symptoms such as fever, nausea and vomiting may also be present.

Epidemiology

Estimation of the true incidence of PID is difficult as subclinical disease is not always identified and the diagnosis

may be missed. Pelvic inflammatory disease identified and managed appropriately in the community is not a notifiable disease although some cases, where an organism such as gonorrhoea is identified, are notifiable.

Estimates of acute PID based upon complications associated with hospital visits have been declining over recent years.^{3,4} In New South Wales this decline is in direct contrast to reports of increasing chlamydial infection. This may represent improved and earlier treatment or increased testing of chlamydia.³ In industrialised nations, the annual incidence of PID peaks in women aged 20–24 years.⁵ In the United Kingdom, PID accounts for 1.7% of general practice attendances of women aged 16–46 years.⁶

Pathogenesis

Sexually transmitted infections (STIs) such as gonorrhoea and chlamydia account for one-third to one-half of PID infections.^{1,7} *Mycoplasma genitalium* has been associated with nonspecific urethritis in men⁸ and PID in women.⁹ There are no commercially available tests for this organism at present.

The role of bacterial vaginosis (BV) organisms in PID continues to be debated. Anaerobic bacteria found in BV including anaerobic gram negative rods, diphtheroides, black pigmented gram negative rods and anaerobic gram positive cocci have been shown to be associated with endometritis and PID.^{10,11}

The role of *Mycoplasma hominis* and ureaplasma urealyticum is less clear. In rare cases, *Haemophilus influenzae*, *Escherichia coli* and streptococci have been isolated from the tubes in salpingitis.¹ As isolates in PID are often polymicrobial, treatment protocols are formulated to reflect common microbiological aetiology.

Nonsexually acquired PID may result from procedures such as intrauterine device (IUD) insertion, dilatation and curettage, and operative termination of pregnancy (TOP) that breach the protective cervical barrier and introduce bacteria from the vagina or cervix directly into the endometrial cavity.¹²

Risk factors

Most risk factors associated with PID are closely associated with those of acquisition of other STIs, with the most important risk factor a past history of either chlamydia and gonorrhoea infection or PID.¹⁴⁻¹⁷ Risk factors include young age, high frequency of partner change, lack of barrier contraception, low socioeconomic group, and smoking.^{6,14,16,17} One study found young women aged 15-19 years were over five times more likely than controls to suffer from PID.¹⁶

Risks associated with IUD insertion have been shown to be limited to the 4 weeks following insertion in women at low risk of STIs.¹³ Bacterial vaginosis can significantly increase the risk of PID following operative TOP and cervical chlamydia can be transported into the uterine cavity.¹² Prior screening for BV and antibiotic treatment can significantly reduced the rate of PID following TOP.

Hormonal contraception such as the oral contraceptive pill has been shown to reduce the severity of PID and may mask clinical recognition of PID.¹⁸ Sex during menstruation or just afterward, following the loss of the cervical plug, may also be a risk factor for PID.¹⁵ Genetic factors may also play a part in the development of infertility following PID.¹⁹

Diagnosis

No symptom or sign is pathognomonic of PID and all symptoms and signs have positive and negative predictive values. The 'gold standard' for diagnosis is laparoscopy, however this is invasive, costly and impractical in addition to underestimating rates of mild disease that present without overt tubal hyperaemia, oedema and exudate.

Strict inclusion of too many clinical criteria for a definitive diagnosis of PID may miss a significant numbers of infections. In a recent study, adnexal tenderness alone was found to be a sensitive marker of endometritis (96%), however the associated specificity was only 4%, suggesting high levels of overtreatment.²⁰ Combining cervical motion tenderness and lower abdominal pain increased the specificity to 20% but decreased the sensitivity to 80%. Newer techniques such as

Table 1. Common signs and symptoms associated with PID

Symptoms	Signs
<ul style="list-style-type: none"> • Lower abdominal pain/discomfort 	<ul style="list-style-type: none"> • Lower abdominal tenderness/guarding/rebound
<ul style="list-style-type: none"> • Vaginal discharge 	<ul style="list-style-type: none"> • Adnexal tenderness or a mass
<ul style="list-style-type: none"> • Abnormal vaginal bleeding 	<ul style="list-style-type: none"> • Cervical motion tenderness
<ul style="list-style-type: none"> • Dyspareunia 	<ul style="list-style-type: none"> • Raised temperature

Doppler ultrasound and magnetic resonance imaging (MRI) may prove useful in the diagnosis of PID in the future.²¹

Common signs and symptoms of PID are shown in *Table 1*. The Centre for Disease Control guidelines suggest treatment for PID when uterine/adnexal tenderness or cervical motion tenderness is present in sexually active young women at risk of STIs where no other cause is identified.²² Empirical treatment for possible PID is unlikely to impair the diagnosis and management of other causes of lower abdominal pain.

Due to the wide spectrum of clinical presentation including subclinical infection, the diagnosis of PID is imprecise. An accurate sexual history as well as a bimanual pelvic examination and relevant investigations should be undertaken (*Figure 1*).

Investigations

There are no specific laboratory tests to diagnose PID. In most cases empirical treatment should be commenced immediately on presumptive diagnosis, while results are pending (*Table 2*). Tests to exclude bacterial STIs should be conducted, including self collected vaginal or urine/cervical swabs for chlamydia polymerase chain reaction (PCR), bacterial cervical culture for gonorrhoea and other organisms, in addition to a high vaginal gram stain from vaginal swab to exclude BV. Onsite wet prep or the laboratory report of vaginal secretions will usually reveal polymorphs. Nonspecific serum markers associated with PID are: erythrocyte sedimentation rate >15 mm/hr, elevated C-reactive protein, and elevated white blood cell count.^{1,24}

Differential diagnosis

The differential diagnosis of lower abdominal and pelvic pain includes endometriosis, ruptured ovarian cyst, dysmenorrhoea and ectopic pregnancy. A pregnancy test should be considered in suspected cases of PID. Other causes of lower abdominal pain include appendicitis, irritable bowel syndrome, urinary tract infection, and gastroenteritis. Careful history and physical examination should exclude most alternative diagnoses. Pelvic ultrasound can aid in excluding other pathology if symptoms do not resolve with antibiotic treatment.

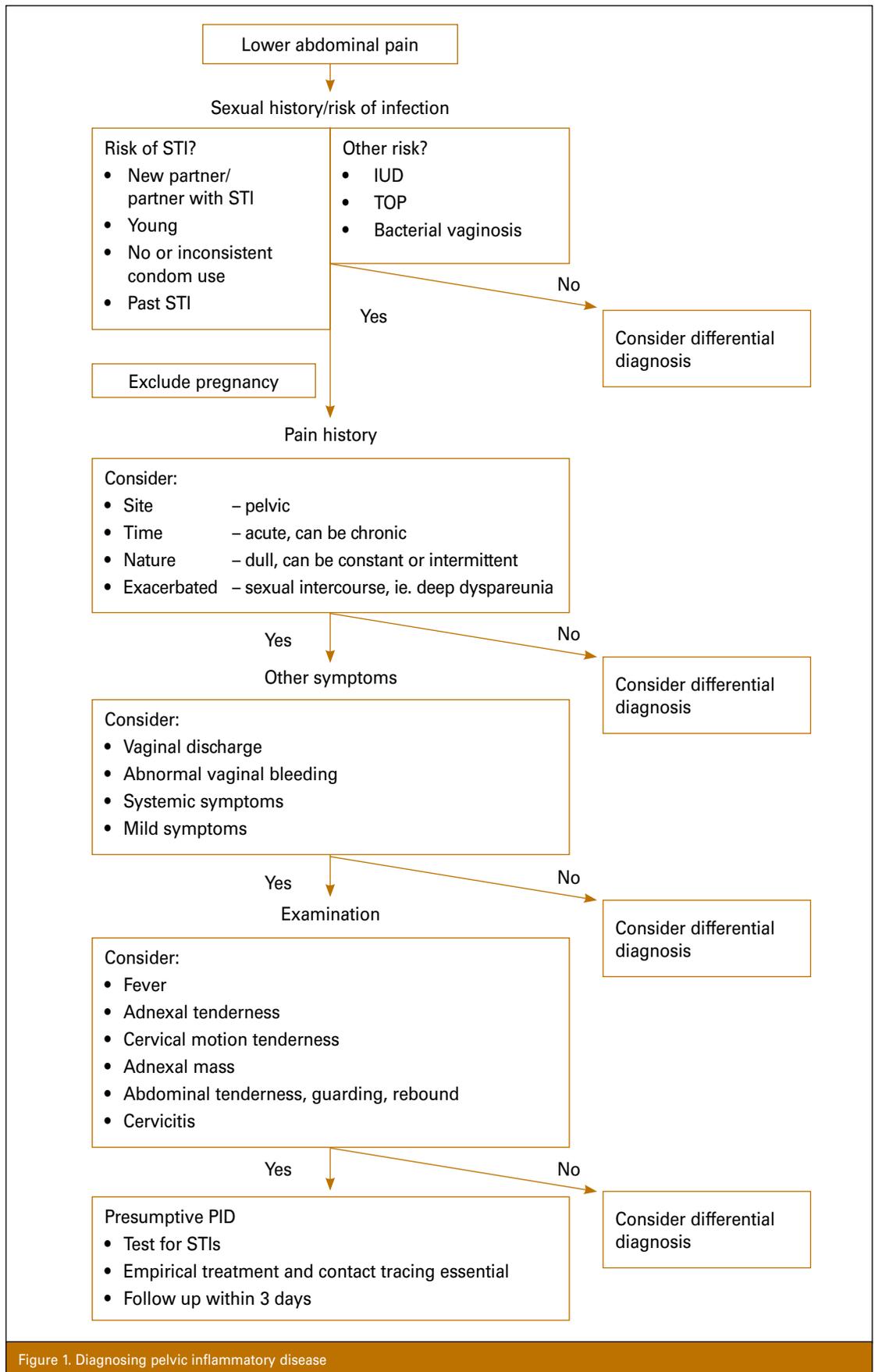


Figure 1. Diagnosing pelvic inflammatory disease

Management and treatment

Antibiotic guidelines for PID treatment provide cover for all common aetiological organisms including both sexually transmitted and facultative organisms. Recommended treatment regimens for outpatient management in Australia are shown in *Table 3*.²³ While gonococcal cover may not be routinely offered due to low rates of gonorrhoea in urban areas of Australia, a recent study from the United Kingdom showed significantly higher rates of clinical cure if ceftriaxone 250 mg intramuscularly was added in all cases of suspected PID.²⁵

The severity of PID determines whether patients are treated as out or inpatients (*Table 4*). There is little difference in long term sequelae such as pregnancy rate, recurrence of PID, chronic pelvic pain and ectopic pregnancy with either out or inpatient treatment for mild to moderate disease.⁷

Following initiation of outpatient treatment close follow up should be available. Duration of treatment depends upon clinical response with a minimum duration of 14 days. Occasionally longer courses may be required.

Complications and sequelae

Pelvic inflammatory disease has significant long term morbidity including tubal factor infertility (20%), chronic pelvic pain (20%) and ectopic pregnancy (10%).²⁶ With each repeated episode of PID the risk of permanent tubal damage and infertility increases 4–6 fold;²⁷ from 8% after one episode, to nearly 20% after two episodes, and 40% after three or more episodes.²⁸ The risk of ectopic pregnancy has been shown to increase 7–10 fold after PID.²⁹ Prompt diagnosis and treatment of women suffering PID may prevent serious sequelae such as infertility and chronic pelvic pain.³⁰

Contact tracing and prevention of PID

Prevention of re-exposure is an integral part of management and treatment of PID. It is not uncommon for partners of women with gonorrhoeal or chlamydial PID to be asymptomatic. Contact tracing and epidemiological treatment of sexual partners is an essential part of PID management.

In one large USA based randomised control community trial, women at increased risk of chlamydia were identified, tested and treated.³¹ This strategy was shown to reduce the risk of PID by up to two-thirds. Medical practitioners should have low thresholds for chlamydia testing young sexually active men and women. The 6th edition of The Royal Australian College of General Practitioners *Guidelines for preventive activities in general practice* ('red book') recommends opportunistic chlamydia screening for all sexually active women under 25 years of age and re-

Table 2. Useful investigations in the management of PID

Microbiological tests for possible specific bacterial aetiology

- Chlamydia trachomatis PCR (vaginal self collected/first catch urine/ endocervical)
- Neisseria gonorrhoea culture (endocervical) or gonorrhoea PCR (vaginal/ cervical/urine) (beware false positives)
- Endocervical swab microscopy/sensitivity/culture
- High vaginal swab and wet mount or gram stain
- Bacterial vaginosis: vaginal pH and 'whiff' test

Tests to assess severity

- Full blood count
- ESR or C-reactive protein

Tests to exclude other causes of pelvic pain

- Pregnancy test
- Mid stream urine
- +/- pelvic ultrasound

Table 3. Outpatient treatment for PID

In young sexually active women with no predisposing factors

azithromycin 1 g orally stat
+
doxycycline 100 mg twice per day for 14 days
+
metronidazole 400 mg twice per day for 14 days
+ (if gonorrhoea is suspected or proven)
ceftriaxone 250 mg IM stat, or ciprofloxacin 500 mg orally stat (depending on local patterns of *Neisseria gonorrhoea* resistance). (Consider adding ceftriaxone in all cases of PID)

Postprocedural PID (including IUD insertion, operative TOP)

doxycycline 100 mg twice per day for 2–4 weeks
Or
amoxicillin 500 mg three times per day for 2–4 weeks
+
metronidazole 400 mg three times per day for 2–4 weeks

screening every 12 months for women considered at high risk, including those with inconsistent condom use or with recent partner change.³²

Conclusion

Pelvic inflammatory disease is an important consequence of both STIs and medical procedures that breach the cervical barrier. Due to the wide clinical spectrum of presentation and difficulty in establishing diagnostic criteria, a high index of suspicion and low threshold for treatment is required. With close monitoring, outpatient treatment is effective and adequate. Chlamydia screening as a public health intervention in all young sexually active women reduces the risk of PID and its sequelae.

Table 4. Hospital admission and PID**Consider hospitalisation where:**

- tubo-ovarian abscess is suspected
- the patient is pregnant
- nonresponsive to oral antibiotic treatment
- unable to tolerate or follow outpatient oral regimen
- suffering severe illness (eg. nausea, vomiting or febrile)
- surgical emergencies such as appendicitis cannot be excluded

Summary of important points

- There is no single diagnostic symptom, sign or investigation for PID.
- Have a low threshold for diagnosing and treating presumptive PID.
- Empiric treatment for presumptive PID is unlikely to impact on diagnosis and management of other causes of pelvic pain.
- Consider adding ceftriaxone intramuscularly to the recommended therapy.
- Screen all sexually active women under 25–30 years for chlamydia. Repeat chlamydia screening at least yearly if there has been a change of sexual partner and/or inconsistent condom use.

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References

1. Weström L, Eschenbach D. Pelvic Inflammatory disease. In: Holmes KK, Sparling PE, Mårdh P, et al, editors. Sexually transmitted diseases. 3rd ed. New York: McGraw Hill 1999;783–809.
2. Marks C, Tideman RL, Estcourt CS, et al. Diagnosing PID: getting the balance right. *Int J STD AIDS* 2000;11:545–47.
3. Chen MY, Fairley CK, Donovan B. Discordance between trends in chlamydia notifications and hospital admission rates for chlamydia related diseases in New South Wales, Australia. *Sex Transm Infect* 2005;81:318–22.
4. Sutton My, Stenberg M, Zaidi A, StLouis ME, Markowitz LE. Trends in pelvic inflammatory disease hospital discharges and ambulatory visits, United States, 1985–2001. *Sex Transm Dis* 2005;32:778–84.
5. Westrom L. Incidence, prevalence, and trends of acute pelvic inflammatory disease and its consequences in industrialised countries. *Am J Obstet Gynecol* 1980;138:880–92.
6. Simms I, Rogers P, Charlett A. The rate of diagnosis and demography of pelvic inflammatory disease in general practice England and Wales. *Int J STD AIDS* 1999;10:448–51.
7. Ness RB, Soper DE, Holley RL, et al. Effectiveness of inpatient and outpatient treatment strategies for women with pelvic inflammatory disease: Results from the Pelvic Inflammatory Disease Evaluation and Clinical Health (PEACH) randomised trial. *Am J Obstet Gynecol* 2002;186:929–37.
8. Totten PA, Schwartz MA, Sjöstrom KE, et al. Association of Mycoplasma genitalium with nongonococcal urethritis in heterosexual men. *J Infect Dis* 2001;183:269–76.
9. Simms I, Eastick K, Mallinson H, et al. Associations between Mycoplasma genitalium, Chlamydia trachomatis and pelvic inflammatory disease. *J Clin Pathol* 2003;56:616–8.
10. Ness RB, Kip KE, Hillier SL, et al. A cluster analysis of bacterial vaginosis-associated microflora and pelvic inflammatory disease. *Am J Epidemiol* 2005;165:585–90.

11. Haggerty CL, Hillier SL, Bass DC, Ness RB. PID Evaluation and Clinical Health study investigators. Bacterial vaginosis and anaerobic bacteria are associated with endometritis. *Clin Infect Dis* 2004;39:990–5.
12. Stevenson MM, Radcliffe KW. Preventing pelvic infection after abortion. *Int J STD AIDS* 1995;6:305–12.
13. Burkman RT. Intrauterine devices and pelvic inflammatory disease: evolving perspectives on the data. *Obstet Gynaecol Surv* 1996;51:35–41.
14. Schofield MJ, Minichiello V, Mishra GD, Plummer D, Savage J. Sexually transmitted infections and use of sexual health services among young Australian women: Women's Health Australian Study. *Int J STD AIDS* 2000;11:313–23.
15. Ness RB, Smith KJ, Chang CH, Schisterman EF, Bass DC. Prediction of pelvic inflammatory disease among young single sexually active women. *Sex Transm Dis* 2006;33:137–42.
16. Simms I, Stephenson JM. Pelvic inflammatory disease: what do we know and what do we need to know? *Sex Transm Infect* 2000;76:80–7.
17. Marks C, Tideman RL, Estcourt CS, Berry G, Mindel A. Assessment of risk for pelvic inflammatory disease in an urban sexual health population. *Sex Transm Infect* 2000;76:470–3.
18. Ness RB, Soper DE, Holley RL, et al. Hormonal and barrier contraception and risk of upper genital tract disease in the pelvic inflammatory disease evaluation and clinical health (PEACH) study. *Am J Obstet Gynecol* 2001;185:121–7.
19. Ness RB, Brunham RC, Shen C, et al. Associations among human leukocyte antigen (HLA) class II DQ variants, bacterial STIs, endometritis and fertility among women with clinical pelvic inflammatory disease. *Sex Transm Dis* 2004;31:301–4.
20. Peipert JE, Ness R, Blume J, et al. Clinical predictors of endometritis in women with symptoms and signs of pelvic inflammatory disease. *Am J Obstet Gynecol* 2001;184:856–64.
21. Barrett S, Taylor C. A review on pelvic inflammatory disease. *Int J STD AIDS* 2005;16:715–21.
22. Centre for Disease Control. Sexually transmitted disease treatment guidelines 2002. *MMWR* 51 (RR-6):48–52.
23. Venereology Society of Victoria, Australasian College of Sexual Health Physicians. National management guidelines for sexually transmitted infections. Revised and updated April 2002.
24. Hagdu A, Westrom L, Brooks CA, Reynolds GH, Thompson SE. Predicting acute pelvic inflammatory disease: A multivariate analysis. *Am J Obstet Gynecol* 1986;155:954–60.
25. Piyadigamage A, Wilson J. Improvement in the clinical cure rate of outpatient management of pelvic inflammatory disease following a change in therapy. *Sex Transm Infect* 2005;81:233–5.
26. Ross J. Pelvic inflammatory disease. *BMJ* 2001;322:658–9.
27. Hillis SD, Owens LM, Marchbanks PA, Amsterdam LF, MacKenzie WR. Recurrent chlamydial infections increase the hospitalisation for ectopic pregnancy and pelvic inflammatory disease. *Am J Obstet Gynecol* 1997;176:103–7.
28. Westrom L, Joesoef R, Reynolds G, Hagdu A, Thompson SE. Pelvic inflammatory disease and fertility: a cohort study of 1844 women with laparoscopic verified disease and 657 control women with normal laparoscopic results. *Sex Transm Dis* 1992;19:185–92.
29. Westrom L. Pelvic inflammatory disease: bacteriology and sequelae. *Contraception* 1987;36:111–28.
30. Hillis S, Joesoef R, Marchbanks P, et al. Delayed care of pelvic inflammatory disease as a risk factor for impaired fertility. *Am J Obstet Gynecol* 1993;86:321–5.
31. Scholes D, Stergachis A, Heidrich FE, Andrilla H, Holmes KK, Stamm W. prevention of pelvic inflammatory disease by screening for cervical chlamydial infection. *N Engl J Med* 1996;334:1362–6.
32. The Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice. 6th ed. South Melbourne: The RACGP, 2005.