The Consultative Council on Obstetric and
Paediatric Mortality and Morbidity (CCOPMM)

# CONGENITAL ANOMALIES IN VICTORIA 2007–2009

# CONGENITAL ANOMALIES IN VICTORIA 2007–2009

To receive this publication in an accessible format phone +61 03 9096 2729, using the National Relay Service 13 36 77 if required, or email clinical.councils@dhhs.vic.gov.au

Authorised and published by the Victorian Government, 1 Treasury Place, Melbourne.



ISSN 2207-2764 (pdf/online)

Where the term 'Aboriginal' is used it refers to both Aboriginal and Torres Strait Islander people. Indigenous is retained when it is part of the title of a report, program or quotation.

Available at https://www2.health.vic.gov.au/hospitals-and-health-services/quality-safety-service/consultative-councils/council-obstetric-paediatric-mortality/congenital-anomalies-register

(1701001)

Recommended citation:

Victorian Congenital Anomalies Register. Congenital anomalies in Victoria 2007–2009. Melbourne: Consultative Council on Obstetric and Paediatric Mortality and Morbidity (CCOPMM), 2017

## Acknowledgements

This report would not have been possible without the particular assistance of:

- midwives in Victoria who provided notification of congenital anomalies on the birth report form or the hospital's electronic system to the Victorian Perinatal Data Collection (VPDC)
- maternal and child health nurses who completed and returned the Birth Defect Notification forms health information managers at all hospitals with maternity services and, in particular, those at hospitals with paediatric services
- The Royal Children's Hospital – Health Information Management, Information Technology, Cardiology Unit, Orthopaedic Unit
- The Royal Women's Hospital – Health Information Management, Information Technology, Fetal Diagnostic Unit, Ultrasound Department
- Monash Medical Centre – Health Information Management, Ultrasound Department and Genetics Clinic, Information Technology
- Genetic Health Services Victoria, Murdoch Childrens Research Institute
- Melbourne Pathology and Cytogenetic Services Victoria.

We thank all of the above for their contributions.

# Contents

# List of tables

# List of figures

# 1. Executive summary

The prevention and management of congenital abnormalities in the community relies on population level surveillance and research into the aetiology, diagnosis and prevention of such conditions. Congenital abnormalities are an important cause of child death and disability and are a leading cause of perinatal mortality in Australia[1], affecting 3.1 per cent of all births[2].

Though the causes of congenital anomalies are unknown in half of all the cases, the factors that contribute to congenital anomalies include genetic factors, socio-demographic factors (such as ethnicity, maternal age, and socioeconomic status), environmental factors like maternal exposure to alcohol, medications, chemicals, radiation and tobacco during pregnancy, infections like syphilis, rubella and zika virus, maternal nutritional status (such as folate deficiency), obesity and pre-gestational diabetes.

The Consultative Council on Obstetric and Paediatric Mortality and Morbidity (CCOPMM), has legislative responsibility to monitor the health outcomes of mothers and babies, and is an expert advisory body to the Victorian Government. CCOPMM maintains the Victorian Congenital Anomalies Register (VCAR) to provide ongoing health surveillance of major congenital anomalies in Victoria. The VCAR also provides data for epidemiological research and for investigations into potential clusters of congenital anomalies.

This report highlights the incidence (new cases of major congenital anomalies), trends and risk of major congenital anomalies in Victoria from 2007 to 2009. Selected congenital anomalies that are either 'lethal', have significant consequences for surviving children and their families, or are relatively common[2,3], are reported in detail.

The reporting of congenital anomalies varies across Australian states and territories with respect to scope (age limits) and the calculation of rates. Despite these differences, congenital anomaly rates in Victoria during the reporting period were consistent with national data.

The long term trend in rates of abnormalities largely reflect the incremental improvement in the ascertainment, identification and notification of congenital anomalies (from 2.7 per cent in 1983 to 4.2 per cent in 2009). Other factors associated with the rates over time include an increase in the maternal age at birth and the prevalence of chronic conditions like pre-gestational diabetes.

The main findings for the reporting period are:

- Incidence and pregnancies affected: Congenital anomalies affected 4.3 per cent of reported pregnancies, a slight increase from the rate of 4.2 percent in 2005 and 2006. In Victoria, the baby had one or more congenital anomalies in one in 25 pregnancies. Among congenital anomaly cases, 22.6 per cent of babies were reported to have had more than one congenital anomaly.
- Most common congenital anomalies: By diagnostic category, hypospadias (abnormality of the urethral opening in males) was the most common congenital anomaly, followed by obstructive defects of the renal pelvis, and trisomy 21 (Down's syndrome), affecting one in 123, 230 and 297 pregnancies between 2007 and 2009 respectively.
- Preterm birth: Babies with a congenital anomaly were 2.6 times more likely to be born preterm (birth before 37 weeks gestation) than babies without a congenital anomaly.
- Risk of perinatal mortality*: The risk of perinatal mortality was three times higher for babies with a congenital anomaly, compared to babies without a congenital anomaly.
- Maternal age: Women aged 35 years and older were 16.0 per cent more likely to have a baby with a congenital anomaly than those younger than 35 years of age.
- Mother's country of birth: Babies of women born in the Middle East were 27.0 per cent more likely to develop a congenital anomaly than babies of women born in Australia and other countries.

* Excludes termination of pregnancy

# 2. Introduction

The Victorian Congenital Anomalies Register (VCAR), formerly known as the Victorian Birth Defects Register was established in 1982 under the *Health Act 1981*[4–9]. The VCAR is a legislative responsibility of the Consultative Council on Obstetric and Paediatric Mortality and Morbidity (CCOPMM). The VCAR is a statewide population-based surveillance system, which aims to provide statistical information on the prevalence of congenital anomalies in Victoria. Data from the VCAR data can assist with:

- planning and provision of healthcare for people with congenital anomalies
- provision of advice to the community/families who are concerned about having a baby with a congenital anomaly
- epidemiological research to increase knowledge in the aetiology and preventability of congenital anomalies
- assessing the effectiveness of primary prevention and screening programs
- responding to concerns about potential clusters or trends in congenital anomalies

## 2.1 Congenital anomalies reported by the Victorian Congenital Anomalies Register

A 'congenital anomaly' (also called a 'birth defect', 'congenital malformation' or 'congenital disorder'), is any abnormality of prenatal origin, either present after conception or occurring before the end of pregnancy. This includes structural, functional, genetic, chromosomal and biochemical abnormalities, and can be detected before birth, at birth or in later years of life[10].

The VCAR contains information on congenital anomaly outcomes in Victoria; from pregnancy† through to children up to 17 completed years of age.

## 2.2 About this report

This report focuses on major congenital anomalies, which have significant medical, social or cosmetic consequences, typically require a medical intervention and are responsible for most of the deaths, morbidity and disability related to congenital anomalies. These are shown in Appendix A.

Minor congenital anomalies (structural changes without significant health, social or cosmetic consequences) have been excluded from this report. Minor congenital anomalies have mainly been derived from the 'external minor congenital anomalies list' provided by the World Health Organization, United States Centers for Disease Control and Prevention and The International Clearinghouse for Birth Defects Surveillance and Research (ICBDSR)[11],shown in Appendix B.

† Regardless of gestational age; stillbirths and livebirths

# 3. Methodology

## 3.1 Sources of notifications

For the period 2007 to 2009, there were 14,253 notifications of congenital anomalies (Figure 1) for a total of 9,445 cases (excluding notifications for isolated minor congenital anomalies as provided in Appendix B). This approximates to an average of 1.5 notifications per child affected by major congenital anomaly (cases).

The sources of notifications are shown in Table 1.

Figure 1: Congenital anomalies notifications and cases in Victoria, 2007–2009

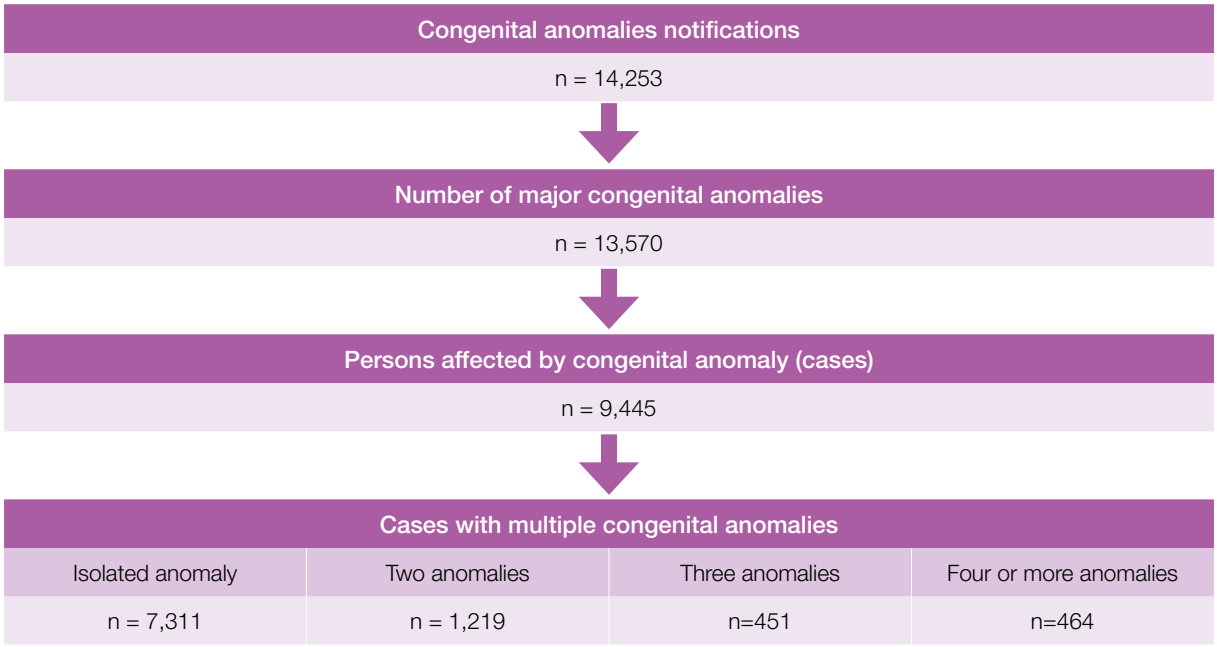



Table 1: Sources of notifications, 2007–2009

| Notification source | Number | Percentage (%) |
|---|---|---|
| Hospital sources | 5,866 | 41.2 |
| Victorian perinatal data collection (VPDC) – birth forms | 4,960 | 34.8 |
| Cytogenetic reports | 1,405 | 9.9 |
| Perinatal death certificates | 842 | 5.9 |
| Maternal and child health nurses | 629 | 4.4 |
| Autopsy reports | 376 | 2.6 |
| Other professionals | 170 | 1.2 |
| Unknown | 4 | 0.0 |
| Other (e.g. parent) | 1 | 0.0 |
| **Total*** | **14,253** | **100.0** |

* Excludes the 1,335 notifications for isolated minor anomalies not included in this report.

## 3.2 Data items

All notifications of congenital anomalies (excluding terminations of pregnancy before 20 weeks gestation and interstate births) are subsequently linked to the Victorian Perinatal Data Collection (VPDC) to obtain the obstetric history for each case.

Health services and maternity care providers must provide data to the VPDC for every birth in Victoria. The routinely collected data items included in the VCAR are listed in Appendix C, with additional data items available for each case over 20 weeks' gestation as reported in the VPDC[12].

## 3.3 Data quality

Data submitted to the VPDC and the VCAR are checked for completeness and validity. Inconsistent or incomplete data is rectified by sending a query to the hospital of birth, or home birth practitioner. Further data cleaning is carried out when all data for the calendar year has been submitted. Validation activities to assess, maintain and improve the quality of the data provided by hospitals to the VPDC and the VCAR are an ongoing commitment of the Department of Health and Human Services (DHHS).

Projects designed to determine the accuracy and completeness of the data submitted to the VPDC and the VCAR are undertaken periodically[13–17], however it is acknowledged that this should be undertaken more regularly. Identification of congenital anomalies can be challenging for termination of pregnancy cases less than 20 weeks gestation[16].

## 3.4 Data analysis

The 2007–2009 Victorian congenital anomaly rates are reported per 1,000 pregnancies‡ for:

- all major congenital anomalies
- major congenital anomalies by maternal and child characteristics
- major congenital anomalies by diagnostic categories.

Trends of major congenital anomalies are reported as well as findings on 29 selected major congenital anomalies considered to be 'lethal, have significant consequences for surviving children and their families, or are relatively common[2,3] by rate, trends and selected maternal and child characteristics.

Relative risk (RR) and its 95 per cent confidence intervals (CI) have been calculated to measure the association of maternal and child characteristics with all congenital anomalies and selected anomalies. A p-value of less than 0.05 was considered significant.

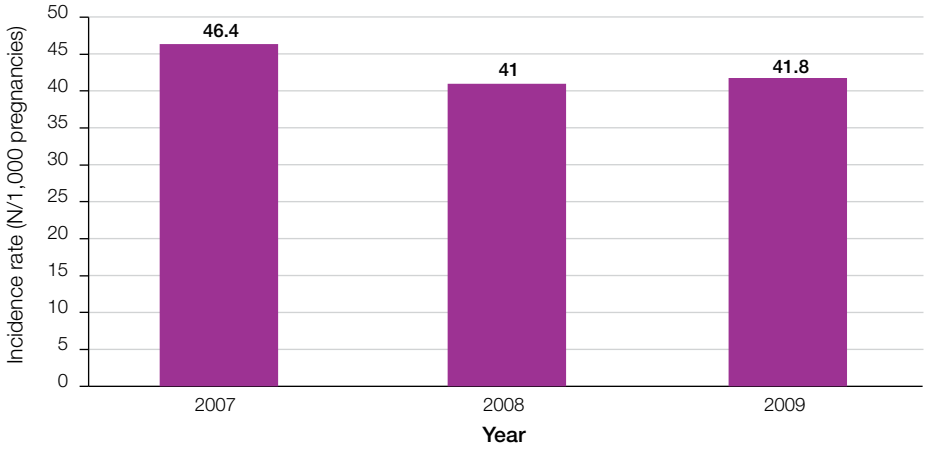
‡ Includes live birth, stillbirth and termination of pregnancy at any gestation

# 4. Findings

## 4.1 Incidence of congenital anomalies in Victoria

In 2007–2009, 4.3 per cent of reported pregnancies (one in 25) were affected by one or more congenital anomalies. The yearly incidence of congenital anomalies is depicted in Figure 2. The five-year (2005 to 2009) incidence of congenital anomalies was 43.4 per 1,000 pregnancies.

Figure 2: Incidence of congenital anomalies per 1,000 pregnancies, 2007–2009

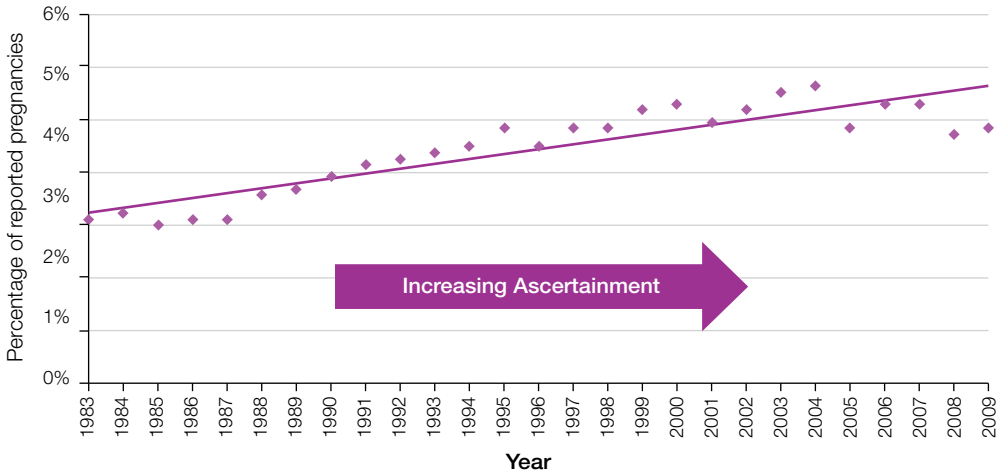



## 4.2 Congenital anomaly trends, 1983 to 2009

Due to a gradual improvement in ascertainment, identification and notification of congenital anomalies, the incidence of congenital anomalies increased from 2.7 per cent in 1983 to 4.2 per cent in 2009. (Figure 3 and Appendix D). National data on congenital anomalies, available for 1981 to 2003[18], depicted a rise from 1.6 per cent between 1981–1992[19] to 3.1 per cent in 2002–03[2].

Other factors associated with this rise in incidence include increased numbers of women with advanced maternal age and the prevalence of chronic conditions, such as pre-gestational diabetes.

Figure 3: Incidence of congenital anomalies, 1983 to 2009



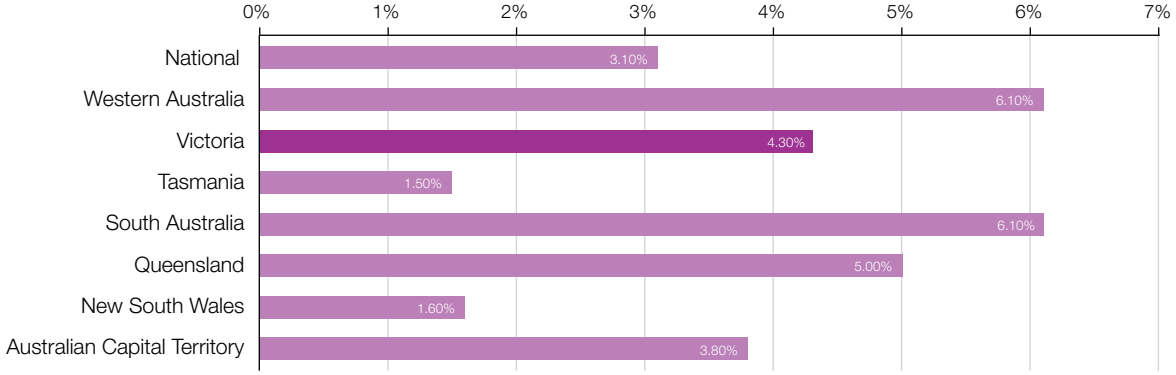
## 4.3 Comparison of congenital anomalies in Victoria with other states and territories of Australia

Congenital anomalies in Victoria from 2007 to 2009 were reported in 4.3 per cent of all pregnancies, which is comparable to the national incidence (3.1 per cent)[2]. The national, state and territory incidence of congenital anomaly is shown in Figure 4 based on data for 2007–2009 or latest available data to 2009.

There is variability among states and territories in the scope of data collection for congenital anomalies, definitions and classifications, denominator selection, sources of notifications and resources for surveillance that impact on the ability to draw comparisons on national rates (Table 2).

In the Northern Territory, congenital anomalies accounted for 1.7 per cent of all the disability adjusted life years (DALYs)[20].

Figure 4: Congenital anomalies in Victoria compared to other states and territories in Australia




Source of the data for various states and territories:

- National: Abeywardana S & Sullivan EA. Congenital anomalies in Australia 2002–2003. Birth anomalies series no. 3 Cat. no. PER 41. Sydney: AIHW National Perinatal Statistics Unit, 2008.
- Western Australia: Bower C, Baynam G, Rudy E, Quick J, Rowley A, Watson L, Cosgrove P. Report of the Western Australian Register of Developmental Anomalies 1980–2014. Western Australian Register of Developmental Anomalies. November 2015.
- Victoria: Victorian Congenital Anomalies Register. Congenital anomalies in Victoria 2007–2009. Consultative Council on Obstetric and Paediatric Mortality and Morbidity (CCOPMM), Victoria, 2016.
- Tasmania: Kids Come First Report 2009. Tasmania Government. 2009.
- South Australia: Gibson CS, Scott H, Scheil W. Birth defects in South Australia 2011. Adelaide. SA Birth Defects Register, Women's and Children's Health Network, 2015.
- Queensland: Endo T, Johnston T, Ellerington J. Data quality and quality issues to be aware of when using the Queensland Perinatal Data Collection to estimate the prevalence of congenital anomalies at birth in Queensland. Health Statistics Unit, Queensland Health. Brisbane 2014.
- New South Wales and ACT: Centre for Epidemiology and Research. New South Wales Mothers and Babies 2009. Sydney: NSW Ministry of Health, 2011 and Australian Capital Territory: Population Health Research Centre, ACT Health. Maternal and Perinatal Health in the ACT 2000–2004, ACT Government, Canberra ACT, 2007.

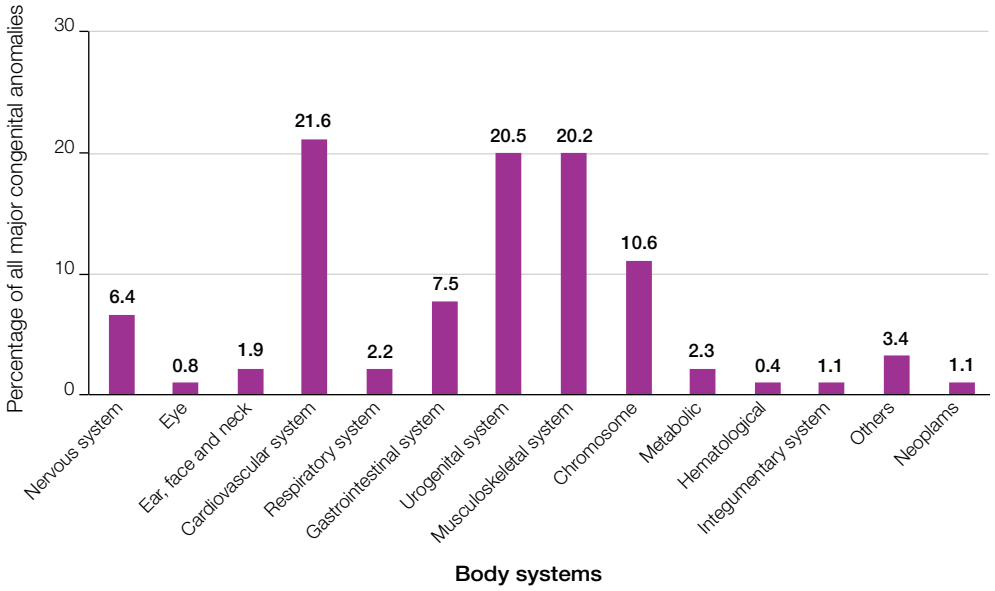
Table 2: Comparison of major congenital anomalies reporting by Australian states and territories

| Reporting | ACT | NSW | NT | Qld | SA | Tas | Vic | WA |
|---|---|---|---|---|---|---|---|---|
| Congenital anomalies included | Structural or anatomical defects that are present at or existing from the time of birth | Conditions that affect the growth, development and health of the baby that are present before birth<br>Conditions due to changes in the number of the baby's chromosomes<br>Four conditions due to changes in the baby's inherited genetic information: cystic fibrosis, phenylketonuria, congenital hypothyroidism and thalassemia major | Structural, genetic, chromosomal and biochemical anomalies | Congenital anomalies agreed upon by the National Congenital Anomalies Steering Committee (NCASC) | Any abnormality, structural or functional, identified up to five years of age, provided that the condition had its origin before birth | Not stated | Any abnormality of prenatal origin including structural, genetic, chromosomal, and biochemical anomalies | Cerebral palsy; or a structural or functional abnormality that is present at conception or occurs before the end of pregnancy and is diagnosed by six years of age |
| Congenital anomalies excluded | Minor congenital anomalies or physiological conditions related to gestational age | Minor anomalies, birth injuries, congenital infections, tumours/cysts and conditions arising from prematurity | Isolated minor anomalies | Those not agreed by NCASC | Most minor malformations, unless they are disfiguring, require treatment or accompany another defect | Not stated | Isolated minor anomalies | Minor malformations unless they are disfiguring or require treatment |
| Pregnancy outcome and gestational age | Births and fetal deaths of at least 20 completed weeks gestation or a birth weight of at least 400 grams | Live births, stillbirths, terminations of pregnancy and spontaneous abortions | Live birth, stillbirth of 20 weeks or more gestation and termination of pregnancy | Live births, fetal deaths, termination of pregnancy | Live births, stillbirths of at least 400 grams birth weight or 20 weeks gestation, and terminations of pregnancies of fetuses with birth defects | Not stated | Live births, stillbirths, terminations of pregnancy at any gestational age | Live births, stillbirths and terminations of pregnancy because of fetal malformation |
| Denominator used in calculation of rates | Births | Live births and stillbirths | Congenital anomalies as percentage of DALYs | Live births plus fetal deaths plus all terminations of pregnancy before 20 weeks duration | Total number of live births and stillbirths, excludes terminations of pregnancy before 20 weeks gestation. | Not stated | Live births, stillbirths and terminations of pregnancy at any gestation | All births |
| Age at which anomaly is notified | At birth | During pregnancy, at birth or till one year of age | Up to 12 months of age | Birth and detected before separation from care | Till 5 years of age | At birth | During pregnancy, at birth or till 18 years of age | Up to 6 years |

## 4.4 Congenital anomalies by body systems

Among all congenital anomalies (not cases), cardiovascular anomalies were the most common (21.6 per cent), followed by anomalies of the urogenital system (20.5 per cent) and musculoskeletal system (20.2 per cent) in 2007–2009. The proportion of congenital anomaly by body systems is shown in Figure 5. Appendix A provides details on the major anomalies according to each body system.

Figure 5: Relative proportion of individual congenital anomalies (not cases) by body systems 2007–2009



## 4.5 Congenital anomalies by diagnostic category

Hypospadias was the most common congenital anomaly reported by diagnostic category, followed by obstructive defects of the renal pelvis and trisomy 21 in 2007–2009 (Table 3).

Table 3: Order of incidence of selected congenital anomalies, 2007–2009

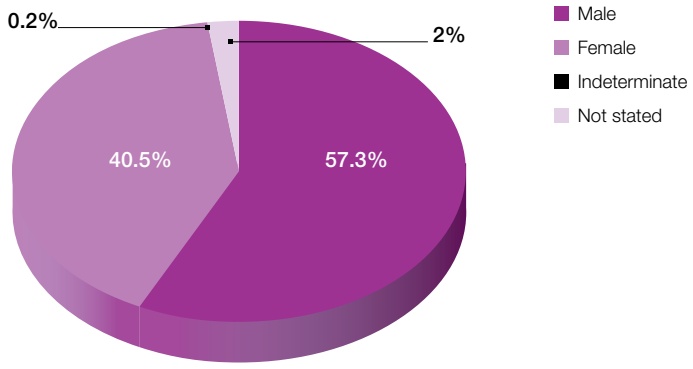
| Defect | N/1,000 pregnancies | 1 in x number of pregnancies |
|---|---|---|
| Hypospadias | 8.1 | 123 |
| Obstructive defects of renal pelvis | 4.3 | 230 |
| Trisomy 21 | 3.4 | 297 |
| Ventricular septal defect | 3.1 | 326 |
| Developmental dysplasia of the hip | 3.0 | 332 |
| All neural tube defects | 1.1 | 910 |
| Trisomy 18 | 1.1 | 922 |
| Hydrocephalus | 0.8 | 1,131 |
| Cleft palate | 0.7 | 1,354 |
| Limb reduction defects | 0.6 | 1,534 |
| Cleft lip and palate | 0.6 | 1,556 |
| Renal agenesis and dysgenesis | 0.6 | 1,556 |
| Transposition of the great vessels | 0.6 | 1,675 |
| Cystic kidney disease | 0.6 | 1,755 |
| Spina bifida | 0.5 | 1,925 |
| Anencephaly | 0.5 | 2,194 |
| Coarctation of the aorta | 0.4 | 2,239 |
| Cleft lip | 0.4 | 2,359 |
| Diaphragmatic hernia | 0.4 | 2,493 |
| Hypoplastic left heart syndrome | 0.4 | 2,493 |
| Trisomy 13 | 0.4 | 2,676 |
| Exomphalos | 0.4 | 2,743 |
| Tetralogy of fallot | 0.3 | 2,965 |
| Anorectal atresia and/or stenosis | 0.3 | 3,180 |
| Oesophageal atresia and/or stenosis | 0.3 | 3,227 |
| Microcephalus | 0.3 | 3,597 |
| Small intestinal atresia and/or stenosis | 0.3 | 3,783 |
| Gastroschisis | 0.2 | 4,302 |
| Encephalocele | 0.1 | 6,649 |

## 4.6 Congenital anomalies by child characteristics

### 4.6.1 Gender

For all congenital anomaly cases reported between 2007 and 2009, 57.3 per cent were males (Figure 6). The rate of congenital anomalies was 48.4 per 1,000 pregnancies for male babies and 36.1 per 1,000 pregnancies for female babies. Male babies were 34.0 per cent more likely to have a congenital anomaly than female babies (RR 1.34, 95 per cent CI 1.28–1.39, p-value < 0.0001).

Figure 6: Congenital anomalies by gender, 2007–2009




### 4.6.2 Gestational age

Between 2007 and 2009, babies with a congenital anomaly were 2.6 times more likely to be born preterm (less than 37 weeks gestation) than those not having a congenital anomaly (RR 2.65, 95 per cent CI 2.54–2.77, p-value < 0.0001). In 20.5 per cent of all congenital anomaly cases, the gestational age was less than 37 weeks (Table 4).

Table 4: Congenital anomalies by gestational age, 2007–2009

| Gestational age (weeks)* | Number of cases with at least one congenital anomaly | Number of reported pregnancies | Congenital anomalies/ 1,000 pregnancies |
|---|---|---|---|
| 20–27 | 703 | 2,520 | 279.0 |
| 28–31 | 295 | 1,774 | 166.3 |
| 32–36 | 937 | 13,957 | 67.1 |
| 37–41 | 6,265 | 196,734 | 31.8 |
| >41 | 96 | 2,915 | 32.9 |

*Excludes terminations of pregnancy less than 20 weeks and those in whom gestational age was not stated

### 4.6.3 Birth weight

The rate of congenital anomalies in low birthweight babies (< 2,500 grams) was 118.3 per 1,000 pregnancies compared with 31.9 per 1,000 pregnancies for babies weighing 2,500 grams or more in 2007–2009 (Table 5). For low birthweight babies, the risk of having a congenital anomaly was 3.7 times higher than for babies weighing 2,500 grams or more (RR 3.70, 95 per cent CI 3.52–3.89, p-value < 0.0001).

Table 5: Congenital anomalies by birth weight, 2007–2009

| Weight in grams* | Number of cases with at least one congenital anomaly | Number of reported pregnancies | Congenital anomalies/ 1,000 pregnancies |
|---|---|---|---|
| < 1,000 | 715 | 2,641 | 270.7 |
| 1,000–2,499 | 1,103 | 12,730 | 86.6 |
| ≥ 2,500 | 6,468 | 202,727 | 31.9 |

*Excludes terminations of pregnancy less than 20 weeks and those cases in whom weight in grams was not stated

### 4.6.4 Birth plurality

By birth plurality, 94.0 per cent of congenital anomaly cases were singleton and five per cent were multiple births, plurality unknown for one per cent of births. In 2007–2009, the rate of congenital anomaly in singleton births was 42.1 per 1,000 pregnancies and 59.8 per 1,000 pregnancies for multiple births. The risk of congenital anomaly was 42.3 per cent higher in multiple births than singleton births (RR 1.42, 95 per cent CI 1.29–1.56, p-value < 0.0001).

Assisted reproductive technology (ART) might have contributed to higher rate of congenital anomalies in multiple births. The VPDC started collecting data on the use of ART from 2009. Data for 2009 indicates that 60.0 per cent of multiple births in women who used ART resulted in a congenital anomaly as compared to 47.0 per cent of multiple births in women not using ART. In medical literature, while some studies supported the positive association of ART with major congenital anomalies[21], other studies found no association[22,23].

### 4.6.5 Perinatal outcomes

Twelve per cent (n = 1,138) of all reported pregnancies with major congenital anomalies were terminated before 20 weeks gestation and 5.9 per cent (n = 555) were terminated at 20 weeks or more[§] gestation between 2007 and 2009. Of babies born at 20 weeks or more gestation with congenital anomaly, 1.1 per cent were stillborn, 1.8 per cent were neonatal deaths and 96.9 per cent lived beyond 28 days (Appendix E).

The risk of perinatal mortality in babies having congenital anomaly was 3.1 times higher than for babies without a congenital anomaly (RR 3.09, 95 per cent CI 2.71–3.52, p-value < 0.0001). The perinatal mortality rate (excluding terminations) in babies with one or more congenital anomalies was 31.3 per 1,000 births compared with a perinatal mortality rate of 10.0 per 1,000 births in babies not having a congenital anomaly.

§ The majority of structural congenital anomalies are not detected until the the 'anomaly ultrasound scan' is undertaken between 18 to 20 weeks gestation.

## 4.7 Congenital anomalies by maternal characteristics

### 4.7.1 Age

Between 2007 and 2009, women aged 35 years and older were 16.0 per cent more likely to have a baby with a congenital anomaly than those younger than 35 years of age (RR 1.16, 95 per cent CI 1.11–1.22, p-value < 0.0001). The rate of congenital anomalies was highest in women aged 40 to 44 years, followed by those younger than 20 years of age (Table 6).

Table 6: Congenital anomalies by mother's age, 2007–2009

| Age group* | Number of cases with at least one congenital anomaly | Number of reported pregnancies | Congenital anomalies/ 1,000 pregnancies |
|---|---|---|---|
| < 20 | 256 | 5,774 | 44.3 |
| 20–24 | 976 | 24,709 | 39.5 |
| 25–29 | 2,294 | 55,871 | 41.1 |
| 30–34 | 2,949 | 75,181 | 39.2 |
| 35–39 | 2,104 | 47,851 | 44.0 |
| 40–44 | 597 | 9,749 | 61.2 |

*Excludes those cases in which neither age nor date of birth was stated

### 4.7.2 Aboriginal women

The rate of congenital anomalies was similar for Aboriginal women (46.6 per 1,000 pregnancies) and non-Aboriginal women (43.5 per 1,000 pregnancies) in 2007–2009, indicating comparable risk of having a baby with a congenital anomaly (RR 1.07, 95 per cent CI 0.88–1.29, p-value 0.47).

### 4.7.3 Parity

The rate of congenital anomaly was 45.6 per 1,000 pregnancies in primiparous women and 42.3 per 1,000 pregnancies in multiparous women between 2007 and 2009. For primiparous women, the risk of having a baby with congenital anomaly was seven per cent higher than for multiparous women (RR 1.07, 95 per cent CI 1.03–1.12, p-value 0.0004).

### 4.7.4 Country of birth

The rate of congenital anomalies was highest for babies of women born in the Middle East and Africa in 2007–2009 (Table 7). Babies of women born in the Middle East were 27.0 per cent more likely to develop a congenital anomaly than babies of women born in Australia and other countries (RR 1.27, 95 per cent CI 1.13–1.44, p-value 0.0001). High number of women born in the Middle East (72.1 per cent) resided in the North and West metropolitan region in 2007–2009.

Table 7: Congenital anomalies by mother's country of birth, 2007–2009

| Mother's country of birth | Number of cases with at least one congenital anomaly | Number of reported pregnancies | Congenital anomalies/ 1,000 pregnancies |
|---|---|---|---|
| Australia | 6,810 | 156,153 | 43.6 |
| Oceania/New Zealand | 285 | 7,519 | 37.9 |
| United Kingdom/Ireland | 234 | 5,469 | 42.8 |
| Europe | 275 | 6,184 | 44.5 |
| Middle East | 253 | 4,583 | 55.2 |
| North America | 47 | 1,364 | 34.5 |
| South America | 38 | 1,272 | 29.9 |
| Africa | 268 | 5,719 | 46.9 |
| Asia | 1,189 | 28,068 | 42.4 |
| Unknown | 46 | 3,090 | 14.9 |

## 4.8 Selected major congenital anomalies

### 4.8.1 Anencephaly

**Definition:** Anencephaly is the total or partial absence of the cranial vault, covering skin and the brain tissue.

**Incidence:** Of all the congenital anomaly cases reported between 2007 and 2009, 1.1 per cent (n = 100) of babies had anencephaly, of which 85.0 per cent (n = 85) were isolated and 15.0 per cent (n = 15) occurred with other congenital anomalies.

**Trend:** From 1998 to 2009, there was no significant change (p-value 0.573) in the incidence of anencephaly (Figure 7).

**Risk factors:** The risk of anencephaly did not vary with maternal age, birth plurality or the gender of the baby (Table 8).

Figure 7: Anencephaly per 1,000 pregnancies, 1998 to 2009

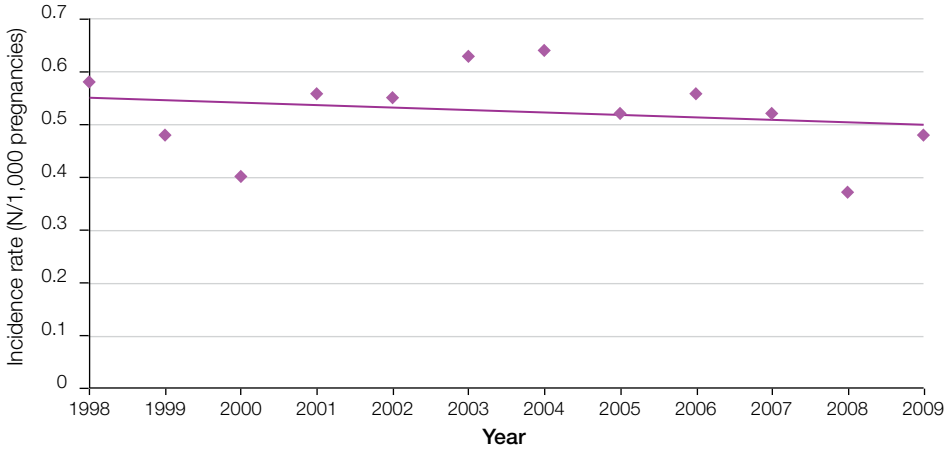



Table 8: Anencephaly by selected maternal and child characteristics, 2007–2009

| Characteristic | | Anencephaly/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.5 | - |
| | ≥ 35 years | 0.4 | 1.25 (0.78–2.01) |
| **Plurality** | Single (Ref) | 0.5 | - |
| | Multiple | 0.7 | 0.65 (0.26–1.62) |
| **Gender** | Male (Ref) | 0.2 | - |
| | Female | 0.2 | 0.82 (0.44–1.53) |

### 4.8.2 Spina bifida

**Definition:** Spina bifida is the herniation or exposure of the spinal cord and/or meninges through incomplete closure of the spine. Hydrocephalus may or may not be present.

**Incidence:** Of all the congenital anomaly cases reported between 2007 and 2009, 1.2 per cent (n = 114) of babies had spina bifida, of which 74.6 per cent (n = 85) were isolated and 25.4 per cent (n = 29) occurred with other congenital anomalies.

**Trend:** There was no significant change (p-value 0.578) in the incidence of spina bifida from 1998 to 2009 (Figure 8).

**Risk factors:** The risk of spina bifida did not vary significantly with maternal age, birth plurality or gender (Table 9).

Figure 8: Spina bifida per 1,000 pregnancies, 1998 to 2009

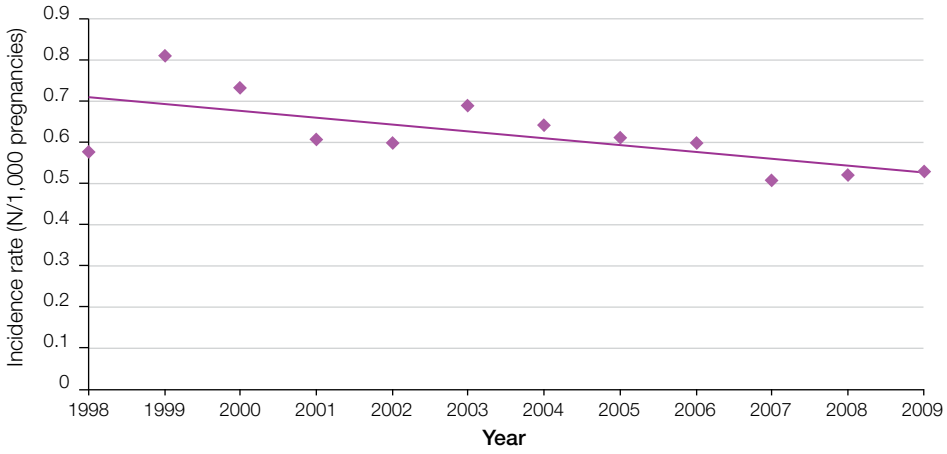



Table 9: Spina bifida by selected maternal and child characteristics, 2007–2009

| Characteristic | | Spina bifida/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.5 | - |
| | ≥ 35 years | 0.5 | 0.94(0.62–1.43) |
| **Plurality** | Single (Ref) | 0.5 | - |
| | Multiple | 0.1 | 3.91(0.54–28.06) |
| **Gender** | Male (Ref) | 0.4 | - |
| | Female | 0.5 | 0.77 (0.52–1.16) |

### 4.8.3 Encephalocele

**Definition:** Encephalocele is the herniation of the brain and/or meninges through a defect in the skull.

**Incidence:** Of all congenital anomaly cases reported between 2007 and 2009, 0.3 per cent of babies (n = 33) had encephalocele, of which 45.5 per cent (n = 15) occurred as an isolated anomaly and 54.5 per cent (n = 18) occurred with other congenital anomalies.

**Trend:** Incidence rates of encephalocele did not change significantly (p-value 0.583) from 1998 to 2009 (Figure 9).

**Risk factors:** The risk of encephalocele did not vary significantly with maternal age, birth plurality or gender (Table 10).

Figure 9: Encephalocele per 1,000 pregnancies, 1998 to 2009

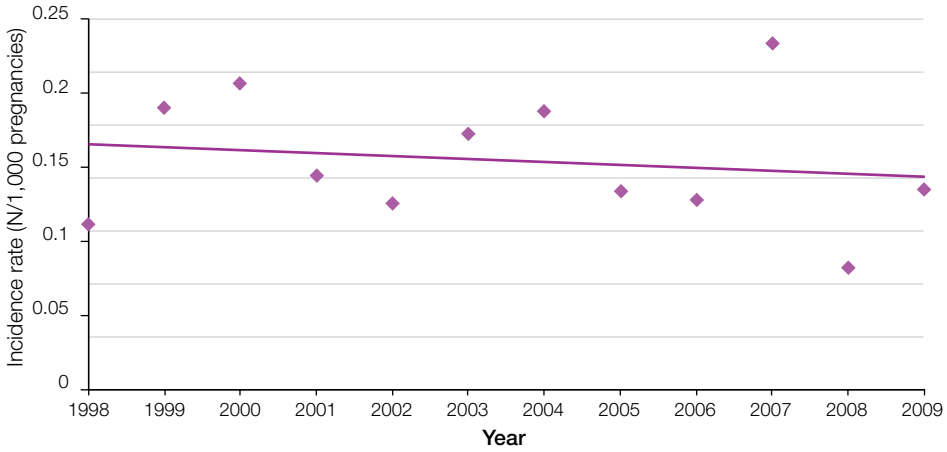



Table 10: Encephalocele by selected maternal and child characteristics, 2007–2009

| Characteristic | | Encephalocele/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.1 | - |
| | ≥ 35 years | 0.2 | 0.94 (0.43–2.03) |
| **Plurality** | Single (Ref) | 0.1 | - |
| | Multiple | 0.4 | 0.34 (0.10–1.13) |
| **Gender** | Male (Ref) | 0.1 | - |
| | Female | 0.1 | 0.83 (0.39–1.74) |

### 4.8.4 All neural tube defects

**Definition:** All cases of anencephaly, spina bifida and encephalocele.

**Incidence:** Of all the congenital anomaly cases reported between 2007 and 2009, 2.6 per cent (n = 241) of babies had neural tube defects.

**Trend:** Although there was a decline in neural tube defects from 1998 to 2009 (Figure 10) this was not statistically significant (p-value 0.514). This finding is comparable to trends in South Australia[24], Western Australia[25] and Queensland[26].

**Risk factors:** The risk of neural tube defects did not differ significantly with maternal age, birth plurality and gender (Table 11).

Figure 10: All neural tube defects per 1,000 pregnancies, 1998 to 2009

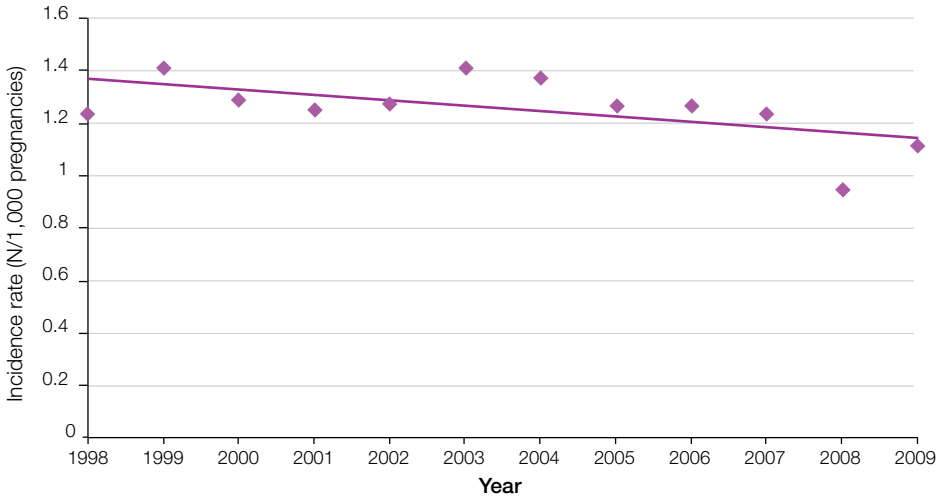



Table 11: All neural tube defects by selected maternal and child characteristics, 2007–2009

| Characteristic | | All neural tube defects/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 1.1 | - |
| | ≥ 35 years | 1.0 | 1.08 (0.80–1.44) |
| **Plurality** | Single (Ref) | 1.1 | - |
| | Multiple | 1.2 | 0.89 (0.45–1.74) |
| **Gender** | Male (Ref) | 0.7 | - |
| | Female | 0.8 | 0.87 (0.64–1.18) |

### 4.8.5 Microcephalus

**Definition:** Microcephalus is the presence of a small cranium defined by an occipito-frontal circumference three standard deviations below the age-sex appropriate distribution curves.

**Incidence:** Between 2007 and 2009, microcephalus was reported in 0.6 per cent (n = 61) of all congenital anomaly cases, of which 50.8 per cent (n = 31) were isolated.

**Trend:** Incidence of microcephalus from 1998 to 2009 did not change significantly (p-value 0.649) (Figure 11).

**Risk factors:** The risk of having a baby with microcephalus did not vary with maternal age; however, multiple births increased the risk by 2.5 times compared with singleton pregnancy and female babies were 1.8 times more likely to develop microcephalus than male babies (Table 12).

Figure 11: Microcephalus per 1,000 pregnancies, 1998 to 2009

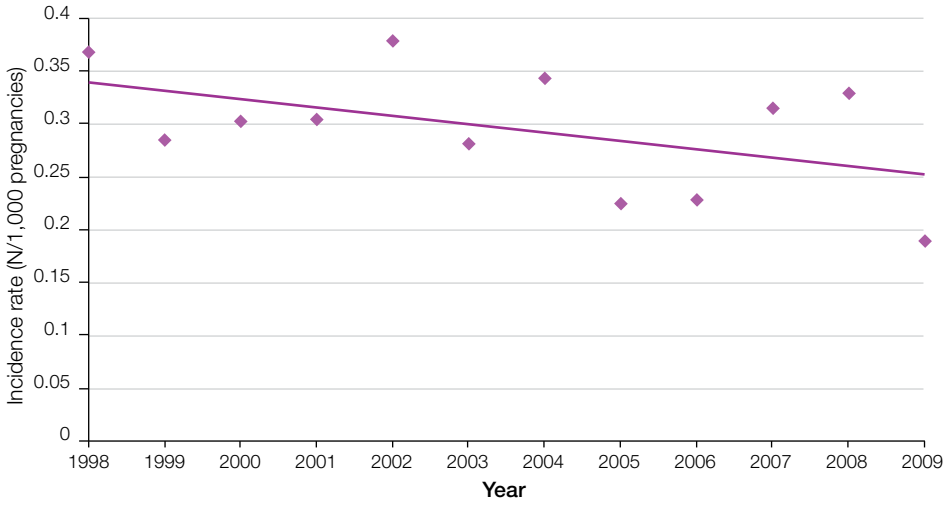



Table 12: Microcephalus by selected maternal and child characteristics, 2007–2009

| Characteristic | | Microcephalus/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.3 | - |
| | ≥ 35 years | 0.3 | 0.78 (0.45–1.34) |
| **Plurality** | Single (Ref) | 0.3 | - |
| | Multiple | 0.7 | 2.57 (1.03–6.41)* |
| **Gender** | Male (Ref) | 0.2 | - |
| | Female | 0.4 | 1.8 (1.06–3.04)* |

*Statistically significant

### 4.8.6 Hydrocephalus

**Definition:** Hydrocephalus is the dilatation of the cerebral ventricles (not associated with primary brain atrophy) with or without enlargement of the head. Diagnosed at birth, these cases exclude hydrocephalus associated with spina bifida or encephalocele.

**Incidence:** Of all the congenital anomaly cases reported between 2007 and 2009, 2.1 per cent (n = 194) of babies had hydrocephalus, of which 42.3 per cent occurred as an isolated anomaly.

**Trend:** From 1998 to 2009, the incidence rate of hydrocephalus did not change significantly (p-value 0.402) (Figure 12).

**Risk factors:** The risk of giving birth to a baby with hydrocephalus did not vary with maternal age; however, multiple births increased the risk by three times. Male babies were 57.0 per cent more likely to develop hydrocephalus than female babies (Table 13).

Figure 12: Hydrocephalus per 1,000 pregnancies, 1998 to 2009

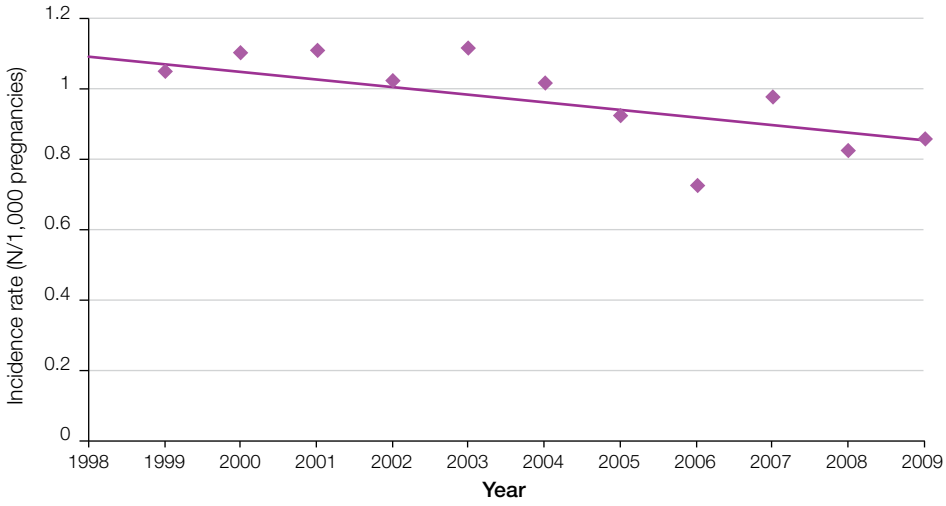



Table 13: Hydrocephalus by selected maternal and child characteristics, 2007–2009

| Characteristic | | Hydrocephalus/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.9 | - |
| | ≥ 35 years | 0.9 | 0.93 (0.68–1.28) |
| **Plurality** | Single (Ref) | 0.8 | - |
| | Multiple | 2.4 | 2.94 (1.81–4.77)* |
| **Gender** | Male (Ref) | 1.1 | 1.57 (1.17–2.10)* |
| | Female | 0.7 | - |

*Statistically significant

### 4.8.7 Transposition of the great vessels

**Definition:** In this anomaly, the aorta exits from the right ventricle and the pulmonary artery from the left ventricle, with or without other cardiac defects. It includes double outlet right ventricle, so-called corrected transposition.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 1.4 per cent (n = 131) had transposition of great vessels, of which 10.7 per cent (n = 14) occurred as an isolated anomaly.

**Trend:** There was no significant change (p-value 0.715) in incidence rates from 1998 to 2009 (Figure 13).

**Risk factors:** The risk of having a baby with transposition of the great vessels did not vary significantly with maternal age or birth plurality; however, male babies were 89.0 per cent more likely to develop this anomaly (Table 14).

Figure 13: Transposition of the great vessels per 1,000 pregnancies, 1998 to 2009

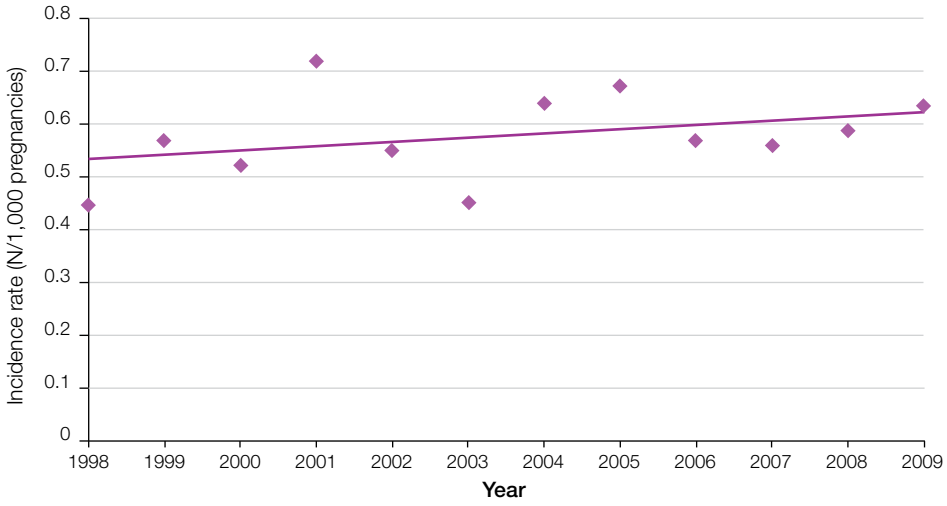



Table 14: Transposition of the great vessels by selected maternal and child characteristics, 2007–2009

| Characteristic | | Transposition of the great vessels/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.6 | - |
| | ≥ 35 years | 0.6 | 0.97 (0.65–1.42) |
| **Plurality** | Single (Ref) | 0.6 | - |
| | Multiple | 0.8 | 1.38 (0.60–3.13) |
| **Gender** | Male (Ref) | 0.8 | 1.89 (1.32–2.72)* |
| | Female | 0.4 | - |

*Statistically significant

### 4.8.8 Tetralogy of Fallot

**Definition:** Tetralogy of fallot is characterised by ventricular septal defect, overriding aorta, infundibular pulmonary stenosis and often right ventricular hypertrophy.

**Incidence:** Of all the congenital anomaly cases reported between 2007 and 2009, 0.8 per cent (n = 74) of babies had tetralogy of fallot, of which 41.9 per cent (n = 31) occurred as an isolated anomaly.

**Trend:** Incidence rates from 1998 to 2009 were comparable (p-value 0.791) (Figure 14).

**Risk factors:** The risk of having a baby with tetralogy of fallot did not vary significantly with maternal age, birth plurality or gender (Table 15).

Figure 14: Tetralogy of Fallot per 1,000 pregnancies, 1998 to 2009

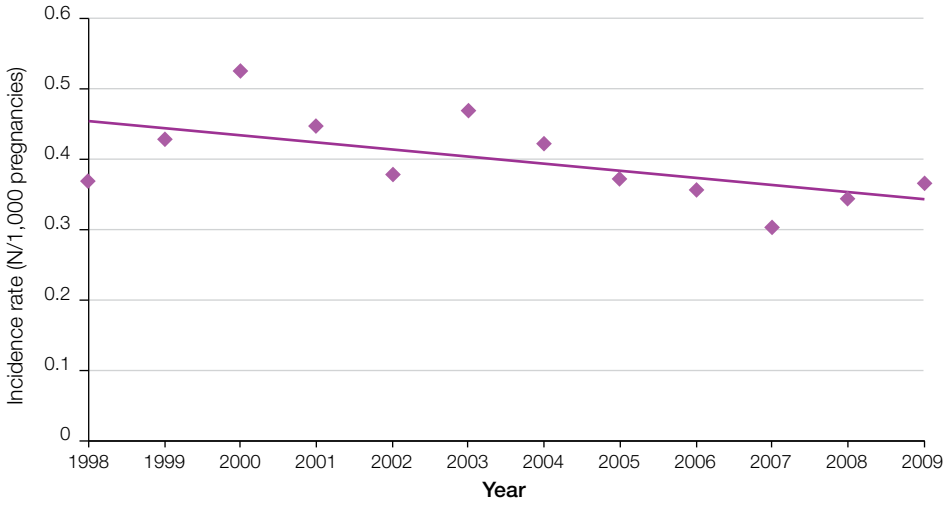



Table 15: Tetralogy of Fallot by selected maternal and child characteristics, 2007–2009

| Characteristic | | Tetralogy of Fallot/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.4 | - |
| | ≥ 35 years | 0.3 | 1.18 (0.69–2.04) |
| **Plurality** | Single (Ref) | 0.3 | - |
| | Multiple | 0.5 | 1.64 (0.60–4.50) |
| **Gender** | Male (Ref) | 0.4 | - |
| | Female | 0.3 | 1.16 (0.73–1.84) |

### 4.8.9 Ventricular septal defect

**Definition:** Ventricular septal defect is characterised by a defect in the septum between the left and right ventricles of the heart, which permits blood to be shunted between them. It excludes ventricular septal defect as part of Tetralogy of Fallot.

**Incidence:** Of all the congenital anomaly cases reported between 2007 and 2009, 7.1 per cent (n = 673) of babies had ventricular septal defect, of which 44.9 per cent (n = 302) occurred as an isolated anomaly.

**Trend:** Rates of incidence of ventricular septal defect decreased significantly (p-value < 0.001) from 1998 to 2009 (Figure 15).

**Risk factors:** The risk of ventricular septal defect was not affected by gender of the baby; however, babies born to women aged 35 years and older were 19.0 per cent more likely to develop the defect compared with those born to women younger than 35 years of age. Multiple births double the risk of ventricular septal defect compared with singleton births (Table 16).

Figure 15: Ventricular septal defect per 1,000 pregnancies, 1998 to 2009

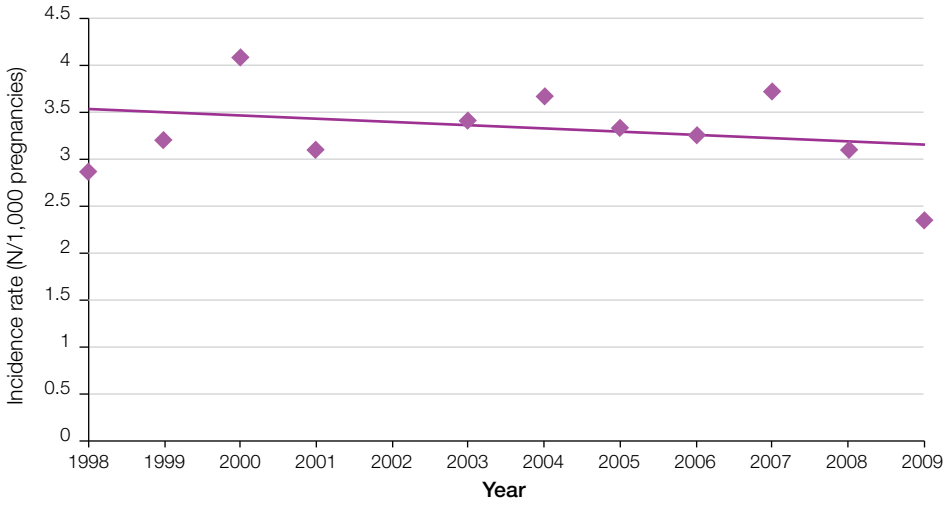



Table 16: Ventricular septal defect by selected maternal and child characteristics, 2007–2009

| Characteristic | | Ventricular septal defect/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 2.9 | - |
| | ≥ 35 years | 3.5 | 1.19 (1.06–1.41)* |
| **Plurality** | Single (Ref) | 3.0 | - |
| | Multiple | 6.0 | 2.05 (1.52–2.78)* |
| **Gender** | Male (Ref) | 3.3 | - |
| | Female | 2.9 | 1.15 (0.99–1.34) |

*Statistically significant

### 4.8.10 Hypoplastic left heart syndrome

**Definition:** Hypoplastic left heart syndrome comprises hypoplastic left ventricle associated with aorta and/or mitral valve atresia.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 0.9 per cent (n = 88) of babies had hypoplastic left heart syndrome, of which 44.3 per cent (n = 39) occurred as an isolated anomaly.

**Trend:** The incidence of hypoplastic left heart syndrome did not change significantly (p-value 0.332) from 1998 to 2009 (Figure 16).

**Risk factors:** The risk of hypoplastic left heart syndrome did not vary with maternal age or birth plurality; however, male babies were 85.0 per cent more likely to develop hypoplastic left heart syndrome than female babies (Table 17).

Figure 16: Hypoplastic left heart syndrome per 1,000 pregnancies, 1998 to 2009

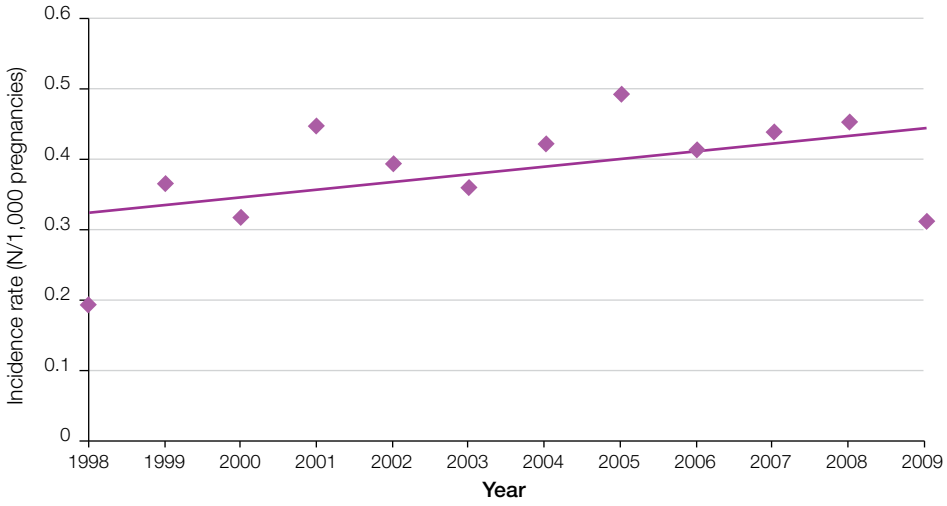



Table 17: Hypoplastic left heart syndrome by selected maternal and child characteristics, 2007–2009

| Characteristic | | Hypoplastic left heart syndrome/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.4 | - |
| | ≥ 35 years | 0.4 | 1.12 (0.69–1.84) |
| **Plurality** | Single (Ref) | 0.4 | - |
| | Multiple | 0.3 | 1.49 (0.36–6.06) |
| **Gender** | Male (Ref) | 0.5 | 1.85 (1.18–2.90)* |
| | Female | 0.3 | - |

*Statistically significant

### 4.8.11 Coarctation of the aorta

**Definition:** In coarctation of the aorta, there is an obstruction in the descending aorta, almost invariably (98 per cent) at the insertion of the ductus arteriosus.

**Incidence:** Among all the congenital anomaly cases reported between 2007 and 2009, 1.04 per cent (n = 98) of babies had coarctation of the aorta, of which 18.4 per cent (n = 18) occurred as an isolated anomaly.

**Trend:** Incidence rates of coarctation of the aorta did not change significantly (p-value 0.342) from 1998 to 2009 (Figure 17).

**Risk factors:** The risk of a baby having coarctation of the aorta does not vary with maternal age, birth plurality or gender (Table 18).

Figure 17: Coarctation of the aorta per 1,000 pregnancies, 1998 to 2009

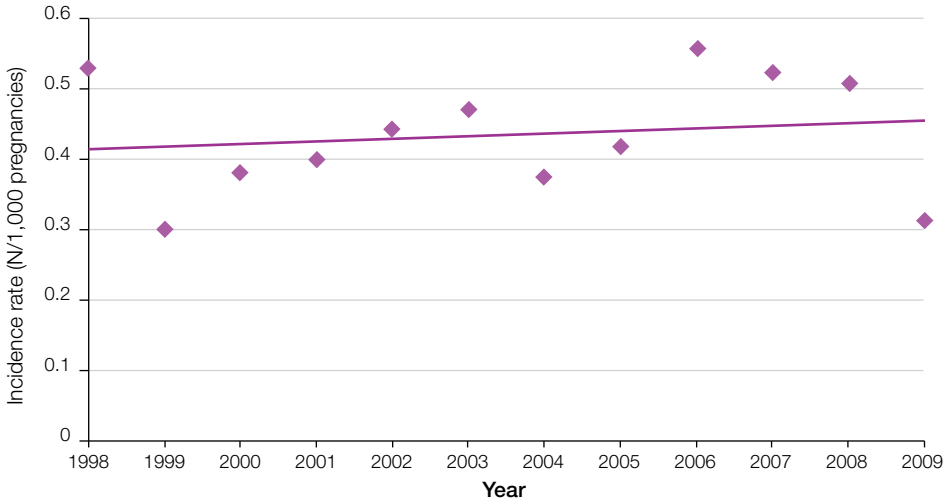



Table 18: Coarctation of the aorta by selected maternal and child characteristics, 2007–2009

| Characteristic | | Coarctation of the aorta/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.4 | - |
| | ≥ 35 years | 0.5 | 0.93 (0.59–1.45) |
| **Plurality** | Single (Ref) | 0.4 | - |
| | Multiple | 0.8 | 0.53 (0.23–1.21) |
| **Gender** | Male (Ref) | 0.5 | - |
| | Female | 0.4 | 1.27 (0.85–1.90) |

### 4.8.12 Cleft palate

**Definition:** Cleft palate is a closure defect of the hard and/or soft palate behind the foramen incisivum without cleft lip; excludes cleft palate with cleft lip, cleft uvula, functional short palate and high narrow palate.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 1.7 per cent (n = 162) had cleft palate, of which 57.4 per cent (n = 93) occurred as an isolated anomaly.

**Trend:** The incidence of cleft palate significantly increased in 2003, decreased to 2007, and increased in 2008 and 2009 (p-value 0.009) (Figure 18).

**Risk factors:** The risk of having a baby with cleft palate did not differ with maternal age or birth plurality; however, female babies were 38.0 per cent more likely to develop cleft palate than male babies (Table 19).

Figure 18: Cleft palate per 1,000 pregnancies, 1998 to 2009

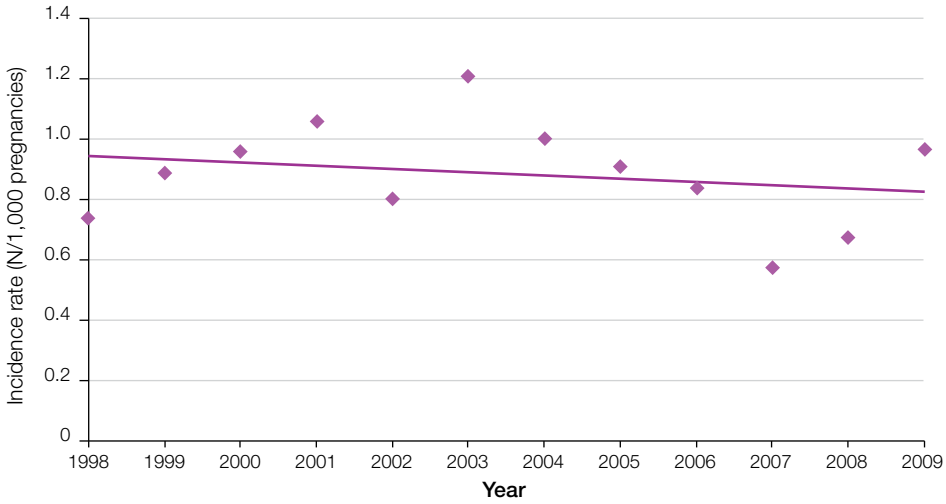



Table 19: Cleft palate by selected maternal and child characteristics, 2007–2009

| Characteristic | | Cleft palate/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.7 | - |
| | ≥ 35 years | 0.7 | 1.04 (0.73–1.48) |
| **Plurality** | Single (Ref) | 0.7 | - |
| | Multiple | 0.5 | 1.37 (0.50–3.69) |
| **Gender** | Male (Ref) | 0.6 | - |
| | Female | 0.9 | 1.38 (1.01–1.89)* |

*Statistically significant

### 4.8.13 Cleft lip

**Definition:** Cleft lip is a closure defect (partial or complete) of the upper lip, excluding the alveolar ridge and palate.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 1.0 per cent (n = 93) had cleft lip, of which 78.5 per cent (n = 73) were isolated and 21.5 per cent (n = 20) occurred with other congenital anomalies.

**Trend:** Incidence rates remained similar (p-value 0.825) from 1998 to 2009 (Figure 19).

**Risk factors:** The risk of cleft lip did not vary with maternal age, birth plurality or gender (Table 20).

Figure 19: Cleft lip per 1,000 pregnancies, 1998 to 2009

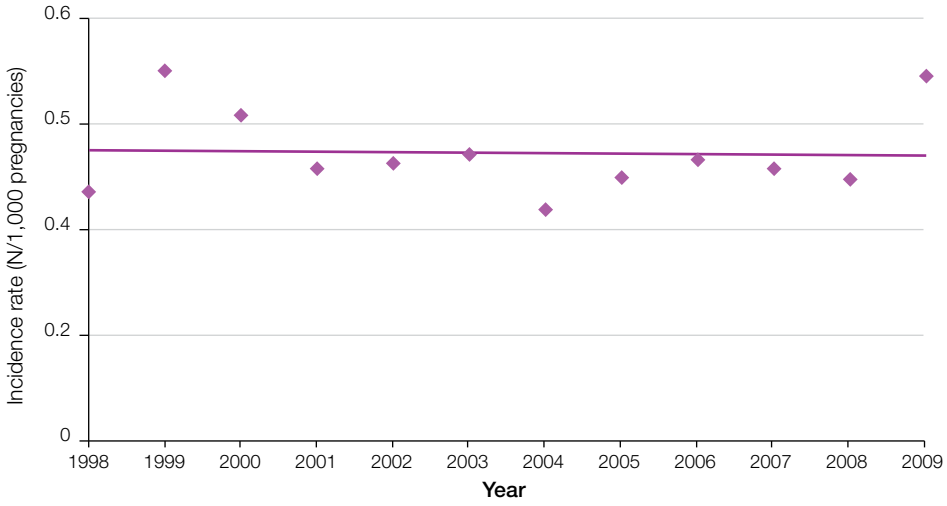



Table 20: Cleft lip by selected maternal and child characteristics, 2007–2009

| Characteristic | | Cleft lip/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.4 | - |
| | ≥ 35 years | 0.4 | 1.04 (0.65–1.67) |
| **Plurality** | Single (Ref) | 0.4 | - |
| | Multiple | 0.3 | 1.57 (0.38–6.40) |
| **Gender** | Male (Ref) | 0.4 | - |
| | Female | 0.4 | 1.14 (0.75–1.72) |

### 4.8.14 Cleft lip and palate

**Definition:** Cleft lip and palate is a closure defect (partial or complete) of the upper lip, alveolar ridge and palate.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 1.5 per cent (n = 141) had cleft lip and palate, of which 66.7 per cent (n = 94) occurred as an isolated anomaly.

**Trend:** Incidence rates of cleft lip and palate remained similar (p-value 0.18) from 1998 to 2009 (Figure 20).

**Risk factors:** The risk of development of cleft lip and palate did not vary with maternal age and birth plurality; however, male babies were 76.0 per cent more likely to have cleft lip and palate than female babies (Table 21).

Figure 20: Cleft lip and palate per 1,000 pregnancies, 1998 to 2009

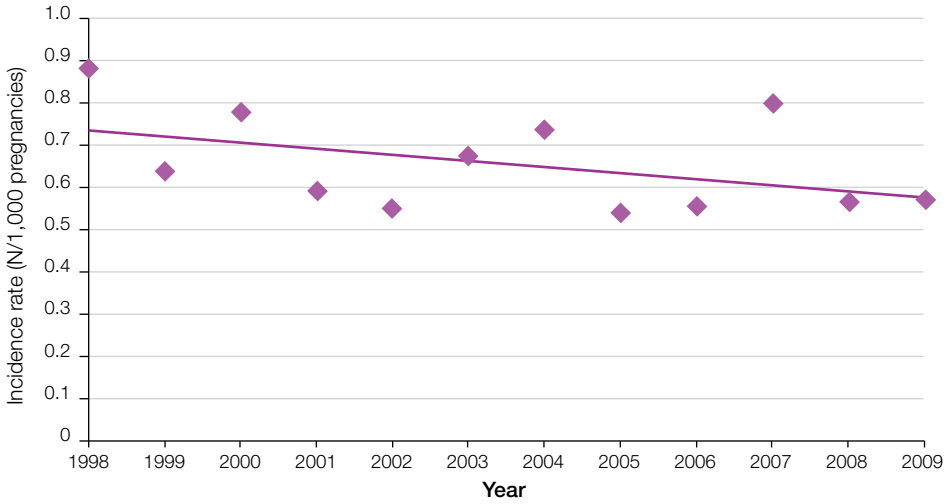



Table 21: Cleft lip and palate by selected maternal and child characteristics, 2007–2009

| Characteristic | | Cleft lip and palate/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.6 | - |
| | ≥ 35 years | 0.6 | 0.99 (0.68 –1.44) |
| **Plurality** | Single (Ref) | 0.6 | - |
| | Multiple | 0.9 | 0.65 (0.30–1.40) |
| **Gender** | Male (Ref) | 0.8 | 1.76 (1.24–2.49)* |
| | Female | 0.5 | - |

*Statistically significant

### 4.8.15 Oesophageal atresia and/or stenosis

**Definition:** Oesophageal atresia and/or stenosis is the absence of continuity or narrowing of the oesophagus, with or without tracheal fistula.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 0.7 per cent (n = 68) had oesophageal atresia and/or stenosis, of which 23.5 per cent (n = 16) occurred as an isolated anomaly.

**Trend:** Incidence rates remained similar (p-value 0.876) from 1998 to 2009 (Figure 21).

**Risk factors:** Women aged 35 years and older were 64.0 per cent more likely to give birth to a baby with oesophageal atresia and/or stenosis compared with women younger than 35 years of age. The risk of oesophageal atresia and/or stenosis in babies did not vary with birth plurality or gender of the baby (Table 22).

Figure 21: Oesophageal atresia and/or stenosis per 1,000 pregnancies, 1998 to 2009

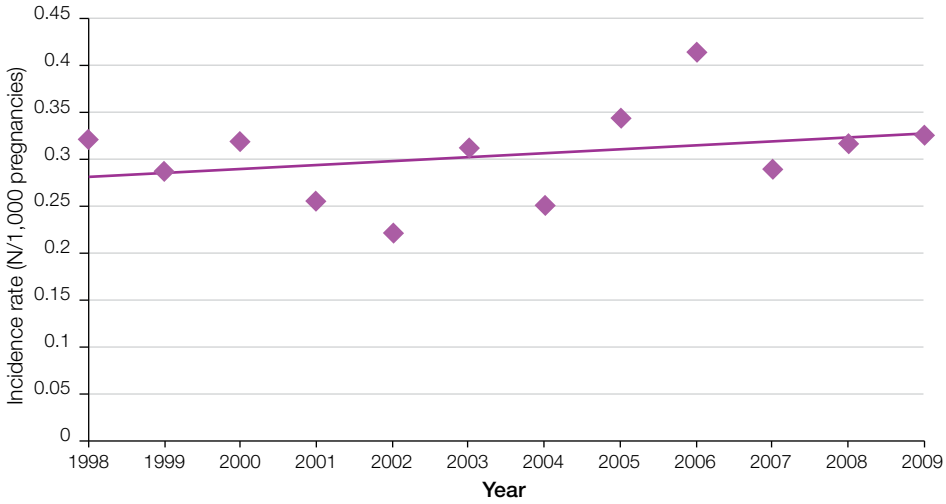



Table 22: Oesophageal atresia and/or stenosis by selected maternal and child characteristics, 2007–2009

| Characteristic | | Oesophageal atresia and/or stenosis/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.3 | - |
| | ≥ 35 years | 0.4 | 1.64 (1.003–2.68)* |
| **Plurality** | Single (Ref) | 0.3 | - |
| | Multiple | 0.5 | 1.80 (0.65–4.94) |
| **Gender** | Male (Ref) | 0.4 | - |
| | Female | 0.3 | 1.37 (0.84–2.22) |

*Statistically significant

### 4.8.16 Small intestinal atresia and/or stenosis

**Definition:** In small intestinal atresia and/or stenosis there is complete or partial occlusion of the lumen of a segment of the small intestine. It can involve a single area or multiple areas of the duodenum, jejunum or ileum.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 0.6 per cent (n = 58) had small intestinal atresia and/or stenosis, of which 41.4 per cent (n = 24) occurred as an isolated anomaly.

**Trend:** The incidence of small intestinal atresia and/or stenosis did not significantly change (p-value 0.897) from 1998 to 2009 (Figure 22).

**Risk factors:** The risk of small intestinal atresia and/or stenosis in babies did not vary with maternal age, birth plurality or gender of the baby (Table 23).

Figure 22: Small intestinal atresia and/or stenosis per 1,000 pregnancies, 1998 to 2009

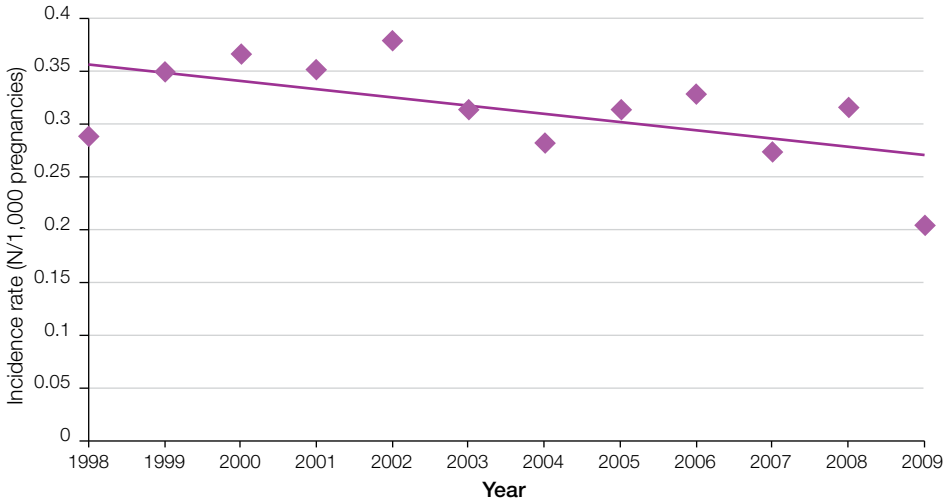



Table 23: Small intestinal atresia and/or stenosis by selected maternal and child characteristics, 2007–2009

| Characteristic | | Small intestinal atresia and/or stenosis/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.3 | - |
| | ≥ 35 years | 0.3 | 0.85 (0.48–1.50) |
| **Plurality** | Single (Ref) | 0.3 | - |
| | Multiple | 0.1 | 1.97(0.27–14.27) |
| **Gender** | Male (Ref) | 0.3 | - |
| | Female | 0.3 | 0.89 (0.53–1.49) |

### 4.8.17 Anorectal atresia and/or stenosis

**Definition:** The absence of continuity of the anorectal canal or communication between the rectum and anus, or narrowing of the anal canal, with or without fistula to neighbouring organs. Excludes ectopic anus.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 0.7 per cent (n = 69) had anorectal atresia and/or stenosis, of which 36.2 per cent (n = 25) occurred as an isolated anomaly.

**Trend:** The incidence of anorectal atresia and/or stenosis did not change significantly (p-value 0.266) from 1998 to 2009 (Figure 23).

**Risk factors:** The risk of anorectal atresia and/or stenosis in babies did not vary with maternal age, birth plurality or gender of the baby (Table 24).

Figure 23: Anorectal atresia and/or stenosis per 1,000 pregnancies, 1998 to 2009

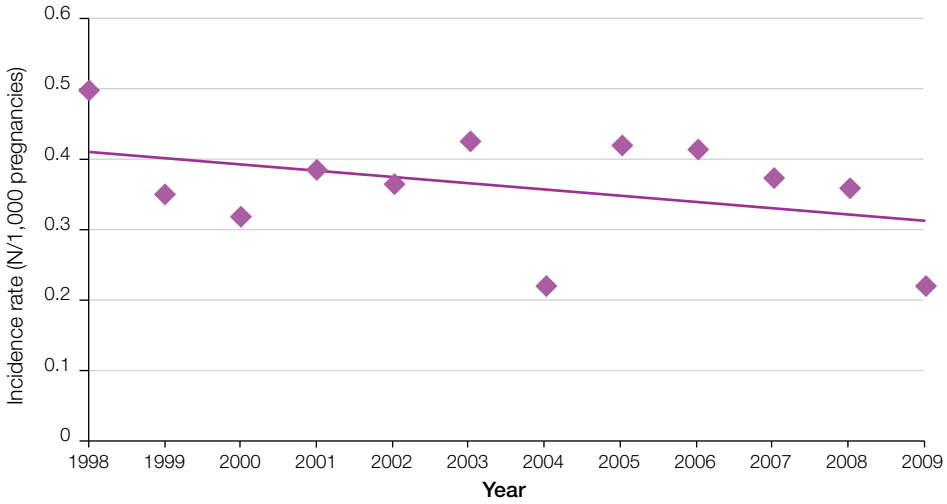



Table 24: Anorectal atresia and/or stenosis by selected maternal and child characteristics, 2007–2009

| Characteristic | | Anorectal atresia and/or stenosis/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.3 | - |
| | ≥ 35 years | 0.4 | 0.80 (0.48–1.35) |
| **Plurality** | Single (Ref) | 0.3 | - |
| | Multiple | 0.3 | 1.16 (0.28–4.74) |
| **Gender** | Male (Ref) | 0.3 | - |
| | Female | 0.3 | 1.17 (0.73–1.89) |

### 4.8.18 Hypospadias

**Definition:** Hypospadias is opening of the urethra on the ventral side of the penis, distally to the sulcus.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 9.2 per cent (n = 867) had hypospadias, of which 86.5 per cent (n = 750) occurred as an isolated anomaly.

**Trend:** The incidence of hypospadias increased significantly from 1998 to 2009 (p-value 0.008) (Figure 24).

**Risk factors:** The risk of hypospadias did not differ with maternal age or birth plurality (Table 25).

Figure 24: Hypospadias per 1,000 pregnancies, 1998 to 2009

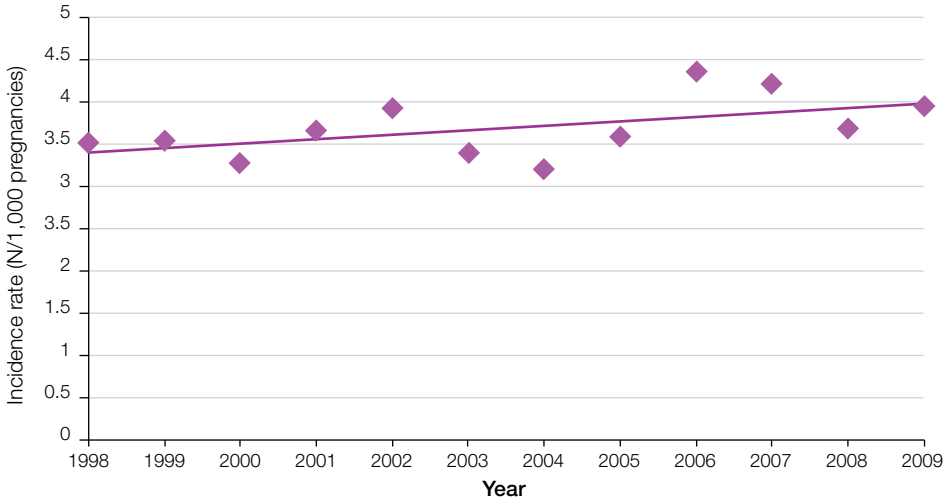



Table 25: Hypospadias by selected maternal and child characteristics, 2007–2009

| Characteristic | | Hypospadias/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 7.9 | - |
| | ≥ 35 years | 7.4 | 1.06 (0.91–1.24) |
| **Plurality** | Single (Ref) | 7.6 | - |
| | Multiple | 10.4 | 1.35 (0.98–1.86) |
| **Gender** | Male (Ref) | 7.7 | |
| | Female | N/A | N/A |

### 4.8.19 Renal agenesis and dysgenesis

**Definition:** This heterogeneous group includes bilateral or unilateral absence of the kidneys, or severe renal dysplasia.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 1.5 per cent (n = 141) had renal agenesis and dysgenesis, of which 45.4 per cent occurred as an isolated anomaly.

**Trend:** The incidence of renal agenesis and dysgenesis did not change significantly from 1998 to 2009 (p-value 0.909) (Figure 25).

**Risk factors:** The risk of renal agenesis and dysgenesis in babies did not differ with maternal age or birth plurality; however, male babies were 43.0 per cent more likely to develop renal agenesis and dysgenesis compared with female babies (Table 26).

Figure 25: Renal agenesis and dysgenesis per 1,000 pregnancies, 1998 to 2009

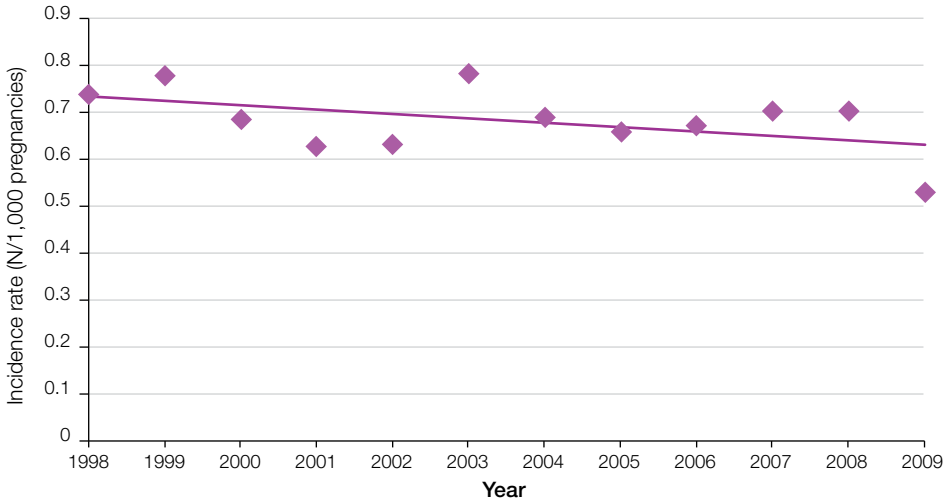



Table 26: Renal agenesis and dysgenesis by selected maternal and child characteristics, 2007–2009

| Characteristic | | Renal agenesis and dysgenesis/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.7 | - |
| | ≥ 35 years | 0.5 | 1.42 (0.94–2.16) |
| **Plurality** | Single (Ref) | 0.6 | - |
| | Multiple | 0.7 | 0.94 (0.38–2.30) |
| **Gender** | Male (Ref) | 0.8 | 1.43 (1.02–2.01)* |
| | Female | 0.5 | - |

*Statistically significant

### 4.8.20 Cystic kidney disease

**Definition:** Cystic kidney disease includes renal cysts of varying size and extent, occurring bilaterally or unilaterally. Polycystic and multicystic kidney diseases are both included.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 1.3 per cent (n = 125) had cystic kidney disease, of which 45.6 per cent (n = 57) occurred as an isolated anomaly.

**Trend:** The incidence of cystic kidney disease did not significantly (p-value 0.404) change from 1998 to 2009 (Figure 26).

**Risk factors:** The risk of cystic kidney disease in babies did not differ with maternal age or birth plurality; however, male babies were 60.0 per cent more likely to develop renal agenesis and dysgenesis compared with female babies (Table 27).

Figure 26: Cystic kidney disease per 1,000 pregnancies, 1998 to 2009

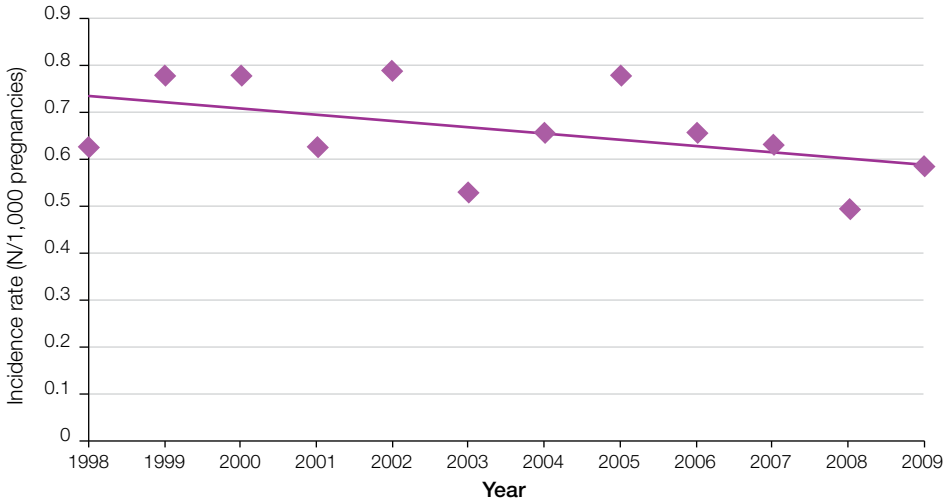



Table 27: Cystic kidney disease by selected maternal and child characteristics, 2007–2009

| Characteristic | | Cystic kidney disease/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.6 | - |
| | ≥ 35 years | 0.5 | 1.34 (0.87–2.07) |
| **Plurality** | Single (Ref) | 0.6 | - |
| | Multiple | 0.8 | 1.45 (0.64–3.29) |
| **Gender** | Male (Ref) | 0.7 | 1.60 (1.11–2.31)* |
| | Female | 0.4 | - |

*Statistically significant

### 4.8.21 Obstructive defects of the renal pelvis

**Definition:** This heterogeneous group includes hydronephrosis and any other defect that results in dilatation of the renal collecting system, bilaterally or unilaterally.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 10.1 per cent (n = 954) had obstructive defects of the renal pelvis, of which 76.4 per cent (n = 729) occurred as an isolated anomaly.

**Trend:** The incidence of obstructive defects of the renal pelvis significantly decreased in 2001 and 2006, increased in 2007 and 2008, and decreased again in 2009 (p-value < 0.001) (Figure 27).

**Risk factors:** Multiple births increased the risk of development of obstructive defects of renal pelvis in babies by 58.0 per cent when compared with singleton births. Male babies were 2.5 times more likely to develop obstructive defects of the renal pelvis than female babies (Table 28).

Figure 27: Obstructive defects of the renal pelvis per 1,000 pregnancies, 1998 to 2009

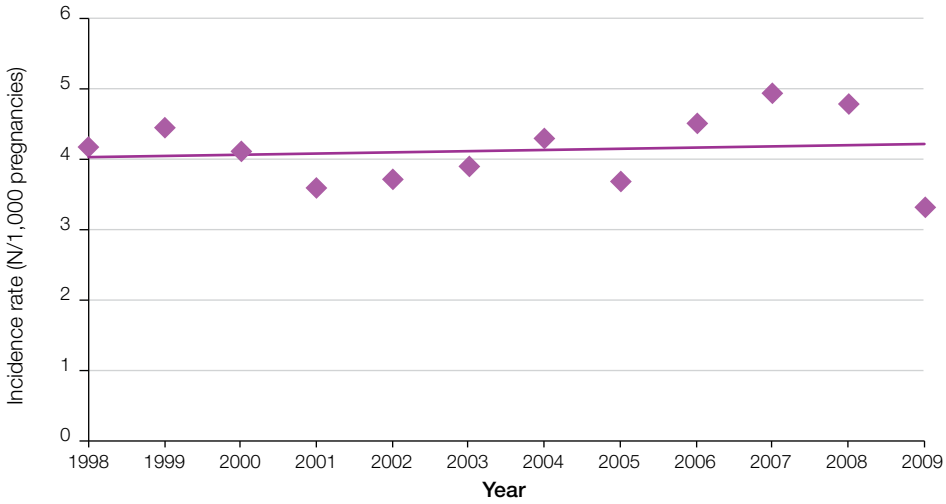



Table 28: Obstructive defects of the renal pelvis by selected maternal and child characteristics, 2007–2009

| Characteristic | | Obstructive defects of the renal pelvis/1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 4.4 | - |
| | ≥ 35 years | 4.1 | 1.05 (0.91–1.22) |
| **Plurality** | Single (Ref) | 4.3 | - |
| | Multiple | 6.7 | 1.58 (1.19–2.11)* |
| **Gender** | Male (Ref) | 6.2 | 2.52 (2.18–2.90)* |
| | Female | 2.5 | - |

*Statistically significant

### 4.8.22 Developmental dysplasia of the hip

**Definition:** Developmental dysplasia of the hip results in the femoral head being displaced (or displaceable) from the acetabulum of the pelvis. This should not be confused with 'clicky hips' which are not included in the VCAR.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 7.0 per cent (n = 661) had developmental dysplasia of the hip, of which 92.6 per cent (n = 612) occurred as isolated anomaly.

**Trend:** The incidence of developmental dysplasia of the hip has varied over time such that that incidence increased in 2003 and 2004, decreased in 2005, increased again in 2006 and 2007, and decreased significantly in 2009 (p-value < 0.001) (Figure 28).

**Risk factors:** The risk of developmental dysplasia of the hip in babies did not differ with maternal age or birth plurality; however, female babies were 3.6 times more likely to have developmental dysplasia of the hip compared with male babies (Table 29).

Figure 28: Developmental dysplasia of the hip per 1,000 pregnancies, 1998 to 2009

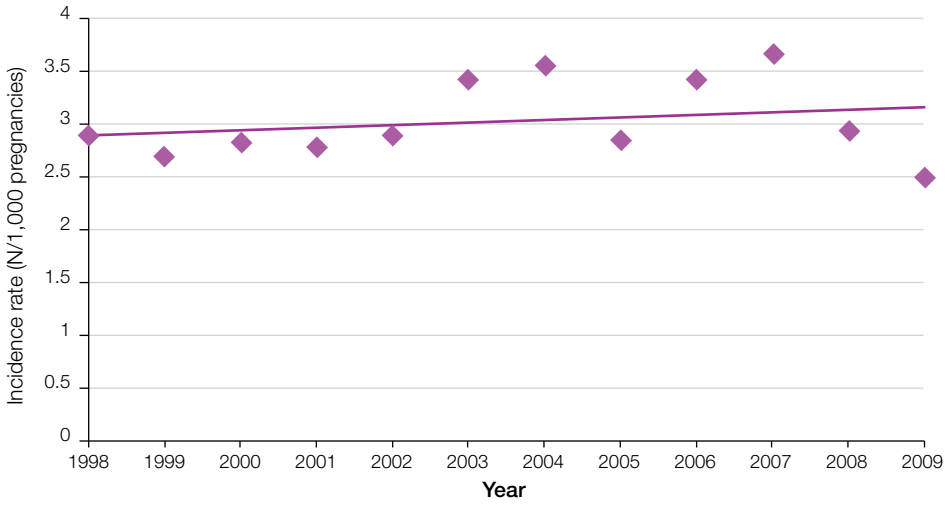



Table 29: Developmental dysplasia of the hip by selected maternal and child characteristics, 2007–2009

| Characteristic | | Developmental dysplasia of the hip/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 3.0 | - |
| | ≥ 35 years | 3.1 | 0.97 (0.82–1.15) |
| **Plurality** | Single (Ref) | 3.1 | - |
| | Multiple | 1.9 | 1.60 (0.94–2.71) |
| **Gender** | Male (Ref) | 1.3 | - |
| | Female | 4.9 | 3.66 (3.04–4.39)* |

*Statistically significant

### 4.8.23 Limb reduction defects

**Definition:** Limb reduction defects range in severity from partial absence of a phalanx to the complete absence of a major skeletal structure such as a whole limb.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 1.5 per cent (n = 143) had limb reduction defects, of which 34.9 per cent (n = 50) occurred as isolated anomaly.

**Trend:** The incidence of limb reduction defects did not change significantly (p-value 0.075) from 1998 to 2009 (Figure 29).

**Risk factors:** The risk of limb reduction defects in babies did not differ with maternal age or gender of the baby; however the risk increased 3.1 times in multiple births compared with singleton births (Table 30).

Figure 29: Limb reduction defects per 1,000 pregnancies, 1998 to 2009

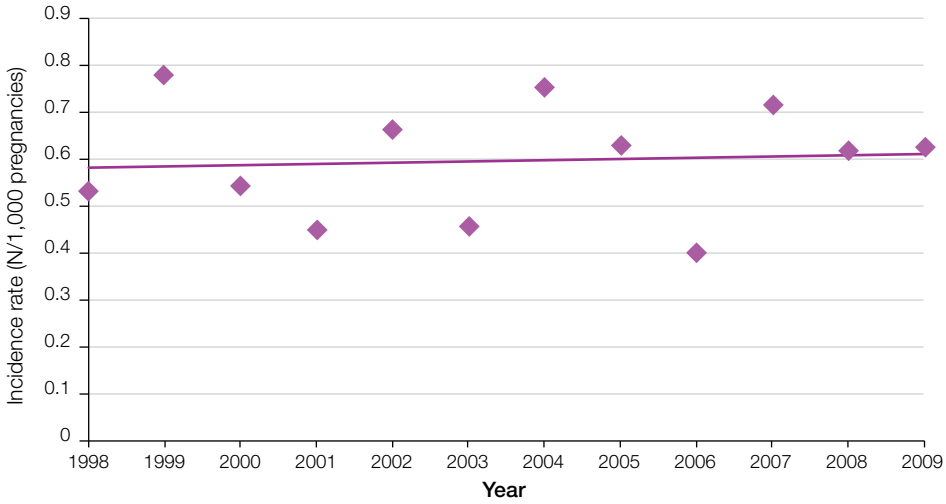



Table 30: Limb reduction defects by selected maternal and child characteristics, 2007–2009

| Characteristic | | Limb reduction defects/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.6 | - |
| | ≥ 35 years | 0.7 | 0.94 (0.65–1.36) |
| **Plurality** | Single (Ref) | 0.6 | - |
| | Multiple | 1.9 | 3.12 (1.80–5.41)* |
| **Gender** | Male (Ref) | 0.7 | - |
| | Female | 0.5 | 1.33 (0.94–1.86) |

*Statistically significant

### 4.8.24 Diaphragmatic hernia

**Definition:** In diaphragmatic hernia, abdominal organs herniate into the thorax through a defect of the diaphragm.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 0.9 per cent (n = 88) had diaphragmatic hernia, of which 37.5 per cent (n = 33) occurred as isolated anomaly.

**Trend:** The incidence of diaphragmatic hernia did not change significantly (p-value 0.541) from 1998 to 2009 (Figure 30).

**Risk factors:** The risk of development of diaphragmatic hernia in a baby did not vary with maternal age, birth plurality or gender of the baby (Table 31).

Figure 30: Diaphragmatic hernia per 1,000 pregnancies, 1998 to 2009

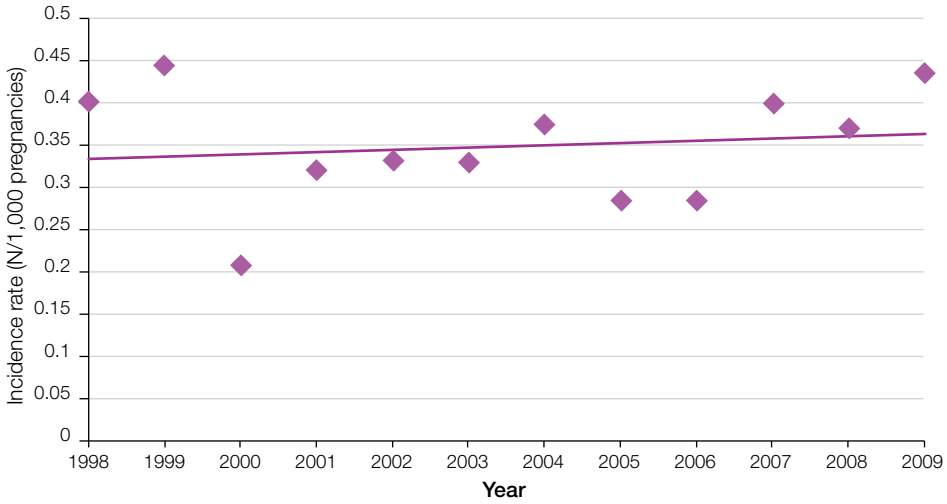



Table 31: Diaphragmatic hernia by selected maternal and child characteristics, 2007–2009

| Characteristic | | Diaphragmatic hernia/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.4 | - |
| | ≥ 35 years | 0.4 | 0.94 (0.59–1.50) |
| **Plurality** | Single (Ref) | 0.4 | - |
| | Multiple | 0.7 | 1.73 (0.70–4.27) |
| **Gender** | Male (Ref) | 0.4 | - |
| | Female | 0.4 | 1.05 (0.69–1.59) |

### 4.8.25 Exomphalos

**Definition:** Exomphalos is the herniation of abdominal contents through the umbilical insertion and covered by membrane which may or may not be intact.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 0.8 per cent (n = 80) had exomphalos, of which 17.5 per cent (n = 14) occurred as an isolated anomaly.

**Trend:** The incidence of exomphalos significantly decreased (p-value 0.014) from 1998 to 2009 (Figure 31).

**Risk factors:** The risk of development of exomphalos in a baby did not vary with maternal age, birth plurality or gender of the baby (Table 31).

Figure 31: Exomphalos per 1,000 pregnancies, 1998 to 2009

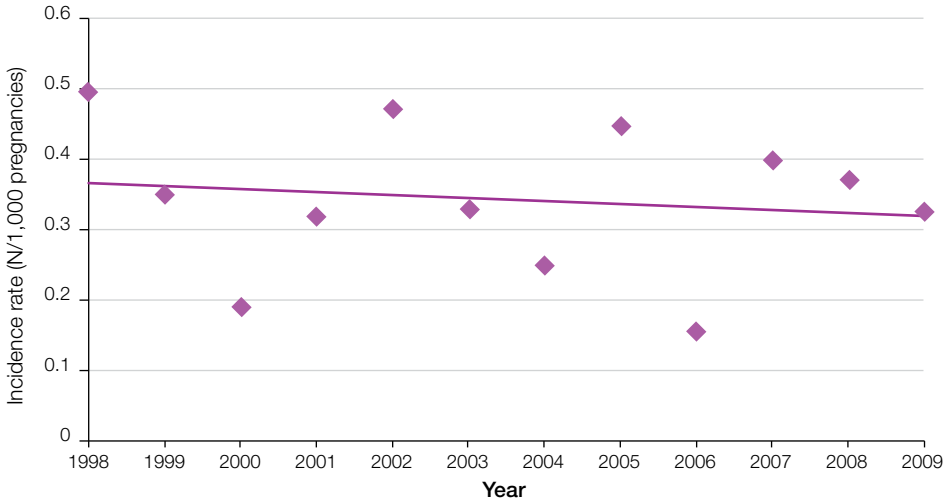



Table 32: Exomphalos by selected maternal and child characteristics, 2007–2009

| Characteristic | | Exomphalos/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.3 | - |
| | ≥ 35 years | 0.4 | 1.56 (0.96–2.55) |
| **Plurality** | Single (Ref) | 0.4 | - |
| | Multiple | 0.5 | 1.53 (0.56–4.20) |
| **Gender** | Male (Ref) | 0.3 | - |
| | Female | 0.4 | 1.34 (0.84–2.14) |

### 4.8.26 Gastroschisis

**Definition:** Gastroschisis is the visceral herniation through an abdominal wall defect lateral to an intact umbilical cord, and not covered by a membrane.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 0.5 per cent (n = 51) had gastroschisis, of which 66.7 per cent (n = 34) occurred as an isolated anomaly.

**Trend:** The incidence of gastroschisis did not change significantly (p-value 0.761) from 1998 to 2009 (Figure 32).

**Risk factors:** The risk of development of gastroschisis in babies varied with maternal age, with women younger than 35 years at 2.2 times greater risk as compared to women greater than 35 years of age, and women younger than 20 years at 10 times greater risk than women aged 20 years and older (RR 10.2, 95 per cent CI 5.2 – 19.8). The risk of gastroschisis did not vary with birth plurality and gender (Table 33).

Figure 32: Gastroschisis per 1,000 pregnancies, 1998 to 2009

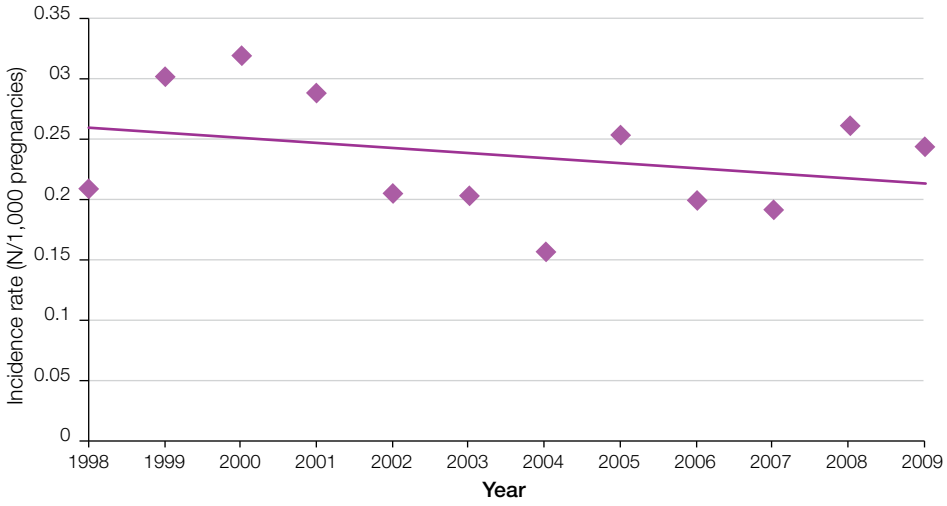



Table 33: Gastroschisis by selected maternal and child characteristics, 2007–2009

| Characteristic | | Gastroschisis/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.3 | 2.22 (1.002–4.93)* |
| | ≥ 35 years | 0.1 | - |
| **Plurality** | Single (Ref) | 0.2 | - |
| | Multiple | 0.0 | 3.57 (0.22–57.89) |
| **Gender** | Male (Ref) | 0.2 | - |
| | Female | 0.2 | 1.23 (0.69–2.18) |

*Statistically significant

### 4.8.27 Trisomy 21

**Definition:** Trisomy 21, also called Down's syndrome, is characterised by an additional chromosome 21.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 7.8 per cent (n = 740) had trisomy 21, of which 64.2 per cent were terminated before 20 weeks of gestation.

**Trend:** The incidence of trisomy 21 increased significantly (p-value < 0.001) from 1998 to 2009 (Figure 33). This is similar to incidence rates observed in South Australia[24] and Western Australia[25]. The incidence of trisomy 21 has declined in Queensland[26] and remained stable in New South Wales[27].

**Risk factors:** The risk of trisomy 21 in babies of women aged 35 years and older was 5.3 times higher than for babies of women younger than 35 years of age. The risk of trisomy 21 did not vary by birth plurality and gender of the baby (Table 34).

Figure 33: Trisomy 21 per 1,000 pregnancies, 1998 to 2009

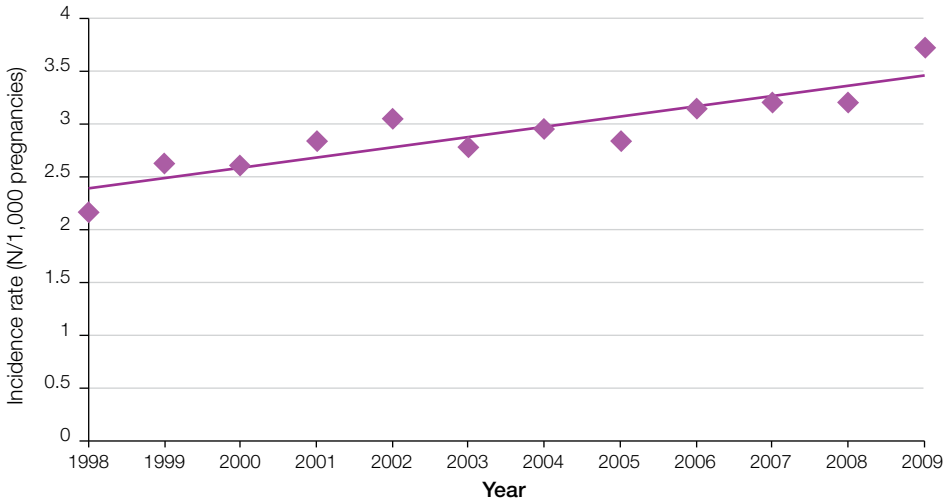



Table 34: Trisomy 21 by selected maternal and child characteristics, 2007–2009

| Characteristic | | Trisomy 21/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 1.2 | - |
| | ≥ 35 years | 6.6 | 5.33 (4.49–6.33)* |
| **Plurality** | Single (Ref) | 3.1 | - |
| | Multiple | 2.3 | 1.31 (0.81–2.12) |
| **Gender** | Male (Ref) | 3.5 | - |
| | Female | 3.2 | 1.08 (0.94–1.25) |

*Statistically significant

### 4.8.28 Trisomy 13

**Definition:** Trisomy 13, also called Patau's syndrome, is characterised by an additional chromosome 13.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 0.9 per cent (n = 82) had trisomy 13, of which 70.7 per cent were terminated before 20 weeks of gestation.

**Trend:** The incidence of trisomy 13 did not change significantly (p-value 0.664) from 1998 to 2009 (Figure 34).

**Risk factors:** Women greater than 35 years of age were almost twice as likely to have a baby with trisomy 13 as compared to women younger than 35 years of age. The risk of trisomy 13 did not vary with birth plurality and gender of the baby (Table 35).

Figure 34: Trisomy 13 per 1,000 pregnancies, 1998 to 2009

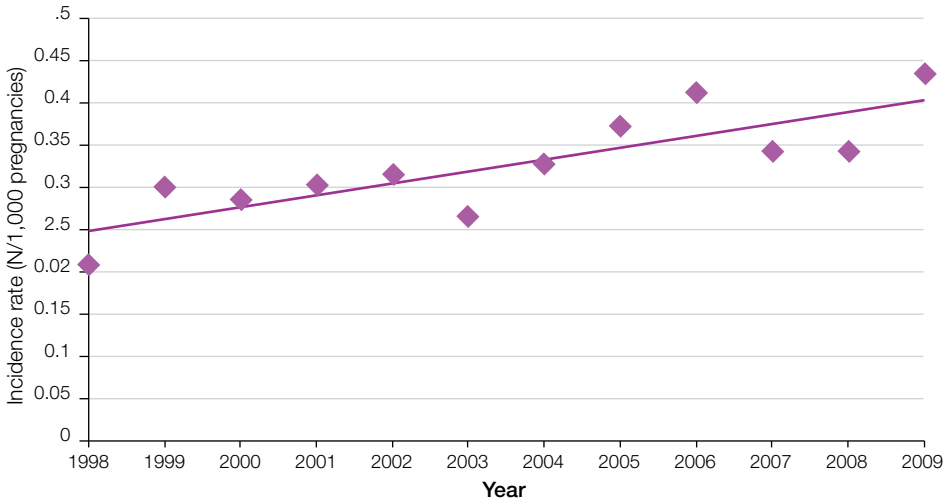



Table 35: Trisomy 13 by selected maternal and child characteristics, 2007–2009

| Characteristic | | Trisomy 13/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.2 | - |
| | ≥ 35 years | 0.4 | 1.91 (1.14–3.16)* |
| **Plurality** | Single (Ref) | 0.3 | - |
| | Multiple | 0.1 | 2.35 (0.32–16.98) |
| **Gender** | Male (Ref) | 0.3 | - |
| | Female | 0.4 | 1.12 (0.72–1.74) |

*Statistically significant

### 4.8.29 Trisomy 18

**Definition:** Trisomy 18, also called Edward's syndrome, is characterised by an additional chromosome 18.

**Incidence:** Of all the congenital anomaly cases reported from 2007 to 2009, 2.5 per cent (n = 238) had trisomy 18, of which 76.5 per cent were terminated before 20 weeks of gestation.

**Trend:** The incidence of trisomy 18 increased significantly (p-value 0.001) from 1998 to 2009 (Figure 35).

**Risk factors:** The risk of trisomy 18 in babies of mothers aged 35 years and older was 6.7 times the risk in babies of women younger than 35 years of age. Female babies were 36.0 per cent more likely to develop trisomy 18 than male babies. The risk of trisomy 18 did not vary by birth plurality (Table 36).

Figure 35: Trisomy 18 per 1,000 pregnancies, 1998 to 2009

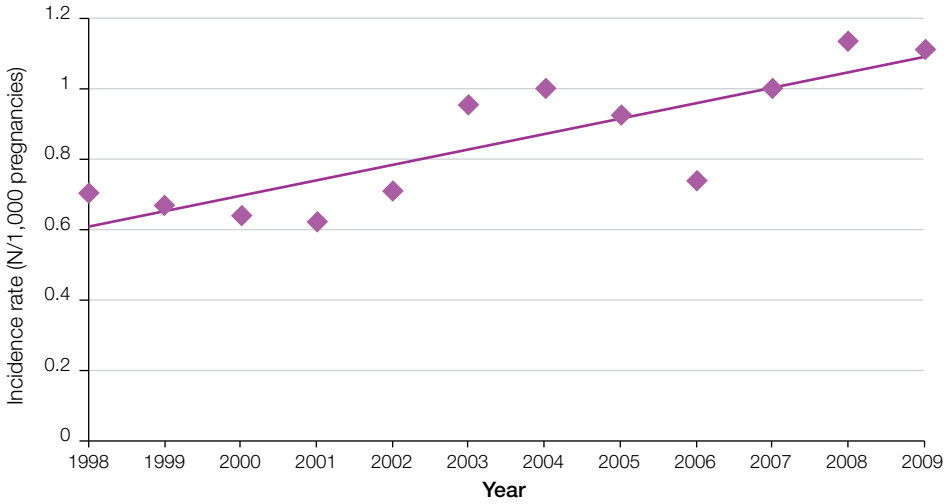



Table 36: Trisomy 18 by selected maternal and child characteristics, 2007–2009

| Characteristic | | Trisomy 18/ 1,000 pregnancies | RR and 95 per cent CI |
|---|---|---|---|
| **Mother's age** | < 35 years (Ref) | 0.3 | - |
| | ≥ 35 years | 2.0 | 6.73 (4.82–9.39)* |
| **Plurality** | Single (Ref) | 0.9 | - |
| | Multiple | 0.5 | 1.73 (0.64–4.66) |
| **Gender** | Male (Ref) | 0.9 | - |
| | Female | 1.2 | 1.36 (1.050–1.76)* |

*Statistically significant

# Appendix A: Major congenital anomalies in Victoria, 2007–2009

The following figures refer to individual congenital anomalies, not cases. The codes are compatible with the British Paediatric Association (BPA) Classification of Diseases Supplement to ICD – 9th Revision. As any one case may have two or more conditions within a particular code range, the number of individual congenital anomalies may exceed the number of cases.

| Defect | Number | N/1,000 pregnancies |
|---|---|---|
| **Nervous system** | | |
| Anencephalus¥ (7400*) | 96 | 0.4 |
| Craniorachischisis (7401*) | < 5 | 0.0 |
| Iniencephaly (7402*) | < 5 | 0.0 |
| Spina Bifida (741**) | 114 | 0.5 |
| Encephalocele (7420*) | 33 | 0.2 |
| Microcephalus (7421*) | 61 | 0.3 |
| Brain reduction (7422*) | 178 | 0.8 |
| Hydrocephalus (7423*) | 194 | 0.9 |
| Other specified anomalies of brain (74240–74299) | 149 | 0.7 |
| Hereditary and degenerative diseases (33000–33799) | 21 | 0.1 |
| Cerebral palsy (34300–34399) | 7 | 0.0 |
| Other disorders of CNS (3499*, 34000–34299) | 0 | 0.0 |
| Disorders of Peripheral NS (35000–35999) | 18 | 0.1 |
| **Total** | **875** | **4.0** |
| **Eye** | | |
| Anophthalmos (7430*) | < 5 | 0.0 |
| Microphthalmos (7431*) | 20 | 0.1 |
| Buphthalmos (7432*) | 11 | 0.1 |
| Other lens (74330–74331, 74333–74339) | < 5 | 0.0 |
| Cataract (74332) | 32 | 0.1 |
| Other specified anomalies of eye (74340–74399) | 45 | 0.2 |
| **Total** | **110** | **0.5** |

| Defect | Number | N/1,000 pregnancies |
|---|---|---|
| **Ear, face and neck** | | |
| Ear-affecting hearing (7440*) | 21 | 0.1 |
| Auditory canal (74400) | 15 | 0.1 |
| Absent auricle (74401) | < 5 | 0.0 |
| Other specified anomalies of the ear (74402–74409) | 5 | 0.0 |
| Other ear (74410–74439) | 74 | 0.3 |
| Accessory auricle (74410–74419) | 20 | 0.1 |
| Microtia (74421) | 16 | 0.1 |
| Other ear (74420, 74422–74429) | 36 | 0.2 |
| Face and neck (74440–74499) | 110 | 0.5 |
| Deafness (389**) | 32 | 0.1 |
| **Total** | **258** | **1.2** |
| **Cardiovascular system** | | |
| Common truncus (7450*) | 19 | 0.1 |
| Transposition of great vessels (7451*) | 141 | 0.6 |
| Tetralogy/Pentalogy of Fallot (7452*) | 77 | 0.4 |
| Common ventricle (7453*) | 22 | 0.1 |
| Ventricular septal defect (7454*) | 673 | 3.1 |
| Atrial septal defect (7455*) | 346 | 1.6 |
| Endocardial cushion (7456*) | 93 | 0.4 |
| Other specified anomalies of cardiac septal closure (74570–74599) | 6 | 0.0 |
| Cardiomyopathy (425**) | 8 | 0.0 |
| Conduction disorder (426**) | < 5 | 0.0 |
| Pulmonary valve (7460*) | 147 | 0.7 |
| Atresia (74600) | 26 | 0.1 |
| Stenosis (74601) | 111 | 0.5 |
| Other (74602–74609) | 10 | 0.0 |
| Tricuspid atresia/stenosis (7461*) | 100 | 0.5 |
| Ebstein anomaly (7462*) | 14 | 0.1 |
| Aortic valve stenosis/insufficiency (74630–74649) | 63 | 0.3 |
| Mitral stenosis/insufficiency (74650–74669) | 35 | 0.2 |
| Hypoplastic left heart syndrome (7467*) | 88 | 0.4 |
| Other specified anomalies of heart (7468*) | 160 | 0.7 |
| Unspecified (7469*) | 125 | 0.6 |
| Patent ductus arteriosus (74701) | 421 | 1.9 |
| Coarctation of aorta (7471*) | 98 | 0.4 |

| Defect | Number | N/1,000 pregnancies |
|---|---|---|
| Other aorta (7472*) | 76 | 0.3 |
| Pulmonary artery (7473*) | 137 | 0.6 |
| Great veins (7474*) | 54 | 0.2 |
| Peripheral vascular (7476*) | 9 | 0.0 |
| Other specified anomalies of circulatory system (7478*) | 16 | 0.1 |
| Unspecified (7479*) | 0 | 0.0 |
| **Total** | **2929** | **13.3** |
| **Respiratory system** | | |
| Choanal atresia (7480*) | 36 | 0.2 |
| Other nose (7481*) | 35 | 0.2 |
| Larynx/trachea/bronchus (74820–74839) | 139 | 0.6 |
| Lung (74840–74869) | 83 | 0.4 |
| Other specified anomalies of respiratory system (74880–74899) | < 5 | 0.0 |
| **Total** | **294** | **1.3** |
| **Gastrointestinal tract** | | |
| Cleft palate (7490*) | 166 | 0.8 |
| Cleft lip (7491*) | 94 | 0.4 |
| Cleft lip and palate (7492*) | 142 | 0.6 |
| Oesophageal atresia/stenosis (7503*) | 68 | 0.3 |
| Other specified anomalies of upper ailmentary tract (75010–75029, 75040–75099) | 193 | 0.9 |
| Meckels diverticulum (7510*) | < 5 | 0.0 |
| Small intestine atresia/stenosis (7511*) | 61 | 0.3 |
| Duodenal atresia/stenosis (75110) | 38 | 0.2 |
| Jejunal atresia/stenosis (75111) | 13 | 0.1 |
| Ileal atresia/stenosis (75112) | 6 | 0.0 |
| Unspecified atresia/stenosis (75119) | < 5 | 0.0 |
| Large intestine rectal anal atresia/stenosis (7512*) | 73 | 0.3 |
| Large intestine atresia/stenosis (75120) | < 5 | 0.0 |
| Rectal atresia/stenosis (75121–75122) | < 5 | 0.0 |
| Anal atresia/stenosis (75123–75124) | 67 | 0.3 |
| Hirschsprung's disease (7513*) | 37 | 0.2 |
| Intestinal fixation (7514*) | 40 | 0.2 |
| Other specified anomalies of intestine (75150–75199) | 94 | 0.4 |
| Other specified dentofacial anomalies (52400–57999) | 44 | 0.2 |
| **Total** | **1013** | **4.6** |

| Defect | Number | N/1,000 pregnancies |
|---|---|---|
| **Urogenital system** | | |
| Ovaries/fallopian etc (75200–75219) | 11 | 0.1 |
| Uterus (75220–75239) | 10 | 0.0 |
| Cervix/vagina/external genitalia (7524*) | 14 | 0.1 |
| Undescended testes ¶ (7525*) | 89 | 0.4 |
| Hypospadias (75260, 75263–75265) | 867 | 4.0 |
| Epispadias (75261) | 12 | 0.1 |
| Chordee (75262) | 57 | 0.3 |
| Indeterminate sex (7527*) | 27 | 0.1 |
| Other specified anomalies of genital organs (7528*) | 94 | 0.4 |
| Unspecified (7529*) | < 5 | 0.0 |
| Renal agenesis/dysgenesis (7530*) | 141 | 0.6 |
| Bilateral (75300) | 32 | 0.1 |
| Unilateral (75301) | 109 | 0.5 |
| Cystic kidney disease (7531*) | 125 | 0.6 |
| Polycystic (75311–75313) | 35 | 0.2 |
| Multicystic (75316) | 87 | 0.4 |
| Other (75310, 75314, 75318) | < 5 | 0.0 |
| Obstructive defects renal pelvis/ureter (7532*) | 994 | 4.5 |
| Hydronephrosis (75320) | 892 | 4.1 |
| Other (75321–75329) | 102 | 0.5 |
| Other specified kidney disorders (7533*) | 202 | 0.9 |
| Other specified disorders of ureter (7534*) | 29 | 0.1 |
| Exstrophy of bladder (7535*) | < 5 | 0.0 |
| Urethra bladder neck atresia/stenosis (7536*) | 46 | 0.2 |
| Urachus (7537*) | < 5 | 0.0 |
| Other bladder/urethra (7538*) | 28 | 0.1 |
| Unspecified (7539*) | 20 | 0.1 |
| Other specified anomalies of genito-urinary system (59200–60899) | 9 | 0.0 |
| **Total** | **2,782** | **12.7** |

| Defect | Number | N/1,000 pregnancies |
|---|---|---|
| **Musculoskeletal system** | | |
| Of skull, face and jaw (7540*) | 40 | 0.2 |
| Of sternocleidomastoid (7541*) | 0 | 0.0 |
| Of spine (7542*) | < 5 | 0.0 |
| Congenital dislocation of hip (75430) | 661 | 3.0 |
| Other hip (75431–75432) | < 5 | 0.0 |
| Genu recurvatum/bowing (7544*) | 10 | 0.0 |
| Of feet (75450–75479) | 549 | 2.5 |
| Other specified musculoskeletal anomalies (7548*) | 7 | 0.0 |
| Polydactyly (7550*) | 303 | 1.4 |
| Syndactyly (7551*) | 97 | 0.4 |
| Reduction deformities upper limb (7552*) | 101 | 0.5 |
| Reduction deformities lower limb (7553*) | 64 | 0.3 |
| Reduction deformities unspecified limb (7554*) | 7 | 0.0 |
| Other upper limb (7555*) | 111 | 0.5 |
| Other lower limb (7556*) | 124 | 0.6 |
| Arthrogryposis Multiplex Congenita (75580) | 14 | 0.1 |
| Other specified anomalies of unspecified limb (75581–75588) | 16 | 0.1 |
| Unspecified (7559*) | 10 | 0.0 |
| Skull, Face and Bones (7560*) | 159 | 0.7 |
| Craniosynostosis (75600) | 78 | 0.4 |
| Other (75601–75602, 75604–75609) | 52 | 0.2 |
| Pierre Robin Syndrome (75603) | 29 | 0.1 |
| Spine (7561*) | 108 | 0.5 |
| Ribs and sternum (75620–75639) | 36 | 0.2 |
| Chondrodystrophy (7564*) | 34 | 0.2 |
| Achondroplasia (75643) | 12 | 0.1 |
| Other dwarfing (75644) | 8 | 0.0 |
| Other (75645–75649) | 13 | 0.1 |
| Osteodystrophies (7565*) | 38 | 0.2 |
| Osteogenesis (75650) | 14 | 0.1 |
| Other (75651–75659) | 24 | 0.1 |
| Diaphragm (7566*) | 92 | 0.4 |
| Other (75660, 75662–75669) | < 5 | 0.0 |
| Diaphragmatic Hernia (75661) | 88 | 0.4 |

| Defect | Number | N/1,000 pregnancies |
|---|---|---|
| Abdominal wall (7567*) | 140 | 0.6 |
| Exomphalos (75670) | 80 | 0.4 |
| Gastroschisis (75671) | 51 | 0.2 |
| Other (75672–75679) | 9 | 0.0 |
| Other specified of muscle/tendon/fascia (7568*) | 17 | 0.1 |
| Unspecified (7569*) | 0 | 0.0 |
| **Total** | **2,744** | **12.5** |
| **Integumentary system** | | |
| Hereditary oedema (7570*) | < 5 | 0.0 |
| Icthyosis congenita (7571*) | 12 | 0.1 |
| Dermatoglyphic anomalies (7572*) | 0 | 0.0 |
| Other specified anomalies skin (7573*) | 130 | 0.6 |
| Specified anomalies of hair (7574*) | 0 | 0.0 |
| Specified anomalies of nails (7575*) | < 5 | 0.0 |
| Specified anomalies of breast (7576*) | < 5 | 0.0 |
| Other specified anomalies of integument (7578*) | < 5 | 0.0 |
| Unspecified (7579*) | < 5 | 0.0 |
| **Total** | **150** | **0.7** |
| **Chromosomal** | | |
| Trisomy 21 (7580*) | 740 | 3.4 |
| Trisomy 13 (7581*) | 82 | 0.4 |
| Trisomy 18 (7582*) | 238 | 1.1 |
| Autosomal deletion (7583*) | 73 | 0.3 |
| Other autosomal (7585*) | 116 | 0.5 |
| Turner's Syndrome (7586*) | 70 | 0.3 |
| Klinefelter's Syndrome (7587*) | 23 | 0.1 |
| Other sex chromosomes (7588*) | 42 | 0.2 |
| Unspecified (7589*) | 50 | 0.2 |
| **Total** | **1,434** | **6.5** |

| Defect | Number | N/1,000 pregnancies |
|---|---|---|
| **Neoplasms** | | |
| Malignant (14000–20899) | 17 | 0.1 |
| Benign (2–22999) | 102 | 0.5 |
| Haemangioma (22809) | 12 | 0.1 |
| Cystic hygroma (22819) | 88 | 0.4 |
| Other (21000–22799) | < 5 | 0.0 |
| Uncertain behaviour (23500–23999) | 30 | 0.1 |
| **Total** | **149** | **0.7** |
| **Metabolic/ endocrine/nutritional** | | |
| Congenital hypothyroidism (2439*) | 84 | 0.4 |
| Other endocrine glands (25000–25999) | 15 | 0.1 |
| Nutritional deficiencies (26000–26999) | < 5 | 0.0 |
| Phenylketonuria (2701*) | 27 | 0.1 |
| Other disorders of amino-acid metabolism (27000, 27020–27099) | 37 | 0.2 |
| Of carbohydrate metabolism (271**) | 12 | 0.1 |
| Of lipid metabolism (272**) | 8 | 0.0 |
| Of plasma protein metabolism (273**) | < 5 | 0.0 |
| Of mineral metabolism (275**) | 9 | 0.0 |
| Cystic fibrosis (2770*) | 64 | 0.3 |
| Other metabolism (27710–27799) | 40 | 0.2 |
| Of immunity (279**) | 16 | 0.1 |
| **Total** | **316** | **1.4** |
| **Haematological** | | |
| Hereditary haemolytic anaemia (282**) | 38 | 0.2 |
| Other anaemias (28100, 28300–28599) | < 5 | 0.0 |
| Coagulation defects (286**) | 11 | 0.1 |
| Other haemorrhagic conditions (28700–28999) | 0 | 0.0 |
| **Total** | **50** | **0.2** |

| Defect | Number | N/1,000 pregnancies |
|---|---|---|
| **Others** | | |
| Spleen (7590*) | 18 | 0.1 |
| Adrenal gland (7591*) | 10 | 0.0 |
| Other endocrine gland (7592*) | 17 | 0.1 |
| Situs inversus (7593*) | 17 | 0.1 |
| Conjoined twins (7594*) | < 5 | 0.0 |
| Tuberous sclerosus (7595*) | 10 | 0.0 |
| Harmartoses nec (7596*) | 7 | 0.0 |
| Multiple so described (7597*) | 10 | 0.0 |
| Other specified syndromes (7598*) | 183 | 0.8 |
| Unspecified (7599*) | 42 | 0.2 |
| Fetal alcohol syndrome (76076) | < 5 | 0.0 |
| Maternal conditions (76079) | 0 | 0.0 |
| Congenital infection (77100–77129) | 40 | 0.2 |
| Toxoplasmosis (77121) | 0 | 0.0 |
| Cytomegalovirus (77119) | 31 | 0.1 |
| Other (77100, 77120–77199) | 6 | 0.0 |
| Hydrops fetalis (non-immune) (7780*) | 100 | 0.5 |
| Developmental delay (7834*) | 0 | 0.0 |
| **Total** | **466** | **2.1** |

* Denotes any number from 0 to 9

Undescended testes in term babies is included in VCAR, but isolated cases have been excluded for the purposes of this report as a minor malformation. This figure refers to cases of undescended testes which are not isolated but occur with other defects.

¥ Anencephalus includes absence of brain, acrania, anencephaly and hemianencephaly.

Denominators do not modify for sex specific conditions (e.g. undescended testes).

# Appendix B: Excluded congenital anomalies

There has been variation in the list of exclusions between 1983 and 2002. Some excluded conditions may be included in this report if they were previously not excluded and occur with other congenital anomalies.

| | |
|---|---|
| Abnormal palmar creases | Macroglossia (large tongue) |
| Accessory nipples | Meckel's diverticulum |
| Anal fissure | Meconium ileus |
| Balanced autosomal translocation (unless occurring with structural defects) | Mental retardations (unless occurring with a syndrome/ structural defect) |
| Birth injuries | Metatarsus varus |
| Birth marks (smaller than 4cm, not including giant naevus) | Micrognathia (unless severe) |
| Bowing of legs (unless severe) | Mongolian spots |
| Blocked tear ducts (dacrostenosis) | Occiput, flat/prominent |
| Brushfield spots | Patent ductus arteriosus (< 37 weeks) |
| Cephalhaematoma | Philtrum, long/short |
| Cleft gum | Plagiocephaly |
| Clicky hips | Pre-auricular sinus |
| Clinodactyly | Prominent forehead |
| Craniotabes (unless severe) | Protruding tongue |
| Dermatogplyphic abnormalities | Ptosis |
| Ear abnormalities (minor) | Retrognathia (unless severe) |
| Epicanthic folds | Rocker-bottom feet (prominent heels) |
| Gastro-oesophageal reflux | Sacral pits, dimples, sinuses |
| Haemangioma (< 4 cm wide) | Short sternum |
| Hernia – inguinal, umbilical | Simian creases |
| High-arched palate | Single umbilical artery/2 vessels in cord |
| Hydrocele | Skin folds/tags |
| Hypertelorism | Slanting eyes |
| Imperforate hymen | Small mouth |
| Laryngeal stridor | Laryngomalacia |
| Low slung/set ears | |

# Appendix C: Routine data items in the Victorian Congenital Anomalies Register

**Maternal data:** postcode, date of birth, method of prenatal diagnosis

**Child/fetus data:** hospital of birth, date of birth (or termination), sex, birth weight, plurality, rank, condition of birth (termination before 20 weeks, termination ≥ 20 weeks, stillbirth, live birth), date of death (if applicable), BPA Codes for congenital defects, position code, source of notification

Other data items available from linkage to the Perinatal Morbidity Statistics Form (Victorian perinatal data collection):

**Maternal items:** UR number, local government area, region, country of birth, Aboriginality, discharge date and status, marital status, number of previous pregnancies, date of completion of last pregnancy, outcome of last pregnancy, maternal medical conditions, obstetric complications, indication(s) for operative delivery, complications of labour birth and postnatal, procedures and operations, type of labour, presentation, method of delivery

**Child data items:** APGAR, time to establish respiration, resuscitation methods, neonatal morbidity

# Appendix D: Congenital anomalies by year, 1983–2009

| Year | Total births, 20 weeks and later | Defects, 20 weeks and later | Defects before 20 weeks (terminations) | N/1,000 pregnancies | Percentage |
|---|---|---|---|---|---|
| 1983 | 60,628 | 1,653 | < 5 | 27.3 | 2.7 |
| 1984 | 60,737 | 1,691 | 9 | 27.9 | 2.8 |
| 1985 | 61,189 | 1,591 | 18 | 26.2 | 2.6 |
| 1986 | 61,253 | 1,604 | 80 | 27.4 | 2.7 |
| 1987 | 61,566 | 1,620 | 55 | 27.1 | 2.7 |
| 1988 | 63,666 | 1,874 | 103 | 31.0 | 3.1 |
| 1989 | 64,255 | 1,930 | 123 | 31.8 | 3.2 |
| 1990 | 66,878 | 2,164 | 132 | 34.2 | 3.4 |
| 1991 | 65,248 | 2,235 | 140 | 36.3 | 3.6 |
| 1992 | 66,305 | 2,295 | 152 | 36.8 | 3.7 |
| 1993 | 64,737 | 2,250 | 203 | 37.7 | 3.8 |
| 1994 | 64,932 | 2,295 | 250 | 39.0 | 3.9 |
| 1995 | 63,717 | 2,444 | 257 | 42.2 | 4.2 |
| 1996 | 62,951 | 2,217 | 272 | 39.3 | 3.9 |
| 1997 | 62,308 | 2,306 | 297 | 41.5 | 4.2 |
| 1998 | 62,091 | 2,332 | 274 | 41.7 | 4.2 |
| 1999 | 62,690 | 2,555 | 294 | 45.2 | 4.5 |
| 2000 | 62,564 | 2,614 | 292 | 46.2 | 4.6 |
| 2001 | 62,149 | 2,394 | 308 | 43.2 | 4.3 |
| 2002 | 63,133 | 2,520 | 327 | 44.8 | 4.5 |
| 2003 | 63,552 | 2,690 | 356 | 47.6 | 4.8 |
| 2004 | 63,700 | 2,774 | 342 | 48.6 | 4.9 |
| 2005 | 66,654 | 2,500 | 339 | 42.3 | 4.2 |
| 2006 | 69,856 | 2,836 | 366 | 45.6 | 4.6 |
| 2007 | 72,474 | 3,013 | 364 | 46.3 | 4.6 |
| 2008 | 72,545 | 2,630 | 360 | 41.0 | 4.1 |
| 2009 | 73,264 | 2,664 | 414 | 41.7 | 4.2 |
| **Total** | **1,745,042** | **61,691** | **6,129** | **38.86** | **3.9** |

These figures may differ from the number of births presented elsewhere due to the inclusion of terminations of pregnancy for psychosocial reasons or congenital anomalies at greater than 20 weeks' gestation, which are excluded from some analyses in other CCOPMM reports.

# Appendix E: Outcomes of selected major congenital anomalies, 2007–2009

| Chromosomal anomalies* | Terminations < 20 weeks | Terminations ≥ 20 weeks | Still births† | Neonatal deaths† | Lived at least 28 days |
|---|---|---|---|---|---|
| Trisomy 21 | 475 | 49 | 8 | < 5 | 204 |
| Trisomy 13 | 58 | 18 | < 5 | < 5 | < 5 |
| Trisomy 18 | 182 | 30 | 12 | 7 | 7 |

| Other selected major anomalies* | Terminations | Still births† | Neonatal deaths† | Lived at least 28 days |
|---|---|---|---|---|
| Anencephaly | 92 | < 5 | 6 | 0 |
| Spina bifida | 75 | < 5 | < 5 | 33 |
| Encephalocele | 24 | < 5 | 0 | 9 |
| All neural defects | 185 | 5 | 9 | 42 |
| Microcephalus | 15 | < 5 | < 5 | 47 |
| Hydrocephalus | 85 | 6 | 10 | 93 |
| Transposition of the great vessels | 26 | < 5 | 6 | 99 |
| Tetralogy of fallot | 8 | < 5 | 6 | 59 |
| Ventricular septal defect | 48 | 12 | 14 | 599 |
| Hypoplastic left heart syndrome | 33 | < 5 | 10 | 44 |
| Coarctation of the aorta | 5 | < 5 | < 5 | 89 |
| Cleft palate | 10 | < 5 | < 5 | 148 |
| Cleft lip | 12 | 0 | < 5 | 79 |
| Cleft lip and palate | 32 | < 5 | 5 | 100 |
| Oesophageal atresia and/or stenosis | 5 | < 5 | 6 | 54 |
| Small intestinal atresia and/or stenosis | < 5 | < 5 | < 5 | 51 |
| Anorectal atresia and/or stenosis | 12 | < 5 | < 5 | 50 |
| Hypospadias | < 5 | < 5 | < 5 | 863 |
| Renal agenesis and dysgenesis | 41 | < 5 | 6 | 92 |
| Cystic kidney disease | 32 | < 5 | 6 | 86 |
| Obstructive defects of renal pelvis | 37 | < 5 | 7 | 906 |
| Developmental dysplasia of the hip | 0 | 0 | < 5 | 660 |
| Limb reduction defects | 49 | < 5 | < 5 | 86 |
| Diaphragmatic hernia | 15 | < 5 | 8 | 63 |
| Exomphalos | 50 | 5 | < 5 | 21 |
| Gastroschisis | < 5 | 5 | < 5 | 41 |

* Congenital anomaly can be isolated or can occur with other anomalies
† Excludes terminations

# Definitions

| | |
|---|---|
| **Birth** | Refers to both live births and stillbirths. |
| **Birth plurality** | Refers to the total number of births resulting from a single pregnancy. Singleton or Single refers to one birth resulting from a single pregnancy and multiple refers to more than one birth from a single pregnancy. |
| **Confinements** | These are the number of pregnancies resulting in at least one birth. Number of confinements are not equal number of births, for example, one confinement may result in two births (twins). |
| **Congenital anomaly** | Any abnormality of prenatal origin, either present following conception or occurring before the end of pregnancy. This includes structural, functional, genetic, chromosomal and biochemical abnormalities. |
| **Congenital anomaly cases** | Refers to the number of live born or stillborn babies or terminations at any gestation affected by at least one congenital anomaly. |
| **Isolated congenital anomaly** | Anomaly that is not related to any other condition and occurs as a single defect example isolated cleft lip. |
| **Live birth** | Complete expulsion or extraction from its mother of a baby of at least 20 weeks gestation or, if gestation is unknown, weighing at least 400 grams who, after being born, breathes or shows any evidence of life such as a heartbeat. |
| **Low birth weight** | Birth weight less than 2,500 grams. |
| **Neonatal death** | A death occurring within 28 days of live birth in an child whose gestation was at least 20 weeks or, if gestation is unknown, weighing at least 400 grams. |
| **Parity** | Number of pregnancies carried to a viable gestational age. A woman who has never carried a pregnancy beyond 20 weeks gestation is called nulliparous, a woman who has carried one pregnancy beyond 20 weeks is called primiparous and a woman who has carried two or more pregnancies beyond 20 weeks is called multiparous. |
| **Perinatal death/mortality** | A stillbirth or neonatal death. |
| **Pregnancy** | Includes live birth, stillbirth and termination of pregnancy at any gestation. |
| **Stillbirth** | Complete expulsion or extraction from its mother of a baby of at least 20 weeks gestation or, if the gestation is unknown, weighing at least 400 grams who did not, at any time after delivery, breathe or show any evidence of life such as a heartbeat. |

# References


1. Hilder L, Zhichao Z, Parker M, Jahan S, Chambers GM. Australia's mothers and babies 2012. Perinatal statistics series no. 30. Cat. no. PER 69. Canberra: AIHW, 2014
2. Abeywardana S, Sullivan EA. Congenital anomalies in Australia 2002–2003. Birth anomalies series no. 3 Cat. no. PER 41. Sydney: AIHW National Perinatal Statistics Unit, 2008.
3. The International Clearinghouse for Birth Defects Surveillance and Research. Annual Report 2005 with data for 2003. International Centre on Birth Defects. Italy, 2006.
4. Riley M, Halliday J. Congenital malformations in Victoria 1983–1994, Perinatal Data Collection Unit, Victorian Government Department of Human Services, Melbourne, 1996.
5. Riley M, Halliday J. Birth defects in Victoria 1983–1998, Perinatal Data Collection Unit, Victorian Government Department of Human Services, Melbourne, 2000.
6. Riley M, Halliday J. Birth defects in Victoria 1999–2000, Perinatal Data Collection Unit, Victorian Government Department of Human Services, Melbourne, 2002.
7. Riley M, Halliday J. Birth defects in Victoria 2001–2002, Perinatal Data Collection Unit, Victorian Government Department of Human Services, Melbourne, 2004.
8. Riley M, Halliday J. Birth defects in Victoria 2003–2004, Perinatal Data Collection Unit, Victorian Government Department of Human Services, Melbourne, 2006.
9. Riley M, Halliday J. Birth defects in Victoria 2005–2006, Victorian Perinatal Data Collection Unit, Victorian Government Department of Human Services, Melbourne, 2008.
10. World Health Organization. Congenital anomalies. 2015. Accessed February 29, 2016. Available at: http://www.who.int/mediacentre/factsheets/fs370/en/
11. World Health Organisation, Centers for Disease Control, International Clearinghouse for Birth Defects Surveillance and Research. Birth defects surveillance: a manual for programme managers. Geneva: World Health Organization, 2014.
12. Victorian Perinatal Data Collection Unit. Victorian Perinatal Data Collection (VPDC) Manual version. 3.0, 2013. Accessed 29 February 2016. Available at: https://www2.health.vic.gov.au/hospitals-and-health-services/quality-safety-service/consultative-councils/council-obstetric-paediatric-mortality/perinatal-data-collection
13. Lumley J, Palma S, Fischer M, Robertson H. The tip of the iceberg: A validation study of the Victorian Congenital Malformations/Birth Defects Register. Australian Paediatric Journal, 1988:24:244–6
14. Kilkenny M, Riley M, Lumley J. Follow-up validation study of the Victorian Congenital Malformations Register. Journal of Paediatric & Child Health, 1995;31:323–25.
15. Riley M, Phyland S, Halliday J. Validation study of the Victorian Birth Defects Register, Journal of Paediatric and Child Health, 2004:40:544–548.
16. Riley M, Howard J, Dale K, Palma S, Halliday J. Validating notifications of pregnancy terminations for birth defects before 20 weeks gestation, Health Information Management, 2001;30: No.2
17. Davey MA, Sloan ML, Palma S, Riley M, King J. Methodological processes in validating and analysing the quality of population-based data: a case study using the Victorian Perinatal Data Collection. Health Information Management Journal 2013; 42(3):12.
18. National Perinatal Epidemiology and Statistics Unit (NPESU). Australian Congenital Anomalies Monitoring System. Last updated November 2016. Available at: https://npesu.unsw.edu.au/data-collection/australian-congenital-anomalies-monitoring-system-acams
19. Lancaster P & Pedisich E. Congenital malformations Australia 1981–1992. Birth defects series no. 1. Cat. no. AIHW 213. Canberra: AIHW, 1995.

20. Zhao Y, You J, Guthridge S. Burden of disease and injury in the Northern Territory, 1999–2003. Department of Health and Families. Darwin, 2009.
21. Wen J, Jiang J, Ding C, Dai J, Liu Y, Xia Y, Liu J, Hu Z. Birth defects in children conceived by in vitro fertilization and intracytoplasmic sperm injection: a meta-analysis. Fertility and sterility. 2012 Jun 30;97(6):1331-7.
22. Yan J, Huang G, Sun Y, Zhao X, Chen S, Zou S, Hao C, Quan S, Chen ZJ. Birth defects after assisted reproductive technologies in China: analysis of 15,405 offspring in seven centers (2004 to 2008). Fertility and sterility. 2011 Jan 31;95(1):458-60.
23. Setti PE, Moioli M, Smeraldi A, Cesaratto E, Menduni F, Livio S, Morenghi E, Patrizio P. Obstetric outcome and incidence of congenital anomalies in 2351 IVF/ICSI babies. Journal of assisted reproduction and genetics. 2016 Jun:1-7.
24. Gibson CS, Scott H, Scheil W. Birth defects in South Australia 2011. Adelaide. SA Birth Defects Register, Women's and Children's Health Network, 2015.
25. Bower C, Baynam G, Rudy E, Quick J, Rowley A, Watson L, Cosgrove P. Report of the Western Australian Register of Developmental Anomalies 1980–2014. Western Australian Register of Developmental Anomalies. November 2015.
26. Endo T, Johnston T, Ellerington J. Data quality and quality issues to be aware of when using the Queensland Perinatal Data Collection to estimate the prevalence of congenital anomalies at birth in Queensland. Health Statistics Unit, Queensland Health. Brisbane 2014.
27. Centre for Epidemiology and Research. New South Wales Mothers and Babies 2009. Sydney: NSW Ministry of Health, 2011 and Australian Capital Territory: Population Health Research Centre, ACT Health. Maternal and Perinatal Health in the ACT 2000–2004, ACT Government, Canberra ACT, 2007.


VICTORIA
State
Government
Health
and Human
Services