Alberta Congenital Anomalies Surveillance System

Ninth Report: 1997–2009

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Government of Alberta

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For more information contact:
Surveillance and Assessment
Community and Population Health
23rd Floor, Telus Plaza North Tower
10025 Jasper Avenue NW
PO Box 1360 STN MAIN
Edmonton, Alberta T5J 2N3
CANADA

Phone: 1 (780) 427-4518
Toll Free: 310-0000 (in Alberta only)
Fax: 1 (780) 422-6663
Email: health.surveillance@gov.ab.ca
Internet: http://www.health.alberta.ca

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Ninth Report

1997–2009

Prepared by:

R.B. Lowry, MD, DSc, FRCPC
B. Sibbald, BA, BN, MSc
T. Bedard, BSc, MPH

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ACASS
R.B. Lowry, MD, Medical Consultant
B. Sibbald, MSc, Manager
T. Bedard, MPH, Research Assistant
J. Anderson, Secretary
A. Preece, Clerical Assistant

Surveillance and Assessment, Alberta Health and Wellness
K. Ness, Executive Director
F-L Wang, Epidemiologist
M. Sanderson, Manager, Monitoring

Alberta Registries, Vital Statistics
L. Speakman, Executive Director
M. Bichai, Director
J. Traill, System Support Administrator
G. Brese, Vital Statistics Specialist

Advisory Committee
F. Bernier, MD, FRCPC, Medical Genetics
M. Brindle, MD, FRCSC, Paediatric Surgery
J. Harder, MD, FRCPC, Paediatric Cardiology
G.N. Kiefer, MD, FRCSC, Paediatric Orthopaedics
J. Midgley, MD, FRCPC, Paediatric Nephrology
H. Sarnat, MD, FRCPC, Paediatric Neurology
R. Sauvé, MD, FRCPC, Neonatology and Community Health Sciences
C. Trevenen, MD, FRCPC, Paediatric Pathology
R. Douglas Wilson, MD, FRCSC, Perinatology/Obstetrics

ACRONYMS FOR ORGANIZATIONS MENTIONED IN THE REPORT

ACASS Alberta Congenital Anomalies Surveillance System

CCASN Canadian Congenital Anomalies Surveillance Network
http://www.phac-aspc.gc.ca/ccasn-rcsac/index-eng.php

ICBDSR International Clearinghouse for Birth Defects Surveillance and Research
www.icbdsr.org

NBDPN National Birth Defects Prevention Network
www.nbdpn.org

AHS Alberta Health Services
www.albertahealthservices.ca

AHW Alberta Health and Wellness
www.health.alberta.ca

Table of contents

1. ACASS Activities and Report Summary

1. This is the ninth in a series of reports detailing the birth prevalence of congenital anomalies in Alberta particularly the years 1997–2009 inclusive.

2. The International Classification of Diseases – 10th Edition (ICD-10) has been adopted by Alberta hospital reporting data systems and ACASS uses the Royal College of Pediatrics and Child Health adaptation of ICD-10. The anomalies outlined in the National Birth Defects Prevention Network's Guidelines for Conducting Birth Defects Surveillance (http://www.nbdpn.org/docs/NBDPN_Guidelines2008.pdf) are reported in this document however, all items from the ICD-10 "Q" codes as well as other sections such as disorders of metabolism are monitored by ACASS. Data on such disorders can be provided to interested parties upon request.

3. The numerator data now includes fetal losses <20 weeks gestation with congenital anomalies. This differs from past reports where live births and stillbirths only were used. Denominator data includes live births and stillbirths only. The reported rates should be more representative of true congenital anomaly rates. Fetal losses have been ascertained since 1997 thus aggregate data are reported from that year. Congenital anomalies data from 1980 onwards, can be accessed at http://www.health.alberta.ca/newsroom/pub-pregnancy-birth.html, however fetal losses will not be included in the numerator.

4. Patent ductus arteriosus (PDA) has been omitted from this report. The data are under review and will be reported in subsequent publications.

5. There may be discrepancies in coarctation of the aorta rates from previous years. This is due to the change from ICD-9 to ICD-10. The current report uses ICD-10 only so the rates will reflect a uniform definition.

6. There may be discrepancies in congenital dislocation of the hip from previous reports as well. ICD-9 did not include hip dysplasia in the congenital dislocation of the hip code 754.3. ICD-10 includes hip dysplasia in the Q65 codes so the rates will reflect this addition.

7. Congenital anomaly rates have remained relatively stable over the years with fluctuations occurring on a year to year basis. There are, however, notable exceptions such as neural tube defects, Down syndrome, and gastroschisis.

8. There appears to be an increasing trend in spina bifida and anencephaly recently which is not statistically significant. The spikes in spina bifida (2006) and anencephaly (2009) were reviewed (**see p.18**). ACASS will continue to monitor the rates to determine whether these are true and sustained increases, or simply factors of normal fluctuations in rates or of ascertainment.

9. Gastroschisis continues to show an increase and is particularly prevalent in young mothers, which is consistent with worldwide observations from other jurisdictions. Omphalocele on the other hand shows no such increases, nor is there an association with low maternal age. In fact there is a higher frequency of omphalocele found in higher maternal ages, that is 40 years of age and older.

10. The increase in Down syndrome is likely attributable to the increased number of women giving birth aged 35 years or older.

11. The percentage of births to women 35 years of age and over continues to increase with approximately 16 per cent of women in this age category giving birth in 2009 compared to four per cent in 1980.

12. The total number of births in Alberta continues to increase from 48,708 in the year 2007 to 51,407 in 2009.

13. A review of congenital heart defects (CHDs) was undertaken to assess the ascertainment quality of ACASS as well as to investigate whether or not folic acid (FA) fortification had any impact on their rates. ACASS ascertainment of severe defects was high (87.5–100 per cent) but lower for anomalies such as bicuspid aortic valve, PDA, aortic valve stenosis, pulmonary valve stenosis and septal defects. Increasing the ascertainment sources as well as the duration of ascertainment (>one year after delivery) increased the ascertainment rates. Although the effects of FA fortification on reducing neural tube defects have been well documented, the effects are mixed with respect to CHDs. Left ventricular outflow tract obstruction (LVOTO) decreased in the post FA fortification period whereas atrial septal defect with or without ventricular septal defect (ASD +/- VSD) increased. These changes, however, were also noted throughout the study period suggesting alternate factors might also be responsible.

14. ACASS continues to be a member of the Canadian Congenital Anomalies Surveillance Network (CCASN) (http://www.phac-aspc.gc.ca/ccasn-rcsac/index-eng.php), a Public Health Agency of Canada initiative, with members of ACASS playing a significant role in Network committees. The Network has been formed to support the development and maintenance of high quality population-based surveillance systems for congenital anomalies.

15. ACASS continues its affiliation with the International Clearinghouse for Birth Defects Surveillance and Research (ICBDSR) (http://www.icbdsr.org/page.asp?p=9895&l=1) and has participated in group studies in a number of congenital anomalies including craniofacial defects, very rare defects, gastroschisis, holoprosencephaly and Down syndrome ascertainment (**see Surveillance and Research Projects, p.31-32**).

16. Four infants with severe unilateral microtia were born in Calgary within a one month period; two on the same day. This unusual event was investigated by ACASS staff at the request of the attending physicians and with the co-operation of the families. There were no common environmental, occupational, genetic or other etiologic factors found. The final conclusion is that this was a statistical chance event.

2. Introduction

This report provides updated data on congenital anomalies ascertained in Alberta from the years 1997–2009 inclusive. For the current release, the anomalies outlined in the National Birth Defects Prevention Network's (NBDPN) Guidelines for Conducting Birth Defects Surveillance (2004) are reported, however, data on other anomalies can be provided upon request. PDA has been excluded from this report pending a review of all cases to ensure consistency in coding practices over the years.

The numerator data now include all fetal losses <20 weeks gestation with congenital anomalies. This differs from past reports where live births and stillbirths only were used. The reported rates should be more representative of the true rates of congenital anomalies in Alberta. Fetal losses have been ascertained since 1997 thus aggregate data are reported from that year forward. Congenital anomalies data from 1980 onwards can be accessed from previous reports at http://www.health.alberta.ca/newsroom/pub-pregnancy-birth.html, however fetal losses will not be included in the numerator. Denominator data includes live births and stillbirths only.

2.1 History

The history of the Alberta Congenital Anomalies Surveillance System (ACASS) has been described in previous reports. Since 1996, funding has been provided by Alberta Health and Wellness (AHW), Surveillance and Assessment Branch. ACASS continues to work closely with Alberta Vital Statistics and relies on them for the provision of notifications of births, deaths and stillbirths (**see Case Ascertainment, p.10**).

2.2 Purpose of a Surveillance System

Public health surveillance in general has been defined by the Centers for Disease Control and Prevention (CDC) in Atlanta, Georgia as the ongoing, systematic collection, analysis and interpretation of data (e.g., regarding agent/hazard, risk factor, exposure, health event) essential to the planning, implementation and evaluation of public health practice, closely integrated with the timely dissemination of these data to those responsible for prevention and control.

The purposes and objectives of surveillance for congenital anomalies (CAs) are to:

1) provide reliable and valid data on the birth prevalence of congenital anomalies in Alberta;
2) investigate any significant temporal or geographic changes in the frequency of congenital anomalies with a view to identifying environmental, and therefore, possibly preventable causes;
3) measure trends;
4) assess the effectiveness of prevention (e.g., folic acid fortification or antenatal screening);
5) assist with health related program planning and development through the provision of data;
6) participate in research into the etiology and natural history of birth defects;
7) assist with research through provision of congenital anomalies data; and
8) provide advice to health care professionals about congenital anomalies, especially with respect to teaching and launching public health campaigns (e.g., folic acid campaign by Community Health in Calgary).

As well as the above, patterns or associations of malformations to determine whether they belong to an existing or new syndrome complex can be explored.

A principle feature of a surveillance system is timeliness; however data collection and analysis should not be accomplished at the expense of an accurate diagnosis. Data are collected to the first birthday, and with the possibility of reporting delays, the data of a given calendar year may not be complete until at least December 31 of the subsequent year although the cases and anomalies are monitored as received.

2.3 Prevention of Congenital Anomalies (Birth Defects)

There is increasing concern about the causes of birth defects and many questions have been raised about relationships between environmental exposures and health problems such as respiratory illness, cancer or birth defects. It is therefore of the utmost importance to keep accurate databases on the occurrence and geographical distribution of illness such as cancer or birth defects which, in the latter case, is the task of ACASS. There is no current indication of any major geographical or environmental clustering of birth defects in Alberta but a preliminary study (Bedard 2011[1]) showed a non significant increase in the birth prevalence of congenital heart disease cases in rural Alberta compared with urban Alberta. Surveillance for trends or clustering of birth defects is a safeguard for the population.

As far as temporal effects and trends are concerned these are also reasonably stable but there are some exceptions (**see Table 4.1.1, p.14**). Neural tube defects have had no significant change since the decline that followed the introduction of folic acid fortification in 1998. There was a peak in spina bifida for the year 2006 and a very small peak for anencephaly in 2009 which are largely unexplained (**see p.18**). There has been a non-significant increase in cleft lip with or without cleft palate but there is a significant downward trend for cleft palate and also for anorectal and large intestine atresia/stenosis.

The main increases are in gastroschisis and Down syndrome. The latter is almost certainly due to the much higher proportion of mothers ages 35 and over who are having babies. In the early 1980's about 4 per cent of all mothers were aged 35 or over whereas now it is 16 per cent. Young maternal age has the opposite association with gastroschisis and occurs mainly in very young mothers (15 to 19 yrs) and to a lesser degree also among the 20 to 24 year age group with relative stability in prevalence among subsequent maternal ages. While the exact cause is unknown there is a correlation with a number of risk factors such as absence of prenatal care, poor nutrition, use of vasoconstrictive recreational drugs such as ecstasy, amphetamines and cocaine, cigarette smoking or marijuana, and genitourinary infections. It is of interest that the increase in gastroschisis is a worldwide phenomenon. There is a mildly significant increase in hypospadias which is discussed on page 27.

Birth defects prevention strategies have been known and implemented with varying degrees of success over the years, the most successful being Rubella immunization and the elimination of congenital Rubella syndrome. Unfortunately there are small pockets in the population who refuse to have their children immunized because of the alleged association between the MMR vaccine and autism which has been proven to be false. Improved glycemic control for diabetic mothers and identification of those with pre gestational diabetes is of the utmost importance as is the avoidance of tobacco, alcohol and drugs that are known to be teratogenic such as thalidomide, antiepileptics, and isotretinoins. Indeed other drugs including recreational drugs even if not proven to be teratogenic should be avoided in pregnancy if possible. If medically needed they should be used only after careful assessment by a physician.

Maternal Obesity

There is substantial evidence that birth defects are more frequent among obese mothers (BMI ≥ 30) and even for overweight mothers (BMI 25.0 – 29.9) than among normal weight (BMI 18.5 – 24.9) women. The increase in birth defects involves many different systems including neural tube defects, heart defects, both septal and conotruncal, orofacial clefts, limb reduction defects, obstructive urinary tract defects and anorectal atresia. (Oddy et al 2009[2]; Stothard et al 2009[3]; Watkins et al 2003[4]).

Multivitamins and Folic Acid/Socio Economic Status

A simple public health measure to reduce the prevalence of birth defects, in addition to neural tube defects, would involve campaigns to increase the use of multivitamins and folic acid prior to conception and throughout the first trimester of pregnancy. Although folic acid fortification of flour and pasta is in place in Canada, the USA and Chile, there continues to be a need for potential mothers to take folic acid and multivitamin pills as well because not all mothers will have an adequate level of red blood cell folate. There is considerable evidence that the use of periconceptional folic acid supplements is suboptimal (Ray et al 2004[5]). Public Health campaigns to make the public aware of the need for folic acid supplementation often do show an improvement which however never rises above 50 per cent of the population and is often quite transient dropping to lower levels very quickly. Among 34 studies surveyed about preconceptional folic acid use the highest rate was found among women in Vancouver, BC (49 per cent) (Morin et al 2001[6]). Although Ray et al (2002[7]) have shown increased red cell folate concentrations of women in Canada after fortification nevertheless there may be some women in whom the red blood cell folate concentration is too low. The institution of therapeutic drug monitoring of RBC folate could be used to identify these women thereby lowering their risk for having a child with a neural tube defect (Tam et al 2009[8]).

Whether folic acid reduces defects other than neural tube defects is still unproven. For discussion on cleft lip and palate (**see pages 19-20**). There is some evidence that folic acid and/or multivitamins may reduce the prevalence of limb reduction defects (Cleves et al 2010[9]), Congenital Heart Defects (Botto et al 2004[10]), Urinary Tract Anomalies (Czeizel 2009[11]) and Anorectal Atresia (Myers et al 2001[12]).

It has long been known that lowered socio economic status (SES) has been associated with an increased prevalence of many types of birth defects. Low SES is a much bigger issue than public health and involves less unemployment, more education, better housing and nutrition. Programs such as the Hungarian optimal family planning program (Czeizel 1988[13]) will not only improve birth outcomes with respect to congenital anomalies but likely reduce preterm births since prevention strategies are similar (Dolan et al 2009[14]). We should strive to educate the public so that most pregnancies are planned and have access to optimal periconceptional prenatal care. Reduction of birth defects will reduce the burden of illness upon the individual, the parents, the family and also society in terms of cost savings across many sectors including health, education, and employment.

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3. Methodology

3.1 Case Definitions

A **congenital anomaly** is an abnormality that is present at birth, even if not diagnosed until months or years later. Most congenital anomalies are present long before the time of birth, some in the embryonic period (up to the end of the seventh week of gestation) and others in the fetal period (eighth week to term). The term "anomaly" covers all the major classes of abnormalities of development, of which there are four major categories as follows:

Malformation – a morphologic defect of an organ, part of an organ or a larger region of the body resulting from an intrinsically abnormal developmental process (e.g., spina bifida, cleft lip and palate).
Deformation – an abnormal form, shape or position of a part of the body caused by mechanical forces (e.g., extrinsic force such as intrauterine constraint causing some forms of clubfoot).

Disruption – a morphologic defect of an organ, part of an organ or a larger region of the body resulting from the extrinsic breakdown of, or an interference with, an originally normal developmental process (e.g., an infection such as rubella or a teratogen such as thalidomide).

Dysplasia – the abnormal organization of cells into tissues and its morphologic result (e.g., Marfan Syndrome, osteogenesis imperfecta).

Other definitions related to pregnancy outcomes for the purposes of this report are as follows:

Live birth – a complete expulsion or extraction from the mother, *irrespective* of the duration of the pregnancy, of a fetus in which, after expulsion or extraction, there is breathing, beating of the heart, pulsation of the umbilical cord or definite movement of voluntary muscle (Alberta Vital Statistics Annual review, 2000).

Stillbirth – a complete expulsion or extraction from the mother, after at least 20 weeks pregnancy (≥20 weeks), *or* after attaining a weight of 500 grams or more (≥500 grams) of a fetus in which, after the expulsion or extraction, there is no breathing, beating of the heart, pulsation of the umbilical cord or unmistakable movement of voluntary muscle (Alberta Vital Statistics Annual review, 2000).

Gestation – completed weeks of pregnancy at delivery.

Preterm birth (aka premature) – a birth before 37 weeks of gestation (<37 weeks).

Termination of Pregnancy (ToP) – for our purposes, any pregnancy loss before 20 weeks gestation (<20 weeks), most of which are therapeutic terminations for congenital anomalies but could include spontaneous abortions or intrauterine fetal deaths with fetal anomalies.

Anomaly definitions are based, for the most part, on those provided by the ICBDSR and NBDPN.

3.2 Case Ascertainment

An infant can be ascertained at any time up to the first birthday. Multiple ascertainment of the same infant can occur and is encouraged, as this frequently improves the quality and reliability of the data.

As several malformations may occur in the same infant, it is advantageous to allow each to be reported so that groups of associated malformations may be studied. This, however, leads to

difficulties since the final tabulations may be reported as total malformations (anomaly rates) or as the total number of malformed infants (case rates).

ACASS obtains information about infants with congenital anomalies from a variety of independent sources. Acquisition of additional reporting agencies is always a priority since the use of multiple sources of information improves not only the ease but also completeness of ascertainment as well as for verification of the diagnostic data. Appendix A.1 (**p. 34**) shows the process of data collection at ACASS.

ACASS screens many Alberta Health and Wellness and Alberta Vital Statistics documents for the presence of a congenital anomaly including:

- Notice of a Live Birth or a Stillbirth and Newborn Record often referred to as the Physician's Notice of Birth (NOB)
- Medical Certificate of Stillbirth
- Medical Certificate of Death

Also, ACASS screens a notification called the Congenital Anomalies Reporting Form (**CARF, Appendix A.2, p.35**) that is completed by all acute care hospitals in the province on live births, stillbirths, admissions or hospital deaths of infants under one year of age as well as pregnancy losses involving one or more congenital anomalies. This form serves as the single most important source of case ascertainment.

Since many children with congenital anomalies are not admitted to hospital, it is very important to obtain out-patient information such as from the Calgary and Edmonton Departments of Medical Genetics.

Ascertainment at a continued high level requires each hospital health record department and each health care provider to co-operate with the system by notifying us as promptly as possible. We are fortunate in having such co-operative agencies and personnel.

3.3 Quality Control Measures

When a copy of a reporting document reaches the ACASS office in Calgary, it is reviewed for content by the Research Assistant and Manager. If the information is unclear, the Manager, on behalf of the Medical Consultant, writes to the physician responsible for the case seeking clarification. A stamped, addressed envelope is included with the letter and the physician is asked to respond at the bottom of the letter thus making the mechanics of replying easy. The response from physicians has been very satisfactory (greater than 90 per cent) and usually this is sufficient to make a decision whether to accept or reject an anomaly or case. Any questionable diagnosis that is not confirmed is not entered into the database. Some cases also not included contain diagnoses that do not belong in a congenital anomaly system or are part of a normal developmental process such as a patent ductus arteriosus or undescended testes in a premature infant. Any reports requiring a medical decision are reviewed with the Medical Consultant. Policy decisions with respect to the acceptance or rejection of a case and its coding are referred to the ACASS Advisory Committee. This body is comprised of a paediatric cardiologist, neonatologist/epidemiologist, paediatric pathologist, medical geneticist (medical consultant) with occasional input from a paediatric neurologist, paediatric nephrologist, paediatric orthopaedic surgeon, paediatric general surgeon and a perinatologist/obstetrician.

3.4 Anomaly Coding

Coding is done at the Calgary office using the Royal College of Paediatrics and Child Health (RCPCH) adaptation of the International Classification of Diseases, tenth edition (ICD-10). Difficult cases are referred to the Medical Consultant (Medical Geneticist). In the past, we were able to code only six anomalies per case but since 1997 we have been coding all eligible anomalies reported to us.

3.5 Data Linkage

Data from ACASS are linked to data from the Alberta Vital Statistics Birth Registry by the birth registration number, with over 99 per cent success. Some maternal risk factor data, such as maternal smoking, drinking and use of street drugs during pregnancy are thus available for babies with congenital anomalies. This linkage enables in-depth data analysis and interpretation.

3.6 Confidentiality and Release of Data

Notifications of Congenital Anomalies are sent to the Surveillance and Assessment Branch, Alberta Health and Wellness (AHW) and from there to the ACASS office in Calgary where the database is maintained. The notifications are handled by the Manager, Research Assistant, Secretary, Clerk and Medical Consultant. The data are treated in a completely confidential manner and the notifications are kept in locked files in a locked room. The database is secured by limited access and is password protected. Should further clarification about a case or anomaly become necessary, we communicate with the attending physician or the physician responsible for ongoing care. Direct contact is never made with the family. When data are requested from us, they are released in aggregate form with no personal identifiers.

Should record level data be required for research purposes, a request should be made to ACASS, however such data are ultimately released through AHW, Planning and Performance Branch Strategic Directions Division. In this situation, it would be appropriate to first contact ACASS with an outline of the proposal to determine the feasibility of the study and whether or not ACASS has the necessary data. An e-mail should then be sent to health.resdata@gov.ab.ca complete with the proposal and appropriate ethics approvals.

3.7 Epidemiological and Statistical Measures

Unless otherwise stated, the birth defect rates presented in this report are calculated using the following formulae:

ANOMALY (DEFECT) RATE =

$$\frac{\text{Number of a particular congenital anomaly among live births + stillbirths + fetal losses}}{\text{Total number of live births and stillbirths}} \times 1000$$

CASE RATE =

$$\frac{\text{Number of individual infants (live or stillborn) or fetuses with} \geq \text{1 congenital anomaly}}{\text{Total number of live births and stillbirths}} \times 1000$$

Confidence intervals (95 per cent) are also included because the rate obtained is actually only a point estimate of the unknown, true population rate. The confidence interval provides information about the precision of the estimate. Thus, the confidence intervals are an estimated range of values within which there is a 95 per cent probability that the true population rate will fall.

Chi Squared Linear Trend Analysis was performed and presented as appropriate.

3.8 Limitations of Data and Analysis

One of the major limitations of the surveillance system is that on its own, the information provided to us does not allow studies to determine etiology. If increasing trends indicate there is a potentially serious problem, then separate investigative studies need to be done. However, it would be possible to conduct linkage studies with other data sources to explore potential causes of specific birth defects.

The ACASS data are collected passively from Vital Statistics, hospitals, and other agencies but are augmented by active ascertainment from physicians and labs, etc. The completeness and accuracy of data are largely dependent on reporting.

4. Patterns of Selected Congenital Anomalies in Alberta

4.1 Birth Prevalence – Time Trends

The following table and graphs of selected sentinel anomalies indicate the trends in congenital anomaly rates in Alberta from 1997 through 2009. Sentinel anomalies are those which the International Clearinghouse of Birth Defects Surveillance and Research (ICBDSR), of which we are a member, watches worldwide with the rationale that they are quite easily identified hence more accurately reported.

Table 4.1.1 Chi Squared Linear Trend Analysis and p-values for Selected Anomalies 1997–2009 Inclusive (Live Births, Stillbirths & ToPs)

Anomaly	Trend Direction	Chi Squared Analysis (χ^2LT)	p-value
Neural Tube Defects	No significant change	0.80	0.3711
Anencephaly	No significant change	1.98	0.1594
Spina Bifida	No significant change	0.04	0.8415
Hydrocephalus	No significant change	2.24	0.1345
Cleft Lip +/- Cleft Palate	Possible slight increase	3.50	0.0614
Cleft Palate	**Decreasing**	9.63	0.0019
Oesophageal Atresia/Stenosis	No significant change	0.60	0.4386
Anorectal & Large Intestine Atresia/Stenosis	**Decreasing**	7.64	0.0057
Hypospadias and Epispadias *	**Increasing**	4.43	0.0353
Limb Reductions	No significant change	0.00	1.000
Gastroschisis	**Increasing**	13.30	0.0003
Omphalocele	No significant change	0.98	0.3222
Down Syndrome	**Increasing**	6.46	0.0110
Renal Agenesis	No significant change	1.71	0.1910
Hypoplastic Left Heart Syndrome	No significant change	0.87	0.3510

*Hypospadias and Epispadias calculated for male births only

Table 4.1.2 presents a comparison of birth prevalence rates between ACASS and a selection of other countries or regions as reported in the International Clearinghouse for Birth Defects Surveillance and Research Annual Report, 2009.

Table 4.1.2 Selected Anomaly Rates for Alberta and Other Jurisdictions Reporting to the ICBDSR, 2003–2007* Rates per 1000 Total Births, ToPs included.

Anomaly	Alberta	Western Australia	Atlanta	Utah	Wales	Texas (2002–2006)
Neural Tube Defects	0.75	1.37	0.71	0.76	1.44	0.67
Anencephaly	0.23	0.53	0.20	0.24	0.54	0.24
Spina Bifida	0.41	0.70	0.40	0.44	0.70	0.35
Hydrocephalus	0.62	0.77	0.62	0.46	0.90	0.60
Cleft Lip +/- Cleft Palate	1.27	1.27	0.91	1.28	1.13	1.08
Cleft Palate	0.65	0.90	0.47	0.71	0.94	0.53
Oesophageal Atresia/Stenosis	0.21	0.45	0.18	0.28	0.30	0.20
Anorectal Atresia/Stenosis	0.48	0.60	0.32	0.36	0.29	0.51
Hypospadias†	2.12	3.27	0.60	0.74	2.23	1.58
Limb Reductions	1.07	0.70	0.39	0.65	0.89	0.53
Gastroschisis	0.47	0.38	0.39	0.54	0.71	0.47
Omphalocele	0.26	0.45	0.19	0.24	0.44	0.20
Down Syndrome	2.27	2.71	1.65	1.49	2.24	1.28
Renal Agenesis	0.13	0.50	0.09	0.37	0.20	0.19
Hypoplastic Left Heart Syndrome	0.32	0.17	0.20	0.34	0.35	0.21

* http://www.icbdsr.org/filebank/documents/ar2005/AR%202009_web.pdf

† Alberta, Western Australia and Wales report all hypospadias; Atlanta, Texas and Utah exclude glanular or 1st degree hypospadias

4.2 Selected Anomalies

4.2.1 Selected Anomaly Definitions

(Adapted from NBDPN guidelines: http://www.nbdpn.org/)

Neural tube defects

- Anencephaly – partial or complete absence of the brain and skull
- Spina Bifida – incomplete closure of the vertebral spine through which spinal cord tissue and/or the membranes covering the spine (meninges) herniated
- Encephalocele – herniation of brain tissue and/or meninges through a defect in the skull

Cleft Lip and Palate

- Cleft Lip – a defect in the upper lip resulting from incomplete fusion of the parts of the lip
- Cleft palate – an opening in the roof of the mouth resulting from incomplete fusion of the shelves of the palate

Abdominal Wall Defects

- Gastroschisis – a congenital opening or fissure in the anterior abdominal wall lateral to the umbilicus through which the small intestine, and occasionally the liver and spleen, may herniated.
- Omphalocele – a defect in the anterior abdominal wall in which the umbilical ring is widened, allowing herniation of abdominal organs, including the small intestine, part of the large intestine, and occasionally the liver and spleen, into the umbilical cord. The herniating organs are covered by a nearly transparent sac.

Chromosome Anomalies

- Trisomy 13 – aka Patau syndrome – the presence of three copies of all or a large part of chromosome 13
- Trisomy 18 – aka Edwards syndrome – the presence of three copies of all or a large part of chromosome 18
- Trisomy 21 – aka Down syndrome – the presence of three copies of all or a large part of chromosome 21

Limb Reductions

Complete or partial absence of upper and/or lower limbs

Anorectal Atresia Stenosis

Complete or partial occlusion of the lumen of one or more segments of the large intestine and/or rectum.

Renal Agenesisi/Hypoplasia

Complete absence or incomplete development of the kidney

Congenital Heart Disease

- Tetralogy of Fallot – the simultaneous presence of a ventricular septal defect (VSD), pulmonic stenosis, a malpositioned aorta that overrides the ventricular septum and right ventricular hypertrophy
- Hypoplastic Left Heart Syndrome – a condition in which the structures on the left side of the heart and the aorta are extremely small. Classically, this condition includes hypoplasia of the left ventricle, atresia or severe hypoplasia of the mitral and aortic valves, and hypoplasia and coarctation of the aorta.

Hydrocephalus

An increase in the amount of cerebrospinal fluid within the brain resulting in enlargement of the cerebral ventricles and increased intracranial pressure.

Oesophageal Atresia/Stenosis

A condition in which the oesophagus ends in a blind pouch and fails to connect with the stomach

Hypospadias

Displacement of the opening of the urethra ventrally and proximally (underneath and closer to the body) in relation to the glans of the penis.

Epispadias

Displacement of the opening of the urethra dorsally and proximally (on the top and closer to the body) in relation to the tip of the glans of the penis.

4.2.2 Neural Tube Defects

There is no significant trend with neural tube defects overall or within the sub groups of anencephaly, spina bifida and encephalocele. It appears that the rates of spina bifida might be inching upward from a low in 2002 (**Figure 4.2.2**) but ACASS will continue to monitor the trends to determine whether there is a true or sustained change in any direction and whether any changes might simply be a factor of normal fluctuations in rates or of ascertainment. There was an increase in the rate of anencephaly in 2009, however the overall trend, although not statistically significant, seems to be continuing to decrease.

Figure 4.2.1 All Neural Tube Defects 1997– 2009

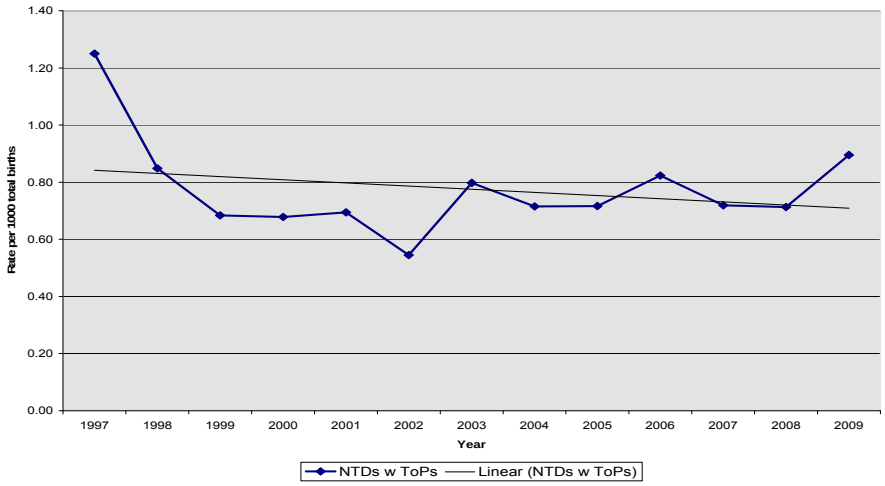
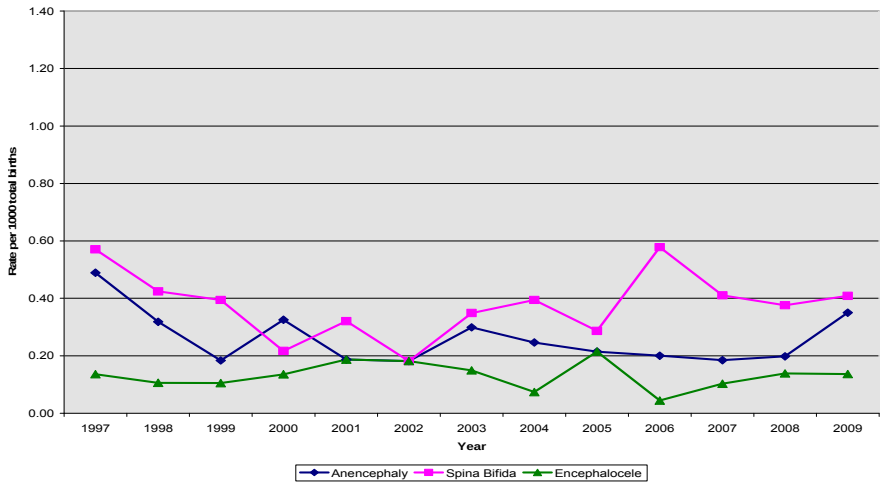


Figure 4.2.2 Neural Tube Defects: Spina Bifida, Anencephaly and Encephalocele 1997–2009



A review of each case of spina bifida for 2006 was done because of the sudden increase. From a total of 26 cases, 15 had a myelomeningocele; three a lipomyelomeningocele; and five a chromosomal etiology (three trisomy 18, one triploidy, one 3q deletion). There was one case each of Limb-body wall malformation; omphalocele-exstrophy-imperforate anus-spinal defects (OEIS); and agenesis of the corpus callosum with myelomeningocele.

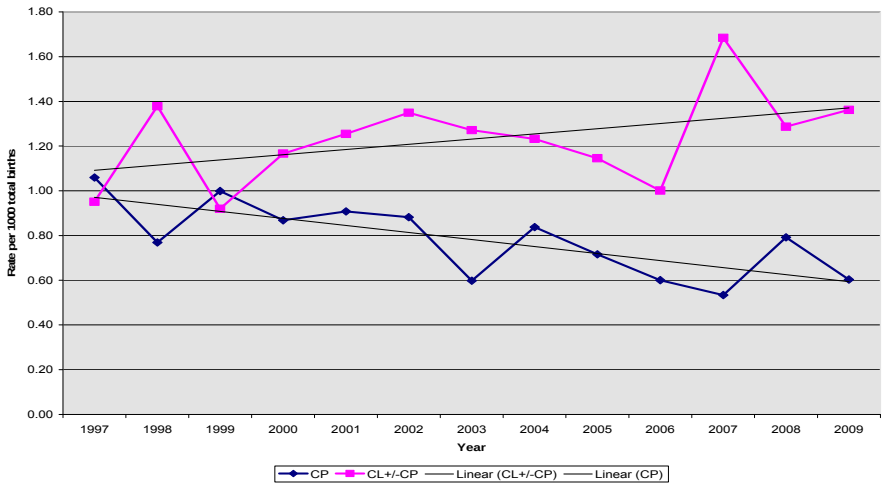
4.2.3 Cleft Lip and Palate

The birth prevalence of cleft lip with or without cleft palate (CL ± CP) remains stable (**Figure 4.2.3**). There are so few ToPs or fetal losses in either group that their addition is not enough to alter the trends in either case (CL ± CP or Cleft Palate (CP) alone). Fetal losses accounted for 4.8 per cent of CL ± CP (33/675) and 2.6 per cent of CP (11/418). The vast majority of the losses had a chromosome etiology or had multiple anomalies with only two apparently isolated cases of CL ± CP. These results and those in Ontario (Ray et al, 2003[i]) suggest that folic acid fortification by itself, has had no effect on the rates of either of the clefting groups. These data do not substantiate the citations that that folic acid fortification has resulted in a decline in CL ± CP prevalence in Canada (Johnson & Little, 2008[ii]; Rozendaal et al, 2011[iii]).

Cleft lip with or without cleft palate has never shown the secular changes that neural tube defects showed. In fact CL ± CP and CP rates have been stable for many years – in British Columbia from 1952–86 (Lowry et al, 1989[iv]) and in Alberta from 1980–2004 (Sibbald & Lowry, 2005[v]). The significant downward trend of cleft palate alone is unexplained.

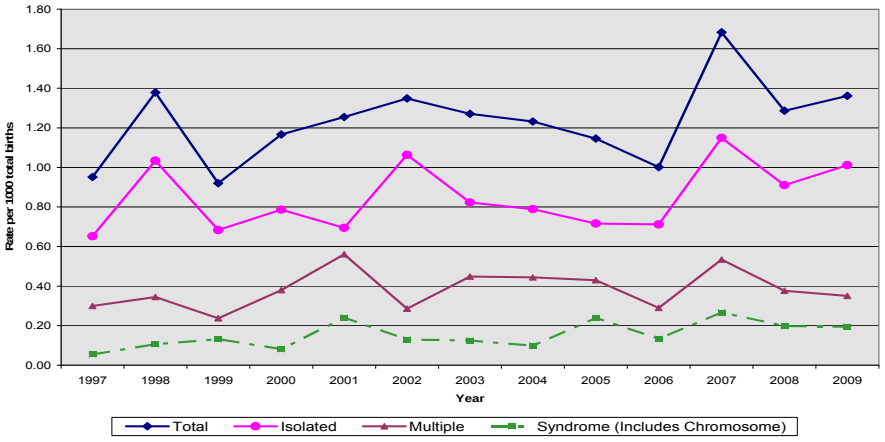
Correct periconceptional use of folic acid (0.4 mg) with vitamins in conjunction with folic acid fortification may be needed to reduce the first occurrence of CL ± CP. The evidence for the effect of folic acid on orofacial clefts is reviewed by Wehby & Murray (2010[vi]) who stressed the need for more studies. There is evidence however, that a high dose of folic acid (6 mg – Czeizel et al, 1999[vii]; or 10 mg – Tolorova & Harris 1995[viii]) can be effective in reducing the recurrence risk of a child with CL ± CP.

Figure 4.2.3 Cleft Lip +/- Cleft Palate (CL+/-CP) and Cleft Palate (CP) Alone 1997–2009



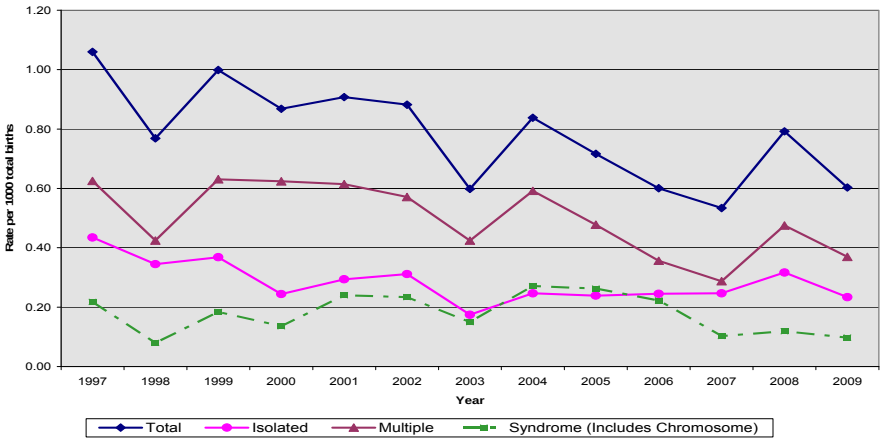
The following graphs (**Figures 4.2.4 and 4.2.5**) illustrate the rates of cleft lip +/- cleft palate and cleft palate alone indicating the proportions that were isolated or multiple cases. The isolated and multiple categories are mutually exclusive. “Isolated” is defined as having a cleft with no other anomalies whereas “multiple” is defined as having a cleft with any other anomalies reported. “Syndrome (including chromosome)” is a subset of the “multiple” category and indicates a known or presumed etiology for the clefting whether it be a recognized syndrome, sequence or chromosome abnormality.

Figure 4.2.4 Cleft Lip +/- Cleft Palate in Total Births, Alberta 1997–2009 Total, Isolated, Multiple and Syndromes*



*includes recognized syndromes as well as chromosome abnormalities

Figure 4.2.5 Cleft Palate Alone, Alberta 1997–2009 Total, Isolated, Multiple and Syndromes*



*includes recognized syndromes as well as chromosome abnormalities

4.2.4 Abdominal Wall Defects

Abdominal wall defects include mainly gastroschisis and omphalocele (**Figure 4.2.6**). Gastroschisis rates continue to increase in keeping with many other jurisdictions world wide. The increase seems to be most obvious in younger mothers, particularly under 20 years of age, and to a lesser extent in the 20–24 year age group (**Figure 4.2.7**). In contrast, there is no significant change in omphalocele annual prevalence rates however they show a reverse maternal age effect in that there is a marked increase in the 35 to 39 year maternal age group and more so in the 40 and over age group. Because omphalocele is more associated with chromosome abnormalities than is gastroschisis, the prevalence in the older mothers might, to some degree, explain the increased rate in mothers over 35 years of age.

Figure 4.2.6 Abdominal Wall Defects – Gastroschisis and Omphalocele in Total Births (Live + Still + ToP), Alberta, 1997–2009

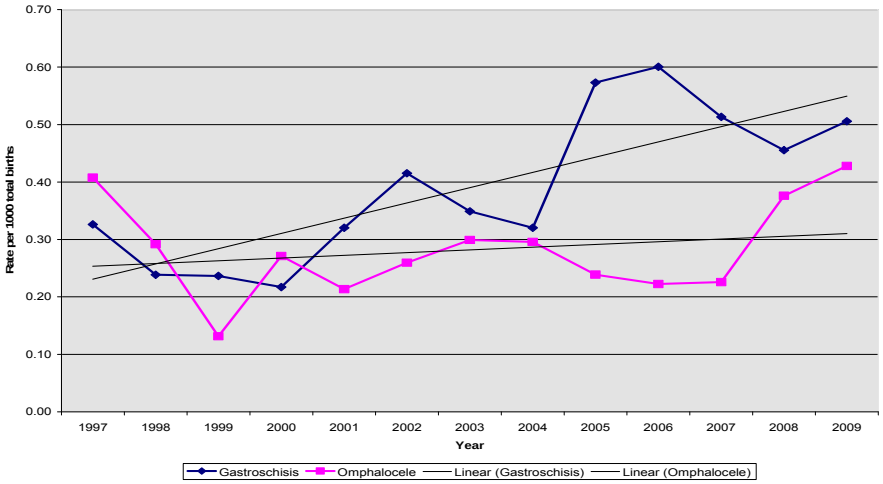
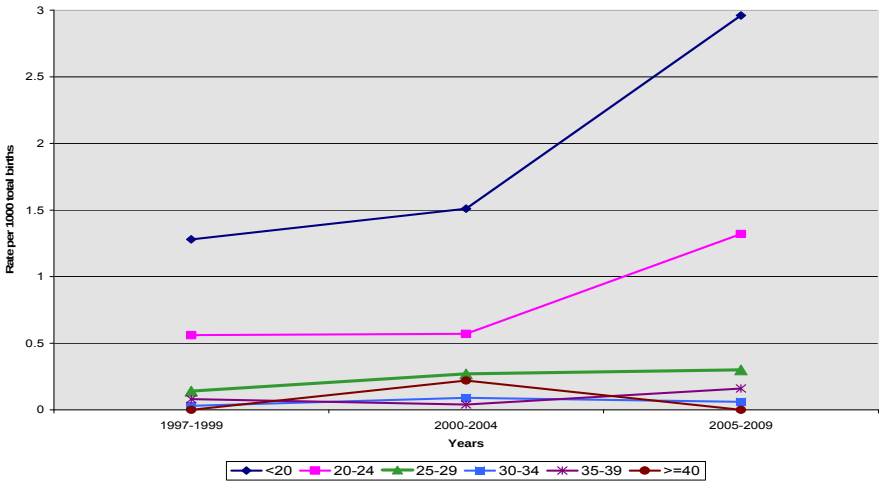


Figure 4.2.7 Gastroschisis by Maternal Age Groups in 5 Year* Increments 1997–2009



*except 3 years for 97–99

4.2.5 Chromosome Anomalies

Down Syndrome is by far the most commonly ascertained chromosome anomaly. As previously reported, rates of Down Syndrome are increasing (**Figure 4.2.9**) but are strongly correlated with increasing maternal age. In 1983, approximately four per cent of mothers were 35 years of age or over whereas in 2009, approximately16 per cent of mothers were in the same age category (**Figure 4.2.8**).

Figure 4.2.8 Maternal Age at birth as a percent of total births, Alberta, 1983–2009

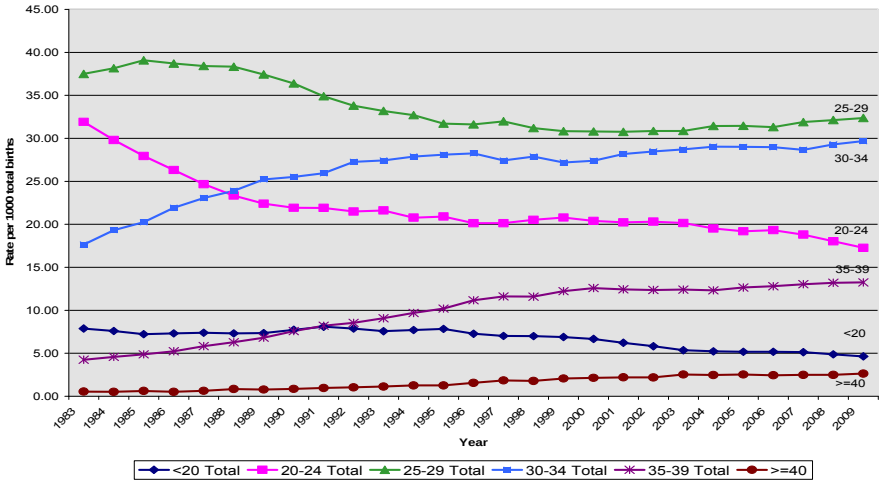


Figure 4.2.9 Chromosome Anomalies: Trisomy 13, Trisomy 18, Trisomy 21, 1997–2009

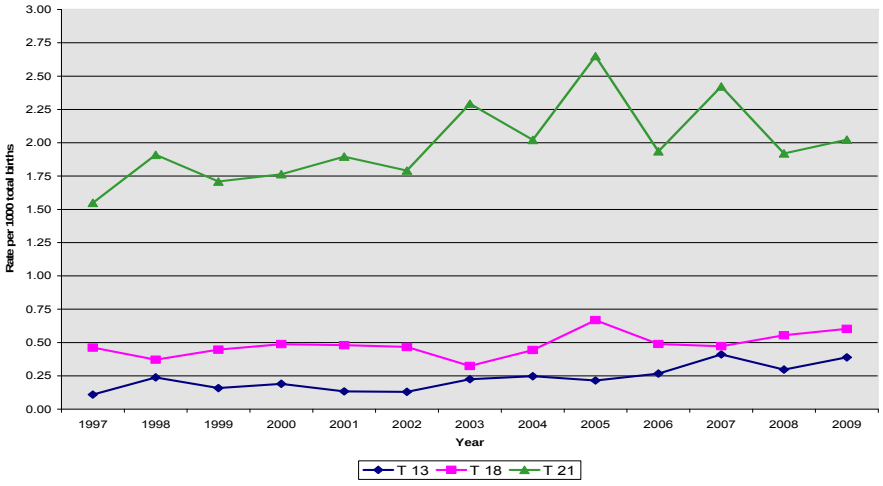


Table 4.2.1 Down Syndrome Rates per 1000 Total Births (Live + Still + ToP) By Maternal Age, 2005–2009

Maternal Age	Year				
	2005	2006	2007	2008	2009
<20	1.85	0	0.80	0.41	0.84
20–24	0.62	0.81	0.76	0.66	0.79
25–29	1.52	0.99	1.03	0.86	1.02
30–34	2.06	1.30	1.94	1.15	1.97
35–39	6.62	5.22	6.31	4.80	4.26
≥40	20.79	17.35	21.49	21.57	14.04
All ages	2.65	1.94	2.42	1.92	2.02

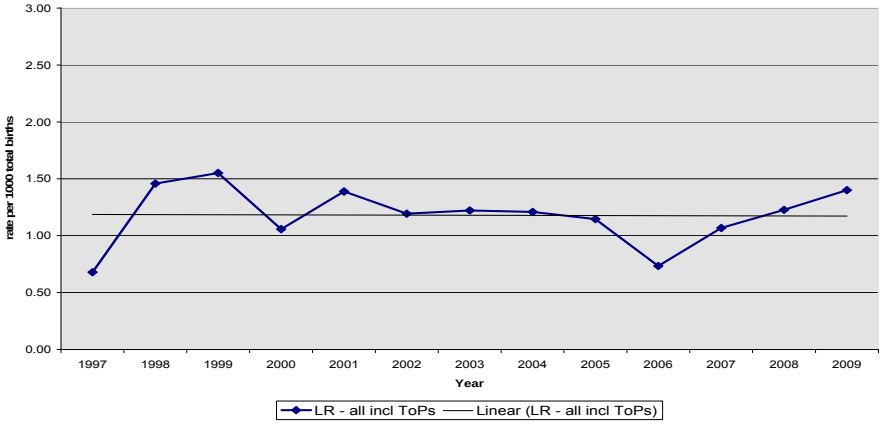
ACASS data were included in an ICBDSR study of ascertainment of Down Syndrome which showed that we compared favourably to many other surveillance systems (Leoncini et al, 2010[ix])

Infants with Down Syndrome often have associated anomalies. ACASS does not code minor anomalies associated with Down syndrome such as single palmar crease, upslanting palpebral fissures, and increased space between the first and second toes. However, other major malformations, if mentioned on the ascertainment documents, are entered routinely into the database.

4.2.6 Limb Reductions

Birth prevalence rates for limb reductions have remained stable from 1997–2009 (**Figure 4.2.10**) with no significant trend evident. ACASS continues to participate in an international study, co-ordinated through the ICBDSR, on the epidemiology of very rare defects. Some of the more uncommon limb reduction defects such as true phocomelia (absence of all limb bones proximal to the hand or foot - the hand or foot attaching directly to the trunk) and amelia (complete absence of one or more limbs) are included.

Figure 4.2.10 Limb Reduction Defects 1997–2009

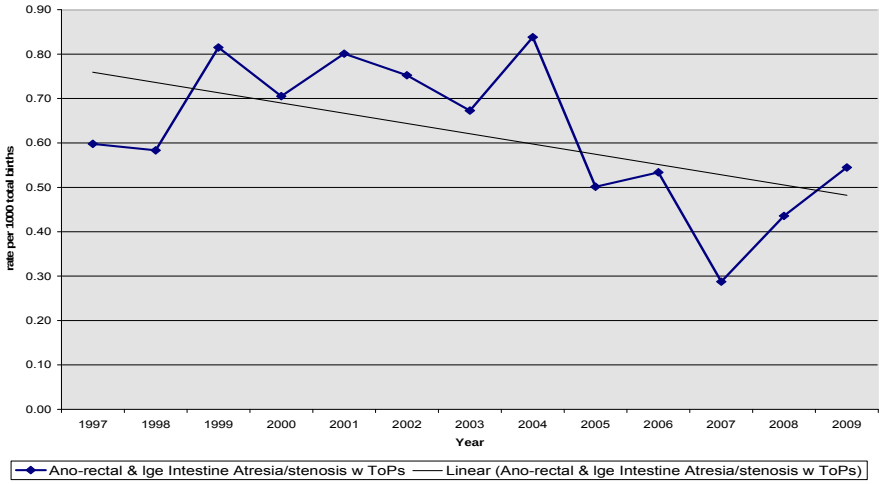


4.2.7 Anorectal Atresia/Stenosis

Anorectal malformations have been decreasing since 1997 (**Figure 4.2.11**). The rates, which appeared to increase in 1998–1999 in both Alberta and Canadian National data, prompted a detailed survey of Alberta data. The results were subsequently published (Lowry et al, 2007[x]) and at that time showed no overall trend. The results indicated that a substantial number of cases belonged in the multiple congenital anomaly VATER/VACTERL.

Our prevalence rates are very comparable to two other large population studies, one from British Columbia and the other from the EUROCAT registries with frequencies in the 1/2200 – 1/2500 range (Spouge et al, 1986[xi]; Cuschieri, 2001[xii]; Cuschieri, 2002[xiii]).

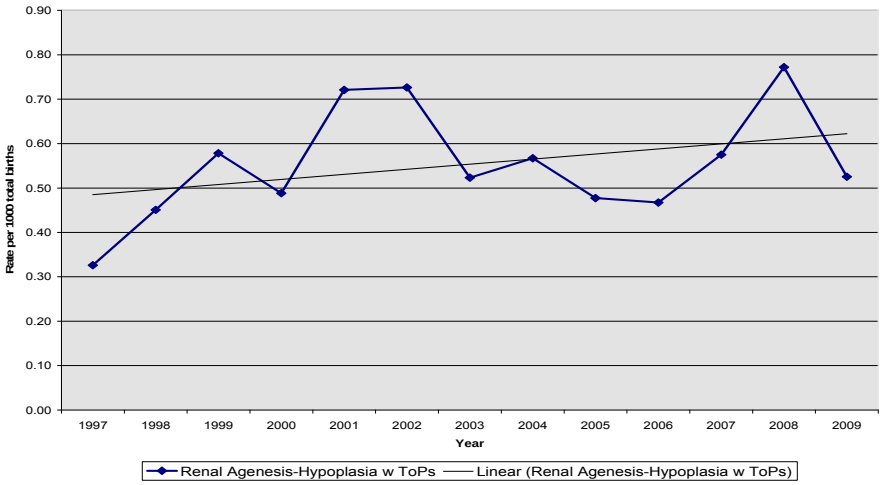
Figure 4.2.11 Anorectal and Large Intestine Atresia/Stenosis1997–2009



4.2.8 Renal Agenesis/Hypoplasia

These rates continue to show an increasing trend. This is likely due to better ascertainment as a result of more ultrasound investigations. (**Figure 4.2.12**).

Figure 4.2.12 Renal Agenesis/Hypoplasia 1997–2009



4.2.9 Congenital Heart Disease

Tetralogy of Fallot (**Figure 4.2.13**) appears to be showing an increase over time whereas Hypoplastic Left Heart Syndrome (**Figure 4.2.14**) shows no significant increase between the years 1997–2009. There is a slight upward trend for both atrial and ventricular septal defects.

ACASS personnel have completed a Quality Assurance Study of congenital heart defects (CHD) for the years 1995–2002. A secondary objective of the study was to see whether folic acid fortification has made any difference to CHD rates, since recent publications suggest that folic acid may have a preventive effect on CHDs and specific types of CHDs.

The overall ascertainment of CHD by ACASS was 45 per cent when compared with the additional ascertainment sources (University of Alberta Pediatric Cardiology via the Western Canadian Children's Heart Network's database, Alberta Children's Hospital Departments of Pediatric Cardiology and Pathology, and the Royal Alexandra Hospital's health records). The ascertainment of the most severe defects ranged from 87.5–100 per cent. Lower ascertainment rates were reported for less severe anomalies which may have been identified outside of ACASS' ascertainment window of one year after delivery. These anomalies include bicuspid aortic valve (15.1 per cent), PDA (21.1 per cent), aortic valve stenosis (26.3 per cent), pulmonary valve stenosis (28.1 per cent) and septal defects (37.5 per cent).

The estimated prevalence of CHD cases in Alberta during 1995–2002 using ACASS cases only was 5.59/1000 total births. When cases from ACASS and the additional sources of ascertainment were included, the prevalence rose to 12.42/1000 total births. The prevalence rates of severe CHDs remained stable throughout the study period and when pre and post folic acid fortification periods were compared. Significant increases were identified for ASDs (32 per cent) and cases with both an ASD and VSD (60 per cent) in the post folic acid fortification period. Left ventricular outflow tract obstruction significantly declined in the post folic acid fortification period (24 per cent). These significant changes were also noted throughout the study period, suggesting that perhaps alternative factors, such as differences in diagnostic and ascertainment practices are responsible for these changes, particularly for rates involving ASDs.

Figure 4.2.13 Tetralogy of Fallot 1997–2009

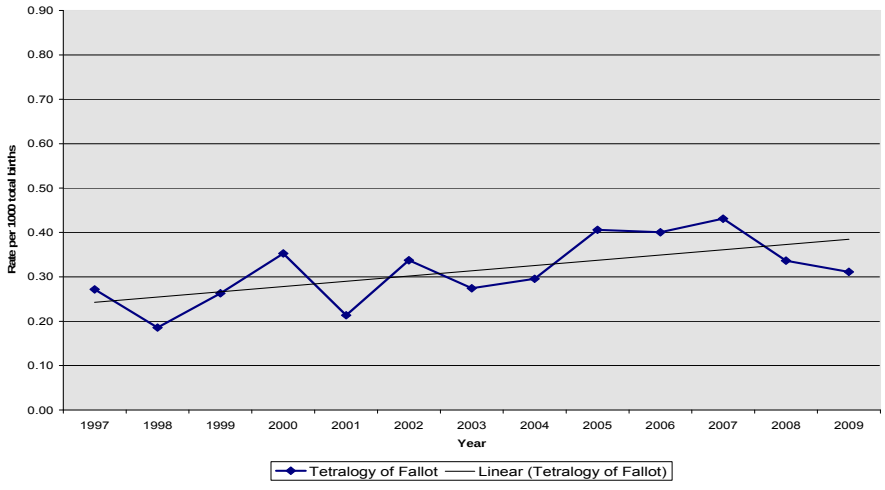
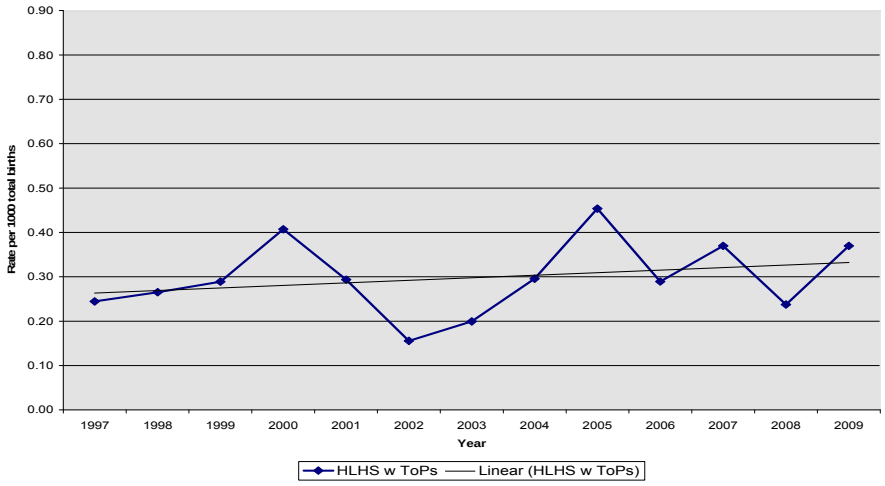


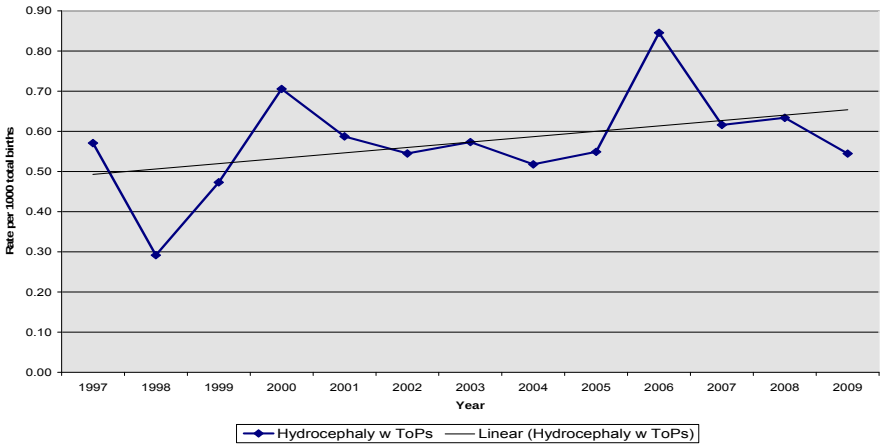
Figure 4.2.14 Hypoplastic Left Heart Syndrome 1997– 2009



4.2.10 Hydrocephalus

Although there appears to be a slight increase in the rates of hydrocephalus since 1997, the trend is not statistically significant (p=0.13).

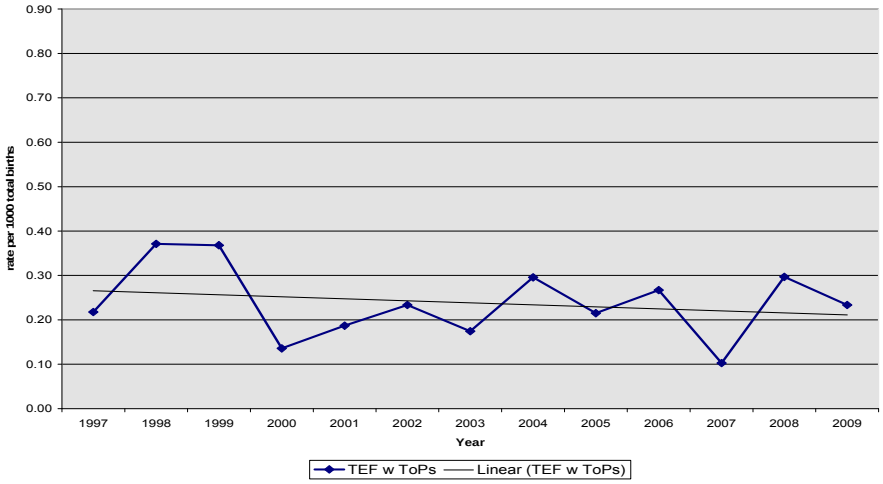
Figure 4.2.15 Hydrocephalus 1997–2009



4.2.11 Oesophageal Atresia/Stenosis

There has been no significant change in the rates of oesophageal atresia and stenosis with or without a tracheo-oesophageal fistula since 1997.

Figure 4.2.16 Oesophageal Atresia/Stenosis 1997–2009



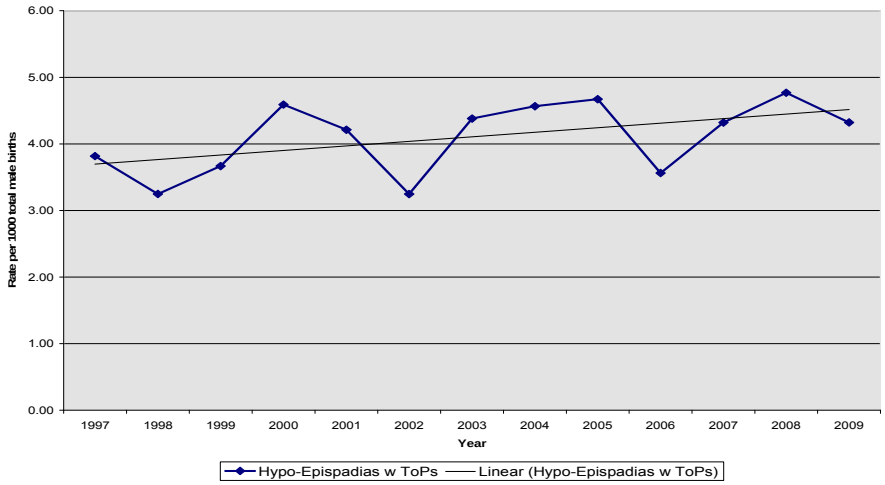
4.2.12 Hypospadias and Epispadias

Ascertainment of hypospadias includes all degrees of severity but excludes congenital chordee without hypospadias. Epispadias is included in the total, originally because the latter had the same three digit code in ICD-9 but also to conform with reporting in the NBDPN. ICD-10 has a separate code for epispadias (Q64.0) whereas different degrees of hypospadias are under Q54. For the 1997–2009 period epispadias represents three per cent of the total (36/1149). Among the 36 there are 25 cases of isolated epispadias, six with bladder exstrophy and five with multiple anomalies. The current data and graph from 1997 is chosen because ToPs and fetal losses are included although the latter make up a very small proportion of the total (only five). It does show a mildly significant rise (p=0.0353) which should be interpreted with caution because our earlier period (1980–2007) showed no significant change. It should also be noted that our rates are expressed as total male births in contrast to many rates in the literature which are cited as live births and do not differentiate the male proportion.

Clearly even a modest increasing trend deserves further study particularly because of the concern in the 1970's and 80's that hypospadias was increasing. Various causes were attributed to that including endocrine disruption factors such as pesticides, herbicides, phthalates, vegetarian diets (phytoestrogens) as well as the breakdown of plastic components (Baskin et al, 2001 [xiv]). Thus there is concern for agricultural workers. The concerns about increasing rates of hypospadias seem to have abated (Fisch et al, 2009 [xv]) but clearly surveillance of more years is necessary before we can conclude that the current increase is sustained.

Dolk et al (2004 [xvi]) have discussed some of the problems of effective surveillance for this anomaly and listed a number of recommendations to improve surveillance reliability. A study in Finland (Aho et al 2000 [xvii]) found that the prevalence there remained constant for the 1970–1986 period though higher than previously reported which was likely due to the incomplete ascertainment. It is of interest that our current 10-year prevalence rate of 4.27 per 1000 total male births is very comparable to that of a study in British Columbia (Leung et al, 1985 [xviii]) where the prevalence was 4.44/1000 male live births for the 1966–81 period. A recent study showed no association has been found between maternal pre-pregnancy obesity and the risk of hypospadias (Adams et al 2011 [xix]).

Figure 4.2.17 Hypospadias/Epispadias 1997–2009



4.3 Summary

ACASS reviews anomalies that have been entered into the database on a regular basis. Detailed studies of some individual anomalies or anomaly groups aid in the assessment and maintenance of the data quality. With intensive review, some cases might be reassigned, recoded or discarded altogether from the database. This continuing review might explain some discrepancies in the data from earlier reports. As well, the current report includes ToPs/fetal losses in the rates for better estimates of true rates. This change from previous reports in which live births and stillbirths only were reported, might also explain potential discrepancies in the data.

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Cleft Lip and Palate

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5. SURVEILLANCE AND RESEARCH PROJECTS

5.1 Surveillance and Research Projects/Collaborations and Consultations/Papers

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    iii. Sibbald B and Lowry RB Down Syndrome in Alberta: Alberta Congenital Anomalies Surveillance System (fall 2003) http://www.phac-aspc.gc.ca/ccasn-rcsac/ct2003/abds_e.html

Alberta Congenital Anomalies Surveillance System

6. Appendices

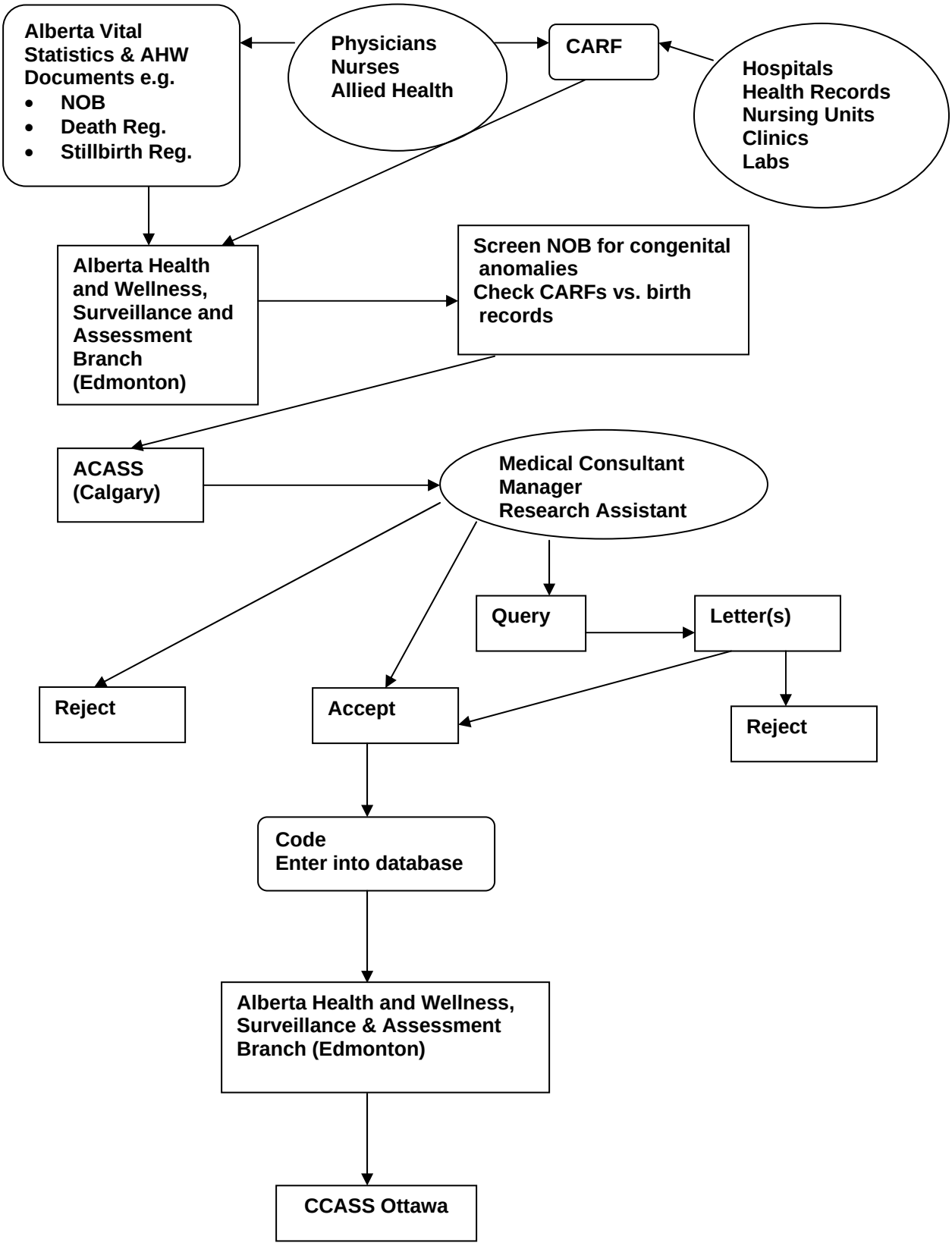
Appendix A.1 Flowchart of the Process of ACASS Data Collection

Appendix A.2 Congenital Anomaly(ies) Reporting Form (CARF)

Appendix A.3 Single and Aggregate Year Anomaly rates

Appendix A.4 Numbers of cases, anomalies and anomalies per case 1980–2004

Appendix A.1 Flowchart of the Process of ACASS Data Collection



Appendix A.2 Congenital Anomaly(ies) Reporting Form (CARF)

Alberta Health and Wellness

Death Reg No	Birth Reg No

Addressograph

Mail parts one and two to:
Alberta Health and Wellness
Surveillance and Environmental Health Branch
PO Box 1360 Stn Main
Edmonton AB T5J 2N3

Congenital Anomaly(ies) Reporting

Fetus / Infant — **PLEASE PRINT CLEARLY**

Name *(Last, First, Initial)*	**Date of Birth** *Month by Name* Day Year

Gender ☐ Male ☐ Female ☐ Unknown	Type of Birth ☐ Livebirth ☐ Stillbirth ☐ Fetus less than 20 weeks gestation	Name of Hospital of Birth
Birthweight *Grams*	Gestation Age *(Completed Weeks)*	Location of Hospital of Birth *(City/Town)*
Child's Personal Health Number		Attending Physician's Name
Plurality of Birth ☐ Single ☐ Twin ☐ First ☐ Second ☐ Triplets ☐ First ☐ Second ☐ Third		Physician Responsible for Ongoing Care *(if different from above)*

Parents

Mother's Name *(Last, First, Maiden)*	**Date of Birth or Age** *(if DOB unavailable)* *Month by Name* Day Year	**Total Number of**
Permanent Address	Mother's Personal Health Number	Livebirths
City/Town	Postal Code	Stillbirths
Father's Name *(Last, First, Initial)*	**Date of Birth or Age** *(if DOB unavailable)* *Month by Name* Day Year	Spontaneous Abortions
		Therapeutic Abortions

Reporting Hospital/Agency/Clinic

Name	Infant's Admission *(If different from birthdate)* *Month by Name* Day Year	Infant's Discharge *Month by Name* Day Year
Location *(City/Town)*		Infant's Death *(If Applicable)* *Month by Name* Day Year

Full description of Congenital Anomaly(ies) and/or **SYNDROME DIAGNOSES** *(If necessary, please attach supporting documents.)*

OFFICE USE ONLY

Completed by	Position	Date

HS0020-112 (2008/06)

Send to Surveillance and Environmental Health

Appendix A.3 Alberta Congenital Anomalies Surveillance System
Q Chapter (Q00–Q99)

Total Births (Live, still and fetal losses) Single Year Anomaly Rates per 1,000 Total Births

Diagnostic Category		2005	2006	2007	2008	2009
Anencephaly	NUMBER	9	9	9	10	18
	RATE	**0.21**	**0.20**	**0.18**	**0.20**	**0.35**
	Lower CI	0.10	0.09	0.08	0.10	0.21
ICD-10 Q00.00, Q00.01, Q00.1	Upper CI	0.41	0.38	0.35	0.36	0.55
Spina Bifida without Anencephaly	NUMBER	12	26	20	19	21
	RATE	**0.29**	**0.58**	**0.41**	**0.38**	**0.41**
	Lower CI	0.15	0.38	0.25	0.23	0.25
ICD-10 Q05..	Upper CI	0.50	0.85	0.63	0.59	0.62
Encephalocele	NUMBER	9	2	5	7	7
	RATE	**0.21**	**0.04**	**0.10**	**0.14**	**0.14**
	Lower CI	0.10	0.01	0.03	0.06	0.05
ICD-10 Q01..	Upper CI	0.41	0.15	0.24	0.28	0.28
Neural Tube Defects (all)	NUMBER	30	37	35	36	46
	RATE	**0.72**	**0.82**	**0.72**	**0.71**	**0.89**
	Lower CI	0.48	0.58	0.50	0.50	0.66
ICD-10 Q00.., Q01.., Q05..	Upper CI	1.02	1.13	1.00	0.99	1.19
Hydrocephalus without Spina Bifida (Excl. hydranencephaly)	NUMBER	23	38	30	32	28
	RATE	**0.55**	**0.85**	**0.62**	**0.63**	**0.54**
	Lower CI	0.35	0.60	0.42	0.43	0.36
ICD-10 Q03	Upper CI	0.82	1.16	0.88	0.89	0.79
Microcephaly	NUMBER	24	16	17	18	21
	RATE	**0.57**	**0.36**	**0.35**	**0.36**	**0.41**
	Lower CI	0.37	0.20	0.20	0.21	0.25
ICD-10 Q02	Upper CI	0.85	0.58	0.56	0.56	0.62
Anophthalmia/microphthalmia	NUMBER	14	5	7	10	4
	RATE	**0.33**	**0.11**	**0.14**	**0.20**	**0.08**
	Lower CI	0.18	0.04	0.06	0.10	0.02
ICD-10 Q11.0, Q11.1, Q11.2	Upper CI	0.56	0.26	0.29	0.36	0.19
Congenital cataract	NUMBER	5	4	9	4	11
	RATE	**0.12**	**0.09**	**0.18**	**0.08**	**0.21**
	Lower CI	0.04	0.02	0.08	0.02	0.11
ICD-10 Q12.0	Upper CI	0.27	0.22	0.35	0.20	0.38
Aniridia	NUMBER	0	0	0	1	1
	RATE				**0.02**	**0.02**
	Lower CI				0.00	0.00
ICD-10 Q13.1	Upper CI				0.10	0.10
Anotia/microtia	NUMBER	12	8	13	11	16
	RATE	**0.29**	**0.18**	**0.27**	**0.22**	**0.31**
	Lower CI	0.15	0.08	0.14	0.11	0.18
ICD-10 Q16.0, Q17.2	Upper CI	0.50	0.35	0.46	0.39	0.50

Appendix A.3 Alberta Congenital Anomalies Surveillance System
Q Chapter (Q00–Q99)

Total Births (Live, still and fetal losses) Aggregate Year Anomaly Rates per 1,000 Total Births

Diagnostic Category		97-99 (3 years)	00-04 (5 years)	05-09 (5 years)	00-09 (10 years)
Anencephaly	NUMBER	37	48	55	103
	RATE	**0.33**	**0.25**	**0.23**	**0.24**
	Lower CI	0.23	0.18	0.17	0.20
ICD-10 Q00.00, Q00.01, Q00.1	Upper CI	0.45	0.33	0.30	0.29
Spina Bifida without Anencephaly	NUMBER	52	57	98	155
	RATE	**0.46**	**0.29**	**0.41**	**0.36**
	Lower CI	0.35	0.22	0.34	0.31
ICD-10 Q05..	Upper CI	0.61	0.38	0.50	0.42
Encephalocele	NUMBER	13	28	30	58
	RATE	**0.12**	**0.14**	**0.13**	**0.13**
	Lower CI	0.06	0.10	0.09	0.10
ICD-10 Q01..	Upper CI	0.20	0.21	0.18	0.17
Neural Tube Defects (all)	NUMBER	104	133	184	317
	RATE	**0.92**	**0.69**	**0.77**	**0.74**
	Lower CI	0.76	0.58	0.67	0.66
ICD-10 Q00.., Q01.., Q05..	Upper CI	1.12	0.81	0.90	0.82
Hydrocephalus without Spina Bifida (Excl. hydranencephaly)	NUMBER	50	113	151	264
	RATE	**0.44**	**0.58**	**0.64**	**0.61**
	Lower CI	0.33	0.48	0.54	0.54
ICD-10 Q03	Upper CI	0.59	0.70	0.75	0.69
Microcephaly	NUMBER	36	76	96	172
	RATE	**0.32**	**0.39**	**0.40**	**0.40**
	Lower CI	0.22	0.31	0.33	0.34
ICD-10 Q02	Upper CI	0.44	0.49	0.49	0.46
Anophthalmia/microphthalmia	NUMBER	25	30	40	70
	RATE	**0.22**	**0.16**	**0.17**	**0.16**
	Lower CI	0.14	0.10	0.12	0.13
ICD-10 Q11.0, Q11.1, Q11.2	Upper CI	0.33	0.22	0.23	0.21
Congenital cataract	NUMBER	21	16	33	49
	RATE	**0.19**	**0.08**	**0.14**	**0.11**
	Lower CI	0.12	0.05	0.10	0.08
ICD-10 Q12.0	Upper CI	0.29	0.13	0.20	0.15
Aniridia	NUMBER	2	3	2	5
	RATE	**0.02**	**0.02**	**0.01**	**0.01**
	Lower CI	0.00	0.00	0.00	0.00
ICD-10 Q13.1	Upper CI	0.06	0.04	0.03	0.03
Anotia/microtia	NUMBER	18	40	60	100
	RATE	**0.16**	**0.21**	**0.25**	**0.23**
	Lower CI	0.09	0.15	0.19	0.19
ICD-10 Q16.0, Q17.2	Upper CI	0.25	0.28	0.33	0.28

Appendix A.3 Alberta Congenital Anomalies Surveillance System
Q Chapter (Q00–Q99)

Total Births (Live, still and fetal losses) Single Year Anomaly Rates per 1,000 Total Births

Diagnostic Category		2005	2006	2007	2008	2009
Common Truncus	NUMBER	2	2	5	3	2
Excludes AP window	**RATE**	**0.05**	**0.04**	**0.10**	**0.06**	**0.04**
	Lower CI	0.01	0.01	0.03	0.01	0.00
ICD-10 Q20.0	Upper CI	0.16	0.15	0.24	0.17	0.13
Transposition of Great Arteries	NUMBER	20	15	13	14	12
	RATE	**0.48**	**0.33**	**0.27**	**0.28**	**0.23**
	Lower CI	0.29	0.19	0.14	0.15	0.12
ICD-10 Q20.11, Q20.3, Q20.5	Upper CI	0.74	0.55	0.46	0.46	0.41
Tetralogy of Fallot	NUMBER	17	18	21	17	16
	RATE	**0.41**	**0.40**	**0.43**	**0.34**	**0.31**
	Lower CI	0.24	0.24	0.27	0.20	0.18
ICD-10 Q21.3.., Q21.82	Upper CI	0.65	0.63	0.66	0.54	0.50
Ventricular Septal Defect	NUMBER	148	121	139	143	157
	RATE	**3.53**	**2.69**	**2.85**	**2.83**	**3.05**
	Lower CI	2.99	2.23	2.40	2.39	2.60
ICD-10 Q21.0	Upper CI	4.15	3.22	3.37	3.33	3.57
Atrial Septal Defect	NUMBER	87	61	73	71	95
	RATE	**2.08**	**1.36**	**1.50**	**1.41**	**1.85**
	Lower CI	1.66	1.04	1.18	1.10	1.50
ICD-10 Q21.1..	Upper CI	2.56	1.74	1.88	1.77	2.26
Endocardial Cushion Defect	NUMBER	15	13	26	24	19
	RATE	**0.36**	**0.29**	**0.53**	**0.48**	**0.37**
	Lower CI	0.20	0.15	0.35	0.30	0.22
ICD-10 Q21.2..	Upper CI	0.59	0.49	0.78	0.71	0.58
Pulmonary Valve Atresia and Stenosis	NUMBER	27	20	33	24	27
	RATE	**0.64**	**0.44**	**0.68**	**0.48**	**0.53**
	Lower CI	0.43	0.27	0.47	0.30	0.35
ICD-10 Q22.0, Q22.1	Upper CI	0.94	0.69	0.95	0.71	0.76
Tricuspid Valve Atresia and Stenosis	NUMBER	1	4	7	4	5
	RATE	**0.02**	**0.09**	**0.14**	**0.08**	**0.10**
	Lower CI	0.00	0.02	0.06	0.02	0.03
ICD-10 Q22.4	Upper CI	0.12	0.22	0.29	0.20	0.22
Ebstein's Anomaly	NUMBER	1	4	2	3	3
	RATE	**0.02**	**0.09**	**0.04**	**0.06**	**0.06**
	Lower CI	0.00	0.02	0.00	0.01	0.01
ICD-10 Q22.5	Upper CI	0.12	0.22	0.14	0.17	0.17
Aortic Valve Stenosis	NUMBER	4	6	10	12	8
	RATE	**0.10**	**0.13**	**0.25**	**0.20**	**0.16**
	Lower CI	0.03	0.05	0.13	0.10	0.07
ICD-10 Q23.0, Q24.4	Upper CI	0.24	0.29	0.43	0.36	0.30

Appendix A.3 Alberta Congenital Anomalies Surveillance System
Q Chapter (Q00–Q99)

Total Births (Live, still and fetal losses) Aggregate Year Anomaly Rates per 1,000 Total Births

Diagnostic Category		97-99 (3 years)	00-04 (5 years)	05-09 (5 years)	00-09 (10 years)
Common Truncus	NUMBER	6	14	14	28
Excludes AP window	**RATE**	**0.05**	**0.07**	**0.06**	**0.06**
	Lower CI	0.02	0.04	0.03	0.04
ICD-10 Q20.0	Upper CI	0.11	0.12	0.10	0.09
Transposition of Great Arteries	NUMBER	39	78	74	152
	RATE	**0.35**	**0.40**	**0.31**	**0.35**
	Lower CI	0.25	0.32	0.24	0.30
ICD-10 Q20.11, Q20.3, Q20.5	Upper CI	0.47	0.50	0.39	0.41
Tetralogy of Fallot	NUMBER	27	57	89	146
	RATE	**0.24**	**0.29**	**0.37**	**0.34**
	Lower CI	0.16	0.22	0.30	0.29
ICD-10 Q21.3.., Q21.82	Upper CI	0.35	0.38	0.46	0.40
Ventricular Septal Defect	NUMBER	285	635	708	1343
	RATE	**2.53**	**3.28**	**2.98**	**3.12**
	Lower CI	2.25	3.03	2.77	2.95
ICD-10 Q21.0	Upper CI	2.84	3.55	3.21	3.29
Atrial Septal Defect	NUMBER	231	458	387	845
	RATE	**2.05**	**2.37**	**1.63**	**1.96**
	Lower CI	1.80	2.15	1.47	1.83
ICD-10 Q21.1..	Upper CI	2.33	2.59	1.80	2.10
Endocardial Cushion Defect	NUMBER	53	99	97	196
	RATE	**0.47**	**0.51**	**0.41**	**0.45**
	Lower CI	0.35	0.42	0.33	0.39
ICD-10 Q21.2..	Upper CI	0.62	0.62	0.50	0.52
Pulmonary Valve Atresia and Stenosis	NUMBER	74	124	131	255
	RATE	**0.66**	**0.64**	**0.55**	**0.59**
	Lower CI	0.52	0.53	0.46	0.52
ICD-10 Q22.0, Q22.1	Upper CI	0.83	0.76	0.65	0.67
Tricuspid Valve Atresia and Stenosis	NUMBER	10	13	21	34
	RATE	**0.09**	**0.07**	**0.09**	**0.08**
	Lower CI	0.04	0.04	0.05	0.05
ICD-10 Q22.4	Upper CI	0.16	0.11	0.14	0.11
Ebstein's Anomaly	NUMBER	7	14	13	27
	RATE	**0.06**	**0.07**	**0.05**	**0.06**
	Lower CI	0.03	0.04	0.03	0.04
ICD-10 Q22.5	Upper CI	0.13	0.12	0.09	0.09
Aortic Valve Stenosis	NUMBER	41	82	40	122
	RATE	**0.36**	**0.42**	**0.17**	**0.28**
	Lower CI	0.26	0.34	0.12	0.24
ICD-10 Q23.0, Q24.4	Upper CI	0.49	0.53	0.23	0.34

Appendix A.3 Alberta Congenital Anomalies Surveillance System
Q Chapter (Q00–Q99)

Total Births (Live, still and fetal losses) Single Year Anomaly Rates per 1,000 Total Births

Diagnostic Category		2005	2006	2007	2008	2009
Hypoplastic Left Heart Syndrome	NUMBER	19	13	18	12	19
	RATE	**0.45**	**0.29**	**0.37**	**0.24**	**0.37**
	Lower CI	0.27	0.15	0.22	0.12	0.22
ICD-10 Q23.4	Upper CI	0.71	0.49	0.58	0.41	0.58
Coarctation of the Aorta	NUMBER	16	13	19	29	21
	RATE	**0.38**	**0.29**	**0.39**	**0.57**	**0.41**
	Lower CI	0.22	0.15	0.24	0.38	0.25
ICD-10 Q25.1..	Upper CI	0.62	0.49	0.61	0.82	0.62
Cleft Palate without Cleft Lip	NUMBER	30	27	26	40	31
	RATE	**0.72**	**0.60**	**0.53**	**0.79**	**0.60**
	Lower CI	0.48	0.40	0.35	0.57	0.41
ICD-10 Q35..	Upper CI	1.02	0.87	0.78	1.08	0.86
Cleft Lip with and without Cleft Palate	NUMBER	48	45	82	65	70
	RATE	**1.15**	**1.00**	**1.68**	**1.29**	**1.36**
	Lower CI	0.85	0.73	1.34	0.99	1.06
ICD-10 Q36.., Q37..	Upper CI	1.52	1.34	2.09	1.64	1.72
Choanal Atresia	NUMBER	7	8	5	6	9
	RATE	**0.17**	**0.18**	**0.10**	**0.12**	**0.18**
	Lower CI	0.07	0.08	0.03	0.04	0.08
ICD-10 Q30.0..	Upper CI	0.34	0.35	0.24	0.26	0.33
Oesophageal Atresia/ Tracheo-oesphageal Fistula	NUMBER	9	12	5	15	12
	RATE	**0.21**	**0.27**	**0.10**	**0.30**	**0.23**
	Lower CI	0.10	0.14	0.03	0.17	0.12
ICD-10 Q39.0 – Q39.4	Upper CI	0.41	0.46	0.24	0.49	0.41
Rectal and Large Intestinal Atresia/Stenosis	NUMBER	21	24	14	22	28
	RATE	**0.50**	**0.53**	**0.29**	**0.44**	**0.54**
	Lower CI	0.31	0.34	0.16	0.27	0.36
ICD-10 Q42..	Upper CI	0.77	0.79	0.48	0.66	0.79
Pyloric Stenosis	NUMBER	42	43	51	57	53
	RATE	**1.00**	**0.96**	**1.05**	**1.13**	**1.03**
	Lower CI	0.72	0.69	0.78	0.86	0.77
ICD-10 Q40.0	Upper CI	1.36	1.29	1.38	1.46	1.35
Hirschsprung Disease	NUMBER	5	12	6	6	8
	RATE	**0.12**	**0.27**	**0.12**	**0.12**	**0.16**
	Lower CI	0.04	0.14	0.05	0.04	0.07
ICD-10 Q43.1..	Upper CI	0.27	0.46	0.26	0.26	0.30
Biliary Atresia	NUMBER	5	2	3	2	5
	RATE	**0.12**	**0.04**	**0.06**	**0.04**	**0.10**
	Lower CI	0.04	0.01	0.01	0.00	0.03
ICD-10 Q44.2	Upper CI	0.27	0.15	0.17	0.14	0.22

Appendix A.3 Alberta Congenital Anomalies Surveillance System
Q Chapter (Q00–Q99)

Total Births (Live, still and fetal losses) Aggregate Year Anomaly Rates per 1,000 Total Births

Diagnostic Category		97-99 (3 years)	00-04 (5 years)	05-09 (5 years)	00-09 (10 years)
Hypoplastic Left Heart Syndrome	NUMBER	30	52	81	133
	RATE	**0.27**	**0.27**	**0.34**	**0.31**
	Lower CI	0.18	0.20	0.27	0.26
ICD-10 Q23.4	Upper CI	0.38	0.35	0.42	0.37
Coarctation of the Aorta	NUMBER	38	63	98	161
	RATE	**0.34**	**0.33**	**0.41**	**0.37**
	Lower CI	0.24	0.25	0.34	0.32
ICD-10 Q25.1..	Upper CI	0.26	0.42	0.50	0.44
Cleft Palate without Cleft Lip	NUMBER	106	158	154	312
	RATE	**0.94**	**0.82**	**0.65**	**0.72**
	Lower CI	0.77	0.69	0.55	0.65
ICD-10 Q35..	Upper CI	1.14	0.95	0.76	0.81
Cleft Lip with and without Cleft Palate	NUMBER	122	243	310	553
	RATE	**1.08**	**1.26**	**1.31**	**1.28**
	Lower CI	0.90	1.10	1.16	1.18
ICD-10 Q36.., Q37..	Upper CI	1.29	1.42	1.46	1.39
Choanal Atresia	NUMBER	13	41	35	76
	RATE	**0.12**	**0.21**	**0.15**	**0.18**
	Lower CI	0.06	0.15	0.10	0.14
ICD-10 Q30.0..	Upper CI	0.20	0.29	0.21	0.22
Oesophageal Atresia/ Tracheo-oesphageal Fistula	NUMBER	36	40	53	93
	RATE	**0.32**	**0.21**	**0.22**	**0.22**
	Lower CI	0.22	0.15	0.17	0.17
ICD-10 Q39.0 – Q39.4	Upper CI	0.44	0.28	0.29	0.26
Rectal and Large Intestinal Atresia/Stenosis	NUMBER	75	146	109	255
	RATE	**0.67**	**0.75**	**0.46**	**0.59**
	Lower CI	0.52	0.64	0.38	0.52
ICD-10 Q42..	Upper CI	0.83	0.89	0.55	0.67
Pyloric Stenosis	NUMBER	76	179	246	425
	RATE	**0.68**	**0.92**	**1.04**	**0.99**
	Lower CI	0.53	0.79	0.91	0.89
ICD-10 Q40.0	Upper CI	0.85	1.07	1.17	1.08
Hirschsprung Disease	NUMBER	14	23	37	60
	RATE	**0.12**	**0.12**	**0.16**	**0.14**
	Lower CI	0.07	0.08	0.11	0.11
ICD-10 Q43.1..	Upper CI	0.21	0.18	0.21	0.18
Biliary Atresia	NUMBER	7	12	17	29
	RATE	**0.06**	**0.06**	**0.07**	**0.07**
	Lower CI	0.03	0.03	0.04	0.05
ICD-10 Q44.2	Upper CI	0.13	0.11	0.11	0.10

Appendix A.3 Alberta Congenital Anomalies Surveillance System
Q Chapter (Q00–Q99)

Total Births (Live, still and fetal losses) Single Year Anomaly Rates per 1,000 Total Births

Diagnostic Category		2005	2006	2007	2008	2009
Renal Agenesis/Hypoplasia	NUMBER	20	21	28	39	27
	RATE	**0.48**	**0.47**	**0.57**	**0.77**	**0.53**
	Lower CI	0.29	0.29	0.38	0.55	0.35
ICD-10 Q60..	Upper CI	0.74	0.71	0.83	1.06	0.76
Bladder Exstrophy	NUMBER	1	1	2	1	1
	RATE	**0.02**	**0.02**	**0.04**	**0.02**	**0.02**
	Lower CI	0.00	0.00	0.00	0.00	0.00
ICD-10 Q64.1 (excl Q64.10)	Upper CI	0.12	0.11	0.14	0.10	0.10
Obstructive Genitourinary Defect	NUMBER	101	102	117	139	133
	RATE	**2.41**	**2.27**	**2.40**	**2.75**	**2.59**
	Lower CI	1.96	1.85	1.99	2.31	2.17
ICD-10 Q62.0 – Q62.3, Q64.2, Q64.3	Upper CI	2.93	2.75	2.88	3.25	3.07
Hypospadias/Epispadias	NUMBER	100	82	107	124	114
denominator male births only	**RATE**	**4.67**	**3.56**	**4.32**	**4.77**	**4.32**
	Lower CI	3.80	2.83	3.54	3.97	3.57
ICD-10 Q54 (excl. Q54.4), Q64.0	Upper CI	5.68	4.42	5.22	5.68	5.19
Reduction Deformity, Upper Limbs	NUMBER	31	19	34	40	47
	RATE	**0.74**	**0.42**	**0.70**	**0.79**	**0.91**
	Lower CI	0.50	0.25	0.48	0.57	0.67
ICD-10 Q71..	Upper CI	1.05	0.66	0.98	1.08	1.22
Reduction Deformity, Lower Limbs	NUMBER	17	14	18	22	25
	RATE	**0.41**	**0.31**	**0.37**	**0.44**	**0.49**
	Lower CI	0.24	0.17	0.22	0.27	0.32
ICD-10 Q72..	Upper CI	0.65	0.52	0.58	0.66	0.72
Gastroschisis	NUMBER	24	27	25	23	26
	RATE	**0.57**	**0.60**	**0.51**	**0.46**	**0.51**
	Lower CI	0.37	0.40	0.33	0.29	0.33
ICD-10 Q79.3	Upper CI	0.85	0.87	0.76	0.68	0.74
Omphalocele	NUMBER	10	10	11	19	22
	RATE	**0.24**	**0.22**	**0.23**	**0.38**	**0.43**
	Lower CI	0.11	0.11	0.11	0.23	0.27
ICD-10 Q79.2	Upper CI	0.44	0.41	0.40	0.59	0.65
Congenital Hip Dislocation	NUMBER	64	82	95	123	109
(incl. dysplasia)	**RATE**	**1.53**	**1.82**	**1.95**	**2.44**	**2.12**
	Lower CI	1.18	1.45	1.58	2.02	1.74
ICD-10 Q65.0-Q65.6	Upper CI	1.95	2.26	2.38	2.91	2.56
Diaphragmatic Hernia	NUMBER	17	12	17	18	20
	RATE	**0.41**	**0.27**	**0.35**	**0.36**	**0.39**
	Lower CI	0.24	0.14	0.20	0.21	0.24
ICD-10 Q79.0.., Q79.11, Q79.12	Upper CI	0.65	0.46	0.56	0.56	0.60

Appendix A.3 Alberta Congenital Anomalies Surveillance System
Q Chapter (Q00–Q99)

Total Births (Live, still and fetal losses) Aggregate Year Anomaly Rates per 1,000 Total Births

Diagnostic Category		97-99 (3 years)	00-04 (5 years)	05-09 (5 years)	00-09 (10 years)
Renal Agenesis/Hypoplasia	NUMBER	51	117	135	252
	RATE	**0.45**	**0.60**	**0.57**	**0.58**
	Lower CI	0.34	0.50	0.48	0.51
ICD-10 Q60..	Upper CI	0.60	0.72	0.67	0.66
Bladder Exstrophy	NUMBER	3	9	6	15
	RATE	**0.03**	**0.05**	**0.03**	**0.03**
	Lower CI	0.01	0.02	0.01	0.02
ICD-10 Q64.1 (excl Q64.10)	Upper CI	0.08	0.09	0.05	0.06
Obstructive Genitourinary Defect	NUMBER	218	471	592	1063
	RATE	**1.94**	**2.43**	**2.49**	**2.47**
	Lower CI	1.69	2.22	2.30	2.32
ICD-10 Q62.0 – Q62.3, Q64.2, Q64.3	Upper CI	2.21	2.66	2.70	2.62
Hypospadias/Epispadias	NUMBER	206	416	527	943
denominator male births only	**RATE**	**3.58**	**4.20**	**4.33**	**4.27**
	Lower CI	3.10	3.81	3.97	4.01
ICD-10 Q54 (excl. Q54.4), Q64.0	Upper CI	4.10	4.62	4.72	4.56
Reduction Deformity, Upper Limbs	NUMBER	94	147	171	318
	RATE	**0.84**	**0.76**	**0.72**	**0.74**
	Lower CI	0.68	0.64	0.62	0.66
ICD-10 Q71..	Upper CI	1.02	0.89	0.84	0.82
Reduction Deformity, Lower Limbs	NUMBER	45	88	96	184
	RATE	**0.40**	**0.45**	**0.40**	**0.43**
	Lower CI	0.29	0.36	0.33	0.37
ICD-10 Q72..	Upper CI	0.54	0.56	0.49	0.49
Gastroschisis	NUMBER	30	63	125	188
	RATE	**0.27**	**0.33**	**0.53**	**0.44**
	Lower CI	0.18	0.25	0.44	0.38
ICD-10 Q79.3	Upper CI	0.38	0.42	0.63	0.50
Omphalocele	NUMBER	31	52	72	124
	RATE	**0.28**	**0.27**	**0.30**	**0.29**
	Lower CI	0.19	0.20	0.24	0.24
ICD-10 Q79.2	Upper CI	0.39	0.35	0.38	0.34
Congenital Hip Dislocation	NUMBER	206	428	473	901
(incl. dysplasia)	**RATE**	**1.83**	**2.21**	**1.99**	**2.09**
	Lower CI	1.59	2.01	1.82	1.96
ICD-10 Q65.0-Q65.6	Upper CI	2.10	2.43	2.18	2.23
Diaphragmatic Hernia	NUMBER	31	80	84	164
	RATE	**0.28**	**0.41**	**0.35**	**0.38**
	Lower CI	0.19	0.33	0.28	0.32
ICD-10 Q79.0.., Q79.11, Q79.12	Upper CI	0.39	0.51	0.44	0.44

Appendix A.3 Alberta Congenital Anomalies Surveillance System
Q Chapter (Q00–Q99)

Total Births (Live, still and fetal losses) Single Year Anomaly Rates per 1,000 Total Births

Diagnostic Category		2005	2006	2007	2008	2009
Trisomy 13	NUMBER	9	12	20	15	20
	RATE	**0.21**	**0.27**	**0.41**	**0.30**	**0.39**
	Lower CI	0.10	0.14	0.25	0.17	0.24
ICD-10 Q91.4-Q91.7	Upper CI	0.41	0.46	0.63	0.49	0.60
Down Syndrome (Trisomy 21)	NUMBER	111	87	118	97	104
	RATE	**2.65**	**1.94**	**2.42**	**1.92**	**2.02**
	Lower CI	2.18	1.55	2.01	1.56	1.65
ICD-10 Q90..	Upper CI	3.19	2.39	2.90	2.34	2.45
Trisomy 18	NUMBER	28	22	23	28	31
	RATE	**0.67**	**0.49**	**0.47**	**0.55**	**0.60**
	Lower CI	0.44	0.31	0.30	0.37	0.41
ICD-10 Q91.0-Q91.3	Upper CI	0.97	0.74	0.71	0.80	0.86

Appendix A.3 Alberta Congenital Anomalies Surveillance System
Q Chapter (Q00–Q99)

Total Births (Live, still and fetal losses) Aggregate Year Anomaly Rates per 1,000 Total Births

Diagnostic Category		97-99 (3 years)	00-04 (5 years)	05-09 (5 years)	00-09 (10 years)
Trisomy 13	NUMBER	19	36	76	112
	RATE	**0.17**	**0.19**	**0.32**	**0.26**
	Lower CI	0.10	0.13	0.25	0.21
ICD-10 Q91.4–Q91.7	Upper CI	0.26	0.26	0.40	0.31
Down Syndrome (Trisomy 21)	NUMBER	194	379	517	896
	RATE	**1.72**	**1.96**	**2.18**	**2.08**
	Lower CI	1.49	1.77	1.99	1.95
ICD-10 Q90..	Upper CI	1.98	2.17	2.37	2.22
Trisomy 18	NUMBER	48	85	132	217
	RATE	**0.43**	**0.44**	**0.56**	**0.50**
	Lower CI	0.31	0.35	0.47	0.44
ICD-10 Q91.0–Q91.3	Upper CI	0.57	0.54	0.66	0.58

Appendix A.4 Numbers of Cases, Anomalies and Anomalies per Case 1997–2009 Live Births (L), Stillbirths (S) and Fetal losses <20 weeks (T)

Year	Alberta Total Births (L & S)	# Cases (L, S & T)	Case Rate/1000 Total Births	# Anomalies (L, S & T)	Anomaly Rate/1000 Total Births	Average # Anomalies/ Case
1997	36797	1145	31.12	2023	54.98	1.77
1998	37715	1213	32.16	2228	59.07	1.84
1999	38044	1226	32.23	2442	64.19	1.99
97–99	***112556***	***3584***	***31.84***	***6693***	***54.46***	***1.87***
2000	36860	1310	35.54	2384	64.68	1.82
2001	37454	1399	37.35	2628	70.17	1.88
2002	38540	1396	36.22	2579	66.92	1.85
2003	40120	1540	38.38	2644	65.90	1.72
2004	40570	1579	38.92	2936	72.37	1.86
00–04	***193544***	***7224***	***37.32***	***13171***	***68.05***	***1.82***
2005	41890	1641	39.17	2937	70.11	1.79
2006	44954	1643	36.55	2747	61.11	1.67
2007	48708	1884	38.68	3113	63.91	1.65
2008	50512	2013	39.85	3437	68.04	1.71
2009	51407	2065	40.17	3553	69.12	1.72
05–09	***237471***	***9246***	***38.94***	***15787***	***66.49***	***1.71***
1997–2009	**543571**	**20054**	**36.89**	**35651**	**65.59**	**1.78**

