Am. J. Hum. Genet. 46:478–485, 1990

Evaluation of Birth Defect Histories Obtained through Maternal Interviews

Sonja A. Rasmussen,* Joseph Mulinare,† Muin J, Khoury,† and E. Kathleen Maloney‡

*University of Florida College of Medicine, Gainesville, FL; †Birth Defects and Genetic Diseases Branch, Division of Birth Defects and Developmental Disabilities, Center for Environmental Health and Injury Control, Centers for Disease Control, Atlanta; and ‡Computer Services Corporation, Huntsville

Summary

Etiologic studies of birth defects often use family history information provided by parents of patients. The validity of this information has not been adequately assessed. Using data from the Atlanta Birth Defects Case-Control study, we evaluated sensitivity, specificity, and positive predictive value of mothers' responses regarding the presence of birth defects in their offspring. A total of 4929 mothers of infants with major structural defects ascertained by the Metropolitan Atlanta Congenital Defects Program and a total of 3,029 mothers of normal infants were asked whether their babies had had a birth defect or a health problem diagnosed during the first year of life. Interviewers and coders of maternal responses were blinded to the case-control status of infants. Sensitivity (the proportion of case mothers who gave responses that could be coded as denoting a major birth defect) was 61%. Specificity (the proportion of control mothers who gave responses that could not be coded as denoting a major birth defect) was 98%. The positive predictive value (the proportion of mothers who gave a major-birth-defect response who in fact had babies with major birth defects) was estimated as 47%. Sensitivity, specificity, and positive predictive value varied by maternal sociodemographic factors such as race and education, as well as by type of defect. These results suggest that family history data obtained through maternal interviews should be cautiously interpreted and, if not properly validated, may alter estimates of recurrence risks.

Introduction

Research on the genetic epidemiology of birth defects often requires obtaining health-related information from interviews with parents of patients. Health care professionals also depend on family history information obtained from family members to make clinical diagnoses and to provide recurrence risk estimates for genetic counseling. The accuracy of these personal sources has not been fully evaluated. To investigate the accuracy of birth defects information obtained from mothers, the present study compares maternally reported birth defects with information obtained by trained medical abstractors from medical records.

Received July 17, 1989; revision received October 11, 1989.

Address for correspondence and reprints: Muin J. Khoury, M.D., Ph.D., Birth Defects and Genetic Diseases Branch, Division of Birth Defects and Developmental Disabilities. Center for Environmental Health and Injury Control, Centers for Disease Control, 1600 Clifton Road, Northeast, F-37, Atlanta, GA 30333.

© 1990 by The American Society of Human Genetics. All rights reserved.
0002-9297/90/4603-0010$02.00

Methods

Study Population

Data from the Atlanta Birth Defects Case-Control (ABDCC) study were used for the present study. Details on the methods of the ABDCC study have been reported elsewhere (Erickson et al. 1984). In this case-control study, parents of 4,929 babies with birth defects and parents of 3,029 babies without birth defects were interviewed. Data on case babies were obtained through the Metropolitan Atlanta Congenital Defects Program (MACDP) registry for the years 1968–80. This program (Edmonds et al. 1981) used multiple ascertainment sources to identify all live-born and stillborn babies (1) with major structural defects diagnosed during the first year of life and (2) whose mothers were residents of the five-county metropolitan Atlanta area at the time of birth. For each case, information about the baby's birth defects was abstracted after review of hospital medical records, and the birth defects were coded using a six-digit modified version of the International

Classification of Diseases—Clinical Modification (ICD-9CM) code and the 1981 British Pediatric Association (BPA) code. Control babies were chosen from a random sample of birth certificates of all live births that occurred in area hospitals during the same period.

Maternal Interviews

Telephone interviews with parents of case and control babies were conducted during 1982 and 1983. Interviews lasted approximately 45 min and were conducted using a computer-assisted telephone interview system. Mothers were informed that a study on potential causes of birth defects was being conducted by the Centers for Disease Control. The first interviewer asked questions about maternal reproductive histories. The second interviewer, blinded to case-control status, asked questions about possible causes of birth defects, including questions regarding occupational history, chemical exposures, medications taken, and chronic diseases. During the first part of the interview, mothers were asked the following question regarding the index child (case or control): "Did [your child] have a health problem at birth or a birth defect that was diagnosed during the first year of his/her life?" When this question was answered affirmatively, it was followed by a second question, which asked what kind of birth defect or health problem it was; and the answer was recorded verbatim. All responses to the second question were coded by one of us (S.A.R.), who was blinded to the case-control status of the respondent's baby. Coding of birth defects consisted of assigning as many as six defect codes (by using the MACDP six-digit coding system).

Methods of Analysis

The coded defects from maternal responses were then compared with the defects recorded in MACDP, to evaluate sensitivity, specificity, and positive predictive value (PPV) of maternal responses. Sensitivity was defined as the proportion of case mothers who gave responses that could be coded as denoting a major birth defect. The criteria for major birth defect were the same as those used in the case-control study—i.e., any defect that affects morbidity, mortality, and/or health prospects of affected infants (Erickson et al. 1984). Specificity was defined as the proportion of control mothers who gave responses that were not coded as denoting a major birth defect. PPV was defined as the proportion of mothers reporting birth defects responses who in fact had babies with major birth defects. PPV was computed by using values of sensitivity, specificity, and frequency of the defect (p) that were obtained from MACDP and by using the Fletcher et al. (1982, p. 53) formula

$$PPV = \frac{\text{sensitivity} \cdot p}{(\text{sensitivity} \cdot p) + (1 - \text{specificity})(1 - p)}.$$

Sensitivity, specificity, and PPV were also calculated for 66 specific birth defects and defect groups (see Results). Two levels of sensitivity were defined: (1) high-level sensitivity, when the mother's response was coded as denoting the same birth defect or defect group as one or more defects coded in MACDP, and (2) low-level sensitivity, when the mother's response could be coded as denoting a major birth defect but not necessarily the same defect or defect group coded in MACDP. Specificity for these groups was defined as the proportion of control mothers who gave responses that were not coded as the specific birth defect or defect group. PPV here was defined as the proportion of mothers reporting a specific birth defect or defect group who had babies with that birth defect or defect group. To calculate PPV for these groups, we used the high-level sensitivity, specificity, and prevalence calculated for each birth defect or defect group.

We also evaluated whether sensitivity and specificity levels were affected by several maternal and infant characteristics. Factors examined included maternal race, education, and age; previous births, previous unproductive pregnancies (defined as pregnancies which did not result in a live birth); period of birth of the index child; and status of birth (live born or stillborn). The effects of these factors were examined for total birth defects and for 10 representative defects chosen from the list of 66 defect groups (see Results). Independent effects of these factors were examined using multiple logistic regression analysis implemented by a standard statistical package (SAS 1985, p. 222). An association was considered to be significant when $P \leqslant .05$.

Results

Sensitivity, Specificity, and PPV for Total Birth Defects

Of 4,929 case mothers, 3,024 gave responses that could be coded as denoting a major birth defect, for an overall level of sensitivity of 61%. Of 3,029 control mothers, 67 gave responses that could be coded as denoting a major birth defect, for an overall specificity of 98%. From these values and given a prevalence of major birth defects of about 3.1%, PPV can be estimated as 47%.

Table 1

Sensitivity, Specificity, and Positive Predictive Value of Maternal Responses, by Defect Category

Defect	Sensitivity		Specificity	PPV	Prevalence for 1968–80	Total No. of Cases
	Low	High				
Central nervous system defects	.73	.61	1.00	.64[a]	.00294	710
Anencephaly and spina bifida	.83	.75	1.00	1.00	.00164	350
Anencephaly	.69	.48	1.00	1.00	.00071	145
Total spina bifida	.92	.88	1.00	1.00	.00103	205
Encephalocele	.74	.39	1.00	1.00	.00019	38
Microcephaly	.51	.14	1.00	1.00	.00097	93
Hydrocephaly	.67	.48	1.00	.60[a]	.00104	206
Eye defects	.70	.36	1.00	.59[a]	.00133	149
Anophthalmia and microphthalmia	.68	.17	1.00	.14[a]	.00032	60
Cataract	.73	.48	1.00	1.00	.00023	44
Ear, face, and neck defects	.58	.17	1.00	.45[a]	.00320	84
Cardiovascular system defects	.55	.38	1.00	.38[a]	.00699	1,116
Conus arteriosus defects	.73	.36	1.00	1.00	.00074	180
Persistent truncus arteriosus	.83	.35	1.00	1.00	.00008	23
Transposition of great vessels	.74	.38	1.00	1.00	.00041	100
Tetralogy of fallot	.66	.33	1.00	1.00	.00026	58
Single ventricle	.68	.07	1.00	1.00	.00012	28
Ventricular septal defects	.55	.14	1.00	.26[a]	.00163	426
Atrial septal defects	.64	.05	1.00	.03[a]	.00085	176
Endocardial cushion defects	.76	.10	1.00	1.00	.00021	42
Pulmonary valve defects	.65	.06	1.00	1.00	.00021	48
Tricuspid valve defects	.54	.17	1.00	1.00	.00015	35
Aortic valve defects	.70	.17	1.00	1.00	.00022	54
Hypoplastic left heart syndrome	.73	.16	1.00	1.00	.00028	73
Dextrocardia	.73	.18	1.00	1.00	.00014	22
PDA (birthweight > 2,500 g only)	.49	.06	1.00	1.00	.00324	405
Aortic atresia/hypoplasia	.72	.00	1.00	. . .[b]	.00022	25
Coarctation of aorta	.65	.29	1.00	1.00	.00034	80
Pulmonary artery atresia	.46	.00	1.00	. . .[b]	.00020	28
Total respiratory tract defects	.61	.22	1.00	.24[a]	.00097	13
Choanal atresia	.57	.22	1.00	.06[a]	.00009	23
Aplasia and hypoplasia of lung	.70	.14	1.00	1.00	.00042	76
Cleft palate	.90	.74	1.00	1.00	.00062	121
Cleft lip with or without cleft palate	.93	.78	1.00	1.00	.00112	238
Total gastrointestinal defects	.75	.63	1.00	.56[a]	.00519	592
T-E fistula and esophageal atresia	.90	.70	1.00	1.00	.00021	50
Pyloric stenosis	.78	.76	1.00	.31[a]	.00134	312
Small intestine atresia/stenosis	.51	.16	1.00	1.00	.00017	75
Duodenal atresia (excluding Down syndrome)	.48	.17	1.00	1.00	.00014	52
Atresia/stenosis of colon, rectum, and anus	.72	.50	1.00	1.00	.00043	94
Hirschprung disease	.87	.81	1.00	.24[a]	.00013	31
Anomalies of intestinal fixation	.66	.26	1.00	1.00	.00025	50
Genital system defects	.57	.38	1.00	.82[a]	.00392	771
Female genital defects	.44	.09	1.00	.08[a]	.00031	43
Male genital defects	.58	.41	1.00	1.00	.00099	683
Undescended testicle	.63	.06	1.00	1.00	.00103	98
Hypospadias	.58	.47	1.00	1.00	.00260	587
Other male genital defects	.74	.00	1.00	. . .[b]	.00020	43
Urinary system defects	.68	.47	1.00	.29[a]	.00146	285
Renal agenesis with or without atresia of ureter	.73	.43	1.00	1.00	.00034	77

(*continued*)

Table 1 (continued)

Defect	Sensitivity		Specificity	PPV	Prevalence for 1968–80	Total No. of Cases
	Low	High				
Polycystic kidney	.67	.19	1.00	1.00	.00012	48
Obstructive defects of urinary tract	.61	.16	1.00	.05[a]	.00046	127
Limb defects	.54	.45	.99	.06	.00100	1,329
Amniotic bands	.69	.19	1.00	1.00	.00009	16
Positional defects of leg	.47	.46	.99	.25	.00409	879
Preaxial polydactyly	.69	.23	1.00	1.00	.00004	13
Reduction defects	.76	.43	1.00	1.00	.00100	156
Flexion contracture of limb	.65	.11	1.00	.08[a]	.00025	37
Craniosynostosis	.90	.33	1.00	1.00	.00040	21
Diaphragmatic hernia	.76	.37	1.00	1.00	.00022	46
Omphalocele	.71	.12	1.00	1.00	.00037	82
Gastroschisis	.81	.19	1.00	1.00	.00013	27
All chromosomal defects	.90	.81	1.00	.72[a]	.00105	288
Other autosomal trisomies	.82	.36	1.00	1.00	.00018	39
Down syndrome	.94	.90	1.00	1.00	.00086	219
Teratomas and malignant neoplasms	.63	.50	1.00	1.00	.00004	8
All defects	.61	.61	.98	.47	.03122	4,929

[a] Calculated using actual specificity values (<1 but rounded to 1.00 in here).
[b] Unable to be calculated—sensivity = 0.

Analysis of Specific Defects

The sensitivity, specificity, and PPV of the maternal response varied with the type of defect. In table 1 we present values of sensitivity (high-level and low-level), specificity, and PPV for 66 individual defects and defect groups. High-level sensitivity varied from a high of 90% for Down syndrome to <10% for certain types of congenital heart defects. Even for defects that are relatively severe and easily ascertained, a varying proportion of cases were not reported accurately by mothers. For example, high-level sensitivities were 78% for cleft lip with or without cleft palate, 74% for cleft palate, 88% for spina bifida, 48% for anencephaly, 70% for esophageal atresia, and 50% for imperforate anus. PPV also varied by type of defect. For example, a PPV of 100% was achieved for anencephaly, spina bifida, Hirschsprung disease, Down syndrome, hypospadias, cleft palate, and cleft lip. In contrast, the PPVs for several congenital heart defects and positional deformities of extremities were relatively low. Specificity was found to be high for all defects evaluated.

Factors Affecting Sensitivity and Specificity

We analyzed a number of potential risk factors in an attempt to identify predictors of maternal responses. Table 2 shows the effects that the maternal and infant factors had on sensitivity and specificity for total birth defects. Of the factors examined, maternal race, maternal education, maternal age, and the length of time between birth and interview had effects on the sensitivity of the mother's response. Specificity levels were high even when examined by each factor, and no statistically significant differences could be found.

The sensitivity of the maternal response differed significantly with maternal race. Sixty-five percent of white case mothers, compared with 49% of nonwhite case mothers, identified their baby as having a major birth defect ($\chi^2 = 99.4$, df = 1, $P < .01$). The sensitivity of the maternal response also differed with the level of maternal education. The sensitivity for mothers with a college education was 68%, compared with 60% for mothers with only a high school education and 58% for mothers with only a grammar school education ($\chi^2 = 22.0$, df = 2, $P < .01$). Maternal age also was a predictor of the sensitivity of maternal response. Sixty-four percent of mothers ≥25 years of age were able to describe their child's defect as a major birth defect, while only 58% of mothers <25 years of age were able to do so ($\chi^2 = 16.5$, df = 1, $P < .01$).

The years of birth of the babies were used to determine the approximate length of time between the births and the interviews, which were conducted during 1982

Table 2

Sensitivity and Specificity of Selected Risk Factors for All Defects

Risk Factor	Sensitivity	Specificity
Maternal race:		
White	.65[a]	.97
Other	.49	.99
Maternal education:		
Grammar school	.58[a]	.98
High school	.60	.98
College	.68	.98
Maternal age:		
<25 Years old	.58[a]	.98
≥25 Years old	.64	.98
Birth period:		
January 68–April 72	.68[a]	.97
May 72–August 76	.64	.98
September 76–December 80	.54	.98
Baby's birth status:		
Live born	.62	.98
Stillborn	.56	Not applicable[b]

[a] $P < .01$.

[b] Because all control babies were live born.

and 1983. Mothers of babies born in the earlier years of the study were more likely to correctly report the presence of a major birth defect than were mothers of babies born during the later years of the study. Sensitivity decreased from 68% for babies born before May 1972 to 54% for babies born after September 1976 ($\chi^2 = 74.7$, df = 2, $P < .01$).

Mothers of live-born infants were more likely to correctly identify their child as having a major birth defect (sensitivity 62%) than were mothers of stillborns (sensitivity 56%); however, this difference was not statistically significant ($\chi^2 = 3.4$, df = 1, $P = .066$). The sensitivity of the maternal response was not affected by the number of previous pregnancies ($\chi^2 = 0.035$, df = 1, $P = .85$) or by the presence of previous unproductive pregnancies ($\chi^2 = 0.017$, df = 1, $P = .90$).

Logistic regression analysis was employed to ascertain risk factors which were independently associated with sensitivity. Maternal race, maternal education, maternal age, and birth period were independently associated with sensitivity for all defects. Maternal race was a significant factor in the high level sensitivity of the maternal responses for spina bifida, cardiovascular system defects, cleft palate, cleft lip, pyloric stenosis, positional leg defects (clubfoot), and Down syndrome. Maternal education was a significant factor in the determination of high-level sensitivity for cardiovascular system defects, hypospadias, and pyloric stenosis. Maternal age was a statistically significant predictor in the high-level sensitivity of Down syndrome. Baby's birth status was a significant determinant of the high-level sensitivity for spina bifida, cardiovascular system defects, and Down syndrome. The relationship between birth period and sensitivity held for the cardiovascular defects category.

Discussion

In the present study, maternal responses regarding birth defects in offspring were compared with information abstracted from medical records. We are aware of one other study in which this comparison was evaluated. Maternal responses on a questionnaire about birth defects were compared with information obtained from the Swedish Register of Congenital Malformations (Axelsson and Rylander 1984). This study, using a small sample, found a sensitivity level similar to that found in our study. Of the 38 infants with malformations who were reported in the registry, 10 were not reported in the questionnaire completed by the mother (sensitivity 74%). Twenty-four malformations noted by the mothers were not recorded in the registry. These defects most often were not present at birth and therefore would not be included in the Swedish registry, which only registers defects observed at birth.

In a recent review of the literature, Harlow and Linet

(1989) addressed the issue of agreement between questionnaire data and medical records. These authors reviewed papers addressing recall of reproductive history, including menstrual history, pregnancy history, and childbirth. No studies dealing specifically with recall of birth defects were found. A study by Wilcox and Horney (1984) evaluated the accuracy of spontaneous-abortion recall. Women who had recorded a spontaneous abortion during their participation in a long-term study of menstrual cycles were resurveyed about the occurrence of spontaneous abortions. Seventy-five percent of women recalled their abortions. The authors suggested that this level of recall represented an upper limit of recall because the women in the study had been sensitized to their reproductive history because of the long-term recording of their menstrual cycles.

Two studies have compared the accuracy of pediatric history information provided by mothers with that obtained from medical records and have found different results. In a study performed by Goddard et al. (1961), 25 mothers were interviewed about the child's health history when their children were between 4 and 5 years of age. The mothers recalled spontaneously only 18 of 34 major illnesses recorded in the medical record. Health events that were documented in the medical record but not recalled by the mother included measles, chicken pox, tonsillectomy, and corrective shoes and braces. On the other hand, in a study published by Hoekelman et al. (1976), in which 59 mothers were asked, when their babies were 9 mo of age, to identify specific problems or illnesses that had the potential for affecting the baby's general health or development adversely, mothers recalled 25 illnesses in 16 babies, and the medical records documented no conditions that the mothers did not recall (Hoekelman et al. 1976).

Our results suggest that the ability of mothers to recall the presence and type of birth defect is heavily influenced by the type of defect and by other sociodemographic factors. Maternal race, maternal education, maternal age, and time elapsed between birth and interview were all found to be significant factors in the sensitivity of the maternal responses in the present study. Maternal factors were also determined to be of importance in the overall accuracy of the responses in the study by Hoekelman et al. (1976), discussed earlier. Maternal education and maternal race significantly affected the accuracy of responses in this study, but maternal age, sex, or birth order of the baby did not.

Our study observed an unanticipated relationship between time elapsed from birth of the child until the interview and sensitivity of the maternal response. One would expect that the sensitivity would decrease with increased time elapsed between interview and the event to be recalled, as was seen in a study by Wilcox and Horney (1984) on recall of spontaneous abortions; however, our study demonstrated the converse. An explanation for this finding is not readily apparent. One possible reason for the difference between these two studies may be that spontaneous abortion is a single occurrence, whereas raising a child with a congenital defect represents a continuing experience.

In our study, status at birth (live born or stillborn) was not a statistically significant predictor of sensitivity for total defects, but it was a significant factor in the determination of sensitivity for certain birth defects (spina bifida, cardiovascular system defects, and Down syndrome). Cartwright and Smith (1979) compared data obtained from medical records with data obtained from maternal interviews and found that the concordance between the two sources was better for live borns than for stillborns. They suggest that this may be due to a reluctance on the part of both the physician and the mother to remember events surrounding a stillbirth.

The type of defect was the most important factor in the sensitivity of the maternal responses. For defects in which treatment is usually instituted shortly after birth and with good results—i.e., defects such as hypospadias and positional defects of the leg—the sensitivity of the maternal response was poor. In addition, the sensitivity regarding a lethal defect (anencephaly) also was low. For congenital malformations that typically have life-long effects on the family—i.e., malformations such as Down syndrome and spina bifida—the sensitivity of maternal responses was much higher. This finding is similar to that of Axelsson and Rylander (1984), who found that the malformations not recalled by the mothers tended to be less serious (three cases of subluxation of the hips, and one case each of hydrocele, hypospadias, ventricular septal defect, preauricular tag, nevus, metatarsus varus, and pes calcaneovalgus). We hypothesize that the effect on the family is one determining factor in the likelihood of a mother's recall of birth defects.

Our study has some potential limitations. First, a possible limitation may be the manner in which the question about birth defects was asked. Parents were first asked about the presence of either a health problem at birth or a birth defect that was diagnosed during the first year of life. Parents may perceive some health problems as being more serious than some congenital defects and therefore might be more likely to recall them. Furthermore, the inquiry about birth defects was asked

as an open-ended question. Mitchell et al. (1986) have shown that open-ended questions are less likely to obtain an accurate response about drug intake. In their study of recall of drug exposure during pregnancy, the authors first asked an open-ended question about drug use during the past year, then asked about drug use for selected indications, and finally asked about the use of specifically named drugs. Of the three prescription drugs studied, 20%–35% of women who acknowledged drug use did so only when asked about a specific drug by name.

Second, a possible limitation was the way in which the maternal responses were recorded. In contrast to the clinical setting, where the interviewers are health professionals usually familiar with congenital defects, the interviewers in the present study were trained only in interviewing techniques. The entry of a verbatim response about something with which the interviewers were not familiar could be a possible source of error. Certainly, a trained health professional, because of his or her familiarity with the defects mentioned, would obtain more accurate information about the birth defects in offspring. In addition, in a clinical situation, appropriate follow-up questions may be asked, which could result in an increased likelihood of accurate response.

Third, information in the present study was collected through a telephone interview rather than through personal interviews conducted in clinical genetics settings. While it is difficult to assess the impact that telephone interviews may have had on the results of the present study, an investigation by Aneshensel et al. (1982) suggests that this may not be a major limitation. Information obtained from in-person and telephone interviews about community health status were compared, and for assessment of health status, illnesses reported for the previous 4 mo, or reports of hospitalization, the authors found no statistically significant differences between the two methods (Aneshensel et al. 1982).

The results of our investigation suggest that using information obtained from interviews with mothers about birth defects in their offspring may introduce errors into studies of familial aggregation of birth defects. The sensitivity of the maternal responses is such that a large proportion of serious birth defects may be missed or not accurately recalled. Even if medical records could be reviewed for all maternal responses suggestive of a major birth defect and all unknown/uncodable responses, we found that 21% of infants with major birth defects were completely missed. Although our study questionnaire included questions about birth defects in other family members, we were unable to evaluate the sensitivity of this information, since medical records data on these individuals were not available. However, it seems likely that this information would be even less accurate. Nevertheless, interviews with family members may be an acceptable alternative in the study of certain defects. For example, 94% of mothers of babies with Down syndrome identified the infant as having a major birth defect, and 90% were able to specify the presence of Down syndrome.

In summary, our study suggests that information obtained from mothers about birth defects in their offspring should be viewed with caution. We emphasize the importance of obtaining medical records not only to verify birth defects identified by parents but to ascertain defects in patients reportedly unaffected. Several predictors of the sensitivity of the maternal response—such as maternal race, maternal education, maternal age, baby's birth status (live born or stillborn), and time elapsed between the birth and interview—were found. The type of birth defect was found to be the most important determinant of the sensitivity of the maternal response, and it therefore should be considered in conducting and analyzing family studies of birth defects.

Acknowledgments

The authors thank Letitia Dy for her help in preparing the manuscript and Philip d'Almada for his assistance in data processing and programming. S.A.R. was supported by NIH short-term training grant HLO748908.

References

Aneshensel CS, Frerichs RR, Clark VA, Yokopenic PA (1982) Telephone versus in-person surveys of community health status. Am J Public Health 72:1017–1021

Axelsson G, Rylander R (1984) Validation of questionnaire reported miscarriage, malformation and birth weight. Int J Epidemiol 13:94–98

Cartwright A, Smith C (1979) Some comparisons of data from medical records and from interviews with women who had recently had a live birth or stillbirth. J Biosoc Sci 11:49–64

Edmonds LD, Layde PM, James LM, Flynt JW, Erickson JD, Oakley GP Jr (1981) Congenital malformations surveillance: two American systems. Int J Epidemiol 10:247–252

Erickson JD, Mulinare J, McClain PW, Fitch TG, James LM, McClearn AB, Adams MJ Jr (1984) Vietnam veterans' risks for fathering babies with birth defects. JAMA 252:903–912

Fletcher RH, Fletcher SW, Wagner EH (1982) Clinical epidemiology—the essentials. Williams & Wilkins, Baltimore

Goddard KE, Broder G, Wenar C (1961) Reliability of pediatric histories: a preliminary study. Pediatrics 28:1011–1018

Harlow SD, Linet MS (1989) Agreement between questionnaire data and medical records: the evidence for accuracy of recall. Am J Epidemiol 129:233–248

Hoekelman RA, Kelly J, Zimmer AW (1976) The reliability of maternal recall: mothers' remembrance of their infant's health and illness. Clin Pediatr 15:261–265

Mitchell AA, Cottler LB, Shapiro S (1986) Effect of questionnaire design on recall of drug exposure in pregnancy. Am J Epidemiol 123:670–676

SAS Institute Inc (1985) SAS user's guide: statistics, version 5 ed. SAS Institute Inc., Cary, NC

Wilcox AJ, Horney LF (1984) Accuracy of spontaneous abortion recall. Am J Epidemiol 120:727–733