Coll. Antropol. 39 (2015) 3: 769–774
Original scientific paper

Palmar Creases: Classification, Reliability and Relationships to Fetal Alcohol Spectrum Disorders (FASD)

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ABSTRACT

A normal human palm contains 3 major creases: the distal transverse crease; the proximal transverse crease; and the thenar crease. Because permanent crease patterns are thought to be laid down during the first trimester, researchers have speculated that deviations in crease patterns could be indicative of insults during fetal development. The purpose of this study was twofold: 1) to compare the efficacy and reliability of two coding methods, the first (M1) classifying both »simian« and Sydney line variants and the second (M2) counting the total number of crease points of origin on the radial border of the hand; and 2) to ascertain the relationship between palmar crease patterns and fetal alcohol spectrum disorders (FASD). Bilateral palm prints were taken using the carbon paper and tape method from 237 individuals diagnosed with FASD and 190 unexposed controls. All prints were coded for crease variants under M1 and M2. Additionally, a random sample of 98 matched (right and left) prints was selected from the controls to determine the reliabilities of M1 and M2. For this analysis, each palm was read twice, at different times, by two readers. Intra-observer Kappa coefficients were similar under both methods, ranging from 0.804–0.910. Inter-observer Kappa coefficients ranged from 0.582–0.623 under M1 and from 0.647–0.757 under M2. Using data from the entire sample of 427 prints and controlling for sex and ethnicity (white v. non-white), no relationship was found between palmar crease variants and FASD. Our results suggest that palmar creases can be classified reliably, but palmar crease patterns may not be affected by fetal alcohol exposure.

Key words: *dermatoglyphics, palmar creases, flexion creases, reliability, fetal alcohol spectrum disorders (FASD)*

Introduction

A normal human palm contains three major creases. The thenar or vertical crease »begins at or slightly below the proximal transverse crease at the radial border of the palm«[1]. The proximal transverse crease »begins at the radial side of the palm… curves proximally, and ends at the medial border of the hypothenar eminence«. Finally, the distal horizontal crease is found closest to the fingers, beginning at the »interdigital space between the index and middle fingers«, curving »gently wristward toward the ulnar side of the palm«, and ending before the proximal crease of the fifth finger (Figure 1)[1]. Several variants of the normal crease pattern exist, including the simian or single transverse palmar crease (STPC), which joins the proximal and distal creases to produce a single horizontal crease across the width of the palm, and the Sydney crease, which represents an extension of the proximal transverse crease to near the ulnar border of the palm.

Palmar creases are laid down during the first or second trimester of fetal life[1–3] and remain unchanged thereafter[4]. Such features are partially influenced by hereditary and partially influenced by environmental processes[5], including maternal stressors, anticonvulsant medications, and alcohol. A fetus exposed to trauma during early development could show increased prevalence of abnormal palmar crease patterns[6], for example, by changing the pattern of locomotion *in utero*[7]. Previous studies classifying STPC and/or Sydney line variants have suggested that aberrant palmar crease patterns are associated with a variety of disorders, most commonly Down Syndrome, but also low birthweight[8,9], deafness[10], childhood leukemia[11], intrauterine methadone exposure[2], and even hyperactivity[12].

Received for publication July 9, 2013

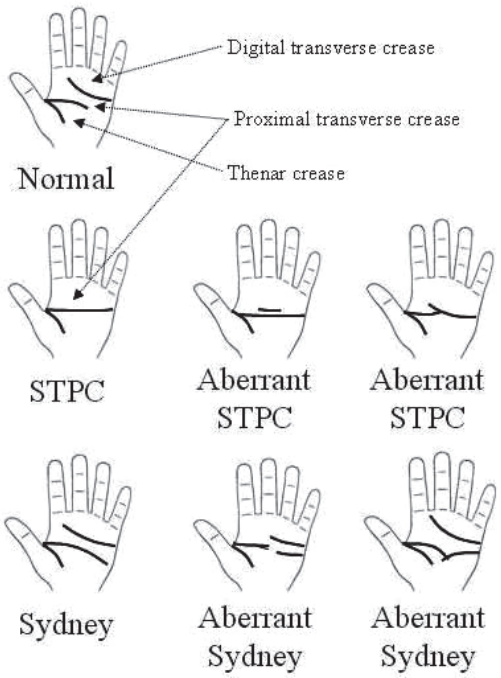

Fig. 1. Examples of palmar crease variants (Method 1) based on the classification system of Dar et al. (1977).

Alcohol is a wide-acting teratogen that causes abnormal fetal growth and development, impaired performance on intelligence and motor tests, and possibly abnormal palmar crease patterns[7,13]. Fetal alcohol spectrum disorders (FASD) are any of a variety of neurological or morphological abnormalities associated with *in utero* exposure to alcohol. These include fetal alcohol syndrome (FAS) and fetal alcohol effects (FAE)[14]. In this study we sought to confirm whether FASD is associated with abnormal palmar creases in a relatively large, contemporary sample consisting of subjects diagnosed with FASD and unexposed controls.

While it is clear that the presence of STPC and Syndey creases are associated with certain fetal insults and genetic disorders, less is known about the relationship of other abnormal crease patterns to these disorders[15]. Moreover, because researchers often use different classification schemes, it is sometimes difficult to compare results across studies. This study sought to compare two methods of palmar crease classification in which STPC, Sydney creases and other variants were included in the classification scheme. The first method (M1) includes classifications for both STPC and Sydney lines and their variants and the second (M2) counts the total number of crease points of origin on the radial border of the hand. While M1 represents the most traditional classification method, M2 has the potential to avoid a number of difficulties inherent in traditional classification schemes, including over-reliance on STPC and misclassification resulting from unclear prints[15]. In this paper we present the inter- and intra-observer reliability of each method and compare the effectiveness of each method in predicting FASD.

Materials and Methods

Data were collected from 237 individuals diagnosed with FAS or FAE. These individuals had been referred to the Fetal Alcohol Syndrome Diagnostic Clinic at the University of Washington for evaluation and enrolled in a follow-up study in 1994–95. At the time of recruitment, age ranged from 6–51 years of age (43% were 6–11 years; 36% 12–20 years and 21% 21–51 years). Additionally, 190 individuals were recruited as unexposed controls from undergraduate students and staff at the University of Washington between 1996 and 2002. As a proxy for prenatal alcohol exposure, individuals were asked whether or not their mother »consumes alcohol as a general policy«. Only individuals who reported that their mother did not drink alcohol were included in the unexposed control sample.

All participants' palm prints were taken using the carbon paper and tape method described by Aase and Lyons[16]. Prints were then digitally scanned at a resolution of 400 dots per inch and later read using Adobe Photoshop (version 8.0, Abobe Systems, Inc., San Jose, CA).

A sub-sample of 98 bilateral prints was randomly selected from the control group. This sub-sample was read twice by two independent readers to determine the inter- and intra-reader reliabilities of M1 and M2. After establishing relatively high inter- and intra-reader reliabilities, one reader then read the entire sample of 427 bilateral prints using M1 and M2, as described below.

Method 1 (crease patterns) – Based on the methods of Dar et al.[2], prints were classified as normal, STPC-normal, STPC-aberrant, Sydney-normal and Sydney-aberrant. A palm print was considered normal if all three creases – proximal, distal, and thenar – were present. If distal and proximal creases formed one solid line across the palm, the print was considered to be STPC-normal. STPC-aberrant palmar prints were those in which the proximal crease was rudimentary, where the STPC was formed by a connection of the distal and proximal creases running in the same direction, or where a STPC was generally present, but with some interruptions and often a rudimentary proximal crease. Sydney variants were considered normal if the proximal crease abutted at no greater than 2 epidermal ridges away from the ulnar border of the palm[2]. Sydney creases were considered aberrant if the Sydney crease was branched, interrupted or broken, or if it extended to, but not beyond, the medial line of the fifth finger. To distinguish between strong palmar creases and weak palmar ridges, we employed a 2-ridge rule, in which a crease was defined by having a thickness at least as great as 2 ridges. We tended towards conservative estimates, classifying palmar prints as normal in cases where hands were highly wrinkled or the classification was otherwise rendered difficult.

Method 2 (points of origin) – Palm prints were further classified by the number of crease points of origin on the radial border of the palm[15]. Points of origin ranged from no less than 1 point (1 crease present) to no greater than 3 points (Figure 2). A third point of origin was only indi-

Fig. 2. Points of origin (Method 2) based on the classification scheme of Chaube (1977).

cated in cases where a clear third point originated from the thenar crease and where the proximal and distal lines originated at the radial border of the palm.

Reliability analysis

The sample of 98 paired prints (left and right) was randomly selected and read by two observers to determine intra- and inter-observer reliability for both methods used in this study. Each print was read twice by Reader A and twice by Reader B. To increase the independence of successive measurements of prints, each set of readings was made at different times, weeks or months apart. Prints were read in a random order and right and left prints from the same individual were not read at the same time.

Results from both readers were compared using the simple Kappa coefficient[17]. Kappa coefficients range from –1 to +1, where 1 indicates complete agreement between the readers (raters), positive coefficients indicate better agreement than expected by chance, and negative coefficients indicate lower agreement than expected by chance. Because Kappa coefficients control for chance agreement, values exceeding 0.5 generally indicate good agreement between observers.

FASD analysis

In addition to the reliability analysis, the entire sample (N=427) was coded by Reader B using both M1 and M2 in order to determine the association between FASD and palmar crease patterns. Logistic regression and simple binary logistic regression models using robust standard errors were built to examine the relationship between crease pattern and FASD and between points of origin and FASD. FASD diagnosis was considered the outcome variable in each model and the crease pattern, or points of origin, the primary independent variable of interest. Models controlled for subjects' sex and ethnicity (white v. non-white) in all analyses. We further tested for an interaction between sex and ethnicity, but as the interaction term did not achieve statistical significance, it was dropped from all analyses. Covariates of interest were re-coded into categorical variables as follows: FASD diagnosis was coded as either affected or unexposed control and ethnicity was coded as white or non-white. All statistical analyses were performed using Stata, version 9.0 (College Station, Texas); p-values of 0.05 or less were considered significant.

TABLE 1
SAMPLE SUMMARY STATISTICS

Sex	N of 427 (%)	
Male	206 (48.2)	
Female	200 (46.8)	
Unk.***	21 (4.9)	
Ethnicity		
White	298 (69.8)	
Non-White	90 (21.1)	
Unk.***	39 (9.1)	
FASD Diagnosis*		
Control	190 (45.0)	
FAS	77 (18.0)	
FAE	143 (33.5)	
Unk.***	17 (4.0)	
Palmar Crease Classifications	Right (%)	Left (%)
Normal	352 (82.4)	344 (80.6)
STPC	5 (1.2)	10 (2.3)
STPC ab**	19 (4.5)	11 (2.6)
Sydney	8 (1.9)	11 (2.6)
Sydney ab**	38 (8.9)	39 (9.1)
Und.***	5 (1.2)	12 (2.8)
Points of Origin		
One	19 (4.4)	19 (4.4)
Two	319 (74.7)	340 (79.6)
Three	87 (20.4)	65 (15.2)
Und.***	2 (0.5)	3 (0.7)

*FAS – fetal alcholol syndrome, FAE – fetal alcohol exposure, **STPC ab – aberrant single transverse palmar crease, Sydnab – aberrant Sydney crease, ***Unk. – unknown, Und – undetermined

Results

Summary statistics of the sample are found in Table 1. The sample was composed of approximately equal numbers of males and females; however, there were more white than non-white individuals (binary test, two-sided, $p<<0.001$). Although over half the sample was diagnosed with a fetal alcohol spectrum disorder, most palmar prints were classified as normal.

Reliability analysis

Kappa coefficients were very high for both intra- and inter-observer reliability. Intra-observer Kappa coefficients were 0.814 and 0.910 for Readers A and B under M1 and 0.804 and 0.885 under M2. Inter-observer Kappa coefficients ranged from 0.582–0.623 under M1 and from 0.647–0.757 under M2. Method 2 produced slightly higher inter-observer reliability estimates, but slightly lower intra-observer reliability estimates compared to M1.

TABLE 2
COMPARISON OF CREASE CLASSIFICATION AND POINTS OF ORIGIN*

	Right, points of origin				Left, points of origin			
Crease Classification	1	2	3	Total	1	2	3	Total
Normal	6 (1.5)	140 (34.2)	44 (10.8)	190	7 (1.7)	150 (36.9)	31 (7.6)	188
Aberrant**	11 (2.7)	169 (41.3)	39 (9.5)	219	12 (2.9)	177 (43.5)	30 (7.4)	219
Total	17	309	83	409	19	327	61	407

*N (% of total), **Includes STPC, STPC aberrant, Sydney and Sydney aberrant prints

There was a high degree of correspondence between the points-of-origins measure (M2) and the standard classification scheme (M1) (right: χ^2= 101.0, p<<0.001; left: χ^2= 96.8, p<<0.001; Table 2). In both palms, where palmar creases were classified as normal, in no instances were there fewer than 2 points of origin. We can thus conclude that a single point-of-origin is indicative of an aberrant crease pattern. Two points of origin were more prevalent than three points of origin in hands classified as »normal« under the standard classification scheme (M1), but this pattern was also found in palms classified as »aberrant« under the same scheme. Thus, the distinction between two and three points of origin may not be useful for determining whether palms are normal or aberrant.

FASD analysis

Table 3 shows the prevalence of crease patterns and points-of-origin in relation to FASD diagnosis for both the right and left hands. Our models never yielded a significant association between palmar crease pattern and FASD, even when sex and ethnicity were controlled for in the models. Table 4 shows an example of the logistic regression results. The only variable with a significant association with FASD diagnosis is sex (males are less likely to be affected by FASD than females). Because the degree to which palmar creases are expected to deviate from normal changes with the timing and degree of fetal trauma, we also analyzed the relationship between Fetal Alcohol Syndrome (FAS), the most severe form of FASD, and crease patterns. Even in this analysis, we were unable to detect a significant association between FAS and crease pattern. Similarly, we could not detect a significant association between points of origin and FAS. A possible explanation for our findings is that abnormal palmar creases were rare in our sample compared to normal palmar creases. This suggests that a larger sample size may be required to detect a relationship between crease patterns and FASD.

TABLE 3
FASD PREVALENCE GIVEN CREASE PATTERNS AND POINTS OF ORIGIN*

	Right		Left	
	Control	FASD	Control	FASD
Crease Classification				
Normal	163 (40.1)	176 (43.3)	153 (41.4)	176 (47.6)
STPC	1 (0.2)	4 (1.0)	4 (1.1)	6 (1.6)
STPC ab	8 (2.0)	9 (2.2)	6 (1.6)	5 (1.4)
Sydney	3 (0.7)	5 (1.2)	2 (0.5)	8 (2.2)
Sydney ab	13 (3.2)	24 (5.9)	2 (0.5)	8 (2.2)
Total	188	218	167	203
	406		370	
Points of Origin				
1 PO	6 (1.5)	11 (2.7)	7 (1.7)	12 (2.9)
2 PO	140 (34.2)	169 (41.3)	150 (36.9)	177 (43.5)
3 PO	44 (10.8)	39 (9.5)	31 (7.6)	30 (7.4)
Total	190	219	188	219
	409		407	

* N (% of total)

TABLE 4
LOGISTIC REGRESSION RESULTS PREDICTING FASD BY CREASE PATTERN, SEX, AND ETHNICITY*

	Right		Left	
	Coef	SE	Coef	SE
Predictor				
STPC**	–.40	.74	.70	1.1
STPC ab**	.67	.53	.38	.59
SYD**	–1.8	1.1	–1.7	1.0
SYD ab**	.11	.40	–.00	.36
Sex***	–.57[b]	.21	–.65[b]	.21
Ethnicity****	–.42	.25	–.47	.25
Intercept	.70[a]	.35	.77[a]	.35

*Coefficients are expressed in log odds; standard errors are robust. R^2 for the right hand is 0.031 on 381 complete observations. R^2 for the left hand is 0.0319 on 376 complete observations. Predictors are considered significant where [a]p < 0.05 and [b]< 0.01, **Reference category is normal, ***Reference is female, ****Reference is white

Discussion

Classification, methods and reliability

During the late 1960s and early 1970s, a flurry of papers attempted to explain the significance of palmar

creases in detecting fetal trauma. Much of this research focused on the relationship of the so-called »simian« crease (STPC) to various disorders, but less attention was paid to other abnormal variants such as the Sydney line and other transitional forms of STPC and the Sydney line. Furthermore, because researchers employed multiple classification methods, comparability across studies was limited[18]. In this study, we performed a direct comparison of two prominent palmar crease classification methods and found that they were similar in reliability and comparable in terms of their relationship to *in utero* alcohol exposure.

Studies of palmar creases differ not only in the scheme employed to classify palms, but also in the method of inspection. The most common method is direct visual inspection of the palm and by ultrasound[2,8,10,19]. However, this method may introduce a bias in that other morphological traits characteristic of certain disorders may influence how palmar creases are classified. The standard ink and inkless methods of examining palmar crease patterns[3,11,20] remove any bias resulting from direct observation of the individual, but may be problematic with respect to the clarity and quality of the print. We used the carbon paper and tape method (analogous to ink methods) to remove bias from direct interaction with subjects and classified palms conservatively if prints were unclear. This may have led to an overestimation of normal palm prints, which may have limited our ability to detect an association between aberrant prints and FASD.

Palmar creases and the relationship to FASD

Dermatoglyphics and palmar crease patterns have long been of interest to physicians because of associations with abnormal development and genetic disorders. Indeed, it has been suggested that palmar crease patterns may provide a reliable cue to physicians of impending dysfunction[1]. Alcohol is a known teratogen during prenatal life, and the long-term effects on morphology and especially, neurobehavior, are well-documented[14,21]. Though we expected to find increased prevalence of aberrant palmar creases in individuals with FASD, we were unable to detect such an association.

There are many factors that may have limited our ability to detect an association between FASD and abnormal crease patterns. By categorizing palmar crease classifications into »normal« versus »abnormal« categories in some models, we may have missed a specific association between a given class of abnormal variant and FASD. It is known, however, that both transitional and complete forms of abnormal creases are associated with high levels of exposure to alcohol *in utero*[7]. Furthermore, binning various forms of aberrant palmar creases seems warranted as heterogeneous manifestations of the same basic crease pattern are known to occur among relatives[8,10]. Finally, even in models where each class of crease aberration was considered separately, we could not detect an association.

It is possible that we were unable to capture an existing association because we did not control for disease severity. It has been suggested that timing and severity of fetal trauma may affect the degree to which palmar creases deviate from normal[6]. Jones et al.[7] found aberrant palmar creases in infants born to chronically alcoholic mothers. It may be that our subjects were all exposed to a subthreshold level of alcohol, in which case palmar creases would not be expected to deviate substantially from normal patterns. We attempted to address this possibility by examining the relationship of severe cases of FAS to aberrant palmar creases, but were unable to detect an association. Nonetheless, further studies examining the timing and degree of trauma with respect to production of aberrant morphologies are warranted.

Palmar creases are known to differ by sex and among ethnicities, independently of their association with a given disorder[2,10,11,22–24]. Indeed, Tsai et al.[25] argue that simian creases could be considered normal in Chinese newborns, as they occur at frequencies of higher than 4%. Even controlling for sex and a crude estimate of ethnicity (white v. non-white), we were unable to detect an association between FASD and aberrant crease patterns.

Conclusion

Based on the results of this research, we suggest that palmar crease variants can be measured reliably. Kappa coefficients for both M1 and M2 were greater than 0.5, suggesting good agreement within and between readers. Using data from the entire sample of 427 prints and controlling for sex and ethnicity, we found no relationship between palmar crease variants and FASD. Various limitations, including small sample size for a rare event, may have hindered our ability to detect such a relationship. These results suggest that while palmar creases can be classified reliably, they may not be useful in predicting fetal trauma due to *in utero* exposure to alcohol in the absence of very large samples.

Acknowledgements

We thank Laura Newell-Morris, Ann Streissguth and Carrie Kuehn for their work in collecting the prints used in this study. This research was supported by grants from NIH NIAAA (R21-AA013704 and R37-AA01455) and a grant from the Alcohol and Drug Abuse Institute, University of Washington.

REFERENCES

1. JOHNSON CF, OPITZ E, Clin Pediatr (Phila), 10 (1971) 392, DOI: 10.1177/000992287101000707. — 2. DAR H, SCHMIDT R, NITOWSKY HM, Pediatr Res, 11 (1977) 103, DOI: 10.1203/00006450-197702000-00004. — 3. CANNON M, BYRNE M, COTTER D, SHAM P, LARKIN C, O'CALLAGHAN E, Schizophr Res, 13 (1994) 179, DOI: 10.1016/0920-9964(94)90100-7. — 4. TAY JS, Ann Hum Genet, 42 (1979) 327, DOI:

10.1111/j.1469-1809.1979.tb00666.x. — 5. SEEMA MA, GANDHI D, SINGH M, Nov Sci Int J Med Sci, 1 (2012) 191, — 6. PURVIS-SMITH SG, HAYES K, MENSER MA, Lancet, 2 (1972) 976, DOI: 10.1016/S0140-6736(72)92508-1. — 7. JONES KL, SMITH DW, ULLELAND CN, STREISSGUTH P, Lancet, 1 (1973) 1267, DOI: 10.1016/S0140-6736(73)91291-9. — 8. DAR H, F. E. CARNEY J, WINTER ST, Acta Paediatr Scand, 60 (1971) 479, DOI: 10.1111/j.1651-2227.1971.tb06691.x. — 9. DAVIES PA, Dev Med Child Neurol, 8 (1966) 729, DOI: 10.1111/j.1469-8749.1966.tb01833.x. — 10. DAR H, WINTER ST, Hum Hered, 20 (1970) 493, DOI: 10.1159/000152350. — 11. WERTELECKI W, Birth Defects Orig Artic Ser, 15 (1979) 455. — 12. LERER RJ, Clin Pediatr, 16 (1977) 645, DOI: 10.1177/000992287701600711. — 13. JONES KL, ROBINSON LK, BAKHIREVA LN, GALINA M, STOROJEV V, STRAHOVA A, SERGEEVSKAYA S, BUDANTSEVA S, MATTSON SN, RILEY EP, CHAMBERS CD, Pediatrics, 118 (2006) e1734, DOI: 10.1542/peds.2006–1037. — 14. STREISSGUTH AP, CONNOR PD, FAS and other effects of prenatal alcohol: Developmental cognitive implications. In: NELSON CJ& LUCIANA M (Eds), Handbook of developmental cognitive neuroscience (MIT Press, MA, 2001). — 15. CHAUBE R, Am J Phys Anthropol, 47 (1977) 7, DOI: 10.1002/ajpa.1330470104. — 16. AASE JM, LYONS RB, Lancet, 1 (1971) 432, DOI: 10.1016/S0140-6736(71)92417-2. — 17. COHEN J, Educ Psychol Meas, 20 (1960) 37, DOI: 10.1177/001316446002000104. — 18. PARK JS, SHIN DS, JUNG W, CHUNG MS, Anat Cell Biol, 43 (2010) 169, DOI: 10.5115/acb.2010.43.2.169. — 19. JEANTY P, J Ultrasound Med, 9 (1990) 131. — 20. DAR H, JAFFE M, Dev Med Child Neurol, 25 (1983) 53, DOI: 10.1111/j.1469-8749.1983.tb13721.x. — 21. STREISSGUTH AP, SAMPSON PD, CARMICHAEL OLSON H, BOOKSTEIN FL, BARR HM, SCOTT M, FELDMAN J, Alcohol Clin Exp Res, 18 (1994) 202, DOI: 10.1111/j.1530-0277.1994.tb00904.x. — 22. BOROFFICE RA, J Hum Evol,10 (1981) 273, DOI: 10.1016/S0047-2484(81)80064-4. — 23. ADETONA MO, OLADAPO, OO, AKINYEMI JO. Afr J Biomed Res, 15 (2012) 93. — 24. SEN J, KANCHAN T, MONDAL N. J Forens Sci, 56 (2010) 109, DOI: 10.1111/j.1556-4029.2010.01554.x. — 25. TSAI FJ, TSAI CH, PENG CT, WU JY, LIEN CH, WANG TR, Acta Paediatr, 88 (1999) 323, DOI: 10.1080/08035259950170114

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NABORI DLANOVA: KLASIFIKACIJA, POUZDANOST I KORELACIJA S POREMEĆAJEM FETALNOG ALKOHOLNOG SPEKTRA (FASD)

S A Ž E T A K

Normalan ljudski dlan sadrži 3 glavna nabora: distalni transverzalni nabor; proksimalni transverzalni nabor i thenarni nabor. Budući da se smatra da uzorci nabora nastaju u prvom tromjesečju, istraživači su nagađali da odstupanja u naborima uzoraka može biti indikativno povredama tijekom fetalnog razvoja. Svrha ovog istraživanja bila je dvostruka: 1) usporediti učinkovitost i pouzdanost dvije metode kodiranja; prva (M1) je klasificiranje »majmunskih« i Sydney varijanti linija i druga (M2) je računanje ukupnog broja točaka podrijetla nabora na radijalnoj granici ruke; i 2) utvrditi odnos između uzoraka nabora dlana i poremećaja fetusnog alkoholnog spektra (FASD). Bilateralni otisci dlanova su snimljeni metodama ugljik papira i trake kod 237 osoba s dijagnozom FASD i 190 kontrolnih pojedinaca. Svi ispisi su kodirani za varijante nabora M1 i M2. Osim toga, odabran je slučajni uzorak od 98 podudaranih (lijeva i desna) otisaka ispisuje između kontrola kako bi se odredila pouzdanost metoda M1 i M2. Za ovu analizu, svaki dlan je očitan dva puta, u različitim vremenima od strane dva čitatelja. Kappa koeficijenti Intra-promatrača su slični po obje metode, u rasponu od 0,804–0,910. Kappa koeficijenata Inter-promatrača u rasponu od 0,582–0,623 pod M1 i od 0,647–0,757 pod M2. Koristeći podatke iz cijelog uzorka od 427 grafika i kontrolirajući varijeble za seks i etnicitet (bijeli v. Ne-bijeli), nije pronađena veza između varijanti nabora dlanova i FASD-a. Naši rezultati ukazuju na to da se nabori dlanova mogu pouzdano klasificirati, ali izloženost fetusa alkoholu ne može utjecati na obrasce nabora dlana.