

# Dupuytren's Diathesis Revisited: Evaluation of Prognostic Indicators for Risk of Disease Recurrence

Sandip Hindocha, MBChB, John K. Stanley, MChOrth,  
Stewart Watson, MBChB, Ardeshir Bayat, PhD

*From Plastic & Reconstructive Surgery Research, The Manchester Interdisciplinary Biocentre, University of Manchester, Manchester, UK; the Centre for Hand & Upper Limb Surgery, Wigan, Leigh and Wrightington NHS Trust, Wigan, Lancashire, UK; and the Department of Plastic, Reconstructive and Hand Surgery, South Manchester University Teaching Trust, Wythenshawe Hospital, Wythenshawe, Manchester, UK.*

**Purpose:** The term *diathesis* relates to certain features of Dupuytren's disease (DD) and dictates an aggressive course of disease. The initial description of DD diathesis included 4 factors: (1) ethnicity, (2) family history, (3) bilateral DD, and (4) ectopic lesions (DD outside the palm). The degree of diathesis is considered important in predicting recurrence and extension of DD after surgical management. Prognostic indicators of risks associated with surgery are important. We aimed to evaluate these 4 factors and known associated risk factors to formulate a statistical predictive value for DD diathesis.

**Methods:** Caucasian patients diagnosed with DD between the ages of 25 and 90 years ( $n = 322$ ) from Northwest England were assessed for DD diathesis with a clinical history and examination. DD diathesis assessment was analyzed by calculating the odds ratios of developing recurrent DD using logistic regression.

**Results:** The observed recurrence rates in the presence of notable risk factors and corresponding odds ratios of recurrent DD were calculated. Of note, recurrent disease was observed in 121 (46%) males, 105 (47%) with bilateral DD, 68 (48%) with a family history of DD, 75 (47%) with age at onset younger than 50 years, 35 (52%) in those with ectopic lesions, and in 26 (63%) with Garrod's pads.

**Conclusions:** The original DD diathesis factors have been evaluated and modified. The original factors of family history, bilateral DD, and ectopic lesions now include 2 additional factors: male gender and age at onset of younger than 50 years. Family history and ectopic disease have now been modified to specify family history with one or more affected siblings/parents and ectopic lesions in the knuckles (Garrod's pads) alone. The presence of all new DD diathesis factors in a patient increases the risk of recurrent DD by 71% compared with a baseline risk of 23% in those DD patients with none of the earlier-described factors. (J Hand Surg 2006;31A:1626–1634. Copyright © 2006 by the American Society for Surgery of the Hand.)

**Type of study/level of evidence:** Prognostic II.

**Key words:** Disease prognosis, Dupuytren's disease, Dupuytren's contracture, Dupuytren's diathesis, ectopic Dupuytren's disease, risk factor, recurrence.

Dupuytren's disease (DD) is a benign, progressive, fibroproliferative disorder that results in the development of abnormal scar-like tissue in the palmar fascia of the hand extending to any digit.<sup>1</sup> In its advanced stages, DD finally leads to an irreversible, permanent, and progressive contrac-

ture of the involved digits. Dupuytren's disease in the hands is commonly bilateral, but Dupuytren's-like fibrotic tissue also can occur in the dorsum of the hand over the knuckles (Garrod's pads), feet (*Lederhose's disease*), and penis (*Peyronie's disease*) of the same individual.<sup>2</sup>

Diathesis describes a condition, constitution, or morbid habit that would predispose an individual to a particular disease.<sup>3</sup> The *DD diathesis* is a term first coined by Hueston,<sup>3</sup> relating to certain characteristics of the disease and dictating an aggressive course and greater tendency for recurrence after surgical treatment. Hueston<sup>3</sup> described 4 factors as part of the DD diathesis: bilateral disease (described as bilateral palmar lesions), family history of DD, ectopic lesions (DD found outside the palmar surface), and ethnicity.

Hueston's<sup>3</sup> study on the DD diathesis noted that patients developed recurrence more frequently than extension. Recurrence can be divided into true and false recurrence. False recurrence can include scar and joint contracture, whereas true recurrence is the development of new DD tissue within the same area of previous surgery for the treatment of DD. Extension describes the development of new DD tissue away from the area of surgery.<sup>4</sup> Foucher et al<sup>5</sup> stated that recurrence at 5.5 years of follow-up evaluation occurred in 41%, extension occurred in 39%, and total disease activity recurred in 55%.<sup>5</sup> Wrong Ref!

The degree of diathesis is considered very important in predicting recurrence and extension of DD after surgical management. Recurrence of DD is problematic for the patient and the surgeon because recurrence after surgical management is not uncommon.<sup>6</sup> We were unable to find clear data regarding the accurate predictive value of various features of DD diathesis. Prognostic indicators of risks associated with surgery are important. Suggestions for the development of recurrence in DD patients after surgery include incomplete excision of the palmar fascia, younger age, and a greater degree of diathesis, especially in those with Garrod's (knuckle) pads, indicating more active disease.<sup>7,8</sup>

In addition to Hueston's<sup>3</sup> 4 diathesis factors there are many environmental factors that have been associated with the cause of DD including a history of smoking,<sup>9</sup> alcohol consumption,<sup>10</sup> frozen shoulder,<sup>11</sup> epilepsy,<sup>12</sup> diabetes mellitus,<sup>13,14</sup> rheumatoid arthritis,<sup>15</sup> carpal tunnel syndrome,<sup>16</sup> history of manual labor,<sup>17</sup> and hand injury.<sup>18</sup> Other factors associated with increased severity include male gender and a young age at onset. None of these factors were included in the original diathesis description by Hueston<sup>3</sup> and may be important in developing recurrent disease.

The presence of the DD diathesis is known to increase the risk of recurrence.<sup>19</sup> We aimed to evaluate Hueston's<sup>3</sup> 4 current DD diathesis factors and known associated risk factors to formulate a statisti-

cal predictive value for DD diathesis and hence provide a more accurate prognostic indicator.

## Materials and Methods

### Study Sample

A hospital-based, retrospective, cohort study design was used to recruit patients with a diagnosis of DD. All patients were enrolled after surgical management for DD, ensuring that the diagnosis of DD was accurate. The presence of DD nodules, cords in the palmar or plantar fascia, with or without contraction of affected digits on examination, was used to confirm the diagnosis. A total of 322 Caucasian patients diagnosed with DD were identified via surgical records from a hospital in the Northwest of England. A total of 262 men (81%) with an age range of 25 to 88 years and a mean age of 63 years (SD = 10) and 60 women (19%) with an age range of 32 to 90 years and a mean age of 62 years (SD = 11) were enrolled in the study. Ethical approval was granted The Wrightington, Wigan and Leigh Local Research Ethics Committee, Wigan and South Manchester Research Ethics Committee, Manchester, UK and all participating patients completed an ethically approved consent form. Each patient was assessed for recurrence of disease for a minimum of 4 years after surgery. To date, no patients have been lost to follow-up evaluation. Assessment was based on a proforma agreed on by all authors involving a thorough history and clinical examination. Each patient was examined by the first and senior authors.

### Diathesis Assessment

A relevant history and clinical examination were performed based on the proforma agreed on by all authors. A family history of DD was documented with a focus on first- and second-degree relatives. The mean onset of disease was in the fifth decade of life,<sup>20</sup> therefore if the age at onset of DD was younger than 50 years, this was deemed important. Bilateral palmar lesions, ectopic lesions (comprising Garrod's pads, Lederhose's disease, and Peyronie's disease), and the presence of digit contractures were examined for in each patient. The following risk factors associated with DD were documented: gender, a history of diabetes (insulin or noninsulin dependent), epilepsy, carpal tunnel syndrome, frozen shoulder, rheumatoid arthritis, smoking and alcohol history, history of manual labor, and history of injury to the hand.

Recurrent disease was assessed as recurrent disease activity; true recurrence was distinguished from false recurrence and extension and noted after surgery. Recurrent disease activity and differentiation of

true, false, and extension of disease were identified via clinical examination by the first author and confirmed or refuted by the senior author. Recurrence was defined as the development of new DD lesions including the smallest palpable nodule irrespective of a presenting contracture in the same area where fasciectomy had been performed.<sup>3</sup> False recurrences must be differentiated from true recurrences. False recurrences are caused by surgical complications and can consist of scar contracture, joint contracture, and extrinsic tendon imbalance.<sup>4</sup> Extension of DD is defined as the development of new DD lesions irrespective of a presenting contracture outside the area where fasciectomy has taken place and in an area where no DD lesions were detected previously.<sup>3</sup>

### Data Analysis

All known associated risk factors and relevant aspects of clinical examination were analyzed to assess which factors (including known associated risk factors and Hueston's<sup>3</sup> diathesis factors) increased the odds ratio of DD recurrence. DD diathesis assessment was analyzed by calculating the observed frequency of recurrence occurring in the presence of associated factors and subsequently calculating the odds ratios of recurrent DD using binary logistic regression. Statistical analyses were calculated using the Microsoft Excel (Redmond, WA) and SPSS software packages (SPSS Inc., Chicago, IL).

All data were transcribed categorically by using binary variables. For alcohol consumption, men who drank more than 28 U/wk and women who drank more than 21 U/wk were categorized as having considerable alcohol consumption. One unit of alcohol is equivalent to 10 mL of pure ethanol. As a rough estimate, 1 small glass of wine, a single measure of spirit, or a half pint of beer each contain approximately 1 U of alcohol. Patients whose age at onset of DD was younger than 50 years were categorized as having a young age at onset.

Odds ratios were first calculated for each individual risk factor unadjusted to other factors by using the chi-square test. For each ratio calculated, a 95% confidence interval was given and a p value was calculated using the Fisher exact test. Binary logistic regression then was used to calculate odds adjusted for all factors; this gives the odds of developing recurrent DD based on an individual factor but takes into account other factors used in the analysis. The final analysis involved calculating the predictive risk of developing recurrent DD by combining the 4 factors in the current diathesis introduced by Hueston<sup>3</sup> with gender and age at onset of disease.

## Results

### Observations

Of the 322 patients, 141 (44%) had recurrent (true recurrence) DD. Each patient had a fasciectomy for treatment of DD. The frequency of observed associated risk factors in all patients was documented (Table 1). Of note, ectopic lesions were found in 77 (24%) patients. There was no Peyronie's disease, 41 (13%) had Garrod's pads, and 36 (11%) had Lederhose's lesions. Bilateral palmar DD lesions were apparent in 225 (70%) patients. A family history was reported by 143 (44%) patients. The mean age at onset of DD was 49 years for all patients, with 154 (47%) patients who were noted to have an age at onset of younger than 50 years.

### Odds Ratio of Recurrent Dupuytren's Disease for Associated Investigated Factors

Unadjusted odds of developing true recurrent DD (Table 1) were significantly greater in those who were men ( $p = .04$ ), had an age at onset of DD of younger than 50 years of age ( $p = .05$ ), presence of Garrod's pads ( $p = .006$ ), and those considered to have high alcohol consumption ( $p = .02$ ). A borderline significant increase in odds of recurrent DD was seen in those with bilateral disease and the presence of any ectopic Dupuytren's lesions ( $p = .07$ ). There was also a significant reduction in the odds of developing recurrent DD in those who had a history of smoking ( $p = .02$ ). Adjusted odds ratios calculated showed similar results except for those observed to have bilateral disease in whom the odds ratio was increased, but not significantly (Table 2).

### Combining Factors to Calculate the Predictive Risk of True Recurrent Dupuytren's Disease

The presence of the 4 factors in the original diathesis combined with male gender and an age at onset of DD of younger than 50 years were used to calculate the predictive risk of true recurrent DD (in percentage terms) from the logistic regression model (Table 3).

There was a general trend of an increased mean predictive risk when a greater number of risk factors was present. Those with all 5 factors of male gender, bilateral disease, the presence of Garrod's pads, an age at onset of younger than 50 years, and a positive family history had a predictive risk of 71% compared with 23% for patients with none of the factors present (Fig. 1).

## Discussion

The odds ratio and predictive risk for developing recurrent DD after surgical management has been

**Table 1. Observed and Unadjusted Odds Ratios of True Recurrent DD**

Factor	Observed Frequency (%)	Observed Recurrent DD (%)	Odds Ratio	95% Confidence Interval		p Value
				Lower	Upper	
Male gender	262 (80.9)	121 (46.1)	1.72	0.95	3.09	.04
Female gender	60 (19.1)	20 (33.3)	—	—	—	—
Bilateral DD	225 (69.4)	105 (46.7)	1.48	0.91	2.42	.07
Age at onset <50 y	154 (47.5)	75 (48.7)	1.47	0.94	2.28	.05
Ectopic lesions (any site)	67 (20.1)	35 (52.2)	1.54	0.90	2.64	.07
Garrod's pads	41 (12.7)	26 (63.4)	2.50	1.27	4.93	.006
Lederhose's Disease	36 (11.1)	16 (44.4)	1.03	0.51	2.07	.54
Positive family history	143 (44.1)	68 (47.6)	1.32	0.85	2.05	.14
Hand injury	22 (6.8)	8 (36.4)	0.72	0.29	1.76	.31
Manual labor	159 (49.1)	67 (42.1)	0.88	0.56	1.36	.32
Smoking	205 (63.3)	81 (39.5)	0.62	0.39	0.98	.02
Considerable alcohol consumption	63 (19.4)	35 (55.6)	1.80	1.04	3.14	.02
Epilepsy	5 (1.5)	2 (40.0)	0.85	0.14	5.18	.61
IDDM	11 (3.4)	5 (45.5)	1.07	0.32	3.59	.57
NIDDM	18 (5.6)	8 (44.4)	1.03	0.39	2.68	.57
Carpal tunnel syndrome	11 (3.4)	7 (63.6)	2.31	0.66	8.06	.15
Frozen shoulder	24 (7.4)	12 (50.0)	1.31	0.57	3.01	.33
Rheumatoid arthritis	8 (2.5)	3 (37.5)	0.77	0.18	3.26	.51

Observed frequency of true recurrent DD and calculated unadjusted odds ratios using the chi-square and the Fisher exact tests. IDDM, insulin-dependent diabetes mellitus; NIDDM, non-insulin-dependent diabetes mellitus.

derived with an implication to modify the current DD diathesis. Patients with features of DD diathesis are at high risk for developing recurrence after treatment. In this study we observed true and false recurrences; those patients with a true recurrence

and not a false recurrence or extension of DD were considered to have recurrent DD after surgical treatment.

It has been suggested that those with a strong diathesis should be treated with aggressive surgery

**Table 2. Adjusted Odds Ratios of True Recurrent DD**

Factor	Odds Ratio	95% Confidence Interval		p Value
		Lower	Upper	
Male gender	2.15	1.07	4.32	.03
Female gender	—	—	—	—
Bilateral DD	1.40	0.82	2.39	.22
Age at onset <50 y	1.47	0.94	2.28	.05
Ectopic lesions (any site)	1.54	0.90	2.64	.07
Garrod's pads	2.50	1.27	4.93	.006
Lederhose's Disease	1.03	0.51	2.07	.54
Positive family history	1.32	0.85	2.05	.14
Hand injury	0.72	0.29	1.76	.31
Manual labor	0.88	0.56	1.36	.32
Smoking	0.62	0.39	0.98	.02
Considerable alcohol consumption	1.80	1.04	3.14	.02
Epilepsy	0.85	0.14	5.18	.61
IDDM	1.07	0.32	3.59	.57
NIDDM	1.03	0.39	2.68	.57
Carpal tunnel syndrome	2.31	0.66	8.06	.15
Frozen shoulder	1.31	0.57	3.01	.33
Rheumatoid arthritis	0.77	0.18	3.26	.51

Calculated adjusted odds ratios using multiple binary logistic regression analysis.

IDDM, insulin-dependent diabetes mellitus; NIDDM, non-insulin-dependent diabetes mellitus.

**Table 3. Observed and Predictive Risks of True Recurrent DD When Combining Risk Factors**

Gender	Bilateral DD	Garrod's Pads	Age at Onset <50 y	Positive Family History	Observed	Predicted	95% Confidence Interval, %	
							Lower	Upper
x	x	x	x	x	33% (2/6)	23%	13	38
✓	x	x	x	x	42% (10/24)	33%	22	45
x	✓	x	x	x	20% (2/10)	30%	18	45
x	x	✓	x	x	100% (1/1)	42%	22	64
x	x	x	✓	x	0% (0/3)	27%	15	45
x	x	x	x	✓	36% (4/11)	28%	16	43
✓	✓	x	x	x	38% (20/53)	41%	31	50
✓	x	✓	x	x	75% (3/4)	53%	33	72
✓	x	x	✓	x	33% (4/12)	38%	26	51
✓	x	x	x	✓	30% (3/10)	38%	26	52
x	✓	✓	x	x	100% (1/1)	50%	28	72
x	✓	x	✓	x	43% (3/7)	35%	21	52
x	✓	x	x	✓	44% (4/9)	35%	22	51
x	x	✓	✓	x	0% (0/0)	47%	25	69
x	x	✓	x	✓	50% (2/4)	47%	27	69
x	x	x	✓	✓	50% (2/4)	32%	18	50
✓	✓	✓	x	x	33% (1/3)	62%	42	78
✓	✓	x	✓	x	48% (23/48)	46%	36	56
✓	✓	x	x	✓	47% (15/32)	46%	35	58
✓	x	✓	✓	x	100% (3/3)	59%	39	76
✓	x	✓	x	✓	100% (1/1)	58%	38	77
✓	x	x	✓	✓	29% (4/14)	43%	30	57
x	✓	✓	✓	x	76% (16/21)	55%	32	76
x	✓	✓	x	✓	50% (2/4)	56%	34	75
x	✓	x	✓	✓	67% (2/3)	40%	25	57
x	x	✓	✓	✓	100% (2/2)	52%	31	72
✓	✓	✓	✓	x	50% (2/4)	66%	49	80
✓	✓	✓	x	✓	100% (1/1)	67%	48	81
✓	✓	x	✓	✓	49% (17/35)	52%	40	62
✓	x	✓	✓	✓	50% (2/4)	64%	45	79
x	✓	✓	✓	✓	100% (1/1)	61%	39	79
✓	✓	✓	✓	✓	88% (14/16)	71%	55	83

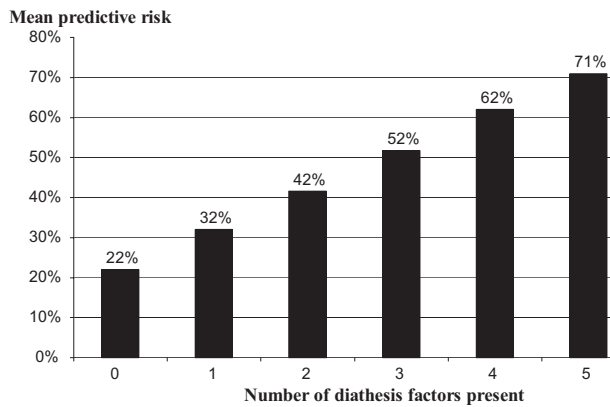
The predictive risk of true recurrent DD (in percentage terms) from the logistic regression model and the corresponding observed percentages of recurrent DD for the 32 combinations of gender, bilateral DD, presence of Garrod's pads, age at onset younger than 50 years, and a positive family history. Checkmark (✓) denotes presence of risk factor or male gender; cross (x) denotes absence of risk factor or female gender.

involving dermofasciectomy,<sup>21</sup> which may reduce the risk of recurrent DD. Patients in this study did not have dermofasciectomy and therefore it was not possible to determine the beneficial effects of this surgical procedure on patients with recurrent DD. Previous reports, however, have shown 100% reduction in recurrence more than 8 years after dermofasciectomy.<sup>22</sup>

It has been postulated that inadequate removal of DD tissue at the time of surgery is a cause of recurrence; this, however, does not explain the progress of a microscopic focus developing into recurrent macroscopic nodules.<sup>3</sup> It has been suggested that if DD tissue is excised incompletely, then myofibroblasts may be further induced, causing local hyperplasia<sup>23</sup>

of the remaining tissue and presenting as recurrent DD. Hueston,<sup>24</sup> however, observed a high rate of recurrent disease in his patients who showed certain characteristics notwithstanding the degree of completeness of the initial surgical excision. He therefore suggested that it is the diathesis that predicts recurrence and so it is the patient who dictates recurrence and not the surgeon who removes the diseased tissue. As a result, incomplete excision of DD tissue may not be a relevant factor in determining recurrent disease.<sup>24</sup> Therefore, DD diathesis has been considered a useful measure of severity and recurrence of disease that may aid in surgical management.

The incidence of DD has been known to increase with advancing age before the establishment of the



**Figure 1.** Bar graph representing the mean predictive risk of developing recurrent DD based on the number of risk factors present from Table 3.

DD diathesis.<sup>25,26</sup> The age at onset of disease is said to be between 40 and 59 years in men and between 40 to 69 years in women,<sup>12</sup> with a mean age at onset in the fifth decade of life.<sup>27</sup> Hence, in this study an age at onset of younger than 50 years in either gender was viewed as important in the risk of recurrent disease. We have shown that there is a 47% increase in the odds of developing recurrent DD after adjustment with other associated factors. It is therefore essential that age at onset of DD is noted as part of the DD diathesis.

Hueston<sup>3</sup> noted an increased frequency of recurrence in those with bilateral disease. This study also observed an increased frequency of recurrence in those with bilateral disease, with a 48% increase in the odds of developing recurrent disease and an increased predictive risk of developing recurrent DD compared with unilateral disease (Table 3); therefore, bilateral disease should be included in the DD diathesis.

The odds ratio of recurrence appears to be considerably greater in men than women. Dupuytren's disease is known to be more prevalent in men; before the diathesis formation Hueston<sup>3</sup> had found an equal incidence of DD in men and women. This may have been owing to the cohort selection of an aging population. The risk of recurrence also may vary in different populations and ethnicities because the prevalence of DD is widely variable around the world.<sup>28,29</sup> The high risk of recurrence in men may be reflected by the fact that there are 4 times as many men with DD than women (Table 1). Although male gender should be accounted for when assessing the DD diathesis, it should be noted that genetics are important in the pathogenesis of DD in women, which has been shown previously.<sup>27</sup> With male gen-

der forming part of the DD diathesis, it should be considered that women with a positive family history of DD will have a strong DD diathesis and a high odds of recurrence.

Ectopic lesions are known to be part of the DD diathesis. Certain ectopic lesions need to be specified, however, when describing the diathesis in each patient. There have been suggestions to include ectopic lesions in the popliteal space<sup>30</sup>; however, it seems that only ectopic lesions on the knuckles are associated markedly with recurrence of DD. Hueston<sup>3</sup> also documented that ectopic lesions were seen most commonly in the knuckles. Lesions in the knuckles also known as *Garrod's pads* were first described by Garrod as moderately soft, mobile lesions on extension of the fingers and firm fixed lesions on flexion of the fingers; with lesions being present on the dorsal aspect of the proximal interphalangeal joints.<sup>31</sup> Recurrence with increasing tenderness is more likely to occur after the excision of ectopic lesions, either plantar nodules (Lederhose's disease) or Garrod's pads, and nonsurgical management has been suggested.<sup>32</sup> Although ectopic lesions formed part of the diathesis introduced by Hueston<sup>3</sup> it is necessary to specify where the ectopic nodules are present, with Garrod's pads alone making a notable contribution to the diathesis. Although there have been reports of ectopic lesions elsewhere in the body including the popliteal space,<sup>30</sup> and case reports have suggested that epilepsy and ectopic lesions in the popliteal space could form part of the diathesis, this has not been elucidated further and is not a formal diathesis characteristic.

Hueston<sup>3</sup> noted that recurrence is more common in the younger age group, which is consistent with this study. He also found that Garrod's pads were more common in the younger age group and therefore strengthened the reason to specify the type of ectopic lesion when describing the DD diathesis. It has been shown previously that the presence of Garrod's pads does not imply a greater predisposition or more aggressive DD,<sup>33</sup> contrary to this we have shown that the presence of Garrod's pads markedly increases the predictive risk of recurrent DD. The presence of an ectopic lesion on the knuckle, plantar surface, or penis results in an increased odds ratio of developing recurrent DD with borderline significance; however, from our findings Garrod's pads appear to play a more significant role in determining diathesis than the other ectopic lesions. Therefore, we recommend indicating the specific form of ectopic lesion when attributing this to diathesis.

Two elements in the etiology of DD clearly continue to be prominent. One is the familial nature of the disease and the other is that DD appears to be an extremely common disorder, mainly affecting Caucasians of northern European ancestry.<sup>1,34</sup> All patients in our sample had northern European ancestry. One hundred and forty-three (44%) patients reported a positive family history; this is consistent with a previous figure.<sup>35</sup> We had found that the presence of a positive family history increases the odds ratio of developing recurrent DD. Although this result was not significant, a positive family history should be included in the DD diathesis. We previously showed that the presence of DD in an individual significantly increases the risk of a sibling developing DD<sup>27</sup>; with this positive heritability, familial aggregation of DD should be included within the diathesis.

It has been shown previously that smoking and alcohol consumption both increase the odds of developing DD.<sup>9</sup> Assessing alcohol consumption can result in ascertainment bias. To reduce this we used a binary figure for the cut-off point, in that those men who drank more than 28 U/wk and those women who drank more than 21 U/wk were said to have major consumption of alcohol.

Contrary to previous reports,<sup>21</sup> we found that the unadjusted and adjusted odds ratios of developing recurrent DD is considerably increased in those who have significant alcohol consumption. At the same time, odds of recurrence in smokers could not be concluded because the quantity of tobacco consumption cannot be specified because of ascertainment bias among patients. It has been concluded previously that although the prevalence of DD is greater in those with considerably high consumption of alcohol, its etiologic importance may be minimal.<sup>10</sup> In view of the conflicting evidence on the role of smoking and alcohol in the cause of DD in the literature, and despite our findings, it was deemed appropriate to exclude alcohol consumption and smoking as part of the DD diathesis. Nevertheless, it may be that certain individuals with heavy smoking and/or alcohol habits may be further predisposed to developing DD because of gene/environment interaction, the scope and relevance of which is currently beyond the context of this article. Both smoking and alcohol consumption require further investigation into their role in the development and recurrence of DD, especially with emerging knowledge regarding susceptibility gene loci and environment interaction.

The prevalence of DD in insulin- and non-insulin-dependent diabetic patients appears to be similar but

with a higher incidence in younger patients with insulin-dependent diabetes.<sup>13</sup> It also has been shown that those with insulin-dependent diabetes mellitus are more likely to suffer severe disease and require surgical management when compared with those with non-insulin-dependent diabetes.<sup>14</sup> We have shown that there is only a marginal increase in the odds ratio of developing recurrent DD, which is higher in insulin-dependent diabetic patients, and therefore from our analysis diabetes should not form part of the DD diathesis.

Occupation has always been linked to the development of DD since its description by Dupuytren himself.<sup>36</sup> This has been refuted by many after a number of epidemiologic studies including one by Hueston<sup>3</sup>, who did not find a notably increased incidence of DD depending on one's occupation.<sup>12</sup> Many have stated that manual work is associated with DD, either as a result of chronic trauma or as a result of retirement from manual labor, preventing "maximum physiological normality."<sup>36-38</sup> Analysis from this study has shown that a history of manual labor is not associated with increased odds of developing recurrent DD.

It has been suggested that injury to the hand may increase the risk of developing DD in those with a positive family history.<sup>18</sup> From our study population only 22 (7%) reported hand injury, with 8 (36%) of these individuals developing recurrent DD. This does not appear to be a factor in the development of recurrent disease, even in those with a positive family history, and should not be included in the DD diathesis.

There has been a general consensus that the incidence of DD is related to epilepsy and anticonvulsant therapy.<sup>12</sup> Our study contained few patients with epilepsy; and analysis showed us that the odds of developing recurrent DD is not increased with a large confidence interval (Table 2), reflecting the uncertainty in the calculated odds ratio. It is therefore suggested that epilepsy should not be included in the DD diathesis.

Cytogenetic analysis of tissue from patients with carpal tunnel syndrome and DD has suggested a common pathologic pathway for the 2 conditions.<sup>16</sup> The odds ratio of developing recurrent DD appeared to be high but with a wide confidence interval the results were statistically insignificant and it was not possible to include carpal tunnel syndrome as part of the DD diathesis.

Although DD is 8 times more likely to develop in those with a history of frozen shoulder,<sup>11</sup> it did not

appear to show a significant increase in the odds of developing recurrent DD after surgical management. There is a negative association between rheumatoid arthritis and DD.<sup>15</sup> Our results are consistent with this finding and there was a decrease in the odds ratio of developing recurrent disease in those with a history of rheumatoid arthritis affecting the hands.

DD diathesis may prove to be a useful clinical tool in predicting prognosis. One of the important factors in determining the outcome of DD surgery may be to determine the degree of DD diathesis.<sup>3</sup> It may prove useful that the patient is educated on the degree of his or her diathesis and help awareness regarding risk of recurrence and expected outcome after any surgical intervention. In those with a great degree of DD diathesis, before any attempt at surgical intervention, the risks of recurrence should be explained and the procedure planned carefully, considering dermofasciectomy to reduce the risk of recurrence.<sup>3</sup>

The authors would like to thank Ms. Sarah Collin of the Research and Development Department, Pennine Acute Hospitals, NHS Trust, for her statistical expertise.

Received for publication June 21, 2006; accepted in revised form September 12, 2006.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

Corresponding author: Ardeshir Bayat, PhD, Clinician Scientist, Plastic & Reconstructive Surgery Research, Manchester Interdisciplinary Biocentre, University of Manchester, 131 Princess St, Manchester, M1 7ND, England, UK; e-mail: ardeshir.bayat@manchester.ac.uk.

Copyright © 2006 by the American Society for Surgery of the Hand 0363-5023/06/31A10-0009\$32.00/0  
doi:10.1016/j.jhsa.2006.09.006

## References

1. Bayat A, McGrouther DA. Management of Dupuytren's disease—clear advice for an elusive condition. *Ann R Coll Surg Engl* 2006;88:3–8.
2. Thurston AJ. Dupuytren's disease. *J Bone Joint Surg* 2003; 85B:469–477.
3. Hueston JT. Dupuytren's contracture. Edinburgh: E&S Livingstone, 1963:51–120.
4. Iselin F. Recurrences in Dupuytren's contracture. In: Hueston JT, Tubiana R, eds. Dupuytren's disease. Edinburgh: Churchill Livingstone, 1974:139–140.
5. Foucher G, Medina J, Navarro R. Percutaneous needle aponeurotomy: complications and results. *J Hand Surg* 2003; 28B:427–431.
6. Lettin AW. Dupuytren's diathesis; a case report. *J Bone Joint Surg* 1964;46B:220–225.
7. Pickren JW, Smith AG, Stevenson TW Jr, Stout AP. Fibromatosis of the plantar fascia. *Cancer* 1951;4:846–856.
8. Hueston JT. Prognosis as a guide to the timing and extent of surgery in Dupuytren's contracture. In: Hueston JT, Tubiana R, eds. Dupuytren's disease. Edinburgh: Churchill Livingstone, 1974:61–62.
9. Burge P, Hoy G, Regan P, Milne R. Smoking, alcohol and the risk of Dupuytren's contracture. *J Bone Joint Surg* 1997; 79B:206–210.
10. Noble J, Arafa M, Royle SG, McGeorge G, Crank S. The association between alcohol, hepatic pathology and Dupuytren's disease. *J Hand Surg* 1992;17B:71–74.
11. Smith SP, Devaraj VS, Bunker TD. The association between frozen shoulder and Dupuytren's disease. *J Shoulder Elbow Surg* 2001;10:149–151.
12. Mikkelsen OA. Dupuytren's disease—initial symptoms, age of onset and spontaneous course. *Hand* 1977;9:11–15.
13. Arkkila PE, Kantola IM, Viikari JS. Dupuytren's disease: association with chronic diabetic complications. *J Rheumatol* 1997;24:153–159.
14. Arkkila PE, Kantola IM, Viikari JS, Ronnema T, Vahatalo MA. Dupuytren's disease in type 1 diabetic patients: a five-year prospective study. *Clin Exp Rheumatol* 1996;14:59–65.
15. Arafa M, Steingold RF, Noble J. The incidence of Dupuytren's disease in patients with rheumatoid arthritis. *J Hand Surg* 1984;9B:165–166.
16. Bonnici AV, Birjandi F, Spencer JD, Fox SP, Berry AC. Chromosomal abnormalities in Dupuytren's contracture and carpal tunnel syndrome. *J Hand Surg* 1992;17B:349–355.
17. de la Caffiniere JY, Wagner R, Etscheid J, Metzger F. [Manual labor and Dupuytren disease. The results of a computerized survey in the field of iron metallurgy.] *Ann Chir Main* 1983;2:66–72.
18. McFarlane RM. Dupuytren's disease: relation to work and injury. *J Hand Surg* 1991;16A:775–779.
19. Abe Y, Rokkaku S, Ofuchi S, Tokunaga S, Takahashi K, Moriya H. An objective method to evaluate the risk of recurrence and extension of Dupuytren's disease. *J Hand Surg* 2004;29B:427–430.
20. Early PF. Population studies in Dupuytren's contracture. *J Bone Joint Surg* 1962;44B:602–613.
21. Smith AC. Diagnosis and indications for surgical treatment. *Hand Clin* 1991;7:635–643.
22. Brotherston TM, Balakrishnan C, Milner RH, Brown HG. Long term follow-up of dermofasciectomy for Dupuytren's contracture. *Br J Plast Surg* 1994;47:440–443.
23. Luck JV. Dupuytren's contracture; a new concept of the pathogenesis correlated with surgical management. *J Bone Joint Surg* 1959;41A:635–664.
24. Hueston JT. Unsatisfactory results in Dupuytren's contracture. *Philosophies of Dr. J. T. Hueston. Hand Clin* 1991;7: 759–763.
25. Hueston JT. The incidence of Dupuytren's contracture. *Med J Aust* 1960;2:999–1002.
26. Gordon S. Dupuytren's contracture: the significance of various factors in its etiology. *Ann Surg* 1954;140:683–686.
27. Hindocha S, John S, Stanley JK, Watson SJ, Bayat A. The heritability of Dupuytren's disease: familial aggregation and its clinical significance. *J Hand Surg* 2006;31A:204–210.
28. Gudmundsson KG, Arngrimsson R, Sigfusson N, Bjornsson A, Jonsson T. Epidemiology of Dupuytren's disease: clinical, serological, and social assessment. The Reykjavik study. *J Clin Epidemiol* 2000;53:291–296.
29. Zerajic D, Finsen V. Dupuytren's disease in Bosnia and Herzegovina. An epidemiological study. *BMC Musculoskeletal Disord* 2004;5:10–14.



30. Wheeler ES, Meals RA. Dupuytren's diathesis: a broad-spectrum disease. *Plast Reconstr Surg* 1981;68:781-783.
31. Garrod AE. Concerning pads upon the finger joints and their clinical relationships. *BMJ* 1904;2:8-9.
32. Hueston JT. The management of ectopic lesions in Dupuytren's contracture. In: Hueston JT, Tubiana R, eds. *Dupuytren's disease*. Edinburgh: Churchill Livingstone, 1974:145-147.
33. Mikkelsen OA. Knuckle pads in Dupuytren's disease. *Hand* 1977;9:301-305.
34. Stradner F, Ulreich A, Pfeiffer KP. [Dupuytren's contracture as a concomitant disease in diabetes mellitus.] *Wien Med Wochenschr* 1987;137:89-92.
35. Skoog T. Dupuytren's contracture with special reference to aetiology and improved surgical treatment, its occurrence in epileptics, note on knuckle pads. *Acta Chir Scand* 1948; 96(suppl 39):25-175.
36. Dupuytren BG. Permanent retraction of the fingers, produced by an affection of the palmar fascia. *Lancet* 1833-34; 2:222-223.
37. Hueston JT, Seyfer AE. Some medicolegal aspects of Dupuytren's contracture. *Hand Clin* 1991;7:617-634.
38. Liss GM, Stock SR. Can Dupuytren's contracture be work related? Review of the evidence. *Am J Ind Med* 1996;29: 521-532.