

# Do Hair Care Practices Affect the Acquisition of Tinea Capitis?

## A Case-Control Study

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**Objective:** To determine the influences of hair-grooming practices and environmental factors as risk factors for the acquisition of tinea capitis (TC) in children.

**Design:** Case-control study comparing children with culture-proved TC with age-, sex-, and race-matched control subjects without scalp disease.

**Setting:** A multicenter study involving 3 urban referral centers in the United States.

**Participants:** A convenience sample of 66 patients aged 12 years and younger presenting to pediatric dermatology clinics with clinical evidence of TC were enrolled as cases. Matched control subjects (n=68), without known scalp disease, were enrolled from the outpatient pediatric clinics at the same institutions.

**Results:** Significant associations with TC in the conditional logistic regression model were a prior history of TC (odds ratio, 3.11; 95% confidence interval, 1.02-9.43;  $P=.04$ ) and exposure to TC (odds ratio, 16.32; 95% confidence interval, 3.55-75.16;  $P=.001$ ). The use of a hair conditioner was statistically significant in the univariable model but not in the multivariable model (odds ratio, 0.46; 95% confidence interval, 0.20-1.08;  $P=.07$ ). Hairstyling, frequency of washing, use of oils or grease, and other hair care practices were not shown to be associated with the presence of TC.

**Conclusions:** Hair-grooming practices do not appear to play a major role in the acquisition of TC. Hair conditioners may be protective in children at risk for TC, but further studies are needed to confirm this finding.

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**T**INEA CAPITIS (TC), a dermatophyte infection of the scalp and hair, is a common infection in children. It has become an important public health problem in the United States.<sup>1,2</sup> The most prevalent causative organism in most of the country is *Trichophyton tonsurans*,<sup>3-7</sup> which presents in various ways, from minimally symptomatic dandrufflike scaling to tender, highly inflamed, purulent nodules known as kerions.<sup>2-6</sup> If untreated, TC may lead to scalp scarring and permanent hair loss.<sup>8</sup> Alternatively, asymptomatic infection may persist undetected for years, with shedding of spores and spreading to susceptible contacts.<sup>9,10</sup> This carrier state, defined as *asymptomatic infection with T tonsurans*, is found to be as high as 15% among urban African American schoolchildren.<sup>11,12</sup>

For unknown reasons, African American children constitute at least 90% of the cases of TC in most series,<sup>2</sup> and have a higher risk of acquisition of TC.<sup>13</sup> Inter-

familial spread is common,<sup>12</sup> and *T tonsurans* can also be cultured from fomites.<sup>1</sup> Some researchers<sup>14-16</sup> have suggested that hairstyling practices may be a factor in acquisition of the disease. Sharp and blunt trauma have been shown to predispose the scalp to dermatophyte infection.<sup>17,18</sup> Hair oils may promote the transmission of the disease,<sup>19</sup> but their precise role has not been determined. To our knowledge, only one other study,<sup>15</sup> published in 1968, has evaluated the role of hair care in patients with TC. Most cases in that study were due to *Microsporum audouinii* infection, not currently the organism responsible, and the use of oils or other hair care products was not evaluated.

In our study, symptomatic children aged 12 years and younger with culture-proved TC were compared with age-, sex-, and race-matched control subjects without clinical evidence of scalp disease to assess factors that might be associated with TC.

The primary objective of this study was to determine the association of hair

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## SUBJECTS AND METHODS

This study was designed as a case-control multicenter study, conducted at 3 urban tertiary pediatric dermatology referral centers. Approval for the study was obtained by the institutional review boards at the University of California, San Francisco, School of Medicine, and the State University of New York Health Sciences Center at Brooklyn; and by the Pediatric Institutional Review Board at the University of Missouri School of Medicine at Kansas City. Subjects were recruited from pediatric dermatology and general pediatric clinics at these institutions. Signed informed consent to participate in the study was obtained from a parent or legal guardian of all subjects.

Patients were enrolled from the pediatric dermatology clinics between July 1, 1996, and June 30, 1999, if they were 12 years old or younger; if they had symptomatic and clinical evidence of TC, such as a kerion, scaling, pustules, crusts, erythema, and/or hair loss; and if the investigators (V.S., N.B.S., R.H., C.T.T., T.A.L., and I.J.F.) had the time and personnel present in the clinic to enroll them into the study. Similarly, age-, race-, and sex-matched control subjects were recruited into the study, at the convenience of the investigator, from general pediatric clinics at the same institutions if they had no clinical evidence of TC and were not presenting for treatment of skin, scalp, or hair disease. We did individual matching, and age was matched within a year. Cases and controls were enrolled at different times within the study period. Investigators did not note anyone refusing to participate in the study. Exclusion criteria for enrollment were as follows: if consent could not be obtained, if there was known congenital or acquired immunodeficiency (eg, human immunodeficiency virus or organ transplantation), or if there was pre-existing scalp or hair disease other than TC. The numbers of patients meeting the exclusion criteria were estimated to have been less than 2 per center. Our sample size was limited to the numbers of subjects who were enrolled by each of the centers during the study period.

All children in the study underwent a visual examination of the hair, scalp, and skin above the neck; palpation of cervical lymph nodes; and fungal scalp cultures. Using the brush-culture method,<sup>20</sup> scalp debris was collected from representative areas of the scalp in children with TC and from 4 areas of the scalp in control children and was inoculated onto Sabouraud dextrose agar or a dextrose agar containing papiac digestive soybean meal, cyclohexamide, and chloramphenicol agar (Mycosel). All positive

culture results were processed by the respective hospital's mycology laboratory. Cultures without fungal growth at 28 days were reported as negative for fungus and discarded. An investigator (V.S., N.B.S., R.H., C.T.T., T.A.L., or I.J.F.) collected demographic data and administered a questionnaire to parents or guardians related to historical and familial factors known to be associated with TC, scalp hygiene, and hair-grooming practices of their child. All children who were diagnosed as having TC were treated by a pediatric dermatologist. If control subjects had a positive culture result, they were also contacted, treated as indicated, and excluded as control subjects.

Our modeling strategy started with simple descriptive statistics. We examined single variable models, and then multivariable models. Our objective was to find a model that gave the best associations among related factors for having TC. Our analysis compared cases with controls using matched groups. A conditional logistic regression model was used to examine the association between case status and several factors. This model controlled for age, sex, and race by having an identifier to group all those with the same year of age, sex, and race category. We did not account for the hospital at which the children were seen in the matching. We dichotomized all the variables, including racial categories, that were designated as African American and other. We looked at continuous variables, such as number of shampoos per month, in several ways, ie, as a continuous variable and as 2 different cut points ( $\geq 15$  and  $\geq 5$  shampoos per month) (data not shown). All analyses yielded similar results.

Candidate variables for these models included the following:

1. Hair care variables: number of shampoos per month; use of a conditioner; hairstyles, such as braids and ponytails; use of a comb, pick, brush, curlers, straighteners, or oil or grease; and sharing of hair utensils.
2. Historical variables: prior history of exposure to TC, medical history of asthma or atopic dermatitis, prior history of griseofulvin use, and household history of TC.
3. Environmental variables: day care attendance, number of people in the household, and pets in the household.

Our analysis began with single variable models. For the multivariable model, we added all significant potential covariates to the model, then we sequentially dropped covariates from the model until all the covariates were significant at  $P < .05$ . Statistical analysis was conducted using Stata for Windows, version 6.0 (Stata Corp, College Station, Tex).

care practices with TC in children. Secondary objectives were to determine the associations of medical history and environmental factors with TC in children.

## RESULTS

Three centers recruited a total of 145 patients into the study; of these patients, 69 were designated as cases and 76 as controls. Because of administrative errors, there were 6 controls without individually matched cases and 3 cases without controls. Two other control subjects had positive culture results, 1 for *T tonsurans* and 1 for *Trichophyton rubrum*. These 11 subjects were excluded from the

statistical analysis. A final total of 134 subjects were analyzed.

Fifty-four (40%) of the cases and controls were recruited from San Francisco, 44 (33%) from Kansas City, and 36 (27%) from Brooklyn. All 66 cases had positive culture results. Sixty-five cultures were positive for *T tonsurans*, and 1 was positive for *T rubrum*. The cultures for *T rubrum* came from a single center in Brooklyn. A description of the study subjects is found in **Table 1**. There were 74 boys (55%), and 132 subjects (99%) were African American; the mean age was 5.1 years for the cases and 5.3 years for the controls (range, 1-12 years). The average number of times cases shampooed was 7.3 per

**Table 1. Description of the 134 Study Subjects\***

Variable	Cases (n = 66)	Controls (n = 68)
Age, mean ± SD, y	5.1 ± 2.3	5.3 ± 2.4
Sex		
Male	37 (56)	37 (54)
Female	29 (44)	31 (46)
Race		
African American	65 (99)	67 (99)
Other (Asian)	1 (1)	1 (1)
Center		
San Francisco, Calif	27 (41)	27 (40)
Kansas City, Mo	20 (30)	24 (35)
Brooklyn, NY	19 (29)	17 (25)
History of exposure to TC	25 (38)	2 (3)
History of having had TC†	20 (30)	8 (12)
Day care attendance	48 (73)	51 (75)
Hairstyle‡		
Braids or pony tails	25 (38)	24 (35)
Natural	38 (58)	38 (56)
Hair oil or grease used§	9 (14)	9 (13)
Conditioner used	22 (33)	35 (51)
Hair utensils shared	39 (59)	43 (63)
No. of shampoos per mo		
Mean ± SD†	7.3 ± 6.1	8.4 ± 7.5
≥5	37 (56)	33 (49)

\*Data are given as the number (percentage) of subjects unless otherwise indicated. TC indicates tinea capitis.

†Two responses are missing.

‡For this variable, percentages do not total 100 because n = 63 for cases and n = 62 for controls.

§In the past 6 months.

month compared with 8.4 per month among controls (range, 1-30 times per month). There was a history of asthma in 14 (21%) of the cases and in 21 (31%) of the controls. A history of atopic dermatitis was noted in 9 (14%) of the cases and in 9 (13%) of the controls. Among the 66 cases, scaling was the most common symptom, recorded in 62 (94%) of the subjects; alopecia and crusting were found in 46 (70%) and 28 (42%) subjects, respectively. Erythema, pustules, and cervical lymphadenopathy each occurred in 22 (33%) of the subjects.

Results of the univariate conditional models for TC stratified for age, race, and sex are shown in **Table 2**. Variables found to be statistically significant in the single variable models were as follows: a prior history of having TC ( $P = .01$ ), a history of exposure to TC ( $P = .001$ ), a history of undergoing treatment for TC ( $P = .03$ ), and the use of a conditioner ( $P = .04$ ). In the final multivariate model, the variables found to be significant were a history of exposure to TC ( $P = .001$ ) and a prior history of having TC ( $P = .04$ ). The statistical significance of using a conditioner declined to a borderline level ( $P = .07$ ). A history of being treated for TC was correlated with a prior history of having TC and was not statistically significant ( $P = .54$ ) in the final model.

#### COMMENT

Tinea capitis is the most common dermatophyte infection in children, with an increasing prevalence.<sup>13,21</sup> Many countries, including the United States,<sup>13</sup> have observed

**Table 2. Conditional Logistic Regression Model for TC, Stratifying for Sex, Race, and Age\***

Variable	Odds Ratio (95% Confidence Interval)	
	Crude	Adjusted
History of exposure to TC	27.0 (5.9-122.7)	16.3 (3.6-75.1)
History of having had TC	3.9 (1.5-10.5)	3.1 (1.0-9.4)
Conditioner used	2.1 (1.0-4.3)	0.5 (0.2-1.1)
Treated for TC	4.6 (1.2-17.7)	1.8 (0.3-11.4)
Frequency of shampoo†	1.0 (0.9-1.0)	1.0 (0.9-1.0)
Length of hair‡	0.8 (0.6-1.1)	...
Hairstyle		
Braids§	0.7 (0.3-1.7)	...
Ponytails	1.4 (0.5-3.8)	...
Hair oil or grease¶	1.6 (0.6-4.1)	...
Hair utensils shared	0.7 (0.3-1.4)	...

\*TC indicates tinea capitis; ellipses, data not computed because these variables were excluded from the final multivariate model.

†Number of shampoos per month.

‡Measured in centimeters.

§Compared with no braids.

||Compared with no ponytails.

¶Used in the past 6 months compared with no use.

an increased incidence of infections in individuals with African ancestry,<sup>22</sup> but the reasons for this are not known. The clinical findings in our patients with TC were similar to those observed in many other studies. Scaling was the most common finding, but our rate of cervical lymphadenopathy (33%) was somewhat lower than that given in previous reports.<sup>23-25</sup> The reasons for this are unclear.

Infection, and reinfection, of individuals within families, communities, and schools is frequent and probably represents the most common mode of acquisition of infection, because of exposure to individuals with active infection and because of reservoirs of the organism in asymptomatic individuals.<sup>1,8,10,11</sup> Our study confirmed this, since a prior history of TC and a history of exposure to TC showed the strongest associations of the many variables assessed.

The study did not, however, confirm associations with many of the factors that have previously been speculated as being, at least in part, responsible for the acquisition and/or spread of TC. Sharing of combs and brushes has been suspected of being a common mode of transmission in infected siblings in several studies,<sup>15,26</sup> but was not a factor in our study. This finding does not exclude the possibility that combs and brushes could act as a vector for infection, but sharing combs and brushes is apparently common—more than 60% in both groups—which may explain the lack of association. Close physical contact has also been proposed as a mode of spread,<sup>12</sup> but we failed to find significant differences between the groups for exposure to sites where close physical contact or prolonged periods of contact are expected, such as day care attendance or families with many children. Similarly, hair-grooming practices, such as the use of oils, gels, and specific hairstyles (eg, braids), were not associated with TC, as has been suggested by some researchers.<sup>14,27</sup> Other researchers<sup>15,16,28</sup> have speculated that improved personal hygiene alone

### What This Study Adds

Tinea capitis (TC) is a dermatophyte infection of the scalp that is an important public health problem in children. Hair care factors are thought to promote transmission of the disease, but their role has not been recently evaluated.

This study explored the role of hair care practices in a case-control study of TC. Our findings of associations of TC with a history of exposure to TC and a prior history of TC support previous research findings. Contrary to earlier research, we did not find TC to be associated with hair care practices. An association of a possible protective effect for the use of hair conditioners needs to be explored further in a larger prospective study.

may play a role in the control of TC. While this may be a factor in institutional settings, we did not find the frequency of shampoos to be associated with TC. This study is limited by the sample size, which, although sufficient to detect large differences, might not have been sufficient to detect smaller differences, and some significant associations may not have been detected.

Surprisingly, the use of a conditioner did appear to have a slight protective effect that was independent of the number of shampoos per month or the type of conditioner. We did not measure the frequency of use of conditioners; we assumed it to be correlated with the frequency of shampoos. Although the effect was of borderline statistical significance, this could reflect the relatively small numbers in the groups. Conditioners allow the hair to be more manageable after shampooing. This effect is achieved by deposition of the conditioner droplets onto the hair and scalp surface. It is possible that these droplets could have a suppressive effect on spore growth or make the hair a hostile environment for the fungus. Future studies could focus on trying inexpensive interventions to prevent this disease. Many shampoos, such as 2.5% selenium sulfide, 1% econazole nitrate, and 4% povidone-iodine shampoo, have shown reductions in fungal carriage in asymptomatic children.<sup>29</sup> In addition, shampooing with selenium sulfide appears to suppress viable spores on the scalp and offers important adjunctive therapy for TC.<sup>30</sup>

In conclusion, our study failed to find an association with hair care styling practices, but did find a possible protective effect for the use of conditioners. Larger studies will be necessary to further study the role of conditioners as a method of preventing the spread of TC.

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### REFERENCES

1. Hebert AA, Head ES, MacDonald EM. Tinea capitis caused by *Trichophyton tonsurans*. *Pediatr Dermatol*. 1985;2:219-223.
2. Lucky AW. Epidemiology, diagnosis and management of tinea capitis in the 1980's. *Pediatr Dermatol*. 1985;2:226-228.
3. Babel DE, Rogers A, Beneke E. Dermatophytosis of the scalp: incidence, immune response, and epidemiology. *Mycopathologia*. 1990;109:69-73.
4. Bronson DM, Desai DR, Barsky S, et al. An epidemic of infection with *Trichophyton tonsurans* revealed in a 20-year survey of fungal infections in Chicago. *J Am Acad Dermatol*. 1983;8:322-330.
5. Aly R. Incidence of dermatophyte in the San Francisco Bay area. *Dermatologica*. 1980;161:97-100.
6. Laude TA, Shah BR, Lynfield Y. Tinea capitis in Brooklyn. *AJDC*. 1982;136:1047-1050.
7. Sinski JT, Flouras K. A survey of dermatophytes isolated from human patients in the United States from 1979 to 1981 with chronological listings of worldwide incidence of five dermatophytes often isolated in the United States. *Mycopathologia*. 1984;85:97-120.
8. Seale E, Richardson J. *Trichophyton tonsurans*, a follow-up of treated and untreated cases. *Arch Dermatol*. 1960;81:87-94.
9. Prevost E. *Trichophyton tonsurans* tinea capitis [letter]. *JAMA*. 1980;244:2522.
10. Honig PJ, Smith LR. Tinea capitis masquerading as atopic or seborrheic dermatitis. *J Pediatr*. 1979;94:604-605.
11. Vargo K, Cohen BA. Prevalence of undetected tinea capitis in household members of children with disease. *Pediatrics*. 1993;92:155-157.
12. Williams JV, Honig PJ, McGinley KJ, et al. Semiquantitative study of tinea capitis and the asymptomatic carrier state in inner-city school children. *Pediatrics*. 1995;96:265-267.
13. Lobato MN, Vulgia DJ, Frieden IJ. Tinea capitis in California children: a population-based study of a growing epidemic. *Pediatrics*. 1997;99:551-554.
14. Sharma V, Hall JC, Knapp JF, et al. Scalp colonization by *Trichophyton tonsurans* in an urban pediatric clinic? asymptomatic carrier state. *Arch Dermatol*. 1988;124:1511-1513.
15. Reid BJ, Shimkin MB, Blank F. Study of tinea capitis in Philadelphia using case and control groups. *Public Health Rep*. 1968;83:497-502.
16. Bocofo FC, Eadie GA, Miedler LJ. Epidemiologic study of tinea capitis caused by *T. tonsurans* and *M. audouinii*. *Public Health Rep*. 1965;80:891-898.
17. Jahan V, Frieden IJ, LeBoit PE. Inflammatory nodule on the scalp. *Pediatr Dermatol*. 1990;7:153-155.
18. Kligman A. The pathogenesis of tinea capitis due to *Microsporum audouinii* and *Microsporum canis*. *J Invest Dermatol*. 1952;18:231-246.
19. Higgins EM, Fuller LC, Smith CH. Guidelines for the management of tinea capitis. *Br J Dermatol*. 2000;143:53-58.
20. Hubbard TW, de Triquet JM. Brush-culture method for diagnosing tinea capitis. *Pediatrics*. 1992;90:416-417.
21. Wilmington M, Aly R, Frieden IJ. *Trichophyton tonsurans* tinea capitis in the San Francisco Bay area: increased infection demonstrated in a 20-year survey of fungal infections from 1974 to 1994. *J Med Vet Mycol*. 1996;34:285-287.
22. Hay RJ, Clayton YM, De Silva M, et al. Tinea capitis in south-east London: a new pattern of infection with public health implications. *Br J Dermatol*. 1996;135:955-958.
23. Hubbard TW. The predictive value of symptoms in diagnosing tinea capitis. *Arch Pediatr Adolesc Med*. 1999;153:1150-1153.
24. Ravits MS, Himmelstein R. Tinea capitis in the New York area. *Arch Dermatol*. 1983;119:532-533.
25. Babel DE, Baughman SA. Evaluation of the adult carrier state in juvenile tinea capitis caused by *Trichophyton tonsurans*. *J Am Acad Dermatol*. 1989;21:1209-1212.
26. Malhotra YK, Garg MP, Kanwar AJ. A school survey of tinea capitis in Benghazi, Libya. *J Trop Med Hyg*. 1979;82:59-61.
27. Frieden IJ, Howard R. Tinea capitis: epidemiology, diagnosis, treatment and control. *J Am Acad Dermatol*. 1994;31:342-346.
28. Wright S, Robertson VJ. Institutional survey of tinea capitis in Harare, Zimbabwe and a trial of miconazole cream versus Whitfield's ointment in its treatment. *Clin Exp Dermatol*. 1986;11:371-377.
29. Neil G, Hanslo D. Control of the carrier state of scalp dermatophyte. *Pediatr Infect Dis J*. 1990;9:57-58.
30. Allen HB, Honig PJ, Leyden JJ, et al. Selenium sulfide: adjunctive therapy for tinea capitis. *Pediatrics*. 1982;69:81-83.