A Double-blind Comparison of Clomipramine and Desipramine Treatment of Severe Onychophagia (Nail Biting)

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Twenty-five adult subjects with severe morbid onychophagia (nail biting) and no history of obsessive-compulsive disorder were enrolled in a 10-week double-blind crossover trial of clomipramine hydrochloride and desipramine hydrochloride. For the 14 subjects who completed the study, clomipramine hydrochloride (mean ± SD dose, 120 ± 48 mg/d) was superior to desipramine hydrochloride (mean ± SD dose, 135 ± 53 mg/d) in decreasing nail biting as measured by a repeated-measures analysis of variance on the Nail Biting Severity, Nailing Impairment, and Clinical Progress scales. The high dropout rate at every stage of the study was in sharp contrast to that seen with psychiatric populations. From a neurophysiologic perspective, similar biologic systems are hypothesized to mediate a spectrum of grooming behaviors, including onychophagia, trichotillomania, and obsessive-compulsive disorder.

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Onychophagia (habitual nail biting) is a widespread behavior among children and adults of all ages, degrees of intelligence, and socioeconomic status. Starting as early as the age of 4 years, nail biting peaks between the ages of 10 and 18 years and is more common in females than males (1.5:1). The prevalence of nail biting in childhood has been estimated to be approximately 50%. By age 17 to 18 years, the behavior decreases somewhat, and the prevalence among 2500 naval recruits was found to be 23%. Although most childhood nail biters probably discontinue this behavior sometime during adulthood, nail biting may persist, and one study found that 4.5% of people in their 60s had retained the habit.

Nail biting appears to be familial. The incidence in twin children whose parents had been nail-biters (most of whom had ceased) was almost three times greater than for those whose parents had not had the behavior. Monozygotic twins had a 66% concordance for nail biting, in comparison with 34% in dizygotic twins. For severe nail biting, monozygotic twins had four times (75%) the concordance that dizygotics did (18%).

Severe pathologic onychophagia is the subject of this report. While most habitual nail biting is considered quite trivial, severe morbid onychophagia may cause both medical and dental problems. The most common complications are chronic subungual infection and recurrent paronychia, but onychodystrophy, secondary bacterial or fungal infections, onychia (acute inflammation), periungual warts, matrix damage resulting in scarring, pterygium, and damage to the nail bed with loss of the nail also occur. Severe nail biters may also bite the cuticles and the skin of the fingers, resulting in inflammation, scars, excoriation, and eventually keloids. Onychotillomania, a compulsive picking or tearing at the nails, may be a variation of the behavior in individuals. Severe nail biting may cause craniofacial dysfunction problems, and it may be a factor in idiopathic tooth apical root resorption, posing a risk for orthodontic treatment. In addition to fingernail damage, biting and/or clipping of the toes and toenails may have similar secondary problems.

A variety of treatments have been reported for onychophagia, including psychotherapy, cue-controlled relaxation, hypnosis, and a plethora of behavioral treatments, such as negative practice, positive reinforcement, covert sensitization, contingency contracting, self-monitoring, habit reversal/competing response, overcorrection with artificial nails, and application of an occlusive dressing or bitter substance. Although short-term reduction in nail biting is reported after these modalities, no systematic studies of any treatment have been carried out. No reports of drug treatment for onychophagia could be found.

During the course of a double-blind comparison of clomipramine hydrochloride and desipramine hydrochloride for the treatment of severe primary obsessive-compulsive disorder (OCD) in adolescents, Leonard et al observed that several patients with concomitant nail biting noted that this too had abated during treatment with clomipramine treatment but was unchanged during the
Individual behavior beyond was with manner ing, and content der OCD specifically, Subjects An Medical/neurologic basis ECG Nail Nail-Nail Rituals. 31, 33 of desipramine. in impulse control (312.29), 29 responded selectively to clomipramine and not to desipramine treatment in a double-blind treatment comparison. An etiologic perspective for OCD has been proposed, on the basis of observations in children, that the most frequent content of the OCD ritualizing behavior, ie, washing, licking, preening, and picking, resembles exaggerated grooming rituals. 30, 32 Severe nail biting, like trichotillomania, has some face validity as an “excess grooming” behavior and is described as an unwanted repetitive irrational behavior engaged in by otherwise sensible individuals, similar to the manner in which patients with OCD describe their compulsive rituals. Therefore, we hypothesized that severe nail biting, like OCD, might belong to a spectrum of behaviors that show a preferential response to clomipramine in comparison with desipramine. We were also interested in determining whether there is a clinical role for drug treatment of severe nail biting. To our knowledge, this is the first pharmacologic study of onychophagia.

SUBJECTS AND METHODS

Subjects

Subjects were recruited through an advertisement in the health section of a local newspaper that requested severe chronic nail biters for a 3-month drug treatment study. There was no mention of OCD. Volunteers had to be 18 years of age or older and had to have started nail biting in childhood. Inclusion criterion were chronic nail biting or nail clipping behavior that was severe enough to cause observable damage and was emotionally distressing to the individual. Subjects had to meet criteria for severe nail biting, defined as fingernails bitten beyond the free edge and with the nail margin below the soft-tissue border. 5 In addition, evidence of nail damage, such as scarring, infections, and bleeding, had to be apparent, or the individual had to have sought treatment for this behavior or its sequelae in the past. Patients were excluded if they had OCD, a medical or neurologic disorder, psychosis, mental retardation, primary major affective disorder, or current alcohol or other drug abuse/dependence. History of an anxiety (other than OCD) or depressive disorder or substance abuse/dependence was not exclusionary. Neither concurrent medication (with the exception of birth control pills) nor concurrent behavioral treatment was permitted. Subject selection became a major focus of the project. From the 346 initial telephone inquiries, 72 callers (20%) could not be reached or did not return our calls. After the study was described by telephone to the remaining 274 callers, 63 (18%) were excluded by the telephone screening, 136 (39%) rejected further contact, and the remaining 75 (22%) were scheduled for screening. Only 52 of the 75 scheduled subjects actually kept their screening appointments. At screening, 15 were excluded for psychiatric (seven subjects) or medical (three) contraindications, lack of severity (three), inability to complete rating scales (one), and desire to become pregnant (one). Of interest, three prospective subjects were excluded for the diagnosis of OCD, one during the initial telephone inquiry and two at screening. While 37 (11%) of the original group met criteria for the study, only 25 (7%) decided to participate. This study was approved by the National Institute of Mental Health Clinical Research Review Committee, and each subject gave informed consent.

Baseline Evaluation

All subjects underwent medical, neurologic, and psychiatric evaluation (Table 1). Photographs of the nails were taken. Psychiatric histories were obtained by means of structured interviews (the Schedule for Affective Disorders and Schizophrenia—Lifetime Version 4 and the Structured Interview for Personality Disorders 5), and these were verified by clinical interview. Additionally, the Leyton Obsessional Inventory was completed. * Laboratory examination included electrocardiogram, complete blood cell count, general chemistry screen, thyroid function tests, hepatitis B antibody, human immunodeficiency virus antibody, and urinalysis. Twenty-five subjects, six men and 19 women, with a mean age of 32.7±6.3 years (range, 21.0 to 42.0 years) entered the study. All subjects had been biting their nails for as long as they could remember. Thus, presumably all had had an onset before age 12 years. All had observable changes in their nails secondary to chronic biting, with distally foreshortened nails and no free nail visible. Cuticles and skin around the nail were frequently mutilated by picking, clipping, and/or biting. Six subjects reported that biting, picking, and/or overclipping the toenails was a significant problem in addition to their fingernail biting, and for one subject this was the predominant symptom. Subjects reported ways in which their nail biting interfered in their lives. For some, nails could be made so painful that they were unable to type, open tops of soda cans, or button their own shirts. One man who overclipped his toenails said that on occasion his toes were so painful that he could not walk.

Table 1.—Clomipramine-Desipramine Treatment Comparison for Nail Biting: Study Design*

<table>
<thead>
<tr>
<th>Measure</th>
<th>Week 0 (Baseline)</th>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 3</th>
<th>Week 4</th>
<th>Week 5</th>
<th>Week 6</th>
<th>Week 7</th>
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<th>Week 9</th>
<th>Week 10</th>
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*NIMH indicates National Institute of Mental Health; and ECG, electrocardiogram.
All 25 subjects had attempted some form of previous treatment, including foul-tasting solutions applied to nails (11 subjects), psychotherapy (seven), hypnosis (five), manicures (five), artificial nails (three), relaxation techniques (two), gloves (two), biofeedback (one), gift incentives (one), and aversive reinforcement with rubber bands (one).

Seventeen of 25 patients met lifetime criteria for one or more Axis I psychiatric diagnosis of major depression (11 subjects), dysthymic disorder (five), late luteal–phase dysphoria (one), substance abuse (alcohol in three and other drugs in three), generalized anxiety disorder (six), and phobic disorder (three). (Seven subjects had a lifetime history of an anxiety disorder, and two of these individuals had both generalized anxiety disorder and phobic disorder.) Four subjects had at least one personality disorder, including dependent (one), avoidant (one), passive-aggressive (two), histrionic (one), and obsessive-compulsive (two). No subject currently met criteria for an affective or anxiety disorder, and seven received no lifetime Axis I or II diagnosis.

The mean ± SD Leyton Obsessional Inventory scores were as follows: total number of "yes" responses, 17.0 ± 12.5; number of symptoms, 10.2 ± 8.8; and personality traits, 6.8 ± 4.4. The mean ± SD resistance score was 11.8 ± 13.4, and the mean interference score was 11.4 ± 14.7. These values are similar to those reported for normal samples.

The lifetime prevalence of nail biting and major psychiatric diagnoses, including trichotillomania and OCD, in each subject's family was ascertained by means of the family history method. The 25 adult subjects had 14 first-degree relatives: parents (50), siblings (60), and children 6 years of age or older (four). Two of the parents were unknown to the probands. Of the 112 about whom something was known, seven (6%) had some severe nail-damaging behavior: four (4%) were reported to be severe nail biters, and three others picked (one) or chewed (one) their hands or feet (one). Two relatives (1.8%) had probable OCD, and the description of symptoms of an additional six relatives (5%) was suggestive of OCD, but there was not enough information available to rate severity and interference. Two sisters (1.8%), of two different subjects, had been treated for anorexia and bulimia. Three relatives (3%) had received inpatient hospitalization, two for unclear reasons and one for compulsive gambling. Two relatives (1.8%) had probable affective disorder.

**Procedures**

The study was a 12-week outpatient study as outlined in Table 1. As shown, after the initial evaluation, a 2-week single-blind placebo trial was initiated, during which any subject having more than 20% improvement on the global impairment scale during the placebo period would be excluded from the study. After the placebo period, a double-blind balanced-order comparison of 5 weeks each of clomipramine and desipramine was conducted. Clomipramine and desipramine were dispensed in identical 25- or 50-mg capsules. The initial dose of 25 mg/d was increased as tolerated during a 5-week period to a maximum of 3 mg/kg per day (not to exceed 250 mg/d). At the end of the first active drug phase, the first drug was tapered during 5 days as the second drug was gradually increased. No other psychotropic medications or concomitant behavioral treatments were allowed.

At the completion of each active drug phase, 12 hours after the last dose of medication, blood was drawn for complete blood cell count, chemistry studies, and plasma drug levels. In addition, an electroencephalogram was obtained at the end of each active treatment phase and compared with that at baseline.

**Clinical Assessment**

As shown in Table 1, subjects were interviewed weekly to evaluate severity of nail biting, depression, and anxiety, to assess any side effects of the medication, and to adjust the dose.

Side effects were rated on the Subjective Treatment Emergent Symptoms Scales, of which each of 23 symptoms was rated as none, slight, or severe. The National Institute of Mental Health Global Assessment Scales for depression and anxiety were used to assess those symptoms. Nail biting ratings were adapted from scales used in the trichotillomania treatment study of Swedo and colleagues for assessing the severity of pathologic hair pulling. The Nail Biting Severity Scale is a five-item measure of the severity of symptoms, with the patients' answers scored from 0 (none) to 5 (most severe). These questions covered the amount of time spent nail biting each day of the past week, the intensity of the nail biting urge, resistance exerted against it, the distress caused by the nail biting, and the degree of interference (from time spent or resultant embarrassment about appearance) in daily life. The Nail Biting Impairment Scale is an 11-point scale for the assessment of overall impairment resulting from the nail biting, which was rated as none (0), minimal (1 to 3), moderate (4 to 6), or severe (7 to 10). The Clinical Progress Scale was rated by a physician; 0 was the absence of symptoms, 10 the pretreatment level of severity, and 20 a hypothetical state of nearly constant nail biting. All ratings were carried out by two of us (H.L.L. and S.E.S.), whose interrater reliability k scores ranged from 0.78 to 1.00 for these three measures.

**Statistical Analysis**

Clinical ratings of nail biting behavior, anxiety, and depression were compared at week 5 of the two drug phases by means of a 2 × 2 repeated-measures analysis of variance for drug and drug order. We hypothesized a greater response to clomipramine than to desipramine on the basis of the observation that several patients with OCD had stopped biting their nails while taking clomipramine and that another grooming behavior, trichotillomania, preferentially responded to clomipramine. Consequently, one-tailed P values for nail biting severity and impairment measures were used. Depression and anxiety ratings, however, were evaluated with two-tailed tests.

The relative frequency of side effects reported during the two drug treatments was examined by means of Fisher's Exact Test (two tailed).

Pearson's product-moment correlation or Spearman's rank-order correlation coefficients (depending on whether the values were normally distributed or not) were performed to evaluate possible predictors of the differential response between clomipramine and desipramine.

**RESULTS**

Only 14 of the 25 subjects who entered the investigation completed the study. One subject with a 66% improvement with placebo treatment was dropped. Ten subjects dropped out of the study. One subject quit during the placebo phase because she believed the study was too time consuming. Nine left the study while taking active medication (four during the clomipramine phase and five during desipramine treatment). Three of the nine (active treatment phase) dropouts occurred during the first week of desipramine treatment in phase B, and it appeared that at least two of these were related to withdrawal of the clomipramine, despite the tapering design. One experienced a flulike syndrome similar to a clomipramine (tricyclic) withdrawal syndrome. The other, a 36-year-old woman, experienced an exacerbation of her migraine headaches both while starting and while tapering clomipramine treatment. The complex migraine she experienced during tapering of clomipramine was severe enough to suggest a transient ischemic attack initially, and she chose to drop out of the study. The dropouts during the clomipramine phase occurred because of vomiting (two), dysphoria and insomnia (one), and stomachache and streptococcal pharyngitis (one); those during desipramine treatment were for stomachache (two), rash (one), migraines (one), and flulike syndrome...
(one), as described above. This is in contrast to our previous studies with these two drugs, in which dropouts were virtually nonexistent.28

Even for those completing the study, it was difficult to reach an adequate dose of either drug. Surprisingly, five subjects (four receiving clomipramine and one receiving desipramine) could not tolerate the side effects resulting from the initial 25-mg dose. When the clomipramine hydrochloride-treated subjects were restarted on a regimen of 12.5 mg, two tolerated this dose and were gradually able to increase, but two were unable to continue. At the end of 5 weeks of treatment, one subject could not tolerate more than 25 mg of clomipramine hydrochloride, and two others were receiving only 50 mg. Two subjects could not tolerate more than 50 mg of desipramine hydrochloride at the end of 5 weeks.

Mean drug doses at week 5 for the two drugs were similar: 120±48 mg/d (range, 25 to 200 mg/d) for clomipramine hydrochloride and 135±53 mg/d (range, 50 to 250 mg/d) for desipramine hydrochloride. The mean clomipramine plasma levels were 151.8±138.3 ng/mL of clomipramine (range, 14 to 418 ng/mL); the desmethylclomipramine level was 194.8±172.4 ng/mL (range, 21 to 629 ng/mL). The mean plasma level of desipramine was 170±205.5 ng/mL (range, 12 to 725 ng/mL).

As shown in Table 2, there was a greater decrease in nail biting during clomipramine treatment than with desipramine as measured on the Nail Biting Severity (F = 3.75, df = 1,12, P < .04), the Nail Biting Impairment (F = 5.27, df = 1,12, P < .02), and the Clinical Progress (F = 7.65, df = 1,12, P < .01) scales. There was no effect of drug order presentation. The Figure illustrates the mean scores for the two drug sequences, as measured on the Clinical Progress Scale. Since no subject was clinically depressed or anxious at the beginning of the study, it is not surprising that neither clomipramine nor desipramine treatment led to a significant reduction in depression or anxiety scores relative to baseline (Table 2).

The 14 subjects who completed the study did not differ significantly from the 11 who dropped out with respect to the frequency or severity of reported side effects to either drug. Four of the seven with a lifetime history of an anxiety disorder were among the completers. Side effects during clomipramine and desipramine treatment, respectively, included dry mouth (12 and eight subjects), fatigue (10 and five), difficulty sleeping (seven and 10), constipation (six and seven), sweating (six and five), and dizziness (five and three). One 38-year-old man had a twofold increase in his serum alanine aminotransferase level during treatment with clomipramine; there were no other clinically significant changes in results of blood chemistry studies, hematologic findings, or electroencephalogram.

There was no significant relationship between age, sex, severity of nail biting symptoms, degree of depression and anxiety, scores on the Leyton Obsessional Inventory, drug dosage, or plasma drug levels and differential improvement between clomipramine and desipramine at 5 weeks of treatment, as measured on any of the nail biting scales.

The clinical impression was of a more modest response to clomipramine treatment than the statistical differences between clomipramine and desipramine scores would indicate. Seven of the 14 subjects completing both phases (and an additional subject who completed only the clomipramine phase) had at least a 30% decrease in

<table>
<thead>
<tr>
<th>Measure</th>
<th>Baseline</th>
<th>Placebo, Week 2</th>
<th>Desipramine Hydrochloride, Week 5</th>
<th>Clomipramine Hydrochloride, Week 5</th>
<th>Clomipramine vs Desipramine</th>
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<tbody>
<tr>
<td>Nail Biting Severity Scale</td>
<td>17.1±3.3</td>
<td>15.0±3.7</td>
<td>15.3±5.6</td>
<td>12.6±5.6</td>
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<td>Nail Biting Impairment Scale</td>
<td>7.6±0.6</td>
<td>7.6±0.7</td>
<td>7.2±1.1</td>
<td>6.4±2.1</td>
<td>5.27</td>
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<tr>
<td>Clinical Progress Scale</td>
<td>10.0±0.0</td>
<td>8.9±1.2</td>
<td>9.0±2.4</td>
<td>7.1±3.0</td>
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<td>NIMH global anxiety</td>
<td>2.8±1.0</td>
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<td>1.7±1.1</td>
<td>1.6±1.2</td>
<td>1.6±0.9</td>
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<td>0.18</td>
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</table>

* NIMH indicates National Institute of Mental Health; and NS, not significant. Values are mean±SD. All comparisons between the drugs were made by repeated-measures analysis of variance. The ratings shown were made after 5 weeks of treatment with each of the two drugs. For description of the various scales, see the "Subjects and Methods" section. P values reported are one tailed for the nail scales and two tailed for the anxiety and depression scales.
amount and severity of nail biting, as measured on the Clinical Progress Scale. Only two subjects stopped biting their nails completely.

Nine (64%) of the 14 subjects completing the study elected to continue taking some medication. One subject recognized his greater improvement with clomipramine but chose desipramine because he had experienced fewer undesirable side effects while taking it. Three chose to continue taking clomipramine; two discontinued clomipramine treatment after 2 months, while a third man continued for 3 months reporting at that time having no biting. Six individuals who had reported a positive response to clomipramine but disliked the side effects were given an open trial of fluoxetine (4.4±3.8 months); only two continued taking fluoxetine, for 6 and 8 months, and reported a 50% improvement. The remainder of the subjects did not believe that their improvement was sufficient to warrant the expense or unpleasantness of continued drug treatment.

On last contact (five to 29 months after completion of the study), no subject was still taking clomipramine; one was taking fluoxetine, one tranylcypromine sulfate, and one bupropion hydrochloride. For these three subjects, the mean Nail Biting Impairment Scale score was 4.0 (range, 2 to 5), compared with 8 (range, 7 to 9) at baseline.

**COMMENT**

The 14 individuals who completed the study had a greater improvement in their severe nail biting with clomipramine than with desipramine as measured on three clinical nail biting scales. This was in contrast to a lack of differential drug effect on ratings of mild anxiety or depression. Two subjects stopped biting their nails completely with clomipramine, and an additional five had at least a 30% decrease in amount and severity of nail biting. Nine patients believed that there had been enough of an improvement to warrant continuing medication at the end of the study. As shown previously for trichotillomania and OCD, there were no significant predictors of drug response, including baseline measures of anxiety, depression, obsessionality, medication dosage, and drug plasma levels.

One of the most striking phenomena in this study was the great difficulty in obtaining subjects. Only 25 (7%) of the subjects who initially inquired entered the study, and only 14 (4%) completed it, in contrast to our previous experience using the same drugs and study design with OCD and trichotillomania. The relative lack of other psychiatric disturbance may have reduced motivation, and therefore tolerance to the drugs, even in this highly select and motivated group of nail bitters. Clearly clomipramine is not the treatment of choice for most patients with onychophagia, although there may be rare exceptions. In the trichotillomania study, 30 there were no dropouts, and the mean final clomipramine and desipramine dosages were 180.8±56.0 mg/d (range, 100 to 250 mg/d) and 173.1±33.0 mg/d (range, 150 to 200 mg/d), respectively, which are significantly higher than those for the subjects with onychophagia, 120±48 mg/d (range, 25 to 200 mg/d) and 135±53 mg/d (range, 50 to 250 mg/d), respectively. Either the onychophagic subjects did not feel troubled enough by their behavior to tolerate side effects or, less plausibly, a nonpsychiatric population may differ in actual physiologic response and tolerance to clomipramine and desipramine. Blood pressure and pulse during drug treatment were similar across the three groups, arguing against an underlying physiologic difference in response to tricyclics. The clomipramine precipitation of migraine headaches during initiation and discontinuation of the drug in one individual has not, to our knowledge, been previously described in the literature, although 25 (7%) of 3525 patients exposed to clomipramine in all US trials had reported this symptom to the manufacturer (Ciba-Geigy Pharmaceuticals, Summit, NJ; data on file). Since serotonergic dysfunction has been implicated in the development of migraine, 41 it is intriguing that clomipramine modulation of the serotonergic system might precipitate a migraine. 42 However, this patient's side effect may be unrelated, because clomipramine was initially reported to be helpful in the prevention and treatment of migraines in an open trial 43 but was not superior to placebo in two subsequent controlled studies. 44,45

The dose, plasma levels, and dropout rates in this nail biting population were comparable with those in clomipramine treatment studies of other nonpsychiatric groups: trials for migraine headaches, 44,45 chronic tension headaches, 46 chronic pain syndromes, 47-49 and fibrositis. 50 Mean daily doses of clomipramine used to treat these various chronic pain syndromes ranged from 30 to 125 mg. 46,48 Only one study reported plasma levels 48 with similar doses, and even lower levels were obtained than in the present study. Several of these studies also cited dropouts from side effects and poor tolerance to clomipramine as significant problems. 45,47,50 In conclusion, the dose and intolerance to the medication in our study appear to be similar to those seen in other nonpsychiatric populations treated with clomipramine.

The association between nail biting and psychiatric disturbance has never been studied systematically. A common assumption is that nail biting is a sign of emotional tension or anxiety in children 51; however, there is little evidence that children who bite their nails are actually more anxious than those who do not. 52 As Kanner 53 said, "It is hardly realistic to assume that two-thirds of our youths (the nail biters) are degenerates, exquisitely psychopathic or walking around with an unresolved oedipus complex." Kanner 54 however, reported that in children, nail biting was most often associated with motor restlessness and that 19% of nailbiters also had motor tics. In adults, the literature is mixed, with a variety of developmental disturbances having been claimed to be associated with nail biting, including anxiety, 55, 56 an unduly irritable disposition, 57 bedwetting, 58 and sociopathy. 59, 60 However, the incidence of nail biting in subnormal psychopaths was similar to that reported for adult military recruits. 61 Nail biting was not associated with either anxiety or stereotyped body movements in a subnormal population. 62 The present study serves to document that the most severe forms of nail biting can occur in the absence of major psychopathologic disorders. In fact, we were impressed by the regularity with which subjects thought that the nail biting in itself was a cause rather than a symptom of their distress, suggesting that the behavior did not stem from an underlying anxiety disorder.

One might argue that the subjects' differential response to clomipramine might be due to a greater nonspecific anxiolytic effect than that of desipramine. Although this is possible, a number of findings argues against this explanation. None of the subjects met diagnostic criteria for an anxiety disorder at baseline; baseline anxiety scores were
low and remained unchanged after treatment with either medication. Those with and without histories of a lifetime anxiety disorder diagnosis dropped out of the study at a similar rate. Although no controlled studies comparing clomipramine with desipramine for generalized anxiety disorder or phobia could be found, clomipramine and desipramine have been reported to be equivalent treatments for panic disorder, implying similar anxiolytic effects.63

The preferential response of severe nail biting to clomipramine, a drug selectively effective in OCD and trichotillomania, does not necessarily imply a common cause with the other disorders responsive to this medication. Moreover, this study sheds no light on the question of “symptom choice” even if a similar biologic system were found to mediate these behaviors. There was no evidence of increased OCD or other anxiety disorders in the families of onychophagic probands, although this was ascertained indirectly. Onychophagia is similar to trichotillomania in that it does not vary in symptom form over time, and both disorders represent self-mutilating behaviors that seem to be in response to an inner urge. The striking feature of childhood OCD is the predominance of washing and “grooming” rituals, posing a provocative parallel with the behaviors in severe hair pulling and nail biting. Demeret64 described both trichotillomania and severe nail biting, from an ethologic perspective, as disorders of “excessive grooming.” Animal models for OCD have only recently received attention.31,33,65,66 A clomipramine treatment study of canine acral lick suggests that a similar central mechanism mediating the excess grooming behavior in this naturally occurring disorder may be involved across species, as the behavior responded selectively to clomipramine and not to desipramine.60 In summary, this pharmacologic study of onychophagia demonstrated significant, although clinically modest, selective efficacy of clomipramine over desipramine. This finding, in conjunction with the noted similarity in the nature of the different behaviors, provides indirect support for the concept that a biologic system mediating grooming behaviors, perhaps of an evolutionary adaptive significance, may be relevant to the understanding of OCD and complex repetitive behaviors.

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References


