type 17—predominant hypersensitivity response.[5] Importantly, observation of DFR in prepubertal patients does not rule out *Malassezia*-induced seborrheic dermatitis as an etiology because prepubertal patients also develop seborrheic dermatitis, albeit at lower rates.[4] The observed rates in children cannot be directly compared to previously published rates of DFR in adults, because misclassification bias may have underestimated the rates in adults. We treat these patients empirically for a seborrheic dermatitis—like etiology of DFR with ketoconazole 2% cream.

Limitations include this study's retrospective nature, single-institution cohort, small sample size, and risk of misclassification bias. Our study, to our knowledge, is the first dedicated pediatric study for DFR. It suggests that DFR may be more common in postpubertal children than in prepubertal children, which may support a seborrheic dermatitis—like etiology for DFR.

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Heritability of tanning addiction: A twin concordance study

To the Editor: Tanning addiction is a dermatologic-psychiatric disorder with characteristics of behavioral addiction, substance use disorder, and other psychiatric disorders. Minimal information exists regarding the unique contribution of genetic components and environmental exposures to tanning addiction. The objective of this study was to estimate concordance rates and heritability of tanning addiction in a twin cohort.

We administered a questionnaire to monozygotic (MZ) and dizygotic (DZ) twin pairs at the 2018 Twins Day Festival (Twinsburg, OH) to evaluate for tanning addiction using the previously validated tanning-modified *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition survey (DSM-IV-TR) (Supplementary Methods; available via Mendeley at http://doi.org/10.17632/k3vwsj4px9.1). We calculated probandwise concordance rates and tetrachoric correlations with 95% confidence intervals (CIs) to measure the degree of concordance for tanning addiction within twin pairs. Structural equation and liability threshold models (Additive genetic variance, Common/shared environmental factors, and individual environmental factors plus measurement Error [ACE model]) (Supplemental Fig 1; available via Mendeley at http://doi.org/10.17632/k3vwsj4px9.1) were created to estimate the liability of additive genetic effects (heritability) and the unique environmental effects of tanning addiction after controlling for age and sex.[1] Both heritability estimates and 95% CIs were determined by using the best-fit model.[2] All analyses were performed using R (R Core Team, Vienna, Austria). This study was approved by the institutional review board of University Hospitals Cleveland Medical Center.

Our sample included 147 DZ and 24 MZ twin pairs; 33.0% of the study population met criteria for tanning addition. Sociodemographic and dermatologic sample characteristics are provided in Table I, Supplemental Table I, and Supplemental

Table I. Sample demographics

Characteristics	Values
Mean age, y, (SD)	34.6 (17.68)
Sex, n (%)	
Male	70 (20.5)
Female	272 (79.5)
Hispanic, n (%)	
Yes	12 (3.5)
No	330 (96.5)
Race, n (%)	
White	303 (88.6)
Black	25 (7.3)
Asian	11 (3.2)
Other	3 (0.9)
Education level, n (%)	
<9th grade	26 (7.6)
Some high school	38 (11.1)
High school graduate	88 (25.7)
Associate's degree	41 (12.0)
Bachelor's degree	84 (24.6)
Master's degree	48 (14.0)
Doctorate degree	17 (5.0)
Employed, n (%)	
Yes	234 (68.4)
No	108 (31.6)
Currently live with twin, n (%)	
Yes	170 (49.7)
No	172 (50.3)
Fitzpatrick skin type, n (%)	
I	34 (9.9)
II	87 (25.4)
III	141 (41.2)
IV	61 (17.8)
V	9 (2.6)
VI	10 (2.9)
Complexion, n (%)	
Light	194 (56.7)
Medium	134 (39.2)
Dark	11 (3.2)
Response to 30 minutes unprotected in sun, n (%)	
Severe sunburn with blisters	14 (4.1)
Painful sunburn, no blisters	72 (21.1)
Mild sunburn and tan	187 (54.7)
Tanned, no sunburn	59 (17.3)
No change in skin color	10 (3.0)
History of blistering sunburns, n (%)	
Yes	139 (40.6)
No	203 (59.4)
Hours of sun exposure, mean (SD)	
Weekdays (SD)	3.4 (4.6)
Weekends (SD)	4.2 (3.4)
History of skin cancer, n (%)	
Yes	27 (7.9)
No	315 (92.1)
History of tanning as medical treatment, n (%)	
Yes	5 (1.5)
No	337 (98.5)

SD, Standard deviation.

Table II. Overall, 34 DZ and 4 MZ pairs were concordant for tanning addiction. Probandwise concordance for MZ pairs was 0.70 (95% CI, 0.60-0.80) and for DZ pairs was 0.50 (95% CI, 0.20-0.80) (Table II). Tetrachoric correlation for MZ pairs was 0.75 (95% CI, 0.56-0.86) and for DZ pairs was 0.38 (95% CI, 0.30-0.45). Controlling for age and sex (ACE model), the estimated heritability of tanning addiction was 75.4% (95% CI, 60.6-90.2), and the estimated contribution of unique environmental effects was 24.6% (95% CI, 9.8-39.4).

The prevalence of tanning addition in this twin cohort study (33.0%) is similar to estimates of the prevalence of tanning addition in the general nontwin population.[3] Moreover, the estimated heritability of tanning addiction in this twin cohort study (75.4%) indicates a substantial genetic component consistent with other addictive disorders, including alcoholism (70.6%), nicotine (65.0%), and illicit substances (68.5%).[4,5] Establishing a heritability estimate is the first step in determining variance in phenotypes. The total heritability estimate is used as a theoretical maximum of variance, which is important for genome-wide association studies to further categorize genetic contributions to tanning addiction and develop potential therapeutic targets.

Strengths of this study include use of a validated statistical methodology to provide a precise heritability estimate using the largest sample of twin pairs in the United States. Limitations include model assumptions, recall bias, and reliance on the efficacy of the DSM-IV-TR as a measure of tanning addiction. Determining the relative genetic and environmental components of tanning addition is critical to better understand the multifaceted pathogenesis of tanning addiction and develop targeted interventions to reduce its morbidity and mortality.

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Table II. Probandwise concordance rates and tetrachoric correlations for tanning addiction and heritability estimate for tanning addiction (controlling for age and sex)

Zygosity	Concordant pairs	Discordant pairs	Unaffected pairs	Probandwise CR (95% CI)	TCR (95% CI)
MZ	34	29	84	0.70 (0.60-0.80)	0.75 (0.56-0.86)
DZ	4	8	12	0.50 (0.20-0.80)	0.38 (0.30-0.45)

Heritability Estimate	Model	A, % (95% CI)	C, % (95% CI)	E, % (95% CI)	AIC
	ACE	75.4 (60.6-90.2)	0.0 (0.0-0.0)	24.6 (9.8-39.4)	401.16
	AE	**75.4 (60.6-90.2)**	—	**24.6 (9.8-39.4)**	**399.16**
	CE	—	70.7 (55.4-86.0)	29.3 (14.0-44.6)	401.43

A, Additive genetic effects (heritability); *AIC,* Akaike information criterion; *C,* common environmental effects; *CI,* confidence interval; *CR,* concordance rate; *DZ,* dizygotic; *E,* unique environmental effects; *MZ,* monozygotic; *TCR,* tetrachoric concordance rate.
Bold values indicate the model chosen for this study, based on lowest AIC.

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The risk of respiratory tract infections in patients with psoriasis treated with interleukin 23 pathway–inhibiting biologics: A meta-estimate of pivotal trials relevant to decision making during the COVID-19 pandemic

To the Editor: The COVID-19 pandemic caused by the severe acute respiratory syndrome virus (SARS-CoV-2) has led to uncertainty regarding the safety of immunosuppressive psoriasis therapy. We recently reported a meta-estimate that showed a 30% to 60% increase in the risk of respiratory tract infections (RTIs) in patients with psoriasis receiving interleukin (IL) 17 biologics compared to placebo.[1] Similar to IL-17, IL-23 is a key cytokine in the maintenance of T-helper 17 cells, which mediate protection against pathogens. Although not believed to be a central cytokine in the defense of viral infections, reduced levels of IL-23 may contribute to impairment of mucosal barrier immunity, resulting in an increased risk of respiratory infections.[2] Biologics that specifically target IL-23 have high efficacy for psoriasis and a favorable risk-benefit profile.[3] However, data on the use of IL-23 inhibitors and their impact on the incidence and outcomes of novel SARS-CoV-2 infection are limited. A study from