Table II. Comparison of *C acnes* phylotypes and clonal complexes in the 2 groups (adult women vs adolescents/teenagers)

Bacteriologic results	Adult women (n = 51)	Adolescents/ teenagers (n = 31)	*P* value*
Sampling area, n face/back	45/0	29/2	
C acnes identification, n (%)	47 (92)	23 (74)	
C acnes phylotype, n (%)			
IA1	32 (68)	19 (83)	.36
IB	5 (11)	1 (4)	
IC	1 (2)	0	
II	9 (19)	2 (8)	
III	0	1 (4)	
C acnes clonal complex, n (%)			
CC3	1 (2)	1 (4)	.43
CC18	25 (53)	15 (68)	
CC28	6 (13)	1 (4)	
CC36	6 (13)	3 (12)	
CC43	0	1 (4)	
CC53	9 (19)	2 (8)	

C acnes, *Cutibacterium acnes*.
**P* value less than .05 is considered significant.

In this letter, we have reported what to our knowledge is the first time that adult acne in women is not related to a specific subtype of *C acnes* because no higher frequency of phylotype or clonal complex or SLST was identified. Previous studies have shown no difference in *C acnes* density on the face between women with early-onset (before age 21) acne and women with late-onset acne[4] or in the densities of the 3 predominant microorganisms (*Cutibacterium spp*, *Staphylococcus spp*, and *Malassezia spp*)[4] and women without acne.[5] In confirmation of recently published results, we found that IA1 was the predominant phylotype associated with acne. Interestingly, the frequency of *C acnes* resistance was similar among the adult women and teenager groups. Limitations of this study include small sample size and the possibility that prior acne treatments may have altered the patients' microbiomes. Our results suggest that differences between acne in adult women and teenagers are more likely related to nonmicrobial factors such as hormonal skin changes, stimulation of innate immunity, or environmental factors.

APPENDIX

Previous studies focusing on adult acne in women and *Cutibacterium acnes* were identified by using the electronic database PubMed with the following terms: *adult woman*, *acne*, and *Propionibacterium acnes* or *Cutibacterium acnes*. Each abstract was verified to identify previous studies. On the basis of this literature search strategy, we can assume that our study is the first report showing no link between adult acne in women and a specific subtype of *C acnes*.

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Funding sources: None.

Conflicts of interest: None disclosed.

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https://doi.org/10.1016/j.jaad.2019.03.040

The proportion of nevus-associated invasive melanoma differs with Breslow thickness: A cross-sectional study of 1087 cutaneous melanomas

To the Editor: Although most melanomas appear de novo, a third of cases are nevus-associated melanomas (NAMs).[1] The proportion of NAMs appears to be slightly higher when the definition is based on information provided by the patient

Table I. Distribution of melanomas according to the absence of nevus-associated melanoma versus common versus dysplastic nevus remnants, and to tumor thickness

		Nevus associated with the melanoma						
		Absence		Common nevus		Dysplastic nevus		
Group	Total, n	n	%	n	%	n	%	*P**
Overall population	1087	796	73.2	144	13.2	147	13.5	
Groups defined by Breslow thickness, mm								
≤1	511	336	65.8	75	14.7	100	19.6	<.001
1.01-2.00	241	170	70.5	42	17.4	29	12.0	
2.01-4.00	188	160	85.1	16	8.5	12	6.4	
>4	147	130	88.4	11	7.5	6	4.1	

**P* value for the Pearson χ^2 test comparing the 3 groups: de novo, common nevus, and dysplastic nevus.

(42%-85%)[2,3] than when it is based on the histologic assessment (4%-72%).[1-4] One explanation for this discrepancy is the possibility of the tumor destroying the preexisting neval component, a phenomenon that is expected to be seen mostly in thick tumors and, perhaps, in nodular melanomas. The present study compared the proportion of NAMs in invasive melanomas based on tumor thickness.

A cross-sectional study was designed based on pathologic information collected in the melanoma database of the institution. Pathology was routinely assessed by a unique pathologist. Only patients with invasive superficial spreading (SSM) or nodular (NM) cutaneous melanomas, diagnosed from 2000 through 2017, were selected. In situ melanomas were excluded to reduce classification bias. Only NAMs with common melanocytic (other than congenital or nevus spilus, ie, flat, Unna, and Miescher nevi) or dysplastic nevus were considered. Three groups were defined (absence of NAM, common NAM, and dysplastic NAM). Breslow thickness was categorized based on the American Joint Committee on Cancer staging system (≤1 mm, 1.01-2.00 mm, 2.01-4.00 mm, and >4 mm). Age (categorized by the median: <55 years vs ≥55 years) and melanoma type-stratified (NM vs SSM) analyses were performed with contingency tables and Pearson χ^2 test. All *P* values were 2 tailed or bilateral, and 2-tailed significance was established at $P < .05$. All statistical analyses were performed with SPSS Statistics, version 21 (IBM, Armonk, NY) statistical program.

Of 1087 patients, 552 were male and 535 female. Overall, the proportion of NAMs decreased with greater tumor thickness, ranging from 34% in melanomas less than 1 mm to 12% in melanomas greater than 4 mm (Table I). SSMs had a higher proportion of NAMs than NMs (Table II). The proportion of NAMs was also increased in the youngest patient group (Table II).

These results suggest that, at least in part, the proportion of NAMs may be underestimated in thicker melanomas. The lower proportion in NMs compared with SSMs suggests that the neval component may have been destroyed by the melanoma or an inflammatory reaction. However, it cannot be ruled out that the differences may also be due to a less aggressive behavior of NAMs. Whether NAMs are less aggressive than de novo melanomas is still debatable. Nevertheless, most studies suggest that differences in prognosis depend on the association with other prognostic features, such as Breslow thickness or ulceration.[5] In a recent study, NAM showed better independent prognosis; however, the validity of this result is questionable because in situ melanomas were included.[1] In conclusion, we confirmed that the proportion of NAMs was greater in thin tumors, in those of SSM type, and in patients younger than 55 years. Because of the possibility that nevi may have been missed during histologic preparation, a prospective study in which a dermatologist assisted with the orientation of the piece would be interesting.

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Table II. Distribution of melanomas according to tumor thickness and to the absence of nevus-associated melanoma versus common versus dysplastic nevus remnants, stratified by age and histologic type

			Absence		Common nevus		Dysplastic nevus		
Characteristic	Breslow thickness, mm	Total	n	%	n	%	n	%	*P**
Age ≤ 55 y		550	368	66.9	96	17.5	86	15.6	
	≤1	302	187	61.9	52	17.2	63	20.9	<.001
	1.01-2.00	137	88	64.2	31	22.6	18	13.1	
	2.01-4.00	71	59	83.1	9	12.7	3	4.2	
	>4	40	34	85.0	4	10.0	2	5.0	
Age > 55 y		537	428	79.7	48	8.9	61	11.4	
	≤1	209	149	71.3	23	11.0	37	17.7	.002
	1.01-2.00	104	82	78.8	11	10.6	11	10.6	
	2.01-4.00	117	101	86.3	7	6.0	9	7.7	
	>4	107	96	89.7	7	6.5	4	3.7	
SSM		813	561	69.0	124	15.3	128	15.7	
	≤1	497	326	65.6	73	14.7	98	19.7	.004
	1.01-2.00	194	137	70.6	34	17.5	23	11.9	
	2.01-4.00	93	74	79.6	14	15.1	5	5.4	
	>4	29	24	82.8	3	10.3	2	6.9	
NM		274	235	85.8	20	7.3	19	6.9	
	≤1	14	10	71.4	2	14.3	2	14.3	.006
	1.01-2.00	47	33	70.2	8	17.0	6	12.8	
	2.01-4.00	95	86	90.5	2	2.1	7	7.4	
	>4	118	106	89.8	8	6.8	4	3.4	

NM, Nodular melanoma; *SSM*, superficial spreading melanoma.
**P* value for the Pearson χ^2 test comparing the 3 groups: de novo, common nevus, and dysplastic nevus.

Drs Reyes, Manrique-Silva, and Folgado contributed equally to this work.

Supported in part by a Universidad Católica de Valencia "San Vicente Mártir" grant (no. 2018-109-001).

Conflicts of interest: None disclosed.

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https://doi.org/10.1016/j.jaad.2019.04.043

Retrospective outcome analysis of 25 alopecia areata patients treated with simvastatin/ezetimibe

To the Editor: Optimism regarding alopecia areata (AA) treatment efficacy with simvastatin/ezetimibe (Vytorin, Merck & Co, Whitehouse Station, NJ) from reports and small series inspired prospective trials with mixed results.[1-4] Its therapeutic mechanism remains unknown, but may involve cytokine reduction, Janus kinase (JAK)/signal transducer and activator of transcription (STAT) pathway inhibition, and increasing regulatory T cells.[5] We examined our experience treating AA with this agent in the context of methotrexate use or failure, or both.

After approval by the University of Pittsburgh Institutional Review Board, a retrospective clinical and treatment outcome analysis of patients with AA receiving once-daily simvastatin 40 mg/ezetimibe 10 mg (S/E) monotherapy de novo, after methotrexate failure, or combined with methotrexate after inadequate response to intralesional steroid (ILS) was performed. Clinical response was classified semi-quantitatively by estimated percentage regrowth: no response (0%), minimal (1%-25%), moderate (26%-75%), or complete (76%-100%).

There were 25 patients (92% white, 68% female; median age, 47 years; range, 13-80 years) who presented with patchy scalp (24%), ophiasis (16%),