a personally relevant treatment goal alongside clinical data did not have as great of an effect as presenting a treatment goal alone. We cannot, however, conclude that we should not give patients clinical data. The potential benefit of improving adherence must be balanced with a patient's right to be informed of all pertinent information.

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Lack of genotype-phenotype correlation in basal cell nevus syndrome: A Dutch multicenter retrospective cohort study

To the Editor: Basal cell nevus syndrome (BCNS), is a rare autosomal dominant disorder (Mendelian Inheritance in Man no. 109400). The most common underlying cause is a heterozygous germline mutation in the Patched-1 (*PTCH1*) gene (chromosome 9q22-31).[1] The mutation detection rate of *PTCH1* in BCNS in clinically confirmed individuals is 40% to 85%.[2] Diagnosis requires 2 major criteria, 1 major criterion and 2 minor criteria, or 1 major criterion and molecular confirmation.[3] To date, no clear genotype-phenotype correlation has been detected. In this study, 83 Dutch patients with a confirmed *PTCH1* mutation were analyzed.

A multicenter retrospective cohort study was conducted at the Maastricht University Medical Center and the VU University Medical Center in the Netherlands. From 1999 until 2015, *PTCH1* variant analysis was performed in 673 patients. In 141 (21.0%) individuals, a *PTCH1* variant was detected.[4] After applications from hospitals outside the Netherlands and nonpathogenic variants were excluded, a pathogenic *PTCH1* mutation was detected in 95 patients. Clinical data could be obtained for 83 patients (37 male, 46 female) from 77 families (mean age, 25.2 years).

Two heat maps were created to investigate a genotype-phenotype correlation. To create robust heat maps, only patients with 2 or more symptoms were included. In the first heat map, the mutations were sorted according to their location within the gene, plotted against 6 clinical symptoms (Fig 1). In the second heat map, the mutations were sorted according to the type of mutation (truncated or missense) against the clinical symptoms (Fig 2). Other symptoms were excluded because specific data were missing for many cases. No specific clustering of symptoms in relation to mutations in a certain exon or part of the *PTCH1* gene could be observed, nor could phenotype similarities be found within families in our population. In accordance with Evans et al,[5] we found that patients with a missense variant in *PTCH1* were diagnosed later with BCNS, were less likely to develop rib anomalies, and had their first basal cell carcinoma at a 3-year older age

Exon	Domain	Change in DNA	Change in protein	Type mutation	BCCs	OKCs	PPP	CFC	SA	MC
1i		c.202-2A>G		splicing						
2	**N-terminal**	c.205A>T	p.(Lys69*)	nonsense						
2	**N-terminal**	c.253_254delAG	p.(Arg85Thrfs*4)	frameshift						
2	**N-terminal**	c.279C>G	p.(Tyr93*)	nonsense						
2	**N-terminal**	c.294C>A	p.(Cys98*)	nonsense						
2	**ECL**	c.385_386dupTG	p.(Trp129Cysfs*9)	frameshift						
3	**ECL**	c.403C>T	p.(Arg135*)	nonsense						
3	**ECL**	c.403C>T	p.(Arg135*)	nonsense						
3	**ECL**	c.403C>T	p.(Arg135*)	nonsense						
3	**ECL**	c.466C>T	p.(Gln156*)	nonsense						
3	**ECL**	c.466C>T	p.(Gln156*)	nonsense						
3	**ECL**	c.479_482delAGAC	p.(Gln160Profs*10)	frameshift						
4i		c.566_584+8del	p.?	splicing						
4	**ECL**	c.591G>C	p.(Trp197Cys)	missense						
4i		c.655-1G>A	p.?	splicing						
5	**ECL**	c.707G>A	p.(Trp236*)	nonsense						
5i		c.747-2A>G	p.?	splicing						
6	**ECL**	c.770_771delCAinsGGTTTGG	p.(Thr257Argfs*14)	frameshift						
6	**ECL**	c.842delT	p.(Met281fs*2)	frameshift						
7	**ECL**	c.1040_1049del	p.(Val347Alafs*17)	frameshift						
8	**ECL**	c.1081C>T	p.(Gln361*)	nonsense						
8	**ECL**	c.1093C>T	p.(Gln365*)	nonsense						
8 and 14	**ECL**	c.[1112delA;2183C>T]	p.[(Met372Cysfs*60);p.(Thr728Met)]	frameshift						
8i		c.1216-2A>G	p.?	splicing						
8i		c.1216-2A>G	p.?							
9	**ECL**	c.1279delC	p.(Leu427Trpfs*5)	frameshift						
9i		c.1347+1G>A	p.?	splicing						
9i		c.1347+1G>A	p.?	splicing						
9i		c.1348-2A>G	p.?	splicing						
10	**SSD**	c.1348_1350delCTC	p.(Leu450del)	inframe deletion						
10	**SSD**	c.1415_1429del15	p.(Ala472_Leu476del)	inframe deletion						
10	**SSD**	c.1450G>A	p.(Gli484Arg)	missense						
11	**SSD**	c.1555G>C	p.(Ala519Pro)	missense						
11	**SSD**	c.1526G>A	p.(Gly509Asp)	missense						
11	**SSD**	c.1526G>A	p.(Gly509Asp)	missense						
11	**SSD**	c.1526G>A	p.(Gly509Asp)	missense						
11	**SSD**	c.1526G>A	p.(Gly509Asp)	missense						
12	**SSD**	c.1691T>G	p.(Leu564*)	nonsense						
12	**SSD**	c.1691T>G	p.(Leu564*)	nonsense						
13	**SSD**	c.1767_1769delGCT	p.(Leu590del)	inframe deletion						
13	**ICL**	c.1804C>T	p.(Arg602*)	nonsense						
13	**ICL**	c.1810G>T	p.(Glu604*)	nonsense						
14	**ICL**	c.1852delT	p.(Cys618fs)	frameshift						
14	**ICL**	c.2098C>T	p.(Gln700*)	nonsense						
14	**ICL**	c.2170G>T	p.(Glu724*)	nonsense						
14	**ICL**	c.2178_2179insA	p.(Cys727Metfs*11)	frameshift						
14	**ICL**	c.2179delT	p.(Cys727fs)	frameshift						
14i_16i		c.2250+1G>T	p.?	splicing						
14	**TRANS**	c.2250G>C	p.(Lys750Asn)	missense						
14i_16i		c.2251-2A>G	p.?	splicing						
14i_16i	**TRANS**	c.(2250+1_2251-1)_(2703+1_2704-1)del	p.(Val751_Gln901del)	inframe deletion						
15	**ECL**	c.2308C>T	p.(Arg770*)	nonsense						
15	**ECL**	c.2359G>T	p.(Glu787*)	nonsense						
15	**ECL**	c.2414T>G	p.(Ile805Arg)	missense						
15	**ECL**	c.2446C>T	p.(Gln816*)	nonsense						
15	**ECL**	c.2447A>G	p.(Gln816Arg)	missense						
15	**ECL**	c.2557C>T	p.(Gln853*)	nonsense						
16-18	**ECL**	c.(2560+1_2561-1)_(3168+1_3169-1)del	p.(Leu855Aspfs*87)	frameshift						
16-18	**ECL**	c.(2560+1_2561-1)_(3168+1_3169-1)del	p.(Leu855Aspfs*87)	frameshift						
16-18	**ECL**	c.(2560+1_2561-1)_(3168+1_3169-1)del	p.(Leu855Aspfs*87)	frameshift						
16-18	**ECL**	c.(2560+1_2561-1)_(3168+1_3169-1)del	p.(Leu855Aspfs*87)	frameshift						
16	**ECL**	c.2619C>A	p.(Tyr873*)	nonsense						
16	**ECL**	c.2619C>A	p.(Tyr873*)	nonsense						
17	**ECL**	c.2748delC	p.(Ser917Alafs*7)	frameshift						
17	**ECL**	c.2824delC	p.(Arg942Glyfs*20)	frameshift						
18	**ECL**	c.3027C>A	p.(Tyr1009*)	nonsense						
18	**ECL**	c.3056_3059delAGCA	p.(Glu1019Glyfs*29)	frameshift						
18	**ECL**	c.3058C>T	p.(Gln1020*)	nonsense						
18	**TRANS**	c.3107_3108del	p.(Leu1036Cysfs*108)	frameshift						
18	**TRANS**	c.3107-3108del	p.(Leu1036Cysfs*108)	frameshift						
18 and 23	**TRANS**	c.[3135delC;4048C>T]	p.[(Phe1046Serfs*3;(Arg1350Trp)]	frameshift						
18 and 23	**TRANS**	c.[3135delC;4048C>T]	p.[(Phe1046Serfs*3;(Arg1350Trp)]	frameshift						
18	**TRANS**	c.3150delC	p.(Trp1051fs*7)	frameshift						
20	**TRANS**	c.3364_3365delAT	p.(Met1122Valfs*22)	frameshift						
1-23		c.(?_-188)_(*3411_?)del	p.0	deletion whole gene						
1-23		c.(?_-188)_(*3411_?)del	p.0	deletion whole gene						
1-23		c.(?_-188)_(*3411_?)del	p.0	deletion whole gene						
exon3-15		c.394+1_395-1)_(2560+1_2561-1)(2)	p.?							

Fig 1. Heat map. The genotype versus clinical symptoms (basal cell carcinomas [BCCs], odontogenic keratocysts [OKCs], palmoplantar pits [PPP], calcification of the falx cerebri [CFC], skeletal abnormalities [SA]; and macrocephaly [MC]) in 78 individuals. Clinical symptoms are shown in red (present), green (absent), and black (unknown). Domains are noted as N-terminal region. *ECL*, Extracellular loop; *ICL*, intracellular loop; *SSD*, sterol-sensing domain; and *TRANS*, transmembrane domain.

compared with patients with other mutations. However, this finding cannot be supported by reliable statistical analysis because of the small number of patients in the subgroups.

A possible cause for the absence of a genotype-phenotype correlation is the fact that specific clinical information was missing because of the retrospective nature of this study. Furthermore, the discriminatory power of the heat maps was limited to only 4 clinical features, which could have skewed the results to be underestimated.

The mutation detection rate in our population was low (21%), probably because genetic tests were easily accessible and patients referred for genetic testing did not necessarily meet formal clinical diagnostic criteria. Moreover, standard genetic tests do not detect genomic rearrangments or deep intronic variants, and Sanger sequencing does not detect mutation loads under 5% (eg, in low-grade

Exon	Domain	Change in DNA	Change in protein	Type mutation	BCCs	OKCs	PPP	CFC	SA	MC
2	**N-terminal**	c.205A>T	p.(Lys69*)	nonsense						
2	**N-terminal**	c.254_255del	p.(Arg85Thrfs*4)	frameshift						
2	**N-terminal**	c.279C>G	p.(Tyr93*)	nonsense						
2	**N-terminal**	c.294C>A	p.(Cys98*)	nonsense						
2	**ECL**	c.385_386dup	p.(Trp129Cysfs*9)	frameshift						
3	**ECL**	c.403C>T	p.(Arg135*)	nonsense						
3	**ECL**	c.403C>T	p.(Arg135*)	nonsense						
3	**ECL**	c.403C>T	p.(Arg135*)	nonsense						
3	**ECL**	c.466C>T	p.(Gln156*)	nonsense						
3	**ECL**	c.466C>T	p.(Gln156*)	nonsense						
3	**ECL**	c.479_482del	p.(Gln160Profs*10)	frameshift						
5	**ECL**	c.707G>A	p.(Trp236*)	nonsense						
6	**ECL**	c.770_771delinsGGTTTGG	p.(Thr257Argfs*14)	frameshift						
6	**ECL**	c.842del	p.(Met281fs*2)	frameshift						
7	**ECL**	c.1040_1049del	p.(Val347Alafs*17)	frameshift						
8	**ECL**	c.1081C>T	p.(Gln361*)	nonsense						
8	**ECL**	c.1093C>T	p.(Gln365*)	nonsense						
8 and 14	**ECL**	c.[1112del;2183C>T]	p.[(Met372Cysfs*60);p.(Thr728Met)]	frameshift						
9	**ECL**	c.1279del	p.(Leu427Trpfs*5)	frameshift						
12	**SSD**	c.1691T>G	p.(Leu564*)	nonsense						
12	**SSD**	c.1691T>G	p.(Leu564*)	nonsense						
13	**ICL**	c.1804C>T	p.(Arg602*)	nonsense						
13	**ICL**	c.1810G>T	p.(Glu604*)	nonsense						
14	**ICL**	c.1852del	p.(Cys618fs)	frameshift						
14	**ICL**	c.2098C>T	p.(Gln700*)	nonsense						
14	**ICL**	c.2170G>T	p.(Glu724*)	nonsense						
14	**ICL**	c.2178_2179insA	p.(Cys727Metfs*11)	frameshift						
14	**ICL**	c.2179del	p.(Cys727fs)	frameshift						
15	**ECL**	c.2308C>T	p.(Arg770*)	nonsense						
15	**ECL**	c.2359G>T	p.(Glu787*)	nonsense						
15	**ECL**	c.2446C>T	p.(Gln816*)	nonsense						
15	**ECL**	c.2557C>T	p.(Gln853*)	nonsense						
17-19	**ECL**	c.(2703+1_2704-1)_(3306+1_3307-1)del	p.?	?						
17-19	**ECL**	c.(2703+1_2704-1)_(3306+1_3307-1)del	p.?	?						
17-19	**ECL**	c.(2703+1_2704-1)_(3306+1_3307-1)del	p.?	?						
17-19	**ECL**	c.(2703+1_2704-1)_(3306+1_3307-1)del	p.?	?						
16	**ECL**	c.2619C>A	p.(Tyr873*)	nonsense						
16	**ECL**	c.2619C>A	p.(Tyr873*)	nonsense						
17	**ECL**	c.2748del	p.(Ser917Alafs*7)	frameshift						
17	**ECL**	c.2824del	p.(Arg942Glyfs*20)	frameshift						
18	**ECL**	c.3027C>A	p.(Tyr1009*)	nonsense						
18	**ECL**	c.3056_3059del	p.(Glu1019Glyfs*29)	frameshift						
18	**ECL**	c.3058C>T	p.(Gln1020*)	nonsense						
18	**TRANS**	c.3107_3108	p.(Leu1036Cysfs*108)	frameshift						
18	**TRANS**	c.3107-3108	p.(Leu1036Cysfs*108)	frameshift						
18 and 23	**TRANS**	c.[3135del;4048C>T]	p.[(Phe1046Serfs*3;(Arg1350Trp)]	frameshift						
18 and 23	**TRANS**	c.[3135del;4048C>T]	p.[(Phe1046Serfs*3;(Arg1350Trp)]	frameshift						
18	**TRANS**	c.3150del	p.(Trp1051fs*7)	frameshift						
20	**TRANS**	c.3364_3365del	p.(Met1122Valfs*22)	frameshift						
4	**ECL**	c.591G>C	p.(Trp197Cys)	missense						
10	**SSD**	c.1450G>A	p.(Gli484Arg)	missense						
11	**SSD**	c.1526G>A	p.(Gly509Asp)	missense						
11	**SSD**	c.1526G>A	p.(Gly509Asp)	missense						
11	**SSD**	c.1526G>A	p.(Gly509Asp)	missense						
11	**SSD**	c.1526G>A	p.(Gly509Asp)	missense						
11	**SSD**	c.1555G>C	p.(Ala519Pro)	missense						
14	**TRANS**	c.2250G>C	p.(Lys750Asn)	missense						
15	**ECL**	c.2414T>G	p.(Ile805Arg)	missense						
15	**ECL**	c.2447A>G	p.(Gln816Arg)	missense						

Fig 2. Heat map. Type of mutation (truncated and missense) versus clinical symptoms (basal cell carcinomas [BCCs]; odontogenic keratocysts [OKCs]; palmoplantar pits [PPP]; calcification of the falx cerebri [CFC], skeletal abnormalities [SA]; and macrocephaly [MC]) in 59 individuals. Clinical symptoms are shown in red (present), green (absent), and black (unknown). Domains are noted as N-terminal region. *ECL*, Extracellular loop; *ICL*, intracellular loop; *SSD*, sterol-sensing domain; and *TRANS*, transmembrane domain.

postzygotic mosaicism). Another explanation could be the occurrence of mutations in other components of the Hedgehog pathway (eg, *SUFU*, *PTCH2*).

In conclusion, our findings support a lack of a genotype-phenotype correlation in BCNS based on a *PTCH1* variant alone and indicate that other factors are likely to contribute.

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Skin cancer and dermatoses in a majority-Hispanic population of solid organ transplant recipients

To the Editor: The number of solid organ transplant recipients (SOTRs) of racial or ethnic minorities is increasing in the United States, with more than 16,000 new nonwhite SOTRs in 2018.[1] Our knowledge that SOTRs have increased incidence of skin cancer and infections primarily derives from non-Hispanic white (NHW) patients, but there is little information published on Hispanic cohorts.[2,3] We sought to address this gap by describing the rates and characteristics of skin cancer and dermatoses in a Hispanic-majority SOTR population. We conducted an institutional review board–approved, single-center retrospective review of 2032 SOTRs (kidney, liver, heart, lung) between 1993 and 2016, including patients with outpatient/inpatient dermatology encounters at our institution, community dermatology visits per outside records, and biopsy-proven skin cancers (including by nondermatologists). Patients were stratified by race/ethnicity as NHW, Hispanic, Asian, or black. Hispanic origin was defined according to US Census Bureau guidelines.[4]

Among all SOTRs, 1058/2032 (52.1%) were Hispanic, and 555/2032 (27.3%) were NHW. Skin cancers or dermatoses were diagnosed in 314 of 2032 (15.5%) patients, of whom 161 (51.3%) were Hispanic (Table I). Among those with dermatologic complications, 41 of 103 (39.8%) NHW and 27 of 161 (16.8%) Hispanic patients had skin cancer. In these two groups, the most common type of skin cancer was squamous cell carcinoma (or in situ), representing 77 of 148 total skin cancers (52.0%). NHW patients had more than twice as many skin cancers as Hispanic patients (106 vs 42, respectively) and more often had multiple skin cancers (21/41 patients (51.2%) vs 7/27 patients (25.9%), respectively). Overall, most skin cancers were on the head/neck (81/148, 54.7%) and upper extremities (32/148, 21.6%) (Fig 1). In Hispanic patients, a greater proportion of skin cancers occurred on the head/neck than in NHW patients (25/37, 67.6% vs 49/97, 50.5%, respectively). There were no between-group differences in time from transplantation to first outpatient visit (median, 24.0 months; $P = .28$) or first skin cancer (median, 3 years; $P = .57$). Rates of inflammatory and infectious dermatoses were similar, except that fungal infections were more frequent in Hispanic patients 45/161 (28.0%) than other groups (NHW, 7/103, 6.8%; Asian, 6/42, 14.3%; black, 1/8, 12.5%; $P < .001$).

In this retrospective study, Hispanics were the majority of all SOTRs and of those with posttransplantation dermatologic complications. Skin cancer was diagnosed in 27/161 (16.8%) of Hispanic versus 41/103 (39.8%) of NHW patients. Superficial mycoses were significantly more common in Hispanic patients than other groups. In a Philadelphia study of majority-nonwhite SOTRs, skin cancer was found in 42% of NHW and 12% of Hispanic SOTRs,[2] comparable to our findings (although only 8% of patients were Hispanic); however, in that study, black and Asian patients had more infections than