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Dependence of Deodorant Usage on *ABCC11* Genotype: Scope for Personalized Genetics in Personal Hygiene

Santiago Rodriguez[1], Colin D. Steer[2], Alexandra Farrow[3], Jean Golding[2] and Ian N.M. Day[1]

Earwax type and axillary odor are genetically determined by rs17822931, a single-nucleotide polymorphism (SNP) located in the *ABCC11* gene. The literature has been concerned with the Mendelian trait of earwax, although axillary odor is also Mendelian. Ethnic diversity in rs17822931 exists, with higher frequency of allele A in east Asians. Influence on deodorant usage has not been investigated. In this work, we present a detailed analysis of the rs17822931 effect on deodorant usage in a large ($N \sim 17{,}000$ individuals) population cohort (the Avon Longitudinal Study of Parents and Children (ALSPAC)). We found strong evidence ($P = 3.7 \times 10^{-20}$) indicating differential deodorant usage according to the rs17822931 genotype. AA homozygotes were almost 5-fold overrepresented in categories of never using deodorant or using it infrequently. However, 77.8% of white European genotypically nonodorous individuals still used deodorant, and 4.7% genotypically odorous individuals did not. We provide evidence of a behavioral effect associated with rs17822931. This effect has a biological basis that can result in a change in the family's environment if an aerosol deodorant is used. It also indicates potential cost saving to the nonodorous and scope for personalized genetics usage in personal hygiene choices, with consequent reduction of inappropriate chemical exposures for some.

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INTRODUCTION

The *ABCC11* gene encodes the multidrug resistance protein 8 (MRP8) (Kruh *et al.*, 2007). Although historically identified as a drug resistance factor, this protein is involved in transport of many small molecules in normal physiology, in a variety of cellular and biological contexts. There is strong evidence suggesting that genetic variation at *ABCC11* has pleiotropic effects. In particular, a functional nonsynonymous single-nucleotide polymorphism (SNP; rs17822931), also known as 538 G→A or G180R, determines human earwax type (Yoshiura *et al.*, 2006) and axillary osmidrosis (Nakano *et al.*, 2009; Martin *et al.*, 2010) and is associated with apocrine colostrum secretion from the mammary gland (Miura *et al.*, 2007). It has also been related to breast cancer risk, although this is more controversial (see Toyoda and Ishikawa, 2010, and references therein).

The rs17822931 genotype AA determines dry earwax type, whereas the presence of at least one G allele (GA or GG) determines wet earwax type in a dominant manner. There are marked differences in rs17822931 allele frequencies across ethnic groups (Yoshiura *et al.*, 2006; Toyoda *et al.*, 2009; Toyoda and Ishikawa, 2010). There is a higher frequency of the A allele in east Asians, and therefore higher prevalence of the dry earwax type. In contrast, the wet earwax type is more prevalent in European and African populations because of higher frequencies of the G allele.

There is a close histological and functional relationship between ceruminous and apocrine sweat glands. This relationship is believed to explain the connection between earwax and axillary odor (Martin *et al.*, 2010). AA homozygous individuals for rs17822931 display a few of the characteristic axillary odorants (Martin *et al.*, 2010; Preti and Leyden, 2010). In contrast, a study of Japanese individuals showed that essentially all individuals with axillary osmidrosis were GG or AG for rs17822931 (Nakano *et al.*, 2009). This indicates that the G allele is necessary and sufficient to cause axillary odor, whereas the AA genotype effectively marks nonodorous individuals.

There is strong evidence supporting the fact that the production of odor associated with rs17822931 follows a dimorphic pattern. It is necessary to have a functional allele (rs17822931 G) in order to secrete amino acid conjugates of

[1]*MRC Centre for Causal Analyses in Translational Epidemiology (CAiTE), and Bristol Genetic Epidemiology Laboratories (BGEL), School of Social and Community Medicine, University of Bristol, Bristol, UK;* [2]*Centre for Child and Adolescent Health, School of Social and Community Medicine, University of Bristol, Bristol, UK and* [3]*Health, Sciences and Social Care, Brunel University, London, UK*

Correspondence: Santiago Rodriguez, MRC Centre for Causal Analyses in Translational Epidemiology (CAiTE), and Bristol Genetic Epidemiology Laboratories (BGEL), School of Social and Community Medicine, University of Bristol, Oakfield House, Oakfield Grove, Bristol BS8 2BN, UK.
E-mail: santi.rodriguez@bristol.ac.uk

Abbreviations: ALSPAC, Avon Longitudinal Study of Parents and Children; SNP, single-nucleotide polymorphism

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human-specific odorants that lead to the production of axillary odor (Martin *et al.*, 2010).

Several studies have addressed various biological aspects of *ABCC11* rs17822931, including the possible mechanism of action and pharmacogenomic aspects (Toyoda *et al.*, 2009; Toyoda and Ishikawa, 2010). However, behavioral aspects related to this genetic polymorphism have been studied to a small extent. In this work, we analyzed the association between *ABCC11* rs17822931 and deodorant usage from a large sample of unrelated children and their parents from the Avon Longitudinal Study of Parents and Children (ALSPAC) (Boyd *et al.*, 2012; Fraser *et al.*, 2012).

RESULTS

No significant deviation from Hardy–Weinberg proportions was found for rs17822931 for white mothers ($\chi^2 = 2.3$, $P = 0.129$) or for white children ($\chi^2 = 0.55$, $P = 0.458$). The analysis of ascertainment bias in rs17822931 (Supplementary Figure S1 online) showed a slight but nonsignificant ($P = 0.129$) excess of rs17822931 AA homozygotes (115 expected vs. 134 observed), again indicating that genotyping error or unrecognized subdivision through admixture or stratification was not an issue in our analyses. Minor allele frequencies were 12.5% for white mothers and 24.9% for non-white mothers. Similar results were observed for the child genotype: 12.7% (white parentage), 15.1% (mixed parentage), and 20.7% (non-white parentage). Average usage of deodorants was 6.1 times per week for mothers (SD = 1.90) and 5.2 times per week for partners (SD = 2.53). There was lower usage among non-white parents by 10–16% ($P < 0.001$).

We describe below a summary of the observed results for nonexperts in statistics. A more detailed description of the results, including specific statistical evidence supporting our findings, can be seen in Supplementary Appendix S1.

Regression analyses

We observed a strong association between deodorant usage and maternal rs17822931 genotype in ALSPAC for both white and non-white participants. The association was highly significant for white mothers ($P = 1.8 \times 10^{-14}$) and for non-white mothers ($P = 1.5 \times 10^{-6}$; Table 1).

However, there was no association between maternal genotype and partners' deodorant usage in whites ($P = 0.9040$). The rs17822931 genotype in children associated with similar effect sizes for deodorant usage by the mother and by the partner. When considering white individuals, the rs17822931 genotype of the child associated significantly with maternal deodorant usage ($P = 0.0004$) and with the use of deodorant by the partner ($P = 0.0235$).

Five other putative confounders were considered in linear regression analysis of maternal deodorant use on maternal genotype (Table 2). Significant associations were observed for four of them (maternal age, maternal education, housing tenure, and hygiene), with no significant effect for paternal social class. The relative effect of the rs17822931 genotype was larger than the effect of each of these variables. The greater effect ranged from 3 times higher than hygiene to 34 times higher than maternal education, as measured by the β-coefficients observed in linear regression analyses (Table 2).

Results from contingency tables

Results from contingency tables (Table 3) allow the identification of more specific effects for each of the categories of deodorant usage in relation to rs17822931. The results for deodorant usage in four combinations of individuals were as follows.

Maternal genotype association with maternal deodorant usage. Table 3a shows that there is a significantly higher than expected frequency of the AA genotype in white mothers with a lower use of deodorant. There is a nearly a 5-fold overrepresentation of AA individuals in the deodorant never use group, with significant overrepresentations in all other categories except in the category of daily deodorant usage. In this category, the observed frequency of AA homozygotes is significantly lower than that expected. Overall, there is a significant ($P = 3.7 \times 10^{-20}$) difference between the observed and the expected numbers in the contingency table for deodorant usage and the rs17822931 genotype. These results are for the additive model. They are very similar to the results observed under the recessive model (Supplementary Table S1 online).

Figure 1 shows the relationship between rs17822931 genotypes and both an odoriferous steroid (data from Martin *et al.*, 2010) and deodorant usage. AA homozygotes (with the lowest production of the odoriferous steroid) showed the highest ratio of never use over daily deodorant use. The converse was found for GG homozygotes. AG heterozygotes showed differences in relation to GG homozygotes, with both lower production of the odoriferous steroid and significantly ($P = 0.002$) higher ratio of never use over daily deodorant use than GG homozygotes.

Child genotype association with maternal deodorant usage. The results from the contingency table analyzing white children's rs17822931 genotype by maternal deodorant usage (Table 3b and Supplementary Table S2 online) are different from the results observed in white mothers. There is an increased proportion of children with AA and GA genotypes in the category of never use of deodorant in white mothers. However, this increase is not as important as the one observed in deodorant usage of white mothers according to their own rs17822931 genotype. Overall, there is a significant ($P = 0.010$) difference between the observed and expected frequencies under the additive model (Table 3b), although these differences are not significant ($P = 0.173$) under the recessive model (Supplementary Table S2 online).

Child genotype association with partner's deodorant usage. Table 3c and Supplementary Table S3 online show partner's deodorant usage according to children's rs17822931 genotype. No significant differences were observed in the contingency tests either under the additive model ($P = 0.170$) or under the recessive model ($P = 0.263$).

Maternal genotype association with partner's deodorant usage. Similar results were observed for the analyses of partner's deodorant usage stratified by maternal rs17822931 genotype (Supplementary Tables S4 and S5 online).

Table 1. Associations between the use of deodorant by the mother at 8m and by the partner during pregnancy and *ABCC11* (rs17822931)

(a) Additive and recessive models

			Additive (copies of A allele)					Recessive (AA genotype)				
	Ethnicity of parent	*N*	B	95% Confidence interval (CI)		*P*	Dev *P*	B	95% CI		*P*	Dev *P*
Maternal genotype												
Use by mother 8m	Non-white	136	−1.52	−2.12	−0.92	1.5×10^{-6}	0.8746	−2.64	−4.02	−1.26	0.0002	0.0021
	White	6,492	−0.37	−0.47	−0.28	1.8×10^{-14}	5.7×10^{-11}	−1.64	−1.98	−1.31	2.2×10^{-21}	0.0007
Use by partner 12w	Non-white	115	−1.18	−1.99	−0.37	0.0046	0.1505	−1.09	−3.20	1.01	0.3051	0.0027
	White	4,952	0.01	−0.14	0.15	0.9040	0.1213	−0.33	−0.84	0.18	0.2026	0.3734
Child genotype												
Use by mother 8m	Non-white	159	−1.06	−1.68	−0.43	0.0011	0.2683	−2.39	−3.89	−0.89	0.0019	0.1356
	White	7,281	−0.16	−0.25	−0.07	0.0004	0.8597	−0.32	−0.65	0.02	0.0640	0.0027
Use by partner 12w	Non-white	119	−1.43	−2.20	−0.67	0.0003	0.5595	−2.85	−4.69	−1.02	0.0026	0.0389
	White	5,563	−0.16	−0.30	−0.02	0.0235	0.6224	−0.41	−0.93	0.12	0.1306	0.0791

(b) Genotype model (effect for GG genotype constrained to be zero)

			GA genotype				AA genotype				Group
	Ethnicity of parent	*N*	B	95% CI		*P*	B	95% CI		*P*	*P*
Maternal genotype											
Use by mother 8m	Non-white	136	−1.47	−2.39	−0.54	0.0021	−3.10	−4.46	−1.73	1.6×10^{-5}	1.0×10^{-5}
	White	6,492	−0.19	−0.30	−0.08	0.0007	−1.69	−2.03	−1.35	2.6×10^{-22}	8.5×10^{-23}
Use by partner 12w	Non-white	115	−1.79	−2.94	−0.63	0.0027	−1.45	−3.50	0.59	0.1610	0.0065
	White	4,952	0.08	−0.09	0.25	0.3734	−0.31	−0.82	0.20	0.2288	0.2988
Child genotype											
Use by mother 8m	Non-white	159	−0.69	−1.59	0.22	0.1356	−2.59	−4.10	−1.07	0.0009	0.0027
	White	7,281	−0.16	−0.26	−0.06	0.0027	−0.35	−0.69	−0.02	0.0397	0.0020
Use by partner 12w	Non-white	119	−1.19	−2.32	−0.06	0.0389	−3.17	−5.00	−1.34	0.0009	0.0013
	White	5,563	−0.14	−0.30	0.02	0.0791	−0.44	−0.97	0.09	0.1032	0.0682

Abbreviations: m, months; w, weeks.
Unadjusted linear regressions of deodorant usage on genotype. Effect sizes are the estimated change in frequency of deodorant use per week.
Dev *P*-value is a test of the deviation from the additive or recessive genetic model by comparison with the genotype model. The group test was a 2 d.f. test.

Table 2. Linear regression analysis of maternal deodorant use on maternal genotype (recessive model) adjusted for five confounders ($N = 4,903$)

		B	95% Confidence interval		*P*-value	Group *P*-value
Maternal	25–29	−0.06	−0.22	0.10	0.475	
age	30+	−0.41	−0.57	−0.24	1.7×10^{-6}	2.6×10^{-10}
Maternal	Medium	0.05	−0.09	0.19	0.472	
education	High	−0.23	−0.37	−0.09	0.002	2.3×10^{-5}
Social	III	0.02	−0.09	0.14	0.679	
class	IV+V	−0.03	−0.21	0.14	0.705	0.776
Housing	Council	−0.26	−0.46	−0.05	0.013	
tenure	Other	−0.36	−0.55	−0.17	2.2×10^{-4}	1.5×10^{-4}
Hygiene	Medium	0.40	0.28	0.53	2.3×10^{-10}	
	High	0.57	0.44	0.70	2.1×10^{-18}	4.1×10^{-18}
Genotype	AA	−1.69	−2.08	−1.30	2.1×10^{-17}	

Hygiene of the mother and partner was estimated in two different ways. For the mother, how often she washed the child (face, hands, and body) was considered, whereas for the partner how often he helped with washing clothes, dinnerware, and utensils was considered. No data were available on how often the parents washed themselves. Reference categories were as follows: <25 for maternal age, low education, paternal social classes I+II, mortgaged/owned, low hygiene, and GG+GA for maternal genotype. Group *P*-values represent a 2 d.f. statistic testing that both parameters for a particular confounder were zero. Analysis restricted to mothers of white ethnic origin.

Prediction of parental deodorant usage on the basis of the child's genotype

Supplementary Figure S2 online shows the expected child genotype–parent phenotype associations based on parents' genotypes, allele frequency, and proportions of never use over daily use of deodorant for each genotypic category, assuming a recessive model and restricting to white mothers. For maternal genotype–phenotype associations, we found good agreement between the observed ratios in children for each genotype and the liability predictions computed from parents.

Percentage of deodorant usage: mothers versus partners

The percentage of ALSPAC partners using deodorant daily or on most days (82.7%) is significantly lower ($P = 5.3 \times 10^{-74}$) than the percentage for mothers using deodorant daily or on most days (93.3%). The figures are computed from Table 3 and are as follows: 945 partners using deodorant approximately once a week or less and 4,502 partners using it on most days or daily; and 433 mothers using deodorant about once a week or less and 6,062 mothers using it on most days or daily.

Table 3a shows that 77.8% of women with the AA genotype still use deodorant (at least once a week week), compared with 80.0% of male parents of children with the AA genotype who use deodorant at least once a week (Table 3c).

Table 3a also shows that the proportion of women with the GG genotype who do not use deodorant (<1 per week or never) is much reduced (4.7%) compared with 13.0% of male parents of children with the GG genotype who never use deodorant or use it <1 per week (Table 3c).

The results in non-white ALSPAC participants can be seen in Supplementary Appendix S1.

DISCUSSION

This study shows a significant association between deodorant usage and a common genetic variation at the DNA level, which to our knowledge is previously unreported. Homozygotes for the rs17822931 A allele within *ABCC11* are almost 5-fold less likely to use deodorant than GG homozygotes and heterozygotes for this SNP. Previous studies offer a biological basis for this association. Homozygotes for the rs17822931 A allele show clearcut differences compared with G allele carriers in the content of axillary secretions (Martin *et al.*, 2010; Preti and Leyden, 2010). The lower frequency of deodorant usage in this group of individuals therefore reflects the lack of need for deodorant usage, a substantially socially determined behavior. Figure 2 shows a diagram relating rs17822931 genotype with axillary odor and deodorant usage.

Nonodorous mothers and partners still use deodorant

On the basis of the production of axillary odorants, we would expect that AA individuals would not use deodorant, whereas GG individuals would use it. However, there are mothers who use deodorant at least once a week without needing it (77.8% of women with the AA genotype). From child genotype, we estimate that 80.0% of fathers of AA genotype children use deodorant with less need to do so.

Deodorant nonuse in the genotypically odorous

In addition, our results indicate a different behavior between men and women in relation to not using deodorant. This seems to be dependent on the rs17822931 genotype. The proportion of women with the GG genotype who, despite needing deodorant, never use it or use it less than once a week is small (4.7%). In contrast, 13.0% of fathers of children with the GG genotype who need to use a deodorant never use it.

Implications

This indicates the influence of other factors such as sex on the behavior of deodorant usage. In northeast Asia, an estimated 7% of individuals use deodorant (Air Sense News, 2008), whereas <1% of individuals are of the GG genotype (dbSNP, 2011). If, as in our UK study, 77.8% of AA genotype women used deodorant, then the total usage would be at least 62–74%. This indicates that sociocultural factors also have a major impact on deodorant usage. In this instance, it is likely that deodorant usage is not widely adopted because there is, for much of the east Asia population, no need for it. The opposite is true in European populations.

The finding that 77.8% of nonodorous (AA) individuals use deodorant at least once a week makes rs17822931 a potential target for personal genomics in relation to deodorant usage. These individuals could avoid the chemical exposures, dermatological (Zirwas and Moennich, 2008) or other health risks

Table 3. Association of genotype with deodorant usage

	GG	GA	AA
(a)			
Never	166 (195.4)	69 (55.0)	20 (4.6)
	4.42	*3.56*	*51.67*
	1	1	1
<1 per week	66 (67.4)	16 (19.0)	6 (1.6)
	0.03	*0.47*	*12.30*
	1	0.58 (0.32–1.08)	0.75 (0.29–1.96)
~1 per week	67 (69.0)	19 (19.4)	4 (1.6)
	0.06	*0.01*	*3.49*
	1	0.68 (0.38–1.22)	0.50 (0.16–1.50)
Most days	608 (650.6)	213 (183.1)	28 (15.3)
	2.79	*4.87*	*10.56*
	1	0.84 (0.61–1.16)	0.38 (0.21–0.70)
Daily	4,070 (3,994.6)	1,084 (1,124.5)	59 (93.9)
	1.42	*1.46*	*12.98*
	1	0.64 (0.48–0.86)	0.12 (0.07–0.20)
$\chi^2 = 110.07$, 8 degrees of freedom, 2-sided *P*-value $= 3.7 \times 10^{-20}$			
(b)			
Never	192 (213.5)	80 (61.9)	8 (4.7)
	2.16	*5.31*	*2.37*
	1	1	1
<1 per week	65 (64.8)	20 (18.8)	0 (1.4)
	0.00	*0.08*	*1.42*
	1	0.74 (0.42–1.30)	N/A
~1 per week	96 (96.1)	27 (27.8)	3 (2.1)
	0.00	*0.03*	*0.38*
	1	0.68 (0.41–1.11)	0.75 (0.19–2.89)
Most days	667 (696.0)	226 (201.8)	20 (15.2)
	1.21	*2.91*	*1.49*
	1	0.81 (0.60–1.10)	0.72 (0.31–1.66)
Daily	4,417 (4,366.7)	1,223 (1,265.8)	88 (95.6)
	0.58	*1.44*	*0.60*
	1	0.66 (0.51–0.87)	0.48 (0.23–1.00)
$\chi^2 = 19.98$, 8 degrees of freedom, 2-sided *P*-value $= 0.010$			
(c)			
Never	408 (413.2)	120 (119.4)	13 (8.4)
	0.06	*0.00*	*2.46*
	1	1	1
<1 per week	132 (132.1)	37 (38.2)	4 (2.7)
	0.00	*0.04*	*0.63*
	1	0.95 (0.63–1.45)	0.95 (0.30–2.97)
~1 per week	165 (176.4)	65 (51.0)	1 (3.6)
	0.74	*3.86*	*1.88*
	1	1.34 (0.94–1.90)	0.19 (0.02–1.47)
Most days	923 (934.0)	281 (269.9)	19 (19.1)
	0.13	*0.46*	*0.00*
	1	1.04 (0.81–1.32)	0.65 (0.32–1.32)
Daily	2,532 (2,504.3)	299 (723.6)	48 (51.2)
	0.31	*0.84*	*0.20*
	1	0.40 (0.32–0.51)	0.60 (0.32–1.11)
$\chi^2 = 11.60$, 8 degrees of freedom, 2-sided *P*-value $= 0.170$			

(a) Maternal genotype with maternal deodorant usage. (b) Association of child's genotype with maternal deodorant usage. (c) Association of child's genotype with partner's deodorant usage. Contingency table showing genotypic frequencies of rs17822931 observed in each category of deodorant usage in white mothers in ALSPAC. Expected numbers are in brackets and contribution to the overall χ^2 in italics. Line 3 within each cell shows the odds ratio and 95% confidence interval (in brackets).

(Farrow *et al.*, 2003), and cost without social disadvantage. We estimate a total unnecessary expenditure of £9million/year (45×10^4 users spending £20 per year) in this 1% of the UK population.

Considering the proposed odor-related mate choice related to the HLA region (Yamazaki and Beauchamp, 2007), *ABCC11* genotype is also a candidate for assortative mating based on its influence on body odor. However, we found no association between maternal rs17822931 genotype and partner's deodorant usage. It is notable that apparently subtle HLA-related genetic traits may influence mate choice, whereas the potent axillary effect of *ABCC11* does not. However, social selection at a low rate over many generations or before the advent of deodorants remains plausible and also a possible mechanism for allelic selection in populations. Other overt genetically influenced traits such as hair (Sabeti *et al.*, 2007), skin color, or coat color in animals (Ludwig *et al.*, 2009), as well as height, are certainly subject to selection or assortative mating.

Heterozygote effect

Although the recessive model was a better fit than the additive model to maternal usage with maternal genotype, there was still a significant deviation reflecting an effect for GA, which was greater than a recessive effect (equal to GG) but less than an additive effect. Indeed, we observed small but statistically significant ($P = 0.002$) differences between rs17822931 GG and GA individuals in relation to deodorant usage. These differences mirror the reported differences in concentration of odorants across rs17822931 genotypes (Martin *et al.*, 2010). Within rs17822931, fewer GA individuals use deodorants than GG individuals, consistent with a slightly lower production of axillary odorants by the former.

Population substructure and self-reported phenotype

Given that there are marked differences in genotype frequencies between white and non-white populations (Yoshiura *et al.*, 2006; Toyoda *et al.*, 2009; Toyoda and Ishikawa, 2010), one could argue that our results could be due to population substructure. However, the associations were equally clearcut when non-white individuals were removed

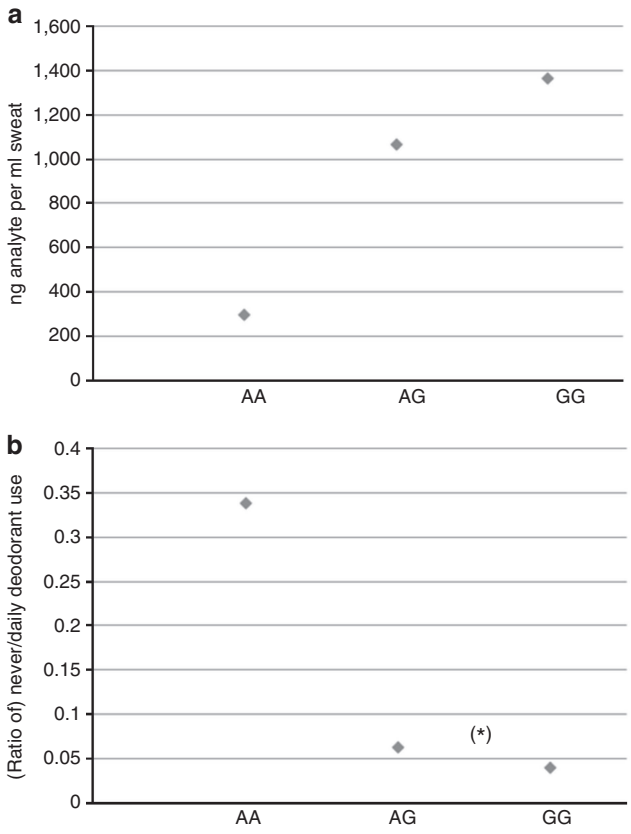

Figure 1. Relationship between an odoriferous steroid and rs17822931 single-nucleotide polymorphism (SNP) genotype and between deodorant use and the rs17822931 SNP genotype. (**a**) Concentration of the odoriferous steroid 5α-androst-16-en-3-one observed for each rs17822931 SNP genotype, as previously described (Martin *et al.*, 2010). (**b**) Ratio of never use over daily use of deodorant in ALSPAC (Avon Longitudinal Study of Parents and Children) white mothers observed for each rs17822931 SNP genotype. *There is a significant ($P=0.002$) difference between the ratios observed in AG and GG individuals.

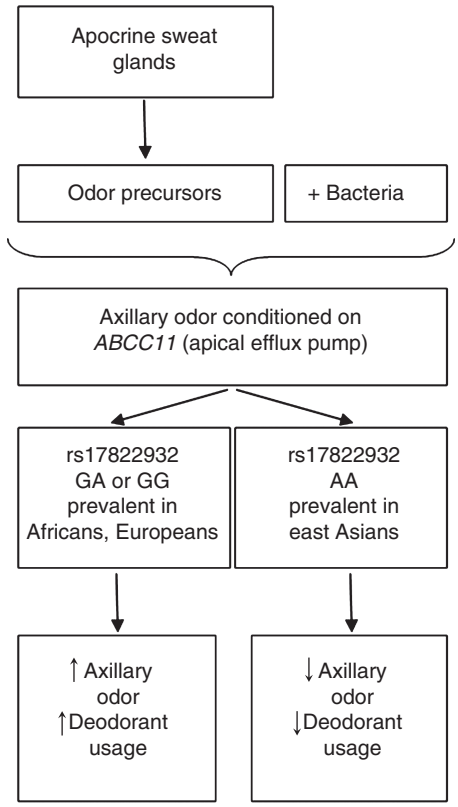

Figure 2. Diagram showing the involvement of genetic variation in *ABCC11* and both axillary odor and deodorant usage.

from the analyses, indicating that the effect attributes to the genotype and not to population substructure. In addition, our Hardy–Weinberg results show no significant difference between the observed and expected frequency of AA homozygotes, with no significant evidence of missingness of this genotype. This represents additional evidence supporting the contention that all the observed AA homozygous mothers who were reported to be white European are indeed white European, and argues against our results being an artifact due to population substructure.

Another potential source of confounding is the fact that deodorant usage is a self-reported phenotype in our study and in previous studies (Mirick *et al.*, 2002). This opens the possibility that participants underreported or overreported deodorant usage. This source of confounding is less likely in the categories of daily and never use, as these are clearcut. We do not have a clear indication about the effect in other categories. To have a significant effect, underreporting or overreporting would have to involve a differential effect of one genotype.

In addition, it is possible that a proportion of the self-reported deodorant usage is actually antiperspirant usage, as most people think that both deodorants and antiperspirants are the same thing. However, whereas deodorants reduce body odor and have an antiseptic action against bacteria, antiperspirants reduce the amount of sweat acting on pores (Benohanian, 2001). The overall result is the same in both instances (reducing axillary odor), but the fact that both deodorants and antiperspirants have different mechanisms of action suggests that they possibly have different biological/genetic relationships.

However, it is recognized for genetic association studies, in contrast with other epidemiological association studies, that genotype cannot be ''caused'' by outcomes or other factors (e.g., confounders) and therefore is an independent/orthogonal factor in such analyses (Smith *et al.*, 2007; Ebrahim and Davey-Smith, 2008). Supplementary Table S6 online shows that the rs17822931 genotype distributed randomly relative to (i.e., not association with) maternal age, maternal education, paternal social class, housing tenure, and hygiene. We do observe a strong association of maternal age, maternal education, housing tenure, and hygiene with deodorant usage, but genotype is independent of these factors. In addition, on the basis of the observed magnitude of effects found, our results are consistent with the relatively larger effect for the rs17822931 genotype compared with the effects observed for all putative confounders, including hygiene.

Non-white individuals

We also analyzed deodorant usage in non-white individuals. In non-whites, we found effects similar to those observed in UK Europeans. rs17822931 AA homozygotes were less likely to use deodorant than GA and GG individuals. This is consistent with the lower production of axillary odorants in AA homozygotes regardless of their ethnic origin and represents an extension, to non-whites, of the results observed in our work in UK Europeans in relation to deodorant usage and rs17822931. However, these results should be interpreted with caution, given the small sample size and the possibility of population substructure within non-whites.

In summary, we have shown that the rs17822931 genotype is a strong predictor of deodorant usage, but despite rs17822931 representing a Mendelian trait, ~80% of genotypically nonodorous white European mothers still use deodorant. The above finding appears to be true for males as well. This is likely driven by sociocultural factors. On the basis of genotype (and/or dry earwax), this group could elect to abandon the chemical exposures and costs of deodorant use. This represents a potential application of personalized genetics in personal hygiene.

MATERIALS AND METHODS

Participants

ALSPAC is a longitudinal, population-based birth cohort study that recruited 14,541 pregnant women residing in Avon, United Kingdom, with expected dates of delivery between 1 April 1991 and 31 December 1992 (Boyd *et al.*, 2012; Fraser *et al.*, 2012). There were 14,062 live-born children. There is questionnaire information for both mothers and their partners. Most of the mother's partners are fathers of the mother's children. The study protocol has been described previously (Jones *et al.*, 2000; Golding *et al.*, 2001), and further details are available on the ALSPAC website (http://www.bris.ac.uk/alspac). Ethical approval for all aspects of data collection was obtained from the ALSPAC Law and Ethics Committee (institutional review board 00003312). Written informed consent for the study was obtained for genetic analysis. Our study adhers to the Declaration of Helsinki Principles.

Genotyping

All ALSPAC mothers and children were genotyped for rs17822931 by KBiosciences (Essex, UK) with their KASPar system. This is a competitive allele-specific PCR SNP genotyping system that uses Fluoresence Resonance Energy Transfer (FRET) quencher cassette oligos (KBioscience, 2009). A total of 8,326 mothers and 9,167 children were successfully genotyped for rs17822931. Deviations from Hardy–Weinberg proportions were calculated with the Hardy–Weinberg equilibrium calculator (http://www.oege.org/software/hwe-mr-calc.shtml) (Rodriguez *et al.*, 2009). This calculator was also used to test possible "missingness" (ascertainment bias) of rs17822931 AA genotypes in white mothers.

Study variables

Deodorant use was assessed by self-completed questionnaires administered during pregnancy for the partner and at 8 months after the child was born for the mother of the study child. Under a section entitled "Chemicals in your environment," each mother was asked the following question: "In the last few months, how often have you used the following (whether at home or at work)?" There was then a list of chemicals, including "deodorants." The partner was asked a similar set of questions during pregnancy, but they were presented with the words "Just before your partner became pregnant, how often did you use the following (whether at home or at work)?" These questions had categorical responses on a 5-point scale ranging from *not at all* to *daily*. These were converted to an estimate of weekly usage: *not at all* (0), *less than once a week* (0.5), *about once a week* (1), *most days* (4), and *daily* (7).

The total number of mothers with information for both deodorant use and rs17822931 were 6,495 and there were 7,132 children. A total of 5,047 partners had both phenotypic information for deodorant use and rs17822931 genotypic information for their children.

Statistical analyses

Linear regression was used to analyze the estimated weekly usage. The results for additive (number of copies of the minor allele) and recessive (effect of homozygotes of minor allele) genetic models are reported. Deviations from these models were tested by comparison with the genotype genetic model.

On the basis of maternal genotype–phenotype associations, we calculated the expected child genotype–parent phenotype associations based on the recessive model. These predictions of child's rs17822931 genotype were based on parents' genotypes, allele frequency, and proportions of never use over daily use of deodorant for each genotypic category in white mothers. They were computed as the probability of transmission of each allele multiplied by the allele frequency and for the ratio of never use over daily use of deodorant.

The χ^2 tests for contingency tables were performed with the software CONTING ver 2.61 (Ott, 1988).

Odds ratios and 95% confidence intervals were computed using the odds ratio calculator (Bland and Altman, 2000).

Linear regression was performed using Stata version 12.2 (Stata Corp, College Station, TX).

CONFLICT OF INTEREST

The authors state no conflict of interest.

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SUPPLEMENTARY MATERIAL

Supplementary material is linked to the online version of the paper at http://www.nature.com/jid

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