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Research paper

Despite identifying some shared gene associations with human atopic dermatitis the use of multiple dog breeds from various locations limits detection of gene associations in canine atopic dermatitis

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ABSTRACT

Canine atopic dermatitis (cAD) is a common, severe pruritic and inflammatory skin disease and is a major veterinary welfare issue. This study genotyped 97 single nucleotide polymorphisms (SNPs) in 25 candidate genes in 659 dogs across eight breeds from three locations (UK, USA and Japan). These genes were selected from hAD literature, and previous cAD gene expression experiments. The aim of this study was to identify any shared gene associations between cAD and hAD.

Only one SNP within the TSLP-receptor was associated with all eight breeds (corrected $p = 0.037$). Five SNPs within Filaggrin, DPP4, MS4A2, and INPPL1 were associated with cAD, but only in certain breeds from different locations. Though these associations are broadly similar to hAD the variability of results across the breeds and locations demonstrates that a candidate gene approach using mixed breeds from different locations is not appropriate. This study therefore suggests that further candidate gene studies in cAD should be breed and location specific to increase the likelihood of finding associations with the disease.

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1. Introduction

Canine atopic dermatitis (cAD) is a common inflammatory skin disease that is considered to be a naturally occurring, spontaneous model of human atopic dermatitis (hAD). Other animal models of hAD can display clinical signs consistent with hAD, but dogs are of particular interest as, unlike rodent models, they largely share the same environment as humans. The pathogenesis of the disease in both humans and dogs is strongly associated with immunological hyper-reactivity, although skin barrier function, microbial colonisation and infection are also considered contributing factors (Olivry and Hill, 2001; Vickery, 2007).

The similarities in the immunological profile and skin barrier function in cAD and hAD imply there is a shared genetic and/or environmental cause.

In humans, it has been established that AD is a complex disease with several genetic factors (Hoffjan and Epplen, 2005). Genetic research into cAD is limited, although the strong breed association provides evidence of genetic links (Sousa and Marsella, 2001; Tarpatki et al., 2006).

Unlike most murine AD models, cAD does not appear to be caused by a single gene defect, and, like hAD, appears to be a polygenic or oligogenic disorder with complex inheritance and interactions with environmental influences. It therefore represents a potentially valuable spontaneous model for hAD. A further advantage is that dog breeds form genetically inbred and isolated homogenous populations which exhibit strong linkage disequilibrium (LD) that extends over long distances (0.8–5 Mb) (Sutter et al., 2004). In contrast, humans exhibit weak LD across the genome (10–100 kb) (Reich et al., 2001). Thus, fewer genetic mark-

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ers and smaller sample sizes can be used to identify disease associations in dogs (Lindblad-Toh et al., 2005).

We previously conducted a genome wide association study (GWAS) and validated the results in a large case–control comparison of cAD (Wood et al., 2009a). Significant associations were found in this study. However the associations were with SNPs in intergenic regions, and therefore how these SNPs contribute to the pathogenesis is unknown. A major disadvantage of the GWAS array we used was that it had large gaps. For example the Filaggrin region, which has been demonstrated to be of great importance in hAD, was not covered by the array. This was also true for many of the 'traditional' candidate genes in cAD. Even the newly available GWAS arrays do not have complete coverage and as there is no HAPMAP (The International HapMap Consortium, 2007) equivalent for dogs we cannot impute the associations for SNPs not included on the array. Therefore to test associations with these genes it is necessary to conduct a candidate gene study.

The aim of this study was a preliminary investigation of the genetics of cAD to identify any shared associations between cAD and hAD using a candidate gene approach. We identified potential candidate genes using previously analysed gene expression data in cAD from mRNA expression microarray (Merryman-Simpson et al., 2008) and qPCR studies (Wood et al., 2009b), as well as data from other publications on the pathogenesis of canine and human AD.

2. Methods

2.1. Case–control selection

DNA from residual blood samples excess to diagnostic requirements stored in the UK DNA Companion Animals Archive (http://www.liv.ac.uk/dna_archive_for_companion_animals/, last accessed 14/10/2009) were selected from 11 Boxers and 11 West Highland White Terrier (WHWT) for a SNP discovery programme conducted as part of this study.

For the SNP genotyping experiment, DNA from atopic and healthy control dogs was derived from residual blood samples excess to diagnostic requirements from British, Japanese and US veterinary clinics. In total, 659 DNA samples from eight dog breeds were selected for SNP genotyping. Table 1 summarizes the breakdown of breeds and their disease status.

Table 1
Samples selected for SNP genotyping.

Breed	cAD				Healthy control				Grand total
	UK	USA	Japan	Total	UK	USA	Japan	Total	
Boxer	11	–	–	11	24	–	–	24	35
GSD	10	9	–	19	23	15	–	38	57
Labrador	23	33	8	64	75	43	11	129	193
G. Retriever	4	23	13	40	28	21	17	66	106
Shiba Inu	–	–	23	23	–	–	33	33	56
Shih Tzu	2	6	39	47	12	2	48	62	109
Pit Bull	–	20	–	20	–	17	–	17	37
WHWT	14	4	–	18	34	14	–	48	66
Totals				242				417	659

The diagnosis of cAD was based on a compatible history, typical clinical signs and exclusion of other causes of pruritus (DeBoer and Hillier, 2001). Briefly, hair plucks, adhesive tape strips, skin scrapes, *Sarcoptes* specific IgG serology, anti-parasite therapy and antimicrobial treatment were used to eliminate ectoparasites, bacterial pyoderma and *Malassezia* dermatitis. A minimum 6-week food trial using a novel home cooked or commercial single protein, or commercial hydrolysed protein diet was used to rule out a cutaneous adverse food reaction. All the dogs with a clinical diagnosis of AD had at least one reaction to indoor allergens on an intradermal test performed and evaluated according to accepted criteria (Hillier and DeBoer, 2001).

DNA was extracted from stored EDTA blood samples using Qiamp DNA blood midi kits (Qiagen, Crawley, UK) as previously described (Clements et al., 2008). DNA was normalized to 50 ng/μl in TE buffer (10 ml 1 M TRIS and 0.2 ml 0.5 M EDTA in 1 L distilled water) for storage.

2.2. Candidate gene selection

In order to identify gene polymorphisms associated with cAD, the literature searches of hAD and cAD were undertaken to identify potential candidate genes, and data from previously conducted mRNA microarray and qPCR cAD studies (Merryman-Simpson et al., 2008; Wood et al., 2009b) were considered. This provided a shortlist of 25 genes (Supplementary Table 1). Ensembl (Curwen et al., 2004) was used to identify published SNPs present in the canine candidate genes. In candidate genes with a low number of available SNPs, a SNP discovery programme was initiated to increase the numbers of SNPs available for genotyping.

2.3. SNP discovery

CMA1, SGPL1, DPP4, SPRR1B, S100A8, IL1RL1, MS4A2 and P-selectin were subjected to a SNP discovery programme. Denaturing high-performance liquid chromatography (DHPLC) (Lilleberg, 2003) followed by DNA sequencing was used to identify SNPs. Specific regions from candidate genes, such as exons and promoters, were selected for SNP discovery analysis. Highly repetitive areas were avoided and the primers were designed around areas spanning 200–700 bp. The primers for the candidate regions were designed using Primer3 plus (<http://www.>

Table 2Individual breed analysis: corrected *p*-value significant SNPs in cAD.

Rs number	Gene region	Breed and geographic region	<i>p</i> -Value	Odds ratio	L95	U95
RS22588227	Filaggrin	UK Labrador	0.009**	5.6	2.1	14.9
RS22986026	MS4A2	Japan Shiba Inu	8.7×10^{-4} *	7.4	2.8	19.9
RS23907319	DPP4	UK Labrador	5.2×10^{-4} **	6.4	2.6	15.4
RS9070190	INPPL1	Japan Shiba Inu	4.6×10^{-5} **	9.2	3.6	23.4
RS9150577	INPPL1	Japan Shiba Inu	1.9×10^{-4} **	0.1	0.1	0.3

* Denotes that this association is significant on trend analysis using the genotypic model statistical tests.

** Denotes that this association is significant on trend analysis using the genotypic model and logistic association using the additive model.

bioinformatics.nl/cgi-bin/primer3plus/primer3plus.cgi). dHPLC analysis was performed as previously described (Short, 2006). Samples with suspected polymorphisms were sequenced to confirm the SNP identity. The samples were prepared for sequencing using Exo-SAPIT® (USB, USA) according to the manufacturer's instructions to purify the sample. Forward or reverse primers from the candidate regions at 0.4 mM were used to sequence the sample. Sequencing was performed using the Applied Biosystems 3730 DNA Analyzer (Applied Biosystems, Foster City, CA, USA). Once the SNP sequences were confirmed they were included in the SNP genotyping study.

2.4. SNP genotyping and statistical analysis

97 SNPs had probe and primer sequences successfully designed for Sequenom-based genotyping (San Diego, CA, USA), see [Supplementary Table 2](#). These SNPs were genotyped in each dog using the Sequenom platform. PCRs were performed in 384-well plate format. A 5 µl reaction volume was used per well: 0.1 µl hot start Taq (5 U/µl, Qiagen), 0.5 µl each of 500 nM forward and reverse primers (Metabion, Martinsried, Germany), 0.325 µl MgCl₂ (25 mM, Qiagen), 0.1 µl dNTPs (25 mM), 0.625 µl × 10 buffer (Qiagen) 0.35 µl distilled water and 2.5 µl of DNA at 10 ng/µl. Amplification conditions were: 94 °C for 15 min; followed by 35 cycles of 94 °C for 20 s, 56 °C for 30 s and 72 °C for 1 min; and a final elongation phase of 72 °C for 3 min. Shrimp Alkaline Phosphatase clean, the iPLEX reaction and the resin clean were all performed as instructed by Sequenom. Spectro chip® arrays were spotted using a nanodispenser, according to the manufacturer's instructions (Sequenom). The SpectroAquire and MassARRAY Typer software packages (Sequenom) were used for interpreta-

tion, and Typer analyser V3.4.0.18 was used to review and analyse all data.

The whole-genome association analysis toolset PLINK (Purcell et al., 2007) was used to analyse the data. All data were subject to quality control procedures; only SNPs which conformed to Hardy–Weinberg expectations (HWE), exhibited ≥90% genotyping success and had a minor allele frequency ≥0.01 were included in subsequent analysis.

Association tests, including basic association, allelic association, logistic regression and the genotypic model statistical test were undertaken on all breeds separately if there were more than 20 cases and 20 controls in the group being analysed. This is to ensure statistical robustness. Meta-analysis (Cochran–Mantel–Haenszel test for $2 \times 2 \times K$ stratified tables) was also undertaken, as this considers the data as a whole taking into account variables such as breed and origin (defined from supplied phenotype information), eliminating false positives due to population substructure (Tian et al., 2008). *p*-Values were corrected for multiple hypotheses testing, using a Bonferroni style correction which uses the number of independent loci (48 in this study) instead of the number of SNPs tested.

3. Results and discussion

Of the 97 SNPs investigated, 6 were significantly associated with cAD after correction for multiple testing. Most SNP associations were restricted to certain breeds and/or dogs collected at particular geographical locations. [Table 2](#) shows the results from the individual breed analyses, detailing corrected *p*-values, odds ratios and confidence intervals obtained from the allelic association tests. Where indicated, the cAD associations were also statistically significant on the logistic and genotypic association tests.

Table 3

Meta-analysis of all breeds clustered by breed and geographic region.

SNP	CHR	BP	Gene	Location	Odds ratio	<i>p</i> -Value	Corrected <i>p</i> -value	Lower 95% CI	Upper 95% CI
RS24613432	X	751050	TSLP-receptor	Splice site, intronic	0.38	8.0×10^{-4}	0.037	0.2	0.7
RS23907319	36	10444284	DPP4	Synonymous coding	1.54	0.004	0.20	1.1	2.1
SPRR.e3.1	17	64883377	SPRR	Exon 3	0.64	0.011	0.51	0.5	0.9
RS22819627	2	45159024	SPINK5	Non-synonymous coding – amino acid change D/E	1.40	0.014	0.67	1.1	1.8
RS21902632	1	16476102	SCCA2	Non-synonymous coding – amino acid change R/S	0.70	0.017	0.84	0.5	0.9
RS22565034	17	64303134	Filaggrin	Non-synonymous coding – amino acid change E/K	0.36	0.026	1.00	0.1	0.9
RS22818977	2	45167469	SPINK5	Synonymous coding	0.75	0.031	1.00	0.6	1.0
RS9150577	21	28912392	INPPL1	Synonymous coding	0.76	0.033	1.00	0.6	1.0
RS9190946	3	15784064	ARTS-1	Synonymous coding	1.33	0.043	1.00	1.0	1.7
RS8877521	21	53631234	MS4A2	Synonymous coding	1.28	0.044	1.00	1.0	1.6

Table 3 shows the SNPs that were associated with cAD in all eight dog breeds following meta-analysis. In each instance, the corrected *p*-value represents the level of significance after correction for multiple testing. After correction, only one SNP (rs24613432) was shown to be statistically significantly associated with cAD in all the dog breeds.

The SNP associated with cAD in all eight breeds by meta-analysis (rs24613432) was found in the thymic stromal lymphopoietin (TSLP) receptor gene. Individual breed associations were not found with TSLP-receptor, the threshold for a statistically significant association may not have been reached because of the limited sample sizes in the individual breed analysis. The TSLP-receptor forms a functional complex with TSLP and the IL-7 receptor. It is involved in the JAK-STAT signalling pathway, which is used by interleukins to transduce the signals that initiate cytokine specific gene transcription, and is therefore of interest in terms of the immunological response in AD. TSLP-receptor's functional complex with TSLP links their functions closely, TSLP has been implicated in allergic inflammation (Liu, 2006), suggesting that TSLP-receptor may also have a role. The TSLP gene has not been characterised in dogs, and was not included in this study. It is therefore a candidate for future studies.

Meta-analysis allowed clustering of the data by breed and point of origin, which allowed independent analysis of these variables while increasing the statistical power. This is the preferred method of analysis for this type of data as it improves statistical confidence by comparing all the cases and controls together. Nevertheless, these data also need to be analysed by breed because there are no assurances that the same disease phenotype in different breeds has the same genetic basis.

Multiple individual breed associations were found, though these varied according to the geographic origin of the dogs. Once the dogs were split down by breed and geographic region the numbers for analysis in each group were small limiting the analysis. The Labrador had the highest number of dogs (UK 23 cases, 75 controls; USA 33 cases, 43 controls), and associations with Filaggrin and DPP4 were found in UK Labradors, though the low numbers does mean the chance of a false positive association is increased. The data was also analysed by breed to increase the numbers for analysis (not broken down by geographic region) but no associations were found. This is of note because if the associations seen in one geographic region are different from another when the data is put together to increase the statistical strength the association is lost. Further analysis by fine mapping and re-sequencing in individual breeds will be required to better characterise the SNPs associated with cAD in this study.

The fact that we have identified associations with different SNPs in different breeds appears to support the decision to analyse the breeds separately, emphasizing the potential importance of different breeds and genotypes in the same disease phenotype. However, the use of eight different breeds from different locations reduced the numbers for single breed and location analysis and meant that only breeds with more than 20 in each group were analysed.

It is therefore possible that some associations may have been missed due to a lack of statistical power and exclusion due to low numbers. Further studies with larger numbers of single breeds from defined geographical locations or case-control selection using ancestry informative markers could strengthen these results.

4. Conclusion

Though these associations are broadly similar to hAD the variability of results across the breeds and locations demonstrates that a candidate gene approach using mixed breeds from different locations is not appropriate. This study therefore suggests that further investigation of cAD by candidate gene study should be breed and location specific to increase the likelihood of finding associations with the disease.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at [doi:10.1016/j.vetimm.2010.07.020](https://doi.org/10.1016/j.vetimm.2010.07.020).

References

- Clements, D.N., Wood, S., Carter, S.D., Ollier, W.E., 2008. Assessment of the quality and quantity of genomic DNA recovered from canine blood samples by three different extraction methods. *Research in Veterinary Science* 85 (1), 74–79.
- Curwen, V., Eyraas, E., Andrews, T.D., Clarke, L., Mongin, E., Searle, S.M., Clamp, M., 2004. The Ensembl automatic gene annotation system. *Genome Research* 14, 942–950.
- DeBoer, D.J., Hillier, A., 2001. The ACVD task force on canine atopic dermatitis (XV): fundamental concepts in clinical diagnosis. *Veterinary Immunology and Immunopathology* 81, 271–276.
- Hillier, A., DeBoer, D.J., 2001. The ACVD task force on canine atopic dermatitis (XVII): intradermal testing. *Veterinary Immunology and Immunopathology* 81, 289–304.
- Hoffjan, S., Epplen Jr, T., 2005. The genetics of atopic dermatitis: recent findings and future options. *Journal of Molecular Medicine* 83, 682–692.
- Lilleberg, S.L., 2003. In-depth mutation and SNP discovery using DHPLC gene scanning. *Current Opinion in Drug Discovery and Development* 6, 237–252.
- Lindblad-Toh, K., Wade, C.M., Mikkelsen, T.S., Karlsson, E.K., Jaffe, D.B., Kamal, M., Clamp, M., Chang, J.L., Kulbokas, E.J., Zody, M.C., Mauceli, E., Xie, X., Breen, M., Wayne, R.K., Ostrander, E.A., Ponting, C.P., Galibert, F., Smith, D.R., deJong, P.J., Kirkness, E., Alvarez, P., Biagi, T., Brockman, W., Butler, J., Chin, C.W., Cook, A., Cuff, J., Daly, M.J., DeCaprio, D., Gnerre, S., Grabherr, M., Kellis, M., Kleber, M., Bardeleben, C., Goodstadt, L., Heger, A., Hitte, C., Kim, L., Koepfli, K.P., Parker, H.G., Pollinger, J.P., Searle, S.M.J., Sutter, N.B., Thomas, R., Webber, C., Lander, E.S., 2005. Genome sequence, comparative analysis and haplotype structure of the domestic dog. *Nature* 438, 803–819.
- Merryman-Simpson, A.E., Wood, S.H., Fretwell, N., Jones, P.G., McLaren, W.M., McEwan, N.A., Clements, D.N., Carter, S.D., Ollier, W.E., Nuttall, T., 2008. Gene (mRNA) expression in canine atopic dermatitis: microarray analysis. *Veterinary Dermatology* 19, 59–66.
- Olivry, T., Hill, P.B., 2001. The ACVD task force on canine atopic dermatitis (VIII): is the epidermal lipid barrier defective? *Veterinary Immunology and Immunopathology* 81, 215–218.
- Purcell, S., Neale, B., Todd-Brown, K., Thomas, L., Ferreira, M.A.R., Bender, D., Maller, J., Sklar, P., de Bakker, P.I.W., Daly, M.J., Sham, P.C.,

2007. PLINK: a tool set for whole-genome association and population-based linkage analyses. *The American Journal of Human Genetics* 81, 559–575.
- Reich, D.E., Cargill, M., Bolk, S., Ireland, J., Sabeti, P.C., Richter, D.J., Lavery, T., Kouyoumjian, R., Farhadian, S.F., Ward, R., Lander, E.S., 2001. Linkage disequilibrium in the human genome. *Nature* 411, 199–204.
- Short, A.D., 2006. The genetics of canine diabetes: a candidate gene study. The University of Manchester, Thesis.
- Sousa, C.A., Marsella, R., 2001. The ACVD task force on canine atopic dermatitis (II): genetic factors. *Veterinary Immunology and Immunopathology* 81, 153–157.
- Sutter, N.B., Eberle, M.A., Parker, H.G., Pullar, B.J., Kirkness, E.F., Kruglyak, L., Ostrander, E.A., 2004. Extensive and breed-specific linkage disequilibrium in *Canis familiaris*. *Genome Research* 14, 2388–2396.
- Tarpataki, N., Papa, K., Reiczgel, J., Vajdovich, P., Vorosi, K., 2006. Prevalence and features of canine atopic dermatitis in Hungary. *Acta Veterinaria Hungarica* 54, 353–366.
- Tian, C., Gregersen, P.K., Seldin, M.F., 2008. Accounting for ancestry: population substructure and genome-wide association studies. *Human Molecular Genetics* 17, R143–R150.
- The International HapMap Consortium, 2007. A second generation human haplotype map of over 3.1 million SNPs. *Nature* 449, 851–861.
- Vickery, B.P., 2007. Skin barrier function in atopic dermatitis. *Current Opinion in Pediatrics* 19, 89–93.
- Wood, S.H., Ke, X., Nuttall, T., McEwan, N., Ollier, W.E., Carter, S.D., 2009a. Genome-wide association analysis of canine atopic dermatitis and identification of disease related SNPs. *Immunogenetics* 61, 765–772.
- Wood, S.H., Clements, D.N., Ollier, W.E., Nuttall, T., McEwan, N.A., Carter, S.D., 2009b. Gene expression in canine atopic dermatitis and correlation with clinical severity scores. *Journal of Dermatological Science* 55, 27–33.
- Liu, Y.-J., 2006. Thymic stromal lymphopoietin: master switch for allergic inflammation. *The Journal of Experimental Medicine* 203, 269–273.

Supplementary Table: Candidate Genes

Gene Name	Gene Abbreviation	Chromosome	Evidence Source	Reference
Interleukin 4	IL-4	11	Human and canine Literature	(Kawashima et al. 1998)
Endoplasmic reticulum aminopeptidase 1	ERAP1 or ARTS-1	3	Canine microarray	(Merryman-Simpson et al. 2008)
Cadherin 13	CDH13	5	Canine microarray	(Merryman-Simpson et al. 2008)
Periostin Precursor	POSTN	25	Canine microarray	(Merryman-Simpson et al. 2008)
Syndecan-1 Precursor (CD138)	SDC1 or SYND1	17	Canine microarray	(Merryman-Simpson et al. 2008)
Phosphatidylinositol-3,4,5-trisphosphate 5-phosphatase 2	INPPL1	21	Canine microarray and qPCR	(Merryman-Simpson et al. 2008;Wood et al. 2009b)
Serum amyloid A protein Precursor (SAA)	SAA_CANFA	21	Canine microarray and qPCR	(Merryman-Simpson et al. 2008;Wood et al. 2009b)
Serpin B4 (Squamous cell carcinoma antigen 2)(Leupin)	SERPINB4 or SCCA-2	1	Canine microarray and qPCR	(Merryman-Simpson et al. 2008;Wood et al.

				2009b)
Plakophilin-2	PKP2	27	Canine qPCR	(Wood et al. 2009b)
Peroxisome proliferator-activated receptor gamma	PPAR γ	20	Canine qPCR	(Wood et al. 2009b)
Cell adhesion molecule Precursor (P-selectin)	P-selectin	7	Canine qPCR and literature	(de Mora et al. 2007;Wood et al. 2009b)
Tumor necrosis factor Precursor (TNF-alpha)	TNFA_CANFA	12	Human and canine literature	(Grewe et al. 1998;Morar et al. 2006;Nuttall TJ et al. 2002)
Nucleotide-binding oligomerization domain-containing protein 1	NOD1 or CARD4	14	Human Literature	(Morar et al. 2006)
Chymase Precursor (Mast cell protease I)	CMA1	8	Human Literature	(Morar et al. 2006)
Filaggrin	FLG	17	Human Literature	(Morar et al. 2007;Smith et al. 2006)
High affinity immunoglobulin epsilon receptor subunit beta (FcERI)	MS4A2	21	Human Literature	(Morar et al. 2006)
Peroxisome proliferator-activated receptor alpha	PPAR α	10	Human Literature	(Sertznig et al. 2008)
Cornifin-B (Small proline-rich protein IB)	SPRR1B	17	Human literature	(Sugiura et al. 2005)
Toll-like receptor 2 Precursor	TLR2	15	Human literature	(De Benedetto et al. 2009)

Cytokine receptor-like factor 2 Precursor	TSLP receptor or CRLF2	X	Human literature	(Yong-Jun Liu 2006)
Signal transducer and activator of transcription 2	STAT2	10	Human literature and canine microarray	(Chen and Khurana Hershey 2007;Merryman-Simpson et al. 2008)
Dipeptidyl peptidase 4 (T-cell activation antigen CD26)	DPP4	36	Human Literature and canine qPCR	(Reinhold et al. 2007;Wood et al. 2009b)
serine protease inhibitor, Kazal type 5	SPINK5	2	Human Literature and canine qPCR	(Moffatt 2004;Wood et al. 2009b)
S100 calcium binding protein A8	S100A8	7	Human literature, canine microarray and qPCR	(Merryman-Simpson et al. 2008;Roth et al. 2003;Wood et al. 2009b)

Gene	SNP	chr	bp	Type position	Reverse Primer	Forward Primer	Amplicon Length	Probe Sequence
MS4A2	MS4A2_E5	21	53634435	Exon 5	ACGTTGGATGTATGCT GCTGACAGTGTGG	ACGTTGGATGCTATTGAA CGCACTGGCAGG	116	TCTCCAGGC TGCCCCA
SCCA2	rs21902631	1	16476541	Downstream	ACGTTGGATGGTCTGT CTCTCTCCTTCTAC	ACGTTGGATGTATCTTAT GTTGAACATTTG	106	ATCTCCCAGT GATAATTCC
SCCA2	rs21902632	1	16476102	Non synonymous coding	ACGTTGGATGGAATGG TGGATGCCTTCAAC	ACGTTGGATGTAGACACC ACAAGATCCTGC	95	ACGCCTTCAA CACAAAGAG
SCCA2	rs21902676	1	16469324	Upstream	ACGTTGGATGTTGGGC ATCTGTGTGGTTGG	ACGTTGGATGTCAGACAG CTCCCATTTAGG	111	AGAGAGTCTC TGGTACCCAG AGAACCCT
SCCA2	rs21941198	1	16475961	Non synonymous coding	ACGTTGGATGCCTGCC CATTGCTCATATTC	ACGTTGGATGGCTGCAGC TTGAAGATCAAC	97	GCTCGTCCAC TCTATTAG
STAT2	rs22000453	10	3625056	Downstream	ACGTTGGATGTTTCCTCA CTGCTTAGGAGAC	ACGTTGGATGAAAGCGAT TCCTAAATGCCC	99	AGGTCAAGGA TGGTGG
STAT2	rs22000474	10	3625180	Downstream	ACGTTGGATGTCTGCTT AACAGTCTCCTCC	ACGTTGGATGAGGCATAT TAGATTGGCAGG	105	TGGCCACACC CATTCTTC
PPAR- α	rs22009467	10	23005117	Splice site, intronic	ACGTTGGATGAGAGAC CTCACCTCTGCTCC	ACGTTGGATGTCTGATCG TGGCATTCTTCC	92	TGCTCCCGTT CTCTCCCGTG TC
STAT2	rs22029672	10	3631328	Non synonymous coding, splice site	ACGTTGGATGATGCAG GGCTGGGTTTCTAC	ACGTTGGATGAGCAAGA GGAGCTATTCCTG	113	GGGTTTCTAC CACGAAG
IL-4	rs22146864	11	23976639	Upstream	ACGTTGGATGAATATG GTTGCAGGGCCTTC	ACGTTGGATGCTCTCCCT ACTGATTTCCTC	101	GCCTTCCTCA TACACTCAC
TLR2	rs22349092	15	54486859	Downstream	ACGTTGGATGGGCTTT AGTAAGCAAGTCTG	ACGTTGGATGTTGTAGCT AAGTCCGCTCTG	100	AAGGACAGTG CGTTCA
TLR2	rs22349093	15	54485784	Synonymous coding	ACGTTGGATGTCCGAC ACAAGAATGCAAAG	ACGTTGGATGCAGGGAA AATGACTCGAGAT	113	ATAACCCGTT GCATCCC
CARD4	rs22374481	14	46159795	Upstream	ACGTTGGATGTTAGTC CCTCCTTGAGGTAG	ACGTTGGATGGGCAGGGC TCAACAGAAAAG	95	TGAGGTAGGA AAACTCACA
CARD4	rs22374487	14	46159824	Upstream	ACGTTGGATGAACTCA CACATCCCAGAGAG	ACGTTGGATGTCCAGTAA TGACAGTGACAG	115	CCAGAGAGAT CATTTTATAC TTTT

TLR2	rs22410121	15	54484550	Non synonymous coding	ACGTTGGATGTTCTTCT CTGTCCTGTGACC	ACGTTGGATGTCTCACAG CTGCTGTGAGCC	111	GCCGCTCCAG ATCTTTGAAC T
Filaggri n	rs22509067	17	64302067	Non synonymous coding	ACGTTGGATGTGGATC CCGAGTCTCGGTG	ACGTTGGATGATCAAGGA CAGCAGGCCAC	80	GTGGGCAGAG TCAGG
Filaggri n	rs22509070	17	64298685	Non synonymous coding	ACGTTGGATGGCCACG AGCAGTCAGGAGA	ACGTTGGATGTGGCCTGC TGTCTTGATGT	80	TGGACAGAGC CAGACAC
Filaggri n	rs22509072	17	64300322	Non synonymous coding	ACGTTGGATGAGTCAG GATGCCCCACGGGT	ACGTTGGATGAGACAGA GCCAGACACGCAG	82	GTGTGGCCTG CTGTCCTGA
Filaggri n	rs22509074	17	64300703	Non synonymous coding	ACGTTGGATGCCAGGA CAAGCAGACATCAG	ACGTTGGATGACGGGTCG TTAAGAATTCTC	100	GGGAGGAATC CAGCCTGA
Filaggri n	rs22509090	17	64301986	Synonymous coding	ACGTTGGATGTGCTGT CCTTGGCGTGACC	ACGTTGGATGAAGCCGCC ACGAGCAGTCA	80	CCCGAGTGAC CCTGCGTGTC T
Filaggri n	rs22511501	17	64300264	Synonymous coding	ACGTTGGATGATCCTG ACTCTGCCCCACCGA	ACGTTGGATGTGGCGGCT TCCTTGTCTTTC	81	ACTATGCCCA CCGAGACTCG GAATC
Filaggri n	rs22511502	17	64298621	Non synonymous coding	ACGTTGGATGTGGCGG CTTCCTTGTCTTTC	ACGTTGGATGATCCTGAC TCTGCCCCACCGA	81	CTTGTCTTTCC CTAGTGCTGG AT
Filaggri n	rs22511504	17	64301968	Synonymous coding	ACGTTGGATGAGTCAG GATGCCCCACGGGT	ACGTTGGATGAGACAGA GCCAGACACGCAG	82	AACGTGTGGC CTGCTGTCCTT G
Filaggri n	rs22511518	17	64301581	Synonymous coding	ACGTTGGATGCACTGG ATTCCCGGTGTTTG	ACGTTGGATGCATCAGCA GTCACAGGACAG	99	ATTCCCGGTG TTTGCTGTTAC C
Filaggri n	rs22511526	17	64298647	Non synonymous coding	ACGTTGGATGTGGATT CCGAGTCTCGGTG	ACGTTGGATGATCAAGGA CAGCAGGCCAC	80	ATTCCGAGTC TCGGTGGGCA GAGTCAGG
Filaggri n	rs22529256	17	64273415	Synonymous coding	ACGTTGGATGAAGAGT CTGGATCTGGAGAG	ACGTTGGATGTGACCATC TGAGTTGGATTC	120	CGATTCTGGC TCCAATAATT CTTCTAG

Filaggrin	rs22542234	17	64296141	Non synonymous coding	ACGTTGGATGATGACC AGATCCCTGTCTTC	ACGTTGGATGGCACAGAA CCAATCATGGAC	102	CTGTCTTCTCC CAATACCAG
Filaggrin	rs22542260	17	64296760	Synonymous coding	ACGTTGGATGTGACTG AACTGGATTCCCG	ACGTTGGATGCATCAGCA GTCACAGGACAG	106	TTCCCGGTGT TTGCT
Filaggrin	rs22542262	17	64296821	Non synonymous coding	ACGTTGGATGACCAGA TCCATGTCCAGTTC	ACGTTGGATGATGGAGAC TCAGATCTCAGC	118	GA CTGCTGAT GGTGG
Filaggrin	rs22542263	17	64296837	Splice site, intronic	ACGTTGGATGCTGTCCT GTGACTGCTGATG	ACGTTGGATGTGACTCTG CCCATGGAGACT	95	TGCCCTTGTCT GTCTA
Filaggrin	rs22542266	17	64301700	Non synonymous coding	ACGTTGGATGACGGTG CTGAGATCTGAGTC	ACGTTGGATGACACACAG GGTCACAGCAAG	101	CAGGCTGCCA ACGGG
Filaggrin	rs22565034	17	64303134	Non synonymous coding	ACGTTGGATGTGCACT ACTGCCACGTTGTC	ACGTTGGATGCACTAGAC TCAGAGACTCAG	100	TTCTCCTCTGA CTGGACCTGG ACCGTT
Filaggrin	rs22588226	17	64297000	Non synonymous coding	ACGTTGGATGTGGATC CCGAGTCTCGGTG	ACGTTGGATGAGGGTCAC GCCAAGGACAG	88	CCGTATCGGT GGGCAGAGTC AGG
Filaggrin	rs22588227	17	64297022	Non synonymous coding	ACGTTGGATGAGTCAG GATGCCCCACGGGT	ACGTTGGATGAGACAGA GCCAGACACGCAG	82	ACCCCGGGTG GCCTGCTGT
Filaggrin	rs22588228	17	64297028	Non synonymous coding	ACGTTGGATGAGACAG AGCCAGACACGCAG	ACGTTGGATGAGTCAGGA TGCCCACGGGT	82	CGACACGCAG GGTCAC
Filaggrin	rs22599138	17	64303187	Non synonymous coding	ACGTTGGATGGATCCG GTAACAGCAAACAC	ACGTTGGATGCTGAGTCT CTGAGTCTAGTG	103	CCGGGAATCC AGTGTCACTC AGGCCA
SYND1	rs22605727	17	18202904	Downstream	ACGTTGGATGTCCGTA GGAGGTTCTCTGG	ACGTTGGATGTGAGAATG GAATCCTGGCAC	116	GGTTCTCTGG GCCCCACTGCC ACTTC
SOCS3	rs22615134	9	5860501	Downstream	ACGTTGGATGAACCTT GGAACAAAGTCCCC	ACGTTGGATGTCAAGCTG GGTAGGTTAGTG	113	TTCAGCCAGA GTGTCCAGCC GTT
SOCS3	rs22655272	9	5852692	Upstream	ACGTTGGATGAATTGT	ACGTTGGATGCCCATCTG	119	TGCACCGAGC

					AGCCAACGTGCACC	GTTGTTCTAAGG		ACTGA
SOCS3	rs22655274	9	5852893	Upstream	ACGTTGGATGCTAGAC AGCAGTTCAGTCTC	ACGTTGGATGTGGAGAAA GCCAGGCTTGCG	94	TTCAGTCTCC AGTCAGTGGG
SOCS3	rs22655276	9	5853219	Upstream	ACGTTGGATGTCTTCCT CCTAATCTCCTCC	ACGTTGGATGTGATGACC ACCCTAGGACAC	99	CCACCCCAA GCCAG
SOCS3	rs22655278	9	5853780	Upstream	ACGTTGGATGAAATCC ATTCCCCCTCTGC	ACGTTGGATGTCGTTCTT CCTCAGCTTCTC	106	AGACCTCCTC CTTACTAC
SPINK5	rs22807086	2	45212191	Downstream	ACGTTGGATGAATGAT GTAGCAGACAGGAC	ACGTTGGATGAGAGGGA ACTGAACCCATGC	113	ATGATGGAAT AAATCATTAG AACAAAAG
SPINK5	rs22818977	2	45167469	Synonymous coding	ACGTTGGATGGATGGC AGGATGCATGGCAA	ACGTTGGATGAAGCTCCA CTTTACCCATCC	120	GGATGCATGG CAATAAATG
SPINK5	rs22819627	2	45159024	Non synonymous coding	ACGTTGGATGTTCCCAT CTGTGCCACAAAC	ACGTTGGATGCGATGATT TCAGGAAAGGAG	101	ACAAACGGCT GCATT
SPINK5	rs22839343	2	45201929	Synonymous coding	ACGTTGGATGTATACTT CTCCATCAGCACC	ACGTTGGATGAACTTTTCG GCAGTACGTGAG	103	TCCATCAGCA CCACTCAC
SPINK5	rs22871624	2	45133450	Upstream	ACGTTGGATGGCACTG GATTGAGACACTTC	ACGTTGGATGCTGGAGAG AGTTACTTAGAG	104	GGAAGCAACT AGAATTTTAG C
SPINK5	rs22871627	2	45133465	Upstream	ACGTTGGATGAGTCAG GTAGAGACTTACAG	ACGTTGGATGCACTTGCA GATGGAAGCAAC	110	CTCACATCTG GAGAGAGTT
SPINK5	rs22871630	2	45133611	Upstream	ACGTTGGATGGGCAAT AGATTAAGCCCAAG	ACGTTGGATGCTGCAAAG TTATATGTCTGG	119	ACTCCATACC TCTATGTAAA AACTGAAT
SPINK5	rs22871646	2	45133786	Upstream	ACGTTGGATGGACAGG ATACTGAAGGATGG	ACGTTGGATGGTTGTTCT GGGTCATGCTAC	87	TGAAGGATGG AAACTAAATT GATGT
SPINK5	rs22872666	2	45162084	Synonymous coding	ACGTTGGATGCGACCT TATGTGAAGGATGG	ACGTTGGATGTTTCCATC AGAGCCAAGGAC	91	GGATGGAAGA CTTGTATGCA C
SPINK5	rs22874976	2	45176838	Splice site, intronic	ACGTTGGATGCCACAT GCCTCTGACTTTTG	ACGTTGGATGAGACAATC TGAGAACACAGC	85	CTCTGACTTTT GAGGAGTTC
SAA-1	rs22908459	21	43872759	Synonymous coding	ACGTTGGATGCTTGAG GAATGTCCACCATC	ACGTTGGATGAGGATGAA GCTTCTCGTAGG	101	ATGTCCACCA TCTCTGGCTG CTGAC

SAA-1	rs22908463	21	43891974	Synonymous coding	ACGTTGGATGGTCTCCT TTTCATGTGTCCC	ACGTTGGATGAGGACCAG GGAGCACAATAG	98	GGGATGAAGC TTCTCGT
SAA-1	rs22910701	21	43875650	Synonymous coding	ACGTTGGATGGACAAA TACTTCCATGCCCG	ACGTTGGATGTGATCACT TTAGCAGCCACG	99	CCGGGGGAAC TATGA
PPAR γ	rs22931896	20	9213048	Upstream	ACGTTGGATGTCCACTT GGAGAATACTGGG	ACGTTGGATGTGTAATAA AGATGCCCCCAC	102	GGGAATTACT GCCATCTGGT ATAT
PPAR γ	rs22931897	20	9213053	Upstream	ACGTTGGATGTAAAGA TGCCCCCACTTAGG	ACGTTGGATGTCCACTTG GAGAATACTGGG	97	AACTATGCCC CCACTTAGGG CGATAT
INPPL1	rs22935368	21	28904622	Downstream	ACGTTGGATGTTTTCTG CGTGGGTTTAGGG	ACGTTGGATGTTATGGCC CAGATCCTTCTC	101	GAGTGAGGGC GGGCACCTTA ATA
INPPL1	rs22976618	21	28912506	Synonymous coding	ACGTTGGATGATCACC CCATCCCCTTCAC	ACGTTGGATGCTAGACCC TTTGATGTGAAC	99	CTTCACCTCC AGGAAG
MS4A2	rs22986026	21	53629181	Upstream	ACGTTGGATGTCCGGG TCACTCTTGGTAGA	ACGTTGGATGTCGGCCTA ATGTTTAGGAGC	95	AGGCGGGAGC TGCGTGTCCA CACAG
MS4A2	rs22986033	21	53631825	Non synonymous coding	ACGTTGGATGATAGGT AACACAAATTCTG	ACGTTGGATGATATTGAT CACGGAGCAGAC	92	GGTAACACAA ATTCTGATTG CCTTG
MS4A2	rs22986082	21	53634938	Synonymous coding	ACGTTGGATGCCCAGA ATAGTGAGAAACAG	ACGTTGGATGCTGCCTTG TGACTCTACTTG	101	GAATAGTGAG AAACAGGATC AT
POSTN	rs23274229	25	6184255	Synonymous coding	ACGTTGGATGGACTCA GGACCCACAATAAC	ACGTTGGATGACCTCCAT GGATCACTTCAG	119	AGGACCCACA ATAACAAAGG TCAAAAT
PKP2	rs23360288	27	19048662	Synonymous coding	ACGTTGGATGCAGCGT CAGACAGTTCTCCT	ACGTTGGATGAACCCAGC GGTGCTCACGTA	104	TCAGACAGTT CTCCTTCTCCA
ARTS-1	rs23593765	3	15767342	Synonymous coding	ACGTTGGATGCTGCAA GTATCTAAGGCCAC	ACGTTGGATGGGTATTCC AGGACTCTCAAG	99	CAAGGAGAGT TGGAGAGAG
DPP4	rs23907319	36	10444284	Synonymous coding	ACGTTGGATGAGAGCT CAGTTTCCCATTCG	ACGTTGGATGTGAGAGGC ACTGGAGATAAC	107	AAAGTAAGAA AGTTCTACAG GTAGG
Cadherin-13	rs24264853	5	71503539	Downstream	ACGTTGGATGTACCCA AACCCAAGAGGATG	ACGTTGGATGCCTATGCA TATGACTAGCCC	103	GTACCTACAT GTGAGTAATT GCTAA

P-Selectin	rs24435410	7	31958066	Non synonymous coding	ACGTTGGATGGACCAT TGGCCATTCTCTTG	ACGTTGGATGACCTGTAG CTTCCATTGTCC	100	TCTTGGCATG CTGTTCTCGCT GATCC
S100A8	rs24438947	7	46517606	Upstream	ACGTTGGATGTTTTCTCCT ACTTCCCCACCTC	ACGTTGGATGTCTGGTTT CTCAACCAGGTG	99	CACCTCCCAT CAAAATTTTT CATTC
TSLP receptor	rs24613432	X	751050	Splice site, intronic	ACGTTGGATGTAGGTC AAACCACTGATCCC	ACGTTGGATGGCCATCTG TCGTAACATTGC	102	CCGTGCTTGC TTTTTTTCC
TSLP receptor	rs24613434	X	750979	Synonymous coding	ACGTTGGATGAGTCAC TTGGGTATGTGTCTG	ACGTTGGATGGGCTTGGA TGCTGAGAAATG	108	TTTCACTCTG GCCCCG
TSLP receptor	rs24630571	X	745241	Synonymous coding	ACGTTGGATGAGGAGG AAGAGGCCTCTGG	ACGTTGGATGGTGAAGCC ACCAAGAGAGAC	114	AAGGCGGCGG TGGCGG
TSLP receptor	rs24630609	X	745403	Synonymous coding	ACGTTGGATGTGGACG ATGAGAGCCTCTTC	ACGTTGGATGACGTGGTC CCCTGAATAAG	90	TCTTCCGGGC CGCAATC
TSLP receptor	rs24639522	X	755348	Synonymous coding	ACGTTGGATGCCTGGA GGCAAAGAAGATG	ACGTTGGATGGTAAGCAC TGATCCACTGAC	111	TCTGGAATGG AACACGTCT
TSLP receptor	rs24657063	X	755447	Synonymous coding	ACGTTGGATGACCACA GGTTAACCAGTGAC	ACGTTGGATGGCAGTGCG ACTTTGTTGAAG	90	AACCAGTGAC GAGACTGATA GCCAATG
ARTS-1	rs24872954	3	15782443	Synonymous coding	ACGTTGGATGAAAGCC ATCCATCCTCTCTG	ACGTTGGATGTCTGAGAG GGATCATGAGTG	117	TCCATCCTCTC TGTGCCACCT GT
SAA-1	rs8488679	21	43871640	Upstream	ACGTTGGATGTGGCTG CTGGAAGCTTATTG	ACGTTGGATGACTGAGGG ATGCAAAGCCTG	110	CTCCCTCCCG CTCCC
Filaggri n	rs8555036	17	64296436	Synonymous coding	ACGTTGGATGAGTGGA CGATTGCTGGTGG	ACGTTGGATGTGACAGCT CTAGGCACTCAG	111	CTGGTGGCGA GATCC
Filaggri n	rs8555044	17	64298566	Synonymous coding	ACGTTGGATGTGCTGT CCTTGGCGTGACC	ACGTTGGATGAAGCCGCC ACGAGCAGTCA	80	TTGACCTGCT GTGTCT
Filaggri n	rs8555045	17	64298442	Non synonymous coding	ACGTTGGATGTGTCCTT GGCGTGACCTG	ACGTTGGATGATCCAGCA CTAGGGAAAGAC	101	ACCCTGCGTG TCTTG
Filaggri n	rs8555046	17	64298495	Non synonymous coding	ACGTTGGATGTGGCGG CTTCCTTGCTTTT	ACGTTGGATGAGCCTGAC TCTGCCACCGA	81	TCCCTAGTGC TGGAT
CMA1	rs8867188	8	7447456	Upstream	ACGTTGGATGTTTATCC TTGTCAGGTGCGG	ACGTTGGATGTTTAACGT GGAGTGGGATGG	82	CAGGTGCGGT GCTTT

MS4A2	rs8877519	21	53631150	Splice site, synonymous coding	ACGTTGGATGCGGAGA GGTACTTCTGAAAG	ACGTTGGATGGAGAGAAT TTTTCTCAGGGC	100	AGTTCAATTT CAGGTGC
MS4A2	rs8877521	21	53631234	Synonymous coding	ACGTTGGATGAATTCC AGCTCCCTCTTCAG	ACGTTGGATGGTGA AAC CCCTTACTGGAG	102	CTCCCTCTTCA GAAACATCAG CCA
PPAR γ	rs8944179	20	9156340	Synonymous coding	ACGTTGGATGGAATGA CCAAGTAACTCTGC	ACGTTGGATGTATTCATC AAGGACGCCAGC	94	AGTAACTCTG CTAAAATATG G
SCCA2	rs9013509	1	16476584	Downstream	ACGTTGGATGCAAATT TAGTAGAAGATAGAG	ACGTTGGATGTCTCCCAG TGATAATTCCTC	114	AGAAGATAGA GTTTTAA AAT ATTTTTTT
SCCA2	rs9013510	1	16476441	Downstream	ACGTTGGATGGCGAGA AACACAGATTCTCT	ACGTTGGATGGAGACAG ACAGGCTATTTCC	117	TTTTTTTTCT TTCCAATCTC A
MS4A2	rs9019919	21	53636716	Downstream	ACGTTGGATGCACTGA GGACCAATTAGGTG	ACGTTGGATGTCCCACTA TGAGCGAACTG	101	CTGAGGACCA ATTAGGTGTT TGAAATT
INPPL1	rs9070190	21	28910870	Synonymous coding	ACGTTGGATGCGCAGG ATGTCCAGGTAGTT	ACGTTGGATGAGAGACTT CTTCCCTTCCTG	117	GTCCAGGTAG TTCTGGTT
TNF- α	rs9095590	12	4078693	Synonymous coding	ACGTTGGATGTTCTTTC CTCTCCCTCCTC	ACGTTGGATGGCAGAGCT CTTACCTACAAC	103	TTCCCTCCTCA GAATCATCTT CTCGAAC
INPPL1	rs9150577	21	28912392	Synonymous coding	ACGTTGGATGAGATCT GTGAGCTCCTTGAG	ACGTTGGATGATGACATC TATGTCTTCGGG	114	CTCGCGGTCA CCCACCGAGT T
ARTS-1	rs9190946	3	15784064	Synonymous coding	ACGTTGGATGTCTTCTG TCTCCATCAGCAC	ACGTTGGATGGGAAAGCC TTTCTGCAGCGT	105	ATTGATGTAA AAACCATGAT GAA
ARTS-1	rs9196801	3	15767484	Frame shift - coding	ACGTTGGATGCCCCGTA CACAGTTGTCATTG	ACGTTGGATGTTCTTGG TTCTGTAGGTGC	105	AGCGCGGGGC ATTTAAATGG ATTT
S100A8	S100A8_P	7	46517638	Promoter region	ACGTTGGATGCAGTGA TTGCCCCATTACCC	ACGTTGGATGCCTAGGCC ATGCAGAGATAG	103	TTGATTGCC CATTACCTG GTTGA
S100A8	S100A8_p2	7	46517792	Promoter region	ACGTTGGATGCCTGCA TATCCTCTGTCAAC	ACGTTGGATGCAAAGCAT GGAGCAGACTGA	93	ATTTTCGGGAA ACCTGGTAAG TAA

S100A8	S100A8_p3	7	46517859	Promoter region	ACGTTGGATGACCTCC CTTAGCTTTCTCTC	ACGTTGGATGTCAGTCTG CTCCATGCTTTG	106	TCCCTTAGCTT TCTCTCCCCTC T
SPRR1 B	SPRR1B_E3 _1	17	64883377	Exon 3	ACGTTGGATGAGGAAG TTTGGTGTTCACAG	ACGTTGGATGACCTCAGG AGCCATTGTTC	99	TTGCGGTAAC TTGGTGTC