

Genetic Polymorphisms of Transforming Growth Factor Receptors (TGF- β RI, TGF- β RII) and Risk Factors Associated with Keloid Scars in Burkina Faso: A Cross-Sectional Study

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Background: The keloids scar is a skin scarring pathology with a higher frequency in the black population. Several genetic polymorphisms, including those of transforming growth factor receptors, have been identified as probable susceptibility genes. This study aimed to characterize the polymorphisms of *TGF- β RI*, *TGF- β RII* and identify risk factors associated with keloid scars in Burkina Faso.

Methods: This was a cross-sectional study that included 141 subjects with keloids and conducted from April 2021 to April 2022. Genomic DNA was extracted using the “PureLink™ Genomic DNA Mini kit”. Genotyping of TGF- β RI rs111854391, rs121918710, and TGF- β RII rs104893807, rs28934568 polymorphisms was performed using real-time PCR. Results were considered statistically significant at $p < 0.05$.

Results: The average age of the study population was 34 ± 15 years and O Rhesus positive blood group (O+) was predominant (36.88%). All subjects (100%) were heterozygous (TG) for rs121918710. Medical or surgical history, family history, blood type, and rs28934568 polymorphism were related to the number of sites of keloids on the body. Patients with a medical history (9.8%) were carriers of the mutant allele C for rs28934568. Mossi ethnic had more than 8 times risk of developing a keloid scar ($p = 0.030$; OR = 8.66 (95% CI: 1.230–60.902)).

Conclusion: The presence in all the patients of the mutated allele for the rs 121918710 polymorphism could explain the involvement of this mutation in the occurrence of keloid scars. In addition, ethnicity and blood type were the risk factors associated with keloids.

Keywords: keloids, transforming growth factors, polymorphisms, alleles, Burkina Faso

Background

Following a cutaneous lesion, a process of reaction of cicatrization is set up by the human organism with the aim of leading to the closing of the lesion, on the one hand, and the reconstitution of the damaged tissue, on the other hand.¹ When the balance of reparative processes is disturbed, wound healing can be impaired, resulting in pathological scarring, which can be chronic wounds or excessive scar formation ranging from hypertrophic scars to keloids.^{2,3} The keloid scar represents a pathology of skin healing corresponding to an abnormal proliferation of fibrous tissue in the dermis and which can be responsible for a functional and aesthetic embarrassment.^{4–6} The pathophysiological process of keloids is not well known, but they can occur in all populations, and there is a high frequency in the black population with



a prevalence between 4.5% and 16%.⁷ In sub-Saharan Africa, where these pathologies predominate, few studies have been conducted, and the frequencies are only hospitalized, including 2.5% in the Congo,⁸ 1.9% in Togo,⁹ and 0.7% in Benin.¹⁰ In Burkina Faso, the frequency is estimated at 10.2% in schools.¹¹ The insufficiency of these data in our regions makes management difficult despite the fact that keloids are one of the most frequent reasons for dermatological consultation.

Numerous studies have established a significant genetic predisposition to the development of keloids. Many candidate genes are targeted, notably certain Human Leucocyte Antigens (HLA) polymorphisms and transforming growth factors β (*TGF- β*) and their receptors (*TGF- β RI*, *TGF- β RII*),¹² intervening in signal transduction through different pathways. *TGF- β* is widely present in various tissues and cells, with diverse biological functions including regulating cell proliferation, differentiation, migration, apoptosis, and extracellular matrix (ECM) synthesis, and degradation.¹³ In keloids, *TGF- β* plays a key role in tissue development, renewal, and repair, and appears to be involved in the pathogenesis compared to other genes.^{14,15} Existing literature lacks data on whether the *TGF- β RI* and *TGF- β RII* polymorphisms observed in other regions have the same functional impact on receptor-ligand binding affinity and downstream signaling within the specific genetic background of African patients.

We aimed to characterize the polymorphisms of transforming growth factor receptors (*TGF- β RI*, *TGF- β RII*) and risk factors associated with the occurrence of keloids in patients from Burkina Faso.

Materials and Methods

Study Setting

This was a cross-sectional study that took place from April 2021 to April 2022 in Ouagadougou, Burkina Faso. The patients were recruited in the dermatology departments of three (3) health centers, including the Yalgado Ouedraogo University Hospital (CHU-YO), the Bogodogo University Hospital (CHU-B), and the Medical Center of the General Camp Aboubacar Sangoulé Lamizana (CMCGASL). The Pietro Annigoni Biomolecular Research Center (CERBA) and the Laboratory of Molecular Biology and Genetics (LABIOGENE) were used for biological and genetic analyses.

Recruitment of Patients

A total 141 patients with keloids were recruited. The sample size was calculated using Schwartz formula and a prevalence value of 10.2% reported by Traore et al in 2019. Any adult patient of both sexes, regardless of age, ethnicity, or religion, confirmed with keloids by the dermatologist and who freely agreed to participate in the study was included in this study.

A case is a patient presenting an abnormal proliferation of scar tissue that forms at the site of cutaneous injury (eg, on the site of a surgical incision or trauma); and that does not regress and grows beyond the original margins of the scar. Exclusion criteria were as follows: any patient with a skin pathology other than keloids; anyone without keloid scars; or any patient who was eligible but refused to give consent to participate in the study. Dermatologists were in charge of the recruitment of participants.

Biological Samples Collection

Whole blood sample was collected in patients included in a tube impregnated with ethylene diamine tetra-acetic acid (EDTA). The samples were then sent to CERBA for processing. The samples were used for blood/rhesus grouping of the patients, using the Beth Vincent technique, then centrifuged at 3500 rpm for 15 min, aliquoted in cryotubes, and stored at -80°C for molecular analyses.

Detection of Transforming Growth Factor Receptor Polymorphisms *TGF- β RI* (*rs111854391*, *rs121918710*) and *TGF- β RII* (*rs104893807*, *rs28934568*)

Genomic DNA was extracted from whole blood using the “PureLink™ Invitrogen Genomic DNA Minikit” kit (ThermoFisher, USA). Genotyping of polymorphisms was performed using real-time PCR with a 10 μL reaction mixture consisting of 2 μL DNA, 5.3 μL sterile water, 2.5 μL TaqMan® Probe Universal qPCR Mix 5x, and 0.2 μL SNP mix

(40x). The PCR program consisted of an initial denaturation step and Taq polymerase activation at 95°C for 10 minutes; followed by 35 cycles of denaturation at 92°C for 15 seconds, hybridization and elongation at 60°C for 1 minute.

Ethical Considerations

Our study obtained the approval of the Health Research Ethics Committee (CERS) of the Ministry of Health (Deliberation n°2021-03-058) and the authorization of the health structures of each site for the recruitment of patients. This study complies with the Declaration of Helsinki. All participants received information and gave their signed consent form to participate in the study. For those who were not able to read, they received information in their local language before giving their consent. A parent or legal guardian of participants under 18 years of age provided informed consent. Anonymity and confidentiality were respected for all patients.

Statistical Analysis

Data were entered using Excel 2016 software. Data were analyzed using Statistical Package for the Social Sciences (SPSS) 21.0 software. Allelic discrimination was performed using TaqMan Genotyper® 1.6.0 software to determine the genotype of each polymorphism. Categorical variables were compared using the Pearson's Chi-square test where cell counts were less than five. To identify independent risk factors for keloid occurrence, variables were entered into a Multivariate Logistic Regression model. Odds ratios and 95% confidence intervals were calculated to assess risk. Results were considered statistically significant for $p < 0.05$.

Results

Socio-Demographic Characteristics

The distribution of the subjects according to sex shows a predominance of the female sex, representing 73.1% of the subjects present at the time of the study with a statistically significant difference ($p < 0.001$). The average age of the study population was 34 ± 15 years with a predominance of the young age group 19 to 29 years (36.2%) ($p = 0.154$). Employees (32.6%), single persons (48.94%), and subjects with higher education (35.5%) were in the majority. The most represented ethnic group was Mossi (59.6%), followed by subjects with parents of different ethnicities (cultural mixing) (16.3%) with a statistically significant difference ($p < 0.001$). Subjects with a medical or surgical history (73.3%) and family history (48.9%) were the most represented with significant differences, respectively, $p < 0.001$ and $p = 0.018$ (Table 1).

Table 1 Sociodemographic Characteristics

| | Number (n=141) | Percentage (%) | p-value |
|-----------------|----------------|----------------|---------|
| Sex | | | |
| Male | 38 | 26.9 | <0.001 |
| Female | 103 | 73.1 | |
| Age (years) | | | |
| < 18 | 13 | 9.2 | 0.154 |
| 19–29 | 51 | 36.2 | |
| 30–40 | 34 | 24.1 | |
| 41–60 | 32 | 22.7 | |
| Plus de 60 | 11 | 7.8 | |
| Profession | | | |
| Pupils/Students | 43 | 30.5 | 0.229 |
| Unemployed | 30 | 21.3 | |
| Informal sector | 17 | 12.1 | |
| Employed | 46 | 32.6 | |
| Retired | 5 | 3.5 | |

(Continued)

Table 1 (Continued).

| | Number (n=141) | Percentage (%) | p-value |
|-----------------------------|----------------|----------------|---------|
| Marital status | | | |
| Single | 69 | 48.94 | 0.154 |
| Married | 68 | 48.23 | |
| Widow/Widower | 4 | 2.84 | |
| Education | | | |
| None | 23 | 16.3 | 0.135 |
| Primary | 22 | 15.6 | |
| Secondary | 46 | 32.6 | |
| Higher education | 50 | 35.5 | |
| Medical or surgical history | | | |
| Yes | 102 | 73.3 | <0.001 |
| No | 39 | 27.7 | |
| Family history | | | |
| Yes | 69 | 48.9 | 0.018 |
| No | 33 | 23.4 | |
| NR | 39 | 27.7 | |
| Ethnic | | | |
| Bissa | 8 | 5.7 | <0.001 |
| Gourounsi | 10 | 7.1 | |
| Mossi | 84 | 59.6 | |
| Cultural mixing | 23 | 16.3 | |
| Others | 16 | 11.3 | |

Abbreviation: NR, No responding.

Frequency of Blood Group and Different Polymorphisms of $TGF\beta$ RI and $TGF\beta$ RII

The grouping according to the type of blood group and Rhesus factor shows a predominance of blood group O (39.01%). The genetic analyses showed results according to the different polymorphisms as follows: Concerning $TGF\beta$ RI rs121918710, all patients (100%) were GT heterozygous, indicating that all had the G mutated allele. Concerning $TGF\beta$ RII rs28934568 68.79% were homozygous wild-type versus 31.21% who were heterozygous and therefore carriers of the C mutated allele (Table 2).

Table 2 Frequency of Blood Grouping and Different Genetic Polymorphisms

| | Number (n=141) | Percentage (%) |
|------------------------------|----------------|----------------|
| Blood grouping | | |
| A | 32 | 22.68 |
| AB | 2 | 1.41 |
| B | 52 | 36.88 |
| O | 55 | 39.01 |
| $TGF\beta$ RI rs111854391 | | |
| CC | 140 | 99.29 |
| CT | 1 | 0.71 |
| TT | 00 | 0.0 |

(Continued)

Table 2 (Continued).

| | Number (n=141) | Percentage (%) |
|--------------------------------------|----------------|----------------|
| <i>rs121918710</i> | | |
| TG | 141 | 100 |
| GG | 00 | 0.0 |
| TT | 00 | 0.0 |
| <i>TGFβRII</i> <i>rs104893807</i> | | |
| CC | 140 | 99.29 |
| CT | 1 | 0.71 |
| TT | 00 | 0.0 |
| <i>rs28934568</i> | | |
| TT | 97 | 68.79 |
| TC | 44 | 31.21 |
| CC | 00 | 0.0 |

Abbreviations: *TGFβRI*, transforming growth factors β receptor I; *TGFβRII*, transforming growth factors β receptor II.

Association Between Parameters and the Number of Keloid Location Sites

Bivariate analysis showed that age, occupation, education, ethnicity, medical or surgical history, family history, blood type, and rs28934568 polymorphism were associated with the number of sites of keloids on the body ($p < 0.001$). We note that 31.1% of Mossi patients had at least 3 sites of keloid localization on the body. Then, 41.1% of the patients who presented medical or surgical history had at least 3 keloid sites on the body versus 16.3% ($p < 0.001$) and 33.3% ($p < 0.001$) of the patients who presented familial history had at least 3 keloid sites on the body versus 23.5% (Table 3).

Table 3 Association Between Parameters and Number of Keloid Sites

| | Number of Location Sites of Keloids n (%) | | | Chi ² | p-value |
|-----------------|---|-----------|-----------|------------------|---------|
| | 1 | 2 | ≥ 3 | | |
| Age (years) | | | | 10.83 | <0.001 |
| < 18 | 1 (0.7) | 1 (0.7) | 11 (7.8) | | |
| 19–29 | 7 (5) | 18 (12.8) | 26 (18.4) | | |
| 30–40 | 3 (2.1) | 10 (7.1) | 21 (14.9) | | |
| 41–60 | 8 (5.7) | 6 (4.3) | 18 (12.8) | | |
| More than 60 | 3 (2.1) | 3 (2.1) | 5 (3.6) | | |
| Profession | | | | 13.23 | <0.001 |
| Pupils/Students | 3 (2.1) | 13 (9.2) | 27 (19.2) | | |
| Retired | 0 (0.0) | 1 (0.7) | 4 (2.8) | | |
| Employed | 6 (4.3) | 10 (7.1) | 30 (21.3) | | |
| Unemployed | 8 (5.7) | 11 (7.8) | 11 (7.8) | | |
| Informal sector | 5 (3.6) | 3 (2.1) | 9 (6.4) | | |
| Education | | | | 22.77 | 0.001 |
| None | 6 (4.3) | 7 (5.0) | 10 (7.1) | | |
| Primary | 8 (5.7) | 2 (1.4) | 12 (8.5) | | |
| Secondary | 6 (4.3) | 8 (5.7) | 32 (22.7) | | |
| Higher | 2 (1.4) | 21 (14.9) | 27 (19.2) | | |

(Continued)

Table 3 (Continued).

| | Number of Location Sites of Keloids n (%) | | | Chi ² | p-value |
|-----------------------------|---|------------|-----------|------------------|---------|
| | 1 | 2 | ≥ 3 | | |
| Marital status | | | | | |
| Single | 6 (4.3) | 18 (12.8) | 45 (31.9) | 8.15 | 0.086 |
| Married | 14 (9.9) | 19 (13.5) | 35 (24.8) | | |
| Widow/widower | 2 (1.4) | 1 (0.7) | 1 (0.7) | | |
| Ethnic groups | | | | | |
| Mossi | 16 (11.3) | 23 (16.3) | 45 (31.1) | 0.18 | <0.001 |
| Bissa | 1 (0.7) | 4 (2.8) | 3 (2.1) | | |
| Gourounsi | 1 (0.7) | 3 (2.1) | 6 (4.3) | | |
| Cultural mixing | 0 (0.0) | 6 (4.3) | 17 (12.1) | | |
| Others | 4 (2.8) | 2 (1.4) | 10 (7.1) | | |
| Surgical or medical history | | | | | |
| No | 4 (2.8) | 12 (8.5) | 23 (16.3) | 1.30 | <0.001 |
| Yes | 18 (12.8) | 26 (18.4) | 58 (41.1) | | |
| Family history | | | | | |
| No | 4 (3.9) | 5 (4.9) | 24 (23.5) | 5.05 | 0.001 |
| Yes | 14 (13.7) | 21 (20.6) | 34 (33.3) | | |
| Blood grouping | | | | | |
| A | 6 (4.3) | 8 (5.7) | 18 (12.7) | 1.01 | <0.001 |
| AB | 0 (0.0) | 1 (0.7) | 1 (0.7) | | |
| B | 7 (5.0) | 16 (11.3) | 29 (20.6) | | |
| O | 9 (6.4) | 13 (9.2) | 33 (23.4) | | |
| TGFβRI rs111854391 | | | | | |
| CC | 22 (15.6) | 38 (26.9) | 80 (56.7) | 0.74 | 0.689 |
| CT | 0 (0.0) | 0 (0.0) | 1 (0.7) | | |
| TT | 0 (0.0) | 0 (0.0) | 0 (0.0) | | |
| TGFβRII rs104893807 | | | | | |
| CC | 21 (14.9) | 38 (26.9) | 81 (57.4) | 5.44 | 0.066 |
| CT | 1 (0.7) | 0 (0.0) | 0 (0.0) | | |
| TT | 0 (0.0) | 0 (0.0) | 0 (0.0) | | |
| TGFβRII rs28934568 | | | | | |
| TT | 18 (12.8) | 27 (19.15) | 52 (36.9) | 2.62 | <0.001 |
| TC | 4 (2.8) | 11 (7.8) | 29 (20.6) | | |
| CC | 0 (0.0) | 0 (0.0) | 0 (0.0) | | |

Abbreviations: OR, odds ratio; TGFβRI, transforming growth factors β receptor I; TGFβRII, transforming growth factors β receptor II.

Risk Factors Associated with Keloids

After univariate and multivariate logistic regression analysis, we found that only Mossi ethnicity was strongly associated with the occurrence of keloids [$p=0.030$; OR=8.66 (95% CI: 1.230–60.902)]. Mossi had more than 8 times the risk of developing a keloid [$p=0.030$; OR=8.66 (95% CI: 1.230–60.902)] (Table 4). Sex, education, occupation, marital status, and family history were not associated with keloids.

Table 4 Multivariate Logistic Regression Analysis of the Risk Factors Associated with Keloids

| | p-value | OR | 95% CI |
|------------------|---------|-------|--------------|
| Sex | | | |
| Male | - | - | - |
| Female | 0.383 | 0.47 | 0.089–2.528 |
| Profession | | | |
| Pupils/Students | 0.135 | 5.92 | 0.575–60.97 |
| Retired | 1 | - | - |
| Employed | 0.620 | 1.66 | 0.225–12.216 |
| Unemployed | 0.566 | 1.75 | 0.261–11.699 |
| Informal sector | - | - | - |
| Education | | | |
| None | - | - | - |
| Primary | 0.055 | 0.17 | 0.029–1.040 |
| Secondary | 0.964 | 0.95 | 0.163–5.635 |
| Higher education | 0.419 | 2.54 | 0.263–24.627 |
| Marital status | | | |
| Single | - | - | - |
| Married | 0.733 | 1.28 | 0.306–5.371 |
| Widower | - | - | - |
| Ethnic Groups | | | |
| Mossi | 0.030 | 8.66 | 1.230–60.902 |
| Bissa | - | - | - |
| Gourounsi | 0.087 | 13.84 | 0.683–280.51 |
| Cultural mixing | - | - | - |
| Others | 0.555 | 2.08 | 0.181–23.93 |
| Family history | | | |
| No | - | - | - |
| Yes | 0.418 | 0.54 | 0.119–2.411 |

Abbreviations: OR, Odds Ratio; 95% CI, 95% confidence interval.

Discussion

Our study is the first in Burkina Faso having treated the polymorphism of the receptors of transforming growth factors beta (*TGFβRI* and *TGFβRII*) with keloids. It allowed us to characterize four (4) polymorphisms including rs111854391, rs121918710 of *TGFβRI* gene and rs104893807, rs28934568 of *TGFβRII* gene. The mean age of the study population was relatively young (34 ± 15 years) with a predominance of the 19 to 29 age group (36.2%) and had more than 3 keloid sites on their bodies. Similar results were found in other studies (31.3 ± 13 years) in Togo¹⁶ and Cameroon (≥ 25 years).¹⁷ Young people seem to be more at risk of developing a keloid and this is because they are more exposed to various skin traumas, leading to lesions that can result in keloids, and also due to the fact that skin tension at these ages is at its maximum.¹⁸ Indeed, skin tension is directly related to the quantity of elastic and collagen fibers in the dermis; a recurrence of trauma would solicit more elastic fibers and therefore lead to the accumulation of extracellular matrix (ECM) constituents and consequently contribute to the occurrence of keloids. The Mossi ethnic group was in the majority. This predominance reflects the ethnic distribution of the population of Burkina Faso, of which 40% are Mossi. We also observed that 16.3% of the study population had parents of different ethnicities. This indicates the existence of cohesion and inclusion of all ethnic groups in Burkina Faso, which represents a cultural wealth for the country. The predominance of patients with a family history of keloids (48.9%) corroborates the existence of a genetic predisposition

to the occurrence of keloids.^{19,20} In contrast, family history was found in only 22.4% of a study population of students.¹¹ Results from other countries show much lower rates of family history including 2.3% in the Democratic Republic of Congo²¹ and 21.9% in Togo.²² This predominance has also been observed in studies conducted in Nigeria and Benin with frequencies of 26.5% and 34.53%, respectively.^{17,23} Indeed, the pre-sternal location is still today the most associated with spontaneous keloid.²⁴ The patients in our study were predominantly of blood group O. This predominance was also reported by Mouhari-Touré and al. in 2012 and Nangole et al in 2021 in Kenya.^{22,25} On the other hand, blood group A predominance has been reported in South India²⁶ and Syria.²⁷ The study conducted in Syria also showed a significant association between blood type A and keloid occurrence. This could be justified by the fact that blood group antigens would facilitate intracellular uptake signal transduction or adhesion through the organization of membrane microdomains.²⁸ Mossi ethnicity, medical or surgical history, and familial history were associated with the number of keloids location. Most of them presented at least 3 keloids location in their body. A study also reported large areas of the body affected by keloids in patients²⁹ suggesting the keloid formation in most part of the body. Keloid formation mainly occurs in parts of the body with high concentrations of melanocytes and is rare on the soles of the feet and palms of the hands where melanocyte concentration is minimal.³⁰

Genotyping of the different variants of the study genes revealed some peculiarities. The entire study population was heterozygotes (TG) so presented the mutated allele (G) for the *TGFβ1* rs121918710 polymorphism. This result corroborates that of Loeys et al in 2005 who reported that patients with Marfan syndrome, a disorder of the extracellular matrix (ECM), were heterozygous and that the mutated allele was absent in family members without the disease. This missense mutation could be responsible for the disruption of extracellular matrix homeostasis due to abnormal signaling through these receptors.³¹ This is evidenced by other results obtained by Loeys et al, who found in heterozygous patients diagnosed with Marfan syndrome an accumulation of connective tissue collagen, which is also characteristic of keloid tissue.³² Furthermore, the identification of 100% heterozygosity for *TGFβ1* rs121918710 may be a population-specific variant and have implications for autosomal dominant inheritance. Indeed, a study reported that the pattern of inheritance in families was consistent with an autosomal dominant mode with incomplete clinical penetrance and variable expression.²⁹ This could explain why, in our findings, family history was not associated with keloid occurrence. In contrast, for the *TGFβRI* rs111854391 and *TGFβRII* rs104893807 polymorphisms, only 0.71% of the study subjects (ie 1/141) carried mutated alleles. These polymorphisms might not be related to the occurrence of keloids. For the *TGFβRII* rs28934568 polymorphism, 31.21% of the study population was heterozygous (TC). Interestingly, our findings show the absence of mutated homozygotes genotypes across all four studied SNPs. This finding is consistent with previous literature reporting a low frequency of the mutated allele in similar cohorts.¹⁴ The paucity of mutated homozygotes suggests that the investigated TGF-β polymorphisms may exert their influence primarily through a heterozygous effect or that the mutated allele is subject to strong evolutionary constraints in this population, precluding the high-frequency emergence of homozygous genotypes. This is consistent with previous studies on the presence of this polymorphism in populations with extracellular matrix disorders such as Marfan syndrome.^{33,34} Note that the polymorphisms in our study are all located in the kinase zone of the beta growth factor receptors, which are proteins with kinase activity. Therefore, any qualitative modification could have an impact on the functions of these growth factors. The association of the rs28934568 polymorphism, age, profession, ethnicity, medical or surgical history, and family history with the number of keloids explains the plurality of factors favoring the occurrence of keloids.

Analysis of the risks of keloid occurrence showed that only Mossi ethnicity had more than 8 times the risk of developing keloids ($p=0.030$; OR=8.66 (95% CI: 1.230–60.902)). This result differs from that of Kouotou et al in Cameroon in 2019, who reported that patients with familial history had more than 4 times the risk of developing keloids.¹⁷ This difference could be due to the fact that the parameter “ethnicity” was not considered in the study population in Cameroon. Thus, the lack of association between family history and the occurrence of keloids in our study would justify the “incomplete clinical penetrance and variable expression” character attributed to the mode of inheritance proposed for keloids.^{35,36} Further large-scale studies would therefore be required to test this hypothesis. Also, conducting a study with a control population would allow for better comparisons and explanation of our results. Our study is limited to the hospital level and might not perfectly represent the entire population of Burkina Faso. In addition, the data

collected only from people with keloids do not allow us to truly associate the polymorphisms and risk factors taken into account in this study with the occurrence of keloids.

Conclusion

Our study characterized for the first time in Burkina Faso the polymorphisms of the *TGFβRI* gene and the *TGFβRII* gene that appear to be involved in the occurrence of keloid scars. All patients in our study carried the T mutated allele of the rs12191870 polymorphism of the *TGFβRI* gene and the Mossi ethnic group was at the highest risk of developing keloid scarring in Burkina Faso. Further studies with a larger population and including control group would be important to better understand the implications of these polymorphisms.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no competing interest in this work.

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