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Céline Viennet · Jacqueline Bride · Vincent Armbruster
François Aubin · Anne-Claude Gabiot · Tijani Gharbi
Philippe Humbert

Contractile forces generated by striae distensae fibroblasts embedded in collagen lattices

Abstract Striae distensae are characterized by linear, smooth bands of atrophic-appearing skin that are reddish at first and finally white. They are due to stretching of the skin, as in rapid weight gain, or mechanical stress, as in weight lifting. The pathogenesis of striae distensae is unknown but probably relates to changes in the fibroblast phenotype. In order to characterize striae distensae fibroblasts, alpha-smooth muscle actin expression and contractile forces were studied. Five healthy women with early erythematous striae and five healthy women with older striae were selected. Paired biopsies were taken from the center of lesional striae and adjacent normal skin. Fibroblasts were obtained by an explant technique and expanded in vitro in Dulbecco's modified Eagle's medium. Contractile forces generated by fibroblasts in collagen lattices were measured with the Glasbox device developed in our laboratory. Alpha-smooth muscle actin expression was studied by immunofluorescence labeling of cells and by flow cytometry. Fibroblasts from early striae distensae were the richest cells in alpha-smooth muscle actin filaments and generated the highest contractile forces. Their peak contractile force was 26% greater than normal fibroblasts. There was a 150% higher level of alpha-smooth muscle actin content in fibroblasts from early striae distensae compared with fibroblasts from normal skin. In contrast, there was no significant difference in force generation between old striae fibroblasts and normal fibroblasts with cells expressing no alpha-smooth muscle actin. The contractile properties of fibroblasts from striae distensae varies depending on the stage of the disease. In early striae distensae, fibroblasts acquire a more contractile phenotype, corresponding to that of myofibroblasts.

Keywords Actin · Contractile force · Fibroblast · Striae distensae

C. Viennet · J. Bride · A.-C. Gabiot · P. Humbert
Engineering and Cutaneous Biology Laboratory,
IFR 133, School of Medicine and Pharmacy,
25000 Besançon, France

V. Armbruster · T. Gharbi
Optical Laboratory, School of Science,
25000 Besançon, France

F. Aubin · P. Humbert (✉)
Department of Dermatology, Engineering and Cutaneous
Biology Laboratory, St Jacques Hospital, 2 pl St Jacques,
25030 Besançon, France
E-mail: philippe.humbert@univ-fcomte.fr
Tel.: +33-3-81219176
Fax: +33-3-81218279

Introduction

Striae distensae (SD) are cutaneous lesions associated with continuous and progressive mechanical stretching of the skin occuring frequently in weight gain during puberty and early adult life [21, 29]. Clinically, the linear lesions evolve with time, passing through an initial red color (red striae distensae, RSD) to a typical white stretch mark (white striae distensae, WSD) [30]. They are a common cause of consultation for dermatologists but have somehow rarely been the subject of research in recent years. Their mechanism of development remains still disputed. Exogenous or endogenous factors have been suggested (hormonal troubles, obesity, traction force...). However, SD sometimes indicate a pathological condition of the connective tissue. In the light of morphological observations and molecular data, SD appear correlated with loss of synthetic capacity of fibroblasts and strong alteration of connective tissue structure. Collagen, elastin and fibrillin fibers are significantly reduced in SD compared with normal skin (NS) [20, 36, 38]. But each dermatologist is aware of the fact that striae distensae, which may be important and unaesthetic, can occur in persons without any trouble of the collagen or without any extrinsic or intrinsic aggravating factor. Hence we suggested that the occurrence of striae distensae might be associated with the particular characteristics of the fibroblasts.

Besides their primarily role in extracellular matrix production, fibroblasts are capable of contraction, especially in granulation tissue of wounds [11, 13, 25]. Previous studies have found that granulation tissue fibroblasts, known as myofibroblasts, exhibit some features of smooth muscle cells such as alpha-smooth muscle actin (αSM actin) filament bundles, suggested that they might produce the force of wound contraction [7, 16, 27]. An in vitro model that is considered to be an equivalent of the process of wound contraction has been established. Bell reported that the incorporation of fibroblasts in a collagen gel induces a progressive contraction of the gel, resulting in the formation of a dense collagen disc, called retracted lattice [2]. Contraction is determined by measurements of gel diameter over time. We have developed a new device, the GlaSbox, which can measure directly the forces exerted by fibroblasts on the collagen gel [32]. The culture model used is a tense lattice in which cells are seeded within a collagen matrix immobilized at its extremities. Contraction is inhibited and lattice formation is associated with the production of internal tension. Such tense lattices are more closely related to skin than retracted lattices in terms of both internal structure and mechanical behavior, because skin is also permanently under tension. Several force measurement devices are described in the literature [5, 6, 9, 10, 17, 19]. Different cell sources have been studied to test the idea that the ability of fibroblasts to generate contractile forces varying between populations [8, 12, 24, 32]. The purpose of this study was to analyze the contractile activity of different populations of fibroblast derived from SD and compare it with fibroblasts of NS. We measured cellular contractile forces, and we examined the expression of αSM actin filaments in cell populations.

Materials and methods

Patients

Five women, between 12 and 22 years of age, with early erythematous SD and five other women, between 27 and 41 years of age, with old SD were enrolled in this study (Table 1). All were in good health. Striae distensae were associated with obesity.

Table 1 Patient characteristics

	Early erythematous striae distensae	Old striae distensae
Number	5	5
Sex of the patients	F	F
BMI of the patients	> 30	> 30
Biopsied body site	Abdomen, thigh	Abdomen, thigh
Age of the striae distensae	< 6 months	> 9 months

Fibroblasts isolation and culture

For each patient, paired 2-mm punch biopsies were taken under local anesthesia from the center of the long axis of SD, and adjacent NS. Each explant was placed in 25-cm^2 flasks and allowed to adhere to the plastic surface for approximately 5 min at 37°C. To each flask were then added 3 ml of Dulbecco's modified Eagle's medium (DMEM) supplemented with 10% fetal calf serum (FCS) and 1% gentamycin (10 mg/ml) (Gibco, Cergy-Pontoise, France). Fibroblast cultures were then incubated at 37°C in 5% CO_2 and 95% air. Subcultures were established by trypsination (trypsin-EDTA (1 X)) (Gibco, Cergy-Pontoise, France) of explant cultures after 3–4 weeks of outgrowth. Culture medium was changed three times a week. Fibroblasts from subculture 3 to 8 were used in this study. According to Burgess, the fibroblasts cultivated in passages lower than 8 preserve their characteristics of in vivo. Beyond, they adapt to the conditions of in vitro [4].

GlaSbox device (growing lattice study box)

The cell chamber was composed of 8 rectangular culture wells (26×33 mm) in which lattices developed (see Fig. 1a, b). Two opposite silicon beams (200 μm thick, 20 mm wide and 45 mm high) hanged down into each well at a distance of 27 mm apart. The lattice was attached to this sensor through a grid directly etched on the lower part of the beams [1]. A strain gauge of 4.5 kΩ resistance was deposited at the beam surface by phase vapor deposition (PVD) etched by photolithography method, and connected to form a Wheastone bridge. The strain gauges signal output was amplified, then converted and collected by a computer, which included an acquisition card and a specific program for giving directly the forces in real time. The GlaSbox was calibrated against weights, which ranged from 0 g to 1.5 g. Calibration showed the high linearity of the system and sensitivity of 0.1 mN. The whole cell chamber was sterilized at 120°C for 20 min.

Lattice preparation and measurement of contractile forces

Fibroblasts were suspended just before confluence with a trypsin-EDTA (1 X) solution. A sample was taken to determine the number of cells obtained. The suspension was centrifuged and a final count was performed before bringing the fibroblasts to the concentration of 8×10^5 C/ml. Fibroblasts were embedded three-dimensionally in hydrated collagen gels using a modified version of the technique developed by Bell [2]. Briefly, 2 ml of 1.76 X concentrated DMEM medium (with gentamycin, 0.1 N NaOH and $NaHCO_3$) were added to 1 ml of a solution of type I rat tail collagen (2 mg/ml) (Jacques Boy, Reims, France), and to 0.33 ml of the
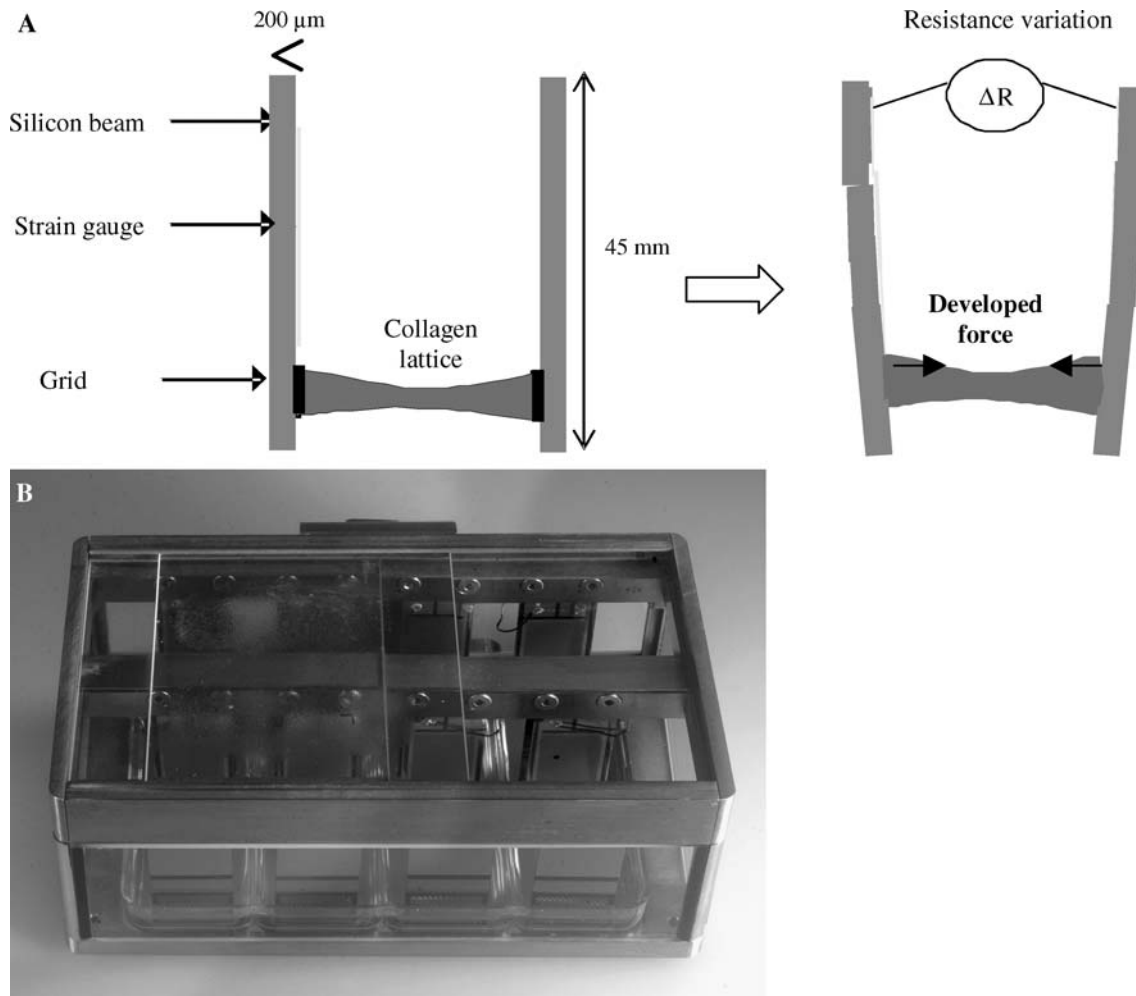
Fig. 1 a Schematic diagram of the GlaSbox. **b** View of the GlaSbox. The cell chamber was composed of eight rectangular culture wells in which lattices developed. Two opposite silicon beams hanged down into each well. The lattice was attached to this sensor through a grid directly etched on the lower part of the beams. A strain gauge was deposited at the beam surface and connected to form a Wheastone bridge. The strain gauges signal output was amplified, then converted and collected by a computer, which included an acquisition card and a specific program for giving directly the forces in real time

suspension of fibroblasts in culture medium. The lattice mixture was poured into a rectangular culture well of the GlaSbox and polymerized in less than 5 min at 37°C. Two milliliters of culture medium were added. The GlaSbox was then placed into an humidified incubator at 37°C, and force measurements were started immediately.

αSM actin immunostaining

Fluorescence microscopy (and labeling of nuclei)

Lattices used in the force measurements and aged 2 days, were fixed under tension in the culture well in 3% PFA in PBS for 30 min. After rinsing in PBS, square-shaped pieces of about 4×4 mm were punched out of the lattice in the area around the center. The fibroblasts in the lattice were permeabilized by the treatment with 0,4 % Triton X100 for 15 min followed by the three changes of PBS. Nonspecific binding sites were blocked at room temperature by a 10 min incubation in 1% glycine in PBS followed by 1 h incubation in 3% BSA solution in PBS containing 10% normal goat serum and 0,1 % Triton X100. Then, samples were incubated overnight at room temperature in a moist chamber with mouse anti-αSM actin monoclonal antibody (diluted at 1:100 in PBS containing 1% BSA and 0.1% Triton) (Sigma, Saint Quentin Fallavier, France). For control, a piece removed of the lattice was incubated in PBS instead of the primary antibody. Excess antibody was removed in the three changes of 10 min each of PBS + 1 % BSA + 0.1 % Triton X 100. The rhodamine-labeled goat anti-mouse IgG polyclonal antibody (diluted at 1:40 in PBS containing 1% BSA and 0.1% Triton) (Sigma, Saint Quentin Fallavier, France) was applied for 1 h at room temperature in a moist chamber. After washing in the three changes of 10 min each of PBS, the lattice fragments were incubated with 2.5 µg/ml Hoechst 33258 (Sigma, Saint Quentin Fallavier, France) in PBS for 10 min in the dark at room temperature.

After washing again, they were gently carried onto an histological slide and coverslipped with Vectashield mounting medium (Biosys, Compiegne, France). Stained preparations were examined under fluorescence microscopy (Olympus IX50).

Flow cytometry

Lattices used in the force measurements and aged 2 days, were digested by collagenase type I from Clostridium Histolyticum (0.150 U/mg) at 37°C for 1 h. Cell suspensions were fixed in 3% PFA in PBS for 10 min, permeabilized in 0,4 % Triton X100 for 15 min and stained for αSM actin as previously described. Isotype controls included cells that incubated only with the secondary antibody. Samples were analyzed on a FACScan flow cytometer (Beckman Coulter) with 488 nm excitation and 610-nm band pass filter for rhodamine. For all flow cytometry analyses, 20,000 cells were analyzed.

Statistical analysis

The data are expressed as mean ± standard deviation (SD). They were compared using the nonparametric Mann–Witney test. A P value less than 0.05 was considered significant.

Results

Force generation

We used a device known as the GlaSbox to measure contractile forces in tense collagen lattices. Force measurements were recorded continuously for 2 days.
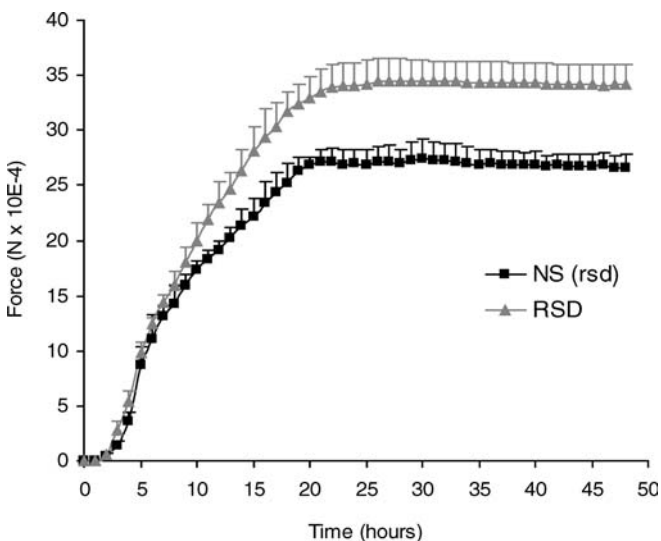
The profiles of contractile force generated by NS and RSD fibroblasts (Fig. 2) and NS and WSD fibroblasts (Fig. 3) were broken down into three phases. The first phase occurred within 2 h of the start of the measurement and did not consist of generated contractile force . The second phase, seen between 2 h and approximately 22 h, represented a near linear increase in force. In comparison with NS fibroblasts, the force generation with RSD fibroblasts was much enhanced . There was no significant difference between the two groups until the 11th hour (p NS). A significant increase for RSD fibroblasts compared with NS fibroblasts was observed from the 12th hour ($P < 0.05$). The peak contractile force developed by fibroblasts from RSD was 26% greater than fibroblasts from NS. In contrast, the contractile force developed by WSD fibroblasts did not differ significantly from that produced by NS fibroblasts (p NS). The third phase resulted in the maintenance of the peak contractile force that was generated. The increase in the RSD fibroblasts was statistically significant ($P < 0.001$).

αSM actin expression

Fibroblasts were cultured within three-dimensional collagen lattices for 2 days, after which they were double stained by immunofluorescence for αSM actin and histofluorescence for nuclei (Fig. 4). Fibroblasts from RSD were stained positively with αSM actin antibody (1a-b). At high magnification, large bundles of αSM actin filaments are visible in RSD fibroblasts (1c). In contrast, fibroblasts from NS do not express αSM actin (2). Fibroblasts from WSD and from NS had no αSM actin filaments (3–4).

Flow cytometric analyses of αSM actin expression on fibroblasts from NS, RSD and NS, WSD showed mean staining intensities of 2.3 ± 0.6, 5.7 ± 0.4, 2.9 ± 0.5 and
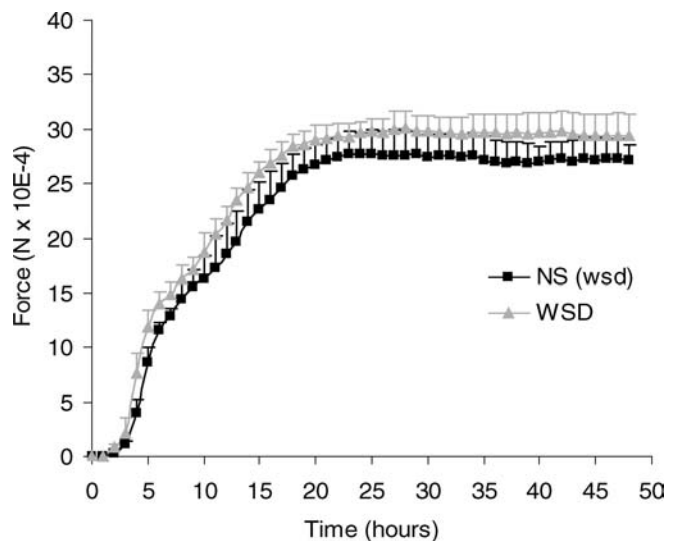
Fig. 2 Forces generated by RSD fibroblasts and NS fibroblasts. All data were expressed as the mean ± SD from five women

Fig. 3 Forces generated by WSD fibroblasts and NS fibroblasts. All data were expressed as the mean ± SD from five women

Fig. 4 Fluorescence staining of cultured fibroblasts from RSD (**1a-c**) and NS (**2**), and from WSD (**3**) and NS (**4**). Fibroblasts were stained with anti-αSM actin antibody and with Hoechst 33258
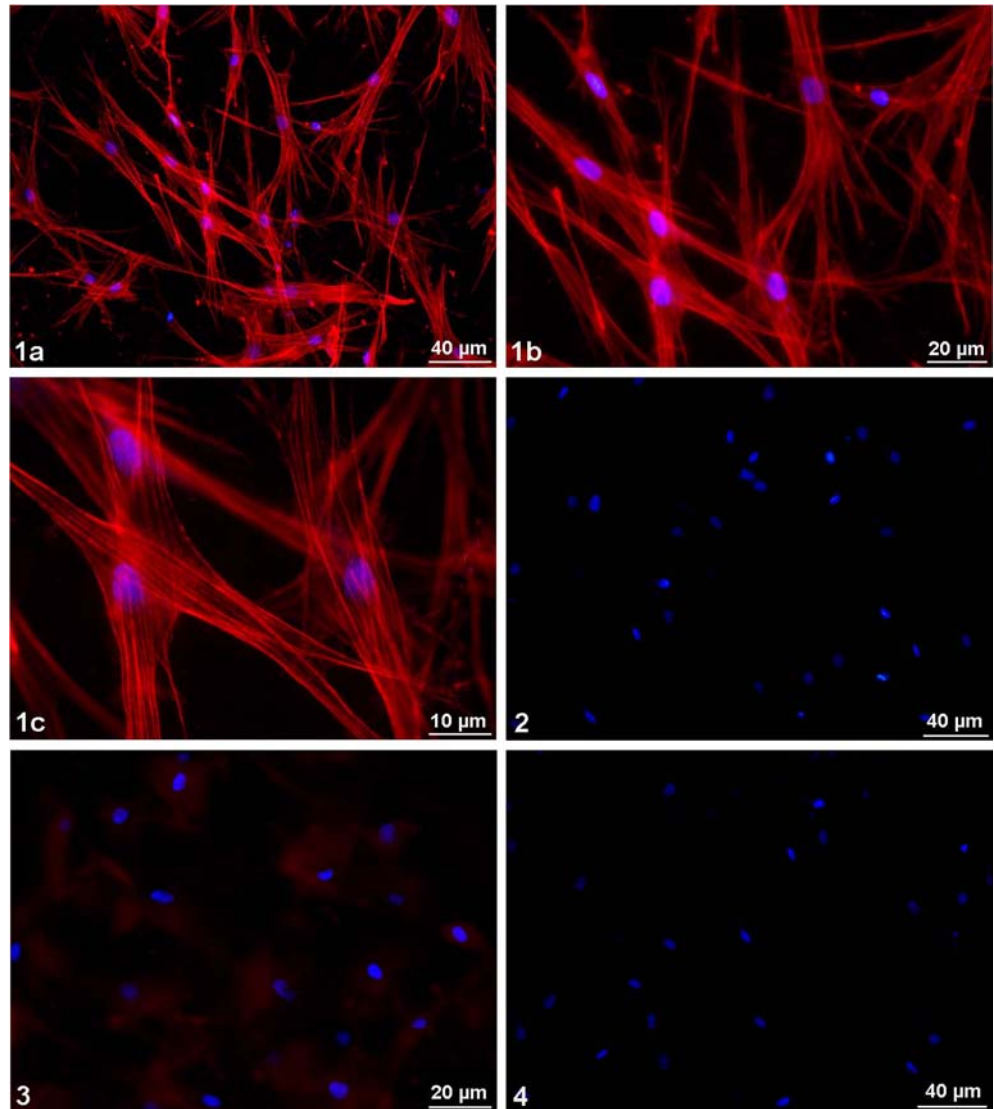
3.7 ± 0.3, respectively (Fig. 5 a, b). Compared with NS fibroblasts, a significant increase of αSM actin was observed for RSD fibroblasts ($P < 0.001$). There was no significant difference between fibroblasts from WSD and fibroblasts from NS (p NS).

Discussion

GlaSbox device

We present a new device allowing the quantification of the force generated in collagen lattices whose contraction has been prevented and which have become tense. The model developed a new method based on a silicon slide, which is smooth enough to be curved by the very weak forces involved in the collagen lattice culture. The slide deformation is measured by strain gauges deposited on its surface. The silicon technology allows the reduction of the component's size to the centimeters range. The lattice is attached to the sensor through a grid directly etched on the silicon stripe. Other groups have developed apparatuses for the same purpose. Kasugai used a square agar mould in which two steel grids were placed opposite each other, one fixed and the other attached to a float connected to a strain gauge [17]. In Devoye's apparatus, two beams were placed opposite each other in a rectangular culture chamber, and covered with Velcro in order to be gripped by the lattice. One of the beam was fixed, the other was connected to a strain gauge [9]. Kolodney's device comprised a culture chamber cut out in a hydrophobic silicone elastomer, into which hung two porous polyethylene holders, one attached to a multistage manipulator, the other attached to an isometric force transducer [19]. Eastwood attempted to improve Kolodney's device by having holders made from hydrophilic microsporous Vyon roughened with a scalpel, and connected to a force monitor by steel suture wires [10]. Campbell's system consists of four vertical cantilever beams with semiconductor strain gauges and can accommodate up to six circuits [5]. All these systems are based on the same
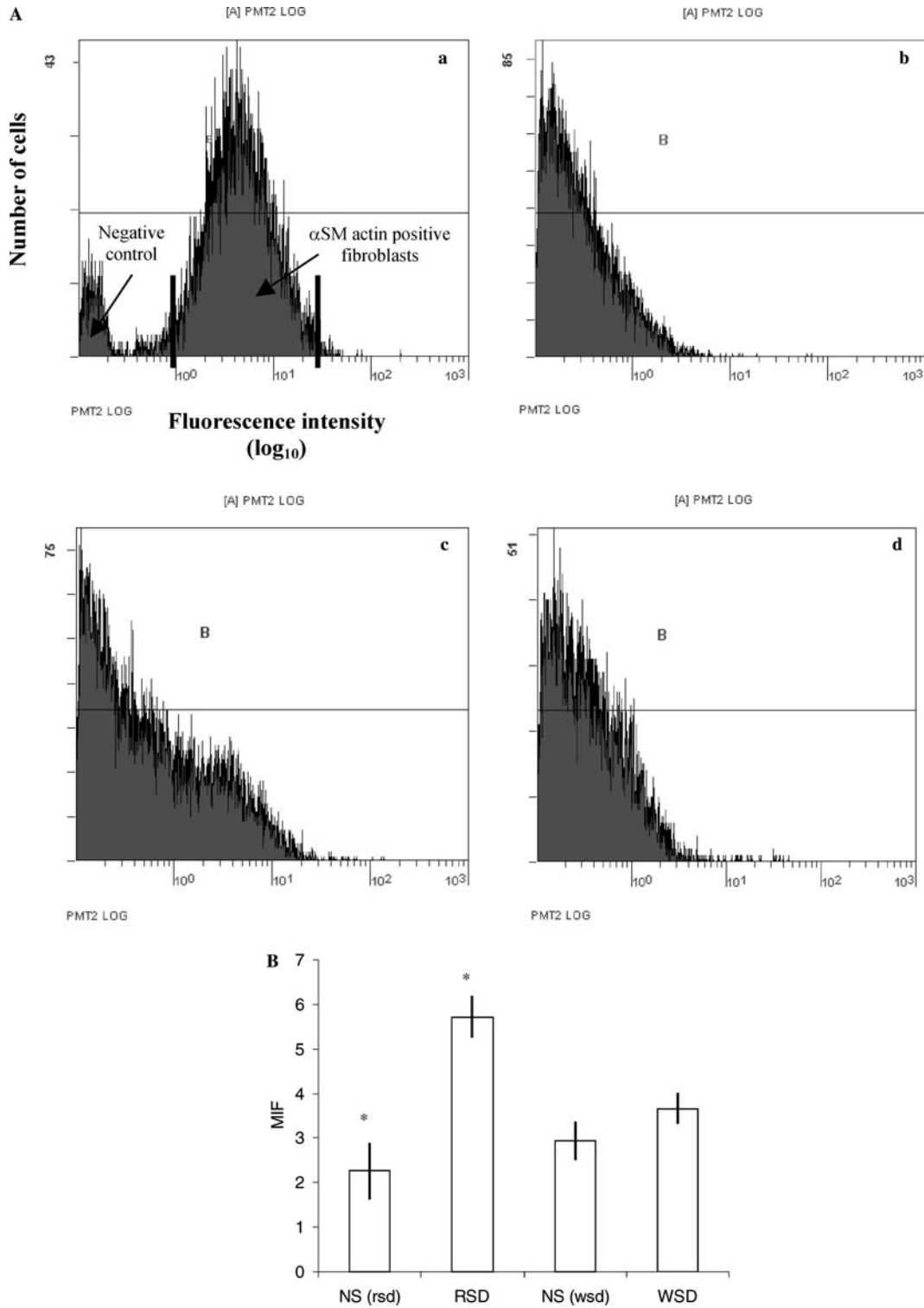
Fig. 5 a Flow cytometry histograms for αSM actin in fibroblasts from RSD (a) and NS (b), and from WSD (c) and NS (d). **b** Flow cytometry analysis of αSM actin expression. All data are expressed as mean intensity of fluorescence (MIF) ± SD from five women. Significant difference between fibroblasts from RSD and fibroblasts from NS is marked as *$P < 0.05$

principle: a strain gauge, generating electrical signals upon tension, assesses the minute displacement of a body attached to the gel. The present report describes a device with improved characteristics. Height lattices can be tested simultaneously. Their preparation requires low volumes of cell suspension and collagen. Contraction forces are of around 1.10^{-8} N/cell, consistent with that found in previous studies. Wrobel has compared the contractile properties of single human dermal fibroblasts and human dermal myofibroblasts by culturing them on flexible silicone elastomers [37]. He found that

fibroblasts and myofibroblasts could produce individual forces of 2–4 μN, which was different from those generated by fibroblasts using the GlaSbox. Differences lie in the culture conditions (monolayer, collagen gel), the sensitivity of the sensor, and the origin of the cells.

The GlaSbox is a useful tool to study the contractility of equivalent dermis composed of various fibroblasts lines. It can facilitate statistical design and analysis of experiments to study the effects of growth factors on cellular contraction and their potential role in scar tissue formation. In addition, this system can be used to examine the relationship between cellular contraction, cell proliferation, and collagen production, which is important in understanding the mechanism for scar tissue formation.

Contractile properties

Though not fully elucidated, the cause of striae distensae, how they form, and what course they take, are matters of considerable interest. There is currently a lack of information about the mechanical properties of such lesions. Rheological characteristics of striae distensae were evaluated in vivo and ex vivo [22, 23]. The aim of this report was to compare in vitro the contractile activity of normal skin and striae distensae. We have studied early and older stages of striae distensae associated with rapid stretching of the skin. We focussed on RSD and WSD fibroblasts placed in culture, and demonstrated that they behave differently from NS fibroblasts. Cells were analyzed at low passage and were, therefore, expected to retain some in vivo phenotype [31]. They were seeded in tense collagen lattices which are known to offer a valuable in vitro model for studying several cellular functions [3]. We used a quantitative and mechanical system (the GlaSbox) to measure the contractile force generated by fibroblast populations. Contractile forces developed by RSD fibroblasts were greater than those of NS fibroblasts. Our data also demonstrate that the force generation with WSD fibroblasts was similar to that of NS fibroblasts. It has been suggested that stress fibers, in particular, αSM actin, play an important role in wound contraction and, their presence has been correlated with the production of contractile force [14, 15]. In addition, the state of the actin cytoskeleton strongly influences the mechanical properties of cells and tissues [34]. Our immunocytological detection of αSM actin as a marker of myofibroblast, revealed a positive staining only in cultured RSD fibroblasts. Fibroblasts from RSD are characterized by the appearance of cytoplasmic bundles of αSM actin filaments. The presence of αSM actin in cultured RSD fibroblasts was confirmed by flow cytometric analyses. The expression of αSM actin correlates positively with the generation of contractile force and varies depending on the stage of the disease. Clinical and experimental studies have shown that fibroblasts can express αSM actin during fibrocontractile diseases and wound healing [28]. The overgeneration of contractile force by RSD fibroblasts appears to be related to characteristics of myofibroblastic differentiation. Our previous studies demonstrated that the expression of $\alpha_2\beta_1$ integrins was increased in RSD fibroblast cultures [33]. $\alpha_2\beta_1$ integrins are receptors involved in the attachment of fibroblasts to collagen fibers and in transmembrane transmission of mechanical signals [35]. Several investigations have concluded that cell lines with myofibroblast-like characteristics express integrins [18, 26].

We have included women with high body mass index (BMI) in the study. Excess mechanical stretching of the skin affected the dermis and caused striae distensae. Our observations indicate that during the early stage of striae distensae, fibroblastic cells modulate toward a myofibroblastic phenotype. They respond to the mechanical tension by expressing αSM actin and producing strong contractile forces. This myofibroblastic phenotype represents a reactive process that causes excess skin stretching. It refers to a quiescence with time, when the lesion takes on a scarlike appearance.

It would be interesting to evaluate αSM actin expression in vivo by immunohistology on biopsies from normal and striae distensae skin.

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