1 Introduction

Arterial dissections may occur spontaneously or nonspontaneously as a result of traumatic and have been observed in several arterial branches including the aorta. Aortic dissections frequently result from an intimal tear (see Ref. [1] and references therein) or from a perforation of the intima as, for example, caused by intramural hemorrhage and hematoma formation [2]. Moreover, mechanical traumatization of the intima due to the cannulation for catheter-based diagnostic and/or therapeutic interventions have been identified as initiating aortic dissections [3]. These intimal defects can cause concentrations of mechanical stress of the pressurized aorta and may be the trigger for the propagation of the medial dissection. In addition, blunt traumatic aortic rupture following motor vehicle accidents carries a high mortality and occurs in 21% of car occupant deaths in the UK [4]. Traumatic aortic dissection is one consequence for blunt thoracic trauma patients [5]. In a population-based long-term study, 86 cases of aortic dissection were found in 84 patients (mean age of 65.7 yr), 66 patients were hospitalized and 18 nonhospitalized [6]. A total of 22.7% of the hospitalized patients died within the first 6 h, 33.3% within 12 h, 50% within 24 h, and 68% in total died within the first two days after admission. Untreated, a dissection may propagate until it runs either back into the lumen, resulting in the reduplication of the aortic lumen (false lumen), or it ruptures through the adventitia, often with a lethal outcome [2]. For example, in the absence of intervention, acute aortic dissections have a 90% chance of mortality, and the majority of these deaths occur within 48 h [7].

The incidence of spontaneous aortic dissection is 5–30 cases per million people per year, and it strongly depends on the presence of risk factors. Among many others, chronic systemic hypertension, atherosclerosis, Marfan’s and Ehlers-Danlos syndromes, aortic dilatation (aneurysm), and coarctation of the aorta are well-established predisposing factors for aortic dissection [2,8]. Chronic systemic hypertension is the most common predisposing factor and has been present in 62–78% of patients with aortic dissection [2].

A common cause of media dissection is balloon angioplasty, an established and effective therapeutic intervention to reduce the severity of atherosclerotic stenosis [9]. Balloon angioplasty involves denudation of endothelium, disruption of the intima and the atherosclerotic plaque with frequent separation from or dissection of the media, and overstretching of nondiseased portions of the arterial wall, see, for example, Ref. [10] and references therein. In particular, dissection is a characteristic form of arterial trauma involving laceration and/or cleavage of the arterial wall. Plaque fracture and/or dissections are major contributors to the gain in lumen following balloon angioplasty [9]. Moreover, dissection has been implicated as a contributing factor to both acute procedural complications (abrupt reclosure, ischemia, myocardial infarction, emergency surgery, and coronary microembolization [11–15]) and chronic restenosis of the treatment site [16]. Luminal gain, tissue injury, and the risk of wall fracture are quantities for consideration with this interventional procedure. Dissection sec-

Dissection Properties of the Human Aortic Media: An Experimental Study

Aortic dissection occurs frequently and is clinically challenging; the underlying mechanics remain unclear. The present study investigates the dissection properties of the media of 15 human abdominal aortas (AAs) by means of direct tension tests (n=8) and peeling tests (n=12). The direct tension test demonstrates the strength of the media in the radial direction, while the peeling test allows a steady-state investigation of the dissection propagation. To explore the development of irreversible microscopic changes during medial dissection, histological images (n=8) from four AAs at different peeling stages are prepared and analyzed. Direct tension tests of coin-shaped medial specimens result in a radial failure stress of \(140.1 \pm 15.9 \text{kPa} \) (mean \(\pm\) SD, n=8). Peeling tests of rectangular-shaped medial strips along the circumferential and axial directions provide peeling force/width ratios of \(22.9 \pm 2.9 \text{mN/mm} \) (n=5) and \(34.8 \pm 15.5 \text{mN/mm} \) (n=7); the related dissection energies per reference area are \(5.1 \pm 0.6 \text{mJ/cm}^2 \) and \(7.6 \pm 2.7 \text{mJ/cm}^2 \), respectively. Although student's t-tests indicate that force/width values of both experimental tests are not significantly different (\(p=0.05, p=0.125\)), the strikingly higher resisting force/width obtained for the axial peeling tests is perhaps indicative of anisotropic dissection properties of the human aortic media. Peeling in the axial direction of the aorta generates a remarkably “rougher” dissection surface with respect to the surface generated by peeling in the circumferential direction. Histological analysis of the stressed specimens reveals that tissue damage spreads over approximately six to seven elastic laminae, which is about 15–18% of the thickness of the abdominal aortic media, which forms a pronounced cohesive zone at the dissection front.
[DOI: 10.1115/1.2898733]

Keywords: aortic dissection, dissection energy, failure stress, media, human abdominal aorta
The rubberlike protein elastin, the stiff fibrous protein collagen, and smooth muscle cells influence the mechanical properties of arterial walls. In the media of elastic arteries (like the aorta), these three components are found to be organized in medial lamellar units [18], each of which is about 10 μm thick [19]. These laminated structures may be prone to separation creating a cleavage plane parallel to the elastic lamellae [20], and hence provide the mechanism to propagate a dissection in parallel to the lumen [21]. Spontaneous aortic dissections typically show steady-state-like failure propagation, which might indicate a kind of fatigue mechanism caused by the pulsatile loading of the aorta. By contrast, balloon angioplasty causes extraordinarily high mechanical loading to the (stenotic) tissue during one or more episodes of treatment, and hence (quasi)static failure mechanisms seem to apply. Although arterial dissection is a frequently occurring phenomenon and a challenging clinical entity, the underlying biomechanical properties remain largely unclear.

The present study was carried out with the goal to investigate the dissection properties of the aortic media. Direct tension and peeling experiments of the media of human abdominal aortas were performed. The direct tension test demonstrates the dissection strength across the lamellae of the media in the radial direction, while the peeling experiment explores the fracture energy required to propagate a dissection. Under certain assumptions, the performed experiments allow an estimation of the constitutive properties of medial dissection within the abdominal aorta. The gathered data have served as a basic input for the finite element modeling of the experiments, presented in the companion paper [22]. From the combined experimental and numerical investigation, an accurate prediction of the dissection properties is feasible. To this end, the material parameters in the finite element model, which affects the dissection process, are varied until the numerical prediction matches the experimental data. In addition, the mechanical study is enhanced by histology at different stages of the peeling test to explore the development of irreversible changes on the microscale level during medial dissection.

### 1.1 Fracture Properties of the Aorta

To our knowledge, the approach in this study is new. However, a variety of methods have been developed and implemented with the goal to explore the failure properties of aortic tissue. A summary of related experimental data is given in Table 1 with respect to tensile, inflation, tearing, splitting, and peeling tests performed on human, porcine, and canine aortic tissues.

#### Table 1 Failure properties of arterial tissue according to tensile, inflation, tearing, splitting, and peeling tests. Mean values are presented with standard deviations.

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Circ. strength (MPa)</th>
<th>Axial strength (MPa)</th>
<th>Radial strength (MPa)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human descending mid thoracic aorta</td>
<td>1.72 ± 0.89</td>
<td>1.47 ± 0.91</td>
<td>—</td>
<td>[23]</td>
</tr>
<tr>
<td>Human abdominal aorta</td>
<td>—</td>
<td>1.21 ± 0.33</td>
<td>—</td>
<td>[24]</td>
</tr>
<tr>
<td>Human descending thoracic aorta</td>
<td>1.76 ± 0.22</td>
<td>1.95 ± 0.60</td>
<td>—</td>
<td>[25]</td>
</tr>
<tr>
<td>Porcine thoracic aorta</td>
<td>—</td>
<td>—</td>
<td>0.061 ± 0.004</td>
<td>[26]</td>
</tr>
<tr>
<td>Human ascending thoracic aorta</td>
<td>1.80 ± 0.24</td>
<td>1.71 ± 0.14</td>
<td>—</td>
<td>[27]</td>
</tr>
<tr>
<td>Human abdominal aorta</td>
<td>—</td>
<td>—</td>
<td>0.140 ± 0.016</td>
<td>Present study</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Ultimate stress (MPa)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human descending mid thoracic aorta</td>
<td>0.114 ± 0.032</td>
<td>[28]</td>
</tr>
<tr>
<td>Human descending thoracic aorta</td>
<td>2.7 ± 1.5</td>
<td>[29]</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Circ. breaking stress (MPa)</th>
<th>Axial breaking stress (MPa)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Porcine upper descending thoracic aorta</td>
<td>2.19 ± 0.57</td>
<td>0.18 ± 0.44</td>
<td>[30]</td>
</tr>
<tr>
<td>Porcine lower descending thoracic aorta</td>
<td>3.64 ± 0.53</td>
<td>0.87 ± 0.34</td>
<td>[30]</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Max. pressure (MPa)</th>
<th>Splitting energy (mJ/cm²)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Porcine descending thoracic aorta</td>
<td>0.077 ± 0.002</td>
<td>15.9 ± 0.9</td>
<td>[31]</td>
</tr>
<tr>
<td>Human thoracic and abdominal aorta</td>
<td>0.079 ± 0.029</td>
<td>0.17</td>
<td>[32]</td>
</tr>
<tr>
<td>Porcine upper thoracic aorta</td>
<td>0.106 ± 0.022</td>
<td>2.84 ± 1.19</td>
<td>[33]</td>
</tr>
<tr>
<td>Porcine lower thoracic aorta</td>
<td>0.109 ± 0.019</td>
<td>2.90 ± 1.21</td>
<td>[33]</td>
</tr>
<tr>
<td>Porcine upper abdominal aorta</td>
<td>0.095 ± 0.037</td>
<td>1.88 ± 0.89</td>
<td>[33]</td>
</tr>
<tr>
<td>Porcine lower abdominal aorta</td>
<td>0.085 ± 0.027</td>
<td>11.34 ± 4.05</td>
<td>[33]</td>
</tr>
<tr>
<td>Porcine thoracic aorta</td>
<td>0.071</td>
<td>—</td>
<td>[20]</td>
</tr>
<tr>
<td>Porcine descending thoracic aorta</td>
<td>0.073</td>
<td>—</td>
<td>[34]</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Circ. dissection energy (mJ/cm²)</th>
<th>Axial dissection energy (mJ/cm²)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human abdominal aorta</td>
<td>5.1 ± 0.6</td>
<td>7.6 ± 2.7</td>
<td>Present study</td>
</tr>
</tbody>
</table>
tions [26], circumferential and axial directions [23,27], inflation tests on circular specimens incorporating bubble inflation techniques [28], inflation tests of intact arteries [29], and tearing tests of arterial strips [30] were performed to determine the tensile strength of aortic tissue. Besides that, in the literature, only a few techniques have been presented to investigate the dissection properties of the aortic media. A commonly used approach has been to create a bleb by infusing a fluid into the media, as it was presented for human aortas decades ago [35,36]. This idea has been adopted by Roach and co-workers to investigate dissection properties of the canine thoracic aortas [21], the porcine aorta [31,33], and the human aorta [32]. Moreover, they applied this method to isolated and pressurized porcine aortas in order to explore the mechanisms of dissection propagation under more physiological circumstances [20,34].

The experimental data from tensile tests on aortas indicate that they are dependent on the anatomical site of the vessel and that the tensile failure behavior is perhaps anisotropic (see Table 1). The aortic strength in the axial direction seems to be slightly lower than in the circumferential direction; however, the large standard deviation makes this finding inconclusive. The failure properties obtained from tensile tests suggest that the tensile strength of the human aorta is decreasing with increasing distance from the heart. Tensile or inflation tests of soft biological tissue are poorly suited to quantify the fracture energy of the tissue, since these testing methods cause unstable (dynamic) failure, i.e., its propagation cannot be controlled. In contrast, tearing experiments, as conducted in Ref. [30], lead to controlled failure propagations of tissues and allow a quantification of the fracture energy. That paper [30] documents that the circumferential “breaking stress” (evaluated through axial tearing experiments) exceeds the axial breaking stress (evaluated through circumferential tearing experiments), which is in accordance with findings from tensile tests. Media splitting showed that the maximum splitting pressure is fairly constant; however, the splitting energy decreases with distal position along the aorta. For comparative purposes, the results from the direct tension and the peeling tests of the present study are included in Table 1. Note that the experimental approach of the peeling test is fundamentally different from the “Tearing test” and the “Media splitting test,” which was noted by the introduction of a separate category in the table.

2 Material and Methods

2.1 Material. In the present study, 19 human abdominal aortas (AAs) (age range 36–75 yr, ten males and nine females) were harvested during autopsy within 24 h from death. We have taken segments from the infrarenal portion of the AA. It was a requirement that pathological changes, such as severe atherosclerosis, were not present in the specimens. In order to satisfy this requirement, the condition and texture of the intima were closely inspected by a trained pathologist. After harvesting, the specimens were stored in a calcium-free and glucose-free 0.9% physiological saline solution at 4 °C until use. Use of autopsy material from human subjects was approved by the Ethics Committee, Medical University Graz, Austria. All tests were performed within 48 h from death.

2.2 Specimen Preparation. We conducted two different mechanical failure tests of the media of the AA specimens—denoted by (i) direct tension tests and (ii) peeling tests. The peripherally attached adipose and loose connective tissues were carefully removed from the adventitia of the aortic patch. The AAs were then cut along the axial direction to obtain flat rectangular tissue sheets. For the anatomical separation of the three layers, we tested various locations at which the layer separation process can easily be initiated. The arterial layers were separated with careful dissection techniques and with the aid of a surgical scalpel. Reflected light microscopy was used to inspect the layer preparation process. The structured media could easily be distinguished from the intimal and the adventitial tissues. In addition, for all specimens considered in the study, related histological images were prepared and investigated in order to confirm that specimens consisted only of medial tissue. This technique has been successfully applied in the past (see Refs. [37,38]). Particular attention has been paid to minimize the mechanical damage of the media including tissue immersion in a 0.9% physiological saline solution throughout the
whole separation process. Figure 1 shows the photographs of the separated intimal, medial, and adventitial layers (from left to right), where it can be seen that even perforating vessels have not caused any difficulties during the separation process.

While intimal and adventitial tissues were not further considered, the medial patches were used to prepare the specimens for the conducted mechanical testing and for subsequent histological investigations. A cylindrical blanking tool was used to punch out eight coin-shaped specimens for the direct tension test (6.0 mm diameter, and 1.16 ± 0.15 mm (mean ± SD), thickness), subsequently denoted as D I–D VIII. These eight radial tension test specimens were obtained from three different AAs. Several specimens were obtained from the same aorta, as indicated in Fig. 1, but note that only about one out of five direct tension tests was successful. A total of 12 rectangular-shaped strip specimens were cut out with a surgical scalpel for the peeling tests, whereas each peeling sample was from a different AA. From Fig. 1, it can be seen that the rectangular strips are either oriented in the circumferential direction θ or in the axial direction z. Five specimens were cut out along the circumferential direction of the aorta, subsequently denoted as PC I–PC V, and seven specimens were cut out along the axial direction of the aorta, subsequently denoted as PA I–PA VII.

In order to get the specimens ready for testing, some final preparations were required. After mounting the specimens for the direct tension test on the testing machine, an incision of about 1.0 mm depth around the circumference of the circular specimen was cut with an especially adapted surgical knife. This undercut provided the site for the initiation of failure (and reduced the tissue diameter from 6.0 mm to 4.0 mm). The specimens devoted to the peeling test were split at one end to get two “tongues” (8–10 mm in length) of about equal thickness for mounting them on the testing machine. We used a superadhesive gel to glue rectangular pieces of sandpaper (Grit: 320) at both sides of the tongues to avoid slipping of the specimen in the clamps of the testing machine during loading. A representative specimen, ready for a peeling test, is shown in Fig. 2. The individual dimensions of the aortic test specimens are summarized in Tables 2–4. Note that the dimensions refer to the prepared medial patches, and that the effective lengths of the peeling test specimens in the circumferential and axial directions are denoted as $L_{pc}$ and $L_{pa}$, respectively.

2.3 Mechanical Testing

2.3.1 Device. Mechanical tests were performed on a computer-controlled, screw-driven high-precision tensile testing machine (Messphysik, μ-Strain Instrument ME 30-1, Fürstenfeld, Austria), which was adapted for small biological specimens (experimental setup shown in Fig. 3). The specimens were investigated in a perspex container filled with 0.9% physiological saline solution maintained at 37.0 ± 1.0°C by a heater-circulation unit (type Ecoline E 200; LAUDA, Lauda-Königshofen, Germany) and the tensile force were measured with a 10 N Class 1 strain gauge-load cell (type TCA 10 N, code CTCA1K5; AEP transducers, Modena, Italy). The upper and the lower fixing clamps of the testing machine are moving in opposite directions, which keeps the center of the specimen fixed in space. A position control resolution of 0.04 μm of the upper and the lower crosshead of the tensile testing machine (see Fig. 3) and a combined error of 0.03% of the 10 N load cell are specified by the manufacturer. For our case, this means that a combined error of 0.6 mN may occur at a maximum occurring force of 2 N.

While the specimens for the peeling test could directly be mounted on the testing machine, an especially developed speci-

---

**Table 2** Thickness of the specimens D I–D VIII for the direct tension test

<table>
<thead>
<tr>
<th>Specimen</th>
<th>Thickness (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>D I</td>
<td>1.34</td>
</tr>
<tr>
<td>D II</td>
<td>1.20</td>
</tr>
<tr>
<td>D III</td>
<td>0.97</td>
</tr>
<tr>
<td>D IV</td>
<td>1.04</td>
</tr>
<tr>
<td>D V</td>
<td>1.27</td>
</tr>
<tr>
<td>D VI</td>
<td>1.07</td>
</tr>
<tr>
<td>D VII</td>
<td>1.34</td>
</tr>
<tr>
<td>D VIII</td>
<td>1.01</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>1.16 ± 0.15</td>
</tr>
</tbody>
</table>

**Table 3** Length $L_{pc}$, width, and thickness of the circumferential peeling test specimens PC I–PC V

<table>
<thead>
<tr>
<th>Specimen</th>
<th>Length $L_{pc}$ (mm)</th>
<th>Width (mm)</th>
<th>Thickness (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PC I</td>
<td>30.1</td>
<td>6.5</td>
<td>0.96</td>
</tr>
<tr>
<td>PC II</td>
<td>31.5</td>
<td>8.3</td>
<td>1.58</td>
</tr>
<tr>
<td>PC III</td>
<td>27.4</td>
<td>8.3</td>
<td>0.93</td>
</tr>
<tr>
<td>PC IV</td>
<td>27.1</td>
<td>9.3</td>
<td>1.15</td>
</tr>
<tr>
<td>PC V</td>
<td>31.0</td>
<td>8.1</td>
<td>1.20</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>29.4 ± 2.0</td>
<td>8.1 ± 1.0</td>
<td>1.16 ± 0.26</td>
</tr>
</tbody>
</table>
men holder was used for the direct tension test. A small cylindrical-shaped recess in each plastic rod, with fixed sandpaper (Grit: 320) therein, leads to a strong specimen-rod connection. The coin-shaped specimens were then glued with cyanoacrylate glue onto the fixed sandpapers on the two plastic rods for mounting on the testing machine (Fig. 4).

2.3.2 Protocol. Direct Tension Test. Cyanoacrylate glue was placed on the sandpaper into the cylindrical recess of the upper and lower rods of the specimen holder. The lower rod was then mounted on the testing machine and the circular test specimen was placed in the cylindrical recess. The upper rod of the specimen holder was mounted on the testing machine, and a compression force of 1.0 N was applied to the specimen for about 5 min to allow the adhesive to react. Before testing, the specimen was moistened with 0.9% physiological saline solution. Throughout the whole test, the extension rate of 1.0 mm/min was controlled and the resisting force recorded. Moreover, the whole test was recorded by a charge coupled device (CCD)-camera and saved in Super Video-Home-System (SVHS) format. After the specimen was completely separated, the zero load level was defined and the failure surface has been visually inspected. Frequently, it happened that failure occurred at the region where the specimen was glued to the specimen holder and not at the location where the incision was placed. If that was the case, the test was rejected. A representative photograph of the end stage of a successful direct tension test is shown in Fig. 5.

Peeling test. Both tongues of the specimen were mounted on the testing machine. Similar to the direct tension test, the extension rate of 1.0 mm/min was controlled, and the measured force recorded. The whole test was recorded, saved in SVHS format, and the zero load level was defined after complete separation of the specimen. In order to account for effects of buoyancy, the testing machine’s “buoyancy-force-displacement” curve was subtracted from the achieved results. This curve was recorded for a particular level of fluid in the perspex container but without a test

<table>
<thead>
<tr>
<th>Specimen</th>
<th>Length $L_{pa}$ (mm)</th>
<th>Width (mm)</th>
<th>Thickness (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PA I</td>
<td>15.0</td>
<td>8.3</td>
<td>2.43</td>
</tr>
<tr>
<td>PA II</td>
<td>18.0</td>
<td>7.8</td>
<td>1.74</td>
</tr>
<tr>
<td>PA III</td>
<td>30.0</td>
<td>8.2</td>
<td>1.53</td>
</tr>
<tr>
<td>PA IV</td>
<td>35.0</td>
<td>8.9</td>
<td>1.43</td>
</tr>
<tr>
<td>PA V</td>
<td>33.5</td>
<td>8.6</td>
<td>1.57</td>
</tr>
<tr>
<td>PA VI</td>
<td>25.0</td>
<td>8.2</td>
<td>1.39</td>
</tr>
<tr>
<td>PA VII</td>
<td>15.0</td>
<td>7.4</td>
<td>1.82</td>
</tr>
</tbody>
</table>

mean ± SD 24.7 ± 8.0 8.2 ± 0.5 1.70 ± 0.36

Table 4 Length $L_{pa}$, width, and thickness of the axial peeling test specimens PA I–PA VII
specimen. A representative image of the medium stage of a circumferentially oriented strip specimen during a peeling test is shown in Fig. 6.

**Measurement of specimen dimensions.** The geometrical dimensions (length, width, thickness) of the test specimens were measured optically before the individual tests by using a PC-based videoextensometer utilizing a full-image CCD camera. For the length and width measurements, the strip samples were positioned in air on a black plate in front of the CCD camera. After calibration of the videoextensometer, its automatic edge recognition capability was used to quantify the dimensions. For the thickness measurement, the strip sample was positioned in air on a black colored object plate of known thickness (1 mm). One lateral side of the gauge region of the strip sample was in plane with the lateral side of the object plate and oriented toward and perpendicular to the CCD camera. Then the thickness of the contour (sample plus object plate) was measured by the videoextensometer within the gauge region. The resulting thickness value of the sample was determined then as the average of the contour thickness along the gauge region minus the thickness of the object plate.

2.4 Histology. In order to investigate irreversible changes of the tissue’s microstructure due to the dissection, eight additional rectangular-shaped strips (four circumferentially and four axially oriented) were prepared from four AAs. In contrast to the aforementioned peeling tests, these strips were not dissected throughout their whole length. We stopped the peeling approximately in the middle of the strip length. For that reason, we designed a specimen holder in order to “freeze” the loading state of the tissue sample (Fig. 7). After mounting the tissue in the specimen holder, it was fixed in 7% neutral-buffered formaldehyde solution (pH 7.4), embedded in paraffin using standard techniques and prepared for histological investigations.

The media, which were partly dissected, were then sectioned at 4 μm and stained with Elastica van Gieson. The applied technique allowed us to investigate, histologically, the gradual development of the damage state in the media subjected to supraphysiological loading, and to trace and study the irreversible effects in the medial tissue due to the mechanical loading.

3 Results and Interpretation

3.1 Direct Tension Test. Figure 8 presents the measured force-displacement response of all eight coin-shaped media specimens during the direct tension test. The thick (solid) curve characterizes the mean response of the individual tissue tests, which are denoted by thin curves. Note that after a radial extension of approximately 4.5 mm, some curves drop down to zero so that less than eight specimens are included in the averaging process above 4.5 mm. The mean curve represents the arithmetic mean calculated with the data analysis software ORIGINPRO 7.5 (Origin-Lab Corporation).

During the increasing displacement, the force-displacement response shows a characteristic sequence, which we subsequently denote by the three regions: S1 for elastic, S2 for damage, and S3 for failure (see Fig. 8). Region S1, i.e., the initial phase of the force-extension curve, is assumed to represent the tissue’s elastic response and is characterized by a steep slope in the force-displacement curve. It is governed by the geometry of the test specimen (diameter, thickness, and incision depth) and the material properties of the media of the human AA. At about 1.25 N, the “elastic limit” is reached. Damage softening (decreasing stiffness with increasing damage of the tissue [39]) and generation of...
microdefects characterize Region S2, which is a region that still has increasing resistance to extension. As illustrated in MacLean et al. [26] (Fig. 4 therein), there may be ruptured elastin layers on a microscale that lead to cavities and to the characteristic pattern for microdefects in the media. The state of damage evolves further until a displacement of about \( u = 0.85 \, \text{mm} \), with the limit force of \( F_{\text{max}} = 1.76 \pm 0.20 \, \text{N} \) (mean \( \pm \text{SD}, n=8 \)), is reached, and microdefects coalesced. Consequently, the media dissects, which is indicated by the decreasing force in Region S3. Our experiments suggest that the primary dissection S3a is followed by a peeling-like failure mechanism S3b, which determines a plateau in the force-displacement curve until it drops to zero (see Fig. 8).

3.2 Peeling Test. The applied peeling test caused a slow and controlled dissection propagation, where the force-displacement curve is characterized by a jagged plateau region. In the following, the results of the peeling of human AA media in circumferential and axial directions are presented separately. Figures 9 and 10 display the force-displacement and force/width versus dissection path for all specimens.
specimens, respectively. The five circumferentially and the seven axially oriented test specimens, i.e., the thick (solid) curves in Figs. 9 and 10, were fitted to the horizontal lines in the force/width-dissection graph. These defined the average peeling force/width of \( F_{pc} = 22.9 \pm 2.9 \) mN/mm (mean \( \pm SD, n = 5 \)) and of \( F_{pa} = 34.8 \pm 15.5 \) mN/mm (mean \( \pm SD, n = 7 \)) (see Table 5) of human abdominal medias along the circumferential and axial directions, respectively. The student's t-test (significance level of \( \alpha = 0.05 \)) gives a \( p \) value of 0.125, which indicates that the force/width values of both tests are not significantly different. However, the strikingly higher resisting force/width obtained for the axial peeling tests with respect to the circumferential peeling tests is perhaps indicative of anisotropic dissection properties of the human aortic media.

### 3.3 Histological Investigation

As mentioned in Sec. 2.4, eight additional rectangular-shaped strips were prepared and dissected up to about the middle of their lengths, and, consequently, the stress state was “frozen” in a specifically designed device. Related histological images were prepared. Figure 11(a) shows the media in a kind of “frozen stress state,” which straightens the elastic laminae, and the intermingled collagen fibers and smooth muscle cells might straighten as well. Figure 11(b) shows the media embedded under stress-free conditions. The parallel sheets of elastic laminae are attached to each other by fine elastin, collagen fibers, and smooth muscle cells, as indicated in the histological images presented in Fig. 11. For a detailed explanation of the transmural organization of the arterial media, see the electronmicroscopical study [40]. We found an average of \( 40 \pm 4 \) (mean \( \pm SD, n = 8 \)) lamellar units in the human abdominal aortic media of the investigated specimens. Note that collagen fiber bundles and smooth muscle cells are barely seen in these images because of the choice of stain, which accentuates elastin fibers.

The microscopical damage to the media during the peeling in circumferential and axial directions is nicely illustrated in Figs. 12(a) and 12(b), as can be seen by the area at the dissection front. In this area, a pronounced cohesive zone has developed, within which fiber bridging might play a dominant role. The elastic laminae are torn apart and the formation of cavities in between is observed (see Figs. 12(c) and 12(d) for a zoom into the dissection

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**Table 5 Peeling force/width defining the circumferential and axial peeling properties of human AA medias:** PC I–PC V and PA I–PA VII refer to the individual specimens for the circumferential and axial peeling tests, respectively. The values \( F_{pc} \) and \( F_{pa} \) denote the arithmetic means of the peeling force/width of the five circumferentially and the seven axially oriented test specimens, respectively.

<table>
<thead>
<tr>
<th>Circumferential peeling</th>
<th>Axial peeling</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specimen</td>
<td>Force/Width (mN/mm)</td>
</tr>
<tr>
<td>PC I</td>
<td>25.2 ( \pm ) 2.2</td>
</tr>
<tr>
<td>PC II</td>
<td>22.6 ( \pm ) 1.8</td>
</tr>
<tr>
<td>PC III</td>
<td>21.0 ( \pm ) 1.6</td>
</tr>
<tr>
<td>PC IV</td>
<td>26.2 ( \pm ) 2.9</td>
</tr>
<tr>
<td>PC V</td>
<td>19.3 ( \pm ) 2.9</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
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</tr>
</tbody>
</table>
| \( F_{pc} = 22.9 \pm 2.9 \) | \( F_{pa} = 34.8 \pm 15.5 \)
Consequently, under radial (tensile) loading of the medial tissue, interconnected fine elastin fibers, and interlaminar muscle cells are torn loose from their attachments to each other and to adjacent elastin (similar to the study [26]); ruptured fine fibers are evident in the photomicrographs. The observed damage spreads over approximately six to seven elastic laminae, which is about 15–18% of the thickness of the abdominal aortic media. Note that we could not observe circular-shaped nuclei of the smooth muscle tips.

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**Fig. 11** Representative histological images of the microstructure of (a) stretched and (b) unstretched human aortic media. Elastica van Gieson (EVG) staining, 4 µm thick sections. Original magnification 800×.

**Fig. 12** Histological images of a representative aortic media during peeling in (a) circumferential and (b) axial directions. Original magnification 20×. Histological images (c) and (d) represent magnifications of the dissection tips and highlight the irreversible mechanism of separation at the micrographic level. Original magnification 400×. EVG staining, 4 µm thick sections.
cells within the damage zone, as discovered in the study [20],
which is most probably due to the low resolution of the histology.

Microscopical images of the generated dissection surface high-
light that peeling in the axial direction creates a remarkably
“rougher” dissection surface compared to that generated by peel-
ing in the circumferential direction (two representative images are
shown in Fig. 13). In particular, the dissection during the peeling
in circumferential direction propagates mainly between adjacent
elastic laminae, while the one in the axial direction frequently
crosses elastic layers. Interestingly, it sometimes happened that
the dissection runs either toward the internal elastic membrane or
toward the external elastic membrane during the peeling in the
axial direction, which, in a way, mimics the process of aortic
dissection, as observed in clinical practice, i.e., where the dissec-
tion runs either back into the lumen or outward into the adventitia.
Consequently, it was not possible to dissect the whole specimen.
This, however, never happened by peeling rectangular-shaped
specimens along the circumferential direction. The histology of
the media might explain the observed anisotropic dissection prop-
erty of the human AA, and the formation of a rougher dissection
surface due to the peeling in the axial direction, as illustrated in
Fig. 13. The smooth muscle cells in the media are oriented fairly
circumferentially [19] and may provide a more pronounced resis-
tance to the dissection in the axial direction (for example, it is
easier to run parallel to the furrows of a ploughed field than across
them). Consequently, the creation of the rougher dissection sur-
face during peeling of axially oriented test specimens might ex-
plain the “higher” resisting force/width and “variance” compared
to the results from peeling in circumferential direction.

4 Discussion

The present study was conducted to explore medial dissection
of the human abdominal aorta. Therefore, direct tension and peel-
ing tests are proposed, which render defined mechanical problems,
and hence allow the quantification of the tissue’s dissection prop-
erties. The failure mechanisms related to the peeling investiga-
tion of damage

region below 10%, and it is most likely that this is too small to
activate mechanisms responsible for stiffness increase at higher
strains. The final peelinglike failure mechanism region, denoted as
S3b, determines a plateau in the force-displacement curve until it
drops to zero, see Fig. 8. It seems that the layered structure of the
media favors the peeling mechanism along the lamellar layers. A
dissection within one lamella throughout the whole dissection
path did not occur, but we observed outward and inward devia-
tions from the lamellar layers. Consequently, the applied experi-
mental approach, i.e., the direct tension test leads to multiple dis-
sections between the different medial layers and a peelinglike
failure mechanism, as it is illustrated in Fig. 5, determines the
force-displacement characteristics at larger displacements.

The following is now a brief discussion of the experimental
findings of this work with respect to the existing literature, where
the radial failure stress and the energy required to propagate a
dissection is investigated in detail.

In order to better quantify the results of the direct tension test
we assume, mainly for simplicity, a homogeneous stress state to
be present. For that case, the (average) radial failure stress can be
estimated as $F_{\text{max}}/(d^2/4) = 140.1 \pm 15.9$ kPa (mean $\pm$ SD, $n=8$),
where $F_{\text{max}} = 1.76 \pm 0.20$ N (mean $\pm$ SD, $n=8$), and $d=4.0$ mm
denotes the effective diameter of the circular specimens. It needs
to be emphasized that the conducted direct tension tests have
caused stress concentrations at the incision of the specimen, and
due to its small size this “edge effect” cannot be neglected. Nev-
ertheless, this approach gives an estimation of the upper limit of
the radial failure stress of the investigated human abdominal aor-
tic media, and we may compare the failure stress with the one
provided in Ref. [26]. That study on porcine thoracic aortas [26]
reported a failure of $61.4 \pm 4.3$ kPa (mean $\pm$ SD, $n=7$), which is a
value approximately half that determined in our experiments on
human AAs. The disagreement may be explained by species dif-
ferences and high variances.

Regarding the peeling tests, there is an oscillation of the force
about some mean “plateau” level (see Figs. 9 and 10), which is
similar to the results obtained from tearing pig descending tho-
racic aortas, as provided in Ref. [30]. The oscillation is due to an
instability inherent in this kind of experiment, which is called
“stick-slip tearing” in rubber mechanics, see, for example, Ref.
[44]. Based on the given results for the peeling test, we may now
estimate the energy required to propagate the medial dissection.
According to the performed experiments, the external work is
found to be $W_{\text{ext}} = 2.0F_{\text{peel}}/\pi R = 1.67 \pm 0.33$ mJ/mm (mean $\pm$ SD, $n=
5$) and $W_{\text{ext}} = 2.0F_{\text{pa}}/\pi R = 2.02 \pm 1.41$ mJ/mm (mean $\pm$ SD, $n=7$),
for the circumferential and axial peeling tests, respectively ($F_{\text{peel}}$
and $F_{\text{pa}}$ are according to Table 5, and $R$, $R_{\text{pa}}$ are the current lengths of
the specimens in the circumferential and axial directions before
separation). Next, we assume that the creation of a new surface
due to the dissection is the only dissipation present during the test,

![Fig. 13 Representative histological images illustrating the “roughness” of the generated dis-
section surface during peeling in the (a) circumferential and (b) axial directions. EVG staining,
4 $\mu$m thick sections. Original magnification 100$\times$.](image_url)
According to this method, the dissection energy is given as
\[ W_{\text{dissect}} = P_{\text{pc}} \left( L_{\text{pc}} - L_{\text{aortic-media}} \right)/L_{\text{medial}} \]
where \( P_{\text{pc}} \) is the peak pressure, \( L_{\text{pc}} \) is the length of the aneurysm, \( L_{\text{aortic-media}} \) is the length of the aortic media, and \( L_{\text{medial}} \) is the length of the medial dissection. For the porcine aorta, this relationship was found to be
\[ W_{\text{dissect}} = 5.1 \pm 0.6 \text{ mJ/cm}^2 \text{ (mean } \pm \text{ SD, } n=5) \] for the upper descending thoracic aorta and
\[ W_{\text{dissect}} = 7.6 \pm 2.7 \text{ mJ/cm}^2 \text{ (mean } \pm \text{ SD, } n=7) \] for the lower thoracic aorta. A quantification of the energy required to dissect the aortic media is provided in several works in the literature. For example, one experimental method generates medial dissections by the creation of a bleb due to the infusion of a fluid into the media. According to this method, the dissection energy is given as
\[ W_{\text{elastic}} = \frac{\text{stress} \times \text{strain}}{2} \]
where the stress is the peak pressure and the strain is the change in length of the aortic media. Hence, these data represent the sum of the dissection energy. The clinical circumstances of dissection are more varied than the controlled protocol of these experiments. Consequently, these data might not be representative for in vivo aortic dissection, where fatigue-like and multiaxial loading is present. However, we think that the data are particularly useful for the estimation of the constitutive response of dissections due to balloon angioplasty, where quasistatic and mainly Mode-I loading conditions appear (Gasser and Holzapfel [45]).

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