Natural history studies have shown that focal articular surface defects in the human knee may progress to degenerative arthritis. Although the risk of defect progression to degenerative arthritis is multifactorial, defect size is important. To date, the “critical” or “threshold” defect size at which biomechanical forces become potentially damaging to adjacent cartilage has not been clearly identified. As a result, the size at which articular surface restoration needs to be considered has not been well defined.

A review of the literature reveals that there is a wide variation in threshold sizes at which authors choose to treat articular surface defects. Brittberg et al, using carbon plugs to reconstruct articular surface defects, defined the minimal defect size as 1.2 cm². In another study using periosteum and cultured chondrocytes, Brittberg et al treated defects 1.6 cm² and larger. Homminga et al used perichondrium to treat defects 1 cm² and larger. Lorentzon et al used periosteum to treat defects 0.75 cm² and larger, and Niederman et al used periosteum to treat defects 3 cm² and larger. Minas used autologous chondrocyte transplantation to treat defects 1.5 cm² and larger.

Table 1 demonstrates this variation in threshold sizes in terms of the area of the defect and the diameter of an equivalent circular defect. Most contemporary algorithms reference the size “2 cm²” when guiding the treatment of...
articular surface defects. However, this is not a threshold size per se but a size guideline for choosing between osteochondral autografting and autologous chondrocyte transplantation.

When defining treatment according to defect size, it appears that the treatment of articular surface lesions is not based on any firm biomechanical data. The purpose of our study was to determine the effect of defect size on defect rim stress concentration, peak rim stress, and load redistribution to adjacent cartilage over the weightbearing area of the medial and lateral femoral condyles in the human knee. Based on our model of cartilage loading redistribution to adjacent cartilage on the weightbearing area of the medial and lateral femoral condyles in the human knee. Based on our model of cartilage loading redistribution to adjacent cartilage over the weightbearing area of the medial and lateral femoral condyles in the human knee. Based on our model of cartilage loading redistribution to adjacent cartilage over the weightbearing area of the medial and lateral femoral condyles in the human knee. Based on our model of cartilage loading redistribution to adjacent cartilage.

**METHODS**

Eight fresh-frozen cadaveric knee specimens were used for this study. They consisted of 5 left and 3 right knees from 5 cadavers. Specimens were selected taking care that no gross malalignment or arthritis was present. Two additional right knees were excluded because of degenerative changes observed in the articular surface. Age ranged from 78 to 91 years, and gender was 75% female and 25% male.

Each knee was loaded to 100 N, and slight adjustments were carried out in the medial/lateral positioning of the knee to ensure that both the medial and lateral compartments of the knee were loaded equally based on total load values under the initial 100 N of compression, as measured by each of the 2 sensors. This position was maintained for the duration of the test. Load on the knee was zeroed before each test while unloading the knee within the loading frame, such that the articular surfaces began to separate. No tethering of the sensors was needed in this testing configuration because the friction between the sensor and the knee was sufficient to maintain positioning. In addition, axial loading was centered on the sensors and did not force them to reposition. A circumferential capsulotomy was next carried out. Each knee with intact articular surfaces was loaded from 100 to 700 N at 100 N/s and held at 700 N for 5 seconds. The knees were not subjected to preconditioning. Dynamic pressure readings were recorded throughout the loading and holding phases. Peak values were used for the analysis of rim stress.

A marker was next used to define the center of each femoral condyle at 30° of flexion. An anterior arthrotomy was made, and digital electronic pressure sensors (K-scan 4000, Tekscan, Boston, Mass) were placed into the medial and lateral compartments of the knee, above the menisci. Prior to use, each sensor was equilibrated and then calibrated in situ in the knee joint. The sensors used in this study were thin (0.089 mm) and flexible, measuring 28 × 33 mm, and considered a high-resolution sensor with 62 sensels/cm². Sensels were oriented in a grid with 1.27-mm spacing between rows and columns.

The knee was next loaded as previously described. This loading was repeated for 8-, 10-, 12-, 14-, 16-, 18-, and 20-mm-diameter defects of constant depth enlarged circumferentially around the initial 5-mm defect. A relaxation period of 30 minutes was allowed between tests, during which the knees were irrigated with physiologic saline solution. A small caliper was used to ensure that the defects were enlarged in a concentric manner. The width of the medial and lateral femoral condyles was recorded for each specimen.

**TABLE 1**

Variation in Threshold Size Used by Different Authors When Choosing to Resurface Focal Articular Surface Lesions

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Method*</th>
<th>Size, cm²</th>
<th>Equivalent Diameter, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Niederman et al²⁷</td>
<td>1985</td>
<td>Periosteum</td>
<td>3</td>
<td>19.5</td>
</tr>
<tr>
<td>Homminga et al²²</td>
<td>1990</td>
<td>Perichondrium</td>
<td>1</td>
<td>11.3</td>
</tr>
<tr>
<td>Brittberg et al¹</td>
<td>1994</td>
<td>Carbon plugs</td>
<td>1.2</td>
<td>12.4</td>
</tr>
<tr>
<td>Brittberg et al²</td>
<td>1994</td>
<td>Periosteum/ACT</td>
<td>1.6</td>
<td>14.3</td>
</tr>
<tr>
<td>Lorentzon et al¹⁹</td>
<td>1996</td>
<td>Periosteum</td>
<td>0.75</td>
<td>9.8</td>
</tr>
<tr>
<td>Messner and Gillquist²⁰</td>
<td>1996</td>
<td>None</td>
<td>1</td>
<td>11.3</td>
</tr>
<tr>
<td>Minas²²</td>
<td>1999</td>
<td>ACT</td>
<td>1.5</td>
<td>13.8</td>
</tr>
</tbody>
</table>

*ACT, autologous chondrocyte transplantation.
Peak pressure distribution around each defect size in the medial and lateral femoral condyles was recorded. A center point was manually determined using the Tekscan graphical interface (ISCAN software) based on the pressure distribution in each condyle. This interface allows the user to query the coordinates of points along a pressure distribution, which can be used to calculate the center of a given region. This center point corresponds with the center of the circular unloaded region, shown as a black circle in the pressure distributions in the bottom of Figure 1. To assess data regarding the location of peak loading in the knee, the radial distance from the center point to the area of peak stress was calculated. The radius of the maximum pressure may be dictated by the pressure distributions along the rim of the defect or by the circumferential loading of other anatomical structures such as the meniscus. This radius was compared to the radius of the defect for each defect size. An analysis of variance (ANOVA) with Bonferroni correction was performed to evaluate the significance of the location of peak stress for each defect size.

RESULTS

Analysis of the data demonstrated that for intact cartilage with no defect, there was a peak stress observed that correlated with the loading of the inside margin of the meniscus. For defect sizes of 5 and 8 mm, peak stresses resembled those observed with no defect in terms of distribution, as illustrated in Figure 2. Effectively, the development of defect rim stress was obscured by loads carried by the meniscus. Hence, for defects 8 mm and smaller, stress was not shown to concentrate around the rims of the defects. Peak stresses in this size range occurred independently and were based on loading patterns dominated by the menisci. These findings were based on observed geometry of the menisci, known defect sizes, and comparisons with the pressure distributions in the intact articular surface.

For defects 10 mm and greater, rim stress concentration was evident as the distribution of peak loads that followed the edge of the defect with a mean distance from the edge of the rim of 2.2 ± 1.5 mm on the medial condyle and 3.2 ± 1.9 mm on the lateral condyle (P = .0025).

A representative example of peak stress and rim stress concentration occurring around a 12-mm defect is shown in Figure 1. The relationship between peak stress and location is demonstrated in Figure 2, with a dot-dashed line to represent the actual radius of the rim of the defect. The distance from the defect rim to the peak stress is illustrated in Figure 3. For defects from 10 to 20 mm, a fairly consistent distance was established, whereas a slight increase in radius was observed at about 20 mm. This slight increase was attributed to collapse of the defect rim as the diameter approached the full width of the condyle. Furthermore, the pattern of distribution observed in the data for defect sizes 8 mm and smaller was distinctly different than for the remaining defect sizes, as demonstrated by the standard deviation, variance, and range values shown in Table 2. Subsequently, these data were each separately analyzed. For defect sizes 8 mm and smaller, an ANOVA with Bonferroni correction was used to compare the locations of peak stresses. Data for this group of defects demonstrate a decreasing trend in radius of peak pressure with increasing defect size (P = .0032), suggesting that as defect size increased from 0, the influence of the defect rim slowly overcame the influence of the meniscus. A second ANOVA with Bonferroni correction was used to compare peak stress locations for various defect sizes of 10 mm and greater. This group of defects demonstrated an increasing radius of peak pressure with increasing defect size (P = .0011), suggesting that the peak pressure follows the rim of the defect. The average medial condylar width was 30.0 ± 7.5 mm, and the average lateral condylar width was 31.3 ± 5.0 mm.

Evaluation of peak contact pressure values around the rims of the defects revealed that for defects in the 10 to 20 mm range, the peak value did not demonstrate a significant change (P = .4502). As the defects were enlarged, peak pressure values remained fairly constant. Figure 4 demonstrates this relationship between defect size and peak rim contact pressure values. Mean peak rim contact pressures around defects were 4844 ± 2096 kPa in the medial femoral condyle and 4327 ± 2197 kPa in the lateral femoral condyle. The mean value was 4585 ± 2155 kPa for both medial and lateral sides combined.

Dynamic evaluation of the loading phase revealed fairly consistent load redistribution and contact area recruitment that kept approximate pace with the central contact pressure distribution, which can be used to calculate the center of a given region.
area loss associated with defect enlargement. Dynamic evaluation of the 5-second holding phase revealed a fairly consistent redistribution of load from the point of peak stress to adjacent areas. Most of this load redistribution occurred rather rapidly within the first second of the holding phase. For each test, this was seen as a slight diminu-

**Figure 2.** The relationship between the radius from the center of the defect to the point at which peak pressure was recorded and the outer rim of the defect. The broken line represents the rim of the defect as the defect was enlarged from 0 to 20 mm in diameter. Note that at a defect size of 10 mm, the relationship becomes relatively constant. Also note that that defect size represents the diameter of the induced defect, whereas the radius of the maximum pressure may be dictated by the pressure distributions on the rim of the defect or by the circumferential loading of other anatomical structures such as the meniscus.

**Figure 3.** The relationship between the distance from the point of peak pressure to the rim of the defect and defect size. Note that at 10 mm, this relationship becomes fairly constant. These distances are derived from data presented in Figure 2 and are used to demonstrate the location of the peak pressure distribution with respect to the rim of the defect.
tion in the peak stress value, with a dissipation of stress to immediately adjacent areas. These observations were not statistically analyzed.

DISCUSSION

The relationship between focal articular injury and osteoarthritis is still under investigation. The similar biological, mechanical, and macroscopic features indicate that both conditions may indeed be a continuum of joint degeneration.\(^5\) This is the primary rational for early intervention in symptomatic patients with focal articular surface damage. Although natural history studies have shown that focal chondral and osteochondral defects progress to osteoarthritis,\(^{15,18,21}\) this claim is difficult to prove in a prospective fashion, likely because of the inability to accurately diagnose articular surface injuries by noninvasive techniques and then follow them out over long periods of time.\(^{23}\) In addition, lesion progression is multifactorial.\(^{22}\) Lesion characteristics such as size, depth, location, chronicity, and response to initial treatment are undoubtedly important in lesion progression. Patient factors such as age, level of activity, obesity, and genetic makeup also contribute. Finally, comorbidities such as cruciate deficiency, limb malalignment, and meniscal damage all factor into a lesion’s risk for progression to osteoarthritis.\(^{5,7,11}\)

### Table 2

<table>
<thead>
<tr>
<th>Defect Diameter</th>
<th>Mean Radius of Maximum Stress (mm)</th>
<th>Standard Deviation</th>
<th>Variance</th>
<th>Quartile 1</th>
<th>Median</th>
<th>Quartile 3</th>
<th>Range</th>
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</thead>
<tbody>
<tr>
<td>0</td>
<td>11.83</td>
<td>2.32</td>
<td>5.3824</td>
<td>9.55</td>
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<td>13.79</td>
<td>7.72</td>
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<tr>
<td>5</td>
<td>9.23</td>
<td>2.56</td>
<td>6.5536</td>
<td>6.38</td>
<td>9.07</td>
<td>11.66</td>
<td>7.58</td>
</tr>
<tr>
<td>8</td>
<td>9.21</td>
<td>2.12</td>
<td>4.4944</td>
<td>8.84</td>
<td>9.16</td>
<td>10.78</td>
<td>6.47</td>
</tr>
<tr>
<td>10</td>
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<td>1.66</td>
<td>2.7556</td>
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<td>8.94</td>
<td>10.70</td>
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<td>1.33</td>
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<td>14</td>
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<td>11.27</td>
<td>5.77</td>
</tr>
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<td>20</td>
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<td>1.1449</td>
<td>10.62</td>
<td>11.11</td>
<td>12.11</td>
<td>3.70</td>
</tr>
</tbody>
</table>

Note the standard deviation, variance, and range of data for the 0-, 5-, and 8-mm defect sizes, suggesting that these data sets are from a different distribution.

**Figure 4.** The relationship between peak pressure and defect size. Note that peak pressure values did not increase significantly as the defects were enlarged but remained relatively constant.
Although a mechanical explanation for the breakdown and degeneration of cartilage adjacent to the edges of defects makes intuitive sense, the relationship between defect size and mechanically induced degeneration secondary to local stress concentration has not been well defined. Brown et al, although showing rim stress concentration around small 2-mm defects in a dog model, were unable to correlate increasing defect size with increasing peak rim stresses. On a biomechanical level, Brown et al demonstrated stress aberrations following imprecise reduction of intra-articular knee fractures, and Lefkoe et al showed that incongruity or step-off of articular cartilage leads to degeneration of cartilage and subchondral bone that mimics osteoarthritis. Radin and Burr suggested that although a mechanical stimulus is necessary for normal articular cartilage maintenance and repair, a reduction in local stress concentrations around defects favors repair. Huber-Betzer et al showed that strong gradients of contact pressure cause elevated shear stress in the deep cartilage layers. Given the importance of pressure-driven, cyclic, interstitial fluid transport in normal cartilage, high contact pressures or shear stresses adjacent to defects may very well interfere with the ability of chondrocytes near these defects to function normally, leading to their demise. Mitchell and Shepard suggested that local compression of cartilage either allows certain chondrocytes to heal or prevents the ingrowth of granulation tissue from the subchondral bone.

Animal studies have provided insight into the relationship between defect size, rim stress, and cartilage degeneration. Studies by Depalma et al, Calandruccio and Gilmer, and Converry et al have shown that small osteochondral defects in animals generally heal with fibrocartilage, and adjacent cartilage does not undergo degenerative change. However, Nelson et al, in an in vivo canine study, showed that the highly compliant fibrocartilaginous tissue filling a 6-mm defect was incapable of reducing contact stress at the rim of the defect. Converry et al, in a study using Shetland ponies, showed that an osteochondral defect 9 mm or larger in the weight-bearing area of the femoral condyle leads to degeneration of the opposing tibial articual surface, as well as the cartilage immediately adjacent to the defect. Jackson et al recently showed that a 6-mm osteochondral defect in a goat model demonstrated resorption of the osseous wall, formation of a large cavitary lesion, and collapse of surrounding bone and cartilage. They postulated that the size of the defect reaches a critical diameter at which increased compressive forces lead to mechanical overloading of surrounding bone and cartilage. O’Driscoll et al and Wei and Messner have shown that the cartilage adjacent to the repair area after chondrocyte/perioseum transplantation shows progressive degeneration. Messner and Gillquist in their critical review of cartilage repair, referred to this detrimental loading adjacent to defects as “edge stress.”

Clinical studies in humans also have pointed to a relationship between larger defect size and degeneration of the articular surface. Linden demonstrated that the presence of a significant chondral defect (ie, clinically detectable osteochondritis dissecans) is associated with a much higher incidence of osteoarthritis than occurs in the general population. He noted that clinically significant disease appears approximately 1 decade earlier than disease associated with idiopathic osteoarthritis. Hughston et al showed that larger osteochondral defects are associated with a poorer clinical outcome than smaller defects. Recently, Messner and Gillquist evaluated 31 patients followed for 14 years. Patients in the study were noted to have unipolar, unicompartmental lesions. The minimal lesion size for inclusion in the study was 1 cm². Although 22 patients continued to have excellent knee scores at a 14-year follow-up, 12 of these patients had radiographic joint space reduction of more than 50%. Although biomechanical, animal, and clinical models all point to a relationship between defect size, local stress concentration, and degeneration of cartilage, this relationship has not been clearly defined. In our current study, the occurrence of rim stress concentration occurring around defects, as well as peak stress occurring slightly outside the rims of these defects, is compatible with the findings of previous studies. However, our study shows that rim stress concentration becomes a factor for defects 10 mm in diameter (0.79 cm²) and greater in size and that biomechanically, a “threshold effect” does indeed exist. This finding is in contrast to indications that a significant biomechanical alteration in local contact stress is appreciated at about 2 cm² (16-mm defect), which is most commonly quoted size in contemporary treatment algorithms. This commonly referenced size seems to be based loosely on clinical studies by Homminga et al and Brittberg et al.

Osteochondral lesions created in the medial femoral condyle showed statistically significant increases in peak rim stress values when compared to lesions in the lateral femoral condyle. Although this finding could be the result of our loading model, this would be unlikely because of the fact that loading began equally between the medial and lateral femoral condyles. It is our assertion that differences such as condylar shape and meniscal morphology contribute to the discrepancy in peak pressures around defects seen between the medial and lateral femoral condyles. Additional biomechanical study would be required to determine the value of these observations and the merits of loading the knee in a locked position versus using a bearing system designed to evenly redistribute the load. However, clinically, this notion that the medial femoral condyle might be more sensitive to altered biomechanics than the lateral condyle is supported by Curl et al, who reviewed 31516 knee arthroscopies and found that grade IV chondral lesions were predominately located on the medial femoral condyle.

Although peak stress values around defect rims did not increase from 10 to 20 mm, our ability to make dynamic observations may offer an explanation for this. We observed stress to redistribute as points of peak contact pressure dissipated load to surrounding cartilage within the first second of the 5-second holding phase of each test cycle. This observation is presumably because of deformation of bone and cartilage adjacent to the defect. As defect size is increased, the rim of the defect may deform more under a given load, especially for larger defects in smaller...
femoral condyles, where there is less medial and lateral osteochondral support adjacent to the defect. The relationship of the defect size to condylar surface area may, therefore, also be a factor as defect size is increased. This deformation, although decreasing local peak contact pressures, may lead to deeper shear stress, as previously discussed. Although the consequence of this shear stress is not clear, it may be detrimental to chondrocyte viability. Although our study did not evaluate the effect of loading or deformation on shear stress, future study may reveal that not only is the absolute value of the contact pressure or stress important but also the deformation of the bone and cartilage and resultant shear stresses that are produced. A 10-mm defect size may indeed be the critical size at which rim stress concentration becomes a factor under compressive loading, but cartilage deformation and shear stress may have an even greater effect on adjacent cartilage with subsequent enlargement of the defect. Further research in this area is needed.

Shortcomings of this study include the following: (1) this is a rather simplistic biomechanical model that can only vaguely approximate what actually occurs in a dynamic living system; (2) our model loaded the defects in a concentric manner, whereas most defects in a living human knee would be loaded in a complex eccentric manner; (3) the study did not quantify the effect of compressive forces on deformation at the edge of the defects or the effect of these compressive forces on shear stresses within the deeper layers of the cartilage; (4) the study did not take into account the capacity for tissue tolerance or repair as occurs in a living system, nor did it address the effect of cumulative stress on cartilage adjacent to the defects; (5) the loading model did not replicate the magnitude of loading seen in a living system, nor did it replicate a typical impact-loading event in the human knee; and (6) the relationship between defect size and condylar width or sagittal radius of the condyle was not evaluated, and this relationship may indeed be crucial.

CONCLUSIONS

Altered loading secondary to focal articular surface defects in weightbearing areas of the knee may have important implications relating to the long-term integrity of cartilage adjacent to these defects and risk for progression to arthritis. Our biomechanical model suggests that a threshold effect does occur at which rim stress concentration becomes a factor around osteochondral defects. Although the risk of lesion progression to arthritis is certainly multifactorial, rim stress concentration and altered loading may cause degeneration of adjacent cartilage. Our biomechanical data may be used as a size reference to guide clinical decision making when choosing whether to treat osteochondral lesions in weightbearing areas of the knee.

ACKNOWLEDGMENT

The authors thank Mamtha Balasubramaniam, the staff biostatistician at the William Beaumont Hospital Research Institute, for her extensive work in the statistical analysis of this data.

REFERENCES


