(thus confirming the value of the model), between groups III and IV but not between groups I-II and III. Biochemistry: active Case was detected only in the contused groups III-IV and latent Case was within the same ranges in the four groups. So, total Case was significantly increased in the contusive group (IV). Its 20% decrease in group III was not significant and the 27% decrease of active Case was near the significant level.

LASE, in this prophylactic schedule, prevents lesions of the contusive cartilage. This 'chondroprotective' effect may be mediated through a pro-anabolic, more than an anti-catabolic mechanism.

**Effect of diacerheine (ART 50) on an experimental post-contusive model of OA**

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The so-called 'chondroprotective' activity of a drug has to be tested in vitro, but also in vivo. As clinical trials are time consuming (years), the use of experimental models of osteoarthritis (OA) are of benefit.

A closed contusion of the patella with a 1 kg-weight dropped from a height of 1 m leads to OA. NZ adult rabbits were sacrificed at day 49 post-contusion. Cartilages of both condyles and patella were assessed in a series of 6 µm sections, stained with hematoxylin and eosin. A morphologic score (macro + microscopic scores) was established using the Colombo scoring system (Arthritis Rheum 1983; 26: 875). Mean score is the mean of both condyles and patella; total score is the sum of the three sites. The 23 rabbits were assigned to four groups: I-controls (N = 6); II-control + ART 50 (N = 5); III-contusion + ART 50 (N = 6); IV-contusion (N = 6). ART 50 (3.5 mg/kg/day) was administered orally during 90 days between contusion and sacrifice, in groups II and III.

The two scores did not show statistical difference between groups I, II and III. The scores showed significant differences (P < 0.03) between groups I and IV (thus confirming the value of the model) and between groups III and IV (P < 0.05).

Diacerheine, with this prophylactic schedule, prevents lesions of the contusive cartilage and has a 'chondroprotective' effect.

<table>
<thead>
<tr>
<th></th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
<th>Group IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean score ± S.D.</td>
<td>3.02 ± 1.10</td>
<td>2.98 ± 0.92</td>
<td>3.91 ± 1.83</td>
<td>8.09 ± 1.62</td>
</tr>
<tr>
<td>Total score ± S.D.</td>
<td>10.57 ± 3.31</td>
<td>8.94 ± 2.75</td>
<td>11.73 ± 5.48</td>
<td>24.27 ± 4.87</td>
</tr>
</tbody>
</table>

**Attempts in vivo to mimic Rumalon’s (R) action in OA cartilage in the Pond-Nuki canine model**

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The ingredients of Rumalon (R) believed to be necessary for full efficacy in human treatment include: (1) sulfated chondroitins and an extract of bone marrow (believed to contain one or more growth factors). In the current study, these components were mimicked by use of a sulfated pyranoside Pentosan (SP-54) and IgF-1, both in combination and alone. Six weeks after AC ligament section and after 3 weeks of treatment with these agents, the study was terminated; articular cartilage was evaluated by gross appearance, histological staining and biochemical analysis.

In comparison to moderately severe lesions in untreated OA animals: (1) gross appearance was near normal only in the group treated with both agents; (2) histological (Mankin) scores indicated less severe disease in the SP-54 (alone) treated group (P < 0.01), and in the IgF-1 + SP-54 treated groups (P < 0.001) than in OA positive control; (3) suppression of active and total neutral metalloproteinase and restoration toward normal of tissue inhibitor of metalloproteinase (TIMP) was seen in these same groups. IgF-1, alone as administered, had no measurable favorable effects. Thus, significantly greater changes occurred with combination therapy. It is postulated that the protease inhibitor SP-54 may have protected the peptide IgF-1 from degradation or that chondrocytes respond to both factors optimally.