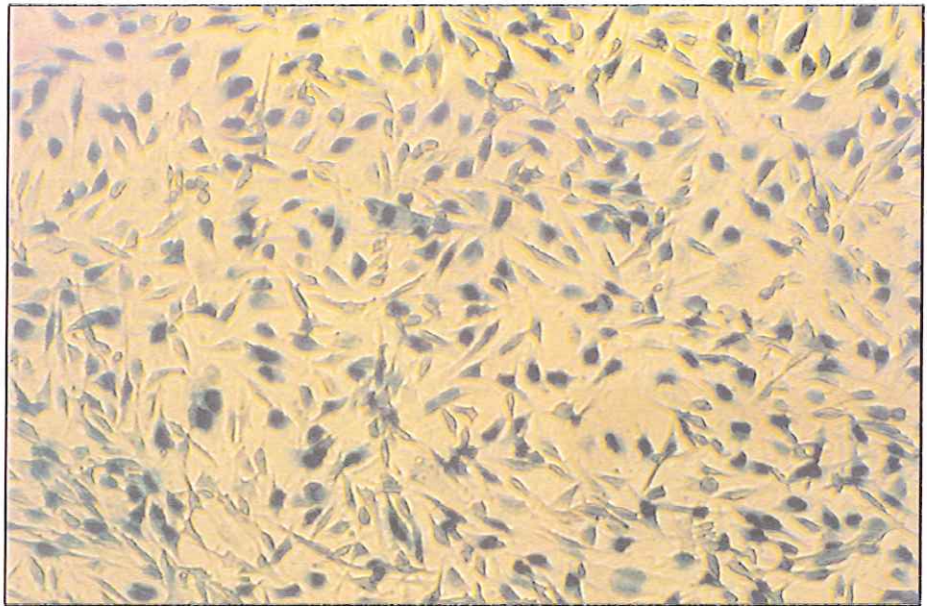


Figure 2a

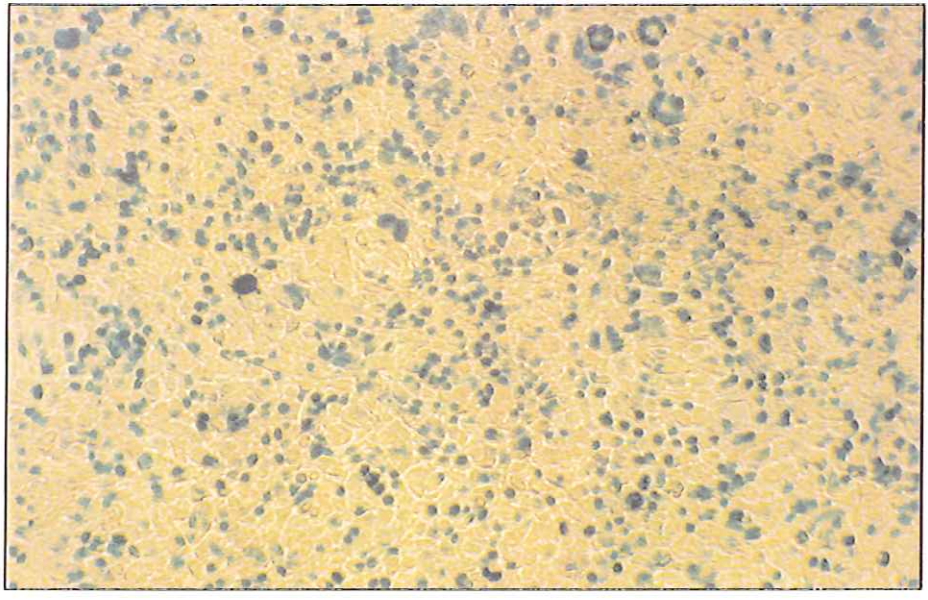


Figure 2b

Chapter 3, figure 3a

β–galactosidase expression in 2 slices of 9L brain tumor after injection of Ad.RSVβgal (a). Rats were injected stereotactically with $2.5x10^9$ pfu's in the tumor. Five days after injection the brains were fixed, cut in slices and stained with X-Gal. Microscopy (x20) of the part shown above (b). The slices were embedded in paraffin, cut in 2 mm sections and counterstained. The majority of the cells show β–galactosidase activity. A few *Lac*Z transduced normal brain cells are visible adjacent to the tumor. (x20) (c).

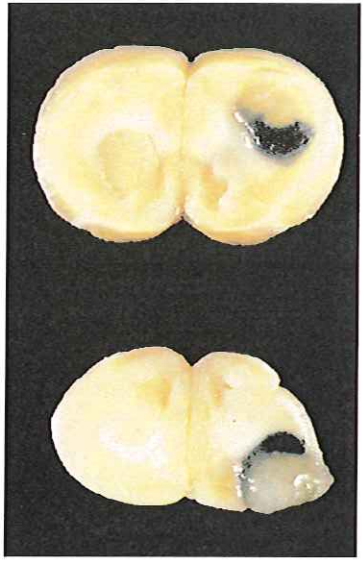


Figure 3a

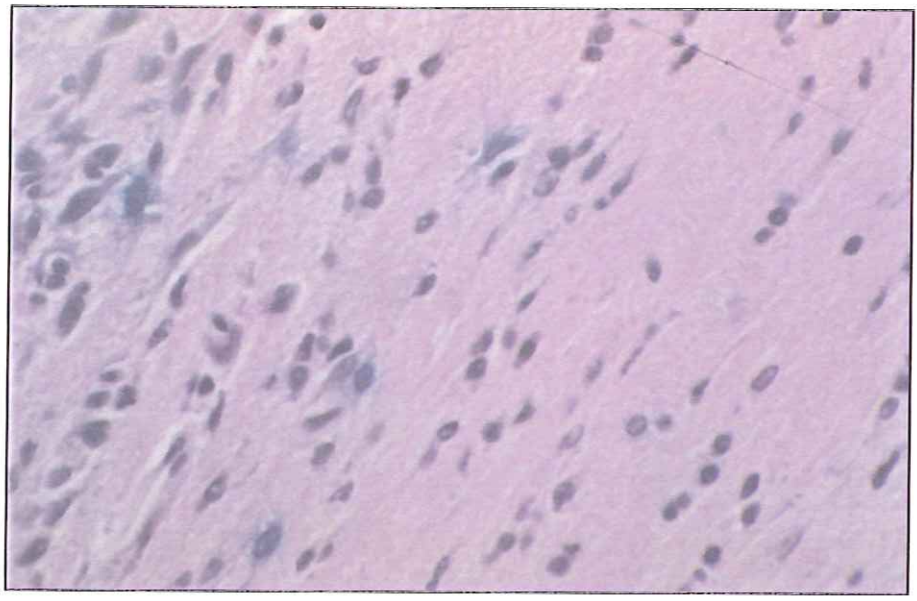


Figure 3b

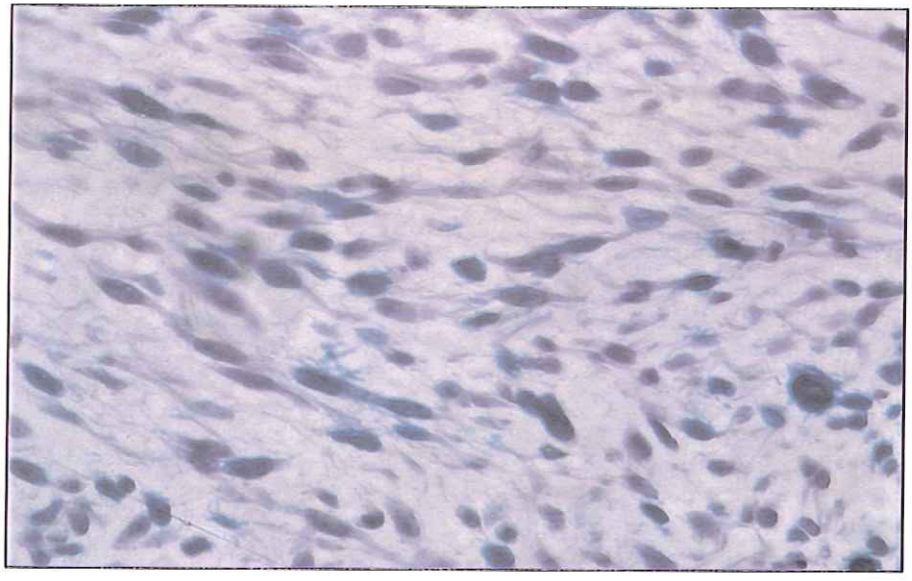


Figure 3c

Chapter 4, figure 4a, 4b, 4c, 4d

Microscopy of X-Gal stained brain and spinal cord containing leptomeningeal tumor cells

Figure 4a

Figure 4b

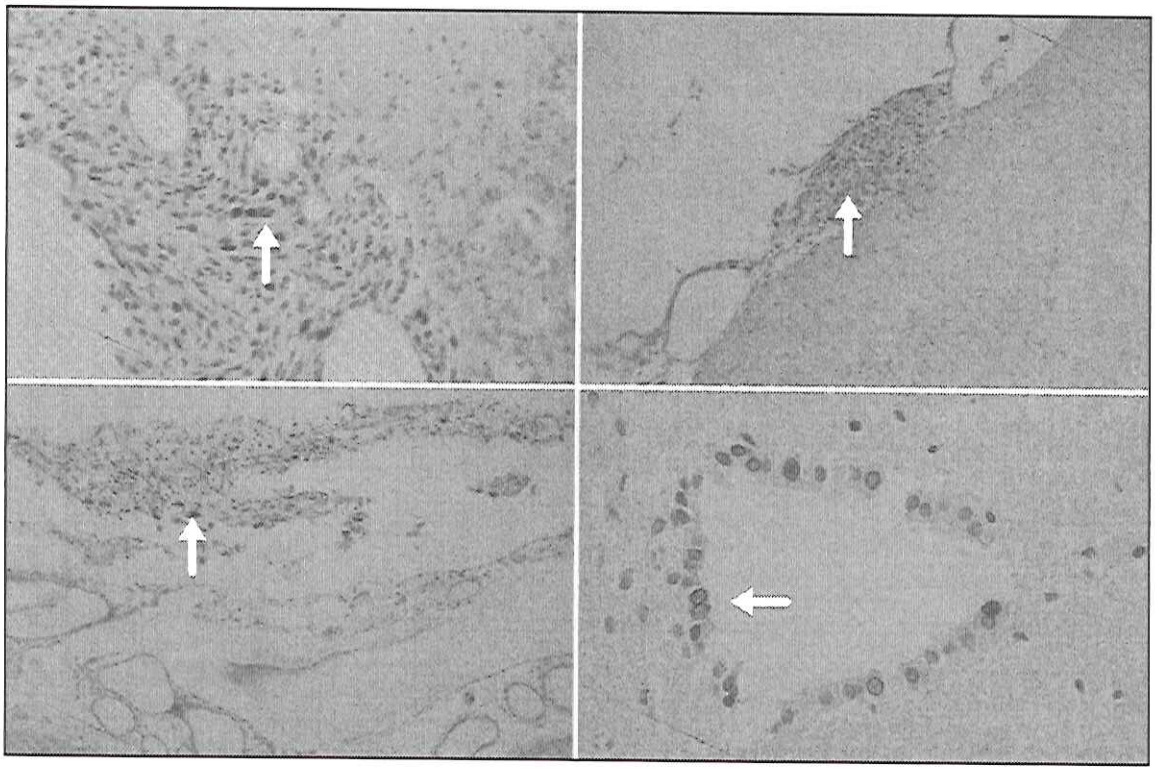


Figure 4c

Figure 4d

Chapter 5, figure 3a, 3b

Microscopy (x200) of human U251 tumor cells infected with IG.Ad.MLP.*Lac*Z (A) or IG.Ad.CMV.*Lac*Z (B) at m.o.i 100. Human tumor cells were plated in 24 well culture dishes (Costar) at a density of 104 cells/well. Cells in triplicate wells were infected at m.o.i. 0, 10, 100 or 1000. To asess β–galactosidase activity, the cells were stained with X-Gal 72 hours after infection and examined microscopically. All U251 tumor cells are transduced with the *Lac*Z gene. IG.Ad.CMV.*Lac*Z infected cells show enlargement of cells and nucleus/plasma ratio is enlarged as compared to the IG.Ad.MLP.*Lac*Z infected cells suggesting cytotoxicity.

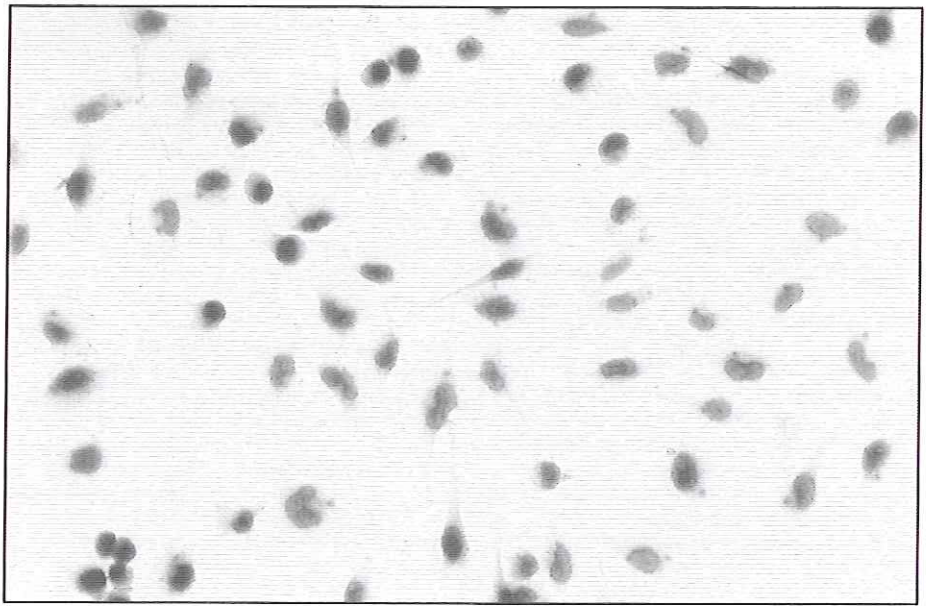


Figure 3a

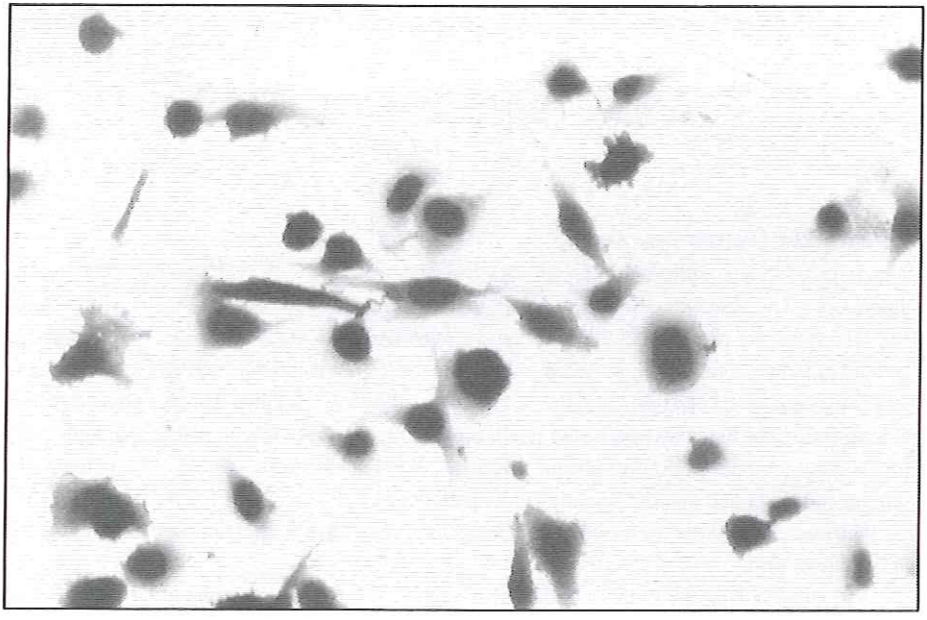


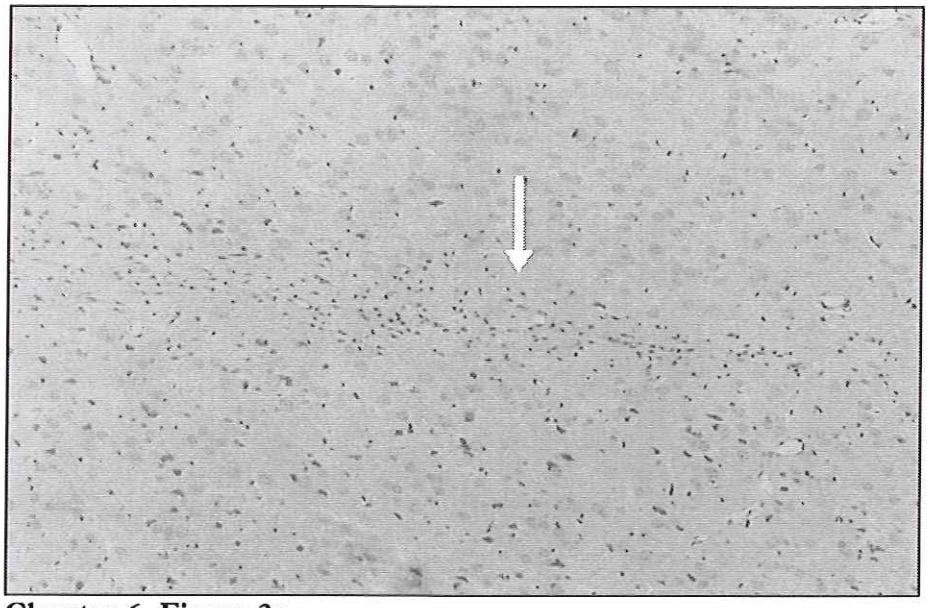
Figure 3b

Chapter 6, figure 2a, 2b

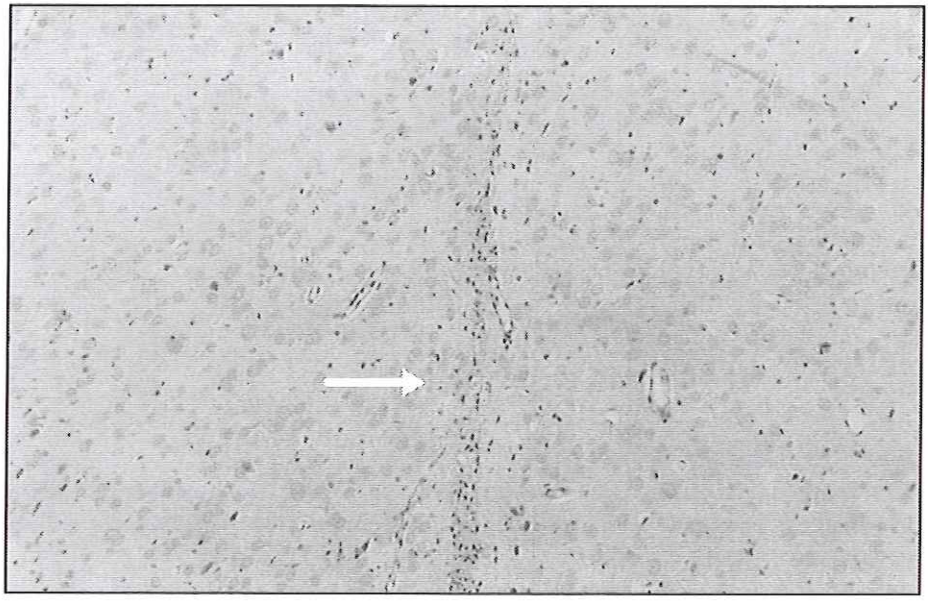
Photographs showing histopathology in rats injected intracerebrally with IG.Ad.MLPI.TK and subsequent GCV treatment. Rats were anaesthetized and injected stereotactically with 10^8 IU (1μl), 10^9 (10μl) IU IG.Ad.MLPI.TK, 10^8 (1μl) IU WtAd5 or PBS via a microliter syringe into the left forebrain, at a depth of 4mm. 24 hours later the rats were treated twice daily for 14 days with either intraperitoneal injections of GCV or PBS. The animals were killed 16 days after intracerebral injection. The brain was perfusion fixed, removed, cut transversely in 6 sections, embedded in paraffin, each section cut into 2 mm sections, counterstained with hematoxylin, phloxin and saffrane and examined microscopically by the pathologist.

2A: Reactive changes due to the injection procedure; mild edema, mononuclear infiltration and glia proliferation,

2B: Mild glia proliferation and mononuclear infiltration in the direct environment of the injection canal (10^8 IU IG.Ad.MLPI.TK+GCV).



Chapter 6, Figure 2a



Chapter 6, figure 2b

Chapter 6, figure 2c, 2d

2C: Moderate to severe glia proliferation, mononuclear proliferation, multifocal perivascular lymphoid cell infiltration, edema in the direct environment of the injection site (10^9 IU IG.Ad.MLPI.TK+GCV).

2D: glia proliferation, mononuclear infiltration , perivascular mononuclear infiltration and edema in the contralateral hemisphere (10^9 IU IG.MLPI.TK+GCV).

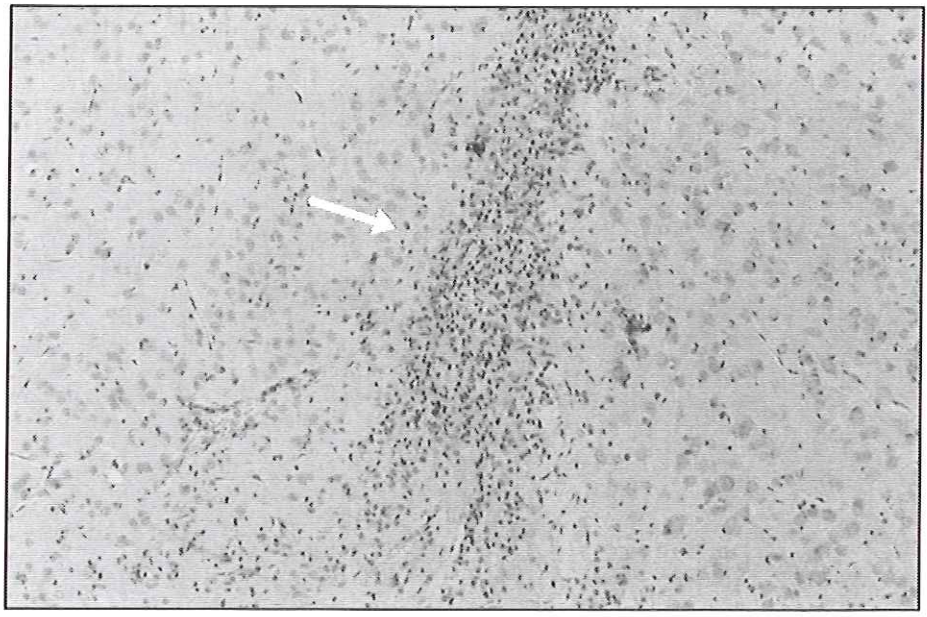


Figure 2c

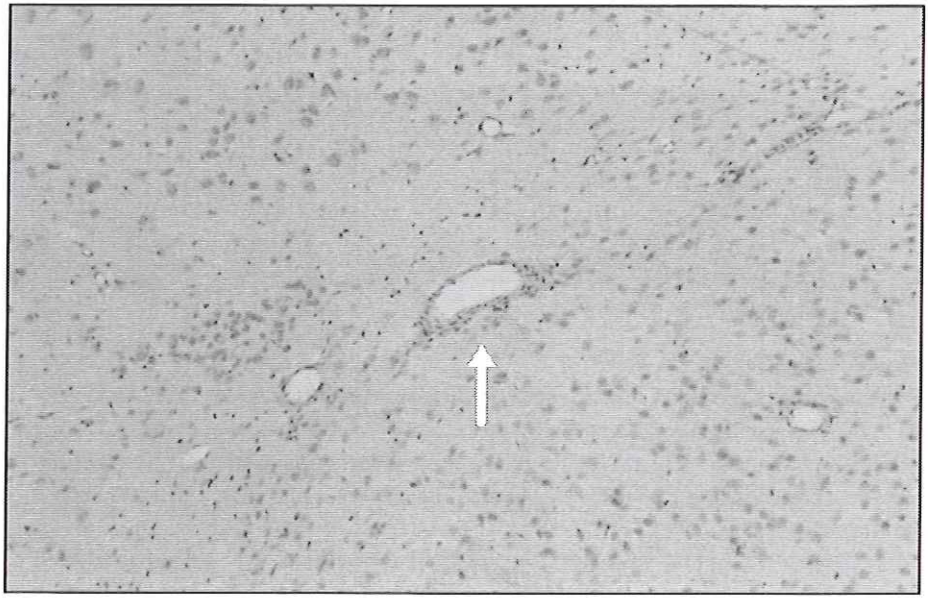


Figure 2d

Chapter 6, figure 4a, 4b

Photomicrographs showing X-Gal stained brain after injection of 10^8 IU IG.Ad.MLP.*Lac*Z.

Rats were injected intracerebrally with 10^8 IU IG.Ad.MLP.*Lac*Z and killed 3 days later. The brain was removed, cut with a vibratome into 100 μm sections and stained with X-Gal. The sections were then counterstained with hematoxylin/eosin and examined microscopically for reporter gene expression (blue cells).

4A: *Lac*Z reporter gene expression along the injection tract and along the meninges near the injection site.

4B: transduced cells along the corpus callosum in the contralateral hemisphere of the injection tract

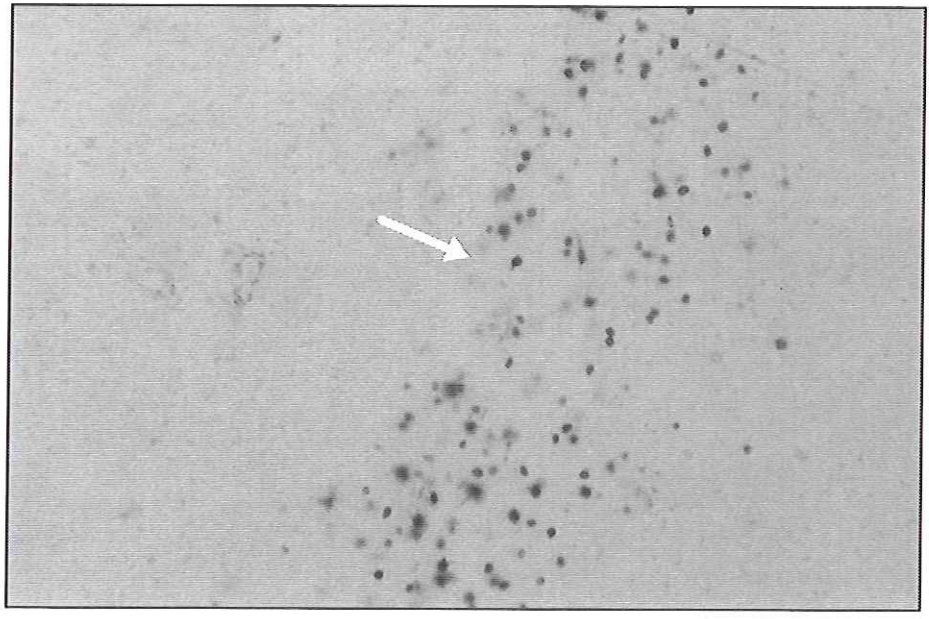


Figure 4a

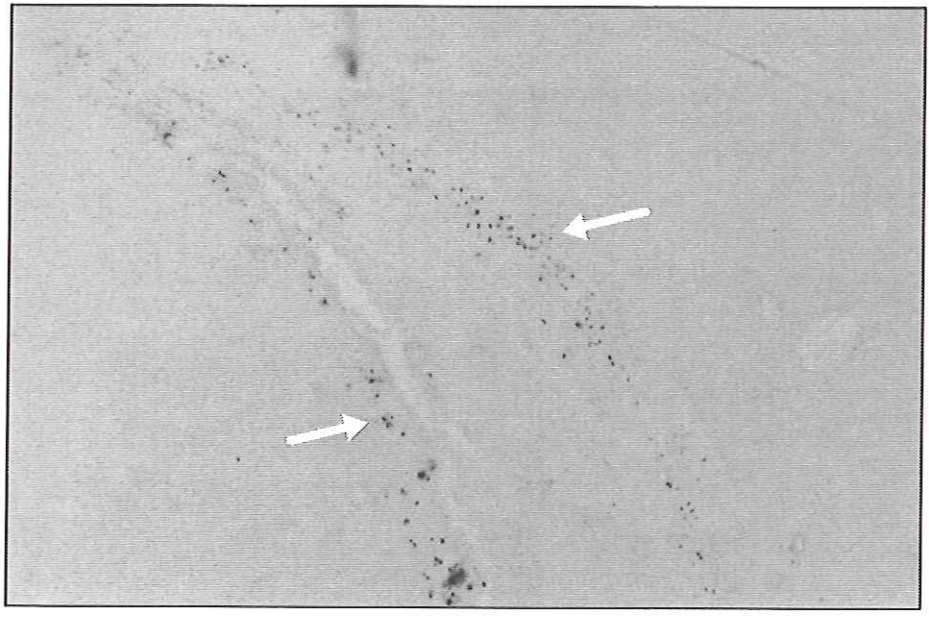


Figure 4b