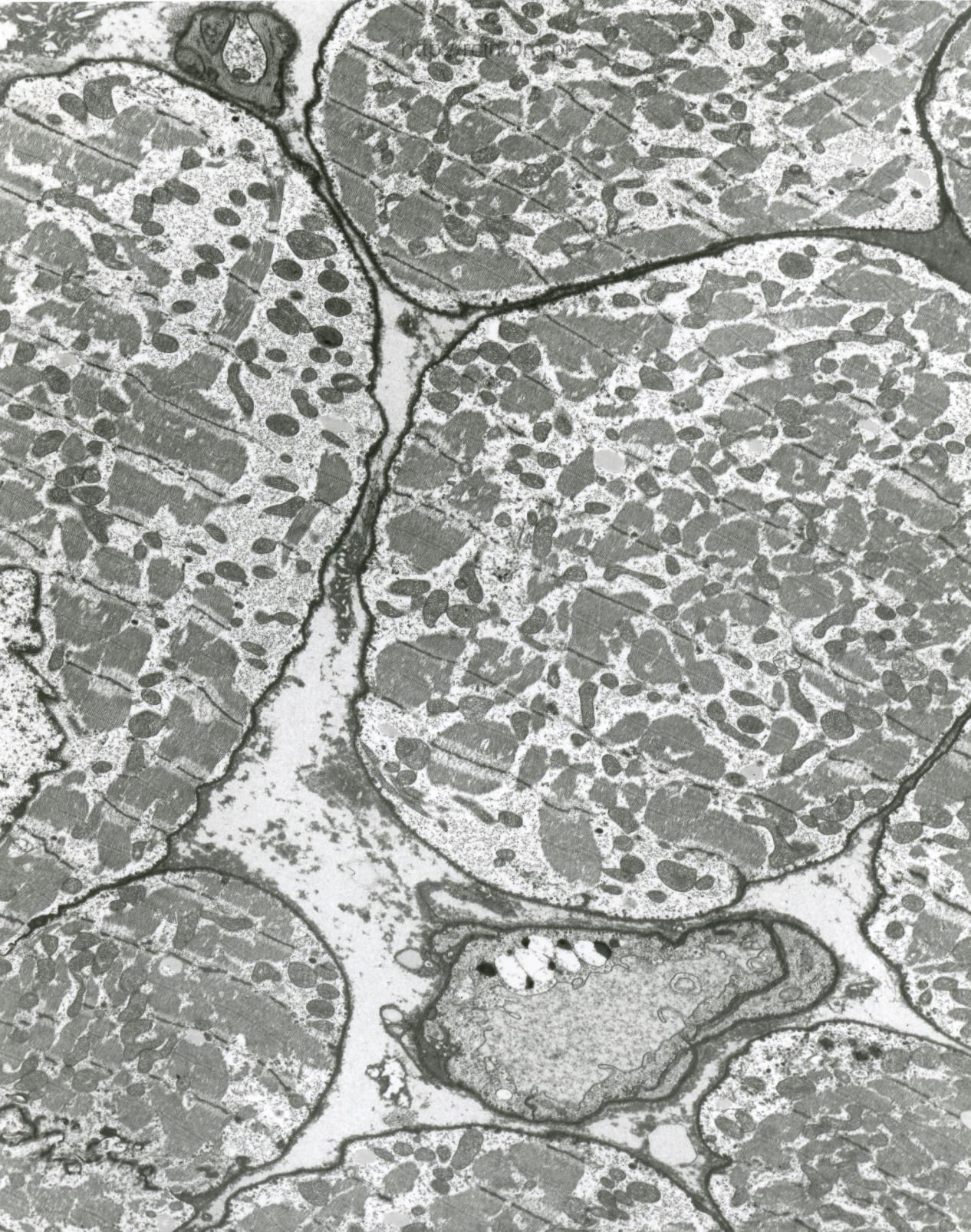


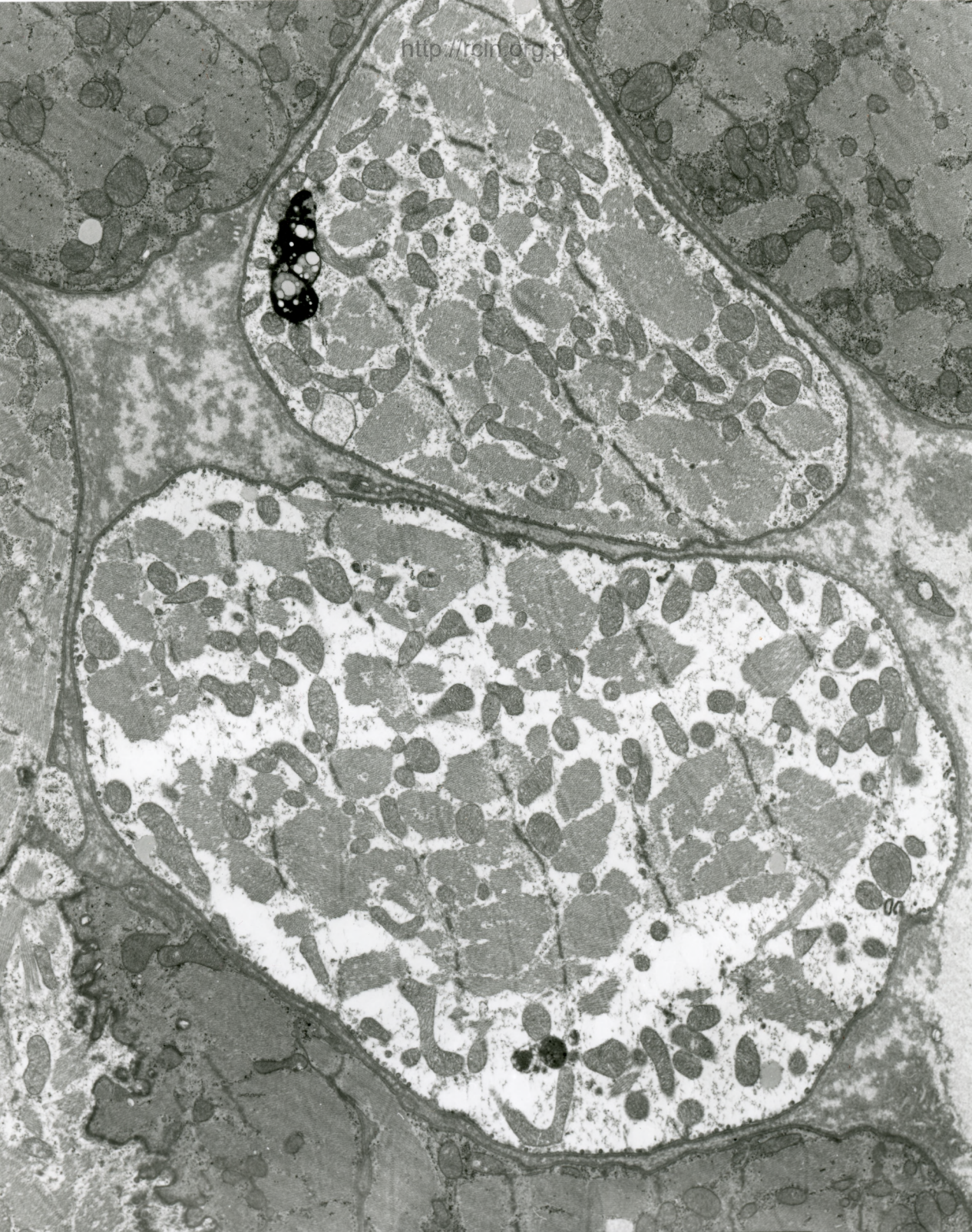
AF 29/89  
091411 80.0KV X2500 2µm

Fig. 1



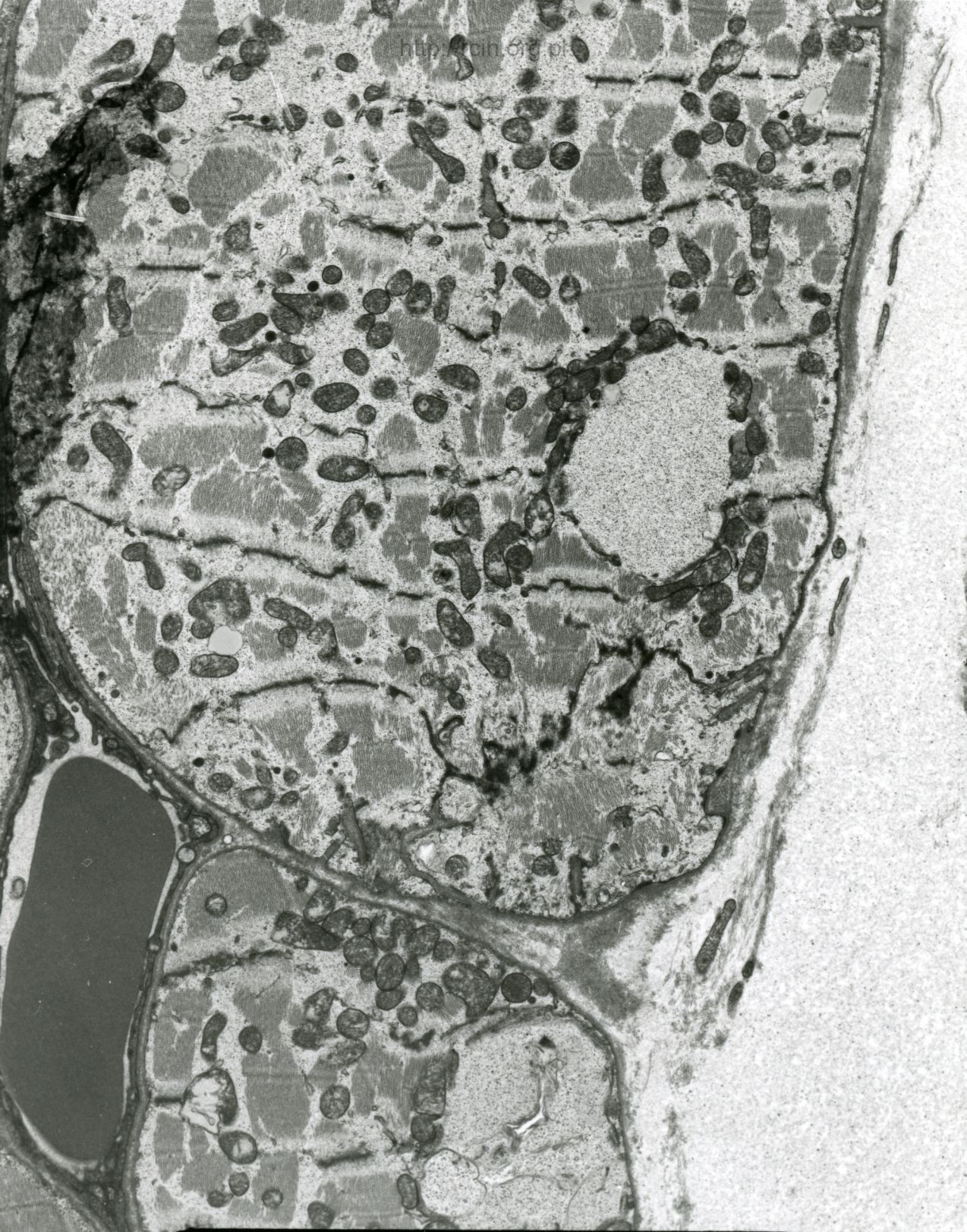
AF 30/09  
091412 80.0KV X2500 2µm

Fig. 2



AF 39/09/SERCE  
091270 80.0KV X3000 24

Fig. 3

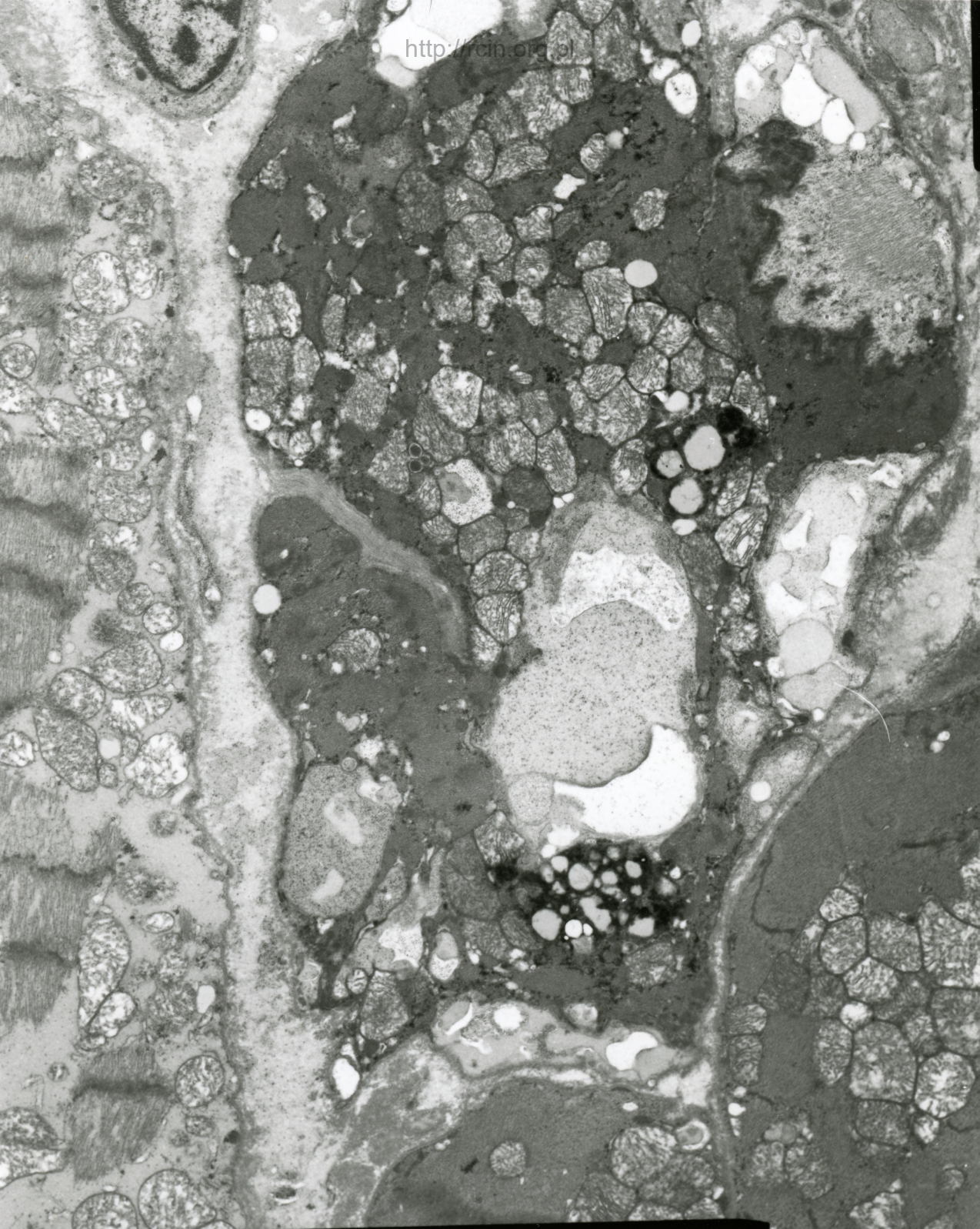


AF 39/09/C/SERCE  
100656 80.0KV X3000



2µm

Fig. 4



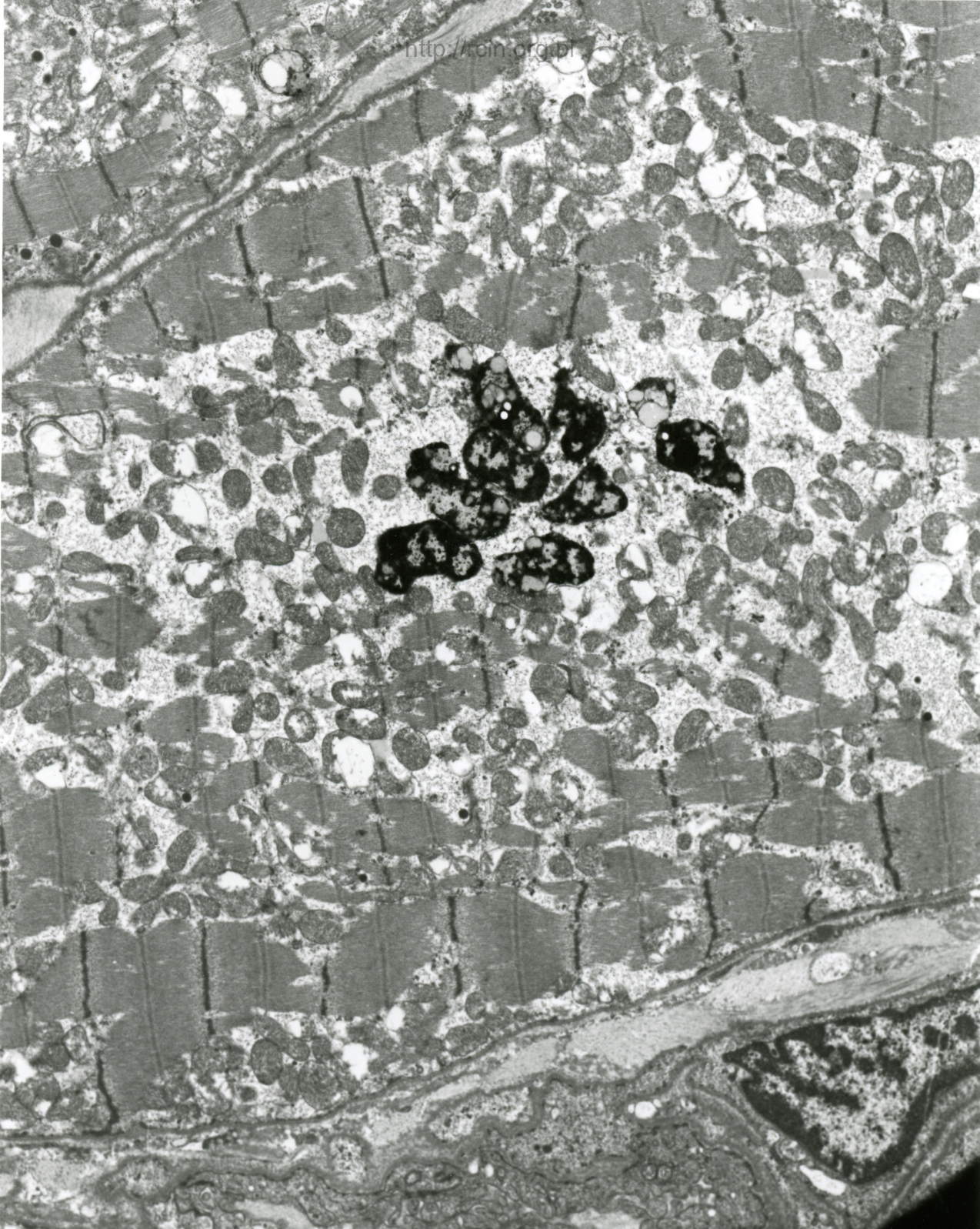
AF 39/09/C/SERCE  
100657 80.0KV X4000 2µm

Fig. 5



AF 39/89/C/SERCE  
100655 80.0KV X3000 2µm

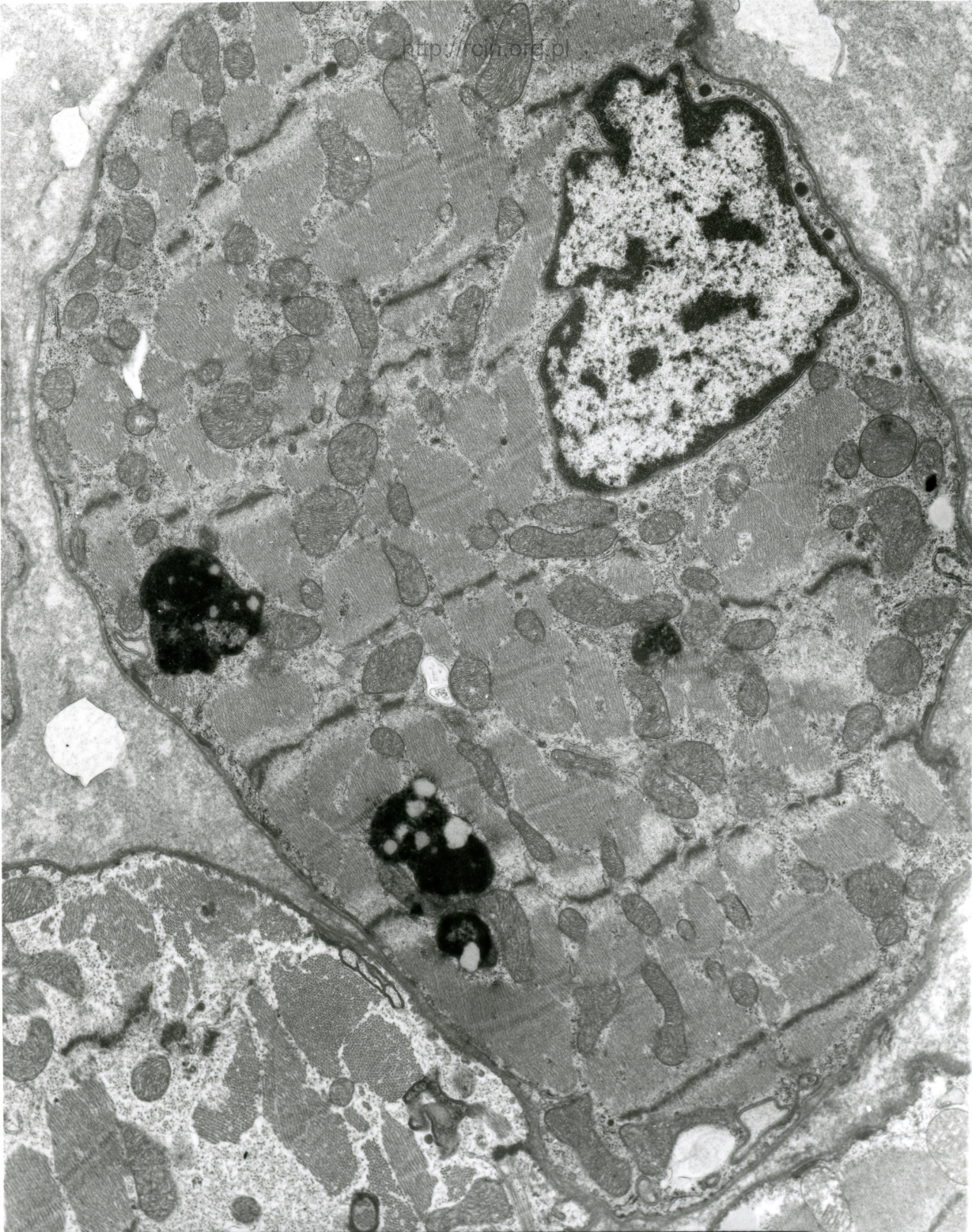
Fig. 6



AF 39/89  
091651 80.0KV X3000



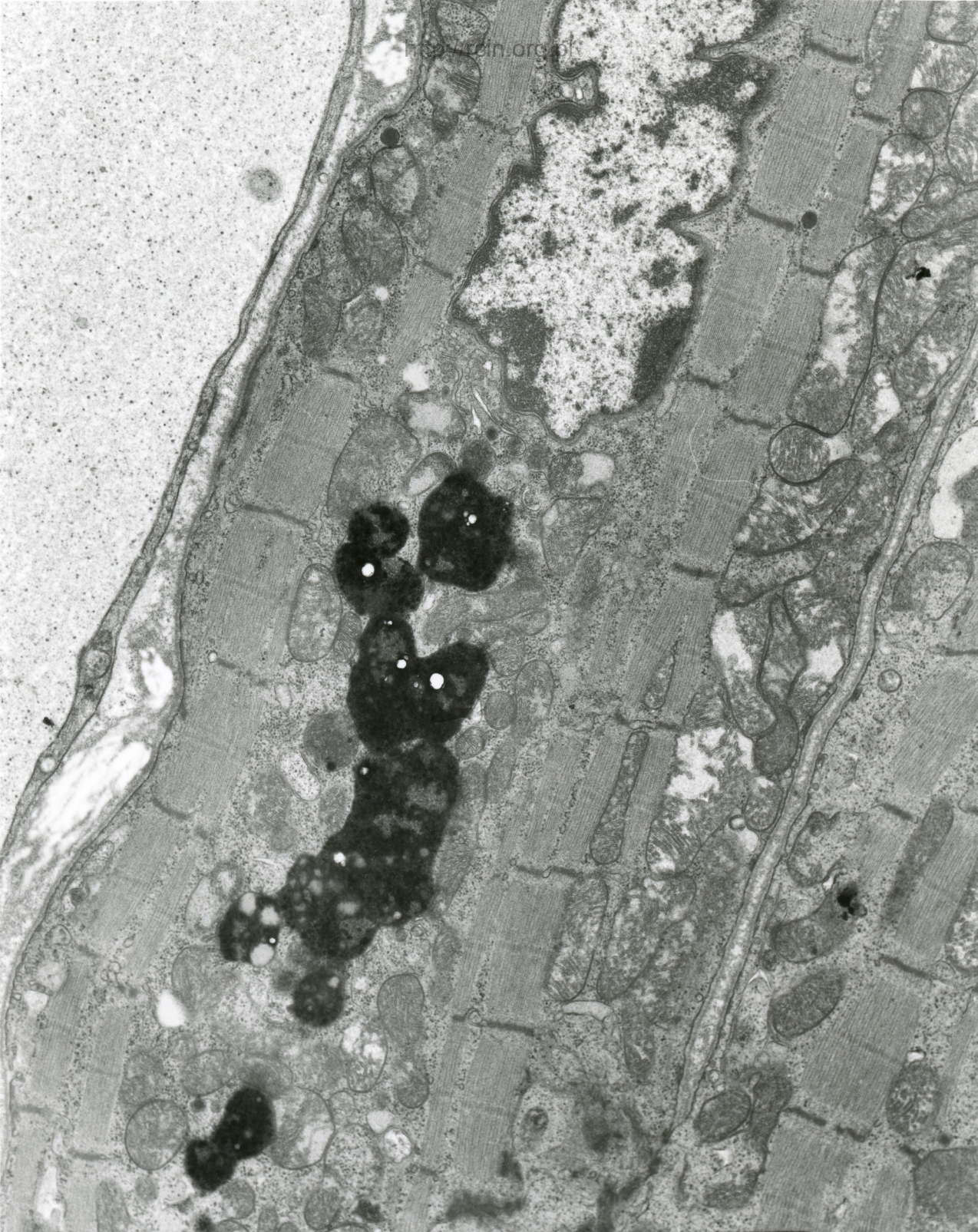
Fig. 7



AF 29/09/B  
AP133 00 OVU Y4000

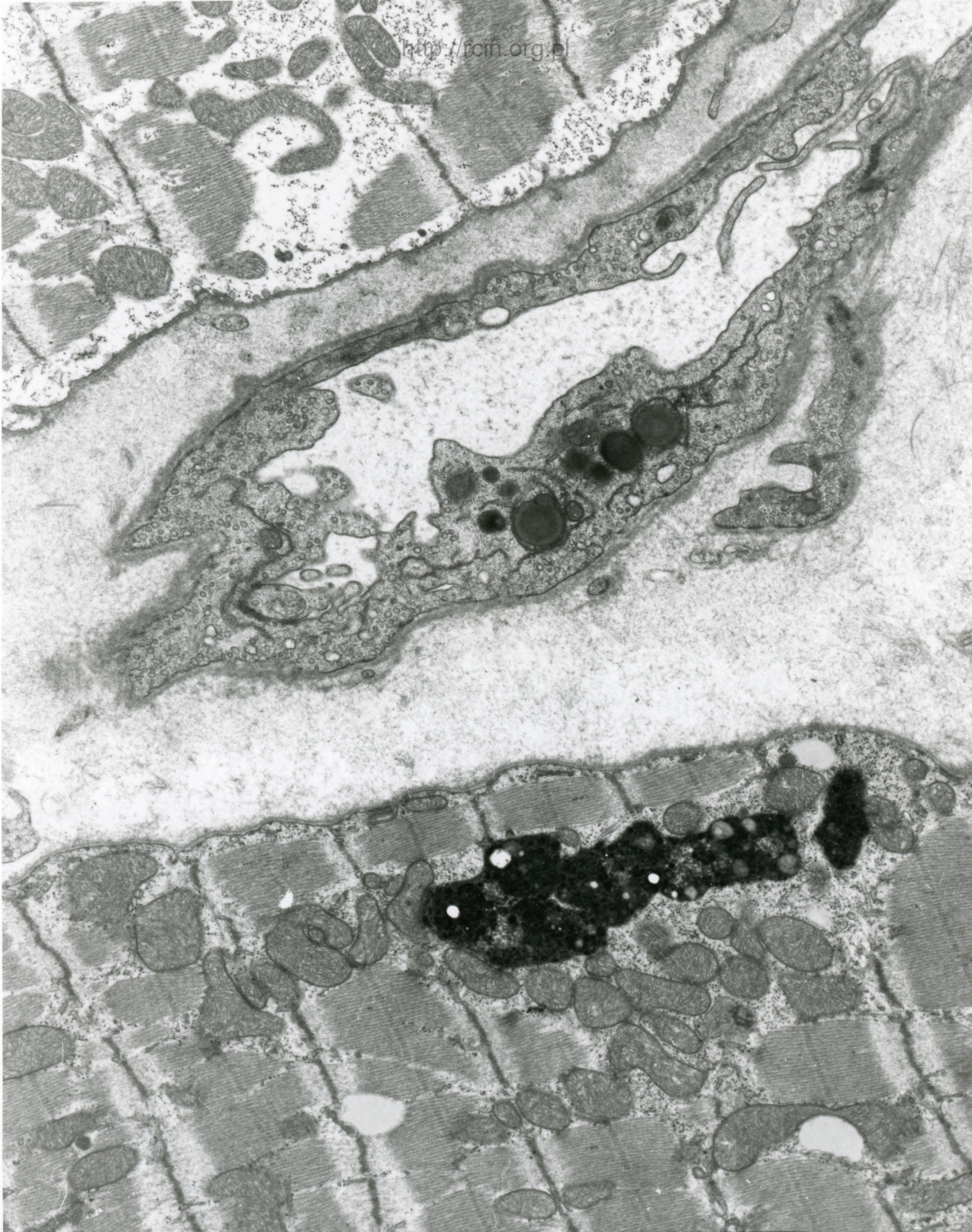
Fig. 8





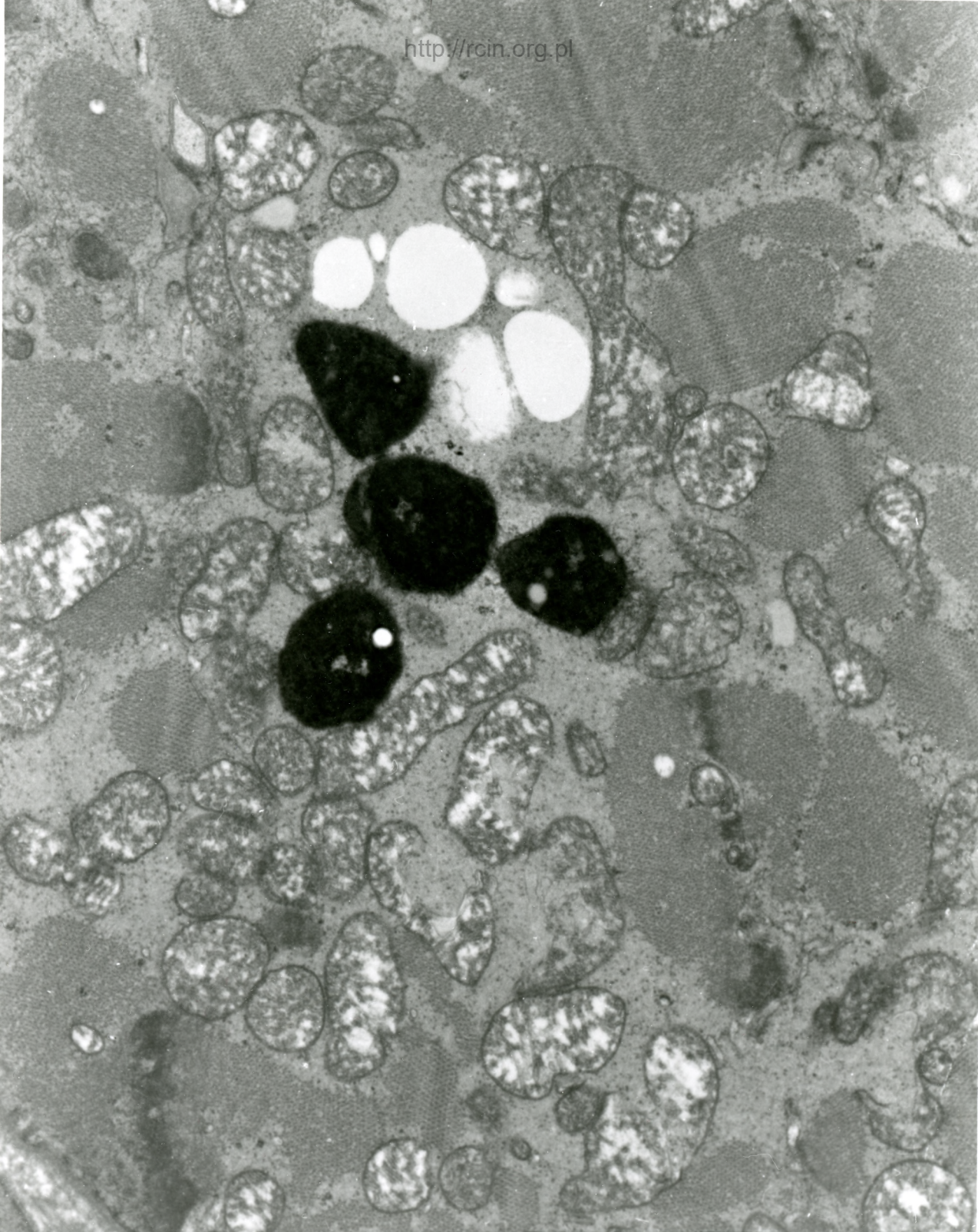
AF 39//09/B  
001202 00 0KU X5000 14

Fig. 9



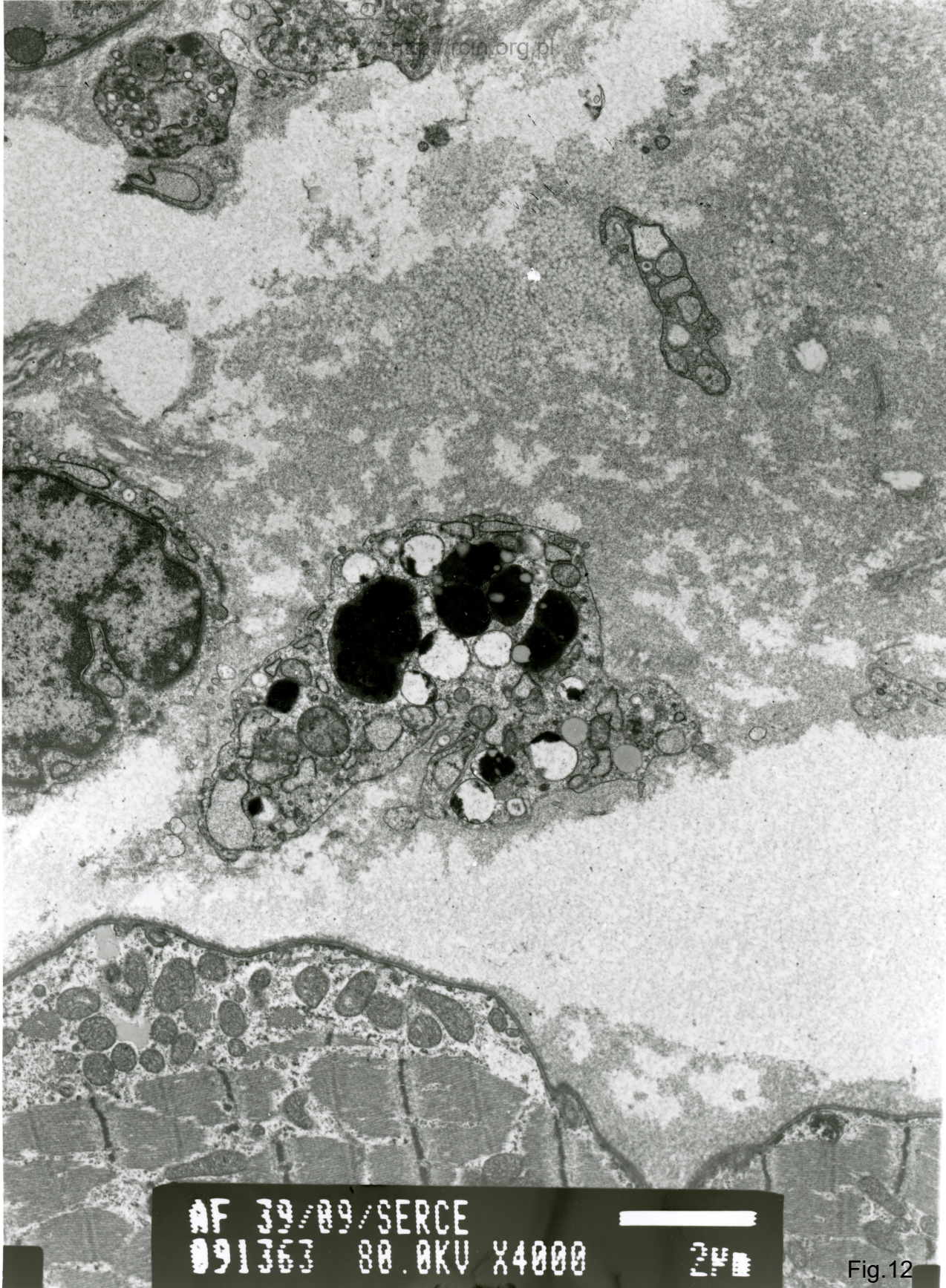
AF 39/09  
091556 80 0KV X5000 1µm

Fig.10

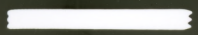


AF 39/89  
091652 80.0KV X7500 1µm

Fig.11

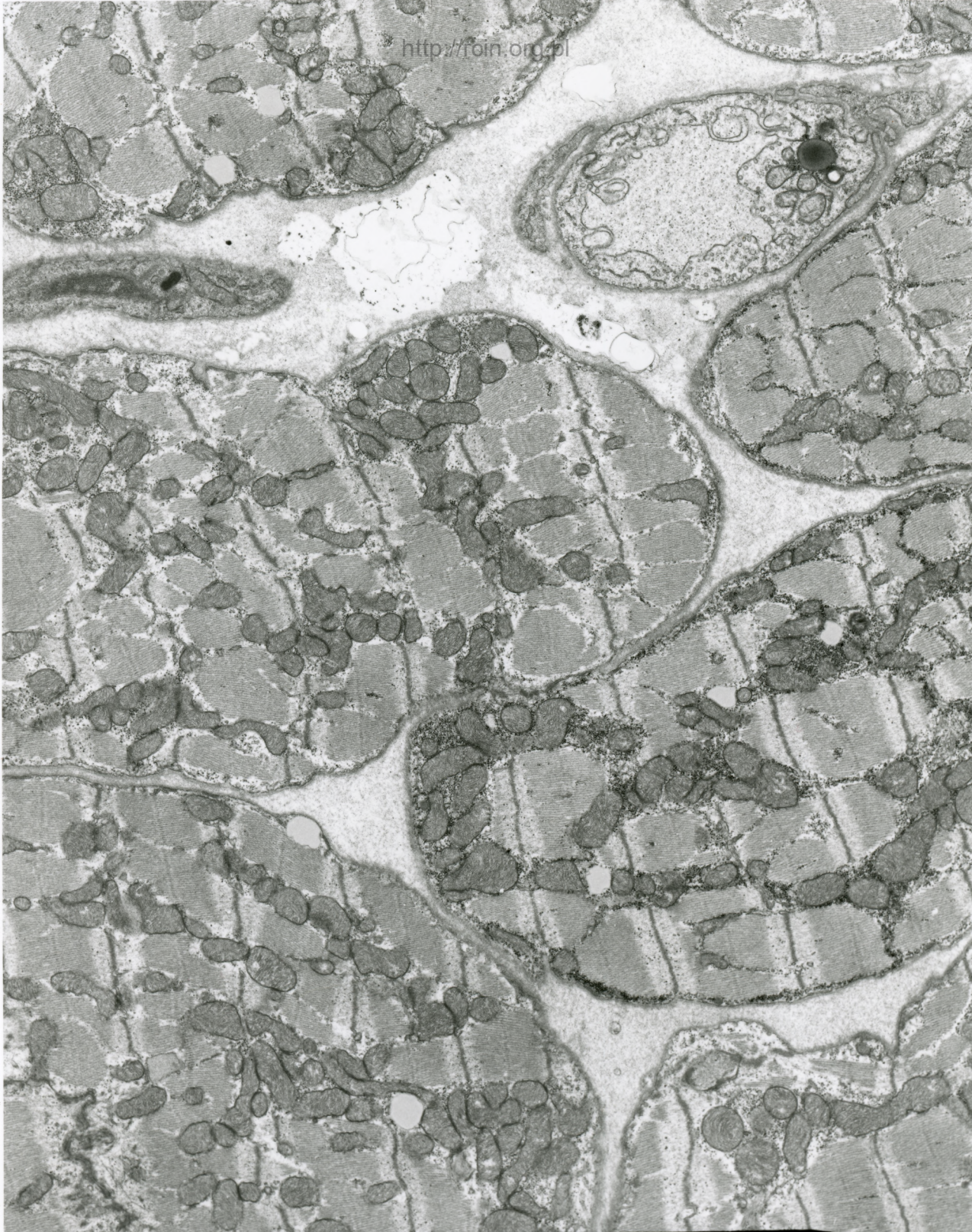


AF 39/09/SERCE  
091363 80.0KV X4000



2µm

Fig.12



AF 29/09/SERCE  
091372 88.0KV X3000 24

Fig.13

39/09 serce, patrz też 40/09

Analiza ultrastrukturalna ujawniła zaburzenia w układzie sarkomerowym oraz dezorganizację, a miejscami znaczny ubytek miofibrili (Fig.1-3). Obserwowano obecność wakuoli (Fig. 4-6). Część mitochondriów wykazywała uszkodzenia grzebieni mitochondrialnych. Licznie występowały lipofuscyny. (Fig. 7-11). W przestrzeni zewnątrzkomórkowej była widoczna, miejscami obfita, tkanka łączna (Fig. 12), a śródbłonki niektórych naczyń kapilarnych wykazywały cechy nekrozy (Fig.13).

Ultrastructural analysis revealed changes in the sarcomere arrangement as well as disorganization and, focally, significant loss of myofibrills (Figs.1-3). The presence of vacuoles was observed (Figs. 4-6). Some mitochondria were altered and characterized by damage of mitochondrial cristae. Many lipofuscin deposits were seen (Figs. 7-11). In the extracellular space quite abundant connective tissue was visible (Fig. 12). Endothelium of some capillaries showed features of necrosis (Fig.13).