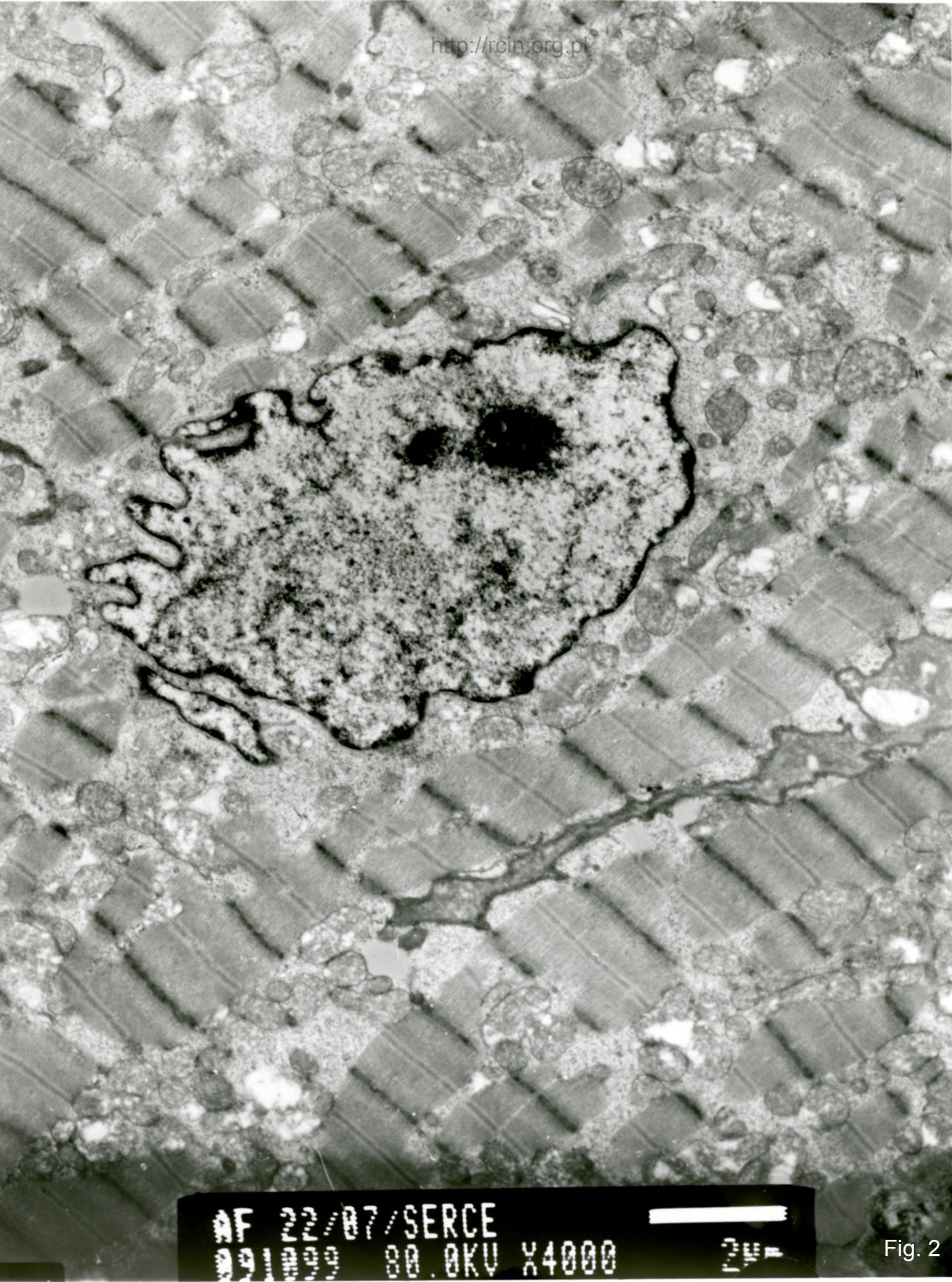




AF 22/07/SERCE  
071672 88 RCU X2500 .

24

Fig. 1



AF 22/07/SERCE  
091899 80.0KV X4000

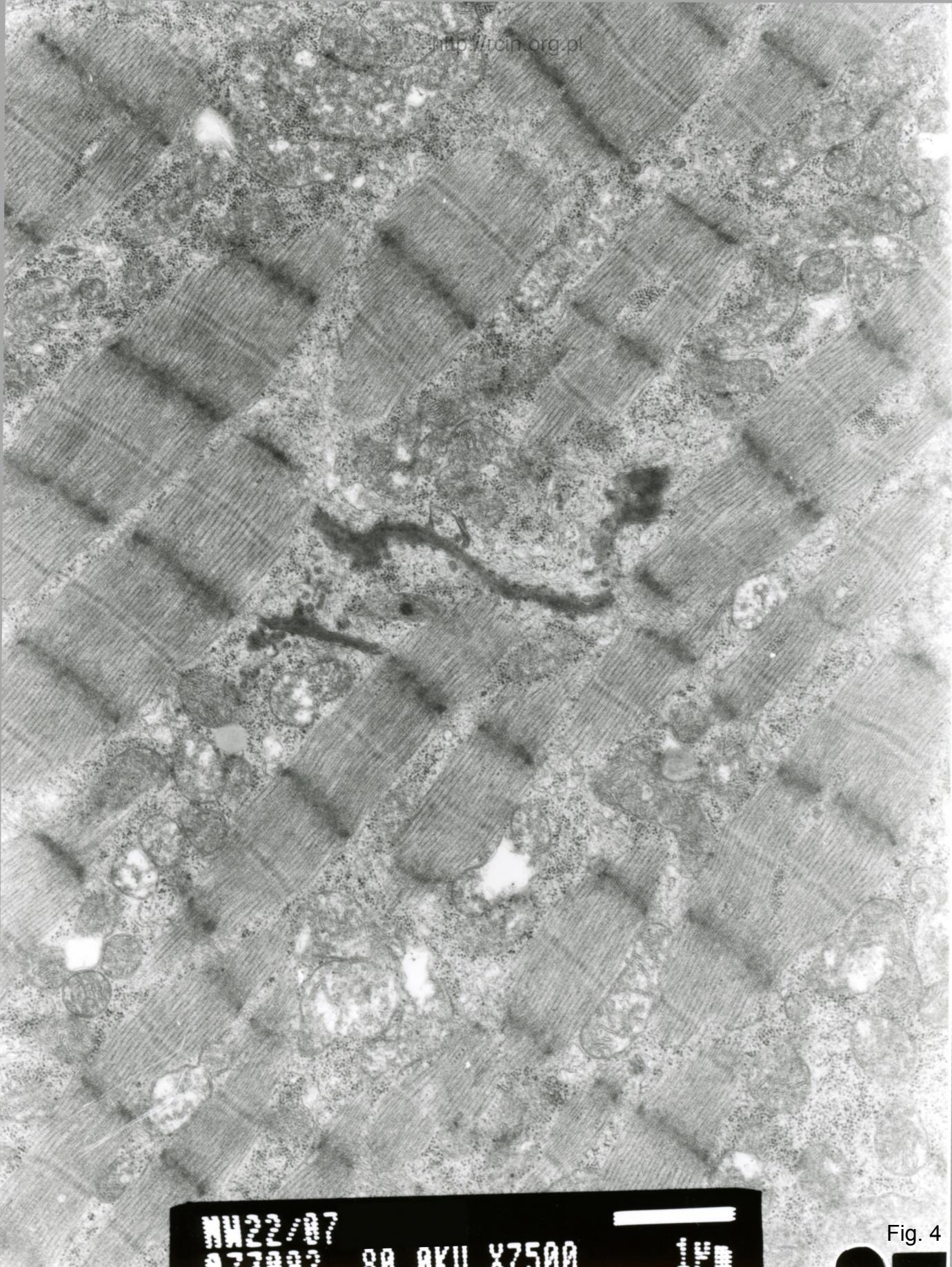
24

Fig. 2

AF 22/07/SERCE  
091088 88.BKV X5000

1μm

Fig. 3



NW22/87  
277800 98 XVII X7500 1µm

Fig. 4

AF 22/87/SERCE  
100297 80.0KV X4000

24m

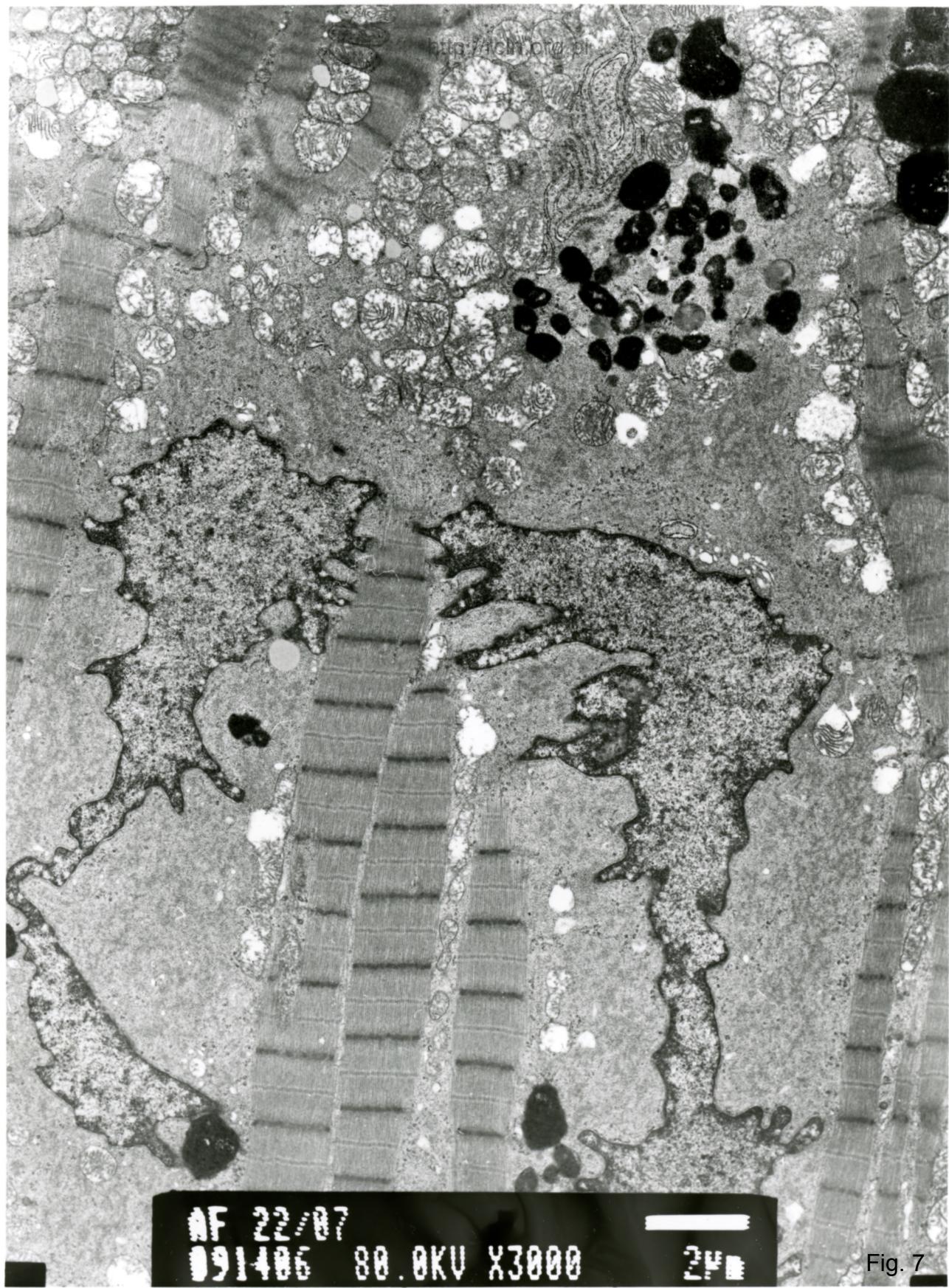
Fig. 5



AF 22/87/SERCE  
29.1.1988 88.8KU X4000

2P

Fig. 6

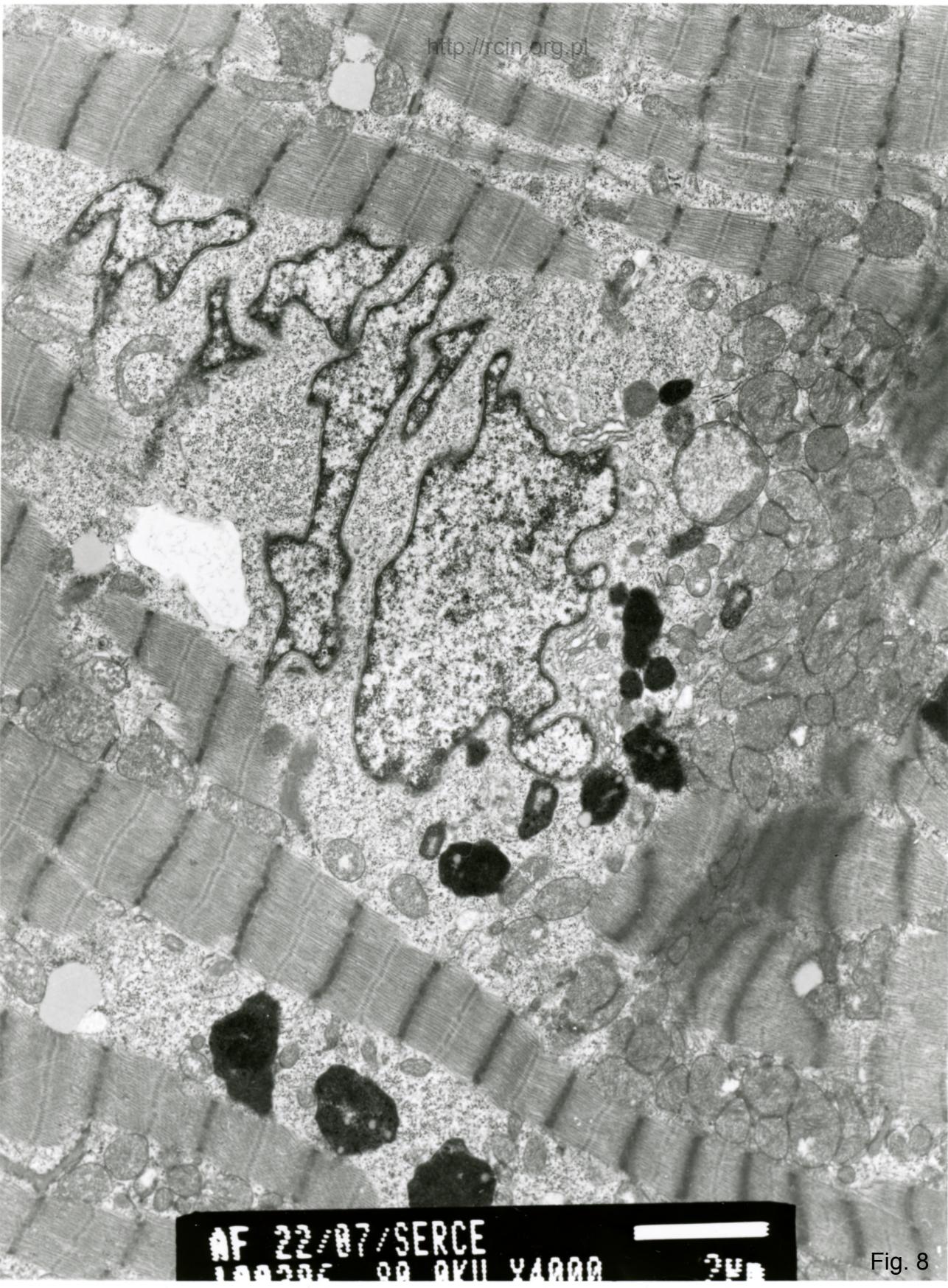


AF 22/87

091486 80.0KV X3000

24

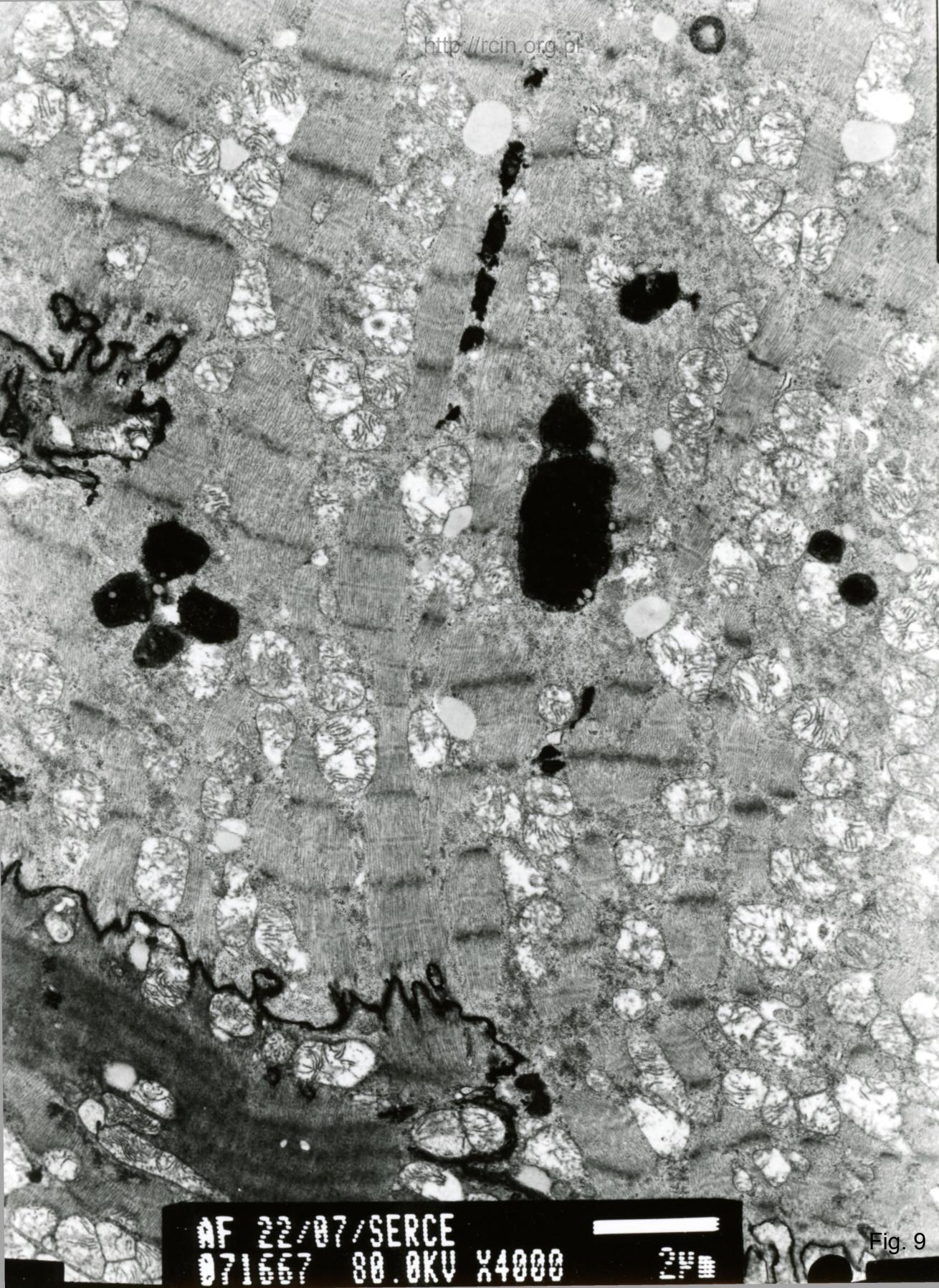
Fig. 7



AF 22/87/SERCE  
199202 00 AKU X4000

2μm

Fig. 8



AF 22/07/SERCE  
071667 80.0KV X4000

24

Fig. 9

AF 22/87/SERCE  
071665 88.BKV X4000

24

Fig.10



AF 22/07/SERCE  
201005 38 AKU X4000

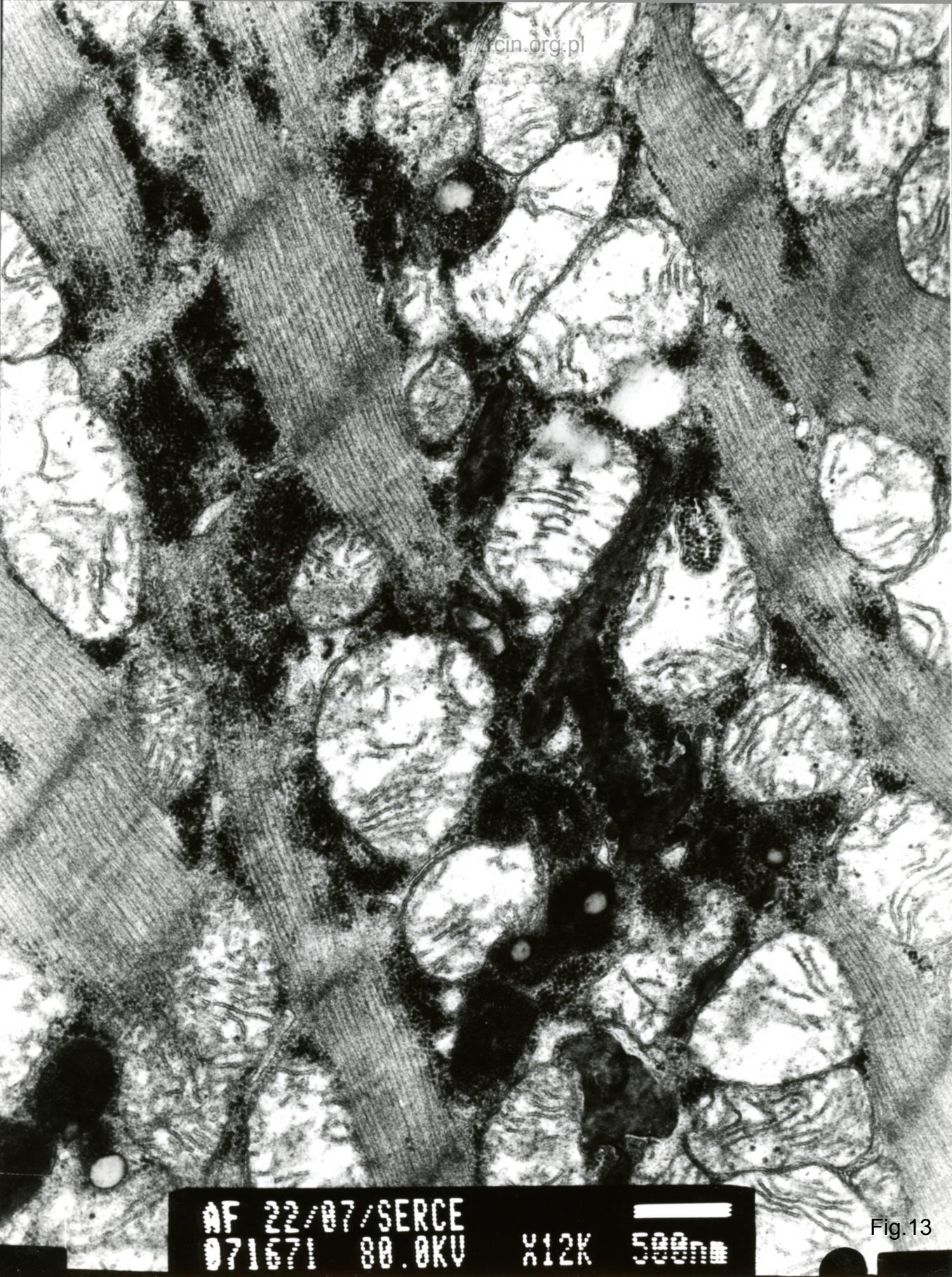
24E

Fig.11

AF 22/87/SERCE  
8912BB 88 BKU X5000

147

Fig. 12



AF 22/07/SERCE  
071671 80.0KV X12K 500nm

Fig.13

AF 22/87/SERCE  
09.1.1993 80.0KV X3000

24

Fig.14

AF 22/87/SERCE  
100295 80.0KV X3000

2μm

Fig. 15

AF 22/07/SERCE  
291089 80.0KV X3000

245

Fig.16

AF 22/07/SERCE  
100300 80.0KV X4000

2μm

Fig.17

AF 22/87/SERCE  
100294 80.0KV X5000

14B

Fig.18

AF 22/07/SERCE  
091200 80.0KV X6000

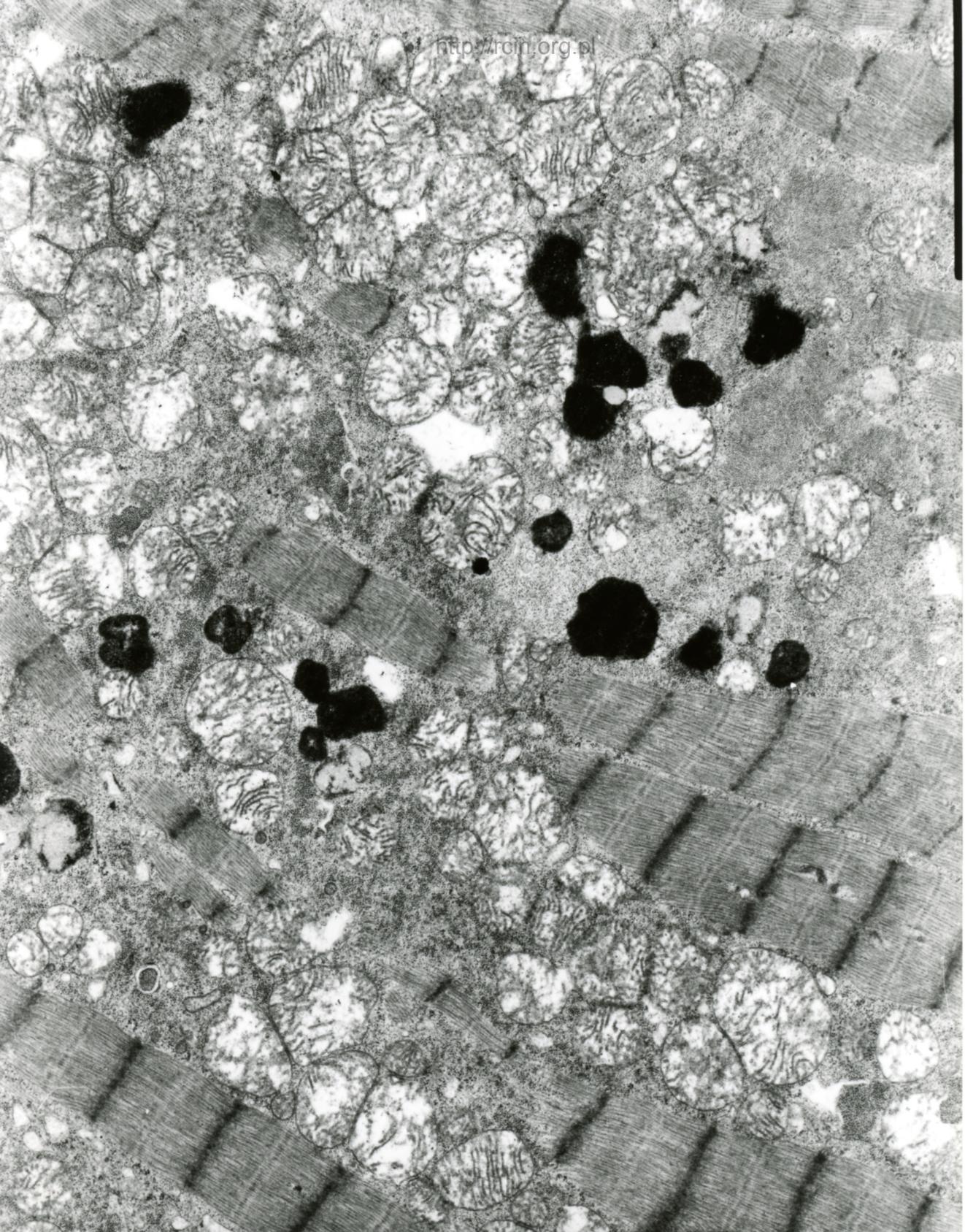
14

Fig.19

AF 22/87/SERCE  
071669 80.0KV X5000

14

Fig.20



AF 22/07/SERCE  
021668 RR PKU X5000

1µm

Fig.21

AF 22/87/  
100264 80.0KV X6000 1μm

Fig.22

AF 22/87/SERCE  
071670 88.BKV X6000

1μm

Fig.23

AF 22/07/SERCE  
091198 80.0KV X10K 500nm

Fig.24

AF 22/07/SERCE  
091092 80.0KV X4000

24

Fig.25

AF 22/87/SERCE  
100298 80.0KV X5000

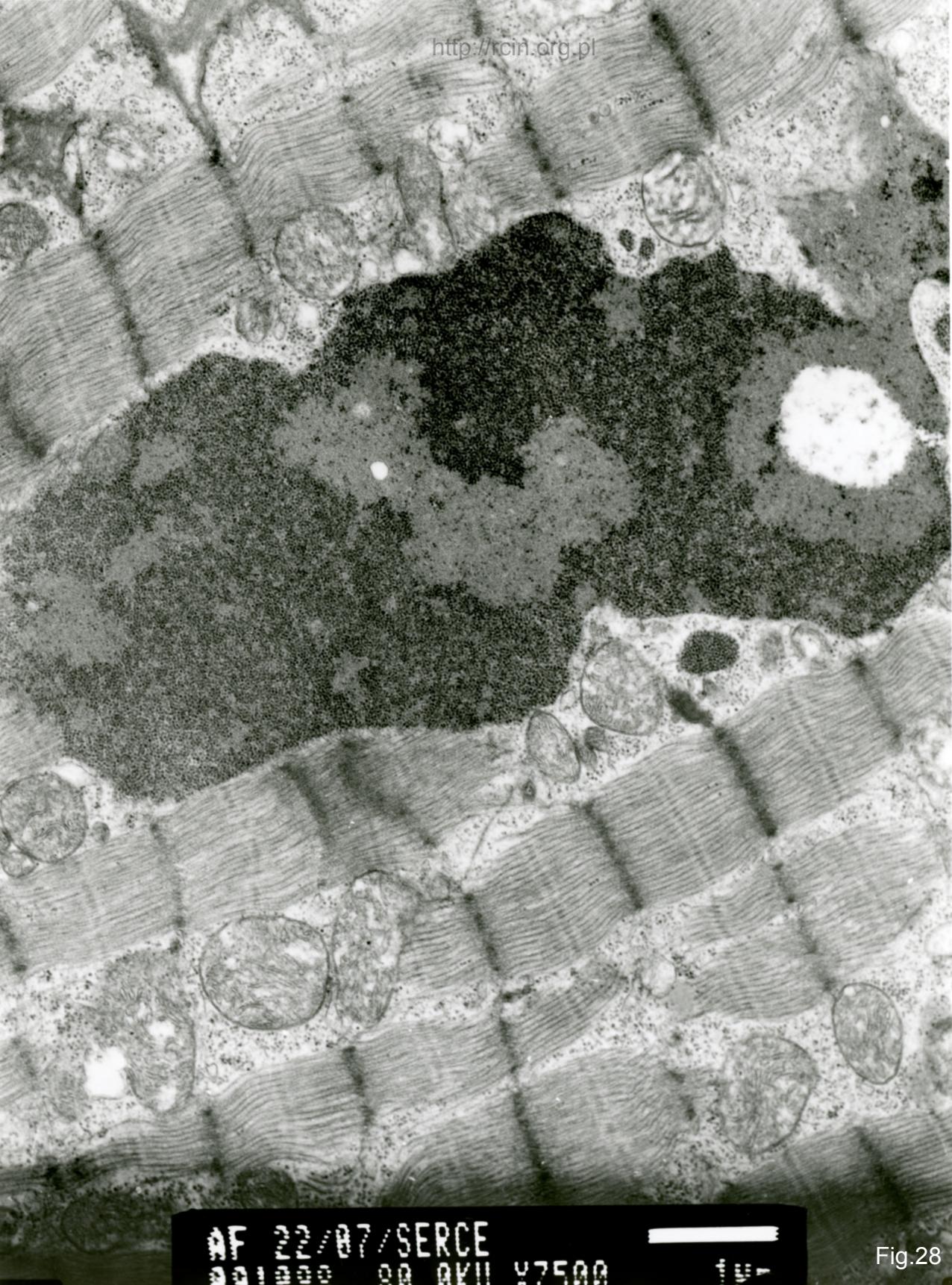
1μm

Fig.26

AF 22/87/  
100265 80.0KV X6000

1µm

Fig.27



AF 22/87/SERCE  
201000 99 AKII 2500

1μm

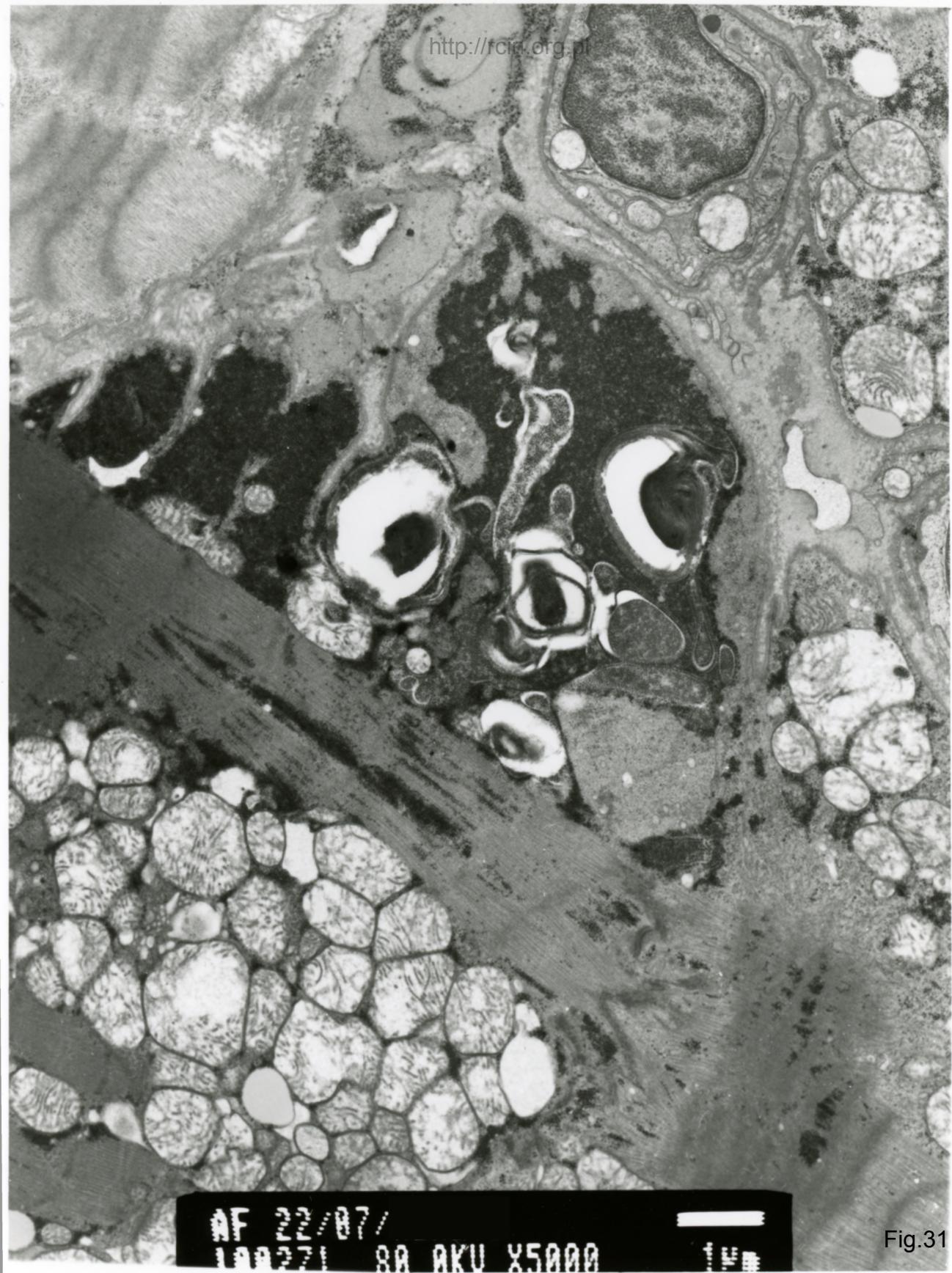
Fig.28

AF 22/87/  
100266 80.0KV X15K 5.0nm

Fig.29

AF 22/87/SERCE  
100299 80.0KV X20K 200nm

Fig.30



AF 22/87/  
198221 88 AKU X5000

1μm

Fig.31

22/07

1. 42

### Kardiomiopatia rodzinna, zaburzenia rytmu serca

Analiza ultrastrukturalna wykazała że w części bioptatu architektura kardiomiocytów nie odbiega znacznie od normy (Fig. 1-4). W niektórych komórkach obserwowano jednak, szczególnie w pobliżu jąder kardiomiocytów, dezorganizację sarkomerów i zanik miofibryli (Fig. 5-8). Wiele mitochondriów wykazywało cechy obrzęku, charakteryzowało się jasną macierzą i ubytkiem grzebieni mitochondrialnych (Fig. 9-13). Licznie występowały obłonione wakuole (Fig. 14-19) oraz złogi lipofuscyny (Fig. 20-24). Ogniskowo obserwowano duże nagromadzenie ziaren glikogenu (Fig. 25-30) oraz autofagię (Fig. 31).

### Familial cardiomyopathy, arrhythmia

Ultrastructural analysis revealed focally unchanged cardiomyocytes architecture (Figs. 1-4). However, disorganization of sarcomeres and atrophy of myofibrils was observed in some cells, especially in the vicinity of cardiomyocytes nuclei (Figs. 5-8). Many mitochondria were swollen and characterized by a bright mitochondrial matrix and loss of cristae (Figs. 9-13). Vacuole-like structures (Figs. 14-19) and lipofuscin deposits (Figs. 20-24) were abundant. Focally, large accumulation of glycogen granules (Fig. 25-30) and autophagy (Fig. 31) were observed.