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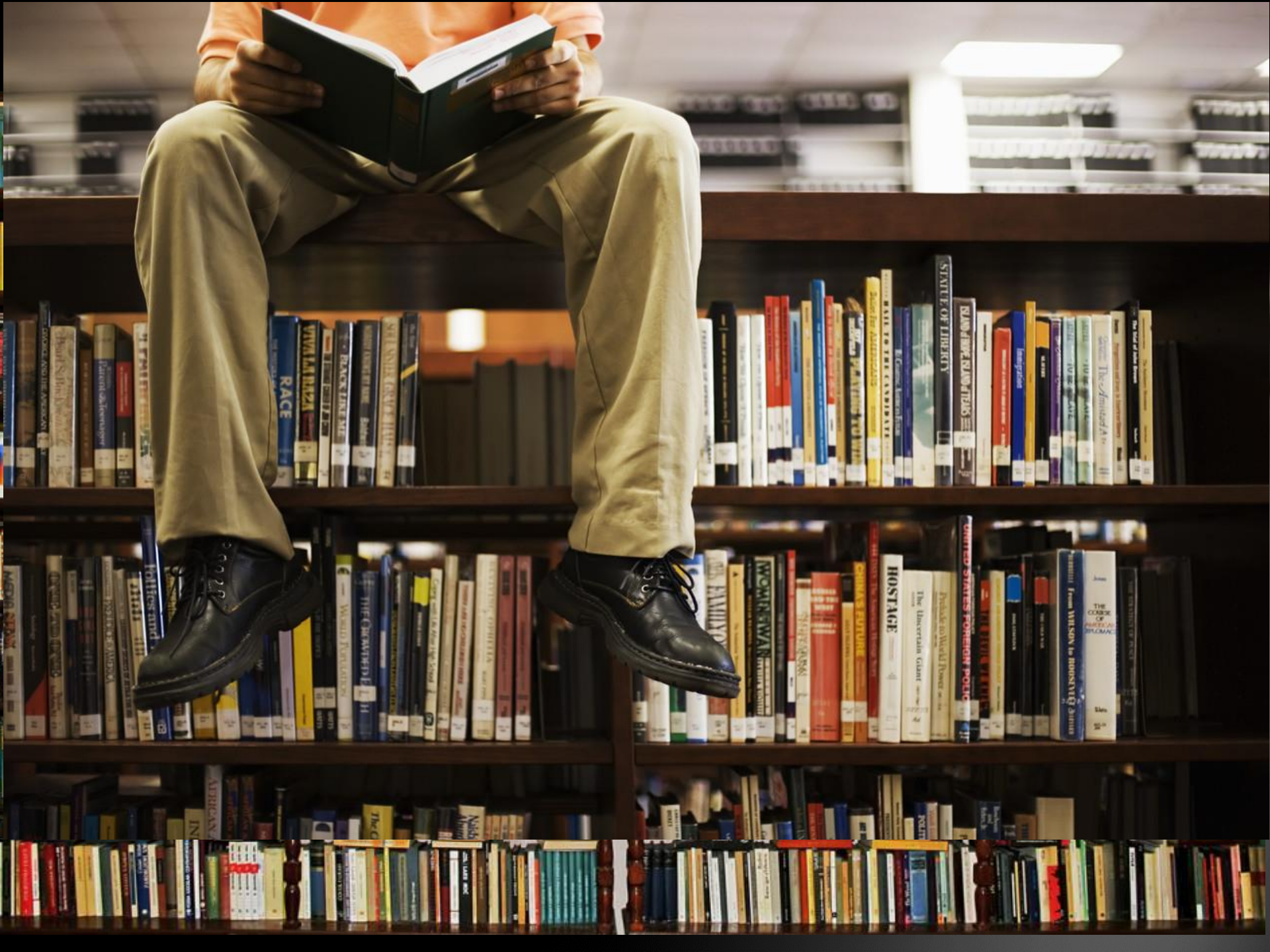
Jadranka Stojanovski

Sveučilište u Zadru, Institut Ruđer Bošković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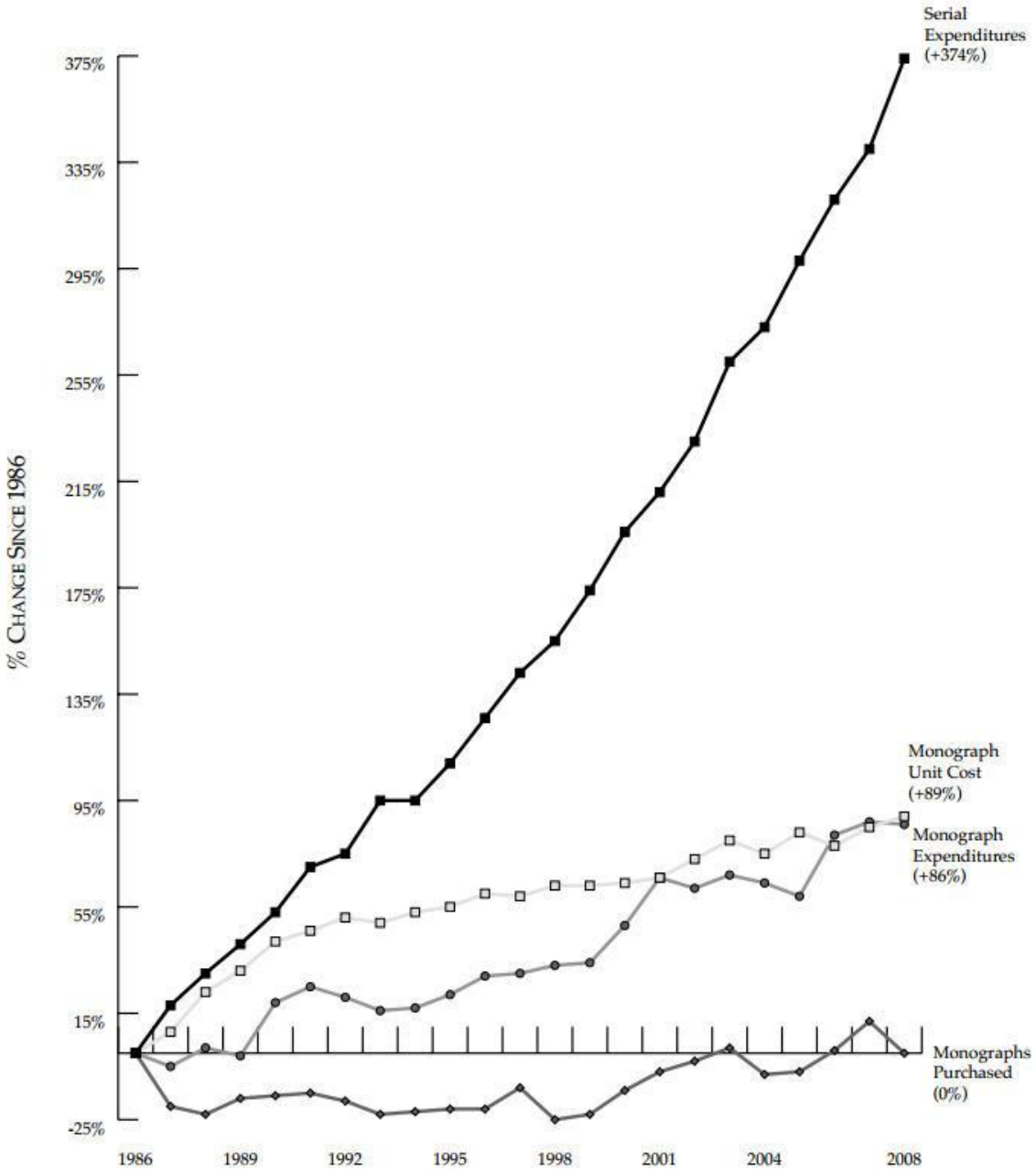

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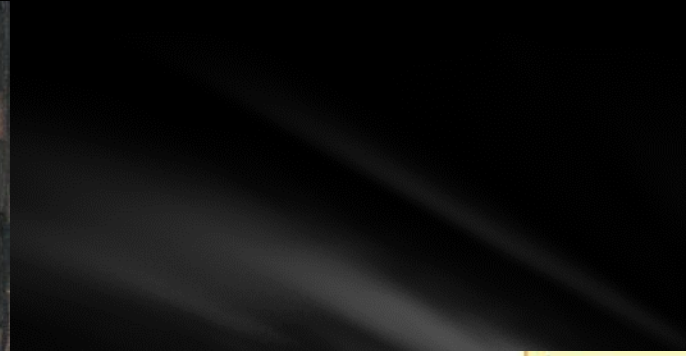

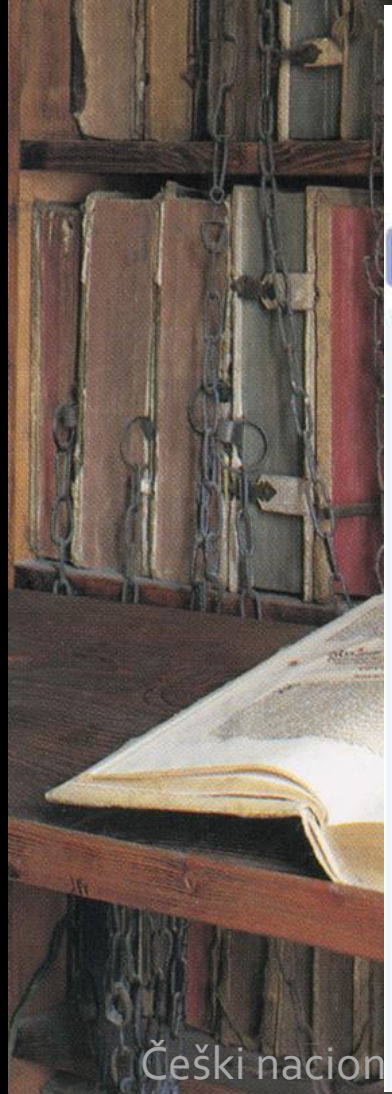

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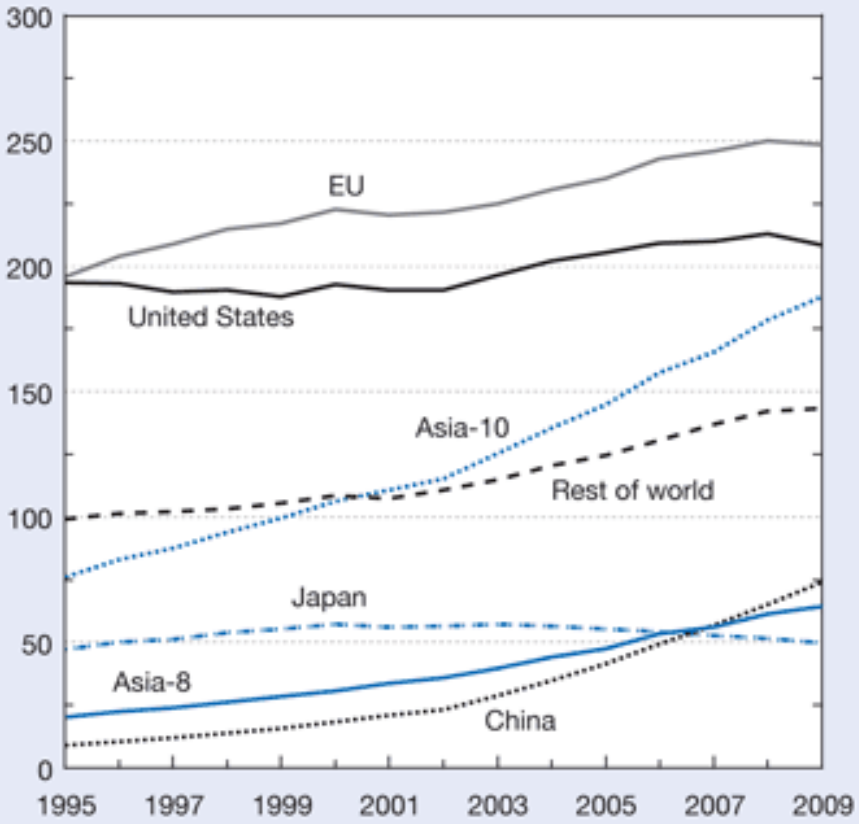

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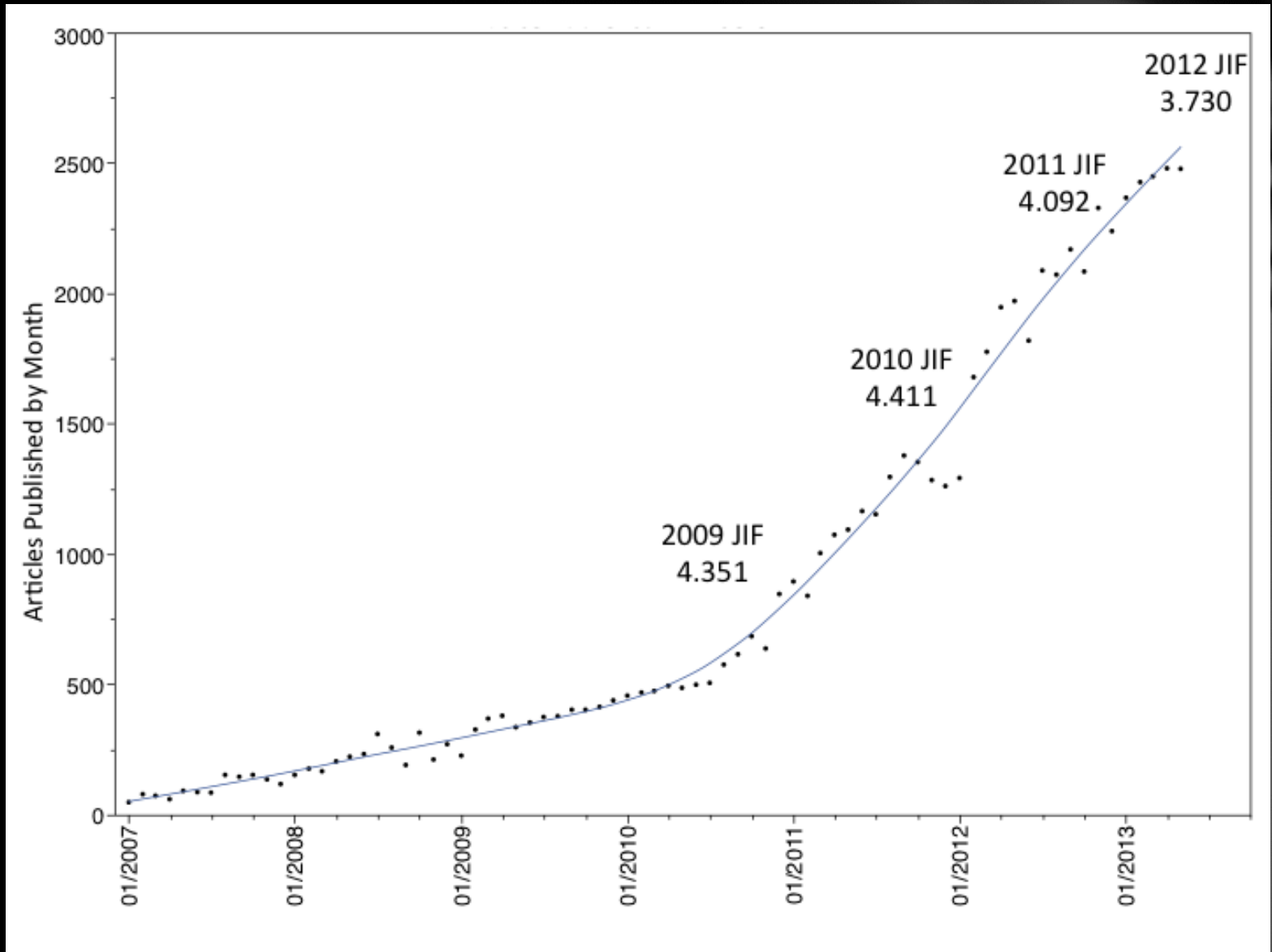
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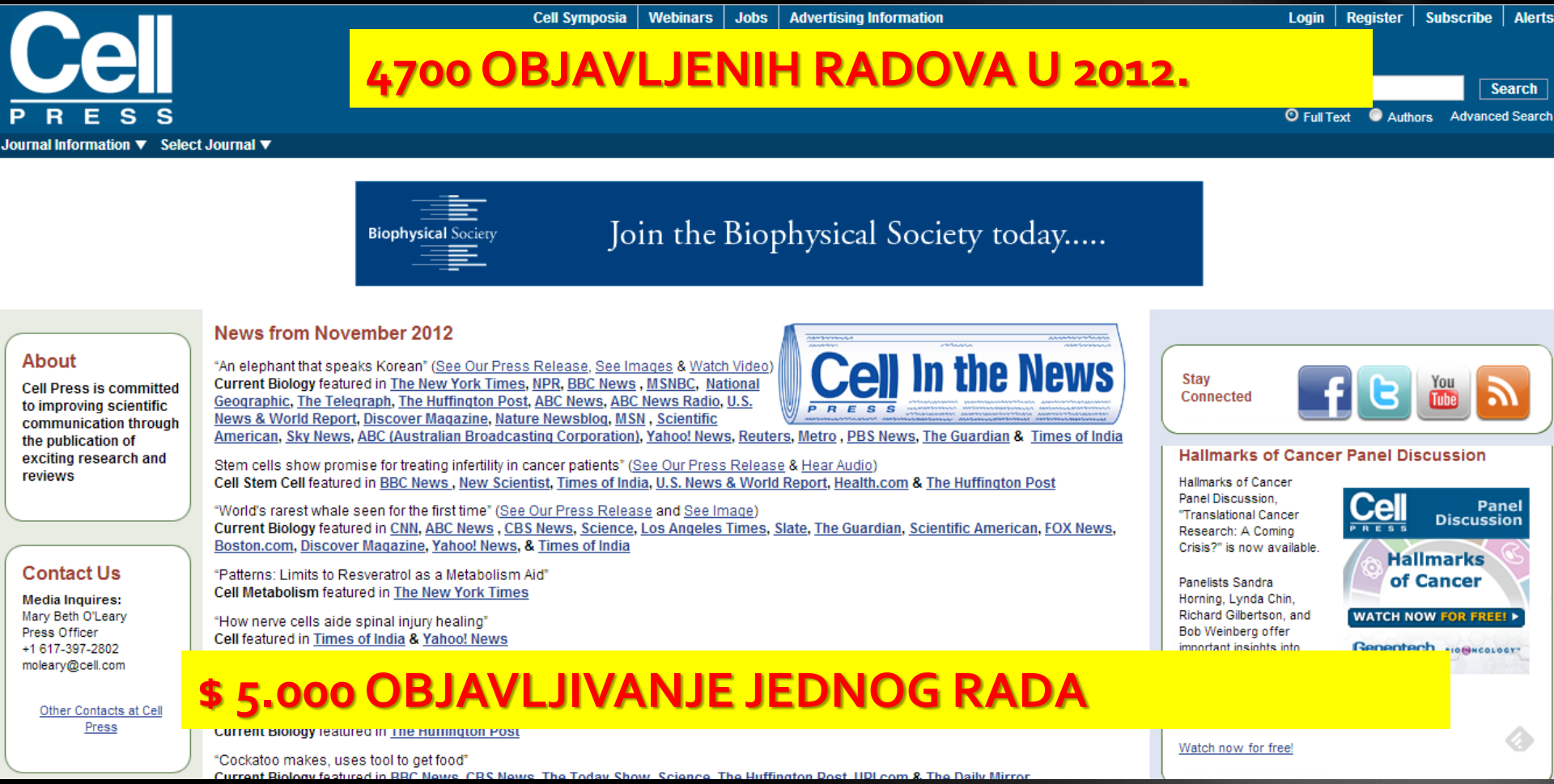

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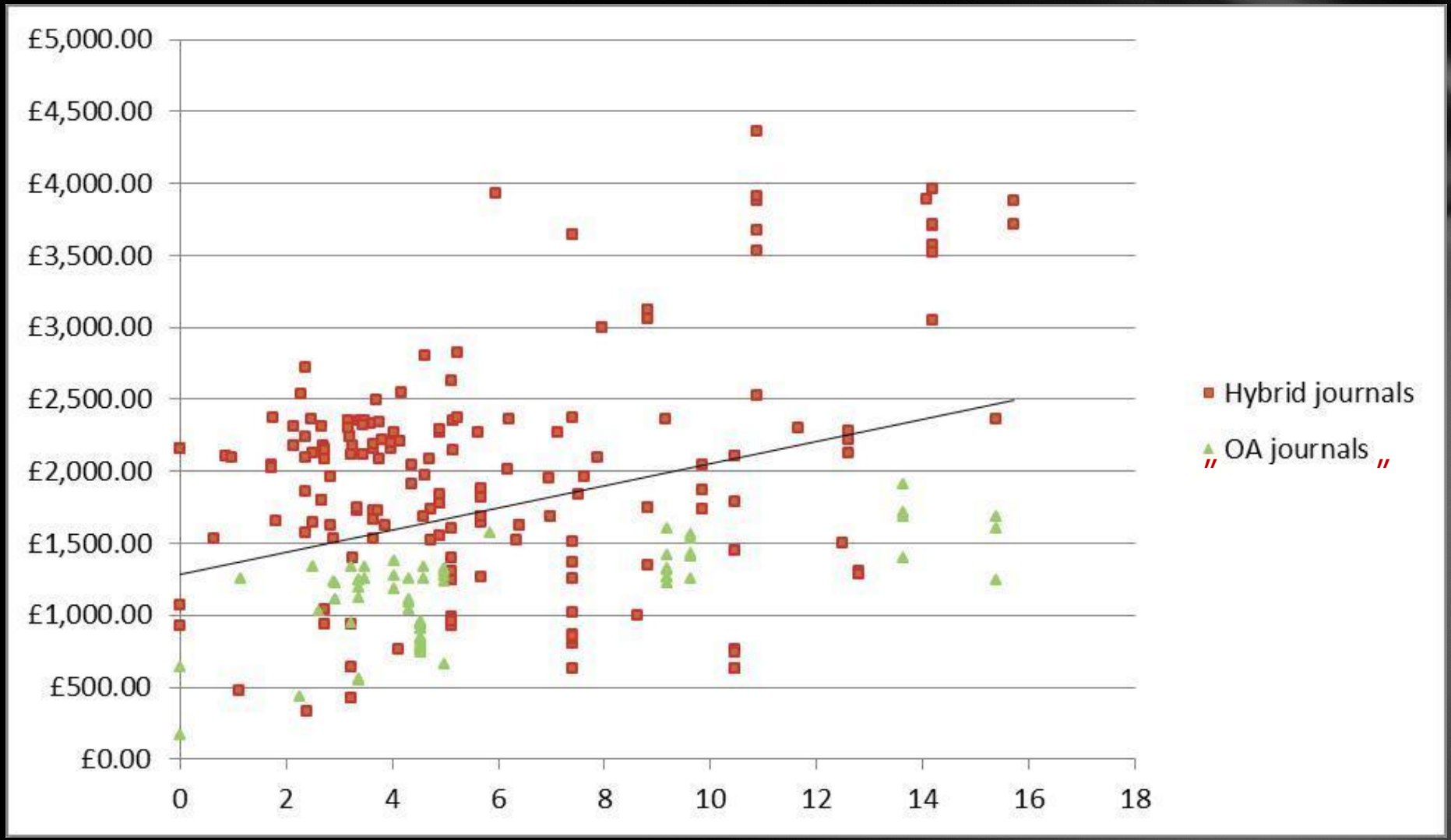

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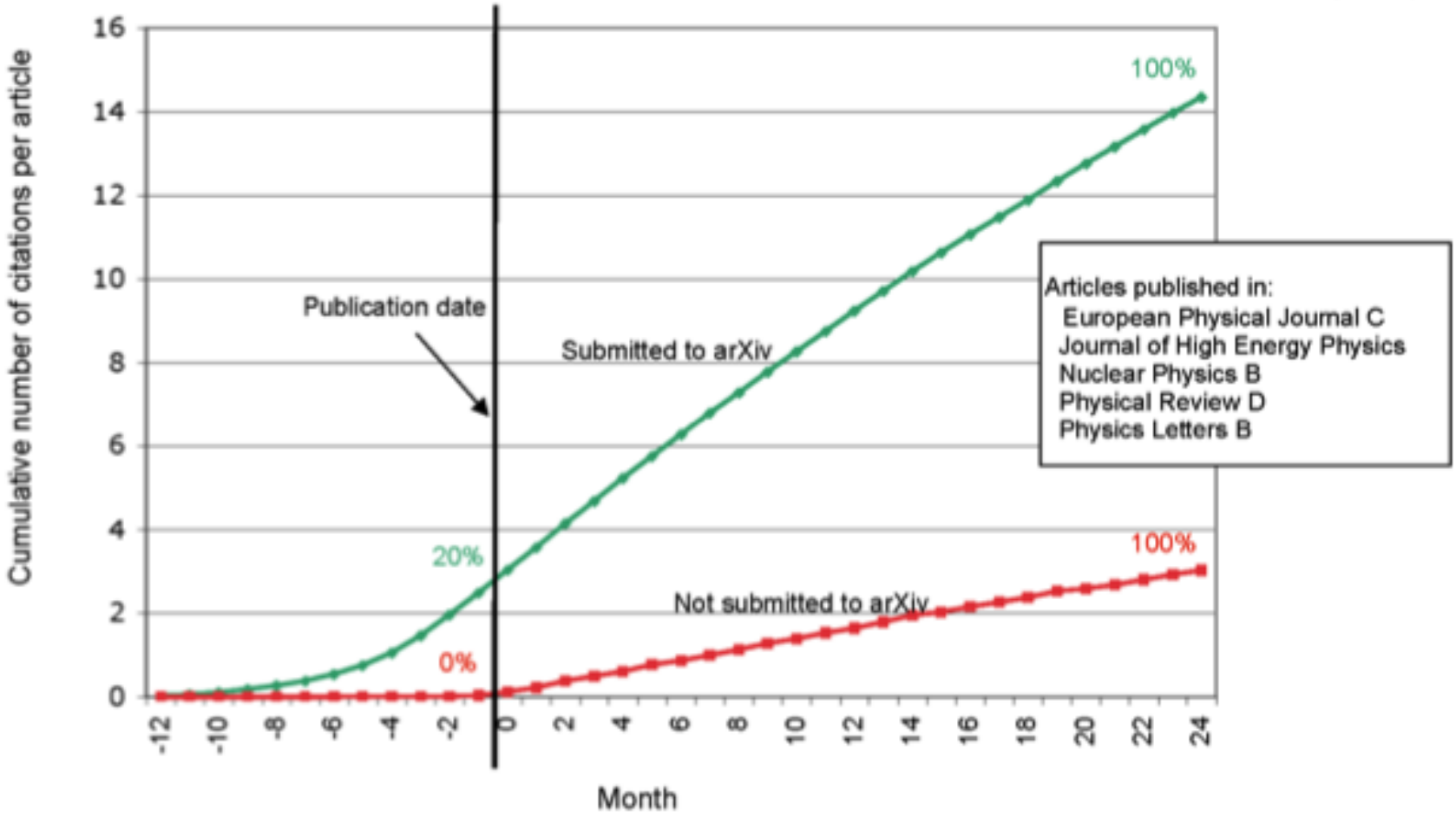

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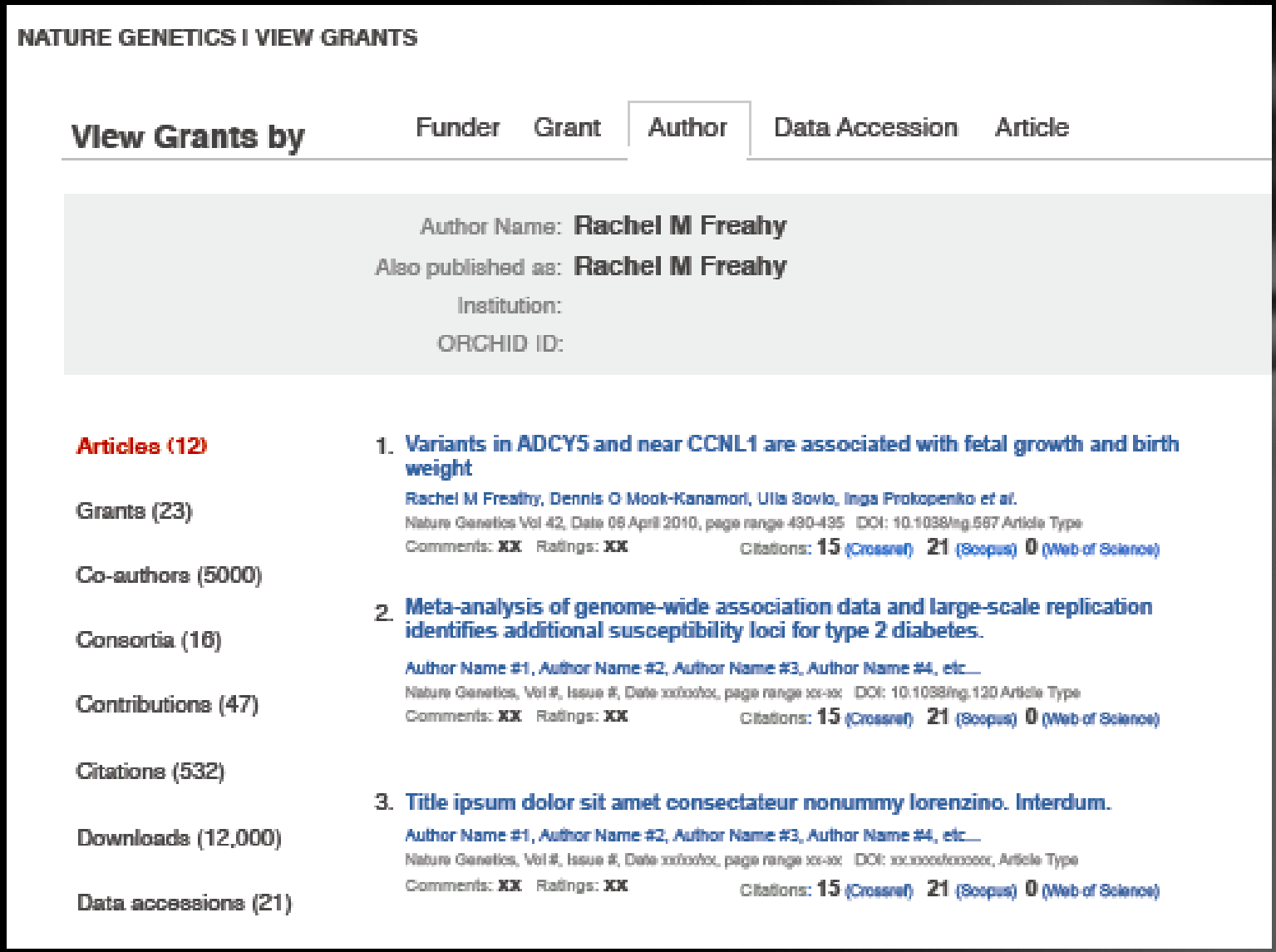

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ANOTACIJA

In the present study, the pH of the acetate buffer used in the TMB incubation medium was adjusted from pH 3.5 up to pH 7.0 in order to ascertain the optimal development of reaction product along with the best tissue preservation.
Regions containing the MB were cut into blocks and processed for electron microscopy according to standard methods (see Materials and Methods, Allen and Hopkins, '88).
Ultrathin sections were cut with a diamond knife and stained with uranyl acetate-lead citrate or left unstained before examination with a Zeiss EM 10A electron microscope.
Nomenclature
The nomenclature of the subicular complex used in the present study corresponds with Meibach and Siegel's ('77) modifications of the initial descriptions of the hippocampal formation by Lorente de N6 ('34). The nomenclature used for the prefrontal cortex follows that proposed by Krettek and Price ('77).
Quantitative analyses
The diameters of labeled axon terminals were calculated by taking the mean of the long and short axes of the terminals as measured directly from electron micrographs (final magnification ~16,000). Since the MB is known to have reciprocal projections to the midbrain (Guillery, '57; Cruce, '77; Takeuchi et al., '85), estimates of the numbers of labeled and unlabeled neurons in the medial and lateral mamillary nuclei were made from 1 µm-thick plastic sections (toluidine blue stained) following injections of WGA-HRP into the midbrain. Only perikarya which were sectioned through the nucleolus were counted. Approximately 1,900 cells were counted from sections cut from selected rostral to caudal levels of the MB in eight animals.

RESULTS
In the present study, injections of WGA - HRP into the region of the MB resulted in dense retrograde labeling in the subicular complex, medial prefrontal cortex, and dorsal and ventral tegmental nuclei. Fewer retrogradely labeled perikarya were observed in the nucleus of the diagonal band of Broca, and small numbers of widely scattered labeled perikarya were found in the lateral hypothalamus. Dense anterograde labeling was observed in the anterior thalamus, dorsal and ventral tegmental nuclei, nucleus reticularis tegmenti pontis, and medial pontine nuclei.
Merents from the subicular complex
Light microscopy. Figure 1 shows the differential distribution of retrogradely labeled neurons in the subicular complex following injections of WGA - HRP into the medial and lateral mamillary nuclei. In one of the cases illustrated in Figure 1, the injection site (inset) was centered in the midline of the medial mamillary nucleus and included most of the subnuclei of the medial nucleus bilaterally. The lateral mamillary nucleus was spared but there was some spread from the principal injection site dorsally into the medial portion of the supramamillary nucleus. Large numbers of retrogradely labeled perikarya were found bilaterally in all layers of the dorsal and ventral portions of the subiculum but no labeled cells were found in the presubiculum or parasubiculum (Figs . 1, 2). A few retrogradely labeled neurons were also found in the deep layers of the retrosplenial granular cortex (Figs . IB , 2). In the second case illustrated in Figure 1, the injection site (inset) was located mainly in the lateral mamillary nucleus with a slight involvement of the lateral part of the medial nucleus. In addition , there was some spread from the injection site dorsally into the lateral portion of the supramamillary nucleus and lateral hypothalamus. Numerous retrogradely labeled perikarya were found mainly ipsilaterally in the presubiculum and parasubiculum (Figs . 1,3). A few labeled neurons were also found in ipsilateral dorsal subiculum as well as in the contralateral presubiculum.
Following injections of WGA - HRP into the subicular complex , anterograde labeling was distributed in distinct horizontal bands or layers across the MB bilaterally (Fig . 4). The horizontal layers of anterograde labeling were present primarily in either dorsal or intermediate or ventral parts of the medial mamillary nucleus depending on the locations of the injection sites in the rostral to caudal parts of the subicular complex . Figure 4A - D shows the results from a representative case in which WGA - HRP was injected into the rostrodorsal portion of the subiculum . The resultant anterograde labeling was present in the medial mamillary nucleus bilaterally and formed a horizontal layer across the dorsal portion of the medial mamillary nucleus (Fig . 4B - D). The anterograde labeling was moderate to light in the anterior (Fig . 4B) and middle (Fig . 4C) thirds of the medial nucleus and heavy in the posterior third of the MB (Fig . 4D). The anteromedial part of the medial nucleus (pars medianus) contained only sparse anterograde labeling (Fig . 4B).
Figure 4E - H shows the results from a representative case in which WGA - HRP was injected into the caudoventral part of the subicular complex which included the presubiculum and parasubiculum . In this case , heavy anterograde labeling was present in the ventral portion of the posterior half of the medial mamillary nucleus bilaterally (Fig . 4G , H), whereas moderate to light anterograde labeling was present in the intermediate and dorsal parts of the anterior half of the medial nucleus bilaterally (Fig . 4F , G). The pars medianus showed very sparse or no anterograde labeling following injections in the caudoventral part of the subicular complex (Fig . 4F). Moderate to light anterograde labeling was also found in the lateral mamillary nucleus mainly ipsilaterally (Fig . 4G). Cases in which the WGA - HRP injections into the subicular complex did not involve the presubiculum and parasubiculum showed no anterograde labeling in the lateral mamillary nucleus (Fig . 4A - D).
Electron microscopy. Following injections of WGAHRP into the subicular complex , labeled axons and axon terminals were observed in both the medial (Figs . 5 - 8) and lateral (Fig . 6C) mamillary nuclei . When DAB was used as the chromogen , labeled axon terminals were characterized by the presence of small amounts of electron - dense reaction product which were located in membrane - bound , lysosomal - like structures (Fig . 5). Identification of labeled axon terminals following DAB histochemistry required careful study of low - contrast , unstained sections with the electron microscope because in stained sections the DAB reaction product , although darker , resembled the staining seen in normal lysosomes . In contrast , when the TMB - DAB procedure was used , amorphous patches of electron - dense reaction product were found in axons and axon terminals in the MB (Figs . 6 - 8). The TMB - DAB - labeled axon terminals could be easily identified in stained sections at low magnifications because the TMB - DAB reaction product formed relatively large complexes and did not resemble normal tissue organelles (Fig . 7). There were, however, some disadvantages with the TMB-DAB procedure in comparison to the DAB procedure. For example , tissue elements were less well preserved and the reaction product was usually so large that it tended to obscure the contents of the axon terminals and the morphology of synaptic junctions following incubations in the standard TMB incubation medium (acetate buffer pH 3.5 - 4.0) (Fig . 6A). These problems were reduced when the pH of the acetate buffer used in the TMB incubations was made less acidic (pH 4.6 - 6.0). This simple modification of the TMB procedure resulted in a noticeable reduction in the amount of reaction product within the axon terminals, allowing visualization of synaptic vesicles and the morphology of synaptic junctions along with a much improved preservation of neural elements (Fig . 6B - D). The number of labeled axon terminals observed at the electron microscopic level was markedly decreased when the pH of the acetate buffer was greater than 6.0.
Labeled axon terminals from the subicular complex ranged in diameter from 0.8 to 2.0 µm , contained mainly round vesicles (diameter = 40 nm), and formed asymmetric synaptic junctions mainly with small - diameter (less than 2 µm) dendrites and dendritic spines . Individual labeled axon terminals occasionally formed synaptic contacts with two adjacent dendrites (Fig . 8). Labeled axon terminals from the subicular complex only rarely contained pleomorphic vesicles and formed synaptic junctions with neuronal somata or proximal dendrites . Unlabeled axon terminals with pleomorphic vesicles and symmetric synaptic junctions with neuronal elements were , nonetheless , readily identified in this material .
Many labeled axon terminals appeared to form two separate synaptic specializations on individual dendritic profiles (Figs . 5, 6B , D , 8A), but serial sectioning of several labeled Merents from the medial prefrontal cortex
Light microscopy. The distributions of retrogradely labeled neurons in the medial prefrontal cortex were mapped following injections of WGA - HRP into the MB . Figure 9 shows the results from a representative case in which retrograde labeling in the medial prefrontal cortex (Figs . 9A , B , 10) was obtained following an injection of WGA - HRP into the medial mamillary nucleus (Fig . 9C).
terminals revealed that two apparently distinct synaptic specializations on the same dendrite were parts of a single continuous synaptic specialization (Fig. 8).
The injection was centered in the medial part of the medial mamillary nucleus with some spread of reaction product laterally into the lateral parts of the medial mamillary nucleus and dorsally into the medial portion of the supramamillary nucleus . The retrogradely labeled cells in the prefrontal cortex were pyramidal - shaped (Fig . 9B) and were distributed from the rostral limit of the prefrontal cortex to a level just rostral to the genu of the corpus callosum (Fig . 10). Most of the retrogradely labeled neurons were located in the deep layers of the infralimbic area while fewer labeled neurons were found rostrally and dorsally in or near layer V of the prelimbic and anterior cingulate areas. A few labeled neurons were also found lateral and ventral to the rhinal fissure. Some of the latter cells were located in the caudal end of the infralimbic cortex where they approached the rostralmost extent of the vertical limb of the diagonal band of Broca .

MJERA DOPRINOSA AUTORA

Nature Genetics **41**, 399 - 406 (2009)
Published online: 22 March 2009 | doi:10.1038/ng.364

Common variants at ten loci influence QT interval duration in the QTGEN Study

Christopher Newton-Cheh[1,2,3,22], Mark Eijgelsheim[4,22], Kenneth M Rice[5,22], Paul I W de Bakker[2,6,22], Xiaoyan Yin[3,7], Karol Estrada[8], Joshua C Bis[9,10], Kristin Marciante[9,10], Fernando Rivadeneira[4,8], Peter A Noseworthy[1], Nona Sotoodehnia[9,11], Nicholas L Smith[9,12,13], Jerome I Rotter[14], Jan A Kors[15], Jacqueline C M Witteman[4,16], Albert Hofman[4,16], Susan R Heckbert[9,12,17], Christopher J O'Donnell[3,18,19], André G Uitterlinden[4,8,16], Bruce M Psaty[9,10,12,17,20], Thomas Lumley[5,23], Martin G Larson[3,7,23] & Bruno H Ch Stricker[4,8,15,16,21,23]

Author Contributions

Framingham Heart Study: M.G.L., C.N.-C., P.A.N., C.J.O., X.Y.

Rotterdam Study: M.E., K.E., A.H., J.A.K., F.R., B.H.Ch.S., A.G.U., J.C.M.W.

Cardiovascular Health Study: J.C.B., S.R.H., T.L., K.M., C.N.-C., B.M.P., K.M.R., J.I.R., N.L.S., N.S.

Broad Institute of Harvard and Massachusetts Institute of Technology: P.I.W.dB., C.N.-C.

Design of QTGEN study: P.I.W.dB., M.E., M.G.L., T.L., C.N.-C., C.J.O., B.M.P., K.M.R., B.H.Ch.S. Genotyping: Affymetrix, C.N.-C., F.R., J.I.R., A.G.U. Statistical analysis and informatics: J.C.B., P.I.W.dB., M.E., K.E., T.L., K.M., C.N.-C., K.M.R., F.R., A.G.U., X.Y. Drafting of manuscript: C.N.-C. Critical revision of manuscript: J.C.B., P.I.W.dB., M.E., K.E., S.R.H., A.H., J.A.K., P.A.N., B.M.P., K.M.R., J.I.R., N.L.S., N.S., B.H.Ch.S., J.C.M.W.

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22. These authors contributed equally to this work.
23. These authors jointly directed this work.

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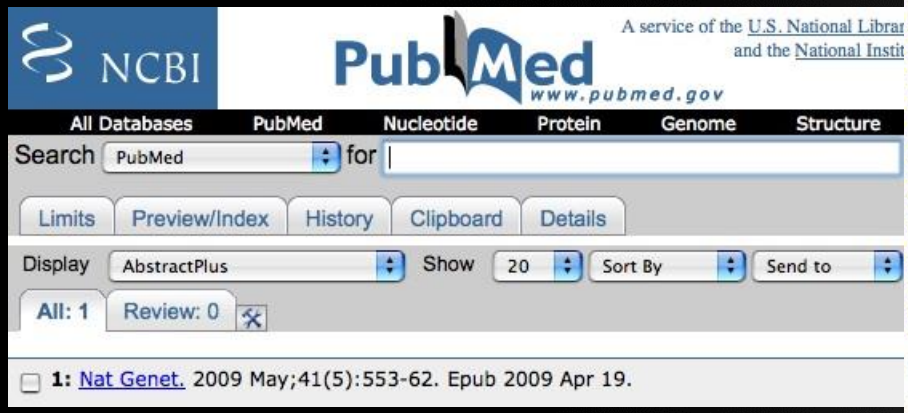
ISCRPNI METAPODACI (IZDAVAČI, KNJIŽNICE...)

Nature Genetics **41**, 553 - 562 (2009)
Published online: 19 April 2009 | doi:10.1038/ng.375

The transcriptional network that controls growth arrest and differentiation in a human myeloid leukemia cell line

The FANTOM Consortium & Riken Omics Science Center[1]



FANTOM Consortium, Suzuki H, Forrest AR, van Nimwegen E, Daub CO, Balwierz PJ, Irvine KM, Lassmann T, Ravasi T, Hasegawa Y, de Hoon MJ, Katayama S, Schroder K, Carninci P, Tomaru Y, Kanamori-Katayama M, Kubosaki A, Akalin A, Ando Y, Arner E, Asada M, Asahara H, Bailey T, Bajic VB, Bauer D, Beckhouse AG, Bertin N, Björkegren J, Brombacher F, Bulger E, Chalk AM, Chiba J, Cloonan N, Dawe A, Dostie J, Engström PG, Essack M, Faulkner GJ, Fink JL, Fredman D, Fujimori K, Furuno M, Gojobori T, Gough J, Grimmond SM, Gustafsson M, Hashimoto M, Hashimoto T, Hatakeyama M, Heinzel S, Hide W, Hofmann O, Hörnquist M, Huminiecki L, Ikeo K, Imamoto N, Inoue S, Inoue Y, Ishihara R, Iwayanagi T, Jacobsen A, Kaur M, Kawaji H, Kerr MC, Kimura R, Kimura S, Kimura Y, Kitano H, Koga H, Kojima T, Kondo S, Konno T, Krogh A, Kruger A, Kumar A, Lenhard B, Lennartsson A, Lindow M, Lizio M, Macpherson C, Maeda N, Maher CA, Maqungo M, Mar J, Matigian NA, Matsuda H, Mattick JS, Meier S, Miyamoto S, Miyamoto-Sato E, Nakabayashi K, Nakachi Y, Nakano M, Nygaard S, Okayama T, Okazaki Y, Okuda-Yabukami H, Orlando V, Otomo J, Pachkov M, Petrovsky N, Plessy C, Quackenbush J, Radovanovic A, Rehli M, Saito R, Sandelin A, Schmeier S, Schönbach C, Schwartz AS, Semple CA, Sera M, Severin J, Shirahige K, Simons C, St Laurent G, Suzuki M, Suzuki T, Sweet MJ, Taft RJ, Takeda S, Takenaka Y, Tan K, Taylor MS, Teasdale RD, Tegnér J, Teichmann S, Valen E, Wahlestedt C, Waki K, Waterhouse A, Wells CA, Winther O, Wu L, Yamaguchi K, Yanagawa H, Yasuda J, Zavolan M, Hume DA; Riken Omics Science Center, Arakawa T, Fukuda S, Imamura K, Kai C, Kaiho A, Kawashima T, Kawazu C, Kitazume Y, Kojima M, Miura H, Murakami K, Murata M, Ninomiya N, Nishiyori H, Noma S, Ogawa C, Sano T, Simon C, Tagami M, Takahashi Y, Kawai J, Hayashizaki Y.

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<Affiliation>RIKEN Omics Science Center, RIKEN Yokohama Institute, Kanagawa, Japan.</Affiliation>
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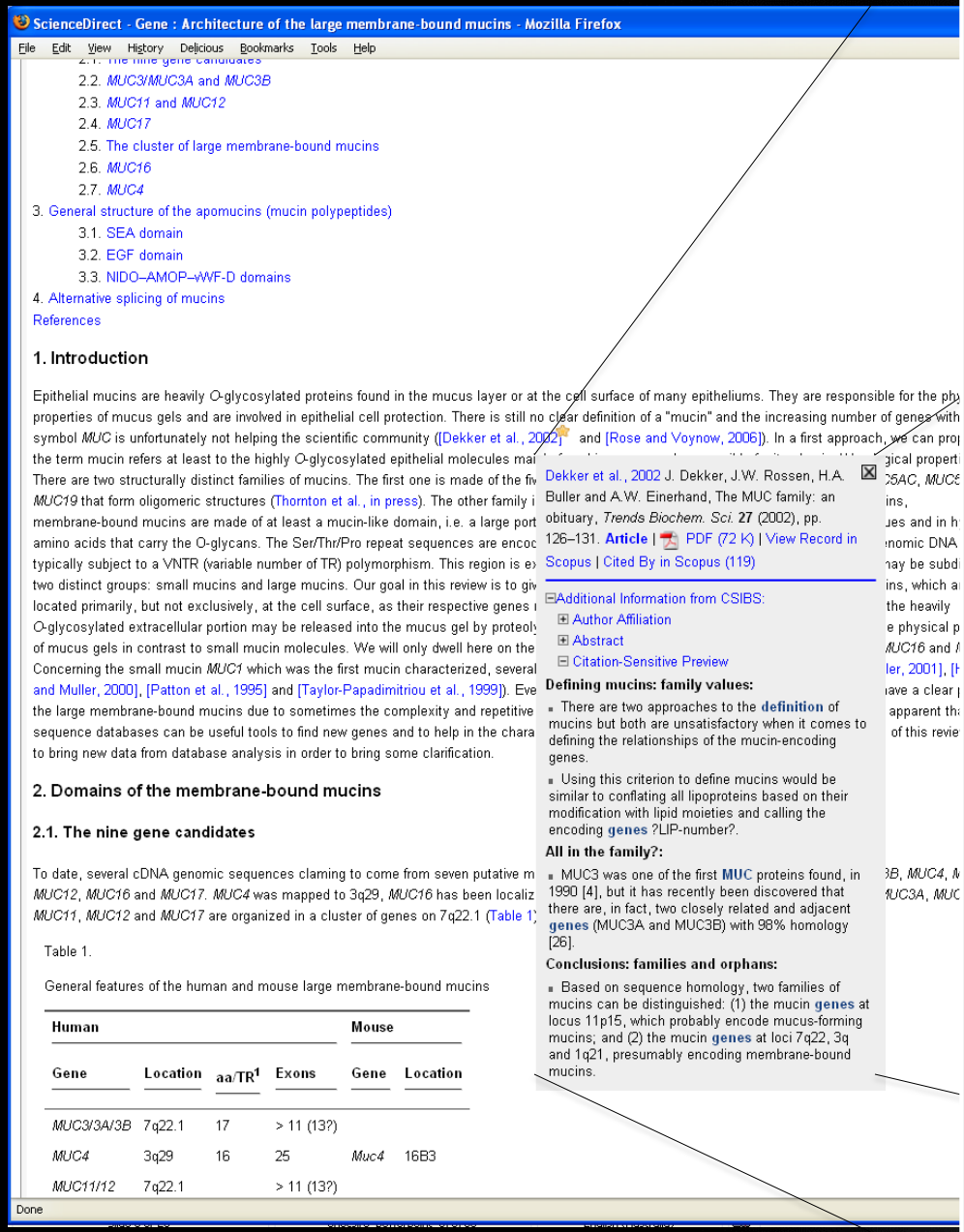

Dekker et al., 2002 J. Dekker, J.W. Rossen, H.A. Buller and A.W. Einerhand, The MUC family: an obituary, *Trends Biochem. Sci.* **27** (2002), pp. 126–131. **Article** | PDF (72 K) | View Record in Scopus | Cited By in Scopus (119)

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Defining mucins: family values:

- There are two approaches to the **definition** of mucins but both are unsatisfactory when it comes to defining the relationships of the mucin-encoding genes.
- Using this criterion to define mucins would be similar to conflating all lipoproteins based on their modification with lipid moieties and calling the encoding **genes** ?LIP-number?.

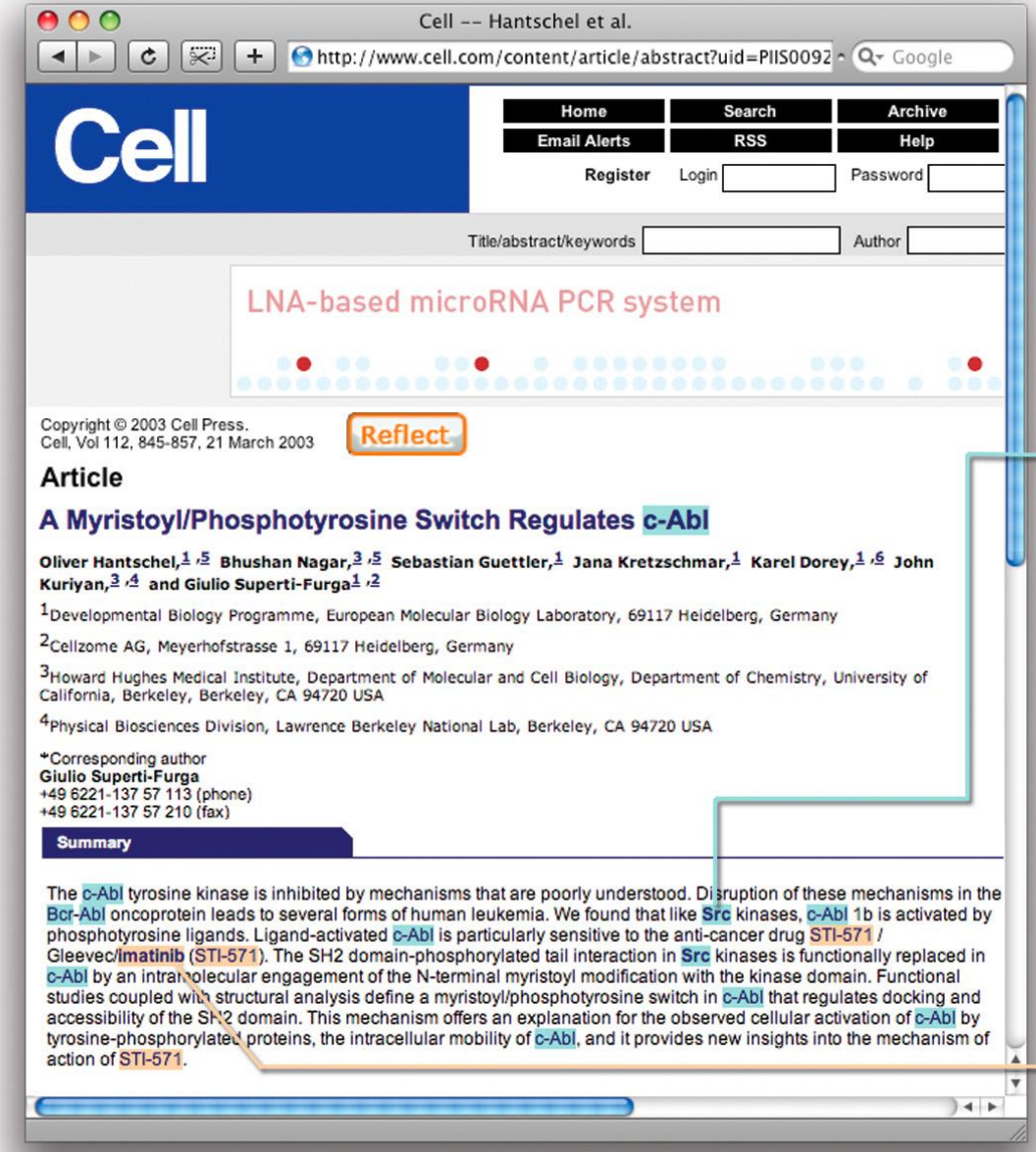
All in the family?:

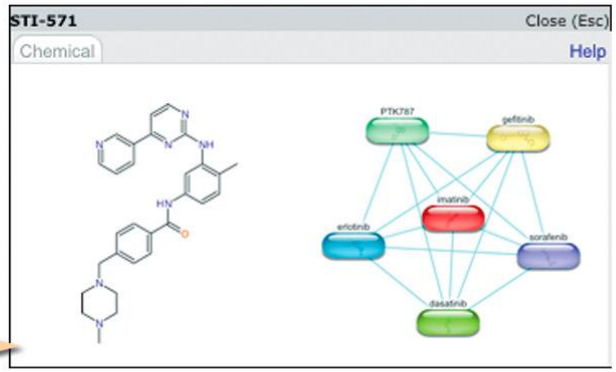
- MUC3 was one of the first **MUC** proteins found, in 1990 [4], but it has recently been discovered that there are, in fact, two closely related and adjacent **genes** (MUC3A and MUC3B) with 98% homology [26].

Conclusions: families and orphans:

- Based on sequence homology, two families of mucins can be distinguished: (1) the mucin **genes** at locus 11p15, which probably encode mucus-forming mucins; and (2) the mucin **genes** at loci 7q22, 3q and 1q21, presumably encoding membrane-bound mucins.

Obogaćivanje članaka multimedijom





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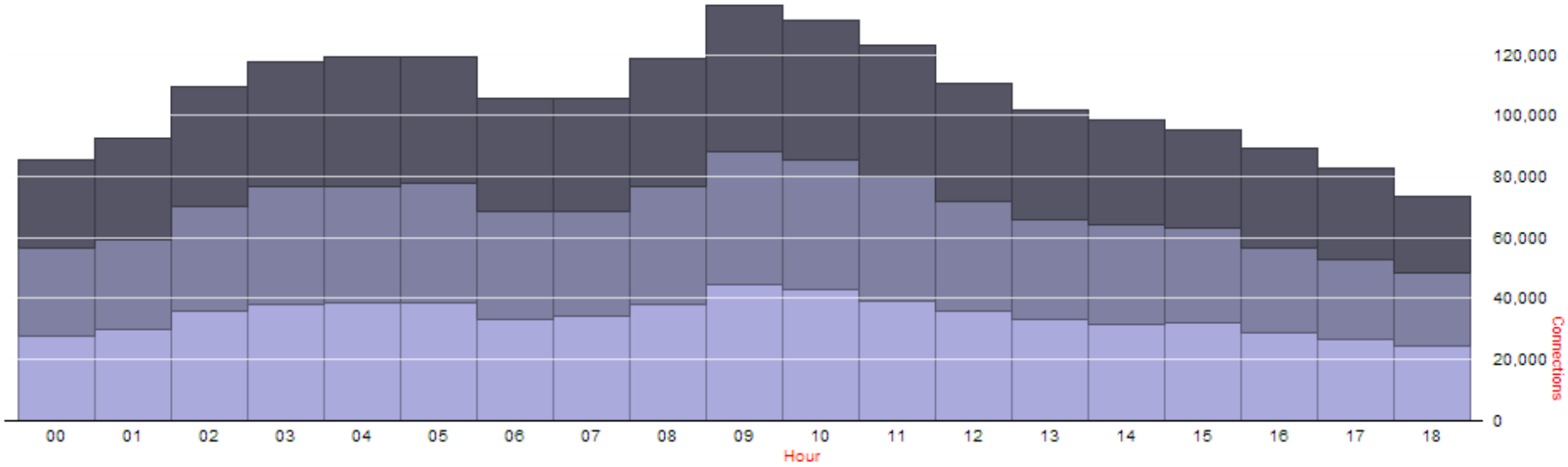

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[b] AIFB, Karlsruhe Institute of

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Abstract

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