

Connecting Research and Researchers

Patricia Hartman

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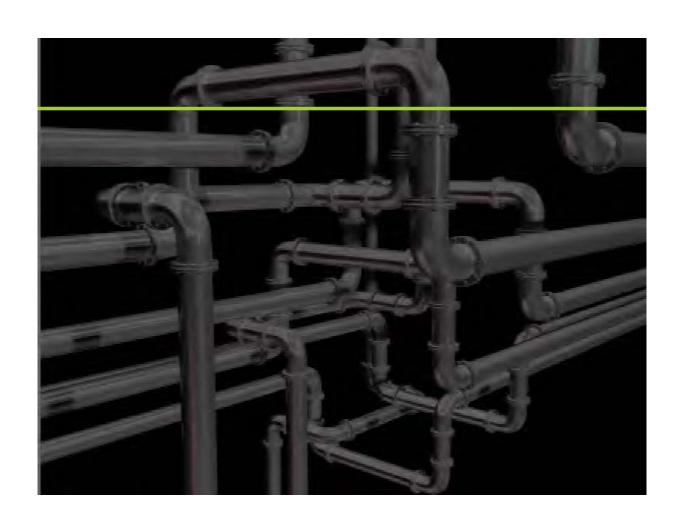
Biological Sciences, Forestry & Wildlife, Mathematics Librarian

What Is ORCID?



- Registry of unique persistent identifiers for researchers
- Open non-profit organization
- Hub enabling machine-readable connections between researchers, research organizations, granting agencies, publishers

Plumbing for scholarly communication infrastructure

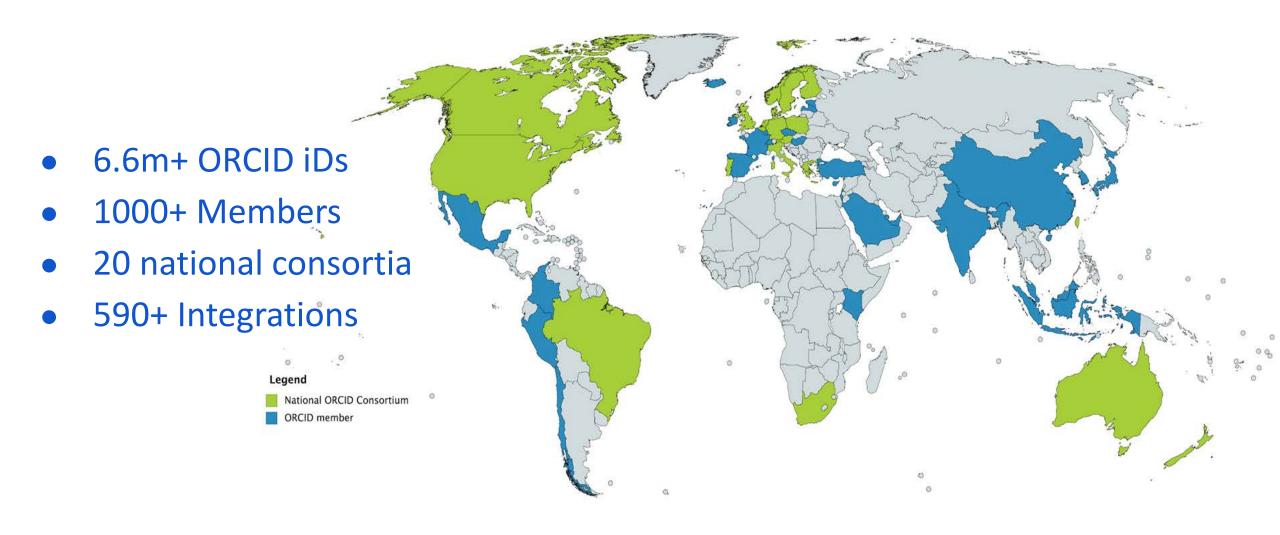


ORCID's VISION

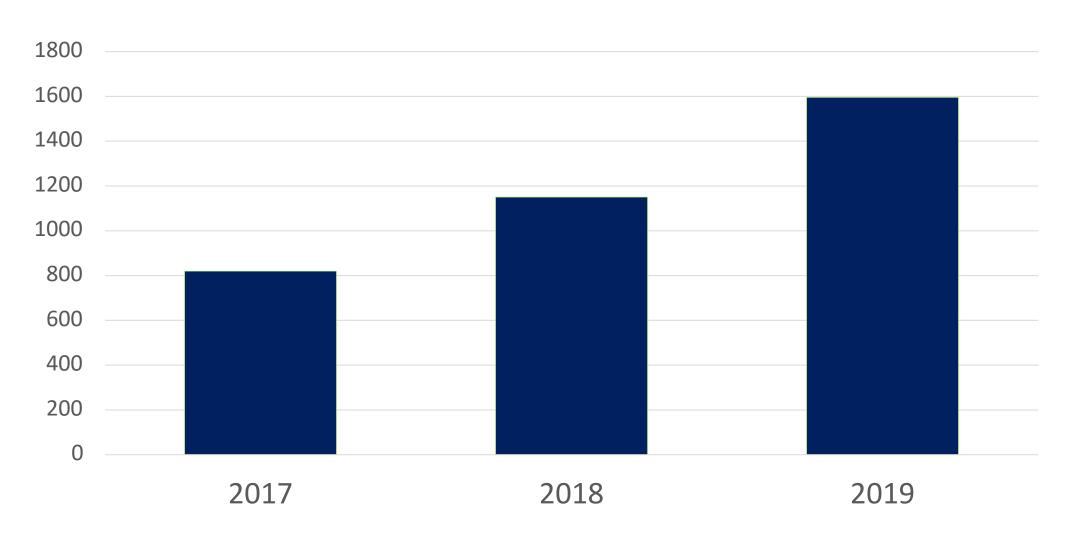
"A world where all who participate in research, scholarship, and innovation are uniquely identified and connected to their contributions across disciplines, borders, and time."



ORCID: Global Picture

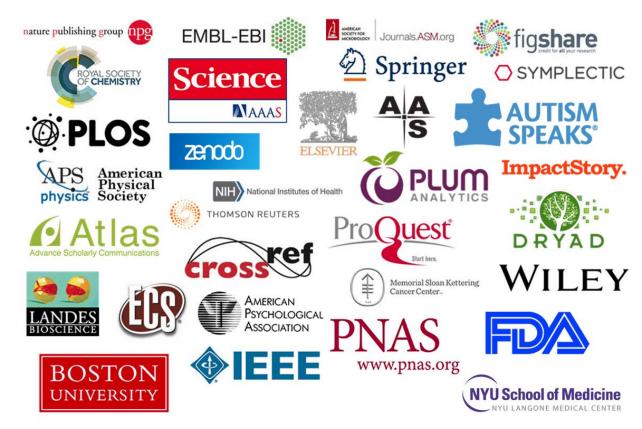


Number of ORCID IDs with auburn.edu email addresses



It's often required

- Journals
- Grant applications
- University IDs



Integration with organizations, publishers and associations

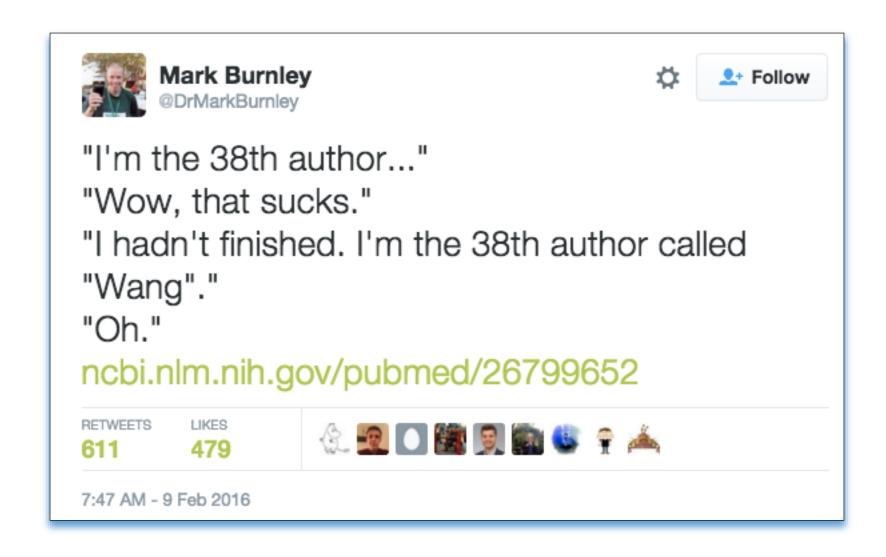
ORCID Open Letter - Publishers

Join the many publishers that have committed to requiring ORCID iDs in the publishing process for their journal(s)!

- Required (80 signatories)
 - Science
 - IEEE
 - Springer Nature
 - Wiley
 - The Royal Society
 - PLoS
 - BMJ Journals
 - Cambridge Univ Press
 - The Company of Biologists...

- Optional
 - Many academic societies
 - Nature Publishing Group
 - Taylor & Francis
 - Proceedings of the National Academy of Sciences
 - Oxford University Press

Why else would you want an ORCID ID?



S NCBI Resources ☑	How To ☑
le .	
Public ed.gov	PubMed \$
US National Library of Medicine National Institutes of Health	Advanced

Abstract → Send to: →

Autophagy. 2016 Jan 2;12(1):1-222.

Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition).

Klionsky DJ, Abdelmohsen K, Abe A, Abedin MJ, Abeliovich H, Acevedo Arozena A, Adachi H, Adams CM, Adams PD, Adeli K, Adhihetty PJ, Adler SG, Agam G, Agarwal R, Aghi MK, Agnello M, Agostinis P, Aguilar PV, Aguirre-Ghiso J, Airoldi EM, Ait-Si-Ali S, Akematsu T, Akporiaye ET, Al-Rubeai M, Albaiceta GM, Albanese C, Albani D, Albert ML, Aldudo J, Algül H, Alirezaei M, Alloza I, Almasan A, Almonte-Beceril M, Alnemri ES, Alonso C, Altan-Bonnet N, Altieri DC, Alvarez S, Alvarez-Erviti L, Alves S, Amadoro G, Amano A, Amantini C, Ambrosio S, Amelio I, Amer AO, Amessou M, Amon A, An Z, Anania FA, Andersen SU, Andley UP, Andreadi CK, Andrieu-Abadie N, Anel A, Ann DK, Anoopkumar-Dukie S, Antonioli M, Aoki H, Apostolova N, Aquila S, Aquilano K, Araki K, Arama E, Aranda A, Araya J, Arcaro A, Arias E, Arimoto H, Ariosa AR, Armstrong JL, Amould T, Arsov I, Asanuma K, Askanas V, Asselin E, Atarashi R, Atherton SS, Atkin JD, Attardi LD, Auberger P, Auburger G, Aurelian L, Autelli R, Avagliano L, Avantagqiati ML, Avrahami L, Awale S, Azad N, Bachetti T, Backer JM, Bae DH, Bae JS, Bae ON, Bae SH, Baehrecke EH, Baek SH, Baghdiguian S, Bagniewska-Zadworna A, Bai H, Bai J, Bai XY, Bailly Y, Balaji KN, Balduini W, Ballabio A, Balzan R, Banerjee R, Bánhegyi G, Bao H, Barbeau B, Barrachina MD, Barreiro E, Bartel B, Bartolomé A, Bassham DC, Bassi MT, Bast RC Jr, Basu A, Batista MT, Behl C, Behrends C, Behrens GM, Behrns KE, Bejarano E, Belaid A, Belleudi F, Bénard G, Berchem G, Bergamaschi D, Bergami M, Berkhout B, Berliocchi L, Bernard A, Bernard M, Bernassola F, Bertolotti A, Bess AS, Besteiro S, Bettuzzi S, Bhalla S, Bhattacharyya S, Bhutia SK, Biagosch C, Bianchi MW, Biard-Piechaczyk M, Billes V, Bincoletto C, Bingol B, Bird SW, Bitoun M, Bjedov I, Blackstone C, Blanc L, Blanco GA, Blomhoff HK, Boada-Romero E, Böckler S, Boes M, Boesze-Battaglia K, Boise LH, Bolino A, Boman A, Bonaldo P, Bordi M, Bornolini C, Braus GH, Bravo-San Pedro JM, Brennan LA, Bresnick EH, Brest P,

Ventura N, Ventura S, Veras PS, Verdier M, Vertessy BG, Viale A, Vidal M, Vieira H, Vierstra RD, Vigneswaran N, Vij N, Vila M, Villar M, Villar M, Villar VH, Villarroya J, Vindis C, Viola G, Viscomi MT, Vitale G, Vogl DT, Voitsekhovskaja OV, von Haefen C, von Schwarzenberg K, Voth DE, Vouret-Craviari V, Vuori K, Vyas JM, Waeber C, Walker CL, Walker MJ, Walter J, Wan L, Wan X, Wang B, Wang C, Wang C, Wang C, Wang C, Wang C, Wang D, Wang F, Wang F, Wang G, Wang HJ, Wang HG, Wang HD, Wang J, Wang J, Wang M, Wang MQ, Wang PY, Wang P, Wang RC, Wang S, Wang TF, Wang X, Wang XJ, Wang XW, Wang X, Wang X, Wang Y, Wa

Resolves name ambiguity issues

- Shared names
- Different versions
- Transliteration
- Accents or other ALT characters
- Name changes

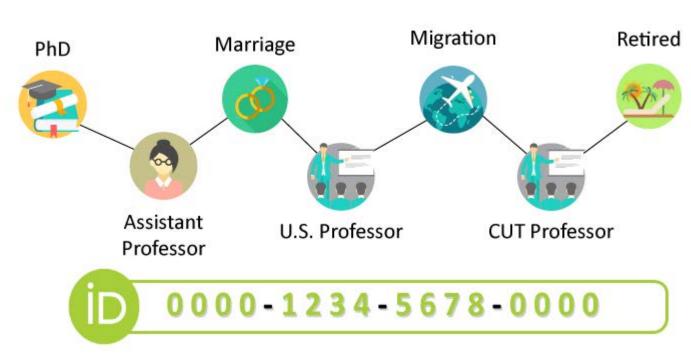






Stays with you wherever you go





Credit for different types of wo series (\$\frac{30}{34}\$) | \frac{1 \lambda{5} \frac{1}{3} \frac{1}{3}

- Articles
- Books
- Grants
- Software
- Musical scores
- Conference posters
- Peer-review



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'. \$file; if (\$color!=='' && (strlen(\$color)!=6 || !ctype_xdigit(\$color)) || intval(\$size)<=0) return FALSE; /* \$size or \$color is not valid */ substr(\$file,-4)!='.bmp')) return FALSE;/* skip non-images and thumbs \$thumbn = dirname(\$file).'/_thumb_'.\$size.(\$color?'_'.lower(\$color): '').'_'.substr(basename(*\$file*).0,strrpos(basename(*\$file*).'.')).'.gif'; if (file_exists(\$thumbn)) return FALSE; if (substr(\$file, -4)=='.gif') \$srci = imagecreatefromgif(\$file);
else if (substr(\$file, -4)=='.png') \$srci = imagecreatefrompng(\$file);
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nature cell biology

Spatial regulation of VEGF receptor endocytosis in angiogenesis

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Masanori Nakayama^{1,8}, Akiko Nakayama¹, Max van Lessen¹, Hiroyuki Yamamoto¹, Sarah Hoffmann¹, Hannes C. A. Drexler², Norimichi Itoh³, Tomonori Hirose⁴, Georg Breier⁵, Dietmar Vestweber⁶, Jonathan A. Cooper7, Shigeo Ohno4, Kozo Kaibuchi3 and Ralf H. Adams1,8

Activities as diverse as migration, proliferation and patterning occur simultaneously and in a coordinated fashion during tissue morphogenesis. In the growing vasculature, the formation of motile, invasive and filopodia-carrying endothelial sprouts is balanced with the stabilization of blood-transporting vessels. Here, we show that sprouting endothelial cells in the retina have high rates of VEGF uptake, VEGF receptor endocytosis and turnover. These internalization processes are opposed by atypical protein kinase C activity in more stable and mature vessels. aPKC phosphorylates Dab2, a clathrin-associated sorting protein that, together with the transmembrane protein ephrin-B2 and the cell polarity regulator PAR-3, enables VEGF receptor endocytosis and downstream signal transduction. Accordingly, VEGF receptor internalization and the angiogenic growth of vascular beds are defective in loss-of-function mice lacking key components of this regulatory pathway. Our work uncovers how vessel growth is dynamically controlled by local VEGF receptor endocytosis and the activity of cell polarity proteins.

The biological activity of growth factor receptors is tightly controlled during growth and patterning processes. While internalization is often seen as a means of terminating signals or degrading receptors, it can also generate qualitatively or quantitatively distinct signalling responses 1-3. Consequently, the positive or negative regulation of endocytosis might facilitate specialized biological activities of certain cells or cell groups within a larger population, as they are frequently seen in morphogenesis4. In the angiogenic vasculature, sprouting involves the sive, and extend filopodia to detect tissue-derived cues such as vascular endothelial growth factors (VEGFs). These ligands (primarily VEGF-A and VEGF-C) trigger the homo- or heterodimerization of their cognate control sprouting and proliferation 5-7. Tip cells are thought to have the highest levels of VEGF receptor signalling because they lead sprouts and cell behaviours are presumably not fixed and rather reflect transient, in-

terconvertible phenotypes and constant competition of endothelial cells for the tip position 6.8. This process involves the Notch pathway, which is thought to downregulate VEGF receptor expression and is therefore presumably less active in tip cells⁹⁻¹¹. Another cell-contact-dependent signalling molecule, the Eph receptor ligand ephrin-B2 (encoded by the Efnb2 gene), promotes the invasive behaviour of endothelial cells and is required for normal VEGF receptor endocytosis and signalling 12-

Physiological angiogenesis also involves the gradual conversion of specialization of endothelial tip cells, which are highly motile and invadothelial cells are increasingly quiescent, show a phalanx-like morphology and are devoid of VEGF-induced activities such as the extension of filopodia or proliferation15. The postnatal vascularization of the retina endothelial receptors (VEGFR2/Flk1 and VEGFR3/Flt4, respectively) in the mouse is an excellent model system for angiogenic sprouting and thereby activate downstream signal transduction cascades that and maturation, because sequentially occurring processes are spatially separated and can be imaged at high resolution 16. Tip- and stalk-cellcontaining sprouts can be found at the peripheral edge of the growing might therefore encounter higher ligand concentrations than trailing vascular plexus next to VEGF-producing tissue regions, whereas the prestalk cells. The latter form the sprout base, maintain a lumenized conviously established, more mature vessels are located in the central retina. nection to the existing vasculature and lack long filopodia. Tip and stalk Here, we show that angiogenesis is controlled by spatially

1 Max Planck Institute for Molecular Biomedicine, Department of Tissue Morohogenesis, and University of Muenster, Faculty of Medicine, D-48149 Muenster, Germany,

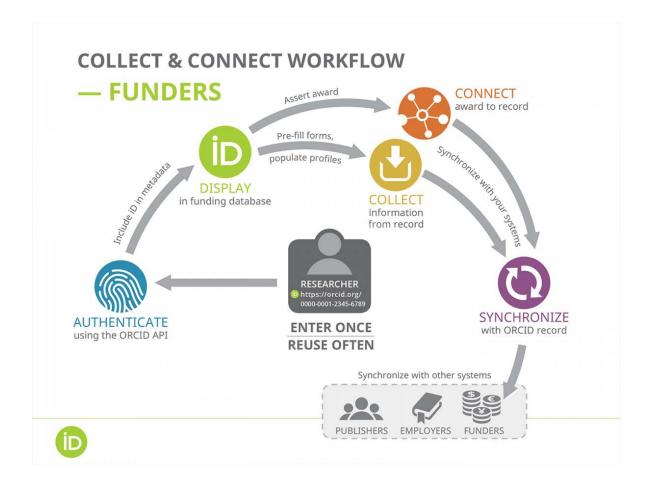
Increases discoverability of your work

- Citable
- Sharable
- Searchable in databases
- Under your control



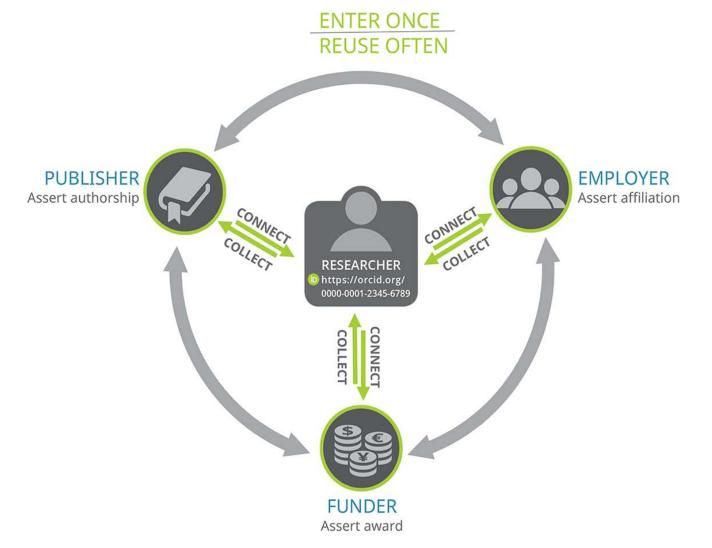
Funding Agencies

- Included in application process
 - National Institute of Health
 - CDC
 - NSF
 - NASA
 - Department of Energy
 - Health Research Alliance
 - World Health Organization
 - ...many more!



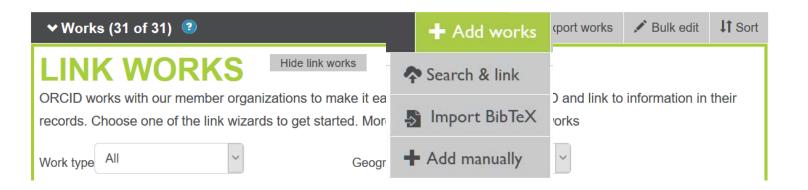
Not in place for all granting agencies **YET**, but it will be a reality soon.

Spend more time working, less filling out forms



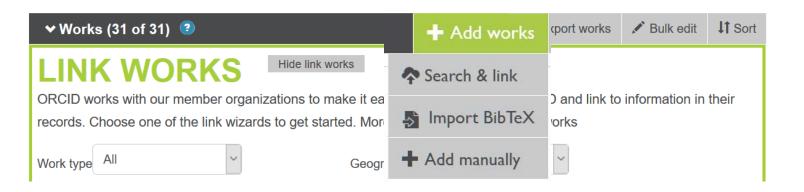
Easy to Add Existing Works

- Google Scholar
- Researcher ID/ Publons ID
- Scopus
- DataCite
- CrossRef



Easy to Add Existing Works

- Google Scholar
- Researcher ID/ Publons ID
- Scopus





Add ulysses@auburn.edu as a trusted user for assistance

ORCID Benefits for Research Institutions

Mitigate confusion caused by name ambiguity

Assess individual contributions & measure institutional impact



Assert trustworthy & accurate affiliations

Save time & reduce administrative burden

Integrating with AU's research processes

- We are members of the US Community Consortium
 - 88 are R1 institutions
- As members, we have 5 application programming interfaces (APIs) at no additional charge



Institutions with existing ORCID integrations

• Total: 75+

R1 Institutions: 58 and growing

Early adopters

Boston University

Cornell

Duke

University of Michigan

SEC Schools

Mississippi State

Texas A&M

University of Missouri

University of Tennessee

Others are in development

How Can Auburn University Use ORCID?

- Create and connect to institutional account
- As members of the US ORCID Community (108 members),
 we have access to features we are not taking advantage of









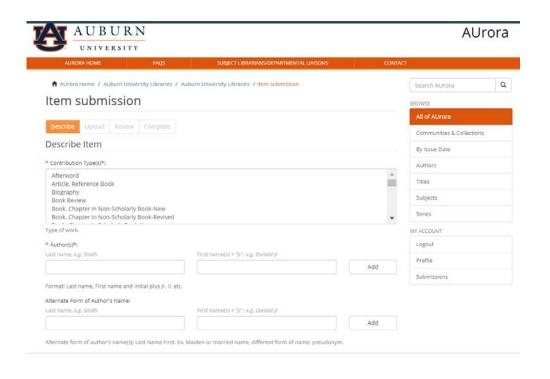
Identity Management

- Standardize institutional affiliation data
- Pull ORCID data directly
- More reliable metadata
- Display in directory
- Showcase researchers



AUrora & ETDs

 Allows automatic addition to ORCID record



Examples of ORCID-Integrated Repositories

Colorado State University

Purdue University

Virginia Tech

Duke University

University of Missouri

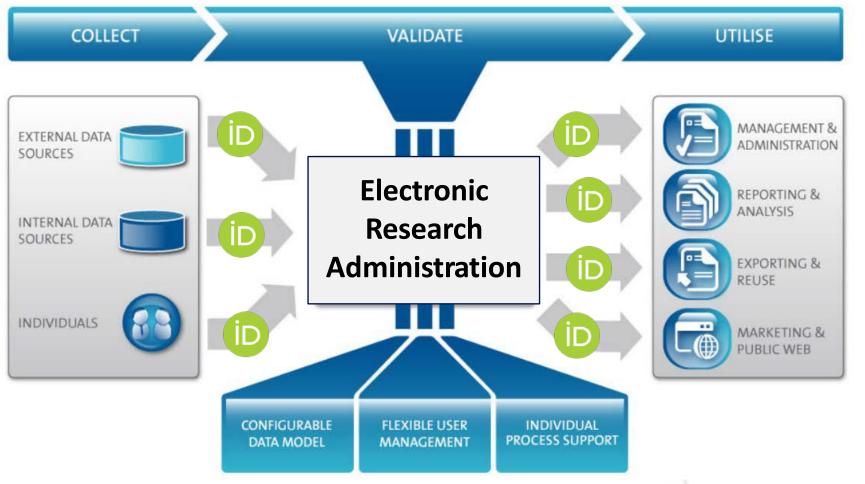
University of Florida

University of Notre Dame

University of Michigan

Texas A&M University

Electronic Research Administration



- Reduces process delays
- Increases consistency
- Improves
 reporting
 compliance and
 accuracy

Status of ORCID Integration at AU

- Administrative support
 - Vice-President for Research
 - Associate Deans for Research
- OIT
 - Banner integration in development
- AU Libraries
 - Prepared to support faculty and students
 - Integrate with AUrora and ETDs

Thank you!

For more information about ORCID at AU, please visit

libguides.auburn.edu/ORCID



Questions? Contact Patricia Hartman (pjh0011@auburn.edu)