

Okazaki, J Vasc 2018, 4:1

Relationship between Deviated Fingerprint Distribution and Vascular Factor Abnormality

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Research Article

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Received date: September 11, 2018; Accepted date: September 27, 2018; Published date: October 4, 2018

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Abstract

Background: Fingerprint patterns are roughly classified into 3 types loop, whorl, and arch. I noticed in the previous study that the fingerprint ridge distribution is closely related to the vein distribution under the fingertip. Therefore, in this study, I intended to prove the relationship between the deviation of fingerprint patterns and the changes/fluctuation of factors relating to vascular formation.

Methods: The subcutaneous vascular distributions of each fingerprint type were observed using a monitoring device, comprising an IR-CCD camera and a near-infrared LED lighting system. By reviewing the literature to date regarding the dermatoglyphic ridge configurations and the vascular factor abnormality of individuals with various diseases, diseases were classified into four groups: ulnar-loop type deviations, whorl type deviations, whorl and arch type deviations, and arch type deviations.

Results: There was a close relationship between the fingerprint type and the subcutaneous vascular distributions. The four groups of fingerprint type deviations were associated with abnormalities of VEGF, EPO, TNFα, and PDGF/PDGFR vascular factors, respectively.

Conclusions: I propose that the shape of the fingerprint is closely related to the subcutaneous vein distribution and the fingerprint type deviations are related to vascular factor abnormalities.

Keywords: Fingerprint; Blood vessel; Genetic disease; Vascular factor; Angiogenesis

Introduction

Fingerprints are ridges wavy aligned at regular intervals on the finger skin surface. They are formed by 10 weeks of gestation in the fetal period and the pattern does not change over lifetime [1-3]. The fingerprints of the palmar distal phalanges are classified based on their pattern into the loop (ulnar and radial types), whorl, and arch types [4]. Since the fingerprint ridge pattern differs among individuals, fingerprints have been used for individual identification, over the world, for more than 100 years [5,6]. In the early half of the 1900s, studies on the distribution of fingerprints in ethnic groups were actively performed in each country from anthropological viewpoints [7-9].

In the middle of the 1900s-early 2000s, with the development of genetic testing, many studies on the relationship between genetic diseases and fingerprints were performed [10-27]. Although fingerprint distributions, characteristic to ethnic groups and diseases were reported, no attention was paid to these data because it was unclear by what fingerprint patterns are controlled.

However, we recently observed the distribution of subcutaneous veins in the distal phalanges of the fingers using a simple system comprised of a near-infrared LED lighting device and an infrared CCD camera and compared these with the ridge patterns, and noticed that they were closely related [28]. In this study, firstly, I carefully observed

the thickness, distribution, location, and distribution density of subcutaneous blood vessels under the loop, whorl, and arch pattern ridges.

In addition, I searched for literature on abnormalities of vasculogenic and angiogenic factors in diseases in which deviations of the fingerprint distribution have been pointed out (particularly, genetic diseases) [29-48]. Based on these findings, I discussed the association between the deviated fingerprint distribution and abnormality of factors involved in angiogenesis in each disease. It was suggested that clarification of the association between the deviated fingerprint distribution and abnormality of factors may lead to the assumption of new abnormalities of vasculogenic and angiogenic factors involved in diseases and clues to the discovery of a new developmental mechanism of diseases previously not known to be associated with these abnormalities of factors.

Materials and Methods

Materials

Fingerprint and subcutaneous vascular distribution patterns were bilaterally investigated from the second to the fifth distal phalanges of 35 healthy adult male/female aged 25-73 years with no genetic diseases. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee, and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Observation and classification of fingerprints

A Power Shot Pro1 optical camera (Cannon, Tokyo) was used to capture super macro photographs of fingerprint ridge patterns under strobe lighting. Fingerprint patterns were then classified into loop (ulnar and radial types), whorl (or annular), arch, or other types.

Observation of subcutaneous blood vessels in the distal phalanges

Photographs of subcutaneous blood vessels distributed several millimeters deep were acquired using a system incorporating the use of a near-infrared output illuminator and an infrared CCD camera. The 850 nm wavelength near-infrared light used for lighting is readily absorbed by blood hemoglobin, allowing the subcutaneous vascular distribution to be observed as black outlines by the near-infrared CCD camera.

A schematic of the acquisition system is shown in Figure 1, and its details and experimental methods are described below. A near-infrared output illuminator (wave length 850 nm, NISSIN ELECTRONIC Co., Ltd., Tokyo) was attached to the dorsal side of the distal phalanx of each finger. Photographs were taken from the palmar side using an infrared CCD camera (XC-E150, SONY, Japan) with an L6000 macro lens (Edmund Optics Japan, Tokyo). The vascular distributions in the acquired images were converted to gray scale for increased clarity.



observation of vascular distribution (The analytical system is consisting of an IR-CCD camera and 850 nm LED illumination).

Literature review strategy

MEDLINE (PuMed) and Google scholar electronic databases were used to search for articles published before December 2017, and total 38 articles were referred in order to clarify the relationship between the fingerprint type deviations and the vascular factor abnormalities.

Results

Relationship between fingerprint patterns and subcutaneous vascular distribution

Following investigation of subcutaneous vascular distributions in the distal phalanges using a system incorporating a near-infrared lighting device and an infrared CCD camera, vein distributions were found to differ among the fingerprint pattern types. Representative photographs of ridge distributions in the ulnar-loop, whorl, and arch types, and subcutaneous vascular distributions are shown in Figure 2A. Regarding the ulnar-loop, whorl, and arch types, the number of cases was increased, and the characteristics of the vascular distributions were determined.

In individuals with the ulnar-loop fingerprint pattern, veins on one side extended toward the distal side compared with veins on the contralateral side, and the distributions of venules and capillary blood vessels in the proximal-distal axis were also asymmetric. On the other hand, the whorl pattern type was typically characterized by symmetrically distributed blood vessels in the proximal-distal axis, and a collection of short veins near the first joint. Blood vessels were also symmetrically distributed in the arch pattern type; however, there were fewer bold veins, and a broad distribution of venules and capillary blood vessels (Figure 2B).



Figure 2: Representative examples indicating the relationship between the fingerprint ridge pattern and vein distribution, and comparison of the vein distribution in ulnar-loop, whorl and arch type fingertip patterns. (A) upper, fingerprint ridge; lower, vein distribution. The white dashed lines indicate the fingerprint ridge pattern; (B) The black solid lines indicate the main subcutaneous blood vessels.

Relationship between fingerprint patterns, genetic diseases, and vascular factor abnormalities

I began by reviewing the literature to date regarding the dermatoglyphic ridge configurations of individuals with various diseases. Diseases were then classified by type of deviations from the standard appearance of the ulnar-loop, whorl, and arch type fingerprint patterns in Table 1 [10-27]. Group I diseases were associated with a high occurrence of deviations of the ulnar-loop fingerprint pattern type, and Group II diseases were associated with a high occurrence of deviations of the whorl and arch fingerprint pattern type. Group II was further subdivided into: IIa, IIb, and IIc, in which the occurrence of deviations from the whorl type, both the whorl and arch types, the arch type, were common, respectively. Group I included type 2 diabetes, Alzheimer's disease, breast cancer, and trisomy 21 (Down's syndrome), and activation of VEGF-related factors, such as

increases in expression of vascular endothelial growth factor (VEGF) and down-syndrome candidate region-1 (DSCR-1) which promotes VEGF expression, were reported in common in these diseases.

In addition, changes/fluctuation of factors influencing blood vessel formation, such as reduction of circulating angiogenic cells (CACs), and reduction and aberration of breast cancer susceptibility gene (BRCA), were reported. Group IIa included acute lymphocytic leukemia, β -thalassemia, and myocardial infarction, and an increase in

erythropoietin (EPO) was confirmed in common in these diseases. Group IIb included cerebral palsy, fragile X syndrome, and Huntington's disease, and an increase in tumor necrosis factor- α (TNF- α) was reported in common in these diseases. Group IIc included autism, epilepsy, and trisomy 18, and abnormalities of platelet-derived growth factor (PDGF) and platelet-derived growth factor receptor A (PDGFRA) were reported in common in these diseases [29-48].

| Fingerprint Pattern | | | | | | Refs | Diseases | Abnormalities of vascular factors |
|---------------------|----|--------|----------|-------|----------|---------|-------------------------------|---|
| | | U-Loor | R-loop | Whorl | Arch | - Reis | Diseases | Abnormanties of vascular factors |
| | | 1 | 1 | Ļ | 1 | [10] | Diabetes Mellitus Type 2 | VEGF ↑ [29] |
| I | | Ť | 1 | 1 | 1 | [11,12] | Alzheimer's Diseases | VEGF ↑[30], CACs ↓ [31], BRCA1 ↓ [32] |
| | | Ť | 1 | Ļ | Ļ | [13,14] | Breast Cancer | VEGF ↑[33] Loss of Heterozygosity i BRCA 1,2 [34,35] |
| | | Ť | 1 | 1 | 1 | [15,16] | Trisomy 21 (Down's Syndrorne) | DSCR-1 ↑ [36,37] |
| II | a. | Ļ | 1 | Ť | Ļ | [17] | ALL | - - EPO ↑[38-41] |
| | | Ļ | 1 | Ť | Ļ | [18,19] | Beta-thalasemia | |
| | | Ļ | 1 | Ť | Ļ | [20] | Myocardial Infarction | |
| | | Ļ | 1 | Ť | 1 | [21] | Congenital Cardiac Disease | |
| | b. | Ļ | 1 | 1 | Ť | [22] | Cerebral Palsy | TNF-α ↑ [42-44] |
| | | Ļ | ↑ | Ť | Ť | [23] | Frangile-X-Syndrome | |
| | | Ļ | 1 | Ť | 1 | [24] | Hantington's Diseases | |
| | C. | 1 | 1 | Ļ | Ť | [25] | Autism | PDGF ↑ [35] |
| | | 1 | 1 | Ļ | Ť | [26] | Epilepsy | Abnormal PDGF and/or PDGFRalph [46,47] |
| | | Ļ | 1 | 1 | ↑ | [27] | Trisomy 18 | PDGFRA promoter haplotypes [48] |

↑: increase; ↓: decrease; ALL: acute lymphoid leukemia; VEGF: Vascular Endothelial Growth Factor; CACs: Circulating Angiogenic Cells; BRCA1: Breast Cancer Susceptibility Gene-1; DSCR-1: Down Syndrome Candidate Region-1; EPO: Erythropoetin; TNF-α: Tumor Necrosis Factor-α; PDGF: Platelet-Derived Growth Factor; PDGFRA: Platelet-Derived Growth Factor Receptor A.

 Table 1: Relationship between fingerprint patterns and genetic diseases associated with abnormal changes/fluctuation of vascular factors.

Discussion

When the subcutaneous vascular distributions in the distal phalanges were observed and compared with the fingerprints using a system combining a near-infrared lighting device and an infrared CCD camera (Figure 1), the vein distribution differed among the fingerprint patterns (Figure 2), suggesting the presence of a close relationship between the fingerprints and subcutaneous vascular distributions.Detailed examination of the positional relationship between fingerprints and blood vessels further clarified the close relationship between each other [28].

Although the specific influences of changes/fluctuation in vasculogenic and angiogenic factors on fingerprint formation and their details are unclear, there may be a close association between deviations of the fingerprint distribution and changes/fluctuation in vasculogenic and angiogenic factors. There previous reports on the deviated fingerprint distributions in various diseases and reports on changes/

fluctuation of angiogenic factor were searched for and organized in Table 1. An extensive literature review revealed that genetic diseases associated with a high occurrence of deviations from the loop type fingerprint patterns (Group I), were associated with increases in VEGF and DSCR1 [29,30,33,36,37]. DSCR1 isoform 1 is VEGF activityenhancing factor [36], and VEGF gene expression is induced by hypoxia, and plays a central role in angiogenesis [49]. Reduced CACs and BRCA expression, and BRCA gene aberration have been reported in Alzheimer's disease [31,32]. CACs are the most basic material for angiogenesis [50], and a recent study demonstrated that BRCA interacts with ER-a to inhibit estrogen-induced VEGF expression and secretion [51]. Reduced BRCA expression may induce the unbalanced upregulation of VEGF, which may cause asymmetric distribution of subcutaneous blood vessels in the loop type fingerprint pattern. Diseases in Group IIa, associated with a high occurrence of deviations in the whorl type fingerprint pattern, demonstrate increase in plasma erythropoietin (EPO) concentration [38-41]. EPO is essential for

erythropoiesis, and a recent study indicates that EPO, and its receptor, also regulate Angiopoetin-1, involved in controlling angiogenesis [52]. An increase in plasma EPO concentration may induce the formation of short and bold veins near the first joint of the finger. Diseases in Group IIb, demonstrating a high occurrence of the whorl and arch type fingerprint patterns, are associated with increased plasma TNF- α concentration [42-44]. TNF- α promotes angiogenesis associated with upregulation of VEGF and hypoxia-inducible factor-1 α [53,54].

An increase in plasma TNF-a concentration may induce the distribution of short bold veins and an increase in capillary blood vessels. Diseases in Group IIc, marked by the high occurrence of arch type fingerprint patterns, were associated with reports of abnormally high PDGF expression in autism patients [45], and abnormal PDGF and PDGFR expression in epilepsy and trisomy 18 [46-48]. PDGF indicates the high-affinity with VEGR [55], and indirectly stimulates capillary blood vessel formation in vitro [56,57]. Abnormal PDGF or PDGFR expression may induce an increase in capillary blood vessels in substitution for bold veins. From these results, it is assumed that dermatoglyphic ridge configuration is closely related to changes/ fluctuations in the expression of angiogenesis-related factors. The association between the deviations of the standard fingerprint patterns and abnormalities of vasculogenic and angiogenic factors in several genetic diseases may provide a novel biomarker for the screening of risk for developing genetic diseases in later life.

References

- Wheeler T, Godfrey K, Atkinson C, Badger J, Kay R, et al. (1998) Disproportionate fetal growth and fingerprint patterns. Brit J Obstet Gynaecol 105: 562-564.
- Dahiya RS, Gori M (2010) Probing with and into fingerprints. J Neurophysiol 104: 1-3.
- 3. Kucken M, Newell AC (2005) Fingerprint formation. J Theor Biol 235: 71-83.
- 4. Penrose LS (1968) Medical significance of finger-prints and related phenomena. Brit Med J 2: 321-325.
- 5. Herschel WJ (1880) Skin furrows of the hand. Nat 23:76.
- 6. Faulds H (1880) On the skin-furrows of the hand. Nature 22: 605.
- 7. Kristine B (1924) Studies on papillary patterns of human fingers. J Genet 15: 1-111.
- Cummins H, Midlo C (1926) Palmar and plantar epidermal configuration (dermatoglyphics) in Europian Americans. Am J Phys Anthropol 9: 471-502.
- 9. Furuhata T (1927) The difference of the index of fingerprints according to race. Jap Med World 7: 162-164 (in Japanese).
- Ravindranath R, Thomas IM (1995) Finger ridge count and finger print pattern in maturity onset diabetes mellitus. Ind J Phys Anthrop Hum Genet 49: 153-156.
- 11. Weinreb HJ (1986) Dermatoglyphic patterns in Alzheimer's disease. J Neurogenet 3: 233-246.
- Berr C, Okra-Podrabinek N, Feteanu D, Taurand S, Hervy MP, et al. (1992) Dermatoglyphic patterns in dementia of the Alzheimer type: a case-control study. J Epidemiol Community Health 46: 512-516.
- 13. Natekar PE, DeSouza EM, Motghare DD, Pandey AK (2006) Digital dermal patterns in carcinoma of breast. Anthropol 8: 251-254.
- Sridevi NS, Wilma Delphine Silvia CR, Kulkarni R, Seshagiri C (2010) Palmar dermatoglyphics in carcinoma breast of Indian women. Rom J Morphol Embryol 51: 547-550.
- 15. Boroffice RA (1978) Down's syndrome in Nigeria: Dermatoglyphic analysis of 50 cases. Niger Med J 8: 571-576.
- 16. Rodewald A, Zankl M, Zankl H, Zang KD (1980) Dermatoglyphs in carriers of a balanced 15;21 translocation. J Med Genet 17: 301-305.

- Bukelo MJ, Kanchan T, Unnikrishnan B, Rekha T, Ashoka B, et al. (2011) Study of finger print patterns in children with acute lymphoblastic leukemia. Forensic Sci Med Pathol 7: 21-25.
- Solhi H, Hashemieh M, Nejad ML, Vishteh HR, Nejad MR, et al (2010) Diagnostic value of fingerprint patterns: an explorative study on betathalassemia diagnosis. Bangladesh Med Res Counc Bull 36: 27-31.
- 19. Rosner F, Spriggs HA (1969) Dermatoglyphic studies in patients with Cooly's anemia. Annals New York Acad Sci 165: 378-386.
- Rashad MN, Mi MP, Rhoads G (1978) Dermatoglyphic studies of myocardial infarction patients. Hum Hered 28: 1-6.
- 21. Brijendra S, Renu G, Dushyant A, Rajneesh G, Sunil K, et al (2014) Dermatoglyphic's in congenital cardiac disease. Int J Biomed Res 5: 610-613.
- Simsek S, Taskiran H, Karakaya N, Fistik T, Solak M, et al. (1998) Dermatoglyphic analyses in children with cerebral palsy. Neurobiol 6: 373-380.
- Simpson NE, Newman BJ, Partington MW, Optiz JM (1984) Fragile-X syndrome: Dermatoglyphic studies in males. Am J Med Genet 17: 195-207.
- Barbeau A, Trudeau J-G, Coiteux C (1965) Fingerprint patterns in Huntington's Chorea and Parkinson's disease. Canad Med Ass J 92: 514-516.
- 25. Walker HA (1977) A dermatoglyphic study of autistic patients. J Autism Dev Disord 7: 11-21.
- Khan K, Jethani SL, Rohatgi RK, Goel D, Ali S, et al. (2014) Qualitative palmer dermatoglyphic patterns in cases of idiopathic generalized epilepsy. Int J Res Med Sci 2: 733-737.
- 27. Shibata K, Waldenmaier C, Hirsch W (1973) The clinical and genetic picture of trisomy 18 (Edwards' syndrome). Z Kinderheilk 116: 13-22.
- Okazaki T (2015) Relationship between fingerprint pattern and vein distribution. Bull Soc Sci Form 30: 2.
- 29. Bonnefond A, Saulnier P-J, Stathopoulou MG, Grarup N, Ndiaye NC, et al. (2013) What is the contribution of two genetic variants regulating VEGF levels to type 2 diabetes risk and to microvascular complications? PLoS ONE 8: e55921.
- Chiappelli M, Borroni B, Archetti S, Calabrese E, Corsi MM, et al. (2006) VEGF gene and phenotype relation with Alzheimer's disease and mild cognitive impairment. Rejuvenation Res 9: 485-493.
- 31. Lee ST, Chu K, Jung KH, Kim DH, Bahn JJ, et al. (2009) Reduced circulating angiogenic cells in Alzheimer disease. Neurol 72: 1858-1863.
- Evans TA, Raina AK, Delacourte A, Aprelikova O, Lee HG, et al. (2007) BRCA1 may modulate neuronal cell cycle re-entry in Alzheimer disease. Int J Med Sci 4: 140-145.
- 33. Saponaro C, Malfettone A, Ranieri G, Danza K, Simone G, et al. (2013) VEGF, HIF-1α expression and MVD as an angiogenic network in familial breast cancer. PLoS One 8: e53070.
- Grillo J, DelloRusso C, Lynch RC, Folkman J, Zaslavsky A, et al. (2011) Regulation of the angiogenesis inhibitor thrombospondin-1 by the breast cancer susceptibility gene-1 (BRCA1). Breast J 17: 434-435.
- 35. Silva JM, Gonzalez R, Provencio M, Dominguez G, Garcia JM, et al. (1999) Loss of heterozygosity in BRCA1 and BRCA2 markers and high-grade malignancy in breast cancer. Breast Cancer Res Treat 53: 9-17.
- 36. Qin L, Zhao D, Liu X, Nagy JA, Hoang MV, et al. (2006) Down syndrome candidate region 1 isoform 1 mediates angiogenesis through the calcineurin-NFAT pathway. Mol Cancer Res 4: 811-820.
- Baek K-H, Zaslavsky A, Lynch RC, Britt C, Okada Y, et al. (2009) Down syndrome suppression of tumor growth and the role of the calcineurin inhibitor DSCR1. Nat 459: 1126-1130.
- Pavlović-Kentera V, Stefanović S, Milenković P, Jancić M, Biljanović-Paunović L, et al. (1976) Erythropoietin level in patients with acute leukaemia. Haematologia (Budap) 10: 455-462.
- Paritpokee N, Wiwanitkit V, Bhokaisawan N, Boonchalermvichian C, Preechakas P, et al. (2002) Serum erythropoietin levels in pediatric patients with beta-thalassemia/hemoglobin E. Clin Lab, PMID: 12465748; 48: 631-634.

- 40. Namiuchi S, Kagaya Y, Ohta J, Shiba N, Sugi M, et al. (2005) High serum erythropoietin level is associated with smaller infarct size in patients with acute myocardial infarction who undergo successful primary percutaneous coronary intervention. J Am Coll Cardiol, PMID: 15862410, DOI: 10.1016/j.jacc.2005.01.043; 45: 1406-1412.
- 41. Gidding SS, Stockman JA, Goldwasser E (1987) Erythropoietin titers in children with cyanotic congenital heart disease. Pediatr Res 21:190.
- 42. Lin CY, Chang YC, Wang ST, Lee TY, LinCF, et al. (2010) Altered inflammatory responses in preterm children with cerebral palsy. Ann Neurop 68: 204-212.
- 43. Garnon J, Lachance C, Di Marco S, Hel Z, Marion D, et al. (2005) Fragile X-related protein FXR1P regulates proinflammatory cytokine tumor necrosis factor expression at the post-transcriptional level. J Biol Chem 280: 5750-5763.
- 44. Hsiao HY, Chiu FL, Chen CM, Wu YR, Chen HM, et al. (2014) Inhibition of soluble tumor necrosis factor is therapeutic in Huntington's disease. Hum Mol Genet 23: 4328-4344.
- 45. Zakareia FA, Al-Ayadhi LY, Al-Drees AA (2012) Study of dual angiogenic/neurogenic growth factors among Saudi autistic children and their correlation with the severity of this disorder. Neurosci 17: 213-218.
- 46. Masuda Y, Miura N, Kawarada Y, Kawagoe M, Shimizu T, et al. (1996) Platelet-derived growth factor B-chain homodimer suppressing a convulsion of epilepsy model mouse EI. Biochem Biophys Res Commun 223: 60-63.
- 47. Ribom D, Andræ J, Frielingsdorf M, Hartman M, Nistér M, et al. (2002) Prognostic value of platelet derived growth factor α receptor expression in grade 2 astrocytomas and oligoastrocytomas. J Neurol Neurosurge Psychiatry 72: 782-787.
- 48. Jauniaux E, Bao S, Eblen A, Li X, Lei ZM, et al. (2000) HCG concentration and receptor gene expression in placental tissue from trisomy 18 and 21. Mol Hum Reprod 6: 5-10.

- Hoeben A, Landuyt B, Highley MS, Wildiers H, Van Oosterom AT, et al. (2004) Vascular endothelial growth factor and angiogenesis. Parmacol Rev 56: 549-580.
- Vaughan EE, O'Brien T (2012) Isolation of circulating angiogenic cells. Methods Mol Biol 916: 351-356.
- 51. Kawai H, Li H, Chun P, Avraham S, Avraham HK, et al. (2002) Direct interaction between BRCA1 and the estrogen receptor regulates vascular endothelial growth factor (VEGF) transcription and secretion in breast cancer cells. Oncogene 21: 7730-7739.
- 52. Kertesz N, Wu J, Chen TH-P, SucovHM, Wu H (2004) The role of erythropoietin in regulating angiogenesis. Dev Biol 276: 101-110.
- Fajardo LF, Kwan HH, Kowalski J, Prionas SD, Allison AC, et al. (1992) Dual role of tumor necrosis factor-alpha in angiogenesis. Am J Pathol 140: 539-544.
- 54. Jing Y, Ma N, Fan T, Wang C, Bu X, et al. (2011) Tumor necrosis factoralpha promotes tumor growth by inducing vascular endothelial growth factor. Cancer Invest 29: 485-493.
- Mamer SB, Chen S, Weddell JC, Palasz A, Wittenkeller A, et al. (2017) Discovery of high-affinity PDGF-VEGFR Interactions: Redefining RTK dynamics. Sci Rep 7: 16439.
- 56. Tomaso Ed, London N, Fuja D, Logie J, Tyrrell JA, et al. (2009) PDGF-C induces maturation of blood vessels in a model of glioblastoma and attenuates the response to anti-VEGF treatment. PLoS One 4: e5123.
- 57. Sato N, Beitz JG, Kato J, Yamamoto M, Clark JW, et al. (1993) Plateletderived growth factor indirectly stimulates angiogenesis in vitro. Am J Pathol 142: 1119-1130.